Epidemiology of childhood mortality

Infant mortality rate is defined as the number of deaths of children under one year of age in one calendar year per 1000 live births in the same calendar year

<table>
<thead>
<tr>
<th>Worldwide infant mortality</th>
<th>In Australia:</th>
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<tbody>
<tr>
<td>- In 1950: 180</td>
<td>- In 1900: 107</td>
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<tr>
<td>- In 2000: 67</td>
<td>- In 2000: 5</td>
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- Chances of dying as an infant decrease with increasing age:
  - 0 – 28 days: 3.4 infants in every 1000
  - 4-52 weeks: 1.6 infants in every 1000
  - 1-4 years: 0.2 infants in every 1000

Most infant deaths occur on the first day of life. Birthdays is the most dangerous day. As in every other statistic, infant males are more likely to die of any cause.

Causes of death in infancy

Newborns: issues of prematurity (eg respiratory prematurity, hemorrhage, immune etc)
1 month to 1 year old: SIDS (improving over the last few years)
After 1 yr of age: trauma; + infection in developing countries

Epidemiology of cardiac arrest in children

VERY FREQUENTLY DUE TO HYPOXIA
And the hypoxia frequently due to ASPHYXIA – eg. inhalation
  Usually, by the time they have a cardiac arrest, they have been hypoxic for a while and they have had enough metabolic acidosis and hypoxia to really screw all their other organs.
The next most frequent cause: circulatory failure
Meaning exsanguination, gastroenteritis dehydration, anaphylaxis, or similar.
Unlike adults: VERY RARELY due to primary cardiac disease

In summary:
- Respiratory obstruction eg. asthma, foreign body, croup
- Respiratory depression eg. convulsions, poisoning, raised ICP
- Fluid loss eg.blood loss or dehydration from burns or vomiting,
- Fluid maldistribution eg. sepsis, anaphylaxis or cardiac failure

Outcome of cardiac arrest in children

Invariably poor. Even worse if you arrive to the hospital apnoeic and pulseless. Typically, a period of acidosis and hypoxia has preceded the arrest anyway.

If CPR has been going on for 20 minutes or longer, there is NO chance of intact neurological survival.
How pediatric resuscitation differs from adult resuscitation

- WEIGHT
- ANATOMICAL SIZE AND SHAPE
- CARDIOVASCULAR, RESPIRATORY and IMMUNE FUNCTION
- INTELLECTUAL ABILITY AND EMOTIONS RESPONSE

**Weight**

3.2 kg at birth = 10.3 kg at year 1

BROSELOW Tape: uses height to estimate weight

Formula: \[ \text{weight} = 2 \times (\text{age} + 4) \]

**Airway**

- In infants, head is large, neck is short. In supine position, the neck will flex and the airway will obstruct.
- The mouth is small
- The teeth may be loose
- The tongue is relatively large
- Floor of the mouth is easily compressible (when chin lifting)

Under 6 months, EVERYONE is an obligate nose breather

So, if the nose is blocked with snot, the airway is obstructed.

After that, in the 3 to 8 yr olds, adenotonsillar hypertrophy becomes a problem

**EPIGLOTTIS IS HORSESHOE-SHAPED**

- It projects posteriorly at 45 degrees.
- This makes intubation more difficult

**LARYNX IS HIGH AND ANTERIOR:** level of C2-C3, instead of 5-6

The trachea itself is soft and short.

A foreign body is equally likely to go into the right or main bronchus

The cricoid ring is the narrowest part of the trachea, not the larynx

Here, it is lined with loose pseudostratified columnar epithelium and this means it is susceptible to oedema

Because this is where the cuff of the tube would lie, in order to avoid oedema, **CUFFLESS TUBES are preferred in prepubertal children**
Breathing
- In the infant, the **lungs are immature** and the total gas exchange area is ~ 3 m²
- **Smaller airways**, thus more easily obstructed
- **Greater reliance on diaphragmatic breathing**: and they have fewer of the type 1 slow twitch oxidative fibres; so they tire more quickly. The more preterm you are, the more quickly you tire.

Circulation
- At birth the ventricles are about the same mass
- 2 months after birth the left ventricle is twice the mass of the right ventricle
- **This is reflected in the ECG**
- As time goes on the
  - P wave enlarges
  - QRS complex elrages
  - QRS duration lengthens
  - P-Q interval duration lengthens

**CIRCULATING BLOOD VOLUME = 70-80 ml/kg**
That’s more than the adult! But the overall volume is less

Body surface area
- Surface area to weight ratio is high: it decreases as you age.
- This means the children are more prone to hypothermia
- At birth the head accounts for 19% of the surface area

Respiratory physiology
- Infants have greater metabolic rate and oxygen consumption
  - Respiratory rate is increased
  - However: **tidal volume remains the same to adulthood**: 5-7ml/kg
- At age 1, rate is 30-40
- At age 2, rate is 25-35
- 2-5, rate is 25-30
- 5-12, rate is 20-25
- In the newborn, the majority of the impedance to expansion of the chest is from the lungs: so, **surfactant is critical to normal respiration**
- Later on, in the adult the compliance of the chest wall comes to play a greater role
- Also:
  - At birth, the oxygen dissociation curve is shifted to the left (p50, the PO2 at 50% Sats is greatly decreased)- because 70% of their Hb is foetal Hb (disappears by 6 months)
  - **Prolonged ventilation of an infant → bronchopulmonary dysplasia: and the potential for oxygen dependence for a year, or perhaps even more!**
Cardiovascular physiology

- Infants have a
  - **ridiculously SMALL STROKE VOLUME:** 1.5ml/kg
  - **But: a HUGE CARDIAC INDEX:** 300ml/min/kg
  - Compartively, adolescents = 100ml/min/kg, and adults = 70-80

  - Stroke volume increases as the heart gets bigger;
  - This underlies the changes in heart rate seen in childhood
  - 110-160 at yr 1
  - 100-150 yrs 1-2
  - 95-140 yrs 2-5
  - 80-120 yrs 5-12
  - After age 12, HR approaches adult values

In infants, stroke volume is small and fixed: this means they can only vary the heart rate to increase their cardiac output.

**THIS MEANS:**

**response to volume therapy is blunted**

Stroke volume cannot increase greatly in response to fluid therapy.

B age 2, you get a more normal response to fluid challenges, and this phenomenon goes away.

**Blood pressure is low at birth, and rises towards adulthood**

This is because systemic vascular resistance is low at birth, and continues to rise.

From 70-90 systolic at birth, it rises to 80-95 by years 1-2, and 80-100 at years 2-5.

At 5-12, it is 90-110.

**Immune physiology**

- Breastfeeding provides protection against respiratory and gastrointestinal pathogens, but otherwise, placental transfer of antibodies stops and the baby takes 6 months to ramp up its own production of antibodies

From the free Google scan of “APLS: the pediatric emergency medicine resource” by Gausche-Hill and Strange, as well as the APLS handbook and www.alsg.org
The structure of pediatric resuscitation

- Things to mention as “**stuff I would ask for when waiting for the ambulance to bring me my critically ill child in a bat-call situation where retrieval have notified me well in advance**”
  - Get the kids age
  - Work out the weight
  - Ensure the help is available
  - Get all the likely fluids, drugs and equipment

**The sequence of events:**
- RESPONSE: unresponsive child = cardiac arrest algorithm
- Primary survey
- Resuscitation
- Secondary survey
- Emergency treatment
- Reassessment (“system control”)
- Ongoing stabilization

**BASIC LIFE SUPPORT**

Main thing to remember: hypoxia is the chief cause of cardiac arrest in children;

**thus:**

**OXYGEN IS THE CRITICAL STEP
NOT DEFIBRILLATION**

- **SAFE approach:**
  - Shout for help
  - Approach
  - Free from danger
  - Evaluate ABCs

- This is a lame way of going through the “DR” part of “DRABC”.
- One rescuer does useful resuscitative things, the other one summons the EMS team.
- If the solitary rescuer has no help after 1 minute of CPR, he’s got to pause and call the EMS team himself.
- **Shout for help** is not the same as get the EMS team. The EMS team has the defibrillator, and in this situation it is more important to get some oxygen into the child. **THUS:** first, you just yell. After a cycle of CPR you call for the EMS team.

When would it be appropriate to contact the EMS people FIRST, and then do CPR?

- **WHEN A HEALTH CHILD HAS A WITNESSED COLLAPSE**
- **WHEN A CHILD WITH KNOWN CARDIAC PROBLEMS HAS A WITNESSED COLLAPSE WITHOUT A BETTER EXPLANATION**
- Basically, whenever you think a defibrillator is critically important

- **?ARE YOU ALRIGHT?** the “R” in DRABC.
  - Weirdly, you ask this question, and THEN you hurt the child.
  - Response to pain is what you’re after, not words. Wiggling and crying is acceptable.
AIRWAY

- A child will frequently organize themselves into a comfortable posture, where they can breathe better (eg in epiglottitis they hold their head at a particular position)

- Attempts to improve a partially protected airway may result in a totally compromised airway. Don’t mess with their position.
  - HEAD TILT AND CHIN LIFT STILL APPLIES
  - The desirable positions are:
    - In the infant: NEUTRAL
    - In the child: SNIFFING

Patency of the airway is then assessed by LOOKING, LISTENING and FEELING for 10 seconds

- In the exam, they expect you to put your ear to the child’s face, looking over the chest to see if it rises and falls

As always, jaw thrust is there for any situation that calls for C-spine immobilization.

Nobody does the finger sweep anymore. You can clog the airway even more by forcing foreign bodies further into the airway.

BREATHING

- If making the airway patent does not result in some sort of breathing,

Up to 5 rescue breaths should be given to achieve 2 effective breaths

- In infants, your mouth goes over both nose and mouth.
- In children, it may be necessary to pinch the nose

  - All children are of different weird shapes
  - Slow breaths
  - Lowest pressure (airway pressure may be high because the airway is narrowed)
  - Chest must be seen to rise

If airway maneuvers do not yield a patent airway, you have to start thinking about foreign body obstruction
Circulation

- **ASSESSMENT:**
  - Definition of “circulatory failure” is no central pulse for 10 seconds
  - Or when the pulse is slow (< 60) and not perfusing
  - Or... by absence of “signs of circulation”, like cough and gag.
  - INFANTS: feel **brachial and femoral** pulses
  - CHILDREN: carotids, like adults

**UNNECESSARY CHEST COMPRESSIONS ARE ALMOST NEVER DAMAGING**

- Chest compressions should go 1/3rd of the depth
- Go at the **lower half of the sternum**
  - INFANTS:
    - use **hand-encircling technique: both thumbs** (when there are 2 rescuers)
    - use one-handed 2 finger technique when alone
  - CHILDREN
    - Use the heel of one hand, or both hands like adults

The rate at all ages is 100 per minute
The rate of compressions is 30 to 2 breaths for a single rescuer
The rate of compressions is 15 to 2 breaths for pair of rescuers

- INFANTS should be compressed with two fingers, or both thumbs (encircling technique)
- CHILDREN can have the heel of one hand, or the heels of BOTH hands for big ones

- After 1 minute of CPR, help should have arrived. If not, this is where you pause to call for an EMS team, to bring a defib et cetera.

- DO NOT interrupt compression for any other reason. Coronary perfusion pressure improves the longer the sequence of compressions

**Recovery position**

- There is no specific position for children
- Stable and lateral is the key

**Infection risk from mouth-to-mouth**

- There’re aren’t many reports of people catching things from mouth-to-mouth
- If you are mouthing a child with menigococcaemia, you should take **rifampicin or ciprofloxacin**
- You seem to be safe from Hep V or HIV- sputum and saliva are low risk fluids
- In fact you are more likely to give the child YOUR HIV.
- High risk fluids are blood, semen, vaginal secretions, amniotic fluid and peritoneal fluid
The child who has inhaled something and is CHOKING

- This is usually the disease of preschool.
- The most inhaled stuffs are foodstuffs.
- It's usually witnessed, and it's usually a sudden onset respiratory distress and stridor.

There are methods for clearing and airway obstruction: BUT if the child has epiglottitis or croup, that will actually destroy their airway, so:

**ONLY ATTEMPT THE FOLLOWING IF:**
- It's obvious that the diagnosis is foreign body obstruction
- AND there has been loss of consciousness, ineffective cough or apnoea
- AND the head tilt / chin lift / jaw thrust have failed to open the airway

- Encourage cough first. Spontaneous cough is more effective than any manoeuvre
- Only move on to manoeuvres if they have satisfied the above criteria, i.e. if they have lost consciousness or become apneic.

**CONSCIOUS CHOKING CHILD**
- Give 5 back blows
- Give 5 chest thrusts
- Repeat assessment

**UNCONSCIOUS CHOKING CHILD**
- Open airway, give 5 rescue breaths
- Start CPR: algorithm is the same.
- Each time reaths are attempted, look for a foreign body in the airway.

**Back Blows and chest thrusts: for INFANTS**
- Put the infant head down on your knee
- Deliver 5 blows to the back with the free hand
- If the obstruction is not relieved, turn them over on their back (still head down)
- Give them 5 chest thrusts: same as cardiac compressions, at a rate of 1 per second

**Back Blows and chest thrusts: for CHILDREN**
- Same as above, but you lay them over your lap

- Each time breaths are attempted, look in the airway – can you see a foreign body?
- Once the child is breathing spontaneously, put them in a recovery position of some sort
ADVANCED PEDIATRIC AIRWAY TECHNIQUES

- At some stage, somebody will ask you what equipment you would like
- This is the list:

  Face masks
  LMAs and Guedel/nasopharyngeal airways
  Self-inflating Laerdel bag
  Endotracheal tube
  Suction device
  Cricothyroidotomy kit

Oropharyngeal and nasopharyngeal airways
  - Oropharyngeal: from mouth to angle of mandible
    - May cause vomiting
  - Nasopharyngeal: from nose to tragus of ear
    - May cause hemorrhage
    - Contraindicated in base of skull fractures

Ok, so you have inserted one of these. Is it successful? If not, URGENTLY reassess your airway.

Laryngoscopy
  - The straight bladed laryngoscope lifts directly, by pushing on the epiglottis
    - This causes vagal stimulation because the epiglottis is innervated by the vagus nerve
  - The curved laryngoscope blade pushes the epiglottis forward
    - Inserted into the vallecula – glossopharyngeal territory, so no vagal stimulation occurs

ET tubes
  - Plain plastic tube before puberty, cuffed ones afterwards
  - Prepubescent cricoid rings are the narrowest parts

  - **There should be a leak:** otherwise the tube is in too tight and there will be oedema

  For over 1 yr olds:
  - Tube Size estimation:
    - **Internal diameter** = \((\text{Age divided by 4}) + 4\)
    - **Oral tube length** = \((\text{Age divided by 2}) + 12\)
    - **Nasal tube length** = \((\text{Age divided by 2}) + 15\)

  NEONATES: tube is 3 – 3.5 mm
  PRETERM: 2.5 mm

  **Magill’s forceps:** to angle the tube, or to remove foreign bodies
  **Tracheal suckers:** French gauge twice the mm diameter of tube; a 3mm tube = 6 french sucker

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Ventilating the child:

- Once again, someone is going to ask you what equipment you want.

  **Oxygen source**
  **Face masks**
  **Self inflating bag**
  **T-piece and open ended bag**
  **Mechanical ventilator**
  **Chest tubes**
  **Gastric tubes**

- **FACE MASKS:**
  - Circular ones give good seal
  - Worst case scenario, turn a Laerdal mask upside-down

- **Self Inflating Bags**
  - 250, 500, 1200ml varieties
  - Without reservoir bag, it’s difficult to give more than 50%

- **T-piece, open ended bag**
  - Only for kids under 20\(\text{kg}\)
  - Entirely useless unless there is a constant pressurized gas supply

- **You check that the tube is in position much like you would with an adult.**
  - Only for kids under 20\(\text{kg}\)
  - Entirely useless unless there is a constant pressurized gas supply
Primary assessment and resuscitation

- Resuscitation management should be concurrent

First, Response: yell at them and hurt them. Are they responding?

- **A and B**
  - Look listen and feel for airway obstruction/respiratory arrest
    - Chin lift/jaw thrust, airway accessories
    - Reassess patency
  - Assess the effort of breathing
    - Recession, stridor, expiratory noises, accessory muscle use
    - Count the respiratory rate
    - Auscultate breath sounds
    - Give rescue breaths, or bag/mask
    - Give high flow oxygen
    - Intubate if the occasion calls for it

- **C**
  - Take stock of the summation of circulatory findings:
    - Heart rate
    - Cap refill
    - Pulse volume
    - Blood pressure
    - Urine output
    - Give high flow oxygen
    - Gain venous or intraosseous access, ad give 20ml/kg of anything you like (crystalloid or colloid)

- **D**
  - “A” should have given you a reasonable AVPU score
  - Look at posture
  - Are they fitting?
  - Look at pupils
  - Check the sugar
  - **Intubate if scoring P or U**
  - **Give glucose if indicated**
  - Give Lorazepam IV, midazolam PO, diazepam rectally
Secondary assessment and emergency treatment

- **Respiratory**
  - Oxygen saturation
  - End tidal Co2
  - Peak flow
  - CXR
  - ABGs
  - Some focussed history
  - **Stridor of croup?** Nebulized adrenaline (5ml of 1:1000)
  - **Drooling?** = epiglottitis, get a senior anaesthetist to intubate
  - **Obvious foreign body inhalation?** Try FB manoeuvres, if they fail move on to direct laryngoscopy and Magills forceps
  - **Anaphylaxis?** IM adrenaline
  - **Children who wheeze have asthma. They need bronchodilators.**
  - **Infants who wheeze have bronchiolitis.** They just need oxygen

- **Cardiovascular**
  - FBCs
  - EUCs
  - ECG
  - Coags
  - **The first bolus of fluid has gone in.** Give another if the first one has failed to yield the desired effect
  - **If the second bolus of fluid is not working,** consider inotropes and CVP monitoring
  - **Give antibiotics to shocked children with no obvious source of fluid loss:** this is probably sepsis
  - **Anaphylaxis?** IM adrenaline 10mcg/kg
  - **ALPROSTADIL** is given to neonates whose shock is due to duct-dependent congenital cardiac disease

- **Neurological**
  - BSL
  - Urine tox screen
  - **Convulsions** = follow the status epilepticus protocol
  - **Raised ICP? Posturing?**
    - Intubate, aim PCO2 40-45
    - 30 degrees head up, head in line
    - Mannitol 0.25 to 0.5g/kg which is 1.25 to 2.5 ml mannitol 20%, infused over 15 minutes, up to a serum osmolality of 325
  - **Maybe they need cefotaxime or acyclovir**
  - **Consider giving naloxone**

- **Further history**
  - Drugs, allergies, etc
PAEDIATRIC CARDIAC ARREST ALGORITHM

So, your patient has arrested and CPR is in progress. The EMS team arrives and attaches defibrillator leads.

1) What's the rhythm? Shockable or non-shockable?

2) Shockable rhythm: VF or VT
   a. DC shock, 2J per Kg
   b. 2 mins CPR while intubating/getting access
   c. Check the monitor
   d. DC shock 4J per Kg
   e. 2 minutes CPR while intubating/getting access
   f. Check monitor
   g. Adrenaline, then DC shock 4J per Kg
   h. 2 minutes CPR
   i. Check monitor
   j. Amiodarone, then DC shock 4J per Kg
   k. 2 minutes CPR
   l. Check monitor
   m. Go back to adrenaline + DC shock, and so it continues

3) Non-shockable rhythm: Asystole or PEA
   a. High flow oxygen
   b. CPR while intubating/getting access
   c. Check the monitor every 2 minutes
   d. Adrenaline every 3 minutes

NON-SHOCKABLE RHYTHM

Asystole
   - The most common arrest rhythm
   - This is what happens to the hypoxic child: bradycardia, then asystole

PEA
   - This is typically a prelude to asystole
   - There may be an identifiable cause, e.g. tension pneumothorax

Secondary assessment
   - Detailed examination of the body
   - Identify wounds / bruising / swelling
   - Reexamine ABCs

4 Hs and 4 Ts
   - Hypoxia
   - Hypovolemia
   - Hypo/hyperthermia
   - Hypo/hyperkalemia

Tension pneumothorax
   - Tamponade (cardiac)
   - Thrombus (cardiac or PE)
   - Toxins (random drug)

ADRENALINE: 10mcg/kg
   AMIODARONE 5mg/kg
Respiratory failure in children

- Why are they susceptible?
  - Haven’t yet acquired immunity to the infections adults are immune to
  - Upper and lower airways are more narrow and thus more easily obstructed by swelling or secretions
  - Chest wall is more compliant and this reduces the efficiency of breathing if there is increased effort
  - In infants, end-expiratory volume is similar to closing volume and so they have small airway closure
  - There are fewer alveoli
  - The respiratory muscles are inefficient: fatigued rapidly
  - Pulmonary vasculature is muscular: pulmonary hypoxic vasoconstriction occurs more readily. This can lead to duct reopening and R→L shunting
  - Foetal hemoglobin is present up to 4th-6ths month of life; dissociation curve shifted to the left: less oxygen is delivered to the tissues because of fHb’s higher affinity for oxygen

Presentation

- Frequently, the only sign in infants is decreased feeding
- Stridor = upper airway obstruction
- Wheeze = lower airway obstruction
- Grunt = pneumonia

The child with stridor

- Most common causes:
  - Croup: viral laryngotracheaitis; barking cough, coryza, fever, hoarseness.
  - Croup: spasmodic; sudden onset, recurrent, with a history of atopy

- Less common causes:
  - Laryngeal foreign body; suddenness of onset
  - Epiglottitis; drooling, with muffled voice
  - Trauma: neck swelling, bruising
  - Retropharyngeal abscess: drooling, septic appearance
  - Airway burns: soot in the mouth
  - Diphtheria: travel to endemic area while unimmunized

- Gurgling is a bad sign. The child is either too drowsy or too tired to clear the secretions with cough.

Loud harsh stridor is usually croup.
Quiet stridor is usually epiglottitis.
Emergency Management of the child with stridor

- Your airway is compromised, but FUNCTIONING.
- Don’t destabilize it.
- Don’t upset the child.
- Get the parents involved.

Partial obstruction by secretions, or decreased level of consciousness

- Suction
- Chin lift / jaw thrust
- Intubate as needed

Croup

- This is usually viral, 95%
- Parainfluenza virus is the commonest pathogen
- Adenovirus is the next most common
- Peak incidence is in the 2nd year of life
- Typical features:
  - Hoarse barking cough
  - Harsh stridor
  - Hoarseness
  - Fever
  - Symptoms worse at night
  - Some children get SPASMODIC CROUP which is associated with ATOPY and ASTHMA
    - This variety may not have fever or coryza
    - The treatment for it is the same

IMMEDIATE MANAGEMENT

- Nebulize adrenaline, 5 ml of 1:1000 (high concentration)
- Neb it in oxygen
- Expect 30-60 minutes of relief while you get a team together

STEROIDS: improvement within 30 minutes!

- Dexamethasone IV or oral : 0.15mg/kg
- Budesonide nebulized

INTUBATE IF YOU NEED TO

- Median duration of ventilation is 3 days
- The younger the child, the longer they stay intubated
- Much smaller tube is required
- Prednisolone 1mg/kg every 12 hrs reduces the duration of intubation
**Bacterial tracheitis**
- “pseudomembranous croup”
- Staph aureus, strep, or H.Influenzae
- Copious secretions, and mucosal necrosis
- The child looks septic, with a croupy cough
- NO DROOLING unlike epiglottitis
- 80% of these kids will need to be intubated
- Fluclox and cefotaxime is called for

**Epiglottitis**
- The onset will be acute, over 3-6 hrs
  - **Unlike croup, cough is minimal or absent**
- The bug is H Influenzae, uncommon in immunized populations
- Most common in 1 – 6 yrs of age
- Typically, the child sits immobile, with their mouth open, chin slightly raised, drooling saliva.
- The child looks pale and septic
- Too painful to swallow or speak
- The epiglottis will be “cherry red”
  - **DO NOT LAY THEM FLAT** or they will die. While conscious, the child will sit upright while they are able.
  - You will need a senior anaesthetist to intubate
  - There will be a gas induction
  - You will need a smaller tube than normal
  - **Cefotaxime or ceftriaxone**
  - Most will be extubated in 24-36 hrs

**Foreign body**
- Inspiratory and expiratory films will show gas trapping
- They will have been eating or playing with small objects
  - “choking child” procedures
  - If they fail, direct laryngoscopy and Magills forceps
  - Get a senior anesthetist and an ENT surgeon
  - Direct bronchoscopic retrieval of the object is called for

**Anaphylaxis**
- Flushing, itching, facial swelling, urticaria
  - Apart from oxygen, INTRAMUSCULAR adrenaline is the key
  - You wont be marked down if you give nebulized adrenaline
  - You also give **chlorpheniramine and steroids**

**Weird stuff**
- Diptheria
- Infectious mononucleosis
- Retropharyngeal abscess
Emergency management of the Wheezing Child

- It's either acute severe asthma or bronchiolitis
  - Before 1 yr of age, its bronchiolitis
  - After 1 yr of age, its acute asthma

### Asthma in the child

- Your asthma is severe when:
  - It happens over several nights
  - Its symptoms are of a long duration
  - You have been to ICU before
  - You are not responding to treatment
    - Severity must be assessed every 4 hrs
    - Pulse, resp rate, SaO₂, use of accessory muscles, agitation
    - Also do 4 hrly peak flows

- **CHARACTERISTICS OF MODERATE ASTHMA**
  - Sats over 92
  - Peak flow over 50%

- **CHARACTERISTICS OF SEVERE ASTHMA**
  - Too breathless to feed or talk
  - Recession, use of accessory muscles
  - Resp rate > 30 (>50 in under-5s)
  - Pulse rate >120 (> 130 in 2-5s)
  - Peak flow <50%

- **CHARACTERISTICS OF LIFE THREATENING ASTHMA**
  - Depressed level of consciousness, or agitated
  - Exhaustion
  - Poor respiratory effort
  - Sats < 92%
  - Peak flow < 30%
  - Hypotension

Respiratory rate, degree of wheeze and pulsus paradoxus are NOT very good signs of severity.

- ABCs come first
- Give oxygen
- Give salbutamol via MDI and spacer
  - If asthma is life threatening, use nebulized salbutamol
  - If the breathing is crap, **give IV salbutamol**

- **Give steroids:** 3-5 day course; = faster recovery
  - No proven benefit of IV over oral steroids
- Give Atrovent as well
  - If the effort of breathing is still high, or they are exhausted, or LOC is low, you should consider bag-masking them
Treatment-refractory asthma in the child

Nothing is working?
• Continuous neb, …or

• Salbutamol infusion
  o Proven to offer an advantage over nebulizer

• “Summon experienced help” is the key phrase

• IV aminophylline 5mg/kg over 20 min
• ..then infuse it at 1mg/kg/hr, while monitoring the ECG
  o Seizures, vomiting or fatal arrhythmia follow rapid infusion

• magnesium sulphate, 25-40 mg/kg over 20 minutes

• intubate with ketamine IF:
  o progressive exhaustion
  o progressive deterioration in clinical condition
  o SaO2 dropping
  o PCO2 rising

• There is no evidence to support the use of Heliox or leukotriene antagonists

Asthma in the child who is beginning to improve: post crisis care

• Reassurance
• Stop IV salbutamol if sats are rising or recession is minimal / PEFR goes over 50% of predicted
• Change from continuous nebs to spacer MDI
• Reduce frequency of nebs

A little more on asthma

• URTIS are the most common precipitant
• Virus cause 90% of these
• Exercise induced symptoms: heat and water loss from the mucosa causes irritation and thus bronchoconstriction
Bronchiolitis in the infant

- RSV is the commonest cause - 75%
- 90% are younger than 1 year
- Wheezing is often present
- There is a history of clear coryza and dry cough
- The chest is hyperinflated
- In one third, the upper lobes are consolidated
- Breathing is disorganised, with periods of apnea
- Management is usually supportive

- **Initial management:**
  - Use sucker to suck out the nose
  - Give high flow oxygen
  - Maintain hydration and nutrition: sucking is too stressful
  - Monitor for hypoventilation in the under-2-month-old

- **Recurrent apnoea? Hypercapnia? Exhaustion? → INTOBATE THEM (2% will need this)**

> In bronchiolitis, there is no evidence for bronchodilators, steroids or antibiotics.

Most children will recover in 2 weeks
About 50% will have recurrent cough over the next 3-5 years
Very few will have bronchiolitis obliterans, and permanent damage

RISK FACTORS FOR A MORE SEVERE BRONCHIOLITIS:
- Younger than 6 weeks
- Prematurity
- Chronic lung disease
- Congenital heart disease
- Immune compromise

How do I tell if this is bronchiolitis or heart failure?
- There is NO MURMUR in bronchiolitis
- CXR = hyperinflation
- Heart looks SMALL in bronchiolitis
The Febrile Child with Respiratory Distress

- It’s probably pneumonia, unless it’s wheezes or drools.

- ABCs, as always
- Give oxygen, as always
- Give antibiotics- who cares if it’s viral
- Rehydrate
- Drain any effusion

PNEUMONIA IN CHILDREN:

- CHARACTERISTICS OF MODERATE ASTHMA
  - Sats over 92
  - Peak flow over 50%

- CHARACTERISTICS OF SEVERE ASTHMA
  - Too breathless to feed or talk
  - Recession, use of accessory muscles
  - Resp rate > 30 (>50 in under-5s)
  - Pulse rate >120 (> 130 in 2-5s)
  - Peak flow <50%

- CHARACTERISTICS OF LIFE THREATENING ASTHMA
  - Depressed level of consciousness, or agitated
  - Exhaustion
  - Poor respiratory effort
  - Sats < 92%
  - Peak flow < 30%
  - Hypotension

Respiratory rate, degree of wheeze and pulsus paradoxus are NOT very good signs of severity.

- ABCs come first
- Give oxygen
- Give salbutamol via MDI and spacer
  - If asthma is life threatening, use nebulized salbutamol
  - If the breathing is crap, give IV salbutamol

- Give steroids: 3-5 day course; = faster recovery
  - No proven benefit of IV over oral steroids
- Give Atrovent as well
  - If the effort of breathing is still high, or they are exhausted, or LOC is low, you should consider bag-masking them
The Child with Heart Failure and Respiratory Distress

- In older children, it is usually myocarditis or cardiomyopathy
  - That sort of thing tends to present with a marked tachycardia

- In infants it is usually due to structural heart disease
  - Falling pulmonary vascular resistance allows increased pulmonary blood flow
  - This causes an increasing left-to-right shunt in kids with a VSD, persistent PDA or truncus arteriosus
  - Thus, there is increased pulmonary congestion and heart failure
  - These kids tend to present with poor feeding, lethargy, sweating and breathlessness

**Duct – dependent heart disease**

- This is a rare thing indeed
- “duct-dependent” because the duct is the only reason there is any flow in the pulmonary or systemic circulation
- The **PULMONARY OBSTRUCTIVE LESIONS** are:
  - Pulmonary atresia, Pulmonary valve stenosis
  - Tricuspid atresia or severe tetralogy of Fallot
  - Sometimes, transposition of the great vessels

- **THE PROBLEM:**
  - There is NO WAY for blood to go from the right ventricle into the pulmonary circulation
  - Thus, the lungs depend on flow via the patent ductus, from the aorta.
    - Occasionally, there is so much aortic coarctation, that the systemic blood flow depends on the patent ductus

- **THE TYPICAL PRESENTATION**
  - These babies tend to present in the first week of life with cyanosis, and with cardiogenic shock
  - There is usually an enlarged liver
    - If the systemic circulation depends on the patent ductus, the child ceases to feed, and collapses.

- **HOW DO YOU KNOW ITS NOT BRONCHIOLITIS?**
  - Heart is enlarged, liver is enlarged, there is no cough or coryza.

**Emergency management of the child with heart failure**

- ABCs come first
  - Treat the cardiogenic shock
  - Give them oxygen via non rebreather mask

- **IF OXYGEN IMPROVES THEIR SATURATION**, it’s a left to right shunt
  - Eg. VSD, ASD, truncus arteriosus
  - Give them diuretics and high flow oxygen

- **IF OXYGEN DOES NOTHING FOR THEM**, it’s probably duct-dependent.
  - **Give alprostadil** – it increases the diameter of the duct temporarily
    - Alprostadil can cause apnoea, so intubate the child
The Child with Anaphylaxis and Respiratory Failure

ABCs come first

AIRWAY:
- If there is a complete obstruction, you intubate or get a surgical airway
- If the obstruction is partial, give adrenaline nebs, and also give it IV, and also give hydrocortisone, and then just keep giving nebs.

BREATHING:
- If the chest is silent, give IM adrenaline, and bag the patient
- If the chest is wheezy, give adrenaline nebs, salbutamol nebs. Hydrocortisone, and aminophylline 5mg/kg IV over 15 minutes

CIRCULATION:
- There may be no pulse! → you know what to do.
- SHOCK calls for INTRAMUSCULAR adrenaline.

There are things which are specifically helpful:

- Chlorpheniramine tds for 48 hrs
- INTRAMUSCULAR adrenaline
- NEBULIZED adrenaline

The Child with Poisoning and Respiratory Failure

- Respiratory rate is increased with:
  - Salicylates
  - Ethylene glycol (antifreeze)
  - Methanol
  - cyanide
The Shocked Child: Which Shock Is That?

- **Definition:**
  - Inadequate tissue perfusion, inadequate removal of wastes

- **Classification:**
  - **CARDIOGENIC**
    - Arrhythmia, cardiomyopathy, valve disease, myocardial contusion… heart failure, in short
  - **HYPOVOLEMIC**
    - Hemorrhage, gastroenteritis, intussusception
    - Also burns, etc
  - **DISTRIBUTIVE**
    - **SEPSIS**
      - Anaphylaxis
      - Spinal cord injury
      - Vasodilating drugs
  - **OBSTRUCTIVE**
    - Tension pneumothorax
    - Cardiac tamponade
    - Pulmonary embolism
  - **DISSOCIATIVE**
    - Anaemia
    - Carbon monoxide poisoning
    - Methaemoglobinemia

### Stages of shock

- **PHASE 1: reversible and compensated**
  - Agitation, confusion, pallor, tachycardia, cool peripheries

- **PHASE 2: reversible and uncompensated**
  - Acidosis, poor peripheral perfusion, tachypnea, hypotension
  - Organ systems are failing; urine output is poor

- **PHASE 3: irreversible**
  - Retrospective diagnosis: the organ systems have failed

### A little about SEPSIS:
- abnormal distribution of blood in the microcirculation
- acidosis depresses cardiac function
- cells do not use oxygen properly: this is a problem at the mitochondrial level:
  - THUS there is an inappropriately narrow gap between arterial and mixed venous oxygen saturation (take that, Rivers study)
- Early sepsis = wide pulse pressure
- Late sepsis =
- You survive sepsis by maintaining a hyperdynamic state
Resuscitating a shocked child:

- **AIRWAY**
  - Well, you obviously need one of those.

- **BREATHING**
  - Everybody who is shocked gets oxygen. Simple as that.

- **CIRCULATION**
  - Get lots of lines in
    - SHORT and WIDE BORE is the key
  - 20ml/kg bolus of crystalloid
  - Don’t use more than 1-2 of these boluses outside of septic shock

  **CEFOTAXIME: 80mg/kg:**
  - In kids, SEPSIS is the commonest cause of shock

  **DON’T FORGET THE GLUCOSE**
  - Hypoglycaemia looks like shock

**THINGS TO ASK THE PARENTS ABOUT:**
- Vomiting, diarrhoea
- Abdominal pain
- Urticaria, angioedema
- Cyanosis
- Major trauma
- Sickle cell disease → haemolysis
- Polyuria, tachypnea

**DISABILITY in shock: meningitis can present with shock**
- Don’t LP them, they cone.
- Maintain normal blood pressure and normocapnea
- MANNITOL 250mg/kg is the key.

**What fluids do I use?**
- Crystalloid – you will need more of, but it improves mortality.
- In adults.
- The less you are going to use, the less the choice matters.
- Colloid corrects intravascular volume depletion more rapidly
- The child has 80ml/kg of blood; a 40ml/kg bolus is already half the circulating volume.
- If larger volumes are needed, 4% albumin is preferred

  **In sepsis, it seems a large bolus early on decreases mortality**
  **In penetrating trauma, you leave fluid until afterwards**
  **In large volume transfusions, fluids should be warmed**
The Child with Fluid Loss
- Infants more susceptible than older children
- Diarrhoea may not be obvious: the stool has not been passed yet
  - Fluid boluses: 20ml/kg of crystalloid x 2
  - The third bolus should be albumin.
  - Also: catheter, chest/abdo xrays, CVC, intubation, inotropes.

The Child with Sepsis
- It's probably menicococcal
- Group B strep and gram negatives also common
- PURPUREAL RASH IS BAD.
- Typically it is preceded by a blanching non-purpureal rash
- Early administration of antibiotics is vital
  - Give antibiotics early
  - The fluid boluses should be albumin 4%
  - Several boluses will be required
  - After the second bolus, consider inotropes
  - Measure CVP: keep it under 12
  - Give bicarb if pH is under 7.2
  - Give glucose at 5ml/kg (10%) if hypoglycemic

The child with duct-dependent congenital heart disease
- You know it's duct dependent because oxygen does not improve the saturation
- They are going to have that huge liver
- There may not be a murmur
- There will be poor pulses, and poor perfusion
  - Oxygen will only make it worse. It causes duct closure.
  - Use only as much as is required to cause an increase in SaO2
  - Give Prostaglandin E2 (alprostenol)
  - This causes apnoea; they will need to be intubated and ventilated

The child with cardiomyopathy
- If they are an infant, it might still be a duct-dependent defect and they would not be HURT by a trial of alprostenol
- Echo will generally point the way to cardiomyopathy
  - Oxygen and diuretics
  - IV dobutamine: make the remaining myocardium work harder

From the free Google scan of “APLS : the pediatric emergency medicine resource” by Gausche-Hill and Strange, as well as the APLS handbook and www.alsg.org
The ridiculously anaemic child

- We are talking Hb less than 50
- THINGS WHICH POINT TO ACUTE HAEMOLYSIS:
  - Dark urine
  - Pale almost white feet and soles
  - Signs of heart failure
  - This tends to happen to sickle cell kids who get septic
  - This also happens in malaria

  Management is simple. Put the blood back in.
  - May also need diuretics. Overhydration will make the heart failure worse.

The ridiculously anaemic child with a sickle cell crisis

- VASO-OCCLUSIVE CRISIS:
  - Abnormal red cells clog all the little vessels
  - Tissue ischaemia ensues

- OTHER CRISES:
  - Acute chest crisis
  - Sequestration crisis (RBCs trapped in the spleen and liver)
  - Aplastic crisis
  - Hyper-haemolytic crisis

- WHY DO THESE HAPPEN?
  - No one knows
  - Infection, hypoxia, dehydration, acidosis, coldness, stress...

  - OXYGEN, rehydration, analgesia, antibiotics.
Tachyarrhythmias

- **CAUSES**
  - Re-entrant pathway (most common)
  - Poisoning
  - Metabolic weirdness
  - After cardiac surgery
  - Long QT
  - cardiomyopathy

Bradyarrhythmias

- **CAUSES**
  - Pre-terminal in hypoxia (most common)
  - Raised intracranial pressure
  - Conduction pathway damage after cardiac surgery
  - Congenital heart block
  - INTUBATION: tickling the larynx causes a vagal response
    - Adrenaline
    - Atropine if it was vagal stimulation

Emergency management

- If there is shock and bradycarida, start CPR
- If there is shock and VT, give a synchronized DC shock, 1 J per Kg
  - If that doesn’t work, progress to 2J/Kg
- You may not be able to synchronize effectively
- An asynchronous shock may cause the rhythm to deteriorate into VF
- If the synchronous shocks are ineffective, use asynchronous shocks

The Child with SVT

- The most common non-arrest arrhythmia
- Its usually 220-300bpm
- Once you’re past 200bpm, you can’t see any P waves anyway
- The longer it continues, the more unstable your blood pressure
- Vagal manoeuvres may be of use
- Adenosine 100mcg/kg, up to a maximum of 500mcg/kg

ONLY USE ONE DRUG:
- Amiodarone 5mg/kg
- Procainamide 15mg/kg
- Flecainide 2mg/kg
- Digoxin…. Maybe
- Verapimil – NOT in the under 1 year olds.
  - DO NOT combine with amiodarone, beta-blockers, or flecainide.
- Propanolol 50mcg/kg – but, it may cause asystole.

The Child with VT

- Look for torsade de pointes - you will be wanting magnesium sulphate.
- If the arrhythmia is due to drugs, it’s better to correct it with DC shock
The unconscious or semiconscious child

- CAUSES
  - Most of the time, its metabolic
  - Very rarely, it’s a structural lesion
  - The metabolic causes may produce false localizing signs; there may be asymmetrical findings.
  - The metabolic cause are
    - Hypoxic injury
    - Epilepsy
    - Meningitis/encephalitis
    - Renal or hepatic failure
    - Reyes syndrome
    - Hypoglycaemia
    - Hypercapnoea

Which coma is that?

- Is there hypertension?
- Is the fontanelle full?
- Is there neck stiffness
- Is there an acidotic pattern of respiration?
- What size are the pupils, and are they reactive?

There are only 3 absolute signs of raised ICP:
- Papilledema
- Bulging fontanelle
- Absence of pulsation in retinal vessels

- Is there hypertension?
- Is the fontanelle full?
- Is there neck stiffness
- Is there an acidotic pattern of respiration?
- What size are the pupils, and are they reactive?

- The first test you is the BSL.

- Everyone gets oxygen
- Everyone is maintained in normoglycaemia
- Keep sodium in the normal range, ? give half-normal saline
- Maintain normothermia
- Pad the eyes so they don’t dry out
Raised intracranial pressure

- **Oculocephalic reflex:**
  - When the head is being turned left to right, the normal response is to move your eyes away from the head movement direction.
  - Also when the head is flexed, the normal response is to move the eyes upwards.
  - If these are absent, the oculocephalic reflex is impaired.

  - **CENTRAL SYNDROME:** coning through the foramen magnum;
    - Neck stiffness and Cushings triad

  - **UNCAL SYNDROME**
    - Third nerve compression and ipsilateral dilated pupil.
    - Oculomotor palsy is next: eye cannot move laterally
    - Hemiplegia may develop

**YOU DO NOT PERFORM LUMBAR PUNCTURE IN THESE CHILDREN.**

When would you reconsider lumbar puncture:

- Prolonged or focal seizures
- Focal signs
- Widespread purpuric rash
- GCS less than 13
- Dilated pupils
- Dolls eye reflex impairment
- Decerebrate or decorticate posturing
- Thrombocytopenia
- Cushings reflex
- Obvious papilledema
- Hypertension

- Nurse head straight, at 30 degrees
- Intubate and sedate
- Keep PCO2 40-45
- Give mannitol 250-500mg/kg
- Think about dexamethasone

A few other things:

- CT head… everyone gets one
- Is there otitis media? Its associated with meningitis
- Subacute hours-long onset suggests meningitis
- Retinal hemorrhage and dodgy story? Maybe contact DOCs
The Child with meningitis or encephalitis
- Except the neonates, the commonest bug is *Neisseria meningitides*
- The next commonest is *Strep pneumoniae*

**IN THE UNDER-3s**
- Signs and symptoms will be those of increased intracranial pressure
- Poor feeding, pyrexia, unexplained crying, cyanosis, purpuric rash

**IN THE OVER-3s**
- Classic signs
  - Treat the ICP
  - Give cefotaxime 80mg/kg, and acyclovir
  - Give dexamethasone 0.15mg/kg BEFORE the antibiotic to reduce the complication of persisting hearing loss

The Child who drank the delicious green methadone syrup
- Naloxone is the key

**HOWEVER:**
- BEFORE you give the naloxone, you want to normalize the CO2; because if you don’t, the naloxone will cause arrhythmias, APO, or asystole.
- This is because opioid antagonists and hypercapnia both stimulate the sympathetic nervous system

The Child in a metabolic coma
- Reye syndrome, or “Reye-like conditions” – rapidly progressive encephalopathy, vomiting, drowsiness, convulsions and coma
- There will be hepatomegaly, hypoglycaemia, high ammonia
- **ASK FOR A SERUM AMMONIA LEVEL** - send in a iced tube
- Reye syndrome is caused by aspirin: that’s why you don’t give it to children.

The Child with malaria
- Diagnosis rests on thick and thin films
- **Give QUININE** loading dose 20mg/kg over 4 hrs in dextrose 5%
- Monitor ECG – QT will elongate
- Give IV antibiotics, eg Cefotaxime
- Transfuse if they hemolysed enough
The child with a seizure

- **Tonic clonic status epilepticus:**
  - Generalized convulsions lasting over 30 minutes; OR
  - Generalized convulsions occurring so frequently over 30 minutes, that the child does not recover consciousness between them

General factoids:
- The longer the episode, the harder it is to terminate
- If its longer than 5 minutes, it wont stop by itself, so use anticonvulsants

Consequences:
- Pulmonary oedema
- Arrhythmias
- DIC
- Rhabdomyolysis
- hyperthermia

Long term consequences:
- the younger you are, the worse it will be
- persistent epilepsy, motor deficits, learning or behavioural problems: 6% of the over-3s, 29% of the under-3s

So what if you are convulsing. Why is it such a problem?
- Cerebral metabolic activity rises 3-fold
- Blood pressure rises initially, and then falls
- When it falls, cerebral perfusion falls, because mid–seizure you lose you ability to autoregulate the cerebral blood flow
- Because of persisting muscle contraction, there is lactic acidosis
- Because of decreased cerebral perfusion, there is cerebral ischaemia, and thus cerebral oedema.

Issues in assessment
- Grunting may be a sign of seizure, not of airway obstruction
- Hypertension is more likely the RESULT of the seizure, not the cause
- Decorticate and decerebrate posturing can be mistaken for the tonic phase
  - **Everyone gets oxygen**
  - **Everyone gets a BSL**
Emergency management of the convulsing child

- Airway, high flow oxygen, glucose
- If there is vascular access, give LORAZEPAM 0.1mg/kg
- If there is no vascular access, give PR DIAZEPAM 0.5mg/kg
  - Or… Buccal MIDAZOLAM 0.5mg/kg

...wait 10 minutes, and give more Lorazepam if there is not benefit

- Then: give PARALDEHYDE 0.4mg/kg PR
- Then: give PHENYTOIN 018mg/kg
- Then: perform an RSI with THIOPENTONE 4mg/kg

what the hell is paraldehyde?
- Cyclic trimer of acetaldehyde molecules
- CNS depressant, hypnotic sedative antiepileptic
- Half-life is roughly 8 hrs in neonates
- Mechanism of action is very similar to the barbiturates: increase the duration of the GABA-chloride-channel opening
- 90% is excreted in the lungs, as it is metabolized into acetaldehyde, then acetate, and then CO2 and water.
- When it is not acting as a sedative, it is an industrial solvent. It can dissolve a plastic syringe in about 15 minutes.
- It is highly flammable
- It can cause rectal irritation
- It tastes like burning, and needs to be mixed with milk.

The drugs:

- LORAZEPAM
  - Less respiratory depression than diazepam; half life 12-24 hrs
  - Poor rectal absorption
- DIAZEPAM
  - Action lasts only 1 hr
  - Fast acting when given PR
- MIDAZOLAM
  - Short acting
  - BUCCAL ADMINISTRATION: squirt it into the area between the lower bottom lip and the gum margin
  - Buccal midaz is twice as effective as rectal diazepam
- PHENYTOIN
  - Measure the levels 60-90 minutes after the infusion has finished
  - Monitor the ECG
  - Peak action is within 1 hr
More drugs:

- **FOSPHENYTOIN**
  - Pro-drug of Phenytoin
  - Can be administered more rapidly: Phenytoin uses propylene glycol as a solvent, and so needs to be administered very slowly
  - There are fewer cardiac side effects because there is no propylene glycol
  - 75mg of this is equivalent to 50mg of phenytoin

- **THIOPENTONE**
  - 4-8mg/Kg
  - NOT an effective long term anticonvulsant

General management

- Normoglycaemia is the key
- Normo-natremia is also the key
- Normothermia is a good idea as well

**RESTRICT FLUIDS:** 60% of maintenance.

- Aspirate the stomach contents, before it ends up in the lungs
- If increased intracranial pressure is present, you will want to be dealing with it.

The child with a systemic hypertensive crisis

- Hypertension is rare in children:
  - Dysplastic kidneys
  - Obstructive nephropathy
  - Glomerulonephritis
  - Coarctation of the aorta

**MEASURING A CHILDS BLOOD PRESSURE IS A PAIN IN THE ARSE**

- Use the biggest cuff that fits comfortably on the upper arm
- Systolic is more accurate
- Diastolic is frequently misleading – you might hear the Korotkoff sound all the way down to zero.
- The measured value needs to be compared against the normal age ranges.
- Aim to reduce the blood pressure to 95% percentile over the next 24-48hrs
- Aim to accomplish 1/3rd of the reduction over the first 8 hrs
- If you lower it any faster than that, you risk infarcting the optic nerve heads
- Drugs of choice:
  - Labetalol infusion – unless they are in heart failure
  - Sodium nitroprusside infusion – and monitor for cyanide poisoning

**Nifedipine:** orally; better when you bite the capsule
Pediatric trauma

General features:
- Tissues are more elastic, less energy is deposited at the site of trauma and more energy is dissipated to other body regions
- As in the adult, there is a sequential approach, and major problems should be addressed as soon as they are discovered

Primary survey

- **AIRWAY** with cervical spine control
  - If they are vomiting, tilt the strap board head-down
  - Intubate with in-line manual stabilization

- **BREATHING** with ventilatory support

- **CIRCULATION** with hemorrhage control
  - Blood pressure takes too long, you need to estimate the requirements for fluid resuscitation
  - Signs suggestive of the need for immediate fluids:
    - Massive brady / tachycardia
    - Falling systolic BP
    - Poor capillary refill time
    - Tachypnea unrelated to thoracic trauma
    - Altered LOC unrelated to head injury

  2 large cannulas are mandatory

  Massive replacement in penetrating trauma is not indicated- it will disrupt the forming clot

  Use 10ml/kg boluses, rather than 20.

  Use saline to begin with.
  After 40ml/kg, start using blood.

- **DISABILITY** with prevention of secondary brain injury
  - Pupil inequality makes you want to do several things:
    - Control CO2 (40-45)
    - Maintain normotension
    - Give mannitol
    - Administer anaesthesia
    - Contact neurosurgery

- **EXPOSURE** with temperature control
Things you need to find and deal with during the primary survey

- Airway obstruction
- Tension pneumothorax
- Open pneumothorax
- Massive haemothorax
- Flail chest
- Cardiac tamponade
- Decompensating head injury

Other things you might do during the resuscitation phase

- Urinary catheter: only if they can’t pass urine spontaneously or if there is a pressing need to measure urine output
- NG tube placement
- Analgesia: IV; no entonox if there is base of skull fracture or pneumothorax

Secondary survey

- “a simple but thorough search for the anatomical features of injury”
- Surface, orifice, cavity, extremity
- This is also where you do the log roll
- LOOK FOR
  - CSF leak
  - Inside and outside the mouth
  - Loose teeth
  - Otoscopy for haemotympanum
  - Ophthalmoscopy for retinal haemorrhage
  - Palpate the C spine
  - Inspect neck veins
  - Auscultate the chest
  - PR is only performed if it is going to change management

- Get CT scans
- Get an ECG

Repeat the ABCDE! Especially if there is a deterioration.

- ABG
- Hemodynamics, hemoglobin, hemostasis
- Urine output monitoring: 1-2ml/kg/hr

METABOLIC SURVEY: EFGH

- Electrolytes
- Fluid balance
- Gastrointestinal
- Hormones
Paediatric chest injury

General features:
- Children have elastic chest walls: they can have massive visceral injuries without rib fractures
- If there ARE rib fractures, it indicates extremely high energies
- High metabolic rate, small functional residual capacity, thus they desaturate quickly
- Flail chest is poorly tolerated because the ribs are more horizontal

During the primary survey, you should pick up:
- Tension pneumothorax
- Massive hemothorax
- Open pneumothorax
- Flail chest – but paradoxical chest movement is less likely to be seen in children because severe pain on breathing will cause the child to splint their chest wall
  - Flail chest requires 2 weeks before it becomes stable
- Cardiac tamponade- Becks triad; muffled heart sounds, distended neck veins, and shock.

Later on, you may discover:
- Pulmonary contusion
  - Oedema and interstitial/alveolar haemorrhage
  - Because the chest wall is so elastic, there is force transmitted directly to the lung
  - Oxygen and physiotherapy
  - It improves in 36 hrs
- Tracheal or bronchial rupture
  - The clue is a persisting air leak through your chest drain
  - There may be subcutaneous emphysema
  - You may need more than one chest drain
  - Your pressure support should be limited so as to allow a small leak to close on its own
- Disruption of the greater vessels
  - If they survive to get to hospital, the tear in the aorta has tamponaded itself within an intact adventitial layer (outermost layer)
  - The commonest site of rupture is at the ligamentum arteriosum, close to the origin of the left subclavian artery
  - They will be shocked and their pulses barely palpable
  - THERE WILL BE A WIDENED MEDIASTINUM
  - Avoid blood pressure fluctuations
- Diaphragm rupture
  - More common on the left
  - More common with penetrating chest injury
Paediatric abdominal injury

General features:
- It was blunt trauma, more than likely
- The children are more susceptible because:
  - They have thin abdominal walls
  - The liver and spleen lie lower and more anteriorly
  - Elastic ribs offer little protection
  - The bladder is abdominal, not pelvic, and therefore more vulnerable when full

Deceleration injury and blunt impact injury
- Solid organs suffer most
- Duodenum may rupture at the duodenojejunal flexure, or it may have a huge hematoma
- Pancreas is at greatest risk from bicycle handlebars
- Straddle injury with perineal hematoma suggest urethral injury

Bits of examination which aid in assessment
- Urethral meatus: is there blood?
- Gastric drainage and decompression: is there blood?
- IDC: only after urethral damage has been ruled out
- Retrograde urethrogram?

Non-operative management
- Not everyone needs a laparotomy
- Maybe you can just sit on the peritoneal hemorrhage, and the solid organs will sort themselves out
- For this to work, you cant be coagulopathic, and there need to be frequent re-examinations

Who needs a laparotomy:
- Penetrating abdominal injury
- Definite signs of bowel perforation
- More than 40ml/kg replaced, and still in shock

A renal pedicle injury probably cant be rescued: warm ischaemia time is only 45-60 min

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# Paediatric head injury

## General features:
- Commonest cause of death from trauma (40%)
- In infancy, the most common cause of head injury is child abuse

## Primary brain injury
- Direct consequence of the impact:

## Secondary brain injury
- Hypotension
- Hypoxia
- Intracranial hypertension, when sutures close at 12-18 months
- Seizures

Normal cerebral blood flow = 50ml blood per 100g brain tissue per minute
A fall below 20ml per 100g will cause ischaemia and thus oedema
A fall below 10 will cause cell wall dysfunction

The most common cause of raised ICP is cerebral oedema

## Factors suggesting a serious injury at triage
- Substantial trauma
- History of lost consciousness
- Focal signs or generalized convulsions
- Penetrating head injury

## When do you intubate them (the UK NICE criteria)
- GCS < 8
- Loss of protective laryngeal reflexes
- Hypoxia or hypercapnia
- Spontaneous HYPERventilation
- Respiratory irregularity

## Glasgow coma scale
- Is modified for the under-4s (mostly verbal response- cooing vs. conversing, crying vs making inappropriate sounds)
- The “best grimace” response for the pre-verbals

## What else to look for
- Retial hemorrhage = child abuse
- Limb tone
- papilloedema

From the free Google scan of “APLS : the pediatric emergency medicine resource” by Gausche-Hill and Strange, as well as the APLS handbook and www.alsg.org
Who gets an urgent CT head:
- GCS less than 13, or 14 at 2 hrs after the injury
- Open or depressed skull fracture
- BOSF
- Focal neuro signs
- Multiple vomiting
- Amnesia of over 30 minutes of events BEFORE the accident
- Dangerous mechanism
- Seizure
- Coagulopathy

What else should you do
- Give analgesia, or they will get hypertensive and develop raised intracranial pressure

For whom will the neurosurgeon get out of bed
- Persisting coma GCS <8 after resuscitation
- Deteriorating LOC, especially changes in motor response
- Focal neuro deficit
- Seizures
- Penetrating injury
- CSF leak
- Unexplained confusion for several hours
Paediatric trauma of the extremities

**General features:**

- Growth plates are 2-5 times weaker than bones and ligaments
- Greenstick fractures: one cortex deforms without fracturing
- There is **DECREASED** chance of fracture propagation
- Comminuted fractures are rare
- Children's bones absorb more force than adults
- Pelvic fractures are uncommon (the pelvis flexes, and the pelvic organs take a hit)
- 20% of your blood volume can leak out into the fractured thigh

Evaluation happens in the secondary survey

**Crush injuries of the abdomen and pelvis**

- Pelvic disruption = life threatening blood loss
- Splint the pelvis
- Put the blood back in
- Embolization of the bleeding vessels may be needed (radiology)

**Traumatic amputation**

- Partial is more threatening than complete: transected vessels go into spasm, lacerated ones do not.
- Tourniquets are out of fashion: they don't stop bleeding from bones. Instead, use an elastic compression bandage, or press on the proximal end-artery
- Dress the stump with a sterile dressing soaked in saline, and splint/elevate the limb.
- Amputated part will remain viable for 8 hours at room temperature, or 18 hrs on ice.
  - Clean it
  - Wrap in moist sterile towel
  - Place in sterile sealed plastic bag
  - Put the bag in an ice box
  - The bodypart should not come in contact with the ice
  - *It has to travel in the same vehicle as the child*
- Keep it even if you don’t plan to graft it.

**Massive open long-bone fractures**

- Open fractures cause twice the blood loss of closed fractures
- Thus, a open femoral fracture = 40% blood loss
Things to do during the secondary survey

- Active movement: don’t passively move their fractured limb
- The key is to identify THE THREATENED LIMB:

VASCULAR PROBLEM
- Pulses present?
- Cap refill present?
- Decreased sensation
- Rapidly expanding hematoma
- A Bruit

COMPARTMENT SYNDROME
Symptoms start when the compartment pressure rises above capillary pressure. Pulse is lost when compartment pressure rises above arterial pressure.
- Pain on passive stretching of the muscle
- Decreased sensation
- Swelling pallor of limb
- Paralysis
- Pulselessness- by this time irreversible damage has occurred
- Usually, it will happen within hours of some sort of crush injury

Emergency treatment

- Alignment
- Reduction
- Immobilization
- Traction

The child with spinal trauma

- Traumatic torticollis should be immobilized in their current position

Factoids

- Upper 3 vertebrae are the ones involved in the young children
- In older children, it’s the lower 3 vertebrae

Guidelines for clinically clearing the C-spine

- No midline tenderness on palpation
- No focal neuro deficit
- Normal level of alertness
- Not intoxicated
- No painful distracting injuries

  - Pseudosubluxation occurs in 9% of 1 to 7 yr olds; usually C2 on C3 or C3 on C4

  Atlantoaxial rotary subluxation is the most common injury; the child presents with torticollis

  if you’re going to use steroids, use them within 8 hrs of the injury
The hideously burnt child

General features:
- Scalds mainly in the under 4s
- Mainly in boys
- Poverty and overcrowding is a risk factor

Features determining severity:
- Temperature
- Duration of contact

At 44 degrees, contact = 6 hours
At 54 degrees for 30 seconds
At 70 degrees within 1 second

Primary survey
- Airway burns
- History of exposure to smoke in a confined space
- High flow oxygen

**BURNS SHOULD NOT CAUSE SHOCK:**
- If they are shocked, they are bleeding from somewhere
- Exposure should be minimal: burnt children lose heat rapidly

Secondary survey
- Estimate the burnt area
  - Rule of 9s cannot be applied to the under-14s
  - Use the palm and fingers: its 1%
- Estimate the depth
- Look for circumferential burns of the limbs or the neck
- Burns of the perineum are more prone to infection

Emergency management
- Analgesia
- If over 10% of surface is burnt, give fluids:

  - **Daily fluid therapy** = percentage burn x weight (kg) x 4
  - **First half in the first 8 hrs**

- Cover the burns in sterile towel +/- cling wrap
- Leave the blisters intact.
- Don’t use cold compresses for longer than 10 minutes; never transfer the child with compresses in place.
Carbon monoxide poisoning

Measure carboxyhemoglobin:

- 2-20% = give oxygen
- Over 20% = put them in a hyperbaric oxygen chamber

Indications for transfer to a burns unit:

- 10% partial and/or full thickness sburn
- 5% full thickness burns
- Burns to “special areas”- hands, feet, face, perineum
- Circumferential burns
- Inhalational burn
- Chemical, radiation, or high voltage burns
The electrocuted child

General features:
- AC is lethal at lower voltages than DC
- Effects of an increase in current:
  - 10mA = tetanic contraction, can’t let go of electrical source
  - 50mA = respiratory arrest due to diaphragmatic tetany
  - 100mA to 50A = primary cardiac arrest (a defib is 10A)
  - 50A and above = severe burns + cardiorespiratory arrest

Relative resistance of body tissues, from least to most:
- Tissue fluid
- Blood
- Muscle
- Nerve
- Fat
- Skin
- Bone
- Nerves, blood vessels, skin and muscle are injured most
- Bone can continue to increase in temperature after the current has stopped
- Swelling of damaged structures can cause compartment syndrome

Primary and secondary survey
- The key is to find the entry and exit point- that way you know what organs got in the way of the current
- There will be burns
- There may also be fractures from tetanic contraction
- There will be rhabdomyolysis in the injured muscle: you may want to alkalinize the urine, and maintain an output of ~ 2ml/kg/
- There will be cardiac arrhythmias, and they will need to be monitored.
The drowned child

General features:

- There is no such thing as “near drowning” anymore
- Definition is respiratory impairment due to submersion

THE DIVING REFLEX

- Bradycardia
- Apnoea
- Due to apnoea, hypoxia and acidosis
- Due to hypoxia and acidosis, there will be tachycardia
- There will also be hypertension
- Eventually, breakthrough breathing will occur
- When inhaled water touches the glottis, immediate laryngospasm occurs
- After the laryngospasm subsides, you just inhale all this water
- Pulmonary oedema ensues
- Severe hypoxia then causes bradycardia and then asystole

THERE MAY ALSO BE SPINAL INJURY AND HYPOTHERMIA.

You won’t have too much of an electrolyte derangement from swallowing pond water

Primary Survey

- Risk of aspiration from swallowed water = intubate immediately
- Decompress the stomach
- Chest x-ray changes will occur later, don’t wait for them
- Patients should be lifted out of the water HORIZONTALLY and not vertically. Vertical recovery may cause cardiovascular collapse due to loss of venous-blood-returning pressure from the surrounding water. Apparently acts as a huge TED stocking.
- Look for hypothermia: shocks may be ineffective below 30 degrees
- Rewarm internally if below 30 (externally if above 30)
- Temperature should rise by 1 degree per hr to reduce chance of hemodynamic instability

ACTIVE INTERNAL REWARMING:

- 39 degree IV fluids
- 42 degree ventilator humidifier
- Gastric, bladder, peritoneal lavage with warm fluids
- Consider extracorporeal blood rewarming

If the initial rhythm was VF, it’s better to keep them cool – at 32-34 degrees
Emergency treatment
- Brain ischaemia precedes cardiac ischaemia
- Organisms that affect them are Pseudomonas and Aspergillus; cefotaxime should be started
- ICP will rise because of hypoxic brain injury
- Normoglycaemia is important

Prognostic indicators
- Duration of hypoxic-ischaemic injury
- Adequacy of initial resuscitation

SIGNS OF GOOD RECOVERY:
- Time to respiratory effort < 3 minutes
- Temperature less than 33 on arrival

SIGNS OF DOOM:
- Immersion time > 8 minutes
- Time to respiratory effort > 40 minutes
- Persisting coma
- Persisting acidosis ~ pH 7.0
- Persisting hypoxia below 60 mmHg

- It doesn’t matter, fresh water or salty

Outcomes
- 70% survive with basic life support
- 40% survive without basic life support
- Of the survivors,
  - 70% will make a complete recovery
  - 25% will have a mild neuro deficit
  - 5% will be in a persisting vegetative state
### Important Formulae for Paediatric Resuscitation

<table>
<thead>
<tr>
<th><strong>WEIGHT ESTIMATION:</strong></th>
<th><strong>ENDOTRACHEAL TUBE SIZE:</strong></th>
<th><strong>DEFIBRILLATOR ENERGY:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight = (Age + 4) x 2</td>
<td>Tube size = ((\frac{\text{Age}}{4})) + 4</td>
<td>2 Joules per Kg first; 4 Joules thereafter</td>
</tr>
</tbody>
</table>

### DRUG DOSES:

<table>
<thead>
<tr>
<th><strong>ADRENALINE INTRAVENOUSLY</strong></th>
<th><strong>ADRENALINE NEBULIZED</strong></th>
<th><strong>ATROPINE</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>10 mcg per kg; = 0.1 ml per kg of 1:10,000 solution</td>
<td>5 ml of 1:1000 solution</td>
<td>20 mcg per kg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>ADENOSINE</strong></th>
<th><strong>AMIODARONE:</strong></th>
<th><strong>FLUIDS</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>100 mcg per kg – and then 200... and then 300...</td>
<td>5 mg per kg</td>
<td>One bolus is 20ml per kg; blood after 2(^{nd}) bolus</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>AMIODARONE:</strong></th>
<th><strong>FLUIDS</strong></th>
<th><strong>BURNS FLUID DEFICIT</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>5 mg per kg</td>
<td></td>
<td>(4ml per kg ) for every 1% of surface area burnt</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>AMILODARONE:</strong></th>
<th><strong>BLOOD</strong></th>
<th><strong>DIAZEPAM</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>5 mg per kg</td>
<td>10 ml per kg</td>
<td>0.25 mg per kg; .......or 0.5 mg per kg when PR</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>PHENYTOIN</strong></th>
<th><strong>DIAZEPAM</strong></th>
<th><strong>10% DEXTROSE</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>20 mg per kg, over 20 minutes</td>
<td></td>
<td>5 ml per kg</td>
</tr>
</tbody>
</table>

### TOTAL FLUID DEFICIT

\[(\%\ \text{dehydration}) \times \text{weight} \times 10\]

### DAILY FLUID DEFICIT

- First 10 kg: 100ml / kg
- Next 10 kg: 50ml / kg
- Rest: 20 ml / kg

### BURNS FLUID DEFICIT

Total deficit is replaced over 24 hours; 50% of this is replaced over the first 8 hours.