DEVELOPMENTAL PHYSIOLOGY OF THE INFANT

- any abnormal physiological/pharmacological stress in,

a.	first trimester (8/52)	~ organogenesis → abnormal organogenesis
b.	second trimester	~ organ function development → abnormal organ function
c.	third trimester	~ weight gain, muscle/fat → smaller organs less muscle/fat mass

- such injuries and stress take the form of,
  a. congenital viral infections
  b. exposure to drugs/toxins
  c. nutritional deficiency - caloric or vascular
  d. other maternal illness

Birth Classifications

a.	preterm	≤ 37 weeks	
b.	term	37-42 weeks	
c.	post-term	≥ 43 weeks	
d.	low birth weight	≤ 2500 grams	§most premie's by definition
e.	small for gestational age	< 10th percentile	
f.	large for gestational age	< 90th percentile	

Gestation	Mean Weight
28	1050 g
32	1700 g
36§	2500 g
40 (term)	3400 g

- assessment of gestational age is by the Dubowitz scale
- subsequent development is assessed by percentile charts
- deviation from a percentile is of greater significance than any absolute value for an infant
- maternal diabetes is often associated with LGA infants
- this is due to the foetal insulin response to maternal hyperglycaemia

Problems Occurring with Increased Incidence

a. ***premature*** - RDS
- apnoea
- hypoglycaemia
- hypocalcaemia
- hypomagnesaemia
- hyperbilirubinaemia
- coagulopathy
- hypothermia

+ in SGA - viral infection
- thrombocytopaenia
- congenital anomalies
- maternal drug addiction
- neonatal asphyxia
- aspiration pneumonia

+ in LGA - transposition of great vessels
- maternal diabetes

b. ***term & post-term infant*** - none

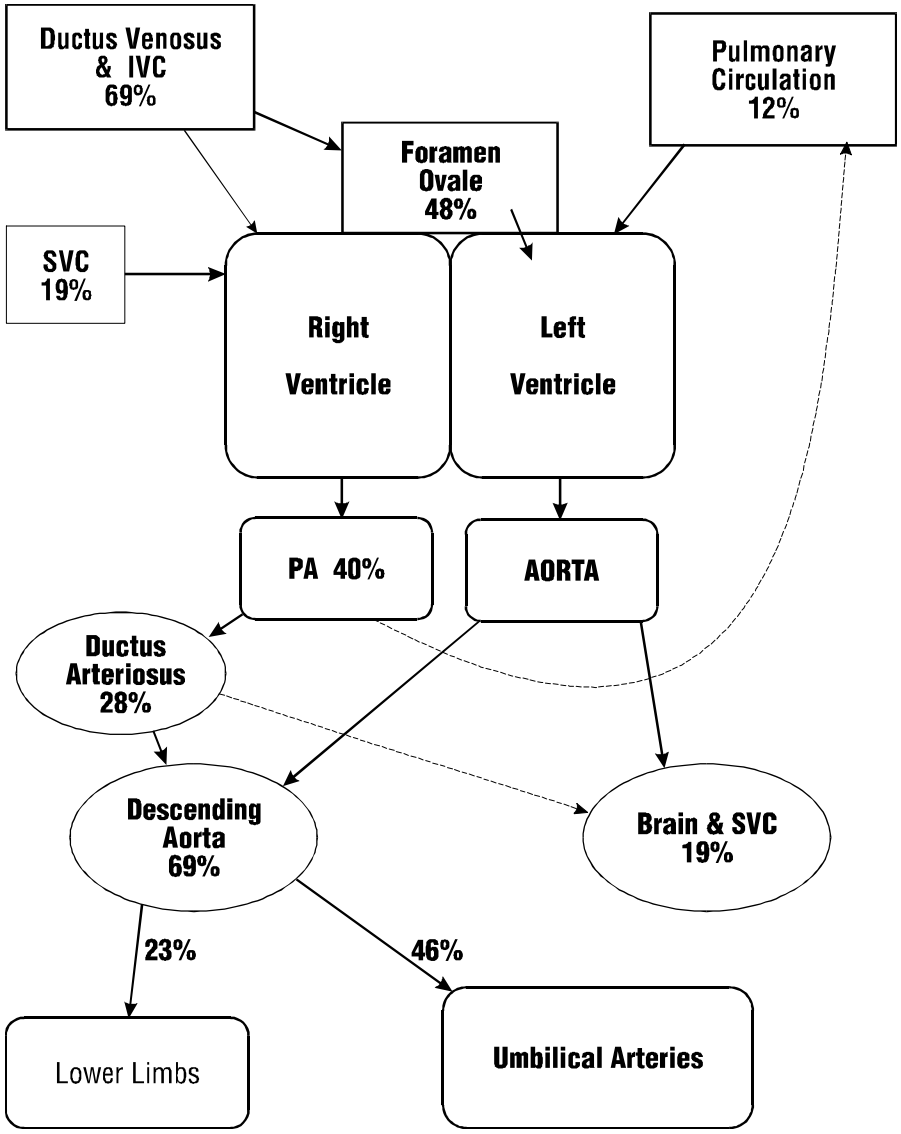
+ SGA infant - viral infection
- thrombocytopaenia
- congenital anomalies
- maternal drug addiction
- neonatal asphyxia
- aspiration pneumonia
- hypoglycaemia

+ LGA infant - birth trauma
- hyperbilirubinaemia
- hypoglycaemia (maternal diabetes)
- transposition of great vessels

Common Causes of Failure to Thrive

a.	genetic	- parental size - chromosomal disorders
b.	nutritional	- inadequate/inappropriate intake - malabsorption, diarrhoea, vomiting - cystic fibrosis - coeliac disease - carbohydrate intolerance - milk protein allergy
c.	malformations	- especially CVS & GUS
d.	infections	- pulmonary - renal - hepatic, enteral - congenital
e.	endocrine	- hypothyroidism - RTA
f.	preterm/SGA infants	
g.	malignancy	

Cardiovascular System



• in utero, most cardiac output is directed from the placenta, across the foramen ovale, into the ascending aorta (oxygenated blood)
• SVC (deoxygenated) blood is directed to both the pulmonary circulation and the ductus arteriosus

Circulatory Changes at Birth

• umbilical vessels have thick, muscular walls that are extremely reactive to trauma, tension, catecholamines, bradykinin, angiotensin and changes in PO_2
• closure of these vessels → increase in foetal TPR and BP
• when flow through the ***umbilical vein*** ceases, the ***ductus venosus*** closes by an unknown mechanism

→ ? ↓ flow & ↓ P_{aO2} to IVC levels

• asphyxia from the cessation of placental circulation and cooling of the body

→ activation of the respiratory centre of the newborn

• with inflation of the lungs, pulmonary vascular resistance falls to ~ $1/10^{th}$ of its intrauterine value
• this is ***not*** caused by the presence of O_2, as inflation with N_2 produces the same ↓ PVR
• however, ***alveolar hyperoxia*** does contribute to vessel dilatation
• PVR continues to fall for the next few years as the architecture of the pulmonary vessels changes
• the LA pressure rises above that of the RA and IVC due to,

a. decrease in pulmonary resistance → increased LA filling
b. ≤ 50% of LV output crosses the PDA, ↑'g pulmonary blood flow & LAP
c. decreased RA filling due to occlusion of the umbilical vein
d. increased LV afterload due to closure of the umbilical arteries

→ abrupt closure of the foramen ovale & fusion in several days

• pulmonary arterial pressure falls to ½ of its intrauterine value → ~ 35 mmHg
• this change, plus the increase in aortic pressure, reverses flow through the ***ductus arteriosus***
• however, within minutes the ductus begins to close producing turbulent flow

→ "physiological murmur of the newborn"

• closure of the ductus is usually complete 1-2 days after birth, and appears to be initiated by the ***raised*** **PaO_2**
• possible mediators being ***prostaglandins***, bradykinin, or adenosine
• successful completion does require ***muscular*** arterial tissue and the deficiency of this in the premature infant partly accounts for the high incidence of patent ductus in this group
• true mechanical closure by ***fibrosis*** does not occur until 2-3 weeks of age
• during this period the infant may revert to the foetal type circulation, so called ***transitional circulation of the newborn***
• factors precipitating transition back to foetal circulation include,

a. hypoxia, hypercarbia, acidosis
b. anaesthetic agent induced changes in PVR/SVR
c. pulmonary pathology - agenysis, dysgenesis
   - infection, collapse, consolidation
d. IPPV - especially non-compliant lungs

• risk factors for a prolonged period of ***transitional circulation*** include,

a. prematurity
b. hypothermia
c. infection, acidosis
d. pulmonary disease - hypercarbia, hypoxia, acidosis
   - meconium aspiration
e. congenital heart disease

Circulatory Changes at Birth

• the structure of the neonatal myocardium differs, in that the percentage cell mass devoted to contractility is significantly less and there are more ***connective tissue*** elements, resulting in,

a. leftward displacement of the cardiac function curve
b. decreased ventricular ***compliance***
c. increased tendency to ***biventricular failure***,
   i. poor tolerance of volume loading
   ii. sensitivity to increased afterload
   iii. CO that is heart ***rate dependent***

NB: myocytes also contain fewer contractile elements cf. other organelles, due to the requirements for growth and cell division

• the myocardium has sparse sympathetic innervation and ***vagal*** influences predominate
• normal parameters in the neonate are,

a. HR ~ 120 ± 20 bpm
b. BP ~ 70/45 mmHg

• the CO is higher as a percentage body weight, with the majority directed to the vessel rich group
• normal ***baroreceptor function*** is abolished under anaesthesia

NB: thus, the neonate adapts poorly to hypovolaemia and BP under anaesthesia is a good guide to volume status

• ***hypoxaemia*** leads to bradycardia and vasoconstriction

• at birth, the two ventricles are about the same weight, having been pumping in parallel in the foetal circuit
• thus the ECG show ***right axis deviation***
• the arterioles of the pulmonary circuit are thick and muscular, maintaining the high pulmonary vascular resistance during foetal life→ - ***pulmonary vascular reactivity***
• after birth, the RV fails to grow to the same extent as the LV, the later becoming predominant and the muscular layer of the pulmonary vessels is lost
• these changes take several weeks

Respiratory Changes at Birth

• the pulmonary system is unable to sustain life until both airway & vascular elements have matured sufficiently **~ 24-26 weeks**
• due to the rapid development of alveoli, the lungs continue to grow up to the age of ~ 8 years
• subsequent growth is in size of alveoli/airways only

Element	Appearance	Maturation
bronchi	16/52	~ 23/52
alveoli	17/52	post-partum
surfactant	24/52*	~ 36/52

* composition is different and production is unstable until 36/52
L/S ratio increases to 2:1 at term
production is ***decreased*** with stress, hypoxia, acidosis, etc.

• the airways and terminal air spaces are filled with ~ 100-200 ml of fluid, which is secreted by the lung and contains mucopolysaccharides and proteins in addition to surfactant
• some of this is expelled during passage through the birth canal, an advantage lost at LUSCS, where the infant frequently has ***transient tachypnoea***
• with the first breath the remainder fluid is drawn down the tracheobronchial tree
• stimulus to first breath includes circulatory changes, (raised TPR), and physical stimuli such as cold, pain, voices, etc.
• with the first gasps against the low compliance, lung **P_{IP} £ -60 cmH_2O**
• however this rapidly decreases as the lung expands and compliance increases

• ***blood gases*** evolve rapidly in the newborn infant,

a. hypoxaemia - corrected by 5 mins
~ 75 mmHg at 5 hours

b. hypercapnia - corrected by 20 minutes
~ 33 mmHg at day 1 & 36 mmHg day 7

c. acidaemia → mixed metabolic & respiratory
~ 24 hours

• ***alveoli*** increase in number,

a. ~ 27 million at birth

b. ~ 300 million by 8 yrs

NB: ~ 10x increase
~ 1 alveolus / second for 8 years

Respiratory Changes at Birth				
Normal Values	At Birth		Adult	
Respiratory Rate	**30-40**	**bpm**	15	bpm
Tidal Volume, TV	7.0	ml/kg (~ 20 ml)	same	(~ 500 ml)
Minute Volume, V_M	**230**	**ml/kg/min**	70	ml/kg/min
Vital Capacity, VC	40	ml/kg	50-60	ml/kg
FRC	27-30	ml/kg	30	ml/kg
Physiological V_D/V_T	0.3-0.5		0.3	
Physiological Q_S/Q_T	**0.1**	(10%)	0.01-0.03	(1-3%)
Lung Compliance, Specific	0.067 67	l/cmH_2O/l ml/cmH_2O/l	same	
Lung Compliance, Absolute	**0.005** **5**	**l/cmH_2O** **ml/cmH_2O** ~ 1/20th adult	0.100 100	l/cmH_2O ml/cmH_2O
Compliance, chest wall	**0.26** **260**	**l/cmH_2O/l** **ml/cmH_2O/l** ~ 5x adult	0.06 60	l/cmH_2O/l ml/cmH_2O/l
Total Pulmonary Resistance	**30-50**	**cmH_2O/l/s** ~ 10x adult	4-5	cmH_2O/l/s
Mean Time Constant (tau)	0.12 s		0.5 s	
PaO_2 (NB: Q_S/Q_T)	65-80	mmHg	98	mmHg
$PaCO_2$	34	mmHg	40	mmHg
O_2 consumption	**7.0** (thermoneutral)	**ml/kg/min**	3.5	ml/kg/min
Airways Resistance:	• high, proportional to $1/r^4$ • obligate nose breather			
Compliance, respiratory:	• similar in infants/adults → ***increased work of breathing***			

Respiratory Changes at Birth

- compliance and airways resistance determine both,
    i. the work of breathing, and
    ii. the resting lung volumes

- the higher the compliance of the chest wall, the lower will be the FRC
- however, volumes measured *in vivo* show that ***FRC ~ 30 ml/kg*** in both infants & adults
- when measured as a percentage of TLC this is actually greater, as infants TLC is less / kg
- this measured increase in expected FRC is due to,
    a. increased intercostal muscle tone during expiration
    b. "laryngeal braking", increasing airways resistance by vocal cord adduction
    c. gas trapping as ***closing volume*** is above FRC

        → increased V/Q mismatch and ***increased shunt***

Respiratory Mechanics

- thus, a number of factors make respiration less efficient in the neonate,
    a. small airway diameter $R \sim 1/r^4$
    b. compliant airways & increased narrowing 2° venturi (Bernoulli) effect
    c. highly compliant/flexible airways & chest wall
        i. functional airway closure
        ii. inability to sustain a high negative P_{IP}
    d. similar dead space but ~ 2-3x the MRO_2 of adults
    e. unequal ventilation and perfusion
    f. less type I muscle fibre → less resistant to fatigue

- despite the narrow airway diameter, most resistance still resides in the upper respiratory tract and is easily influenced by posture etc.
- ~ 25% of the total resistance is in the nasal passages, cf. 60% in the adult
- in the premature infant the ***work of breathing* ~ 3x** that of adults and this can further be increased by hypothermia or partial airway obstruction
- the distribution of ventilation and perfusion improves,
    a. as the infant loses water from the lung
    b. as the lungs mature

- 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
- the composition of the diaphragmatic and intercostal muscles also differs

• ***type I fibres***, which generate ATP by oxidative phosphorylation, are fatigue resistant and comprise,

a. in the neonate ~ 25% diaphragm
~ 46% intercostal

b. in the adult ~ 60% in both

• the greater percentage of fast contracting, type II fibres in the neonate are better suited to meeting the rapid respiratory rates
• however, these are more prone to fatigue under conditions of increased load
• ***work*** of breathing is given by the volume of gas moved against respiratory compliance, and the work to over come resistance to airflow,

$$W = V/C + R.F$$

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

a. deficient surfactant

b. increased discrepancy between ventilation/perfusion

c. compliance ~ 0.00025-0.001 l/cmH_2O ↓ 5-20x

d. resistance ~ 100-250 cmH_2O/l/s ↑ 5-10x

e. increased work of breathing

f. propensity to pneumothorax / barotrauma

Respiratory Control Centres

• in general, during infancy, central responsiveness to,

a. stimulatory inputs increases - hypoxia
- hypercarbia
- acidosis

b. inhibitory inputs decreases - chest wall deformation
- laryngeal stimulation

• newborns have a ***biphasic*** response to ***hypoxia***
• breathing a hypoxic gas with arterial PaO_2 ~ 50 mmHg,

a. 1st minute ~ 30% increase in V_M

b. ~ 2-3 minutes ~ 30% decrease in V_M below baseline
- may progress to ***apnoea*** in some

• this appears to result from an inhibitory process in the central integrating area, and ***does not*** result from,

a. peripheral chemoreceptive adaptation

b. respiratory muscle fatigue

- this response also depends upon the thermal environment
- the ***hypothermic*** neonate responds only with respiratory ***depression***
- adults subject to the same conditions increase V_M ~ 30% but sustain the increase
- 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***

- afferent inputs from the larynx (superior laryngeal n.) reach the brainstem and are potent inhibitors of respiration
- even small quantities of fluid in the larynx may cause prolonged apnoea
- as the child matures this reflex changes character, in adults, resulting in coughing/swallowing
- chest wall distortion, (eg. 2° respiratory obstruction), may also result in prolonged apnoea
- in the older infants this results in only brief respiratory pause,

→ both the ***incidence*** & ***duration*** of apnoea decrease during infancy

- in young infants, the increased,

a. ***incidence*** relates mainly to the increased sensitivity to inhibitory inputs that trigger apnoea

b. ***duration*** relates to the decreased central responsiveness to stimulatory afferents which promote recovery from apnoea

The Apgar Scoring System

Score	0	1	2
HR	absent	< 100	> 100
Respiration	absent	irregular, shallow	good, crying
Reflex irritability	no response	grimace	cough, sneeze
Muscle tone	flaccid	good tone	spontaneous flexion
Colour	blue or pale	body pink / extremities blue	entirely pink

Anaesthetic Considerations - Respiratory

- anaesthesia alters respiratory mechanics primarily by,
    a. decreasing muscle tone in the airway, chest wall & diaphragm
    b. decreasing CNS hypoxic / hypercapnic responses

- studies indicate a higher incidence of hypoxaemia & airway obstruction in infants cf. children
- 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***

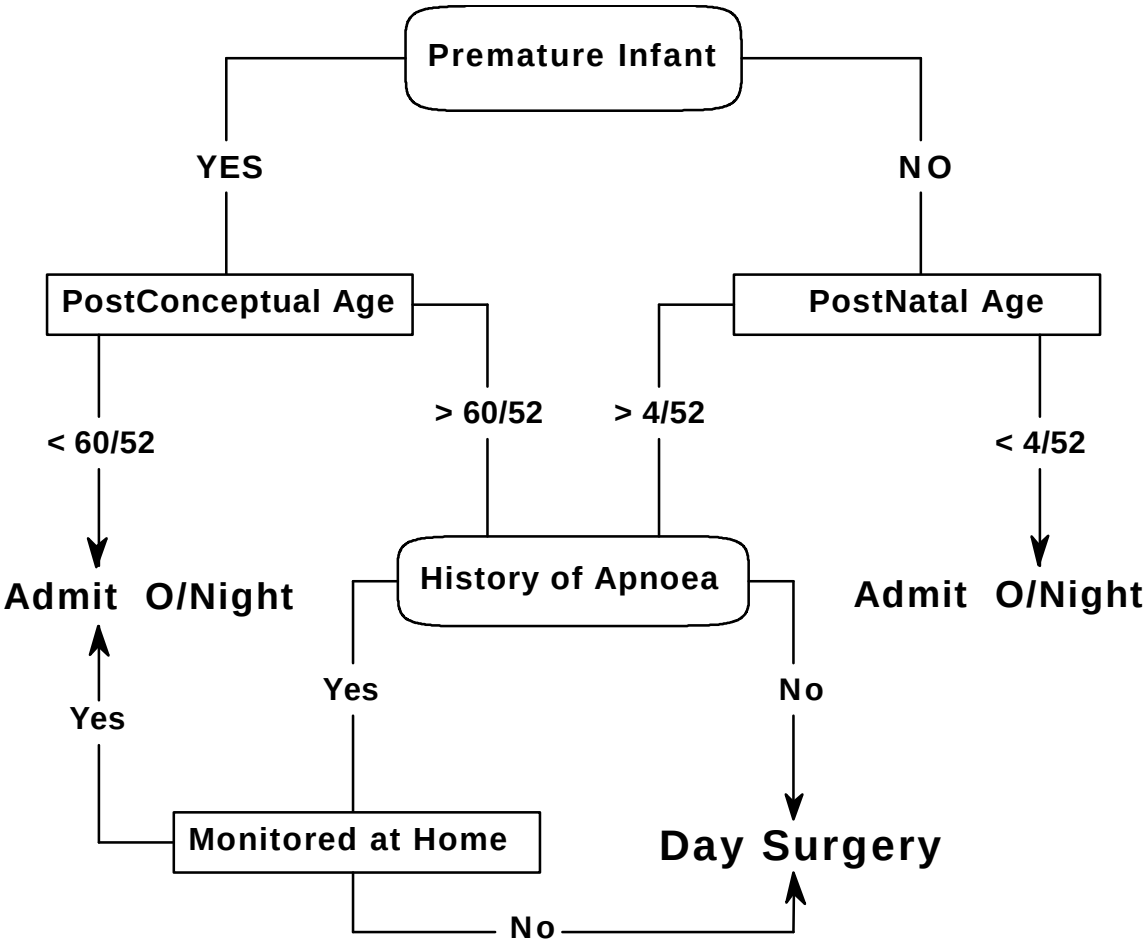
- Cohen (1990) 5 year review of intra/postoperative respiratory complications,
    a. most were ***obstructive*** in nature
        - croup
        - laryngospasm
        - bronchospasm
        - airway obstruction
        - apnoea
    b. frequency decreased with increasing age
    c. incidence in ***neonates*** ~ 4-5x that of 1-5 yr group

- recommendations for neonate/young infant,
    i. set time sampling interval on oximeter to 2-3 cycles
    ii. intubate all infants ≤ 1 year unless procedure is very brief
    iii. pre-O_2 for 2-3 minutes prior to laryngoscopy
    iv. use the pulse oximeter to limit the duration of laryngoscopy
    v. ?? assist ventilation during induction/emergence
    vi. control ventilation during maintenance (preserves FRC)

- 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,
    i. ***brief*** ~ 5-15 s
    ii. ***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

Selection Criterea for Day Surgery



1. was the infant was premature, ie. ≤ 37/52 gestation and/or < 2500g birthweight
2. if the infant is > 60/52 postconception and not on a cardiorespiratory monitor at home, then they may be considered for day surgery
3. all infants < 6 months of age are monitored continuously in the recovery room for 2 hours, alarms being set for,
   i. apnoea > 15 secs
   ii. bradycardia < 90 bpm

• the incidence of postoperative apnoea may be reduced by the administration of ***caffeine 10mg/kg*** IV after induction of anaesthesia
• aminophylline ~ 1.0 mg/kg is also effective
• the incidence of postoperative apnoea ***may*** be less with ***spinal anaesthesia*** cf. general anaesthesia

Intubation

• the larynx is composed of a series of cartilages, suspended from the base of the skull by ligaments,

a. hyoid

b. thyroid

c. cricoid - articulates post. with inf. cornu of the thyroid c.

d. arytenoid - paired, triangular, articulate with sup/post. aspect of the cricoid
- protected by the thyroid c.

e. the laryngeal tissue folds are,

i. paired aryepiglottic folds - epiglottis to sup. arytenoid

ii. paired vestibular folds - thyroid c. to sup. arytenoid
= *"the false cords"*

f. paired vocal cords - thyroid plate to arytenoids

g. interaytenoid fold - posteriorly

h. thyrohyoid fold - hyoid to thyroid c.

• ***sensory*** innervation of the larynx is from,

i. ***internal*** branch, sup. laryngeal nerve

ii. recurrent laryngeal nn.

• ***motor*** innervation of the larynx is from,

i. ***external*** branch, sup. laryngeal nerve → cricothyroidei

ii. recurrent laryngeal nn. - ***all*** except the cricothyroidei

• the major differences which make the neonate potentially 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, therefore the tube easily dislodged, or positioned in right main bronchus, especially with head movement

• relatively large nose → nasal and oropharyngeal airways ~ the same diameter

Spontaneous Respiration

- the infant tends to be an ***obligate nose breather*** due to,
    a. immaturity of coordination between respiration and oropharyngeal muscles
    b. the larger tongue rests against the roof, which is much shorter, of the mouth during quiet respiration
    c. the rostrally located larynx decreases the distance between tongue, hyoid and epiglottis

    NB: these tend to disappear by ***3-5 months***

- thus factors which result in ***nasal obstruction*** may significantly compromise respiration, eg. choanal atresia, stenosis or nasal congestion → ***oropharyngeal airway***
- the larynx, trachea and bronchi are more compliant and as such more subject to normal compressive and distensive forces
- for the trachea, these forces differ for the intra-thoracic and extra-thoracic portions
- during ***inspiration***, intrathoracic pressure becomes more negative and,
    i. the intrathoracic trachea dilates and stretches
    ii. the extrathoracic trachea is slightly narrowed & elongated
        - patency is maintained by muscles & soft tissues of the neck
    iii. increased separation of the vocal and vestibular folds of the larynx ≡ elongation (insp. stridor of laryngospasm)

- during passive ***expiration*** intrathoracic pressure remains negative, maintaining the patency of the bronchial tree & intrathoracic trachea
- during forced glottic closure, there is also contraction of the laryngeal muscles, which results in,
    i. a marked reduction of the interarytenoid distance
    ii. anteromedial movement of the arytenoids with ***apposition*** of the cords
    iii. longitudinal shortening of the larynx → obliteration of the space between the aryepiglottic, vestibular and vocal folds
    iv. contraction of the thyrohyoid muscle, leading to further closure

- ***extrathoracic*** upper airway obstruction, such as occurs with foreign body, croup or epiglottitis, alters the normal balance,
    i. dilatation of the intrathoracic airways
    ii. dynamic closure of the extrathoracic airways below the level of the obstruction
        → greatest at the ***thoracic inlet***

- ***intrathoracic*** upper airway obstruction, such as occurs with foreign body or a vascular ring
    → stridor on both inspiration & expiration
- in lower airway obstruction, such as asthma or bronchiolitis, significant dynamic intrathoracic obstruction occurs with further prolongation of the expiratory phase and gas trapping
- these dynamic changes are exacerbated during crying or forced breathing, therefore efforts to calm the child are important
- however, the use of sedative or narcotics prior to intubation, decreases voluntary effort and may worsen obstruction

Mechanical Ventilation

• most neonates breathe at 30-60 bpm, with an I:E ratio of ~ 1:1
• 5x the average time constant being ~ 0.6 sec
• thus when 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***
• also, volume cycled ventilators function poorly at high respiratory rates
• 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:FiO_2 is proportional to the mean airway pressure
• however, at some point this becomes excessive and is detrimental to oxygenation (analogous to "best-PEEP")
• centres vary, however, approximate guidelines for ventilation 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*** reduced

• PEEP increases mean P_{AW} and improves FRC at low lung volumes
• excessive PEEP is detrimental on pulmonary blood flow, CO, and V/Q mismatch
• increasing PEEP without increasing the peak P_{AW} decreases the tidal volume & minute ventilation
• at rapid respiratory rates (> 60 bpm) significant gas trapping occurs
• minute ventilation reaches a plateau, then decreases as the rate exceeds ~ 75 bpm on some ventilators
• 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. bronchopulmonary dysplasia
b. retrolental fibroplasia

• the aim should be to reduce the FiO_2 to ≤ 0.6 ASAP

• there are few studies on the effects of gas flow rates
• clinical experience suggests that there is an increased incidence of barotrauma associated with high initial gas flows
• weaning is facilitated by the use of intermittent mandatory ventilation
• this improves pulmonary blood flow by generating a "negative" intrathoracic pressure
• the general aims of weaning should be to,

a. reduce FiO_2 ≤ 0.6 prior to other reductions
b. reduce peak P_{insp} ≤ 20 cmH_2O
c. reduce the IMV rate
d. reduce 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

• in fact, these forms of ventilation are 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

• 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

Renal Changes

Normal Values	**Neonate**	**Adult**
GFR • ***premature*** • at birth • at 1 month	10-20 ml/min/m^2 0.7-0.8 ml/min/m^2 1-2 ml/min/m^2 50 ml/min/m^2	60-80 ml/min/m^2 (70kg → 1.7m^2)
Maximum Urine Concentration	450-600 mosmol/l	1400 mosmol/l
Plasma Creatinine	• maternal at birth[1] • infant ~ 18-35 µmol/l • child ~ 30-60 µmol/l • youth ~ 45-90 µmol/l	• male ~ 55-120 µmol/l • female ~ 45-95 µmol/l • pregnant ~ 30-80 µmol/l
pH	7.35	7.4
[HCO_3^-]	20 mmol/l	25 mmol/l

[1] decreases due to low muscle mass and high rate of anabolism

• the newborn has the same number of nephrons as the adult, and similar to the liver, the organ mass as a percent of body weight is ~ 2x the adult
• however, renal function in the neonate is markedly diminished due to,

a. low perfusion pressure
b. immaturity of glomerular & tubular function

• the glomeruli mature in utero considerably at ***~ 34-36 weeks***
• GFR in infants born ***prior*** to this age matures at a much slower rate than the term infant
• at birth GFR ~ 2-4 ml/min (term) but may be as low as 0.7-0.8 ml/min/m^2 (prem)
• this increases after the first few days to 8-20 & 1-2 ml/min/m^2 respectively
• by the end of ***1 month*** renal function is ~ 80-90% mature
• maturation is nearly complete by ~ 8-9 months

• drugs eliminated by GFR include,

a. long acting muscle relaxants
b. the aminoglycosides
c. H_2-receptor antagonists
d. digoxin

• ***tubular function*** is also immature and maturation lags slightly behind that of GFR, reaching adult values by ~ 35 weeks
• this is reflected in the lower tubular transport rates for,

a. bicarbonate, glucose, phosphate

b. xenobiotics - penicillin
- sulphonamides

c. digoxin

NB: thus, the ***physiological acidaemia*** of the newborn reflects the lower tubular threshold for ***bicarbonate*** → "effective RTA"

• the tubular threshold for ***glucose*** is lower in the neonate, such that rises in plasma glucose may lead to glycosuria and ***osmotic diuresis***
• ***urea*** excretion is always low due to protein anabolism, and this contributes to the decreased ***concentrating ability*** of the neonatal kidney
• there is limited excretion/conservation capability for salt, water and acid-base alterations
• the newborn is better able to tolerate moderate fluid overload than ***dehydration***, such as from prolonged preoperative fasting or vomiting
• RBF may be compromised by increases in intra-abdominal pressure, eg. post omphalocele repair, and renal failure may ensue
• growth retardation may be associated with a significantly malfunctioning kidney
• such children may be below the third percentile for age, however the reasons for this are unclear

Hepatic Function

• all metabolic functions are dependent upon adequate ***hepatic blood flow***
• in the neonate this may be reduced by,

1. low perfusion pressure
2. shunting via the ductus venosus
3. raised intra-abdominal pressure

• most enzyme systems for drug metabolism are developed but not yet induced
• with growth hepatic function increases in two ways,

a. hepatic blood flow is increased
b. resident enzyme systems are induced

• at birth the liver ~ 4% of body weight, cf. 2% in the adult, thus as blood flow and enzyme systems increase metabolic activity increases above adult levels
• ***conjugation reactions*** are frequently impaired in the neonate

→ bilirubin, sulphonamides, chloramphenacol, paracetamol & meprobamate, etc.

• therefore, most metabolism is via ***phase I reactions***
• of the phase I reactions ***N-demethylation*** appears to be the most deficient, as in theophylline and caffeine metabolism
• hydroxylation reactions are reduced to a lesser extent, and are more inducible → phenobarbital, mepivacaine, lignocaine, nortriptyline
• unconjugated ***hyperbilirubinaemia*** is common in the first 2 weeks,

a. term infants ≤ 105 mmol/l (6 mg/dl)
b. prem infants ~ 170-255 mmol/l (10-15 mg/dl)

NB: ***CNS damage*** may be sustain at: preterm infant ≥ 100-150 mmol/l
term infant ~ 350 mmol/l

• this increased risk of ***kernicterus*** is exacerbated by,

a. immature BBB
b. hypoxia, acidosis, hypothermia, hypoalbuminaemia
c. displacement by drugs
   - diazepam
   - sulphonamides
   - frusemide

NB: R_x phototherapy, exchange transfusion

• reduced activity of degredation reactions leads to prolonged drug plasma half lives
• it is common to have longer drug elimination half lives in the neonate and shorter in the infant and small child, cf. the adult
• ***glycogen synthesis*** begins at ~ 14/40, however most of the hepatic stores are laid down in the last 2 weeks of gestation

• thus, the premature neonatal liver has minimal glycogen stores and is unable to handle large ***protein loads***, which predisposes to,

1. hypoglycaemia - commonly stressed / SGA neonate, or maternal IDDM
   ≤ 2.2 mmol/l R_x 10% dextrose ~ 5 mg/kg/min
   ~ 50% of symptomatic → neurological damage
2. acidaemia
3. failure to gain weight with high dietary protein

• infants of diabetic mothers are prone to rebound hyperinsulinaemia & subsequent hypoglycaemia, therefore dextrose should be administered slowly
• plasma levels of albumin, prothrombin & other carrier proteins are lower, especially in the premature neonate, associated with,

a. neonatal ***coagulopathy*** & requirement for vitamin K
b. higher plasma free drug fractions & altered pharmacokinetics

Factors in Neonatal Jaundice
• Excess bilirubin production ∝ RBC breakdown
• Impaired liver uptake of bilirubin
• Impaired conjugation of bilirubin
• Defective bilirubin excretion
• Increased enterohepatic circulation of bilirubin

Pathological Causes of Jaundice in the Neonate	
Antibody induced haemolysis	• ABO & Rh
Hereditary RBC disorders	• G6PD
Infections	• neonatal hepatitis • generalised sepsis • severe UTI
Internal haemorrhage	• ICH • retroperitoneal
Biliary atresia	
Metabolic causes	• hypothyroidism • galactosaemia

Gastrointestinal System

• gastric pH at birth is ~ 7.4, ie. ***alkalotic***
• levels of enterokinase and lipase steadily increase throughout gestation, but are lower at birth than in the older child
• thus the preterm, and to a lesser extent the term infant, are unable to handle large protein or fat loads
• acid production begins shortly thereafter, reaching "normal" pH by 2-3 days
• the ability to co-ordinate respiration and swallowing does not mature until 4-5 months of age
• peristalsis is absent from the distal oesophagus in normal infants and "normal" LOS pressures are not reached until 3-6 weeks
• developmental abnormalities of the GIT usually manifest within 24-36 hrs,

a. upper GIT → regurgitation/vomiting
b. lower GIT → distension & failure to pass meconium

• these anomalies include,

a. oesophageal atresia and tracheo-oesophageal fistula
b. intestinal atresia and stenosis (esp. duodenal)
c. duplication and diverticulum
d. Hirschprung's disease
e. peritoneal bands = malrotation, often duodenojejunal
f. omphalocele & gastroschisis

Meconium

• consists of desquamated epithelial cells, bile, pancreatic and intestinal secretions, and water
• usually passed within the first few hours after birth but may be delayed for up to 48 hours
• its presence in the amniotic fluid usually indicated ***intrauterine asphyxia***
• ***aspiration*** may result in,

a. pneumonia
b. pneumothorax
c. transitional circulation of the newborn
d. respiratory failure

• ***meconium ileus*** is associated with cystic fibrosis (~ 10%) and with Hirschprung's disease

→ ***inspissation & obstruction***

Calcium Metabolism

- Ca^{++} is actively transported across the placenta to meet the requirements of the growing foetus
- transport accelerates near term
- neonatal parathyroid function is immature & vitamin D stores inadequate
- therefore, ***hypocalcaemia*** is prone to occur with,

a. premature / SGA infant

b. birth trauma

c. neonatal asphyxia, severe illness

d. blood transfusion - citrated WB or FFP

e. correction of metabolic acidosis with $NaHCO_3$

f. renal failure

NB: ***normal range*** Ca^{++} ~ 1.7 mmol/l total
Ca^{++} ~ 1.0 mmol/l ionized

R_x Ca-gluconate 10% ~ Ca^{++} 60 mg/kg/day

Pancreatic Function

- the placenta is ***impermeable*** to both insulin and glucagon
- islets of Langerhans secrete insulin from the 11^{th} week, increasing with age
- maternal hyperglycaemia results in islet cell hyperplasia with subsequent derangement of lipid metabolism
- this, and possibly the increase in maternal amino acids, results in the characteristic large infants of diabetic mothers
- ***hyperinsulinaemia*** will persist beyond birth and may result in ***rebound hypoglycaemia***

- ***SGA infants*** are frequently hypoglycaemic

a. ? 2° in utero malnutrition

b. inappropriate insulin secretion / glucose level

c. deficient liver glycogen stores

d. deficient gluconeogenesis

- abnormal fasting BSL's during the first 3 days are,

a.	term infant	≤ 1.95mmol/l	35 g/dl
b.	preterm	≤ 1.4 mmol/l	25 g/dl
c.	after 3/7	≤ 2.5 mmol/l	45 g/dl

Haemopoietic System

- the blood volume of the infant is determined by the time of cord clamping,
    a. immediate ~ 80 ml/kg
    b. delayed ~ 93 ml/kg
    c. preterm ~ 100 ml/kg

- within the first hour plasma volume contracts → ***haemoconcentration***
- this effect is greater with a greater initial blood volume
- the normal ***Hb* ~ 15-20 g/dl**, capillary (heel-stick) values may be higher by as much as 6 g/dl
- erythropoietic activity decreases immediately after birth
- initial reticulocyte count ~ 5% decreases to < 1%
- returns to ~ 1-2% by the 12^{th} week, where it remains during childhood
- Hb reaches a nadir → ***10^{th} week ~ 10 g/dl***
- in premature infants the fall is greater and occurs earlier, ~ 4-8/52 and levels ~ 7-10 g/dl

- ***vitamin E*** is essential for RBC membrane integrity
- premature infants (esp. < 1500g) are deficient and may develop ***haemolytic anaemia*** at 6-10 weeks of age
- the WBC count may reach ~ 21,000 after birth and 12,000/mm^3 at week 1
- increased susceptibility to ***sepsis*** in part relates to decreased leukocyte function
- infection may be associated with minimal response or even leukopaenia

Coagulation

- vitamin K dependent factors are ~ 20-60% of adult levels and less in premature infants
- thus, prolonged prothrombin times seen in infants are normal
- even with administration of vitamin K, liver synthesis is decreased and does not reach adult values for several weeks
- K_1 administration at birth is essential to prevent haemorrhagic disease of the newborn
- this effect is exacerbated with maternal anticonvulsant therapy & K_1 deficiency

- English study showed increased incidence of childhood cancer with IM administration of K_1
- therefore now recommended to be given orally, however there is no such preparation & most are giving 3 oral doses of Konakion

Central Nervous System

• nociceptive pathways and anatomic substrates for pain transmission are present in the neonate and although ***myelination*** is incomplete, the senses are active
• with growth & development during childhood there is further refinement of sensory modalities and intracortical connections
• as recently as 1976 Lippmann *et al.* wrote, with regard to surgical repair of PDA, "anaesthetic or analgesic agents in our experience are unnecessary"
• a review article in the Gauntlet (1987) reported that, in response to survey, only 85% of anaesthetists believed that newborns perceived pain

• anaesthetic agents appear to act via the same mechanisms as for adults, however, in general drug requirements are less in the neonate
• these differences relate to possible immaturity of the BBB and altered conformation of specific drug receptor sites
• the retina of the preterm infant is easily damaged by exposure to high PaO_2, especially

a. infants ≤ 32 weeks PCA

b. infants ≤ 1600 gms

NB: maintaining SpO_2 at ~ 90-95% is generally considered safe, however, SpO_2 is a poor monitor of ***hyperoxia*** therefore, better to maintain a P_{aO2} ~ 50-70 mmHg

Causes of Mental Handicap	
Infections	• meningitis, encephalitis • TORCH
Head injury	
Hypoxaemia	• birth asphyxia • near drowning, CO poisoning
Metabolic	• hypoglycaemia, hypernatraemia • chronic malnutrition • inborn errors of metabolism • hypothyroidism
Toxic	• lead poisoning • addictive drugs
Degenerative CNS disease	
Cerebral tumour, CVA	
Congenital malformations	

Temperature Regulation

= the balance between heat production and heat exchange

a. ***production***
   i. MRO_2
   ii. voluntary activity
   iii. non-shivering thermogenesis
   iv. shivering

b. ***exchange***
   i. radiation **~ 40%**
      - ***major form*** of loss normally
      - all objects > zero kelvin and mainly IR from humans
      - net balance of emitted/absorbed
      - ∴use radiant lamps, heated surroundings, reflective coverings

   ii. convection **~ 30%**
      - high turnover in O/T increases conductive & evaporative losses to air
      - static air = effective insulator
      - ∴use blankets, drapes, etc.

   iii. evaporation **~ 29%** (higher in infants cf. i & ii.)
      - ∝ latent heat of vaporization ~ 0.58 Cal/g H_2O (580 kcal/kg)
      - ~ 20-30% of normal heat loss
      - ~ 8-10% of MRO_2 in a 3.0 kg intubated infant is used to humidify dry gases
      - ∴use heat/moisture exchangers or humidifiers

   iv. conduction **~ 1%**
      - ∝ δT skin-contacts air
      - effective ~ 0.5 m^2 SA size
      - up to 60% conductive loss from uncovered infants head
      - ∴use warming blankets, warm irrigation solutions, thermal insulators

NB: respiratory losses ~ 10%

   i. humidification ~ 8%
   ii. convection ~ 2%

- ***Deficits in Regulation:***

a.	minimal hypothalamic control of	- cutaneous vasomotor tone - sweating
b.	high SA/weight ratio	~ 2x adult
c.	high evaporative losses	- high RR & MV - thinner skin, more H_2O permeable
d.	inability to shiver effectively	
e.	inability to take evasive action	

- ***Gains in Regulation:***

NB: "brown fat"

i. heat 2° to uncoupling of oxidative phosphorylation & increased mitochondria
ii. neck, back, axillae, inguinal, perinephric
iii. ~ 2-3% of body mass in a term neonate
iv. mediated by NA on α & β-receptors
v. ~ 25% of the CO may be directed to brown fat
vi. requires an increased MRO_2 ~ 60%

• the increase in NA will also increase PVR, tending to increase right to left shunting through the foramen ovale and the ductus arteriosus
• peripheral vasoconstriction results in the mottled appearance of the skin
• neither neonates, nor adults can temperature regulate via white fat

→ neonates must be kept in ***thermoneutral zone***

i.	naked	~ 32-35 °C	
ii.	clothed	~ 24 °C	
iii.	premature infants	~ 36-36.5°C	***skin temperature***

• ***hypothermia*** will markedly increase the MRO_2 of the newborn infant
• a fall of only 2°C may result in a 2 fold increase

NB: Coté states ~ 12% δBMR / °C

Monitoring

a. ***central***

i.	nasopharyngeal	~ internal carotid artery - easily dislodged - cooling via ET tube
ii.	tympanic membrane	- good correlation - potential for damage to TM - errors from wax etc.
iii.	oesophageal	- lower third of oesophagus - less prone to movement ~ heart/aortic arch

b. ***peripheral***

i.	axillary	
ii.	rectal	- altered by rectal contents - affected during cystoscopy
iii.	forehead	~ 1-2°C < core with vasoconstriction
iv.	skin	- digital/trunk

PHARMACOKINETICS & PHARMACODYNAMICS

Absorption - Oral

- several factors alter oral bioavailability in the newborn period,
    a. slow gastric emptying and irregular peristalsis
    b. relative ***achlorhydria*** during the first few days
        - decreased absorption of acidic drugs
        - increased absorption of basic drugs
    c. variability in splanchnic blood flow
    d. gradual maturation of biliary function
    e. variable colonisation of the GIT with bacteria
    f. high levels of b-***glucuronidase*** (~ 7x adult)
        - ? increased enterohepatic circulation of morphine
        - may contribute to neonatal jaundice

    NB: beyond the neonatal period, no significant differences exist which would alter oral absorption cf. the adult

Parenteral

- IM absorption depends upon regional blood flow which may vary in the child
- cf. oral, absorption of the following are ***decreased*** when given IM,
    a. diazepam
    b. digoxin
    c. gentamicin

Percutaneous

- absorption is significantly higher in newborns & children cf. adults
- this relates to skin hydration and thinness of the stratum corneum
- this is also responsible for the ***toxic reactions*** seen with,
    a. boric acid powders
    b. naphthalene
    c. salicylic acid

- the topical use of other agents, such as GTN, has been little studied

Absorption - Rectal

- venous blood drains to the sup/mid/inf. rectal veins
    a. sup. rectal vein → portal circulation
    b. inf. & mid. rectal vv. → IVC & systemic circulation

- thus, agents absorbed from the ***lower 2/3*** bypass hepatic 1st pass metabolism
- this increases bioavailability for most anaesthetic agents
- where the main action is via a metabolite, this is obviously disadvantageous
- aqueous & alcoholic formulations are more rapidly absorbed than suspensions or emulsions
- although the area is much smaller than the stomach, higher peak plasma levels have been observed with,
    a. diazepam
    b. theophylline

Placental Transfer

- factors which affect drug transfer include,
    a. physicochemical properties of the drug
        i. lipid solubility
        ii. degree of ionization
        iii. molecular weight
        iv. protein binding - for most maternal > foetal
            - reverse ≡t "trapping"
    b. foeto-placental factors
        i. maternal placental blood flow
        ii. foetal placental blood flow
        iii. metabolism by placental enzymes
        iv. extraction by the foetal liver

Variations in Body Composition

- the body composition of the neonate varies from the adult in that,

				ECF	ICF
a.	TBW is higher				
	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

- higher proportion of TBW in younger children 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
- the net result of these differences is that,

a. water soluble drugs have a higher V_{dSS} and may require a higher initial dose (relaxants)

b. drugs which redistribute into the ***lipid*** compartment will have a prolonged clinical effect (thiopentone)

c. drugs which redistribute into ***muscle*** will have a prolonged clinical effect (fentanyl)*

NB: *despite this, saturation of muscle has not been demonstrated

- the other factors which affect drug handling in the neonate include,

a. delayed excretion 2° larger V_{dSS}

b. immature hepatic and renal function

c. less plasma protein
- lower protein binding
- less drug metabolism

d. complications of prematurity
- sepsis
- cardiac failure
- poor nutritional status

- in comparison, ***older children*** tend to have,

a. more mature hepatic and renal function

b. normal adult values for protein

c. fat & muscle content approaching adult values

d. a greater fraction of CO diverted to liver & renal blood flow

e. a greater % body mass for liver & kidneys

- the net effect of all of these factors is that,
    a. most drugs have a prolonged $t_{½\beta}$ in pre & term infants
    b. most have a shorter $t_{½\beta}$ in children ~ 2 yrs cf. adults
    c. as the child approaches adulthood the $t_{½\beta}$ lengthens

Plasma Proteins

- the principle binding proteins are,

a.	albumin	- preferentially ***acidic*** drugs - antipyretics, antiepileptics - benzodiazepines & barbiturates - ***bilirubin***
b.	α_1-acid glycoprotein	- preferentially ***basic*** drugs - neuroleptics, narcotics - muscle relaxants, local anaesthetics - β-blockers

- protein levels are lower in the neonate and also exhibit lower drug affinity
- levels of these proteins are affected in ***disease states*** in a similar fashion to the adult,
    1. ↓ albumin levels
    2. ↑ α_1-acid glycoprotein levels

- binding can also be influenced by ***competition*** between drugs, eg.
    1. antibiotics and bilirubin
    2. phenytoin and valproate

    NB: alterations in protein binding have the greatest effect for drugs which are ***highly*** protein bound, ie. have a small free fraction

Drug Interaction

1.	absorption and distribution	
2.	renal function	- alteration of GFR - competition for tubular transport
3.	metabolism	- induction - competition for metabolism
4.	protein binding & free fraction	

Inhaled Anaesthetics

- recent studies have shown that,
    a. MAC is lower for the premature than for the term neonate
    b. MAC is lower for the newborn than for the 3 month infant
    c. infants have greater anaesthetic requirements than older children or adults (~ 30%)

    NB: the later, combined with the requirement for a deeper plane of anaesthesia for intubation, places the infant at risk of ***overdosage***

- some use the MAC for endotracheal intubation (MAC_{EI}), which for halothane is ~ 1.33%
- early administration of opioids / muscle relaxants may avoid this problem
- the uptake & excretion of volatile agents is ***increased*** due to,
    a. higher respiratory rates
    b. higher V_M:FRC ratio
    c. higher cardiac index
    d. higher percentage of CI directed to vessel rich group
    e. differences in blood:gas partition coefficients
    f. the anaesthetic circuit type (Mapleson D) $\rightarrow$
        i. more rapid alteration (increase/decrease) in $F_{A.gas}$
        ii. added risk of overdosage of infants / small children
        iii. minimal metabolism of halothane / isoflurane

- factors which will ***decrease*** the rate of volatile uptake include,
    a. ventilation perfusion mismatch
    b. large intracardiac shunts
    c. upper airway obstruction
        - croup, epiglottitis
        - sub-glottic stenosis
        - endobronchial F.B.

- these factors are inconsequential for N_2O due to its insolubility
- all of the volatile agents depress ventilation and increase the risk of ***apnoea***
- intercostal muscle activity is abolished and ventilation becomes solely ***diaphragmatic***
- assisted / controlled ventilation should be employed for all but very short procedures

Halothane

• of the three potent agents commonly used, this has the least noxious smell
• studies show no significant differences between rates of induction / emergence
• however, there are less airway related problems → preferred agent
• a 1987 report described 7 cases of halothane hepatitis, 1 fatal, concluding children should not undergo repeated halothane exposure
• this report did not take into account actual incidence, or risk benefit, which far exceeds agents such as penicillin
• other authors dispute the existence of this in the paediatric group
• if ***halothane hepatitis*** does occur in the paediatric age group, the true incidence is probably less than the adult population **~ 1:30,000**
• another concern is sensitisation of the myocardium to arrhythmias, 2° to endogenous/exogenous catecholamines
• in general, ***arrhythmias*** are usually accompanied by either ***hypercarbia*** or ***light planes*** of anaesthesia
• children tolerate higher levels of adrenaline than adults
• tachycardia and hypertension are more common side effects than arrhythmias
• several studies have shown ***adrenaline*** **< 10 µg/kg** carries minimal risk
• halothane is a potent myocardial depressant, especially for the neonate or the child with congenital heart disease
• this differs from that caused by isoflurane, which causes less direct depression and lowers SVR, thus preserving CO providing preload is maintained
• the use of supplemental short acting opioids usually overcomes this problem
• halothane markedly increases cerebral blood flow and is relatively contraindicated in most patients with raised ICP

Enflurane

• not favoured for gaseous induction due to the unpleasant smell, therefore usually only used after IV induction
• concentrations > 2.5% with hypercarbia induces seizure-like EEG activity
• however, it has been used safely in children with seizure disorders
• it is 3 times less sensitising with catecholamines than halothane
• like halothane it increases CBF and is contraindicated in raised ICP
• with long exposure there is some F^- induced decrease in renal concentrating ability

▪ ***Isoflurane***

• suggested advantages over halothane, as yet not clearly demonstrated, include,

1. less myocardial depression
2. preservation of heart rate
3. greater reduction in cerebral MRO_2

NB: the ↑ HR and maintenance of CO seen in adults ***is not*** observed in the paediatric group → ***hypotension & bradycardia*** ≡ halothane

• beneficial factors include greater potentiation of NMJ blockade, less sensitisation to catecholamines and less increase in CBF
• the main disadvantage being the more ***pungent odour*** and the greater incidence of laryngospasm and coughing

▪ ***Methohexital***

• short acting barbiturate
• dose ~ 1-2 mg/kg, supplied in 1.0% solution
• problems with IV induction include,

i. burning
ii. hiccup
iii. apnoea
iv. extrapyramidal type movements

• main advantage over STP is the shorter elimination half life
• may be used for induction PR, using 10% MOX ~ 25-30 mg/kg, inducing sleep in ~ 10 minutes
• this is usually limited to children < 20 kg, 10 mths to 6 yrs, who are afraid to be separated from their parents
• because it can cause seizure activity it is contraindicated in patients with temporal lobe epilepsy
• other children given seizure medications usually require larger doses

▪ ***Thiopentone***

• the induction dose, in healthy unpremedicated children **~ 5-6 mg/kg**
• termination is by redistribution to fat & doses should be tailored in patients with low fat stores (premature, malnourished)
• although termination of action is by redistribution, children do metabolise STP at ~ 2x the rate of adults
• thus, they tolerate total doses £ **10 mg/kg** reasonably well
• STP, 30 mg/kg can also be used rectally if MOX is contraindicated
• as for adults its weak vasodilatory action and myocardial depression require caution in hypovolaemic patients

- ***Ketamine***

• doses as low as 1.0 mg/kg produce sedation sufficient for induction of GA
• higher doses, ~ 10 mg/kg IM, allow insertion of invasive monitoring pre-induction (cardiac anaesthesia)
• relatively large interpatient variability
• major side-effect is ***salivation***, requiring routine administration of an antisialogogue
• other effects include ***vomiting*** and ***dreaming/dissociation***, though, this occurs more in the older than the younger child
• the later may be reduced by the concurrent administration of benzodiazepines
• its sympathomimetic properties are useful in the hypovolaemic patient, however, the incidence of dreaming is such that other agents are preferable
• relative ***contraindications*** to its use include,

a. active URTI
b. raised ICP
c. open globe injury
d. presence of a seizure or psychiatric disorder
e. as a sole agent in a patient with a full stomach

• it ***does not*** preserve the gag reflex and thus should not be used as the sole agent in patients with a full stomach or hiatus hernia
• it may also be administered rectally or nasally ≤ 10 mg/kg

- ***Droperidol***

• actions are primarily ***extrapyramidal***, therefore side-effects include,

a. muscle rigidity
b. visual disturbance
c. hallucinations
d. oculogyric crisis
e. dysphoria

• thus, the use of high doses of droperidol has little place in paediatric anaesthesia due to the prolonged sedation which may occur in some patients
• the primary use is as an antiemetic ~ 75 µg/kg, due to the increase in CNS dopamine levels
• ?? anti-emetic effects in adults seen at doses as low as 5-15 µg/kg (ie. 300 µg to 1.0 mg)
• other actions include α-blockade & hypotension

▪ ***Benzodiazepines***

- diazepam has a more rapid oral absorption in the child
- Miller states that 0.1-0.3 mg/kg orally almost always provides excellent sedation within one hour
- in neonates the $\mathbf{t_{½b}}$ **~ 80 hrs**, being relatively contraindicated ≤ 6 months
- it should also be used with caution in patients with hepatic disease
- midazolam being water soluble is less painful IV and readily absorbed after IM administration
- doses ~ 0.025-0.05 mg/kg IV, or 0.05-0.1 mg/kg IM will usually produce desired sedation
- it may also be administered orally or nasally

▪ ***Opioids***

- the newborn is more sensitive to ***morphine***, due in part to,
    a. immaturity of the BBB
    b. altered pharmacokinetics with decreased clearance
    c. changes in receptor ontogeny

- these statements are based on a number of papers, usually involving experiments on rats,
    a. Kupferburg & Way (1963) — - LD_{50} lower in newborn rats
- higher brain:plasma concentration ratio
    b. Way *et al.* (1965) — - IM morphine in infants
- greater ventilatory depression cf. morphine
    c. Zhang *et al.* (1981) — - low & high affinity morphine receptors in rats

        i. ***low affinity*** — ∝ ***respiratory depression***
- high concentration at birth
- remain high for first ~ 18 days
        ii. high affinity — ∝ analgesia
- low concentration at birth
~ 50% of adult level by 15 days

- 1981, Robinson & Gregory *et al*. demonstrated the safety of fentanyl in ***PDA ligation***

    NB: **Anand** *et al*., (Lancet 1987), evaluated biochemical markers and clinical outcome variables, comparison study dTC + N_2O/O_2 ± fentanyl, establishing importance of anaesthesia for ***neonates***, the fentanyl group showing,

        i. attenuation of hormonal stress response
        ii. ↓ protein breakdown
        iii. ↓ incidence of postoperative apnoea, bradycardia & hypoperfusion
        iv. ↓ pulmonary hypertensive episodes 2° airway manipulation in neonates

• Hertzke et al. (1989), looked at age related depression with fentanyl, using pancuronium + N_2O/O_2 + fentanyl 21 µg/kg,

a. infants 1-12 months

b. children 3-5 years * ***highest*** incidence of apnoea

c. young adults

• however, all but one of the infant group were > 3 months, thus the issue of neonatal depression is still unresolved
• other studies with alfentanyl have similarly shown these agents to be safe by 3, and probably 2, months of age
• Anand & Hickey (1992) in anaesthesia for CHD,

a. comparing morphine & halothane with high dose sufentanyl

b. greater stress response and increased morbidity & mortality with former

▪ ***Morphine***

• morphine should be administered with caution to neonates & infants ≤ 6 months age
• infants > 6 months appears to have an "adult" response, however, more studies are required
• as opioids of greater lipophilicity, eg. pethidine → fentanyl, are used the effects of the BBB become less significant and the clinical effect more predictable

▪ ***Fentanyl***

• fentanyl, in doses of ≤ 10-20 µg/kg has been shown to,

a. prevent the CVS response to surgical simulation

b. prevent pulmonary hypertension associated with stress

c. cause little if any CVS instability

• supplementary doses are required at ~ 60 minutes
• with these doses postoperative observation is necessary as ***rebound*** in plasma levels may be observed
• initial termination of action of fentanyl is by ***redistribution***, higher doses by elimination which approaches that of the longer acting agents
• the pharmacokinetic profile appears to be ***more variable*** for neonates than for older children
• the rate dependent CO of the neonate must be remembered with fentanyl induced ***bradycardia***
• alfentanyl and sufentanyl have greater potency and shorter duration of action than fentanyl but are otherwise similar

- ***Oral Transmucosal Fentanyl Citrate OTFC***

= the "fentanyl lollipop"

• effective, non-threatening premedication in surgical patients
• easier parent separation & induction of anaesthesia
• also effective as an analgesic in paediatric cancer patients undergoing invasive procedures, LP/BMB
• some studies have shown a higher incidence of side-effects, including,

a. longer wake-up times
b. higher incidence of nausea & vomiting
c. respiratory depression

• this may reflect the high dose used in some studies (20-25 µg/kg cf. average ~ 15-20 µg/kg)

- ***Alfentanyl***

• a fentanyl cogner, with a rapid onset of action and short elimination half-life, due to,

a. ***less lipophilic*** than fentanyl due to its low pK_a
b. ~ 90% ***unionized*** at physiological pH
→ more is available for diffusion into the CNS
c. highly ***protein bound*** → small V_{dSS} & short $t_{½\beta}$

• studies of newborn infants (≤ 3/7) show altered kinetics,

a. larger V_{dSS}
b. lower clearance → longer $t_{½\beta}$

- ***Nasal Transmucosal Sufentanyl***

• similarly effective to OTFC
• doses ~ 3.0 µg/kg have been associated with truncal rigidity and decreased ventilatory compliance
• studies comparing IV versus transmucosal sufentanyl show a similar time of onset for sedation, but slightly faster with IV
• by 20 minutes sedation is the same and by 30 minutes plasma levels are identical

Spinal Axis Opioids

• spinal axis administration allows analgesia without motor or sympathetic blockade
• ***lipid solubility*** of the opioid is critical in determining the rate of onset, duration and degree of spread
• caudal morphine (0.075 mg/kg) has been shown to provide analgesia for up to 2-12 hours (~ 6), and to decrease the 24 hour opioid requirements for children undergoing open heart surgery
• Krane *et al*. (Anesthesiol. 1989) found the optimal dose of ***caudal*** morphine (**~ 0.03 mg/kg**),

a. age groups 1-17 years
b. provided analgesia for ~ 22 hours
c. minimal incidence of side effects

NB: equivalent to ~ 2.0 mg for a 70 kg adult

• ***side effects*** of spinal opioids include,

a. urinary retention
b. nausea & vomiting
c. pruritus
d. delayed respiratory depression

20-40%

• fentanyl, sufentanyl and other highly lipophilic opioids bind rapidly to the spinal cord and reduce the incidence of delayed respiratory depression
• however, the duration of analgesia with these agents is similarly decreased and administration by continuous infusion is more appropriate

Patient Controlled Analgesia

Standard PCA Settings	
Concentration	• ~ 1.0 mg/kg Morphine / 60 ml PCA syringe* • ~ 0.015 mg/kg per dose/ml
Loading dose	• nil, except postoperative pain protocol
Bolus	• 1.0 ml stat
Lockout	• 5' ? Davis recommends 10-15'
Background	• 1.0 ml/hr

Syringe Concentrations by Weight		
Weight	Morphine (mg)	Concentration
15-20 kg	18 mg	0.3 mg/ml
21-26 kg	24 mg	0.4 mg/ml
27-32 kg	30 mg	0.5 mg/ml
33-38 kg	36 mg	0.6 mg/ml
39-44 kg	42 mg	0.7 mg/ml
45-50 kg	48 mg	0.8 mg/ml
51-56 kg	54 mg	0.9 mg/ml
≥ 57 kg	60 mg	1.0 mg/ml

NB: for ***pethidine*** simply times all concentrations/doses by a factor of 10

NB: for ***morphine infusions***, use the formula →

0.5 mg/kg / 50 ml syringe @ 1-4 ml/hr
≡ᵗ 0.01 - 0.04 mg/kg/hr

Berde (ASA) → < 6 months ~ 0.015 mg/kg/hr (~ 50% decrease < 6/12)
> 6 months ~ 0.025 mg/kg/hr

• studies of PCA show better pain scores & patient acceptance, but ***no*** decrease in total dose, time to oral intake, N & V, or incidence of urinary retention
• PCA well accepted at ***age*** ³ **7**, occasionally at 5-6 years but increased failure rate
• ***Ketorolac*** 0.5 mg/kg q6h IV decreases opioid requirements, but optimal dosing unknown

Muscle Relaxants in Children

• throughout childhood there is physical and biochemical maturation of the NMJ
• the composition of the contractile proteins of skeletal muscle change and there is an increase in the % body mass as skeletal muscle
• synaptic transmission is relatively slow in the neonate & young infant, further the rate at which ACh is made available during repetitive stimulation is markedly reduced,

a. ***train of four*** → ***not*** sustained at 100%
b. ***frequency sweep EMG*** → marked fade at higher frequencies

≡[T] ***myasthenic response***

• the net effect is a reduced "safety margin" for NMJ transmission in the infant

▪ ***Succinylcholine***

• being a highly water soluble drug, with rapid distribution in ECF, the dose varies with postconceptual age,

a.	neonates & infants	~ 2-3	mg/kg
b.	children	~ 1-2	mg/kg
c.	adults	~ 1-1.5	mg/kg

• the reduction of immediately available stores of ACh and the reduced synthesis / mobilisation with tetanic stimulation seen in neonates contributes to the greater dose requirement
• in neonates and young infants the responses to further administration also vary,

a.	tachyphylaxis	≥ 4.0 mg/kg
b.	phase II block	≥ 6.0 mg/kg

• these occur at lower doses in older children
• although the infant has ~ ½ the plasma ***pseudocholinesterase*** level of the adult, at equipotent doses the recovery phase in not prolonged
• in the absence of IV access, suxamethonium may also be given IM, reliable relaxation occurring within 3-4 minutes of 5 mg/kg in infants and 4 mg/kg for children > 6 months
• following IM administration relaxation may last up to 20 mins

• ***arrhythmias*** frequently accompany IV administration (sinus bradycardia, rarely sinus arrest), particularly during halothane anaesthesia
• IV (not IM premed) administration of ***atropine*** decreases the incidence
• ***sinus arrest*** may follow the first dose but is more common following repeat doses, and may occur in patients of any age
• Miller states it is probably advisable to administer atropine IV to all children about to receive suxamethonium and that it should be available for other patients

Complications of Suxamethonium	
Fasciculations Muscle Pains Hyperkalaemia (mild) Myoglobinaemia Bradyarhythmias Raised intraocular pressure Raised intracranial pressure Raised intragastric pressure Raised gastric acid secretion Increased bronchial secretion Salivation	• ***Common***
Malignant hyperpyrexia Prolonged apnoea Anaphylaxis Anaphylactoid reactions Pulmonary oedema Severe hyperkalaemia Masseter spasm Sinus arrest / asystole	• ***Rare***

• ***intragastric pressure*** rises in direct proportion to the strength of muscle fasciculations
• in adults pressures ≤ 40 cmH_2O have been recorded, with the cardio-oesophageal sphincter becoming incompetent at ~ 20 cmH_2O
• in children, however, rises ≤ 4 cmH_2O are seen and pressure occasionally decreases

• ***intraocular pressure*** rises in both groups, the predominant factor being the contraction of the ***extraocular muscles***
• after IV administration pressure rises within 60 secs, reaching a maximum at 2-3 minutes and falling to baseline by 5-7 minutes

• severe ***hyperkalaemia*** is usually only seen in association with other factors (see over)
• normal subjects show ~ 0.3-0.5 mmol/l increase
• strong fasciculations are not necessary to produce severe hyperkalaemia in susceptible individuals
• there is no data to suggest the susceptible small child is less prone to massive K^+ efflux
• there is a high incidence of ***myoglobinaemia*** following SCh 1.0 mg/kg in prepubertal children
• this is rarely seen in adults
• similarly, elevation of ***creatine phosphokinase*** is also seen in children
• both of these occurring in the ***absence*** of strong fasciculations
• thus far there is no good explanation for this

- ***Succinylcholine***

• the occasional patient will develop ***trismus*** as the earliest sign of MH
• masseter spasm is seen with a frequency ~ 1% in 2 retrospective studies
• it is seen most often in children given a mask halothane induction and SCh
• tachycardia, occasional PVC's and mild metabolic acidosis frequently occur
• the implication of this is uncertain, as the definition of masseter muscle spasm / rigidity varies with the investigator
• there is a range of response for the masseter muscles following SCh, which may be defined as follows,

a. subclinical "jaw stiffness" $\equiv^{t}$ normal response
- only demonstrable with strain devices
- virtually none of these are MH prone

b. "jaw tightness interfering with intubation"
~ 1% of children
- a small ***undetermined*** percentage are at risk of MH

c. "extreme jaw rigidity, unable to open the mouth"
= ***masseter muscle rigidity***, MMR
~ 50% are biopsy determined at risk for MH

• the last group is that often quoted as showing ~ 50% positive for MH with a caffeine ± halothane contracture test
• this association is frequently associated with the second group, which would lead to an expected MH susceptibility frequency of ~ 0.5% !!
• given the number of triggering anaesthetics given and the low incidence of MH, this is clearly an erroneous association
• the actual incidence of true MMR is uncertain
• Miller suggests the incidence of MMR is actually less than the quoted 1%, and there is a need for formal prospective studies
• the problem is then of how to manage the child who displays MMR,

a. Rosenburg (1988)
- stop anaesthesia
- administer dantrolene
- monitor for rhabdomyolysis
- muscle biopsy

b. Gronert (1988)
- continue with safe agents
- monitor for rhabdomyolysis
- monitor $ETCO_2$, temp., etc.
- muscle biopsy

c. Littleford (1991)
- continue (triggering) anaesthesia
- look for other rigidity
- monitor $ETCO_2$, temp., etc.
- muscle biopsy

• the later is clearly the most controversial and it would seem idiotic to continue when equally effective, safe alternatives are available
• thus, the intermediate approach cf. Gronert would seem the most reasonable

- ***Default Plan of Action***

1. "jaw tightness"
   i. continue with safe agents
   ii. monitor for rhabdomyolysis
   iii. monitor $ETCO_2$, S_pO_2, ECG, temp., etc.
   iv. muscle biopsy
2. "masseter muscle rigidity"
   i. stop anaesthesia
   ii. monitor for rhabdomyolysis
   iii. monitor $ETCO_2$, temp., etc.
   iv. muscle biopsy

NB: administer ***dantrolene*** in either case if positive signs of MH

Contraindications to Suxamethonium	
Altered Neuromuscular Function	• the myotonias • disuse atrophy • spinal cord injuries (transection) • muscular dystrophy • Guillain-Barré syndrome
Risk of Severe Hyperkalaemia	• severe burns • massive trauma • sepsis syndrome • closed head injuries • spinal cord injuries • preoperative hyperkalaemia • hyperkalaemic familial periodic paralysis
Risk of Prolonged Neuromuscular Blockade	• atypical plasma cholinesterase • altered plasma cholinesterase function • prior administration of an anticholinesterase
Risk of Malignant Hyperpyrexia	
Open Eye Injury	
Encephalitis	• only if raised ICP

Muscle Relaxants - Nondepolarizing

• like other agents neonates are more sensitive to these agents and display greater interpatient variability
• the neonatal receptor is quite sensitive, however the drugs volume of distribution is larger
suggested doses for these agents is as follows,

Doses of Neuromuscular Agents

Agent	Intubation Dose
atracurium	• 0.5-0.6 mg/kg
vecuronium	• 0.1-0.15 mg/kg
pancuronium	• 0.1-0.15 mg/kg
dTC	• 0.8 mg/kg
neostigmine[1] edrophonium	• 20-60 µg/kg • 0.3-1.0 mg/kg
atropine[2] glycopyrrolate	• 10-20 µg/kg • 5-10 µg/kg

[1] ***Neostigmine*** 2.5 mg / 5 ml = 0.5 mg / ml / 10 kg
= 50 µg/kg

[2] ***Atropine*** 1.2 mg / 5 ml = 0.24 mg / ml / 10 kg
= 24 µg/kg

NB: Miller recommends reversal of all neonates and small children, even if they have clinically recovered

■ ***Mivacurium***

• a short-acting agent, rapidly metabolised by ***plasma cholinesterase*** at ~ 70% of the rate of SCh
• metabolites have no pharmacological action
• at doses ~ 2xED_{95} it appears to be devoid of CVS side-effects
• larger doses may be associated with a transient decrease in BP 2° to ***histamine*** release
• at "intubating" doses, ~ 0.25 mg/kg, the duration to onset is ~ 1 minute and the duration of clinical blockade ~ 10 min
• it is a shorter acting agent in children than adults
• like other non-depolarising agents, blockade is facilitated by the volatile anaesthetic agents
• plasma cholinesterase activity is the major determinant of infusion requirements at all ages
• there are minor differences associated with altered kinetics
• following infusion there is ***no*** evidence of apparent accumulation

▪ ***Atracurium***

• an intermediate duration agent, metabolised by non-specific esters and undergoing spontaneous ester hydrolysis $\equiv^{T}$ ***Hofmann elimination***
• both processes are pH and temperature dependent
• under normal physiological conditions, breakdown is predominantly by ***ester hydrolysis***, Hofmann elimination playing a minor role
• on a dose/weight basis, infants (1-6/12) had a similar requirement to adolescents, whereas children had a ***higher*** dose requirement
• on a dose/surface area basis, children and adolescents were similar, with infants having a lower requirement
• as for other agents, this reflects the susceptibility of the neonatal NMJ receptor, but the greater volume of distribution
• as for other agents, there is potentiation by the volatile agents
• no accumulation is seen with prolonged infusions, requirements being,

a. thiopentone / opioid ~ 8-10 µg/kg/min
b. volatile agents ~ 4-5 µg/kg/min

NB: both following an initial bolus

▪ ***Vecuronium***

• a medium duration agent, steroid based, related to pancuronium
• taken up by the liver and excreted largely unchanged by the hepatobiliary system ~ 40-50%
• small amounts are excreted through the kidneys ~ 4-14%
• these routes of elimination are affected at the extremes of life, such that the times to 90% recovery are,

a. infants ~ 73 minutes
b. children ~ 35 minutes
c. adults ~ 53 minutes

→ vecuronium ***does not*** have an intermediate duration in infants

• this is a function of the larger volume of distribution, slower beta elimination and greater sensitivity of the motor end plate
• like atracurium, little or no accumulation is observed with infusion
• infusion rates for children 2-10 years are ~ 2x those of adults

- ***Doxacurium***

• duration of action similar to pancuronium, however apparently devoid of CVS side-effects
• excretion is predominantly unchanged through the ***kidneys***, with a marked prolongation of $t_{½\beta}$ in patients with renal failure
• times to recovery of TOF to 75% are,

a. 1 x ED_{95} ~ 63 ± 33 min

b. 1.8 x ED_{95} ~ 108 ± 25 min

• there were no significant changes in HR / BP with either bolus dose

- ***Pipercuronium***

• a new steroidal relaxant with a similar duration of action to pancuronium
• like doxacurium it appears to be devoid of CVS side-effects
• times to clinical recovery (T_{25}) following repeated dosage are ~ 20 minutes for infants and ~ 30 minutes for children

Anticholinergics

• ***scopolamine*** (10 µg/kg) cf. ***atropine*** (20 µg/kg) has,

a.	vagolytic properties	~ equal	
b.	antisialogogue	~ 2-3x	(NB: MCQ)
c.	***CNS depressant***	~ 5-15x	
NB:	doses being slightly > adult	~ 5-7 µg/kg	

• both decrease the ability to sweat and thus increase the core ***temperature***
• clinical indications for their use include,

a. diminish secretions preoperatively
b. block laryngeal & vagal reflexes
c. minimise suxamethonium induced bradycardia
d. antagonise the muscarinic effects of neostigmine
e. oculocardiac reflex
f. arrhythmias in WPW syndrome

• some literature suggests Down's are more susceptible to atropine, however this is disputed
• there is a higher incidence of narrow angle ***glaucoma*** in Down's and atropine should be administered cautiously under these circumstances
• scopolamine should therefore be limited to cases where the sedative effects are desirable
• the CNS sedation and antisialogogue effects are useful in combination with ***ketamine***
• the CNS effects of both agents are antagonised by physostigmine
• their routine use as premedicants has fallen into disfavour because,

a. they are ***painful*** on injection
b. the ***timing*** of injection/induction rarely coincides
c. the modern volatiles produce less secretions

▪ ***Glycopyrrolate***

• a synthetic ***quaternary ammonium*** compound & potent anticholinergic
• it has some advantage over the 3° agents as,

a. it produces less CNS effects
b. the duration ~ 3-4 hours cf. atropine ~ 2.5 hrs
c. minimal δHR and less arrhythmias
d. ± reduced gastric volume & acidity in some children (doesn't occur in adults in clinical doses)

NB: usual reversal dose ~ 5-10 µg
ie. ~ ½ atropine dosage

NPO STATUS & PREMEDICATION

• Winternitz (1920) was the first to describe the association between gastric acid and the syndrome of pulmonary aspiration
• Hall (1940) first described this problem for the parturient, however ***Mendelson*** (1946) is credited with having described the pathophysiology of pulmonary aspiration of gastric contents
• this subsequently led to a number of measures,

a. regional anaesthesia where possible
b. "awake" intubation
c. the development of cuffed endotracheal tubes
d. barbiturate/suxamethonium induction
e. cricoid pressure
f. the fasting period prior to elective procedures
g. administration of clear antacids or IV H_2 blockers
h. the use of gastrokinetic agents - metoclopramide

• the commonly accepted ***risk factors*** for gastric aspiration occur in **~ 76%** of paediatric patients, irrespective of their inpatient/outpatient/fasting status, ie.

a. gastric pH ≤ 2.5
b. gastric volume ≥ 0.4 ml/kg

• these parameters were actually based on unpublished data from Rhesus monkeys
• the clinical incidence of aspiration is actually very low despite this apparent high risk
• Raidoo (1988) suggested the volume of aspirate required was ~ 0.8 ml/kg
• cimetidine 7.5 mg/kg ~ 1 hour prior to surgery will markedly improve this, however its use should be restricted to high risk groups

- ***Risk Factors for Aspiration***

a. recent food intake
b. obesity
c. gastrointestinal pathology
d. mechanical bowel obstruction
e. narcotics
f. trauma / pain
g. neurological & neuromuscular dysfunction
h. prior oesophageal surgery
i. difficult airway
j. "operator inexperience"

→ especially in paediatrics

• the simple act of postponing emergency procedures for 4 hours has been demonstrated to markedly reduce the gastric residual volume in children,

a. surgery 1-4 hours from admission ~ 1.1 ml/kg

b. surgery 4-8 hours from admission ~ 0.5 ml/kg

• this act does not negate the need for management as a "full stomach"
• even the emergency patient may benefit from the administration of ***clear antacids*** pre-induction

Risk of Aspiration

• reported incidences vary between 1-10 per 10,000 **~ 4-5:10,000**
~ 1:2,000
• cf. adults, Olsson (1986) found a 3x high risk in patients £ **10 yrs**
• in this group (185,358), 7 of 10 episodes were associated with 1 or more episodes of ***laryngospasm*** → ? gaseous distension as a major contributing factor
• of those who aspirated, the majority had minimal postoperative sequelae

Optimal Fasting Period

• half the volume of normal saline is emptied from the stomach within 11 minutes
• the rate of emptying is modified by,

a. osmolality

b. presence of fat

c. presence of glucose

• a number of recent studies have shown no alteration of the gastric contents in infants / children / teenagers administered unlimited clear fluids within 2-3 hours of operation
• some have actually shown lower volume / higher pH levels cf. the standard fasting group

Paediatric Fasting Guidelines

	Milk/Solids		Clear Fluids[1]	
	old	new	old	new
0-6 months	4	4	2	2
6-36 months	6	6	6	3
≥ 36 months	8	6	8	3

[1] includes water, fruit juice, ± jelly

Premedication

- ***Reasons***

1. to allay fear & apprehension
2. to induce quiescence
3. to reduce airway secretions
4. to prevent / minimise vagally mediated reflexes
5. to raise gastric pH & decrease volume

NB: essentially to facilitate a smooth transition from the awake state to anaesthesia

- ***Ideal Premedicant***

a. painless & acceptable to children
b. rapid onset with short-term duration of action
c. 100% effective
d. have no adverse CVS/RS/etc. side effects
e. provide for the smooth transition from awake to GA

• the vast majority of studies show that whatever agent is used,

a. efficacy ~ 80-90%
b. placebo ~ 30-40%

• further, nearly all agents have some drawback, either,

a. the potential for airway obstruction
b. hypoventilation
c. discomfort on administration
   - taste
   - IM
   - burning, irritation
d. excessive time delay between administration and maximal effect

Route	Advantages	Disadvantages
oral	painless	slow onset bioavailability
IM	rapid onset reliable	painful sterile abscess
IV	most reliable	± painful threatening overdose
rectal	rapid onset reliable	± painful local trauma defecation bioavailability
nasal	reliable	uncomfortable parent/child objection
mucosal/oral (fentanyl)	reliable	slow onset vomiting desaturation

- all assessments of premedication stress the importance of the ***preanaesthetic visit***,
    a. transmission of ***anxiety*** from parents to the child
    b. difference between ***anaesthesia & sleep***
    c. that the child ***will waken*** at the end of surgery
    d. that ***pain*** will not be felt during surgery
    e. that adequate pain relief will be provided postoperatively

- ***anticholinergic*** agents are not routinely administered as they ***do not*** reduce the frequency of laryngeal reflexes during induction
- some authors have reported that atropine 20 µg/kg, orally/IM with premedication ~ 45 minutes from induction does reduce the incidence of ***hypotension*** with the volatile agents
- this is only the case for infants ≤ 6 months (ie. rate dependent CO)

Premedication Guidelines			
Route	Drug	Dose	
Oral	***trimeprazine***	**3-4**	mg/kg
	promethazine	0.5	mg/kg
	pethedine &	2-3	mg/kg
	pentobarbital	**2-4**	mg/kg
	pethedine &	2-3	mg/kg
	diazepam	0.2	mg/kg
	midazolam	**0.5-1.0**	mg/kg
	ketamine	6-10	mg/kg
Transmucosal	fentanyl	15-20	µg/kg
Nasal	midazolam	0.2	mg/kg
	ketamine	3.0	mg/kg
Rectal	STP[1]	10-25	mg/kg
	methohexitone[1]	10-20	mg/kg
	midazolam	0.3-0.4	mg/kg
IMI	***morphine*** &	0.1-0.15	mg/kg
	pentobarbital	2-4	mg/kg
	ketamine	2-4	mg/kg

[1] Induction dose, smaller doses provide edation

Induction of Anaesthesia - Gaseous

• mask induction is usually used in the age group ≤ 12 months, as they usually readily separate from their parents
• once anaesthesia is induced it is important not to overdose the child prior to the establishment of IV access
• this is best achieved by reducing ***halothane*** £ **1.5%**
• once IV access is established anaesthesia may be deepened or muscle relaxation added to enable intubation
• the rapidity with which F_{Agas} may be changed with a Mapleson D circuit must be remembered & discontinuation of anaesthetic during intubation is reasonable

• should the child present a potential problem at intubation they should receive only light sedation before mask induction, thus allowing breath sounds to assist intubation
• if the normal anatomy is disturbed and the child is not spontaneously breathing considerable advantage is lost

1. the child with ***intrathoracic*** obstruction generally has ***expiratory*** stridor and a prolonged expiration phase,
   i. bronchiolitis
   ii. asthma
   iii. intrathoracic foreign body
2. the child with ***extrathoracic*** obstruction generally has ***inspiratory*** stridor,
   i. epiglottitis
   ii. larhyngotracheobronchitis
   iii. laryngeal foreign body

• when agitated the later patients have dynamic airway collapse which may worsen the obstruction and lead to hypoxia and failure

→ ***gaseous induction*** with halothane and 100% oxygen

• stridor may be helped by mild CPAP, however maintenance of spontaneous ventilation is essential
• induction will be slow and any associated full stomach / regurgitation risk is secondary to the airway problem, "crash" induction being ***contraindicated***

NB: the Melbourne group will use RSI with IV induction in ***epiglotitis***

• the child with epiglottitis or croup will usually require a tube which is 0.5-1.0 mm smaller in internal diameter than usual
• (though S. Keeley says use a normal size for age in epiglottitis, 0.5-1.0 mm smaller in LTB)
• intubation may be assisted by the use of a stylet
• cuffed tubes are contraindicated

- ***Rectal Administration***

• a number of agents can be used via this route, the main advantage being that the child may fall asleep in their parents arms → ***STP ~ 25-30 mg/kg***

- ***Intramuscular Administration***

• the advantage of this route is reliability, the disadvantage being pain
• other problems include sterile abscess formation

- ***Intravenous Administration***

• this is the most reliable and rapid means of induction, however, gaining IV access can be difficult in the younger age groups
• options include,

a. two catheter system - 25G "butterfly" & catheter
b. use of LA via fine bore needle
c. use of EMLA cream

The Full Stomach

• the child should be managed the same as the adult, ie. RSI with cricoid pressure
• additional difficulties with the child include,

a. higher MRO_2/FRC ratio
b. often refuse preoxygenation → rapid desaturation
c. difficulties with IV access
d. reflex bradycardia 2° SCh ± atropine 0.02 mg/kg IV

ETT Tube Selection			
Patient Age	ETT ID[1]	Laryngoscope Blade - straight	Distance of Insertion[2] (cm)
prem < 1,250g	2.5	0	6-7
term	3	0-1	8-10
1 yr	4	1	11
2 yrs[3]	5	1-1.5	12
6 yrs	5.5	1.5-2	15
10 yrs	6.5	2-3	17
18 yrs	7-8	3	19

[1] ID = tube internal diameter in mm

[2] Insertion of the tube is relative to the ***alveolar ridge*** of the mandible

[3] Age > 1 $ID_{ETT} \sim (Age + 17)/4$

Endotracheal Tube Sizes (NTM)					
Age	Weight[1] kg	ID mm	Oral cm	Nasal cm	Suction FG
prem	1-2.5	2.5-3.0	6-7	7.5-9	5-6
newborn	3.5	3.5	8.5	11	7-8
6/12	7	3.5-4.0	10	12	8
1	10	4	11	14	8
2	12	4.5	12	15	10
4	16	5	14	18	10
7	23	6	16	20	12
10	32	6.5 (cuffed)	17	21	12

[1] Approximate weight Age < 9 years (2 x age) + 9 kg
Age > 9 years 3 x age kg

IV Fluid & Transfusion Therapy

Body Compartment Volumes			
Normal Values	Premature	Term	Adult
Total Body Water	80%	75%	55-60%
ECF ICF	45% 35%	40% 35%	20% 40%
Blood Volume	90-100 ml/kg	85 ml/kg	~ 70 ml/kg
H_2O/day - at 1 day - at 1 week[1]		50 ml/kg/d 150 ml/kg/d	

[1] increases until 6/52, then decreases to adult values

- clinical assessment of ***dehydration*** may be approximated by,

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

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

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

NB: ≥ 20% may result in ***coma***

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

[1] "4-2-1 rule"/hr, or "100-50-20"/day

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

- under normal circumstances ~ 100 ml of water being required for every 100 Calories expended
- this formula takes into account the higher metabolic rate of infants and small children, together with their larger surface area/weight ratio
- most sources suggest that the neonate, in the first few days, is at risk of ***hypernatraemia*** due to,

a. low urinary Na^+ losses

b. high insensible water losses

c. a high solute load from a high MRO_2

→ day 1 ~ 5% or 10% dextrose at 2 ml/kg/hr
≥ day 2 ~ 5% dextrose + Na^+ 40 mmol/l
+ K^+ 20 mmol/l

- Coté is actually more concerned with ***hyponatraemia***, due to the kidneys inability to conserve Na^+, even in the presence of a low plasma level

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	10-15 ~ 20	g/kg/d g/kg/d

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

10 g/kg/d = 200 ml D_5W/kg, ∴ use $D_{10}W$

IV Fluid & Transfusion Therapy

• the preceding table does not include,

a. fluid deficits or third space losses

b. modifications for hypothermia / hyperthermia

c. other modifying factors
- sepsis
- burns
- major trauma

• ***fluid deficits*** from elective preoperative fasting may be calculated from the above formula for basal requirement

• deficits from other pathology may be calculated by assessment of the degree of dehydration (see above) and the total deficit replaced at,

a. 50% in the first hour

b. 25% in each of the next two hours

NB: ***plus*** maintenance & 3rd space losses

• ***third space*** losses are procedure dependent, and may be approximated,

a. 0-2 ml/kg/hr - superficial procedures

b. 3-5 ml/kg/hr
- intracavity procedures
- moderate tissue manipulation

c. ≤ 15 ml/kg/hr
- extensive abdominal procedures
- eg. repair of an omphalocele

NB: → replacement with balanced salt solution ± 5% protein if low

• postoperative fluid management is generally aimed at providing maintenance water only, with other losses being assessed & replaced as required

→ D_4W & N/5 saline + KCl 20 mmol/l

Glucose

• the addition of glucose to the maintenance fluid is presently uncertain due to,

a. animal data suggesting a worse CNS outcome with a high BSL

b. the concerns of unrecognised hypoglycaemia

c. true incidence of hypoglycaemia in all groups of food restricted children is unknown

d. different groups have used different reference ranges for defining hypoglycaemia (varying with age)

• all data on the detrimental effects of ***hyperglycaemia*** in humans has been based on retrospective studies of diabetic patients who developed ischaemic strokes & had poor outcomes

• newborns/premature infants should be considered separately from other children, ie. assume hypoglycaemia until proven otherwise
• different studies have shown a variable incidence of ***hypoglycaemia*** in healthy children ≤ 6 years of age fasted for ≤ 10 hours
• further, that these children may not demonstrate clinical signs of hypoglycaemia in the preanaesthetic period and that a proportion do not respond to the stress of surgery with an increase in blood glucose
• administering the NPO deficit as D_5W invariably raised the BSL, however occasionally excessively with resultant ***glycosuria***

• various institutions use varying combinations, reasonable practice including,

a. replacement of all existing deficits & 3rd space losses with balanced salt solution Hartmann's, Plasmalyte

b. maintenance with 2.5% dextrose in Hartmann's
   - not commercially available
   - add $D_{50}W$ to later = 50 ml of $D_{50}W$ per litre

Blood Product Replacement

▪ *Red Blood Cells*

• the ***estimated blood volume*** (EBV) should be calculated prior to anaesthesia,

a.	neonate	~ 90-100	ml/kg
b.	**term infant**	**~ 80-90**	ml/kg
c.	3/12 to 1 yr	~ 70-80	ml/kg
d.	over 1 yr	~ 70	ml/kg

• blood loss can be replaced with crystalloid (3:1), colloid or blood (1:1), or a combination
• the safest lower level for the haematocrit depends upon the type of surgery, the patients level of activity and their underlying medical condition,

a.	infant & young child	≥ 25%
b.	newborn	≥ 40%

• using these criteria, the ***maximal allowable blood loss*** can be calculated by,

$$\text{EBV} \times \frac{(\text{Hct.} - \text{minimum Hct.})}{\text{Hct.}} = \text{MABL}$$

• ***monitoring*** of the adequacy of blood volume replacement may be done by,

a.	HR & BP	
b.	urine output	
c.	direct arterial BP	* pulse pressure / ventilation
d.	CVP	

• once blood loss exceeds the MABL, replacement of RBC's is required
• slight overtransfusion from a single blood pack is preferable, rather than risking the need for a second transfusion postoperatively
• some transfusion services split packs into "quad packs", so that multiple transfusions may be given from a single donor pack

■ ***Fresh Frozen Plasma***

- contains all ***clotting factors***, except when FVIII is harvested prior to freezing (noted on the unit)
- after 6 hours the labile factors (V/VIII) begin to diminish (ie. use ASAP)
- contains proportionally more ***citrate*** than whole blood
- indications for use,

a.	massive blood transfusion	- clinical evidence of bleeding, not surgical - laboratory evidence of coagulopathy
b.	von Willebrand's disease	
c.	haemophilia A	- rarely, usually FVIII concentrates

■ ***Platelets***

- causes for a reduction in platelet numbers,

a.	***reduced production***		- marrow failure (aplastic) - marrow infiltration
b.	***sequestration***		- splenomegaly
c.	***accelerated destruction***		
	i.	massive transfusion	- dilutional (≥ 1 BV)
	ii.	consumptive	- coagulopathy (DIC)
	iii.	autoimmune	- SLE, lymphoma, HIV, ITP
	iv.	drug induced	- aspirin, heparin

NB: → 2 groups, gradual vs. rapid reduction in platelet numbers

- requirement for platelets depends upon cause and rate of development,

a.	1 unit of platelets	~ 7,000-11,000 / mm^3 / m^2 SA increase
b.	0.1-0.3 units/kg (standard dose)	~ 20,000-70,000 / mm^3

- important points,

a. antibody production is ∝ to units transfused
   → limited effectiveness of future transfusions

b. not all hospitals have platelets readily available

c. in the thrombocytopaenic patient, they should be administered immediately preoperatively

d. they should ***not*** be run through a micropore filter

e. single donor platelets may be required where the patient has become refractory to random donor platelets, but ↑↑ cost & need HLA matched donor

- ***Cryoprecipitate***

a. contains ~ 20-50% of the original F-VIII levels
   - but in a much smaller volume, ∴ best means of replacement in factor deficiency

b. also contains F-XIII and ***fibrinogen***

c. the principal use is in the R_X of ***haemophilia A***

- ***Factor Therapy***

- patients with haemophilia B (F-IX deficiency) are managed with commercial concentrates which contain F-VII, IX and X
- concentrates are from pooled donor sources and have a greater risk of ***transmissible disease***

Massive Transfusion

Def'n: replacement of the patients blood volume 1 or more times / 24 hours, or, > half the BV in 2 hours

1. ***factor deficiency*** - WB deficient F-V,VIII (~ 20-50%)
   - packed cells minimal quantities
   - bleeding increased at ≤ 30% normal
   → FFP ~ 20-30% for each BV lost

2. ***thrombocytopaenia***
   - bleeding diathesis occurs at < 50,000-75,000 for acute loss
     < 10,000-15,000 for chronic disease
   - depends upon the starting count ~ 2 BV's in children
   - ***thrombocytopathy*** with massive transfusion (GP1b)
   - therefore require baseline & subsequent ***clotting studies***

3. ***hyperkalaemia*** ∝ to the shelf life of the unit
   - generally only with whole blood
   - rate of infusion important → ≤ 1.5-2.0 ml/kg/min
     → ≤ 100 ml/min in an adult
   - monitor by ***ECG*** at higher rates
   - ABP's better and check unit $[K^+]$ for neonates
   - Red Cross recommended expiry for neonatal use **~ 7 days**

4. ***hypocalcaemia*** ∝ to citrate content of unit
   ∝ rate of infusion
   - ≤ 1.5-2.0 ml/kg/min rarely a problem ~ 100 ml/min in adults
   - FFP has more %citrate than WB → < 1.0 ml/kg/min
   - monitor by ***ECG*** at higher rates

5. ***acid-base*** - depends upon reason for T_X
   i. hypovolaemic shock & acidosis
   ii. ongoing surgical losses, without shock
   - acidosis is rarely a problem providing ***hypovolaemia*** is avoided
   - most acid in WB is CO_2 → lungs
   - $NaHCO_3$ may have be harmful → use according to AGA's only
6. ***hypothermia*** → L-shift of HbO_2 curve
   - all banked products ~ 2-6°C
   - all T_X should be warmed 38-40°C
   - ≥ 42°C results in RBC destruction
7. **HbO_2 *dissociation*** ∝ pH, Temp., and 2,3-DPG
   - ***citrate*** is metabolised to HCO_3^- → L-shift
   - WB & FFP have the greatest effect
   - stored blood deficient in ***2,3-DPG*** → L-shift
   - however, CO_2 / H^+ load → R-shift
8. ***pulmonary effect***
   - microthrombi embolism greatly reduced with micropore filters
   - however, incidence of ARDS ***unaffected***
9. ***infective risk***
   i. viral transmissible - HIV, CMV, EBV, HBV, HCV
   ii. bacterial contamination
   iii. other - plasmodium, spirochetes etc.

Monitoring

a. routine - ECG, BP, Temp., SpO_2, $ETCO_2$
b. urinary output
c. IABP
d. CVP

NEONATAL ANAESTHESIA

Factors of Special Concern in the Neonate

- Thermal stability
- Airway and breathing mechanics
- Blood volume, Circulation & Cardiac function
- Transitional Circulation
- Metabolic and electrolyte imbalance
- Renal function
- Hepatic function & jaundice
- Coagulopathy
- Immunoparesis
- Associated medical conditions
- Nutrition
- Monitoring

- ***Thermal Stability***

1. transport in incubators
2. O/T temperature
3. overhead radiant heaters
4. warming blanket
5. heat loss from head
6. heater/humidifiers
7. monitoring

- ***Airway/Breathing***

1. ***intubation***
   - i. large, poorly supported head
   - ii. large tongue
   - iii. large, mobile epiglottis
   - iv. anterior/high larynx
   - v. angled vocal cords
   - vi. funnel larynx, narrow cricoid
   - vii. short trachea → movement of ETT with head

2. ***respiration***
   - i. small FRC
     - high MRO_2 / FRC → rapid desaturation
     - ↓ FRC / CC ratio → gas trapping & ↑ V/Q mismatch
   - ii. large shunt fraction ~ 10%
   - iii. high compliance of chest wall/angle of ribs
   - iv. loss of laryngeal brake with ETT
   - v. fatigue of muscle
   - vi. abdominal organomegaly/stomach
   - vii. immaturity of respiratory centres
     - airway reflexes
     - hypoxic reflexes
     - sensitivity to opioids

3. ***risk of BPD***

- ***Blood Volume/Circulation***

   - i. small blood volumes
   - ii. greater %effect of any losses
   - iii. poor compensation for hypovolaemia / decreased preload
   - iv. poor compensation for increased afterload (no ↑ contractility, fixed SV)
   - v. HR dependent CO (NB:hypoxaemia)

- ***Transitional Circulation***

   - i. highest risk in first 2/52 and in premature / SGA infants
   - ii. avoid factors which ↑ PVR
     - hypoxia, hypercarbia, acidosis
     - high mean airway pressures, PEEP
   - iii. patent ductus arteriosus in older child

- ***Metabolic***

  i. risks of hypo/hypernatraemia
  ii. potassium balance/renal function
  iii. hypocalcaemia - multiple causes
  iv. fluid balance *weight change/urine output
  v. neonatal $NaHCO_3$ = 0.5 mmol/ml (risk of IV haemorrhage)
  vi. narrow limits for plasma glucose

- ***Renal Function***

  i. creatinine more useful than BUN - anabolic
  ii. depressed by hypoxaemia / ischaemia
  iii. congenital abnormalities
  iv. indomethacin for PDA - PG's opposed ADH tubular effects
  - disordered GTB
  v. altered drug excretion
  vi. fluid overload/imbalance

- ***Hepatic Function***

  i. altered drug handling
  ii. drug side effects
  iii. hyperbilirubinaemia & drug effects

- ***Coagulopathy***

  i. ***all*** should receive vit. K_1 1mg x 3 ? orally not parenterally
  ii. maternal anticonvulsant therapy
  iii. sepsis / DIC / thrombocytopaenia

- ***Immunoparesis***

  i. depressed antibody & cell mediated
  ii. bacterial & viral susceptibility
  iii. non-specific clinical signs
  iv. need for aseptic techniques

- ***Other Conditions***

NB: with ***any*** anomaly there is an increased risk of others

- ***Nutrition***

i. ? CVC placement if TPN required
ii. alternatively insert long line

- ***Retinopathy of Prematurity***

i. multifactorial origin - FiO_2, P_{aO2}, duration, ventilation, other factors
ii. lack of consensus as to ideal SaO_2 - especially upper limit of safety
iii. most agree ~ 90-95% range
iv. definition of neonatal ***hyperoxia*** $\rightarrow$ P_{aO2} > 80 , > 90 or > 100 mmHg

Anaesthetic Management

• once all of the above have been considered, the main question is whether or not the child is an ***aspiration risk***
• if so, the choice is then between rapid sequence induction and awake intubation
• awake intubation is recommended in infants with upper airway abnormalities, or those who are moribund for any reason
• for rigorous infants with normal airways, RSI is preferred
• breath sounds must be checked after intubation and after repositioning of the head
• the ETT moves ***in with flexion*** and ***out with extension*** of the neck
• there are a number of conditions where ***inhalational induction*** with spontaneous respiration are recommended,

a. TOF
b. congenital lobar emphysema
c. bronchogenic / pulmonary cysts

• differing responses to the anaesthetic agents should be noted
• need for postoperative ***apnoea*** monitoring in neonates receiving opioids or volatile agents
• increased incidence of,

a. ***intraventricular haemorrhage*** with sustained hypertension
b. ***retrolental fibroplasia*** with excessive O_2
c. ***bronchopulmonary dysplasia*** with long-term high airway pressures

CONGENITAL HEART DISEASE

- ***Incidence***

  ~ 6-8:1000 live births

- ***Classification***

i.	***acyanotic***	VSD	~ 25%	(30)
		PDA	~ 17%	(10)
		ASD	~ 7%	(7)
ii.	***cyanotic***	Fallot's tetralogy	~ 11%	(5)
		transposition	~ 8%	(5)
		tricuspid atresia	~ 3%	
iii.	***obstructive***	PS	~ 7%	(7)
		coarctation	~ 6%	(6)
		AS	~ 4%	(5)

Indices of Critical Impairment in CHD	
SaO_2	< 75%
Q_{PUL}/Q_{SYS}	> 2:1
	L→R shunt ≥ 50%
LV outflow tract δP	> 50 mmHg
RV outflow tract δP	> 50 mmHg
PVR	> 480 dyne.sec.cm^{-5}/m^2
	> 6 Woods units
polycythaemia (Hct)	> 60%

- patients meeting 1 of these criteria are at risk of decompensation during anaesthesia
- patients meeting ≥ 2 criteria require extensive evaluation prior to anaesthesia
- the following are general ***risk factors*** for cardiac anaesthesia,

  a. any severe form of an isolated lesion

  b. complex lesions

  c. concurrent infectious disease

  d. metabolic disturbance

  e. congestive cardiac failure

  f. previous palliative procedures

  g. acute haemodynamic deterioration

  NB: where possible, preoperative correction of these factors should be attempted

Congenital Heart Disease

- many procedures commonly labelled "corrective" do not leave the patient with a "normal" CVS
- the stress of subsequent anaesthesia/surgery may result in decompensation
- in general, the only defects following which CVS reserve may be considered normal are,

1. patent ductus arteriosus
2. atrial septal defect - secundum type, without a patch

NB: when these procedures are performed ***early*** in childhood

True correction	Correction	Palliation
• patent ductus • ASD	• aortic coarctation • transposition • VSD • tetralogy of Fallot • pulmonary stenosis • aortic stenosis • AV canal repairs	• conduits • transplantation • pulmonary atresia • Fontan operation • pulmonary obstructive disease

a. ***true correction*** - normal life expectancy
- normal CVS reserve
- no further medical R_X

b. ***correction*** - markedly prolonged life expectancy
- minor limitation in CVS reserve
± further medical/surgical R_X

c. ***palliation*** - prolonged life expectancy
- definitely ***abnormal*** CVS function
+ further medical / surgical R_X

Preanaesthetic Evaluation

a. ***history*** - cyanosis, intermittent vs. continuous, stress related
- squatting, sweating, syncope, tachypnoea
- exercise tolerance
- feeding intolerance
- failure to thrive

* drug therapy

- hospitalisation for CCF / systemic diseases

b. ***examination*** * murmur, cyanosis, arrhythmia, respiratory distress (major signs)
- respiratory pattern, tachypnoea, SpO_2
- cyanosis, clubbing (± differential cyanosis with PDA)
- pulses & character (coarctation, sacrifice of a subclavian)
- height / weight percentiles
- CCF, liver, spleen
- usual factors (IV access, airway, behaviour, etc.)

c. ***investigations***

i. CXR
- situs and position
- pulmonary blood flow
- cardiac contour
- aortic contour
- lung fields
- skeletal factors (rib notching)

ii. CBP
- Hct. on admission ~ best indicator of R→L shunting
- polycythaemia with Hct > 60% increases risk

iii. catheterisation
- right heart pressures
- position & size of shunt
- pressure gradients & obstructive lesions
- response of PVR to FiO_2 = 100% (reversibility of pulmonary hypertension)

* effects of sedation/anaesthesia on data

iv. echo
- current, non-invasive data quickly
- screening in neonates with other congenital anomalies
- RV/LV function, valvular competence, shunt flows

Premedication

- avoid heavy sedation in patients with severe CCF or cyanosis
- generally unnecessary in infants < 6 months, or in calm, cooperative older children
- older children with less critical circulation → oral diazepam (0.1 mg/kg)
- where heavy premedication is required in the sicker patients,

a. ***pentobarbitone*** ~ 2-4 mg/kg orally an hour prior to IM injection

b. ***morphine*** ~ 0.1-0.2 mg/kg
± atropine/scopolamine

CHD - General 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 MRO_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^+

CHD - Specific Problems in Anaesthesia

▪ ***Severe Hypoxaemia / Cyanosis***

• problems include those from ***adaptations*** to chronic hypoxaemia,

a. polycythaemia
b. increased blood volume
c. neovascularization
d. alveolar hyperventilation / chronic respiratory alkalosis
e. increased 2,3-DPG

• polycythaemia has a ***biphasic effect***, depending upon the level,

a. Hct ≤ 60% increases C_{cO2} (60% ~ 20 mg/dl)
b. Hct > 60% increases SVR with decreasing CO & DO_2
c. Hct > 70% generally require preoperative R_X, due to,
   i. risk of ***thrombosis*** - renal
      - cerebral
      - pulmonary
      - coronary
   ii. ***coagulopathy*** - 2° to polycythaemia

• R_X includes erythrophoresis and cautious hydration & harvesting of ***autologous blood***
• with a Hct > 60%, prolonged periods without oral intake, both pre & postoperatively, should be avoided unless adequate IV hydration is supplied
• as the Hct rises, PVR increases disproportionately ***greater*** than SVR

• ***hypoxaemia*** generally results from inadequate pulmonary blood flow, 2° to,

a. aortopulmonary shunts
b. right to left intracardiac shunts
c. problems with ventilation of the lung

- therefore, treatment is aimed at one of three factors,

a.	reducing PVR	- FiO_2 = 100% - ensure bilateral gas exchange - hyperventilation - trial of mild PEEP
	tetralogy spell	- IV esmolol (outflow obstruction) - increase volatile anaesthetic
b.	increase SVR	- phenylephrine / metaraminol - manual abdominal aortic compression - manual knee-chest posture
c.	reduce MRO_2	- sedation / pain relief - general anaesthesia - mild hypothermia ~ 32-35°C - muscle paralysis

NB: the only exception to this is in ***transposition of the great vessels*** in newborns, where inadequate intracardiac ***mixing*** may result in hypoxaemia,
→ aim for L & R sided pressures ~ equal

▪ ***Cardiac Shunting***

- Rudolph (1974) described distinction between cardiac shunts,

1. ***dependent***
   - i. size & direction of shunt are dependent on the PVR / SVR relationship
   - ii. chamber pressure differential is small
   - iii. PDA, simple ASD/VSD, aortopulmonary windows
   - iv. operative systemic-pulmonary shunts (Blalock-Taussig, Waterson, Glenn)
2. ***obligatory***
   - i. shunt is independent of SVR/PVR ratio
   - ii. resistances tend to be fixed
   - iii. chamber ***pressure differential*** is large
     - LV→RA in complete AV canal
     - peripheral AV fistulae
   - iv. alternatively, with ***outflow obstruction***
     - mitral/tricuspid atresia → unidirectional atrial shunting
     - aortic/pulmonary atresia → similar

NB: in complex CHD, both types of shunt may ***coexist***
this description assumes defects are large and flow is unimpeded
with ***restrictive shunts*** the relative effects of PVR & SVR are proportionately less

• generally, acute increases in **L® R** shunting during anaesthesia are of clinical significance in only a few situations,

1. hypoplastic left heart syndrome
2. pulmonary atresia with a widely patent ductus arteriosus

NB: here a substantial "steal" of systemic blood flow can occur

→ ↓ mean diastolic & coronary perfusion pressure

• shunting from **R® L** is always accompanied with some degree of arterial desaturation and is relatively poorly tolerated

■ ***Excessive Pulmonary Blood Flow***

• results in ***volume overload*** of both ventricles, limiting cardiac reserve
• increased pulmonary blood volume limits gas exchange by,

a. airway compression at various levels
b. decreased pulmonary compliance
c. increased lung water

• permanent changes in the structure of the pulmonary vasculature can result in reversal of the shunt when PVR is sufficiently raised
• anaesthesia may further increase pulmonary blood flow, leading to,

a. CCF
b. hypotension
c. myocardial ischaemia
d. decreased gas exchange

• therapeutic measures aimed at ***increasing PVR*** include,

a. minimise FiO_2 ~ 21%
b. mild hypoventilation & respiratory acidaemia
c. PEEP
d. in severe cases
   i. endobronchial intubation / ventilation
      → hypoxic pulmonary vasoconstriction in one lung
   ii. direct manipulation, banding

Congestive Cardiac Failure

- results from either increased ***pressure*** or ***volume loads*** on the ventricles
- the child may compensate, however, severe failure results in growth retardation
- the reversibility of ventricular dysfunction in CHD varies depending on,

a. the type of defect

b. the severity of the anomaly

c. how amenable it is to correction
   - medical
   - previous surgical

d. the duration of dysfunction

- treatment in the acute phase is directed at,

a. minimising the pressure load
   - preload
   - afterload

b. avoidance of myocardial depressants

c. inotropic support

d. R_X of intercurrent disease

- in children with an ***unrestrictive*** ASD/VSD, or a single atrium or ventricle, the **CVP ~ LVEDP**
- PA catheters rarely provide additional information in these circumstances and are difficult to insert without fluoroscopy

Arrhythmias

- frequently accompany CHD and may be either concurrent or iatrogenic
- in children requiring demand pacing, ***asystole*** will often result if the pacemaker is interrupted
- choosing the ***asynchronous*** pacemaker mode will protect against sensing electrocautery signals
- rarely complete pacemaker failure has been seen, with "burnout" of the pacemaker unit
- therefore, alternative facilities/action should be available,

a. external pacing

b. temporary wire

c. the use of bipolar cautery

d. positioning of the ground-active axis at 90 ° to the pacing wire

e. use of the minimal effective current

f. use for short bursts & minimal duration effective

■ ***Outflow Obstruction***

• CHD lesions producing ***LV outflow obstruction*** include,

i. interruption of the aortic arch
ii. coarctation of the aorta
iii. aortic stenosis - subvalvular/valvular/supravalvular
iv. mitral stenosis & atresia
v. hypoplastic left heart syndrome (HLHS)

• these patients may have,

i. LV hypertrophy - except MS and HLHS
ii. myocardial ischaemia
iii. limited systemic vascular reserve

• ventricular fibrillation may occur, particularly in younger patients
• older children with less severe forms may present with,

i. arrhythmias ± syncope
ii. dyspnoea, fatigue
iii. chest pain

• levels of mean diastolic pressure are adequate to ensure myocardial perfusion, unless the lesion is above the coronary ostia

• CHD lesions producing ***RV outflow obstruction*** include,

i. tetralogy of Fallot
ii. pulmonary atresia
iii. pulmonary stenosis, subvalvular stenosis
iv. pulmonary artery stenosis
v. pulmonary vascular obstructive disease

• these patients may have,

i. RV hypertrophy and hypertension
ii. RV myocardial ischaemia
iii. RV failure as pressure approaches systemic

• similarly, acute treatment is aimed at providing adequate myocardial perfusion, usually with α-adrenergic agonists (metaraminol/phenylephrine)
• lowering PVR is beneficial, providing it can be accomplished without lowering MAP
• patients with ***pulmonary hypertension*** of any duration suffer,

i. cor pulmonale or arrhythmias
ii. sudden onset pulmonary hypertensive crises ± RVF/death
iii. increased incidence of sudden death

Anaesthetic Considerations

- most will tolerate GA, however their tolerance of mishaps is severely limited, eg.,
    a. loss of airway
    b. hypoventilation
    c. inappropriate fluid therapy
    d. inappropriate choice & dose of anaesthetic agent
    e. major surgical insults

- once CVS collapse occurs, resuscitation may be extremely difficult
- therefore, the aim of anaesthesia is to ***prevent*** any such occurrence
- in general, no single anaesthetic technique can be recommended over another

Volatile Agents

- conventional inhalational induction is well tolerated in the milder forms of heart disease (functional classes A & B)
- the margin of safety is greatly reduced in severe disease (C & D) and the potent inhalational agents should probably be avoided
- studies of inhalational ***halothane*** induction in normal children have shown,
    a. ~ 50% incidence of hypotension and bradycardia
    b. ~ 38% decrease in LV stroke volume and ejection fraction

- this is added to by the greater anaesthetic requirement in infants (1-6 mth) MAC ~ 1.2% and their increased myocardial sensitivity
- in neonates the CVS is even more sensitive, though, the MAC ~ 0.87%
- ***atropine*** prior to induction reduces the CVS effects by limiting the associated bradycardia
- ***isoflurane*** may be less depressive, however studies in infants < 6 months have shown,
    a. ~ 40% decrease in MAP, and
    b. ~ 32% decrease in HR

- in addition to this there is a higher incidence of ***laryngospasm*** (~ 30%)
- airway problems rapidly lead to hypoxia / hypercarbia & raised PVR

    NB: ***isoflurane*** has limited advantage in paediatric heart disease & anaesthesia, cf. its preference in the adult population

▪ ***Nitrous Oxide***

• potentially a problem due to,

1. its effect only any ***air bubbles*** entering the circulation
2. increase in ***PVR*** (reported in adults, ***no*** paediatric studies)
3. reduction in PaO_2 due to reduction in FiO_2
4. negative inotropic effect - especially in combination with ***fentanyl***
   - may be significant in abnormal hearts

NB: it is generally tolerated well in most cases, however, should be avoided in those with severely depressed myocardial function and post-CPB

▪ ***Ketamine***

• is generally well tolerated in children with severe CCF or cyanosis
• it requires the use of atropine/scopolamine to offset ***secretions***
• there have been reports of raised PVR in adults
• this is not seen in children as long as the airway is maintained & ventilation supported
• it may be administered IM (5-10 mg/kg) or IV (1-2 mg/kg)
• contraindications to its use may include,

a. ***coronary insufficiency*** - anomalous LCA
b. aortic stenosis > 50 mmHg gradient
c. hypoplastic left heart & aortic atresia with hypoplasia of the ascending aorta
   i. prone to VF / tachycardia
   ii. 2° to coronary insufficiency

▪ ***Fentanyl***

• in high doses (50-100 µg/kg), with pancuronium and either O_2 or air/O_2, is suitable for all forms of severe CHD
• changes in CI, SV, SVR, PVR are ***insignificant***
• with fentanyl or the other opioids, the negative inotropic effects of N_2O may become clinically significant
• the use of ***pancuronium*** is recommended to offset the vagotonic effects of fentanyl
• when administered slowly there are no significant changes in HR or BP
• an intubating dose administered as a bolus will result in tachycardia, however, this is frequently desirable in children with CHD & fixed SV
• the CVS stability seen with this technique may not be observed with other relaxants
• atracurium and vecuronium have been little studied in children with CHD
• clearly this technique is suited to the sicker infants where extubation is not expected at the end of the procedure

Atrial Septal Defect

• usually benign in childhood, except for large defects
• even the larger ostium primum defects are usually only mildly symptomatic, unless associated with ***mitral regurgitation*** ($\equiv^{T}$ incomplete AV canal)
• PVR usually remains normal and PA pressures are only mildly elevated during L→R shunting
• the shunt flow is not large because the pressure differential is small
• thus, transient reversal of the shunt is common,

a. during valsalva & other causes of increased thoracic pressure
b. anaesthetic mishaps - loss of airway
- severe hypoventilation

• Eisenmenger's complex is rarely seen as repair is usually done at a younger age
• repair is uncomplicated except for ***sinus venosus*** type ASD, where insertion of a patch is required

Ventricular Septal Defect

• as PVR decreases after birth and RV pressures fall, the ***dependent*** shunt fraction increases
• large lesions generally result in severe CCF early in life
• small lesions behave more like a ASD
• failure is worse and anaesthetic problems greater when $\mathbf{Q_P:Q_S > 2:1}$
• only low concentrations of volatile agents will be tolerated without systemic hypotension
• when RV pressures are elevated, even small decreases in systemic pressure may result in reversal of the shunt, hypoxia and further myocardial dysfunction
• IV induction techniques using fentanyl or ketamine are generally safer
• regardless of the induction technique & size of the VSD, airway obstruction or hypoventilation may raise PA pressures & cause ***shunt reversal***
• should this occur during anaesthesia, management includes,

1. $FiO_2 = 1.0$
2. mild hyperventilation
3. systemic vasopressors - metaraminol, phenylephrine, noradrenaline

• the greater the tendency toward shunt reversal, the greater the anaesthetic technique should tend toward lowering PVR & maintaining systemic BP
• specific problems which may be encountered ***post-bypass*** include,

a. LV/RV outflow obstruction - redundant muscle
- position of the patch
b. arrhythmias - injury to the bundle of His
c. residual muscular type VSD's - undiagnosed pre-operatively
- difficult to find from RV
- difficulty in weaning
* $\mathbf{P_{PA}O_2 > P_{RA}O_2}$

Tetralogy of Fallot

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

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

▪ ***Clinical Features***

a.	symptoms	- syncope (~ 20%) - dyspnoea - growth retardation
b.	signs	- finger clubbing - PS murmur (no VSD murmur ∝ non-restrictive)
c.	ECG:	- RAH, LVH
d.	CXR:	- "boot-shaped" heart, ***coeur en sabot*** - large aorta ~ 25% have right sided aortic arch & descending aorta - small PA's, oligaemic lungs
e.	central catheter	* RAEDP ~ LVEDP
f.	complications	- endocarditis - cerebral abscess (10%) - other systemic emboli - thrombotic stroke (polycythaemia) - epilepsy - growth retardation - increased risk/severity of "tet" spells if uncorrected
g.	non-cardiac congenital anomalies occur in 20-30%	

▪ ***Management***

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

a.	neonate	- maintain oxygenation - PDA, high SVR until shunt
b.	severe infant	- Blalok-Taussig shunt
c.	child without shunt but increasing "spells"* β-blockers	

• operability is primarily dependent upon the size of the pulmonary arteries
• there are varying degrees of aplasia / dysplasia of the RV outflow tract & PA's
• small PA's preclude primary repair and mandate a palliative procedure

- ***cyanotic spells*** are associated with,
    1. cyanosis
    2. right to left 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 = increase SVR & reverse shunt
    3. morphine 0.1mg/kg - decrease sympathetic drive

- severe attack,
    1. 100% O_2
    2. morphine 0.1mg/kg - decrease sympathetic drive
    3. IPPV - increase P_{aO2}
       - decrease MRO_2
    4. paralysis - decrease MRO_2
    5. hypocapnia - pulmonary vasodilator
    6. maintain RV perfusion pressure
    7. peripheral vasopressors - metaraminol
       - increase SVR
       * avoid β-agonists
    8. pulmonary vasodilators
        i. ***PGI_2*** ~ 0.1-0.2 µg/kg/min
           - also systemic vasodilator
           - closes PDA
           - fever
           - decreased platelet adhesiveness
        ii. NO^- - inhaled at 10-80 ppm

- β-agonists may increase infundibular dynamic obstruction, reduce RV coronary perfusion and increase cardiac MRO_2 (tachycardia)
- ***propranolol*** may therefore be used for prophylaxis

Anaesthetic Considerations

• if patients are very cyanotic and polycythaemic preoperatively they will require adequate hydration ± venesection
• maintenance of systemic pressures & a patent airway are paramount during induction
• either fentanyl/pancuronium/100% O_2, or ketamine/100% O_2 are satisfactory
• increases in PVR with ketamine in children are probably 2° to hypoventilation and hypercarbia, not the drug itself
• ***cyanotic spells*** occurring during anaesthesia,

a. $FiO_2 = 1.0$
b. hyperventilation
c. systemic vasopressors - metaraminol, phenylephrine
d. morphine 0.1-0.2 mg/kg - less effective during anaesthesia, especially fentanyl/pancuronium
e. propranolol 0.005 mg/kg - initially, increasing as required
   - ***esmolol*** / atenolol
f. problems occurring during, after repair include,
   i. residual RV ***outflow obstruction***
   ii. pulmonary insufficiency - 2° to resection of the stenosis
   - 2° to a transannular outflow patch
   iii. partial or complete ***heart block***
   iv. residual VSD's
   v. ***RV dysfunction*** - ventriculotomy, hypoplasia of RV
   - RBBB, pulmonary insufficiency
   + dysfunction 2° to CPB

Coarctation of The Aorta

- wide range of presentations, from CCF/acidosis in the newborn to asymptomatic child
- the critically ill neonate will require **PGE_1** infusion to maintain patency of the ductus

  → increased systemic flow & reduction in acidaemia

- induction should be minimally CVS depressant in the face of the large R→L shunt & desaturation of the lower body
- some reserve is attributable to the better saturation of the upper body, however, depressants are still poorly tolerated
- the potential for shunting in the older child is virtually nil & induction is generally well tolerated
- retraction of the lung in the lateral position is poorly tolerated by the neonate, who's circulation is still transitional
- cross clamping of the aorta is generally well tolerated in the neonate, as the isthmus has little flow in this group
- in the older child, AoXC may result in upper body pressures ≥ 200 mmHg
- although aggressive R_X may appear indicated, high upper body pressures are required for perfusion of,
  i. spinal cord
  ii. liver & kidneys

- while the use of vasodilators improves perfusion of organs in the upper body, experimental work in dogs has resulted in ***paraplegia***
- pressure changes on removal of the clamp are generally small, providing clamping times are short and the child in not hypovolaemic

Patent Ductus Arteriosus

- also present from CCF in the neonate to asymptomatic older child
- induction must consider L→R shunt with relative pulmonary hyperperfusion
- diastolic hypotension and pulmonary "steal" from the systemic circulation may result in hypoperfusion of vital organs
- pulmonary hypertension and reversal of shunt flow are uncommonly seen in PDA
- transient reversal of shunt flow, with desaturated blood going to the lower body, may occur as in the neonate with persistent foetal circulation

Postoperative Management

1.	extubation	- degree of CVS insult - tendency to shunt reversal - effects of residual anaesthetic - extent of surgical insult (pain and hypoventilation)
2.	hydration	- chronic hypoxia and polycythaemia - nausea and vomiting
3.	inotropic support	
4.	***tamponade***	- post-CPB, especially small children
5.	sedation ± paralysis	- decrease MRO_2 - facilitation of ventilation - minimisation of shunt flow - stress response to ETT suction etc.

RESPIRATORY - NEONATAL CONDITIONS

Cleft Palate

•

Choanal Atresia

• failure of perforation of either the ***bony*** or ***membranous*** portions of the nasopharynx ~ 1:8,000
• rarely a problem if unilateral, but bilateral may result in acute respiratory distress at birth
• first presentation may be cyanosis and choking at feeds due to obligate nasal breathing
• surgical correction is indicated early
• associated congenital anomalies are rare & the remaining airway usually normal

Laryngeal / Tracheal Web

• an incomplete fibrous membrane may occlude the laryngeal opening, or more commonly the sub-glottic trachea
• signs of acute respiratory distress are present at birth, stridor being the most prominent
• occasionally an ETT with a stylet may be passed through the membrane
• if intubation is not possible then cricothyroid puncture may be required
• the obstruction is usually resected via a bronchoscope without sequelae

Congenital Subglottic Stenosis

• presentation is usually less acute than that for a web
• if the narrowing is severe, or involves a long segment of trachea, then management with cricothyroid puncture may be necessary
• long-term management involves repeated bronchoscopic resection, dilatations, stenting and steroid injection
• anaesthetic management at the first presentation is usually most critical
• induction and maintenance cf. upper airway obstruction, with assisted ventilation on volatile/O_2, followed by bronchoscopic evaluation
• ***tracheostomy*** is usually required with decannulation achieved by 2-5 years

Post-Traumatic Subglottic Stenosis

• usually due to the use of too small an ETT
• an adequate air leak should occur with 20-30 cmH_2O

- ***Subglottic Haemangioma***

• rarely presents at birth but develops over weeks as the tumour grows
• occurs more commonly than teratoma or cystic hygroma
• may present with respiratory symptoms, particularly intermittent ***stridor*** or noisy breathing, or acute respiratory arrest
• upper airway ***infection*** is more frequent and trauma from coughing may make intubation hazardous in terms of haemorrhage
• therefore, in cases of acute obstruction from an unknown cause, initial intubation should be accomplished with a smaller tube than usual

Oesophageal Atresia & Tracheo-Oesophageal Fistula

• first described in 1697, however, the first survivor and surgical repair was performed in 1939 by Leven (staged repair)
• the first primary repair was in 1943 by Haight & Townsey
• the incidence is ~ 1:3,000 live births, M ~ F with no racial prevalence
• the risk for subsequent siblings is ~ 0.5%
• when present as the sole anomaly, the survival rate postoperative repair is up to 100%
• associated congenital anomalies (~ 30-50%) include,

a.	CVS	~ 35%	- VSD, ASD, PDA - Fallot's tetralogy - aortic coarctation	(~ 2x ↑)
b.	MSS	~ 30%		
c.	GIT	~ 20%	- imperforate anus - duodenal atresia	
d.	GUS	~ 10%		
e.	craniofacial	~ 4%		
f.	VATER syndrome		- **V**ertebral anomalies or **V**SD - **A**nus, imperforate - **T**OF - **E**sophageal atresia - **R**adial or **R**enal anomalies	

- ***Classification***

a.	Type A	- oesophageal atresia / °TOF
b.	Type B	- oesophageal atresia / proximal TOF
c.	**Type C**	- oesophageal atresia / ***distal*** TOF * most common, opening proximal to carina
d.	Type D	- oesophageal atresia / proximal & distal TOF
e.	Type E	- no oesophageal atresia / TOF = "H type"
f.	Type F	- oesophageal stenosis / °TOF

- ***Clinical Presentation***

a. intrauterine - suspect with ***polyhydramnios***

b. at birth
- inability to pass ***NG catheter***
- presence of excessive, foamy oral secretions

c. neonatally
- usually at the first feed
- coughing, choking, cyanosis

d. with type C
- tympanic, overdistended abdomen
± diaphragmatic splinting and respiratory embarrassment

e. with type "H"
- may present in childhood / adolescence
- coughing spells
- recurrent respiratory infections
- "asthma"
* abdominal distension with ETT & IPPV

NB: if ***oesophageal atresia*** is shown by inability to pass a gastric tube, and there is air in the GIT on AXR, then a TOF ***must*** be present

- ***Preoperative Management***

NB: prevent / minimise ***respiratory complications***

1. standard ABC
2. stop oral feeds
3. nurse in the semi-upright position
4. continuous naso-remnant suctioning
5. thorough assessment of post-gestational age,
   * ***prematurity*** increases morbidity & mortality,
   i. hypoglycaemia, hypocalcaemia
   ii. IRDS
   iii. hyperbilirubinaemia
   iv. immunoparesis
6. exclude other anomalies,
   i. CXR, AXR, abdominal U/S
   ii. ECG, echo, ± cardiac catheter study
   iii. IDC, renal U/S ± IVP
   iv. check CBP, U&E's, LFT's
7. institute IVT, G&M ~ 250 ml WB

NB: if severely premature, then gastrostomy tube under LA, definitive repair when weight increases

- ***Anaesthetic Considerations - TOF***

1. ***hypothermia*** → increased MRO_2 & metabolic acidosis
   - operating room temperature ~ 27°C
   - warm all IVT + humidified, warmed gases, warming blanket
   - rectal temperature probe
2. ***monitoring*** - ECG, SpO_2, NIBP + IABP, $ETCO_2$, serial BSL's, Hct.
   - left axillary precordial stethoscope (***dependent lung***)
3. ? premedication with ***atropine***
4. ***suction oesophageal pouch***
5. ***induction / intubation***
   - i. ***gaseous induction*** & intubation under volatile
     - aspiration risk with deep volatile anaesthesia
     - careful aspiration & emptying of the oesophageal remnant pre-induction
     - if ***bronchoscopy*** required, use ***lignocaine*** spray to airway (4 mg/kg)
     - if IPPV → gastric distension then SV & assist, or gastrostomy ± catheter in distal remnant NMJ blockade & IPPV
     - if IPPV → adequate ventilation then NMJ blockade & IPPV
   - ii. ***awake intubation*** + N_2O / O_2 / volatile & gentle IPPV
   - iii. ***RSI*** - O_2 + fentanyl 1.0 µg/kg (? 3-5 µg/kg)
     - cricoid pressure
     - suxamethonium 1.5-2.0 mg/kg
     + N_2O (?) / volatile

NB:
1. deliberate ***endobronchial intubation***, then withdraw to two lung ventilation
2. beware of effects of volatiles in neonates
3. if ***premature*** then SpO_2 ~ 90-95% / PaO_2 < 100 mmHg
4. suction catheters for small tubes as secretions / bleeding → blockage
5. ***gastric insufflation*** & **N_2O** - gastric rupture and pneumoperitoneum
   - abdominal distension & decreased ventilation
   - decreased venous return and cardiac output

---

6. ***position*** → left lateral for right thoracotomy
   → early ligation of fistula & 1° anastomosis
7. ***IVT***:
   - i. maintenance ~ 4 ml/kg/hr 5% dextrose/saline
   - ii. operative & 3rd space ~ 6-8 ml/kg/hr Hartmann's
   - iii. blood T_X ∝ Hct £ **30%**
     losses ³ **15%** blood volume

Congenital Bronchogenic & Pulmonary Cysts

- isolation of primordial respiratory tissue from the developing lung
- presentation will be determined by size and location (carina > peripheral)
- both types have the propensity to cause acute respiratory failure & arrest
- many only present following rupture,

→ ***haemorrhage*** & ***bronchopleural fistula*** formation

- most bronchogenic cysts are centrally located, ~ 5-10% of posterior mediastinal masses
- carinal cysts may produce a syndrome similar to congenital lobar emphysema, 2° to mainstem bronchial obstruction & ***air trapping***
- paratracheal and parenchymal cysts produce a more insidious course, with ***infection*** & abscess formation
- management is directed at minimising enlargement of the cyst preoperatively,

1. maintain ***spontaneous ventilation*** whenever possible
   i. gaseous induction
   ii. SV anaesthesia until the chest is opened
   iii. once chest opened change to relaxant anaesthesia
2. if require IPPV use lowest peak inspiratory pressures possible
3. N_2O is ***contraindicated*** → Air/O_2
4. FiO_2 should be minimised according to blood gases
5. if cyst is fluid filled & soiling is possible
   i. deliberate endobronchial intubation
   ii. temporary surgical clamping of affected bronchus/artery
   iii. bronchial blocking with a Fogarty catheter

NB: neither anomaly is associated with other congenital disorders and the postoperative prognosis is ***good***

Congenital Lobar Emphysema

- usually unilateral, most commonly ***left upper lobe***
- there is co-existing incidence of ***CHD ~ 10%***
- clinical presentation is usually of,
    i. progressive respiratory distress
    ii. unilateral thoracic hyperexpansion
    iii. atelectasis of the unaffected lung
    iv. mediastinal shift ± cardiovascular decompensation

- primary management is as for pulmonary cysts above,
    i. spontaneous ventilation whenever possible
    ii. minimal airway pressures
    iii. low I:E ratio to allow expiration
    iv. N_2O is ***absolutely contraindicated***

Diaphragmatic Hernia

- incidence ~ 1:2,500 births, and ~ 25% of these have associated anomalies, most = CHD
- due to premature return of the midgut to the abdominal cavity
- therefore characterised by the presence of the abdominal viscera in the thoracic cavity
    → stomach, intestines, SI ± liver/spleen

- herniation is usually through the ***pleuroperitoneal sinus*** = foramen of Bochdalek, rarely through the substernal sinus (Morgagni)
- outcome is determined by the associated degree of ***pulmonary hypoplasia***
    i. ipsilateral volume ~ 10-20%
    ii. contralateral volume ~ 60-70% of normal

- severe cases, presenting within the first 6 hours of life still have a ***mortality ~ 50%***
- clinical presentation is usually soon after birth,
    a. cyanosis, tachypnoea & chest retractions
    b. decreased movement & breath sounds over the affected hemithorax
    c. shift of the cardiac impulse to the opposite side
    d. a scaphoid abdomen
    e. CXR: - gas filled loops of bowel in the hemithorax
        - mediastinal shift to the opposite side
        - lung hypoplasia
    f. AGA's: - severe hypoxaemia, acidaemia & hypercarbia
    g. rarely may present with signs 2° to intestinal obstruction

• infants with ***bilateral*** pulmonary hypoplasia & severe preductal shunting

1. severe, unresponsive hypercarbia
2. poor prognosis, mortality ≥ 90%

• acute respiratory decompensation may be the result of ***pneumothorax*** of the contralateral lung, before/during/after surgery
• the order of management is determined by the degree of cardiorespiratory distress
• for severe cases,

1. awake intubation to establish airway/ventilation
2. high RR, low TV/airway pressure ventilation
3. NG tube to deflate the stomach
4. IV access ± IA access (should not delay surgery)
   ± bicarbonate depending on the degree of acidaemia
5. surgical decompression as soon as practicable
6. preferred technique is opioid/relaxant
   i. N_2O is contraindicated
   ii. volatile agents are avoided due to CVS depression
   iii. volatile may be used as supplements after the chest is open

• one of the major determinants of outcome is the reversibility of ***pulmonary hypertension*** in the postoperative period
• this may result in ***persistent foetal circulation*** with progressive hypoxia & acidaemia
• thus, factors which increase pulmonary vascular resistance are avoided,

a. hypoxia, hypercarbia, acidaemia
b. hypothermia
c. nociceptive stimuli
   - post-surgical
   - ETT suctioning

• specific management to reduce PVR include,

1. hyperventilation →
   - hypocapnic pulmonary vasodilatation
   - risks of pulmonary barotrauma
2. prolongation of "anaesthesia"
   - opioid ± relaxant infusion
3. pulmonary vasodilators
   - PGE_1
   - tolazoline, isoproterenol
   - GTN, SNP

   → inconsistent results

   • multiple case reports with the use of ***nitric oxide*** & selective pulmonary vasodilatation
   • results appear promising, but formal studies of altered outcome are required
4. other management
   - inotropic support of CO/SVR
   - ECMO/ECLS

Bronchial Foreign Body

1. ***age group*** ~ 6 months to 3 years
2. ***presentation***
   - i. pharyngeal - coughing, gaging, dysphagia, drooling
   - ii. laryngeal - cough, stridor
   - iii. oesophageal - swallowing & wheeze
     - dysphagia, drooling
   - iv. tracheal - expiratory wheeze, cough
   - v. bronchial - wheeze, recurrent infection
     - persistent pneumonia, lobar collapse
   - • complete obstruction & respiratory arrest may occur at any stage
3. ***diagnosis***
   - • best made from the ***history***, usually choking while eating, and examination
   - • P-A and lateral CXR's only demonstrate radiopaque objects
   - • inspiratory and ***expiratory*** films may show localised ***air trapping***
4. ***non-operative management***
   - i. holding the child upside down while supporting the airway
   - ii. backblows
   - iii. finger sweep of the pharynx
   - iv. chest thrusts, and abdominal thrusts - Heimlich manoeuvre
     - • not recommended in infants < 12 months
     - • may be tried cautiously in the older child
   - v. direct laryngoscopy, bronchoscopy, and emergency intubation
5. ***bronchoscope***
   - i. Storz - 15 mm gas connector, closed system, well suited to SV
     - straight cylinder with eyepiece or integral telescope
   - ii. Negus - conical taper, wider proximally, M.Peacock says better for suction
     - open-ended, either nipple/hose connection or jet ventilation
     - better suited to paralysis and jet insufflation
6. ***SV anaesthesia***
   - i. keep child calm, distress will increases dynamic obstruction
   - ii. administer supplemental O_2 if tolerated
   - iii. secure reliabe IV access
   - iv. induce in theatre with surgeon readily available
   - v. SV induction with ***halothane & O_2***
   - vi. LA spray to airway
     - • lignocaine 4.0% topical ~ 4.0 mg/kg total
     - • dilute to 3-4 mls volume for administration
     - • sequential spraying of laryngeal inlet / cords / proximal trachea

GASTROINTESTINAL CONDITIONS

- general factors in the neonate include,
    a. often advanced obstruction
    b. sepsis and abdominal distension enhance ***respiratory embarrassment***
    c. marked intravascular ***volume depletion***, ***acid base disturbance*** and ***CVS depression***
    d. association with other congenital malformations

    NB: therefore they are frequently ***critically ill***

- anaesthetic management therefore requires,
    1. aggressive fluid and electrolyte resuscitation
    2. correction of acid-base disturbances
    3. frequent cardiorespiratory support pre/post-operatively
    4. GA which minimises CVS depression

Neonatal Emergencies

1. omphalocele and gastroschisis
2. hypertrophic pyloric stenosis *medical ***not*** surgical
3. duodenal atresia
4. atresia of the small intestine
5. malrotation and midgut volvulus
6. necrotising enterocolitis
7. Hirschprung's disease
8. imperforate anus

Omphalocele & Gastroschisis

a. incidence ~ 1:5,800 for omphalocele
~ 1:15,000 for gastroschisis

b. pathology
   i. failure of return of the yolk-sac membranes & gut to the abdominal cavity
   ii. thus, a membrane covers the herniated viscera which is morphologically & functionally normal
   iii. ? due to occlusion of the ***omphalomesenteric artery***
   → abdominal wall defect (R>>L)

c. associations ~ 40% are low birth weight infants
~ 30% are premature
- ***Beckwith-Wiedemann syndrome*** & omphalocele (organomegaly / hypoglycaemia)
- extrophy of the bladder
~ 10% also have ***CHD***

d. clinical - omphalocele usually ruptures with birth
- severe heat & fluid losses
→ ***hypovolaemia & hypothermia***

e. management - minimise further fluid / heat loss
- fluid resuscitation (Hartmann's/NSA) ~ 20 ml/kg then PRN
- NG tube for stomach decompression
? awake intubation / ? RSI
- muscle relaxants are essential
- ventilated postoperatively

f. complications - degree of evisceration determines respiratory compromise
- basal atelectasis & respiratory failure
→ either 1° or staged repair
- those associated with ventilation
- those associated with premature LBW infants
- those associated with TPN

Hypertrophic Pyloric Stenosis

a.	incidence	~ 1:300-500 births - M:F ~ 4:1 - more common in first born male infants & affected parents
b.	pathology	- hypertrophy of the ***muscularis*** layer - irritation & mucosal oedema with gastric outlet obstruction - fluid and electrolyte depletion → hypochloraemic, hypokalaemic ***metabolic alkalosis*** hypovolaemic & mildly hyponatraemic ~ 40% will have "near-normal" U&E's - urine [Cl^-] > 20 mmol/l → good marker of resuscitation ? jaundice ≤ 8% ∝ ↓ glucuronyl transferase & starvation
c.	clinical	- usually by 4-6 weeks of age - projectile vomiting following feeds - "olive-sised" epigastric mass * barium NG study if equivocal (U/sound)
d.	anaesthesia	- not a surgical emergency → fluid & electrolyte resuscitation * anaesthetic considerations for the ***neonate*** - NG catheter suctioning - NB use of barium, as this may produce aspiration pneumonitis - RSI & relaxant anaesthetic or ? awake intubation - volatile vs. narcotic no real difference - obstruction is incomplete, N_2O OK - extubation should be done awake

Duodenal Atresia

a.	incidence	~
b.	associations	- high incidence of other malformations - especially CHD & trisomy 21 ± oesophageal atresia & imperforate anus
c.	pathology	? failure of recanalization - some are extrinsic compression from an annular pancreas
d.	clinical	- maternal polyhydramnios - bilious vomiting soon after birth
e.	AXR:	- "double-bubble" stomach & duodenum & absence of intestinal air
f.	anaesthesia	- high aspiration risk, even with NG tube - fluid and electrolyte abnormalities - RSI / ?awake intubation - NB: associated anomalies

Small Intestinal Atresia

a.	associations	- low incidence of other malformations - malrotation & volvulus - abdominal wall defects - prematurity
b.	pathology	- usually mesenteric vascular occlusion - not usually with defective embryogenesis
c.	clinical	- maternal polyhydramnios - bilious vomiting soon after birth - abdominal distension & dehydration greater than that with duodenal atresia
d.	AXR:	- "double-bubble" stomach & duodenum & absence of intestinal air
e.	anaesthesia	- high aspiration risk, even with NG tube - fluid and electrolyte abnormalities - RSI / ?awake intubation

Malrotation & Midgut Volvulus

a.	associations	- low incidence of other malformations - prematurity
b.	pathology	- incomplete migration from the yolk-sac - rotation around the fixed mesentry with vascular obstruction ± formation of atretic segments - ileocecal valve → RUQ
c.	clinical	- rapidly increasing abdominal girth ± passage of blood at stool → need for urgent surgery - fluid & electrolyte losses ± maternal polyhydramnios
d.	AXR:	- "double-bubble" stomach & duodenum & absence of intestinal air
e.	anaesthesia	- high aspiration risk, even with NG tube - fluid and electrolyte abnormalities may need to be corrected intraoperatively - RSI / ?awake intubation - relaxant anaesthetic, N_2O contraindicated - long-term ICU / TPN postoperatively

Necrotising Enterocolitis

a. onset
- usually within the first 2-3 weeks
~ 16% occur on the first day

b. ***risk factors***
- low birth weight infants
- PROM, chorioamnionitis
- birth asphyxia
- shock (any cause decreasing GIT perfusion)
- respiratory distress syndrome
- PDA
- recurrent apnoea
- UA/UV catheterization, hypertonic fluids
- feeding with hyperosmolar formulas

c. signs
- abdominal distension
- reflux, vomiting, bloody diarrhoea
- apnoea spells & bradycardia
- ***thrombocytopaenia*** is often present

d. AXR:
- fixed loop dilatation
- mucosal oedema
* ***pneumatosis intestinalis***, intramural air
- if severe may →air in portal vein

e. management

i. early * prior to necrosis and perforation
- nil orally, N/G suctioning
- IV fluid & electrolyte replacement
- antibiotics

ii. surgery * progression to perforation or peritonitis / uncontrolled sepsis
- thus requires repeat AXR's
- stools monitored for occult blood
- abdominal wall oedema / hyperaemia

iii. GA ~ peritoneal burn
- fluid and electrolyte resuscitation
- ≥ 1 blood volume of FFP may be required
- therefore need "large" bore IV access
- platelets & FFP for coagulopathy, DIC
- correction of acid-base, hypocalcaemia
- placement of arterial & CV catheters
- inotropic support (usually dopamine)

f. prognosis
- with medical therapy alone is very good
- a number will develop colonic strictures
- mortality is high with perforation

Meconium Ileus

• ~ 10-15% of patients with ***cystic fibrosis***
• symptoms of GIT obstruction begin soon after birth
• presents as either terminal ileal obstruction or as intestinal atresia
• thus, all infants with SI atresia should be screened for CF
• most cases can be managed with hyperosmolar enemas (gastrografin)
• this causes 3rd space loss and may necessitate IVT
• occasionally require operative removal & gut irrigation with acetylcysteine

Hirschprung's Disease

• absence of the ***parasympathetic ganglion*** cells of Meissner's and Auerbach's plexus
• non-peristaltic segment varies in length, usually extending proximally from the rectosigmoid, with a tonically contracted anal sphincter
• those with functional obstruction present in the neonatal period
• others present with increasing constipation
• progression results in "toxic megacolon" & entercolitis of the normal bowel, with secondary septicaemia & endotoxaemia
• corrective measures to restore IV volume and treat sepsis are essential prior to surgery
• once septic shock supervenes mortality increases to ~ 30-35%

Imperforate Anus

• ~ 25-75% have other associated ***congenital anomalies*** and these present the problems to anaesthesia (apart from being a neonate)
• ~ 10% have ***oesophageal atresia ± TOF***
• some will have the ***VATER*** constellation of symptoms,

v.	**V**ertebral anomalies	or **V**SD
a.	**A**nus, imperforate	
t.	**T**OF	
e.	**E**sophageal atresia	
r.	**R**adial	or **R**enal anomalies

• ***genitourinary*** anomalies are the most frequent association but only the most serious (bilateral dysgenesis / agenesis) significantly increase mortality
• most undergo colostomy and definitive repair at a later stage

NEUROSURGICAL ANAESTHESIA

Physiology

- see neuroanaesthesia notes
- in the presence of open fontanelles & cranial sutures, these effects may be accompanied by an increase in ***head circumference***
- ultimately herniation of the brain occurs, even in the presence of open sutures
- in children the signs of raised ICP, hypertension / bradycardia and pupillary dilatation may not occur, or may occur at relatively normal ICP
- when associated with raised ICP they are generally late & ominous signs
- ***papilloedema*** may not be present, even in children dying from intracranial hypertension
- a diminished level of ***consciousness***, especially if associated with abnormal posturing or response to painful stimuli, frequently indicates raised ICP
- as for adults, normal ICP ~ 15 mmHg
- children with a "normal" baseline may exhibit pathological pressure waves, indicative of reduced intracerebral compliance
- the later being a better guide of intracerebral pathology
- monitoring is similar to adults, except that,

    a. intraventricular catheters may be difficult to insert in the presence of raised ICP & small ventricles

    b. SA bolts are difficult to stabilise in small children due to the thin calvarium

Anaesthetic Management

- preoperative history should include,

    a. allergies - important for contrast agents

    b. drugs - anticonvulsant agents $\rightarrow$ enzyme induction

- examination should concentrate on,

    a. evidence of raised ICP

    b. ability to maintain airway reflexes, cough

    c. muscle power and pulmonary reserve

    d. associated conditions
        - SIADH, electrolyte disorders
        - dehydration
        - malnutrition, muscle wasting

- laboratory investigations may include,

    a. U&E's, LFT's

    b. CBP, G&M

    c. CXR, ECG

Anaesthetic Management

• premedication is frequently withheld
• possible exceptions include ***aneurysms***, where minimal agitation is essential
• the effects of opioids and sedatives in patients with raised ICP are unpredictable & these should therefore be avoided
• the vagolytic effects of atropine have frequently dissipated by the time of surgery & if required, atropine may be administered with induction
• as an alternative to IM administration, rectal methohexitone (20-30 mg/kg), will result in good sedation
• PR barbiturates probably lower ICP cf. IV administration providing the airway is maintained
• MOX should be avoided in patients with known seizure disorders, or in those at significant risk of such

• minimal ***monitoring*** should include,

1. stethoscope - precordial or oesophageal
2. temperature
3. ECG
4. NIBP ± IABP
5. FiO_2, SpO_2, $ETCO_2$

6. NMJ blockade
7. precordial doppler
8. EEG, evoked potentials
9. ICP
10. urine output

→ same indications cf. adults

• use of the sitting position is less frequent in paediatric neurosurgery, and is not used at all in many centres
• extremes of head position may result in malposition of the ETT
• appropriate padding/supports for the eyes/ears are required
• freedom of motion of the abdominal wall is essential in the prone position
• the risk of ***air embolism*** is proportional to the height between the heart and the operative field
• however, these have occurred in the supine position, especially in small children during craniotomy

• the relative sensitivity of monitoring to detect air embolism is,

1. transoesophageal echo
2. precordial doppler 4-5th ICS ? R > L
3. $ETCO_2$ ~ PAWP
4. RAP
5. BP
6. oesophageal stethoscope
7. respiratory pattern
8. ECG

• a ***precordial doppler*** should be used in all patients in the sitting position ± a CVC line positioned just above the RA
• position of the CVC catheter can be made by CXR, ECG or by injection of NaCl or CO_2 and monitoring of the doppler
• administration of CO_2, as for adults, may not be benign in children with potential for R→L shunts (embolism → increased PVR)

Management of Air Embolism

1. notify the surgeon → cover the operative field with NaCl
2. FiO_2 = 1.0 (cease N_2O)
3. lower the operative field ~ heart
4. aspirate air from the CVC, if present
5. CPR if no CO / severe hypotension
6. IVT - volume ± vasopressors

Induction

• ideally any child with raised ICP should have an IV induction
• if attempts to secure access are met with extreme difficulty, then the child probably has sufficient intracranial compliance to tolerate a gentle gaseous induction
• voluntary hyperventilation with O_2 and mannitol may be used in the cooperative child
• the ***barbiturates*** are the agents of choice due to their beneficial effects on $CMRO_2$, CBF and ICP
• small supplementary boluses are effective just prior to intubation
• ketamine should be avoided as it has been associated with sudden dramatic rises in ICP
• ***suxamethonium*** causes a small, but clinically insignificant rise in ICP
• if immediate control of the airway in not required then non-depolarising agents may be used to facilitate intubation

• mechanisms for decreasing the rise in ICP associated with intubation include,

1. supplemental doses of barbiturate (1-2 mg/kg)
2. rapidly acting opioids (fentanyl 1-2 µg/kg) ?? need 3-5 µg/kg
3. lignocaine (1.5 mg/kg) several minutes prior to intubation
4. controlled hyperventilation
5. osmotic agents (mannitol 0.25-1.0 g/kg)
6. loop diuretics (frusemide 1 mg/kg)

• rapid administration of mannitol may result in vasodilatation and a decrease in MAP and CPP
• renal dysfunction may result if the plasma osmolality rises above 340 mosm/l

• if surgery is elective and there is no significant increase in ICP, then induction may be facilitated by either,

1. gaseous induction with halothane & deep intubation, or
2. rectal administration of barbiturates

NB: the deep planes of volatile anaesthesia required for intubation simultaneously decrease MAP and increase ICP, with a large decrease in CPP

Maintenance / Emergence

• all of the volatile agents are vasodilators and high concentrations should be avoided in the presence of raised ICP
• at similar depths of anaesthesia, ***isoflurane*** results in the smallest increase in CBF
• N_2O may also increase ICP in patients with poor intracerebral compliance
• however, this effect is easily prevented by the concomitant use of barbiturates or diazepam

• awakening and extubation should also be atraumatic and is facilitated by the same manoeuvres used prior to intubation
• NMJ blockade should always be reversed since residual blockade may result in significant hypercapnia

Craniotomy

• brain tumours are second only to leukaemia in childhood cancers
• they are the most common solid tumour and most are ***posterior fossa***
• when proximal to the pituitary / hypothalamus there may be associated hormonal disturbance
• brainstem manipulation may produce CVS instability intraoperatively, or result in postoperative neurological deficit

Head Injury

- accidents, with head injury, are the leading cause of ***death*** in children
- small children frequently have associated ***spinal cord injury without radiological abnormality***
- therefore, great care should be taken with intubation / positioning
- the cribriform plate is easily fractured & nasal intubation should be avoided in the presence of a midfacial injury, or nasal discharge of blood or CSF
- in contrast to adults, head injury is infrequently associated with intracranial haematomas
- also hypotension may result from head injury alone, or from associated bleeding from scalp lacerations

Craniofacial Deformities

- craniosynostosis, alone or in association with facial anomalies (Apert's, Crouzon's syndromes), may require extensive surgery with massive blood loss
- intracranial hypertension may occur if there are multiple suture fusions

Hydrocephalus

- to allow for growth, the distal end of the shunt is usually placed intraperitoneally
- raised ICP is usually present preoperatively

Dyspharism

- in the spine (meningomyelocele) or brain (encephalocele) are usually associated with hydrocephalus and neurological deficit
- hydrocephalus results from a coincident ***Arnold-Chiari malformation*** (downward displacement of the brainstem 2° to an abnormality of the cervical spine)
- extremes of head position may result in brainstem compression similar to that produced by posterior fossa tumours
- vocal cord paralysis, leading to stridor & upper airway obstruction may occur
- other congenital anomalies may occur but are not common

REGIONAL ANAESTHESIA

- as a supplement to GA, regional anaesthesia,
    a. decreases the total amount of GA required
    b. hastens wake-up times
    c. provides for a more rapid & pain free recovery

- indwelling catheter techniques are capable of providing continued relief from acute pain
- cited ***disadvantages*** include,
    a. performance of a second anaesthetic technique & associated risks,
        i. LA toxicity / allergy
        ii. nerve / vessel / pleural / dural damage
        iii. haematoma, infection
    b. as most require the prior administration of GA, there is the need for skilled assistance

- contraindications to RA include,
    a. infection at the proposed site of blockade
    b. systemic sepsis & catheter techniques
    c. systemic coagulopathy
    d. allergy to LA's
    e. anatomical abnormality at the site of blockade
    f. patient / parent refusal

Pharmacology of Local Anaesthetics

- toxicity is dependent upon the total dose and the rate of absorption
- thus, the site of administration determines the maximal dose
- the highest blood levels are recorded after,
    1. intrapleural
    2. intercostal
    3. tracheal (topical) administration

- the free plasma level is also determined by,
    1. protein binding
    2. volume of distribution
    3. plasma clearance

• neonates and infants ≤ 3 months have immature metabolic pathways and significantly reduced hepatic blood flow
• most LA's are bound to a_1-***acid glycoprotein*** and ***albumin***, both of which are reduced in neonates
• therefore, toxicity is more prone to occur with the amide LA's, especially ***bupivacaine***
• the ester agents are metabolised by plasma cholinesterase
• this is present at ~ ½ the adult level, however clearance is not prolonged
• approximate elimination half lives in ***children*** are,

a. lignocaine ~ 2 hrs (~ 1.8 hrs)
b. bupivacaine ~ 4.5 hrs (~ 3.5 ± 2 hrs)
   • neonates ~ 8.1-14 hrs

• plasma levels following caudal administration of 1 ml/kg of,

a. lignocaine 1.0% ~ 1.25 µg/ml (10 µg/ml)
b. bupivacaine 0.25% ~ 2.0 µg/ml (4 µg/ml)

NB: these are well below the toxic levels (*) for adults
CECANZ, toxicity level neonates ~ 2.0 µg/ml

• for continuous techniques, supplemental doses ~ 0.5-0.6x the initial dose are recommended
• for bupivacaine £ **0.4 mg/kg/hr** total dose (70 kg → 28 mg ~ 0.25% at 13 ml/hr)
• toxic side effects are similar to those seen in adults,

a. CVS collapse, arrhythmias
b. seizures, coma, respiratory failure

NB: these may be ascribed to other factors in anaesthetised children

• children with ***cyanotic CHD*** (R→L shunts) are particularly susceptible to lignocaine toxicity

Agent	Dose limit[1]	Duration
2-chloroprocaine	20.0mg/kg	short
procaine	10.0mg/kg	intermediate
lignocaine (+ adrenaline)	7.0 mg/kg	intermediate
mepivacaine	7.0 mg/kg	intermediate
tetracaine	1.5 mg/kg	long
bupivacaine	3.0 mg/kg[2]	long

[1] based on lean tissue mass

[2] Berde < 6 months < 1.5 mg/kg < 0.2 mg/kg/hr
> 6 months < 2.5 mg/kg < 0.4 mg/kg/hr

Test Doses

- ***adrenaline*** 5 µg/ml was used as a marker of IV administration
- recent studies have shown that this will ***not*** reliably produce tachycardia in all halothane anaesthetised children
- isoprenaline may be a more reliable agent, however studies confirming this are still lacking
- the safest procedure is to use ***fractionated doses***

Caudal Epidural Anaesthesia

- most commonly performed regional technique for urologic, orthopaedic and general surgical procedures below the diaphragm
- has also been used for continuous sympathetic blockade in children with intense vasoconstriction (purpura fulminans)
- complications are rare, and unlike adults, children < 8 years rarely develop hypotension, even with thoracic levels of blockade
- dural puncture is uncommon as the dura, even in the small child, rarely extends beyond S_2
- potential problems include,

    1. local anaesthetic accumulation with continuous administration
    2. toxicity from intravascular injection
    3. urinary retention

- following a single shot caudal ~ 5% take longer than 8 hrs to void
- ***bupivacaine***, (1 ml/kg 0.25% or 0.5 ml/kg 0.5%), will provide 2-4 hours of surgical anaesthesia
- repeat doses ~ 0.6-0.7x the original are usually sufficient and do not lead to accumulation
- ***opioids*** are also effective via the caudal route, morphine having been shown to be effective for analgesia following abdominal thoracic and cardiac surgery
- Krane *et al*. have shown that 0.03 mg/kg ≡ 0.1 mg/kg for ***analgesia***, though the higher dose does prolong the duration (10 ± 3.3 vs 13.3 ± 4.7 hrs)
- the incidence of side effects was the same for the two groups, though, one patient in the higher dose group developed late respiratory depression
- a number of studies have demonstrated ventilatory depression for up to 18 hours following epidural morphine
- the risk of late depression can be avoided by the use of lipid soluble agents, such as fentanyl, with or without dilute bupivacaine
- these agents have a shorter duration of action and are frequently administered by continuous infusion, fentanyl 1-10µg/ml @ 0.2-1.0 ml/kg/hr
- monitoring for respiratory depression is still required

Lumbar Epidural Anaesthesia

- ***advantages*** compared to the caudal epidural approach,
    1. the volume of drug to reliably block the lower thoracic dermatomes may be excessive
    2. of potential faecal soiling with catheter placement
    3. the relative resistance to lumbar epidural blockade of the L_5-S_1 nerve root is not observed in paediatric patients
    4. the low incidence of hypotension in young children (< 8yrs)

- there are a number of ***disadvantages***,
    1. placement is technically more difficult cf. caudal
    2. greater risk of dural puncture & headache
    3. small potential for nerve root / cord damage

Subarachnoid Anaesthesia

- fell into disrepute early 1900's but repopularised for inguinal hernia repair in neonates
- following the administration of volatile agents, opioids, sedatives or ketamine, many newborns are at risk of developing ***apnoea***, especially,
    a. newborns < 60 weeks postconceptual age
    b. those born prematurely
    c. those with a history of neonatal apnoea

- spinal and caudal anaesthesia ***may*** produce less apnoea and thus be safer
- however, large controlled prospective studies have not yet been done and at risk infants should still be admitted for overnight observation
- during IH repair, spermatic cord/peritoneal traction requires spinal blockade to the thoracic dermatomes, or a very cooperative surgeon
- the block is best performed in the lateral position with the legs flexed and the neck ***extended***
- the electrocautery plate should be place prior to blockade, as raising the legs after administration may result in rostral spread
- in the neonate the cord extends to L_3 and dural entry above L_4 may be associated with cord injury

Recommended Maximal Doses (mg/kg)

Drug		Spinal	Caudal/Lumbar	Peripheral
Lignocaine	0.5-2.0%	1-2.5	5-7[1]	5-7
Bupivacaine	0.0625-0.5%	0.3-0.4	2-3	2-3

[1] higher doses are recommended only with the use of ***adrenaline***

- ***Intercostal Block***

• administered during or following thoracotomy, these may,

1. reduce opioid requirements
2. improve postoperative respiratory function
3. erncourage early ambulation

• the usual choice is bupivacaine 0.5% with adrenaline ~ 1-5 ml/segment
• recent work has shown a more rapid rise of serum levels cf. adults
• the site of injection may be either paravertebral or mid-axillary
• the most common technique is to "walk-off" the rib inferiorly
• the associated problems include pneumothorax & excessive absorption

- ***Penile Block***

• useful for pain relief post circumcision, urethral dilatation or hypospadias repair
• usually use bupivacaine 0.25-0.5% ***without adrenaline*** ~ 1-4 ml
• landmarks are midline, ~ 1 cm above the symphysis at an angle of 30°, piercing the penile fascia

- ***Brachial Plexus Block***

• very useful in paediatric anaesthesia, advantages include,

1. ease of insertion
2. low incidence of side-effects, delayed morbidity
3. suitability for plastic & orthopaedic work to the hand/forearm

• there are various techniques, those relying on ***paraesthesia*** being relatively ***contraindicated*** in paediatric patients
• usually, the axillary artery is readily palpable & LA may either be placed in its proximity or it may be transfixed
• the need for subcutaneous infiltration to block the musculocutaneous & intercostobrachial nerves is quite variable
• PNS may be used, however frequently is not required
• a volume of 0.5-1.0 ml/kg of solution is generally adequate, with the total dose adjusted for age
• the use of bupivacaine in children has greatly reduced the need for postoperative opioids

- ***IV Regional Anaesthesia Bier's Block***

• first described in 1908
• provides rapid onset & brief duration of anaesthesia
• disadvantages include the possibility of LA toxicity 2° to torniquet failure and the need for IV access in an awake child
• the use of double torniquets reduces the likelihood of accidental toxicity
• lignocaine 3-5 mg/kg as the 0.5% solution is generally satisfactory

MALIGNANT HYPERTHERMIA

• ***autosomal dominant*** inherited metabolic defect, with reduced penetrance and variable expression
• some patterns of inheritance do not follow this, therefore ? transmitted by more than one gene & more than one allele
• estimated incidence is ~ 1:50,000-100,000 adult anaesthetics
• the reported incidence in ***children ~ 1:3,000-15,000***
• this large difference may be due to retrospective study design or the age at which most surgery is performed
• the incidence is greater with the use of halothane and suxamethonium
• the syndrome was first accurately described by Denborough & Lowell in 1960
• the mortality at that time was ~ 90%
• the recognition of ***dantrolene*** in prevention and management was made in 1979

• most of the study of the pathophysiology has been done in pigs
• the metabolic defect is in the transport of Ca^{++} across the ***sarcoplasmic reticulum***
• during normal contraction, muscle membrane depolarization reaches the terminal sac of the SR via the t-tubules and leads to release of Ca^{++}
• Ca^{++} is also released from the mitochondria and the sarcolemma
• the normal resting cytosolic $[Ca^{++}] \sim 10^{-7}$ M, which increases ~ 10x during depolarization
• the main defect lies in the inability of the SR to store Ca^{++}, with the cytosolic $[Ca^{++}]$ ~ 3-4x normal
• upon triggering MH this increases up to ~ 17x (~ 6x normal depolarization)
• this large increase in $[Ca^{++}]$ leads to,

a. ATP'ase activation with conversion of ATP to ADP
b. inhibition of ***troponin***, enabling muscle contraction
c. activates phosphorylase kinase & glycogenolysis → ATP & heat
d. increased mitochondrial $[Ca^{++}]$ & increased aerobic glycolysis

→ ATP depletion & increased muscle metabolism
anaerobic metabolism & lactic acidosis
high MRO_2, VCO_2, heat production

• heat dissipation & muscle energy requirements will initially be met but later blood flow will be shunted away from the skin in order to increase
• muscle blood flow → dramatic rise in core body temperature
• later, muscles swell with rhabdomyolysis and efflux of Ca^{++} and K^+
• the resulting hyperkalaemia is the major source of mortality
• Ca^{++}-channel blockers have produced evanescent elevations of K^+ in swine and are thus contraindicated in the management
• cardiac arrhythmias are frequent and relate to,

1. hypoxia, hypercapnia and acidosis
2. hyperthermia
3. autonomic hyperactivity

• if the patient survives the acute episode, other problems may occur,

1. consumption coagulopathy
2. haemolysis & haemoglobinuria ± ATN
3. myoglobinaemia & myoglobinuria ± ATN

• the cause of death is variable and relates to the time of death in relation to the onset of the disorder,

a. early (hours) - VF, hyperkalaemia
b. intermediate - following resuscitation
- pulmonary oedema
- coagulopathy
- acid-base / electrolyte imbalance
c. late (days) - MOSF, renal failure
- brain damage

NB: the height of the fever ***does not*** correlate with outcome

■ ***Diagnosis***

• clinical ***signs*** of MH include,

1. tachycardia
2. tachypnoea &/or respiratory acidosis
   → tachycardia & raised $ETCO_2$ under anaesthesia
3. metabolic acidosis
4. hyperthermia
5. hyperkalaemia
6. cyanosis
7. coagulopathy

• ***presentation*** forms of MH,

a. masseter rigidity
b. suxamethonium induced muscular rigidity
c. "full blown" syndrome - intraoperatively
- in recovery
d. intraoperative fever alone
e. neuroleptic malignant syndrome
f. heat stroke
g. episodic fever
h. SIDS

• masseter spasm, depending upon the criteria of diagnosis, may have a susceptibility to MH in 50-80% of cases

• it is more common in myotonia congenita and Deuchenne muscular dystrophy
• most authors would not treat rigidity after suxamethonium alone, but would closely monitor for hypermetabolism and have dantrolene available

■ ***Muscle Testing***

• excised muscle is placed on stretch in a bath at 37°C
• optimal length tension is established, then caffeine ± halothane are added
• the muscle is then stimulated supramaximally and the contracture amplitude measured,

a. halothane → increased
b. caffeine → reduced ***MH susceptible***

• the responses to caffeine 2 mmol/l and halothane ≤ 2% are the only tests that ***unequivocally*** discriminate between MH survivors and controls
• simultaneous exposure produces too greater an overlap
• the problem is the patient who is muscle biopsy negative but clinically positive
• a wide range of expression of the disorder has been observed in both pigs and humans
• thus, most laboratories trend toward false positive results as the consequences of a false (+)'ve are less than a false (-)'ve

Anaesthetic Considerations

• the majority of cases are unsuspected, however, there are a number of conditions associated with an increased risk of MH,

a. diseases almost certainly related = ***central core disease***
b. diseases possibly related
   i. Deuchenne muscular dystrophy
   ii. King-Denborough syndrome
   iii. other myopathies
      - Schwartz-Jampel syndrome
      - Fukuyama muscular dystrophy
      - Becker muscular dystrophy
      - periodic paralysis
      - myotonia congenita
      - SR-ATP deficiency syndrome & mitochondrial myopathy
c. diseases coincidentally related
   i. SIDS
   ii. neuroleptic malignant syndrome
   iii. others
      - lymphomas
      - osteogenesis imperfecta
      - glycogen storage disease

• other tests are non-conclusive but helpful in assessment
• serum CPK is elevated in 60-70% of MH patients even at rest

• the usefulness of this in assessment lies in the absence of other causes of elevation and on the degree of elevation
• in the absence of other explanations, a ≥ 10x rise in the clinical setting of hypermetabolism is diagnostic of MH until proven otherwise
• other proposed investigations include,

1. post-ischaemic tetanic stimulation = "torniquet test"
2. lowered CvO_2 in the ischaemic arm
3. halothane induced platelet ATP depletion

NB: results from these have not been consistently reproducible

Management - Acute

a. stop all ***triggering agents*** immediately
   * continue with safe agents if surgery cannot be immediately ceased

b. ***hyperventilate*** with **100% O_2**
   * use new soda-lime & change to "clean MH-machine"

c. administer ***dantrolene*** **2.5 mg/kg** immediately
   i. continue until vital signs normalise
   ii. total dose up to 10-20 mg/kg
   iii. continue with 1 mg/kg q6h ó post-operatively for 48-72 hrs

d. ***bicarbonate*** **1-2 mmol/kg** stat, then follow AGA's

e. initiate cooling
   - iced saline, cooling blanket
   - body cavity lavage
   - extracorporeal circulation

f. manage hyperkalaemia
   - HCO_3^-
   - insulin dextrose

g. manage arrhythmias
   - procainamide 3 mg/kg if persistent
   - to maximum of 15 mg/kg

h. monitoring
   - AGA's, $ETCO_2$, SpO_2, ECG, core T°
   - U&E's, Ca^{++}, CK, myoglobin
   - APTT/PT, platelets, FDP's

i. maintain urine output
   - IVT ± mannitol/frusemide

j. transfer to ICU
   - observe for 24-48 hours

k. counsel family
   ± investigate

NB: no anaesthetic should be given without access to 36 vials of dantrolene & a clean anaesthetic machine (Kaplan)

- ***Dantrolene***

• dantrolene takes ~ 6 minutes to have any effect
• continuation of any signs of persistent MH should be treated with further dantrolene
• the actions of dantrolene include,

a. no effect on NMJ transmission

b. muscular weakness, which may potentiate NMJ blockade
~ 5-15 mg/kg produces significant muscular relaxation

c. ? prevents the release of Ca^{++} from the SR

d. ? antagonises the effects of Ca^{++} at the actin/myosin - troponin/tropomyosin level

e. up to 15 mg/kg there is no significant effect on the CVS

f. up to 30 mg/kg there is no significant effect on respiration

NB: there is no evidence of ***toxicity*** when administered acutely

• the reduction in muscle rigidity results in rapid normalisation of serum biochemistry, especially hyperkalaemia, and cardiac function
• cardiac arrhythmias & arrest are almost always 2° to hyperkalaemia/acidosis, the myocardium is not directly involved in MH pathology
• these can usually be managed by treating the 1° disturbance
• $CaCl_2$ can be used as a last resort for hyperkalaemia
• Ca^{++}-channel blocking agents should not be used as they may result in cardiovascular collapse in the presence of dantrolene

Management - Elective

a. preparation of theatre personnel

b. a "clean" anaesthetic machine

c. a MH cart

i.	drugs	- dantrolene 36 vials + 1000 ml sterile H_2O - $NaHCO_3$ - dextrose 50% - mannitol 25%, frusemide - procainamide - chlorpromazine
ii.	equipment	- T° probes - NG tubes - soda lime for circle - disposable breathing circuit - urinary catheters - blood collection tubes - syringes/needles, AGA syringes - CVC cannulation equipment

d. use of safe anaesthetic agents

e.	monitoring	- $ETCO_2$, SpO_2, ECG, NIBP, FiO_2 - T° core & peripheral ± urinary catheter
f.	adequate recovery	≥ 4 hrs duration → ward or home
g.	alert back-up support	- anaesthetic staff - local ICU

Anaesthetic Agents for MH	
Unsafe	**Safe**
all modern volatile agents • halothane = worst • enflurane • isoflurane • desflurane & sevoflurane	barbiturates propofol ketamine etomidate N_2O
	opioids droperidol benzodiazepines
suxamethonium	non-depolarising relaxants anticholinesterases anticholinergics
	local anaesthetics[1]
	catecholamines digoxin Ca^{++}

[1] amides raise $[Ca^{++}]_{ICF}$ but do not trigger MH, therefore, they may theoretically worsen an ongoing episode

MH - Prophylaxis

• though it would seem prudent to use prophylaxis in all patients, dantrolene may be associated with,

1. phlebitis
2. lethargy
3. nausea & vomiting
4. severe muscle weakness in some disease states
5. potentiation of neuromuscular blockade
6. uterine atony postpartum
7. placental transfer and neonatal hypotonia

• therefore, prophylaxis may be recommended for,

a. prolonged procedures ≥ 2 hours

b. physiologically stressful procedures

c. in the presence of underlying disease states which are intolerant of a hypermetabolic state or myoglobinuria

NB: dantrolene 2.5 mg/kg IV 30 mins to 2 hrs pre-anaesthesia
dantrolene 5 mg/kg ó 24 hours post-operatively

Management - Family

• most important is adequate information and support
• biopsy of the patient is reasonable after an appropriate interval
• biopsy of the remaining family members is ***not essential***, as most anaesthetists will treat them as susceptible irrespective of the biopsy result
• biopsy at the time of incidental surgery is therefore logical
• prior to biopsy dantrolene & droperidol should be avoided as they "normalise" the abnormal responses of MH susceptible individuals

BURNS

• results in destruction of part of the largest organ of the body, upon which we depend for,

a. themoregulation
b. fluid and electrolyte conservation
c. microbiological defence

NB: → ***systemic*** reactions to localised lesions

• paediatric patients have a larger SA/weight ratio and the extent of the burn is frequently underestimated

Rule of Nines				
	Adult	10-14 yrs	5-9 yrs	1-4 yrs
Head & Neck	9%	13%	15%	**19%**
Thorax	36%	32%	32%	32%
Legs (each)	18%	18%	17%	15%
Arms (each)	9%	9.5%	9.5%	9.5%
NB: * note the large area of the head/neck in the small child				

▪ ***Cardiac Effects***

• immediately following a significant burn CO is markedly reduced
• this may result from,

a. massive fluid shifts from the intravascular compartment
b. the compressive effects of circumferential burns impeding venous return
c. circulating myocardial depressant factor
   i. some patients have low CO despite high filling pressures
   ii. especially with extensive 3° degree burns

• 3-5 days after the injury there is a hypermetabolic state, when CO may be increased 2-3 fold, which persists for up to 3 weeks
• ensuing gram (-)'ve sepsis may result in depressed CO in those affected

- ***Respiratory Effects***

• thermal inhalational injury to the larynx/trachea may result in upper airway obstruction 2° to oedema formation
• the lower bronchi & alveoli may be damaged by inhalation of toxic fumes, such as nitrogen dioxide & sulphur dioxide, which form their corresponding acids in the tracheobronchial tree
• inhalation of products of cotton & wool result in formation of acid aldehyde, which may result in pulmonary oedema at ≤ 10 ppm
• combustion of polyurethane products releases hydrogen cyanide, which may lead to histotoxic hypoxia and death
• the overall effects of pulmonary inhalation are,

a. necrotising bronchitis with bronchial swelling
b. alveolar destruction with protein exudate & surfactant loss
c. reactive bronchospasm

→ ***bronchopneumonia*** & decreased pulmonary compliance

• all of these lead to V/Q mismatch, with ensuing hypoxia and hypercapnia
• this, combined with reduced cardiac output, leads to poor tissue oxygenation

- ***Renal Effects***

• tissue destruction results in myoglobinuria & haemoglobinuria
• the former is most common with electrical burns and the later with severe cutaneous burns
• hypotension and hypovolaemia will further aggravate the renal effects
• patients with ≥ 40% SA burns demonstrate renal tubular dysfunction
• even during hyperosmolar states there is no antidiuresis, suggesting a resistance to ADH and aldosterone
• episodic or persistent hypertension occurs in children, 2° to renin and catecholamine release

- ***Hepatic Effects***

• the liver may be damaged by,

1. hypoperfusion during the early phase
2. hypotension
3. inhaled or absorbed chemicals or toxins
4. sepsis

• during the hypermetabolic phase there is an increase in blood flow, gluconeogenesis, and protein turnover
• with the onset of sepsis, glucose output and alanine uptake may be sharply reduced, though, blood flow and oxygen utilization remain increased
• fatty infiltration has also been described
• altered protein levels, receptor sensitivities, and haemodynamic parameters make the effects of drugs unpredictable in the severely burned patient

- ***Central Nervous System***

• this may be adversely affected by hypoxic encephalopathy or inhalation of neurotoxic chemicals other factors are sepsis, hypovolaemia and hyponatraemia
• there is potential for cerebral oedema formation early in the course and evidence for raised ICP sought

- ***Haematological Effects***

• blood viscosity may increase 2° to haemoconcentration, due to fluid shifts, and due to alteration of plasma protein content
• anaemia may occur from RBC destruction and ongoing intravascular haemolysis, which is not uncommon
• thrombocytopaenia may occur early, 2° to increased aggregation in the lung
• this is followed by an increase in platelet numbers at 10-14 days
• FDP's may be increased for the first 3-5 days, indicating a mild degree of DIC
• F-VIII and F-V are also increased 4-8x normal and may remain elevated for 2-3 months following a major burn

- ***Gastrointestinal Effects***

• prophylaxis against stress ulceration may require larger than usual doses of H_2-receptor antagonists
• transient ileus is common, and patients NG drainage should be employed due to the potential dangers of gastric aspiration
• TPN will be required in those unable to feed enterally

- ***Metabolic Effects***

• there is increased utilization of fat, protein and glucose
• this is accompanied by an increase in MRO_2 and VCO_2
• centrally or sepsis mediated hyperthermia also raises MRO_2, which may persist following healing of the wounds
• the plasma ionised Ca^{++} is low for up to the first 7 weeks post burn
• this is accompanied by ***hypophosphataemia*** and ***hypermagnesaemia***
• thus, the usually reciprocal relationship between ionised Ca^{++} and inorganic phosphate is not observed and supplemental Ca^{++} is essential

- ***Skin Effects***

• extensive destruction predisposes to hypothermia and fluid and electrolyte imbalance
• contraction of the forming eschar may result in compartment syndromes, or decreased thoracic compliance
• late effects include restrictive scar formation

- ***Circulatory Assessment***

• there are a number of formulas to estimate fluid requirements in burns,

1. Parkland ~ (4ml Hartmann's) x $SA_{\%burn}$ x TBW_{kg}
2. Brooke ~ (SPPS 0.5ml + Hartmann's 1.5ml) x $SA_{\%burn}$ x TBW_{kg}

→ plus normal ***maintenance*** requirements

• half of the calculated replacement + 24hr maintenance is given in the first 8 hours
• the remainder divided over the next two 8 hour periods
• the syndrome of hyperosmolar, hyperglycaemic, nonketotic coma may occur with burns and has a high mortality
• fluid replacement should aim at maintaining a good urine output
• ATN in the setting of severe burns has a very high mortality
• overzealous hydration may, however, result in pulmonary oedema, especially in the presence of an inhalational injury
• urine output should be carefully monitored, remembering the increased secretion of ADH and the possibility of tubular dysfunction
• severe cases may thus require CV ± PA catheter monitoring
• the daily evaporative losses from full thickness burn

a. child ~ 4000 ml/m^2
b. adult ~ 2500 ml/m^2

• concomitantly, in the child, ~ 2,500 $kcal/m^2/d$ of heat are lost
• minimising caloric expenditure and providing nutritional supplementation are therefore paramount
• there is a great tendency for children to become poikilothermic in the absence of the skin barrier
• therefore, concentrated efforts are required to prevent heat loss, especially during the initial resuscitation

Circumferential Burns

• large burns involving the thorax and abdomen have the potential to produce life threatening respiratory and cardiac compromise
• those involving limbs may result in compartment syndromes and subsequent loss of the limb
• these lesions frequently require urgent escharotomies

Electrical Burns

- the extent of injury is frequently not immediately obvious
- there is a high association with loss of limbs
- the surface injury may be small and the underlying muscle necrosis massive
- there is a high association with other injuries, including,
    a. long bone fractures
    b. myocardial injury
    c. visceral injury
    d. coma and seizures reflecting CNS damage
- muscle tissue surrounding bone is often more affected than superficial muscles
- massive myonecrosis may result in haemo/myoglobinuria and ATN
- late complications may occur up to 3 months following a major injury,
    a. neurologic dysfunction
    b. ocular damage
    c. damage to the GIT
    d. delayed haemorrhage from a large vessel
- the pattern of injury is comparable in its temporal relationship to that following irradiation

DAY SURGERY

- paediatric patients are generally good candidates for day surgery because,
    a. they rarely have any serious systemic disorder
    b. most surgical procedures are uncomplicated
    c. parental separation is minimised
    d. reduced exposure to hospital acquired infections

Patient Selection Criteria

- there must be a well defined selection scheme which all admitting surgeons adhere to
- for children there are three 1° factors requiring consideration,
    a. the ***patient***
        i. the child should be in good health, or have a chronic condition which has been stable for a defined period
        ii. some centres confine day surgery to ASA I&II
        iii. others allow ASA III&IV providing they have been well controlled & the planned surgery is not major
        iv. many chronic children prefer outpatient management
        v. the premature infant < 46 weeks PGA is unsuitable (see p.12)
        vi. safe ages vary with author → 46, 50 or 60/52 PGA
        vii. clearly these need to be individualised for the patient

    b. the ***parent***, must be willing, reliable and capable of following instructions

    c. the ***procedure***
        i. should involve minimal bleeding or physiological trespass
        ii. procedure length *per se* is not a significant drawback
        iii. most workers agree that almost any procedure not involving the cranial vault, thorax or abdominal cavities is OK
        iv. patients with infective lesions are not good candidates due to the requirement for a separate area in recovery

- recent experience is the USA has suggested that T&A's can safely be done on an outpatient basis
- there being little gained by observing these patients > several hours
- clearly this is operator dependent !

Preoperative Screening

NB: this is essentially the same as for inpatients

a. complete history and examination
b. appropriate laboratory investigations
c. specialist consultation where required
d. preoperative instructions
    i. fasting
    ii. medications
    iii. transport
    iv. activities

NB: → it is desirable to complete as many as possible ***prior*** to admission

- the level of screening prior to admission varies with institution, from,
    a. screening by the outpatient surgical clinic
    b. telephone history prior to admission
    c. medical/anaesthetic questionnaire by D/S nurse ± filtering to anaesthetic staff
    d. all patients seen by anaesthetic staff prior to admission

- on the day of surgery all patients should be screened for,
    1. recent acute illness
    2. adequate NPO status
    3. recording of vital signs

- despite adequate screening unexpected conditions will infrequently arise
- two common problems include,
    1. the "runny nose"
    2. the presence of a heart murmur
        i. even in the absence of any physical signs or symptoms, all such patients should be reviewed by a cardiologist
        ii. by definition day surgery is totally elective
        iii. children with confirmed lesions will then require antibiotic prophylaxis

- ***Childhood URTI'S***

• ~ 20-30% of all children will have a runny nose for a significant part of the year
• some will have simple vasomotor rhinitis, requiring no deferment of surgery
• those with a prodromal infectious condition should have their surgery postponed
• 3 recent prospective studies looking at URTI's in children,

1. DeSoto *et al.* - Anesth.1988
   - significant risk of arterial desaturation in the infected group
2. Cohen & Cameron *et al.* - A&A.1991
   - 2-7x increase in respiratory related adverse events
   - 11x increase if required ETT
3. Tait & Knight *et al.* - Anesth.1987
   - ***no difference*** in children undergoing myringotomies/tympanostomies

• simple nasopharyngitis, it can usually be safely rescheduled for ***1-2 weeks***
• "flu-like" illnesses involving the upper & lower respiratory tracts should be deferred ***~ 1 month***
• when anaesthesia must be performed,

1. interruption of vagally mediated reflexes with ***atropine*** is desirable
2. monitoring S_pO_2 intra & postoperatively is desirable

Preoperative Preparation

• the time interval between arrival and surgery should be kept to a minimum
• the majority of children do not require pharmacological premedication
• however, this will result in the occasional very frightened child
• ***preinduction agents*** refers to the use of rapidly acting agents shortly before induction of anaesthesia, eg.,

a. rectal methohexitone/thiopentone
b. intranasal midazolam
c. transmucosal fentanyl citrate - intranasal/transbuccal
d. ketamine 2 mg/kg IM

Induction

• smooth induction of anaesthesia in the unpremedicated child is probably the most difficult part of paediatric outpatient anaesthesia
• no single approach is superior,

1. ***inhalational*** induction
    i. most commonly used technique
    ii. halothane produces the most rapid & acceptable induction
    iii. with prolonged procedures, recovery is longer with halothane cf. isoflurane
    iv. nausea and vomiting are not common
    v. transparent masks & scented coatings may aid acceptance
    vi. slower in larger children

2. ***intravenous*** induction
    i. method of choice for most older children
    ii. relatively larger doses (5-6 mg/kg) may be required
    iii. studies with propofol indicate a smooth induction and a lower incidence of side-effects in recovery
    iv. however, pain on injection can be a problem

Perioperative Fluid Management

• the requirements for preoperative fasting have been revised, such that clear fluids are allowed up to 2-3 hours pre-op
• this has reduced the necessity for IV fluid therapy in all but extended procedures
• other advantages include,

1. less irritability prior to induction
2. less hypotension on induction
3. less concern about hypoglycaemia

• IVT should probably be instituted for

a. procedures > 60 minutes
b. those associated with significant blood loss
c. those with a high incidence of postoperative N&V
d. small children who have fasted for extended periods

Postoperative Analgesia

- the requirement for analgesia is dependent upon the nature of the surgery and the pain threshold of the patient
- it does not depend upon whether the patient is a day case
- methods for pain management include,

a. simple analgesics — < 6 months general nursing
   - paracetamol 20-30 mg/kg PR q4h
   - codeine 0.5 mg/kg q4h

b. opioid analgesics — - IV administration allows titration
   ? use short acting agents

c. regional analgesia
   i. placement after induction & prior to surgery reduces GA & opioid requirements
   ii. pain free recovery, allows earlier ambulation and return to a normal diet

Discharge Criteria

a. appropriateness and stability of vital signs
b. absence of respiratory distress
c. ability to take oral fluids
d. ability to cough / demonstrate a gag reflex
e. ambulation consistent with developmental age
f. absence of excessive nausea & vomiting
g. conscious state appropriate for developmental age