**Anaesthesiology Summaries Sean Thornley SIC 2011**

**1) SIDE EFFECTS OF OPIOIDS:**

1. Respiratory Depression
2. Severe PONV
3. Tolerance
4. Miosis
5. Constipation
6. Addiction potential
7. Histamine release
8. High doses – Purple, rigid patient
9. Severe pruritus if used as spinal

**Depression, PONV, Tolerance, Miosis, Constipation, Addiction, Histamine, Rigidity, Pruritus**

**2) Factors that influence MAC:**

|  |  |
| --- | --- |
| **INCREASE MAC** | **DECREASE MAC** |
| Chronic Alcohol Usage | Analgesics eg Opioids/sedatives |
| Hyperthyroidism? | Hypothyroidism? |
| Hyperthermia | Hypothermia |
| Infants/Puberty | Geriatric |
|  | Low cardiac output |
|  | Pregnancy |

**3) Metabolic Equivalents and requirements for surgery:**

1 met at rest = 3ml/kg/min O2 consumption (+- 210ml O2 if 70kg)

Require a minimum of 4 mets for safe surgery = 12ml/kg/min = +- 840ml O2/ min.

**4**)Bradycardia in a child is hypoxia until proven otherwise!!!

**5)** Never give an IV induction agent or muscle relaxant in a compromised airway

**6)MAP** = (s + 2d)/3 --------------- aim for > 60mmHg in normotensive patient. (perfusion to brain maintained between 60-180 mmHg). Do not let BP decrease by > 20-30% to maintain regulation.

**7) Factors shifting O2-haemoglobin curve:**

|  |  |
| --- | --- |
| **LEFT shift (02 TIGHTLY BOUND)** | **RIGHT shift (02 JUMPS OFF EASIER)** |
| ↓ Temperature | ↑ Temperature |
| ↓ [H+] (alkalosis - ↑ pH) | ↑ [H+] (Acidosis - ↓ pH) |
| ↓ 2,3 DPG | ↑ 2,3 DPG |
| ↓PaCO2 | ↑ PaCO2 |

8) RBC have no mitochondria, don't use O2, only transport ---- they consume lactate

9) **Draw an annotated normal Capnogram:**

A-B) post inspiration/dead space exhalation

B) is the start of alveolar exhalation (anatomical dead space) (Phase 1)

B-C) the exhalation upstroke where dead space gas mixes with lung gas (Phase 2)

C-D) is the continuation of exhalation/ plateau(all the gas is alveolar now, rich in C02). (Phase 3)

D) is the end-tidal value – the peak concentration

D-E) is the inspiration washout. (Phase 0)

**10) NERVE STIMULATORS:**

** Normal Scoline Non-Depolarisers**

1. **Single Twitch**
2. **Tetanus (TET) – give stimulus then maintain it.**
3. **Train of Four (TOF)**
4. **Post-Tetanic Potentiation**

**(PTP) --- give a tetanus then a single twitch**

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1. **PEEP –** maintains a high FRC, keeps FRC:closing capacity high. Method of keeping small airways open.
2. **Oxygen Flux Equation:**

**DO2  = Cardiac Output X CaO2 (content of arterial O2)**

= CO X (Hb-bound O2 + dissolved O2) Dissolved Negligible

 = Stroke volume x HR [(Hb x SaO2 x 1.34) + (0.03 x PaO2)] Hb most NB.

 = ± 70ml x 70bpm [(Hb x SaO2 x 1.34) + (0.03 x PaO2)]

 = ± 5 ( 197ml O2/l + 3 ml O2/l)

 = 5 (200) = ± 1000 ml O2/minute to all cells of the body

Remember 1 met = 3ml/kg/min therefore demand = 210ml and supply is almost 5 x that (1000ml)

1. **Types of Hypoxia**: based on Oxygen Flux Equation
2. Cardiogenic – stagnant hypoxia (Heart Failure, tamponade etc)
3. Anaemic - ↓[Hb]
4. Toxic – binding capacity of Hb changes. 1.34 changes (amount of ml able to bind to 1g Hb).
5. Hypoxic hypoxia - ↓ PaCO2
6. (Histotoxic hypoxia: O2 reaches the tissues but tissues cannot utilise it eg Cyanide poisoning)
7. **Mapleson Classification of circuits**

Good for spontaneous ventilation. FGF ± 5 litres/min

Ineffective for controlled ventilation (uneconomical)

Spontaneous ventilation in kids <20kg

F) Modified Mapleson-E circuit. Bag with hole attached to expiratory side. Spontaneous and controlled ventilation.

Valveless, low resistance(↓effort needed for child), small dead space volume.

Needs high FGF to eliminate CO2 --- 2.5X minute volume.

 **CIRCLE SYSTEM:** Economical (↓FGF/inhalants). ↓ pollution. Heated & humidified gases.

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1. **Airway management**

|  |  |
| --- | --- |
| **5 metal objects** | **5 plastic objects** |
| 1) Laryngoscope | 1) Face mask |
| 2) Introducing stylet | 2) Tracheal tube |
| 3) Magill’s forceps | 3) Guedel airway |
| 4) Artery forceps | 4) Catheter mount |
| 5) Stethoscope | 5) Suction |

1. **Pulse Pressure Variation (PPV) (requires an A-line)**

[(Highest SBP – Highest DBP) – (Lowest SBP – Lowest DBP) / mean PP] X 100

EG:

Highest SBP – 135

Highest DBP – 90 Therefore: 135-90 = 45

Lowest SBP – 115

Lowest DBP – 75 Therefore 115-75 = 40

[(45-40)/(45+40/2) X 100 = PPV

5/42.5 X100 = **11.7%**

**Therefore:** PPV **> 12%** is an indication of HYPOVOLAEMIA (volume responders).

 PPV **8-12%** : grey area

 PPV **< 8%:** HEART FAILURE hypovolaemia (non-volume responders).

1. **Fluid Replacement:**

**4:2:1 maintenance rule:** 4ml/kg/hr (up until 10 kg). 2ml/kg/hr (10-20kg). 1ml/kg/hr thereafter.

Ie. 70 kg: 40ml + 20ml + 50ml = 110ml/hr.

If kept NPO for 8hrs ----- 110 x 8 = 880ml. Give half in first hour. Then ¼ in 2nd hour, final ¼ in 3rd hour.

**Therefore:**  70kg person NPO for 8 hours requires 880ml replacement : 440ml in 1st hour, 220 ml in 2nd hour and 220ml in 3rd hour.

**Acute losses (eg blood):** Replace crystalloid 3:1 (R/L) or colloid 1:1 (eg Voluven)

1. **Complications of blood transfusions:**

|  |  |
| --- | --- |
| **IMMUNE MEDIATED** | **NON IMMUNE MEDIATED** |
| **Haemolytic** | **Acute:** **Delayed** | **ABO incompatibility**:Shiver, fever, nausea, chest or flank pain, skin blush, hypotension, tachycardia**Rh/minor incompatibility**:2/52 post-op anaemia & jaundice. Mild | **Metabolic** | **pH**  changes: met alkalosis (citrate to bicarb)**Citrate** Toxicity: bind calcium -**hypocalcaemia**(long QT, brady, hypotension) Rx CaCl2 10mg/kg iv**Hypomagnesaemia**: also bound to citrate**Hyperkalaemia**: especially if renal impairment**↓2,3DPG**: left shift O2Hb curve |
| **Infective** | Hepatitis, HIV, EBV, CMV, malaria. Bacterial contamination if aseptic technique avoided |
| **Micro - aggregates** | Plt/WC that form after 3-5/7. Cause pulmonary obstruction and **TRALI** |
| **Non -Haemolytic** | **Allergic** Rxns: stop, give antihistamine**Pyrexial** Rxns:**Graft vs Host Disease**: very bad**Post-transfusion purpura**: acute thrombocytopaenia | **Bleeding Tendency** | Dilutional thrombocytopaenia eg massive blood loss and replace all but clotting factors/platelets |
| **Hypo-thermia** | Not adequately heated before transfusion. Left shift, citrate toxicity, dysrhythmias |
| **Circulatory overload** | Volume exceeds patients ability to pump it. |
| **Massive transfusion** | Transfusion of 1 whole blood volume within 24 hrs. All above complications are more likely  |

1. **LARYNGOSPASM: Management.**

Avoid irritating stimuli eg: blood, mucous etc

Give 100% O2, CPAP mask, jaw thrust with painful stimulus to TM joint.

Deepen anaesthesia (propofol and lignocaine)

INTRACTABLE: Muscle relaxant (Scoline) and intubation

1. **MYOCARDIAL O2 SUPPLY VS DEMAND**

|  |  |
| --- | --- |
| **SUPPLY** | **DEMAND** |
|  **OXYGEN**:  | **HEARTRATE** |
|  FIO2 | **CONTRACTILITY** |
|  Haematocrit  | **WALL TENSION:** |
|  02 Dissociation |  Preload |
| **CORONARY ARTERIES:** |  Afterload |
|  Atherosclerosis | **BASIC METABOLIC NEEDS** |
|  Spasm |  Fever |
|  Endothelial damage |  Thyrotoxicosis |
|  Vasoactive substances |  Cold |
| **CORONARY PERFUSION PRESSURE:** |  |
|  Aorta DP - LVEDP |  |
| **PERFUSION TIME:** |  |
|  Tachycardia (diastolic time) |  |

**3 THINGS TO CONSIDER:**

1)Severity of disease

2)Type of surgery

3)Pt functional capacity

**Supply: O2** needs to get into the **arteries** at a certain **pressure** for a certain period of **time**.

**Demand:** How fast? How strong? How much tension to push out/against? Normal needs?

1. **Transfusion Triggers:**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **SITUATION** | **Patient****Or----🡪** | **Haemodynamic****(Tachy/hypotensive)** | **Myocardial Ischaemia** | **Oximetry** **(see below)** | **Hb** **Trigger (g/dl)** |
| **THEATRE** | ALL | Rel ↑HR/↓MBP | ECG | Yes | 6 |
| >80 YEARS | Rel ↑HR/↓MBP | ECG | Yes | 7 |
| CVD | Rel ↑HR/↓MBP | ECG | Yes | 8 |
| ↑T, metabolism | Rel ↑HR/↓MBP | ECG | Yes | 8 |
| INFANTS | ------ | ------ | ------ | 9 |
| **WARD** | ALL | Rel ↑HR/↓MBP | Clinical | N/A | 6 |
| >80 YEARS | Rel ↑HR/↓MBP | Clinical | 7 |
| CVD | Rel ↑HR/↓MBP | Clinical | 8 |
| ↑T, metabolism | Rel ↑HR/↓MBP | Clinical | 8 |
| INFANTS | ------ | ------ | ------ | 9 |

**Tachycardia-** HR ↑ by 120-130% of baseline or to > 110-130bpm

**Hypotension-** MBP < 70-80% baseline or < 60mmHg (55 if young/healthy); < 70-80mmHg if CVD/HT

**Oximetry (1 or more)-** PvO2 < 32mmHg, SvO2 < 50%, O2ER > 50%, ↓VO2 > 10%

 **(** O2ER – oxygen extraction ratio --- (SaO2 – SvO2)/SaO2 ---Normal about 25%)

1. **02Hb Dissociation curve**

P90 – Partial pressure of O2 at which the saturation is 90%. If the value is < 60mmHg –HYPOXIA

P50 – Pressure when the Sats are 50% --- usually ± 26.5 mmHg.

If the P50 is higher --- R shift of the curve: Hb does not bind as readily to O2, releases easier. This is good when you need O2 eg: Temperature, Drop in pH, increase in 2,3DPG

L shift is opposite (binds tighter) – only 2 situations where this is beneficial: 1) Altitude 2) Fetus

1. **PaO2 and PF Ratio:**

PF ratio = PaO2/FiO2.

Normal PaO2 is 80-100mmHg. But, inspired fraction of oxygen FiO2 is only 21% on room air. Therefore, if you increase the FiO2 to 100%, a 5 times greater result can be obtained and a PF ratio of 400-500 is possible

* PF ratio of < 300 is ALI
* PF ratio of < 200 is ARDS
* PF ratio of < 100 is massive shit
1. **Valvular Heart Disease: GIVE ANTIBIOTICS IN THIRD WORLD!!!** Kefzol/Clindamycin/Gentamycin
2. **Regurgitant** lesions: FULL, FAST, FORWARD, FORCEFUL with a low PVR

Therefore: Preload, increase HR, give inotropes

Spinal is a good option (↓PVR)

1. **Stenotic** Lesions: NEVER do a spinal

Slow-normal HR, normal fluid levels (use CVP), normal PVR

1. **POTASSIUM --- Normal is 3.5-5.5**

**HyperK+** – weakness, tetany.

ECG – loss of p-waves; prolonged PR interval; widened QRS, peaked T-waves; v-fib; asystole

Management:

1. If > 5.5 but < 6.5 🡪 home Rx: Diuretics (Furosemide/HCTZ) and K-exelate 15-30g qid
2. If > 6.5 but NO ECG changes: shift it: Insulin/dextrose infusion (10IU/50ml 50% dextrose over 20 minutes) ---- (apparently it’s better to give it stat?)
3. If > 6.5 WITH ECG changes: VERY BAD and needs following management:

1st) Insulin/Dextrose

2nd) CaCl2 10ml of 10% solution

3rd) Create an alkalosis – NaHCO3 (8.4%) 50-100ml. OR hyperventilate

**HypoK+:** ECG: T-wave flattening, u-waves, prolonged PR interval, 1st/2nd degree blocks

**Rx:** Peripheral line – not more than 40mmol K+/litre NaCl. Central (60). Rate: 20mmol over 30 minutes with ECG monitoring. K+ extremely irritating to blood vessels, phlebitis/burning.

1. **Ischaemic Heart Disease:**

**Dx**: Clinical Symptoms; ECG- ST-changes, T-wave inversion, arrhythmias, **Q-WAVES.**

Stress ECG 🡪 Angiography.

 Pharmacological stress test: Dobutamine.

1. **HT URGENCY/EMERGENCY**

BP > 240/120 WITHOUT/WITH acute symptoms of end-organ damage.

Brain/Heart 1st, Kidney/Eyes 2nd.

1. **Perioperative Cardiac Arrhythmias**

**Narrow-complex SVT –** WITH haemodynamic compromise: electrical/chemical cardioversion

WITHOUT compromise: Rate control (esmolol); CCB – verapamil. Consider amiodarone/adenosine.

**Broad-complex SVT –** Distinguish between ventricular or atrial origin (different treatments). Do not give adenosine in order to distinguish the origin. Consider lignocaine/amiodarone.

**Ventricular Tachycardias –** risk factors: R on T phenomenon; bigeminus; 3 extrasystoles in a row; multifocal/polymorphic complexes; > 5 extrasystoles/min.

**VT:** lignocaine, amiodarone, MgSO4

**VF/Unstable VT:** defibrillate

**2nd/3rd degree heart block:**IV pacemakers

1. **ATROPINE VS GLYCOPYRROLATE**

|  |  |
| --- | --- |
| **ATROPINE** | **GLYCOPYRROLATE** |
| TERTIARY AMINE – Crosses Barriers | QUATERNARY AMINE- Does not cross barriers |
| Bad Side Effects – CNS: Confusion etc | Fewer Side Effects |
| Faster Onset – good for emergency | Slower Onset but works longer |
| Can be given ORALLY eg for premed | Can only be given IV |
| ↑ HR more than Glycopyrrolate | Less ↑ in HR than Atropine |
| Does not ↓ secretions as well | ↓ secretions better (4x) |
| Not safe in Glaucoma | Safe in Glaucoma |
| Avoid or be very careful in IHD/elderly. | No real contra-indication |
| Cheaper | More expensive |

1. **Diabetes Mellitus**

**END-ORGAN DAMAGE:**

1. **CVS:** Atherosclerosis – micro/macroangiopathy

 Silent Ischaemia. MI/HT common. ↑ CCF

 Autonomic Neuropathy: orthostatic hypotension, loss beat-to-beat variation.

 Sudden peri-operative death

1. **RENAL:** Glomerulosclerosis, papillary necrosis, chronic renal failure.

↑ risk acute failure post-op. ↑ risk for urosepsis

1. **NEUROPATHY:** Peripheral

Autonomic: diarrhoea, neurogenic bladder, impotence, post hypotension

1. **GASTRO-INTESTINAL:** Delayed stomach emptying (aspiration risk)
2. **STIFF JOINTS:**  Atlanto-occipital movement restricted (difficult tubing)
3. **METABOLIC DECOMPENSATION:** Hypoglycaemia; DKA; Non-ketotic hyperosmolar crisis; lactic acidosis (especially if on Biguanide eg Metformin)

**EXAMPLE:** Patient coming in for amputation of leg for sepsis. Problem is severe dehydration and in DKA. Resuscitate first before surgery. Must resus glucose, ketones, fluid, pH . Give lots of fluids, watch K+. Put up CVP, catheter. Do RSI at our level for diabetics (aspiration risks).

Resus fluid: Use Ringers rather than N/S, rather deal with lactate 🡪 glucose than acidotic saline

pH of 7.2 or >; Urine output at least 0.5ml/kg/hr; Normalise K+ ; Glucose < 15; correct acid-base.

1. **OBESITY**

Anatomical Differences:

-Difficult intubation: short, thick neck; excessive pharyngeal soft-tissue; large tongue; anterior larynx. Require an intubation pillow and equipment for difficult intubation.

-Difficult to mask ventilate/drip/blood pressure cuff (A-line sometimes necessary)

-↑ risk aspiration- delayed gastric emptying. RSI safest

- Careful positioning: pressure sores/DVTs/nerve injury. Early mobiliation NB.

Respiratory: ↑O2 use/ CO2 PRODUCTION. ↑minute volume. ↑ work of breathing.

 ↓FRC – pre-oxygenation is essential.

 ↓Chest-wall compliance

 Do better with controlled ventilation

 Look out for Sleep Apnoea

 Extubate awake/administer post-op O2

Cardiovascular: ↑ blood volume & CO

 HT common. Ventricular Hypertrophy .OSA may have Pulmonary Hypertension

 ↑ incidence IHD, DM, high cholesterol, Metabolic Syndrome.

1. **PORPHYRIA:**

-↑ precursors in the heme-synthesis pathway after a stressful occurrence/trigger.

-Certain drugs induce the enzyme eg Sodium Thiopentone/Sulphurs/Steroids

-Precursors cause acute neuropathies + - skin problems, chronic causes skin problems:

CVS: severe hypotension/dysrhythmias

 Severe abdominal pain mimicking appendicitis

 CNS: Convulsions, depression, psychosis

-RX: ABCs; Carbohydrates (10% Dextrose saline) slow process (high carb diet/glucose drip in theatre).

- Haematin 3mg/kg/day

-RX: N+V , convulsions, pain, anxiety, hydrate patient well

1. **MALIGNANT HYPERTHERMIA:**

-Inherited disease. Problem at Ryanodine Ca2+ channel receptor causing uncontrolled release of intracellular sarcoplasmic Ca2+ with resultant contraction of muscles. 50% Masseter Muscle spasm.

-Severe rigidity; consumes energy, release CO2 ,myoglobinuria, metabolic then respiratory acidosis

-Children 1:15 000 (be careful in children with muscle disease, strabismus, cleft lip/palate).

 Adults 1: 50 000.

* Triggered by SCOLINE and all VOLATILES (except N2O)
* **SNX**: Rigid, Tachy, ↑ CO2, ↑ Temp.
* **DIFF**: **Think hypoventilation!!!!!** Endocrine – hyperthyroidism, thyroid storm, phaechromocytoma. Neuroleptic malignant syndrome. Sepsis. Iatrogenic (BairHuggers).
* **RX**: **STOP TRIGGER!** Can’t stop Scoline. Stop Vapour and switch to TIVA (propofol).

Cool the patient down 🡪 run in cold fluids, lavage with fluid, Ice at neck/groin/forehead. Cool room.

**RX Acidosis:** Hyperventilate + bicarb. **RX Hyperkalaemia. RX Myoglobinuria:** Diuresis/dialysis.

**RX Dysrhythmias:** as they occur.

**DEFINITIVE: DANTROLENE SODIUM.** Ca2+ antagonist. Give through blood filter

**When Stable:** Take to ICU, can recur. Must have genetic test as well as family. Muscle biopsy.

1. **Propofol Infusion Syndrome (PRIS):**

Decreased CO, severe metabolic acidosis, increased blood lipids.

1. **Causes of Delayed Awakening**

Eg Patient for hysterectomy: gave **midazolam** premed. Gave **sufenta**, induced with **propofol**, intubated after giving **scoline**. After 5 minutes, gave **vecuronium** and maintained with **sevoflurane**. Delayed Awakening.

1)Midazolam: Reverse with flumazenil

2)Opioids: Sufenta – pinpoint pupils. Reverse with Naloxone (but short-acting).

3)Propofol: Possibly in TIVA or Obese patient

4)Scoline – Apnoea

5)Vecuronium – inadequate reversal (test with nerve stimulator to differentiate)

6)Sevoflurane: Forgot to turn off. ↓ clearance if obese

Also remember: DELAYED AWAKENING CAN ALSO BE:

-Hypothermia; Acid-Base derangements; ↑CO2 derangement (CO2 narcosis); ↓Na+/Ca2+; hypermagnesaemia (eg PET patients on MgSO4); Cirrhosis, Renal Failure; Muscle Diseases; MI/Stroke; Hypothyroidism; Comas – hypoglycaemic, DKA etc.

1. **SCOLINE APNOEA:**
2. INHERITED: homozygous (both genes) – can take up to 6 hours to reverse

 Heterozygous: (one gene) – takes faster than homozygous

1. ACQUIRED: Pregnancy: (↑ body water, ↑VOD, dilution of pseudocholinesterase).

 Neonates: underdeveloped liver, ↑ body water.

 Plasmaphoresis

**RX:** Support patient. **VENTILATE + SEDATE (amnesia).** Give pseudocholinesterase in theory in FFPs but risk of infection so not really in SA. Must wear medic alert bracelet.

1. **MUSCLE RELAXANTS:**

-Hypokalaemia/Hypermagnesaemia potentiate muscle relaxants

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **CLASSIFICATION** | **DRUG****(FRIDGE)** | **TYPE** | **ONSET/DURATION OF ACTION** | **OTHER:** |
| **ULTRA – SHORT ACTING** | **SCOLINE**(Suxamethonium) | Depolarising | 1 minute5 minutes |  |
| **SHORT ACTING** | **MIVACURIUM** | Non- DepolarisingIso-steroid | 2-3 minutes15 minutes | Possibly Mivacurium Apnoea |
| **MEDIUM ACTING** | **ATRACURIUM** | Non-DepolarisingBenz-isoquinolone | 2-3 minutes25 minutes | Good- renal failureBad-Histamine release |
|  | **VECURONIUM** | Non- DepolarisingIso-steroid | 2 minutes45 minutes | CVS stable |
|  | **ROCURONIUM** | Non- DepolarisingIso-steroid | 1 minute45 minutes | Rarely histamine release |
| **LONG ACTING** | **PANCURONIUM** | Non- DepolarisingIso-steroid | 2-3 minutes> 45 minutes | Tachy + hypotension(avoid in IHD/renal disease). Vagolytic |

**Depolarisers: SCOLINE:** Looks like 2 molecules Ach. Works as an AGONIST 🡪 causes muscle twitching/fasciculations. Broken down by pseudocholinesterase in blood THEREFORE works longer than Ach.

There are two phases to the depolarizing block. During phase I (depolarizing phase), they cause muscular [fasciculations](http://en.wikipedia.org/wiki/Fasciculation) (muscle twitches) while they are depolarizing the muscle fibers. Eventually, after sufficient depolarization has occurred, phase II (desensitising phase) sets in and the muscle is no longer responsive to acetylcholine released by the [motorneurons](http://en.wikipedia.org/wiki/Motoneuron). At this point, full neuromuscular block has been achieved.

**Non-depolarisers:** ANTAGONISTS. Block Ach from receptor. Reverse by ↑ Ach, kicks Non-depolariser off, Ach can bind again. 🡪 use Neostigmine : Stops Ach-Esterase 🡪 Therefore an Ach-E inhibitor 🡪but non-specific (works at muscarinic as well as nicotinic) so counteract the side-effects of Neostigmine by giving atropine/glycopyrrolate BEFORE giving Neostigmine.

ED95 – Effective Dose that will ↓ a twitch by 95% --- give 3x ED95 in order to intubate.

Snx inadequate relaxation: Surgeon not happy (patient pushes). Spontaneous breathing on capnogram (curare cleft. Tearing. Nerve-stimulator recovery. ↑ in airway pressure.

Can only give neostigmine when 3+4 are back on train-of-four. Can extubate when 4:1 is 80%

Adequate reversal: Lift head for 5 seconds; open eyes, strong grip, cough adequately etc

Inadequate: (Opposite) – ptosis, fish on dry land, tracheal tug, ineffective cough, paradoxical breathing

**SCOLINE –Side Effects and Contra-Indications**

1. Scoline Apnoea. No/little pseudocholinesterase to break down Ach.

 Hereditary -------> (Genetic)

 Acquired ---------> Physiological (Pregnancy)

 Pathological (Organophosphate exposure/poisoning)

1. Massive POTASSIUM release : ↑Ach receptors, overstimulation with acetylcholine, K+ release

ABSOLUTE C/I : Spinal Cord Transsection (quad/paraplegia)

 Burns: 1-60 days

RELATIVE C/I : Severe intra-abdominal sepsis Disuse atrophy

Massive Trauma Prolonged immobilisation CNS infection/injury (10-60 days post insult)

1. Myalgia : Fasciculations, may be debilitating. Give small dose non-depolariser eg 1mg rocuronium before giving scoline (followed by higher doses scoline)
2. Malignant Hyperthermia : Rare, pathological release of Ca 2+ from muscle. ↑Temp, ↑ CO2 levels, ↑HR, Metabolic Acidosis. RX Dantrolene asap (Dantrolene ring of hospitals). More common is masseter muscle spasm in kids. Monitor temp, myoglobin and CK levels in high care.
3. Hypersensitivity Reactions
4. Arrhythmias: Tachy and brady-arrhythmias. Give atropine in children before first dose (2nd dose in adults).

**Iso-steroids in general: DONT** CAUSE HISTAMINE RELEASE (RARELY ROCURONIUM)

If you cannot Intubate/ventilate, you need to make an emergency airway

Only use Pancuronium if there is a long surgery (>3 hours)

Atracurium, 10 isomers, > chance histamine release ( cis-atracurium has only 1 isomer)

Atracurium, broken down by Hoffman Elimination (spontaneous due to temp and pH in body – forms laudanosine (convulsions in dogs but not seen in humans) and ester hydrolysis. Therefore good choice in renal failure patients. (Cis-atracurium only by Hoffmann Elimination, therefore duration of action longer than atracurium).

1. **Local Anaesthetics**

Lignocaine – fast onset (minutes), short offset (1 hour).

Bupivicaine (Macaine) – slower onset (30 min), lasts longer (2-4hours)

|  |  |
| --- | --- |
| **AMIDES (broken down by liver)** | **ESTERS (ester hydrolysis-pseudocholinesterase)** |
| Lignocaine (Xylocaine) | Procaine |
| Bupivicaine (Macaine) | Chloroprocaine |
| L – Bupivicaine (Chirocaine) | Tetracaine |
| Ropivicaine | Cocaine |

Addition of vasoconstrictor: eg adrenaline ---slows down absorption of drug, lowers risk for toxicity, prolongs the block.

Decreasing order of risk of absorption:

Intercostal > caudal >epidural >brachial plexus >spinal

**NOTE**: the concentration of the local anaesthetic is written as a % on the vial.

Convert: 1% = 1g/100ml = 1000mg/100ml = 10mg/ml

(rule of 10 ------ x10)

**MAXIMUM DOSES: vasoconstrictor (adrenaline, 5ug/ml ----- 1:200 000)**

1. Lignocaine (without adrenaline) --------------------3-4mg/kg
2. Bupivicaine, L- Bupivicaine, Ropivicaine-----------2mg/kg max (150mg)
3. Lignocaine (with adrenaline) ------------- 7mg/kg
4. Same Bupivicaine, L- Bupivicaine, Ropivicaine--- 2mg/kg max (150mg)

**Complications of administration of local anaesthetics :**

* **Immediate**: Hypotension
	+ - Respiratory Distress
		- Pain on injection

|  |  |  |  |
| --- | --- | --- | --- |
|  | **CNS** (more lignocaine) |  | **CVS** (more bupivicaine) |
| **INITIAL PHASE** | Circumoral paraesthesia | **INITIAL PHASE** | Hypertension |
| Tinnitus | Tachycardia  |
| Confusion |  |
| **EXCITATORY PHASE** | Convulsions (RX diazepam 5-20mg) + scoline + intubation/ventilation) | **INTERMEDIARY PHASE** | Myocardial Depression |
| ↓Cardiac Output |
| Hypotension |
| **DEPRESSIVE PHASE** |  | **TERMINAL PHASE** | Peripheral Vasodilation |
| Loss of consciousness | Severe Hypotension |
| COMA | Sinus Bradycardia |
| Respiratory Arrest | Conduction defects/VD/VF |

* + - Toxic (nerve toxicity, myotoxicity, systemic-see table)

**RX TOXICITY**: Identify symptoms; resus equipment available; give O2/ventilate; hypotension PEP 50-100ug/adrenaline 5-10ug boluses; convulsions diazepam – scoline – intubate and ventilate; VF – intralipid (effective antidote for bupivicaine-induced CVS collapse). (Ropivicaine / L-bupivicaine better).

* **Intermediate:** Motor paralysis + urinary retention
* **Late:** 1) Neurological damage – nerve trauma, ant spinal aa syndrome, arachnoiditis, spinal cord pressure

2) Pneumothorax

3) Headache

1. **Pre – operative assessment**

PRE-OPERATIVE ASSESSMENT -------------**AGE NAAS PC**

1. **A**naesthetics. Previous, complications, allergies, difficult airways, ICU, type etc.
2. **G**MHx. THREAD. TB, Hypertension (ACE-I can cause severe hypotension, any anticoagulants, Wafarin stopped 5 days pre-op. INR minimum 1.5), Rheumatoid Arthritis (difficult airway), Epilepsy (drug interactions and clearance), Asthma (corticosteroids and Addisonian Crisis, bronchospasm, control etc), Diabetes (insulin during NPO period, glycosylation of joints).
3. **E**xamination. According to history, as for internal medicine. Check for valve lesions (eg fixed CO with stenotic heart valves).
4. **N**PO. 6 hours solids, 4 hours fluids, 2 hours absolutely NPO.
5. **A**irway. Look at patient. Retracted mandible? Obese? Pregnant? Neck swelling? Previous scarring? BONES : Beard, Obese, No teeth, Elderly, Snoring HX . Fingers between teeth (<3 may be difficult). Mallampati score (1-4). Patil test (Thyromental distance should be >6.5cm). Sternomental height with neck in full extension should be > 12.5 cm.
6. **A**SA Score. 1) Healthy, no comorbidities.
7. Mild Systemic Illness eg HT/DM well controlled
8. Systemic illness not controlled/previous MI but patient is not in immediate danger.
9. Patient requires surgery urgently, threat to life.
10. Moribund, organ donor, won’t live> 24 hours.
11. E for any of the above ------EMERGENCY
12. **S**pecial Investigations: ECG/CXR if necessary, glucose, FBC and HCT if anaemic or electrolyte disturbance, lung function tests if thoracic surgery.
13. **P**remedication and **C**onsent

**NB:**  The aim of the **pre-medication** is to allow the patient to enter the theatre complex **relaxed** and well **sedated** but still **cooperative**. OTHER goals are:

* **Analgesia** – opioids, morphine/pethidine
* **Amnesia**(anterograde) /**Anxiolysis** ­- BZD, midazolam 7.5mg
* Reductions of **secretions** and vagal activity. Atropine/Glycopyrrolate
* ↑ Gastric pH and accelerate stomach emptying. Sodium Citrate
* Metoclopramide (↓N + V)
* Insulin sliding if a diabetic etc.

**Mallampati Score**

1-2 relatively easy

3-4 potentially difficult

1. **OBSTETRIC ANAESTHESIA**

|  |  |
| --- | --- |
| **Organ System where changes occur** | **PHYSIOLOGICAL CHANGE** |
| **HAEMATOLOGICAL**More plasma, more blood, but less HB relatively | 1. ↑ plasma volume (45%)
 |
| 1. ↑Total blood volume (35%)
 |
| 1. Dilutional Anaemia (Hct = 35%)
 |
|  |
| **CARDIOVASCULAR** | 1. ↑CO by 30-50%
 |
| More blood out at faster rate, blood vessels squashed, less resistance to pump against | 1. Aorto-caval compression (10%)
 |
| 1. HR ↑ (15-30%)
 |
| 1. PVR ↓ 15%
 |
|  |
| **RESPIRATORY** | 1. ↑Alveolar ventilation (70%)
 |
| Faster, more ventilation but less reserve, less amounts of CO2 | 1. ↓FRC (20%)
 |
| 1. RR ↑ 15%
 |
| 1. ↓PaCO2 (30%)
 |
|  |
| **GASTROINTESTINAL** | 1. Delayed gastric emptying
 |
| CONTENTS IN LONGER, CONTENTS COME UP | 1. ↓LES tone with GORD
 |
|  |
| **ALTERED RESPONSE TO DRUGS** | 1. ↓amounts inhalants ↓ MAC
 |
| (LESS AMOUNTS REQUIRED) | 1. ↓LA for spinal/epidural
 |
| 1. ↓Thiopentone requirements
 |

**Haematological, CVS, resp, GI, drugs**

**NB: RAPID SEQUENCE INDUCTION:**

1. PRE-O2 (3 MINUTES)
2. INDUCTION AGENT QUICKLY
3. CRICOID PRESSURE
4. SCOLINE 1mg/kg
5. INTUBATE
6. CHECK FOR TRACHEAL INTUBATION (no first sound over epigastrium, listen to axillae, confirm bilateral, check capnograph, see vapour in ET tube, chest expansions seen.
7. RELEASE CRICOID PRESSURE

**NB: ↑RISKS FOR ASPIRATION IN PREGNANCY**

1. ↑ abdominal pressure ----- Large uterus
2. ↑ gastrin production. ↑ volume and acid content of secretions
3. ↓motilin secretion, delayed gastric emptying
4. Change in cardio-oesophageal angle, sphincter less efficient
5. ↑release progesterone, relaxes smooth muscle, ↓ gastric emptying.

**NB: PREVENTION OF REGURGITATION/ASPIRATION**

1. Keep NPO from start of labour
2. Few or no opiates given
3. Avoid GA if possible (or else use rapid sequence induction, avoiding lithotomy position)
4. Experienced anaesthetist
5. Give sodium citrate, maxolon, ranitidine to prevent acid pneumonitis.

**NB:** PATIENT IN LEFT-LATERAL TILT (30®) -----PREVENT **AORTO-CAVAL COMPRESSION**. CAN’T DO CPR UNLESS SHE’S IN THIS POSITION!!! **Clinically** : paleness, hypotension, sweating, reflex bradycardia in aorto-caval compression.

**PRE-ECLAMPSIA IN PREGNANCY (HT, PROTEINURIA, GENERALISED OEDEMA)**

Cerebral/pulmonary oedema ; IC bleeds ; IV dehydration (hypovolaemia) ; renal failure + oliguria ; cardiac failure ; coagulopathy (don’t do epidural/spinal!)

* Low-dose **aspirin** decreases incidence in those who are prone
* **Epidurals** are of benefit – uterine-placental blood flow ↑, good pain relief, decrease stress response, ↓BP. (must be normal clotting, optimal fluid status, foetal monitoring, no Aorto-caval compression.
* Prevent HT response in GA with **esmolol** ( beta blocker) , nitroglycerine or **alfentanil/remifentanil**, rx HT with **methyldopa.**
* **MgSO4**is good at therapeutic level of 2mmol/l. Don’t go higher 🡪 risk asystole. Antidote is CaCl2 or gluconate.
* **NB: Difficult airway; if urea >10 plt function decreased; HELLP syndrome**

**NB: ANAESTHESIA FOR CAESARIAN SECTIONS and POST-SPINAL HEADACHE (PDPH)**

* Spinal (subarachnoid) is method of choice.
* Opiates and local anaesthetics only.
* +- 12mg bupivicaine (2.5ml of 0.5% bupivicaine)

**NB** **Post-Dural puncture headache** ----🡪 leakage of CSF ---USUALLY when larger needles used

* Needle size – 26g mandatory (not in kalafong!!!)
* Bevel introduced sideways.
* Use pencil point needles.

**Treatment of PDPH**

* Conservative – bed rest and fluids
* NSAIDS/Caffeine
* Epidural blood patch 24 hours after initial puncture 🡪 STERILE, 20ml injected.

**NB POST-SPINAL HYPOTENSION (marked increase in Heart Rate)**

* Fluid + O2 + left lateral tilt
* Ephedrine 5-10mg bolus (↓ HR)
* PEP 50-100ug bolus (especially if tachycardic)
* Adrenaline 5-10ug bolus if above 2 don't work. (↓HR)

**NB Contra-indications to Epidurals/spinals**

* **Absolute**
	+ Patient refuses
	+ Coagulopathy/bleeding diathesis
	+ Trauma/shock/hypotension
	+ Raised Intracranial pressure (ICP)
	+ Infection over sight
	+ Fixed Cardiac Output (stenotic valve/tamponade)
	+ Allergies to local anaesthetic
* **Relative**
	+ Intracranial infection (meningitis/encephalitis)
	+ Spinal cord abnormality eg scoliosis or (congenital/acquired defect)
	+ Inexperienced operator
	+ Severe foetal distress ?
	+ Abruptio Placentae ?
	+ Myopathies/Motor Neuron Disease?
1. **Complications of Neuraxial Blocks**
* Adverse/exaggerated physiological response
* IV drug injection
* Nerve root damage/cauda equina/spinal or epidural haematoma
* Post-dural puncture headache
* High block
* Urinary retention
* Cardiac arrest
* Inflammation (arachnoiditis); inflammation (meningitis)
* Anterior spinal artery syndrome
* Horner’s syndrome
1. **DIFFERENTIAL DX OF INTRA-OPERATIVE BRONCHOSPASM:**

-Mechanical obstruction of ET Tube (kinking, secretion, overinflation)

-Inadequate depth of anaesthesia

-Endobronchial intubation

-Pulmonary aspiration

-Pulmonary oedema

-Pulmonary Embolus

-Pneumothorax

-Acute asthma attack

**RX:** Deepen anaesthesia (vapours potent bronchodilators), if ineffective – MgSO4 35mg/kg (careful as drops BP & potentiates DOA of muscle relaxants), Ketamine 0.25mg/kg (with glycopyrrolate to ↓ secretions), β-agonist 🡪adrenaline, if still refractory 🡪 lignocaine through ET Tube

1. **MODIFIED ALDRETE SCORE IN RECOVERY ROOM:**
2. ACTIVITY: Moves all 4 limbs: 2

 Moves 2 extremities: 1

 Cannot move limbs: 0

1. BREATHING: Able to breathe deeply and cough freely: 2

 Dyspnoea, shallow, limited breathing: 1

 Apnoea 0

1. CIRCULATION: BP within 20mmHg of preop level: 2

 BP 20-50 mmHg of preop level: 1

 BP > 50mmHg of preop level: 0

1. CONSCIOUSNESS: Fully awake: 2

Arousable on calling: 1

Unresponsive: 0

1. SATURATION: Saturation > 92% on room air: 2

Needs O2 to maintain sats > 92% 1

Sats < 90% on O2  0

MINIMUM SCORE OF 9 TO BE DISCHARGED FROM RECOVERY ROOM......

**Limited at does not include: PONV/PAIN/BLEEDING/DYSRHYTHMIAS/HYPOTHERMIA**

1. **STRUCTURES A SPINAL NEEDLE PASSES THROUGH**

Skin🡪Subcutaneous Tissue🡪Supraspinous Ligament🡪Interspinous Ligament🡪Ligamentum Flavum🡪Epidural Space🡪Dura🡪Subarachnoid Space

1. **FORMULAE:**
* WEIGHT: AGE x 2 +9
* ET Tube: AGE/4 + 4
* ET Tube depth: AGE/2 +12
* Acute Blood Loss > 10% replace with packed cells using either formula:
1. MAXIMUM ALLOWABLE BLOOD LOSS:

1.5 X kg X (Hct 2 – Hct 1 ) OR EBV X ( Hct 2 – Hct 1 )/Change in Hct

EBV = wt x blood volume = 90ml/kg Neonate

 85ml/kg Toddler

 75ml/kg Adult Male

 65ml/kg Adult Female

1. **ASPIRATION 🡪 not all results in syndrome : usually >25ml, pH 2.5 or <, particulate fluid**
2. ↑intra-abdominal pressure : pregnant, ascites,
3. Full stomach : NPO, trauma patients
4. Delayed Gastric Emptying : DM (neuropathy), trauma patients (pain delays gastric emptying), uremic patients.
5. Abnormality of GIT: Tumour, hernia, stenosis, diverticulum
6. Cannot protect own airway: Head injury, CP, intoxicated

**IDENTIFY:** sats drop, ausculate and hear a wheeze (diff dx: asthma, bronchospasm, pneumothorax etc)

**RX:** Suction what you can, give PEEP, recruitment breath, ↑FiO2, chemical pneumonitis therefore NOANTIBIOTICS**.** Give steroids to reduce inflammation. Bring back very seldomly for BAL if they desaturate further (might be food plug, collapsed segment etc)

1. **DRUGS RELEASING HISTAMINE**

-Thiopentone, Morphine/Pethidine, Atracurium/Cis-atracurium, NB is antibiotics!!!

1. **RESTRICTIVE (INSPIRATORY PROBLEM) VS OBSTRUCTIVE (EXPIRATORY PROBLEM) LUNG DISEASE**

|  |  |  |
| --- | --- | --- |
|  | **RESTRICTIVE** | **OBSTRUCTIVE** |
| **EXAMPLE** | Obesity/Pleural Effusion/ALI/ARDS | ASTHMA/COPD |
| **PROBLEM** | Inspiration | Expiration |
| **TIDAL VOLUME** | 4-6ml/kg | 10ml/kg  |
| **RESPIRATORY RATE** | Faster (maintain CO) | Slower (maintain CO) |
| **I:E RATIO** | 1:1-1.5 | 1:2.5-4 |
| **PEEP** | Do give PEEP | Like PEEP! +- 8 |
| **FiO2** | ↑FiO2 | Permissive hypoxia (88-92%) |

1. **Causes of continued CPR : Hs and Ts**

**Hs**

* [**H**ypovolemia](http://en.wikipedia.org/wiki/Hypovolemia) - A lack of circulating [body fluids](http://en.wikipedia.org/wiki/Body_fluids), principally [blood](http://en.wikipedia.org/wiki/Blood) volume. This is usually (though not exclusively) caused by some form of [bleeding](http://en.wikipedia.org/wiki/Bleeding), [anaphylaxis](http://en.wikipedia.org/wiki/Anaphylaxis), or [pregnancy](http://en.wikipedia.org/wiki/Pregnancy) with gravid uterus. [Peri-arrest treatment](http://en.wikipedia.org/wiki/Cardiac_arrest#Peri-arrest_period) includes giving [IV fluids](http://en.wikipedia.org/wiki/Intravenous_therapy) and [blood transfusions](http://en.wikipedia.org/wiki/Blood_transfusions), and controlling the source of any [bleeding](http://en.wikipedia.org/wiki/Bleeding) - by direct pressure for external bleeding, or emergency surgical techniques such as [esophageal banding](http://en.wikipedia.org/w/index.php?title=Esophageal_banding&action=edit&redlink=1), [gastroesophageal balloon tamponade](http://en.wikipedia.org/w/index.php?title=Gastroesophageal_balloon_tamponade&action=edit&redlink=1) (for treatment of [massive GI bleeding](http://en.wikipedia.org/w/index.php?title=Massive_GI_bleeding&action=edit&redlink=1) such as in [esophageal varices](http://en.wikipedia.org/wiki/Esophageal_varices)), [thoracotomy](http://en.wikipedia.org/wiki/Thoracotomy) in cases of penetrating trauma or significant shear forces applied to the chest, or [exploratory laparotomy](http://en.wikipedia.org/wiki/Exploratory_laparotomy) in cases of penetrating trauma, spontaneous rupture of major blood vessels, or rupture of a hollow viscus in the abdomen.
* [**H**ypoxia](http://en.wikipedia.org/wiki/Hypoxia_%28medical%29) - A lack of [oxygen](http://en.wikipedia.org/wiki/Oxygen) delivery to the [heart](http://en.wikipedia.org/wiki/Heart), [brain](http://en.wikipedia.org/wiki/Brain) and other [vital organs](http://en.wikipedia.org/wiki/Vital_organ). Rapid assessment of airway patency and respiratory effort must be performed. If the patient is mechanically ventilated, the presence of breath sounds and the proper placement of the endotracheal tube should be verified. Treatment may include providing oxygen, proper ventilation, and good [CPR](http://en.wikipedia.org/wiki/Cardiopulmonary_resuscitation) technique. In cases of [carbon monoxide](http://en.wikipedia.org/wiki/Carbon_monoxide) poisoning or [cyanide](http://en.wikipedia.org/wiki/Cyanide) poisoning, [hyperbaric oxygen](http://en.wikipedia.org/wiki/Hyperbaric_oxygen) may be employed after the patient is stabilized.
* [**H**ydrogen](http://en.wikipedia.org/wiki/Hydrogen) ions ([Acidosis](http://en.wikipedia.org/wiki/Acidosis)) - An abnormal pH in the body as a result of [lactic acidosis](http://en.wikipedia.org/wiki/Lactic_acidosis) which occurs in prolonged hypoxia and in severe infection, [diabetic ketoacidosis](http://en.wikipedia.org/wiki/Diabetic_ketoacidosis), [renal failure](http://en.wikipedia.org/wiki/Renal_failure) causing [uremia](http://en.wikipedia.org/wiki/Uremia), or ingestion of toxic agents or overdose of pharmacological agents, such as [aspirin](http://en.wikipedia.org/wiki/Aspirin) and other [salicylates](http://en.wikipedia.org/wiki/Salicylates), [ethanol](http://en.wikipedia.org/wiki/Ethanol), [ethylene glycol](http://en.wikipedia.org/wiki/Ethylene_glycol) and other [alcohols](http://en.wikipedia.org/wiki/Alcohols), [tricyclic antidepressants](http://en.wikipedia.org/wiki/Tricyclic_antidepressants), [isoniazid](http://en.wikipedia.org/wiki/Isoniazid), or [iron sulfate](http://en.wikipedia.org/wiki/Iron_sulfate). This can be treated with proper ventilation, good [CPR](http://en.wikipedia.org/wiki/Cardiopulmonary_resuscitation) technique, buffers like [sodium bicarbonate](http://en.wikipedia.org/wiki/Sodium_bicarbonate), and in select cases may require emergent [hemodialysis](http://en.wikipedia.org/wiki/Hemodialysis).
* [**H**yperkalemia](http://en.wikipedia.org/wiki/Hyperkalemia) or [**H**ypokalemia](http://en.wikipedia.org/wiki/Hypokalemia) - Both excess and inadequate potassium can be life-threatening. A common presentation of hyperkalemia is in the patient with [end-stage renal disease](http://en.wikipedia.org/wiki/End-stage_renal_disease) who has missed a [dialysis](http://en.wikipedia.org/wiki/Dialysis) appointment and presents with [weakness](http://en.wikipedia.org/wiki/Weakness), [nausea](http://en.wikipedia.org/wiki/Nausea), and broad [QRS complexes](http://en.wikipedia.org/wiki/QRS_complex) on the [electrocardiogram](http://en.wikipedia.org/wiki/Electrocardiogram). (Note however that patients with [chronic kidney disease](http://en.wikipedia.org/wiki/Chronic_kidney_disease) are often more tolerant of high potassium levels as their body often adapts to it.) The [electrocardiogram](http://en.wikipedia.org/wiki/Electrocardiogram) will show tall, peaked T waves (often larger than the R wave) or can degenerate into a sine wave as the QRS complex widens. Immediate initial therapy is the administration of [calcium](http://en.wikipedia.org/wiki/Calcium), either as [calcium gluconate](http://en.wikipedia.org/wiki/Calcium_gluconate) or [calcium chloride](http://en.wikipedia.org/wiki/Calcium_chloride). This stabilizes the electrochemical potential of cardiac myocytes, thereby preventing the development of fatal arrhythmias. This is, however, only a temporary measure. Other temporary measures may include [nebulized](http://en.wikipedia.org/wiki/Nebulizer) [salbutamol](http://en.wikipedia.org/wiki/Salbutamol), intravenous [insulin](http://en.wikipedia.org/wiki/Insulin) (usually given in combination with [glucose](http://en.wikipedia.org/wiki/Glucose), and [sodium bicarbonate](http://en.wikipedia.org/wiki/Sodium_bicarbonate)) which all temporarily drive potassium intracellularly. Definitive treatment of hyperkalemia requires actual excretion of potassium, either through urine (which can be facilitated by administration of [loop diuretics](http://en.wikipedia.org/wiki/Loop_diuretics) such as [furosemide](http://en.wikipedia.org/wiki/Furosemide)) or in the stool (which is accomplished by giving [sodium polystyrene sulfonate](http://en.wikipedia.org/wiki/Sodium_polystyrene_sulfonate) enterally, where it will bind potassium in the GI tract.) Severe cases will require emergent [hemodialysis](http://en.wikipedia.org/wiki/Hemodialysis). The diagnosis of [hypokalemia](http://en.wikipedia.org/wiki/Hypokalemia) (not enough [potassium](http://en.wikipedia.org/wiki/Potassium)) can be suspected when there is a history of [diarrhoea](http://en.wikipedia.org/wiki/Diarrhoea) or [malnutrition](http://en.wikipedia.org/wiki/Malnutrition). Loop [diuretics](http://en.wikipedia.org/wiki/Diuretic) may also contribute. The [electrocardiogram](http://en.wikipedia.org/wiki/Electrocardiogram) may show flattening of T waves and prominent U waves. [Hypokalemia](http://en.wikipedia.org/wiki/Hypokalemia) is an important cause of acquired [long QT syndrome](http://en.wikipedia.org/wiki/Long_QT_syndrome), and may predispose the patient to [torsades de pointes](http://en.wikipedia.org/wiki/Torsades_de_pointes). [Digitalis](http://en.wikipedia.org/wiki/Digitalis) use may increase the risk that [hypokalemia](http://en.wikipedia.org/wiki/Hypokalemia) will produce life threatening [arrhythmias](http://en.wikipedia.org/wiki/Arrhythmia). Hypokalemia is especially dangerous in patients with [ischemic heart disease](http://en.wikipedia.org/wiki/Ischemic_heart_disease).
* [**H**ypothermia](http://en.wikipedia.org/wiki/Hypothermia) - A low [core body temperature](http://en.wikipedia.org/wiki/Body_temperature), defined clinically as a [temperature](http://en.wikipedia.org/wiki/Temperature) of less than 35 degrees Celsius. The patient is re-warmed either by using a [cardiac bypass](http://en.wikipedia.org/wiki/Heart-lung_machine) or by irrigation of the body cavities (such as thorax, peritoneum, bladder) with warm fluids; or warmed [IV](http://en.wikipedia.org/wiki/IV) fluids. [CPR](http://en.wikipedia.org/wiki/CPR) only is given until the core body temperature reached 30 degrees Celsius, as [defibrillation](http://en.wikipedia.org/wiki/Defibrillation) is ineffective at lower temperatures. Patients have been known to be successfully resuscitated after periods of hours in hypothermia and cardiac arrest, and this has given rise to the often-quoted medical [truism](http://en.wikipedia.org/wiki/Truism), "You're not dead until you're warm and dead."
* [**H**ypoglycemia](http://en.wikipedia.org/wiki/Hypoglycemia) or [**H**yperglycemia](http://en.wikipedia.org/wiki/Hyperglycemia) - Low blood glucose from overdose of [oral hypoglycemics](http://en.wikipedia.org/wiki/Oral_hypoglycemics) such as [sulfonylureas](http://en.wikipedia.org/wiki/Sulfonylureas), or overdose of [insulin](http://en.wikipedia.org/wiki/Insulin). Rare endocrine disorders can also cause unexpected hypoglycemia. Generally, hyperglycemia is itself not fatal, however [DKA](http://en.wikipedia.org/wiki/Diabetic_ketoacidosis) will cause pH to drop, and [nonketotic hyperosmolar coma](http://en.wikipedia.org/wiki/Nonketotic_hyperosmolar_coma) leads to a severely hypovolemic state. Hypoglycemia is corrected rapidly by intravenous administration of concentrated glucose (typically 25 ml of 50% glucose in adults, but in children 25% glucose is used, and in neonates 10% glucose is used.) However, the patient will often require a continuous intravenous drip until the causative agent is completely metabolized. In DKA, the goal is correction of acidosis. In NKH, the goal is adequate fluid resuscitation.

**Ts**

* [**T**ablets](http://en.wikipedia.org/wiki/Tablets) or [**T**oxins](http://en.wikipedia.org/wiki/Toxins) - [Tricyclic antidepressants](http://en.wikipedia.org/wiki/Tricyclic_antidepressant), [phenothiazines](http://en.wikipedia.org/wiki/Phenothiazines), [beta blockers](http://en.wikipedia.org/wiki/Beta_blocker), [calcium channel blockers](http://en.wikipedia.org/wiki/Calcium_channel_blocker), [cocaine](http://en.wikipedia.org/wiki/Cocaine), [digoxin](http://en.wikipedia.org/wiki/Digoxin), [aspirin](http://en.wikipedia.org/wiki/Aspirin), [acetominophen](http://en.wikipedia.org/wiki/Acetominophen). This may be evidenced by items found on or around the patient, the patient's medical history (i.e. drug abuse, medication) taken from family and friends, checking the [medical records](http://en.wikipedia.org/wiki/Medical_records) to make sure no interacting drugs were prescribed, or sending [blood](http://en.wikipedia.org/wiki/Blood) and [urine](http://en.wikipedia.org/wiki/Urine) samples to the [toxicology](http://en.wikipedia.org/wiki/Toxicology) lab for report. Treatment may include specific [antidotes](http://en.wikipedia.org/wiki/Antidote), fluids for volume expansion, vasopressors, [sodium bicarbonate](http://en.wikipedia.org/wiki/Sodium_bicarbonate) (for [tricyclic antidepressants](http://en.wikipedia.org/wiki/Tricyclic_antidepressant)), [glucagon](http://en.wikipedia.org/wiki/Glucagon) or [calcium](http://en.wikipedia.org/wiki/Calcium) (for [calcium channel blockers](http://en.wikipedia.org/wiki/Calcium_channel_blocker)), [benzodiazepines](http://en.wikipedia.org/wiki/Benzodiazepine) (for [cocaine](http://en.wikipedia.org/wiki/Cocaine)), or [cardiopulmonary bypass](http://en.wikipedia.org/wiki/Cardiopulmonary_bypass). Herbal supplements and over-the-counter medications should also be considered.
* [Cardiac **T**amponade](http://en.wikipedia.org/wiki/Cardiac_tamponade) - Blood or other fluids building up in the [pericardium](http://en.wikipedia.org/wiki/Pericardium) can put pressure on the heart so that it is not able to beat. This condition can be recognized by the presence of a narrowing [pulse pressure](http://en.wikipedia.org/wiki/Pulse_pressure), muffled [heart sounds](http://en.wikipedia.org/wiki/Heart_sounds), distended neck veins, [electrical alternans](http://en.wikipedia.org/wiki/Electrical_alternans) on the [electrocardiogram](http://en.wikipedia.org/wiki/Electrocardiogram), or by visualization on [echocardiogram](http://en.wikipedia.org/wiki/Echocardiogram). This is treated in an emergency by inserting a needle into the [pericardium](http://en.wikipedia.org/wiki/Pericardium) to drain the fluid ([pericardiocentesis](http://en.wikipedia.org/wiki/Pericardiocentesis%22%20%5Co%20%22Pericardiocentesis)), or if the fluid is too thick then a [subxiphoid window](http://en.wikipedia.org/w/index.php?title=Subxiphoid_window&action=edit&redlink=1) is performed to cut the pericardium and release the fluid.
* [**T**ension pneumothorax](http://en.wikipedia.org/wiki/Tension_pneumothorax) - The build-up of air into one of the [pleural cavities](http://en.wikipedia.org/wiki/Pleural_cavity), which causes a [mediastinal](http://en.wikipedia.org/wiki/Mediastinum) shift. When this happens, the [great vessels](http://en.wikipedia.org/wiki/Great_vessels) (particularly the [superior vena cava](http://en.wikipedia.org/wiki/Superior_vena_cava)) become kinked, which limits [blood](http://en.wikipedia.org/wiki/Blood) return to the [heart](http://en.wikipedia.org/wiki/Heart). The condition can be recognized by severe air hunger, [hypoxia](http://en.wikipedia.org/wiki/Hypoxia_%28medical%29), jugular venous distension, hyperressonance to percussion on the affected side, and a tracheal shift away from the affected side. The tracheal shift often requires a chest [x-ray](http://en.wikipedia.org/wiki/X-ray) to appreciate (although treatment should be initiated prior to obtaining a chest x-ray if this condition is suspected). This is relieved by a needle [thoracotomy](http://en.wikipedia.org/wiki/Thoracotomy) (inserting a needle catheter) into the 2nd [intercostal space](http://en.wikipedia.org/wiki/Intercostal_space) at the mid-[clavicular](http://en.wikipedia.org/wiki/Clavicle%22%20%5Co%20%22Clavicle) line, which relieves the pressure in the [pleural cavity](http://en.wikipedia.org/wiki/Pleural_cavity).
* [**T**hrombosis](http://en.wikipedia.org/wiki/Thrombosis) ([Myocardial infarction](http://en.wikipedia.org/wiki/Myocardial_infarction)) - If the patient can be successfully resuscitated, there is a chance that the [myocardial infarction](http://en.wikipedia.org/wiki/Myocardial_infarction) can be treated, either with [thrombolytic therapy](http://en.wikipedia.org/wiki/Thrombolysis) or [percutaneous coronary intervention](http://en.wikipedia.org/wiki/Percutaneous_coronary_intervention).
* [**T**hromboembolism](http://en.wikipedia.org/wiki/Thrombosis) ([Pulmonary embolism](http://en.wikipedia.org/wiki/Pulmonary_embolism)) - hemodynamically significant pulmonary emboli are generally massive and typically fatal. Administration of [thrombolytics](http://en.wikipedia.org/wiki/Thrombolytics) can be attempted, and some specialized centers may perform [thrombolectomy](http://en.wikipedia.org/w/index.php?title=Thrombolectomy&action=edit&redlink=1), however, prognosis is generally poor.
* [**T**rauma](http://en.wikipedia.org/wiki/Trauma_%28medicine%29) - cardiac arrest can also occur after a hard blow to the chest at a precise moment in the cardiac cycle, which is known as [commotio cordis](http://en.wikipedia.org/wiki/Commotio_cordis). Other traumatic events such as high speed car crashes can cause sufficient structural damage to induce arrest.

**THEREFORE**:

-Hypoxia, Hypovolaemia, Hypo/Hyperglycaemia, Hypo/Hyperkalaemia, Hypothermia, H+ Ions (acidosis)

-Tablets/Toxins, Thromboembolism, Tamponade, Tension Pneumothorax, Thrombosis (MI), Trauma

1. **ACID-BASE CONTROVERSY**

**Severe Acidosis leads to:**

* ↓ Myocardial function, suppresses SA Node, ↓ diastolic depolarisation, ↓ threshold of VF, - inotropy, ↓ response to catecholamines, inhibits the CPR response 🡪 **SO SOMETIMES MUST GIVE NaHCO3 BUT: NaHCO3 causes:**
* Hyper**N**atraemia N
* Intracellular **A**cidosis A
* **H**yperosmolarity H
* Hyper**C**apnia C
* ↓ release of **O2** from the Hb to tissues 03
1. **RATE OF UPTAKE OF VAPOUR INTO BLOOD DEPENDS ON:**
	1. **↓ BGPC** - ↓ solubility, higher increase in alveolar partial pressure, quick induction/recovery
	2. **Cardiac Output:** Slower the output, faster the rise in alveolar partial pressure
	3. **[ ] of vapour given**
	4. **Ventilatory rate**
	5. **Potency (MAC):** MAC higher, higher fat solubility, ↑ potency
2. **INDICATIONS FOR INTUBATION: (ABSOLUTE INDICATION)**
3. AIRWAY MAINTENANCE: Competition for airway, adverse positions, difficulty holding mask
4. AIRWAY PROTECTION: Gastric Aspirate (RSI), pus/blood/saliva
5. VENTILATION: Muscle Relaxants, prolonged procedures
6. **ICU**: Tracheal toilet, ARDS, head injury
7. Extremes of age ?
8. **Assessment of correct ET Tube Placement**

-Visualise: Tubes pass through vocal cords, chest rising, bag moving, vapour in tube

-Ausculate: Axillae, apices, epigastrium,

-No abdominal distension

-Capnogram: Gold Standard (end-tidal CO2 trace)

1. **Management of unanticipated failed intubation (NB PASS FAIL QUESTION)**

-Only allowed **3 attempts** at Laryngoscopy

-Between each attempt, MUST **ventilate** with bag and mask

-Between attempts **improve** the situation by: improving LOV by flexing head more, using a stylet or introducer, change size ET tube, change laryngoscope blade.

-Have **suction** ready to remove secretions.

-If unable to ventilate with a mask, place an **LMA**.

-If can’t intubate, can’t ventilate 🡪 **cricothyroidotomy** (tracheostomy is NOT emergency procedure).

 **ALGORITHM FOR FAILED INTUBATION**

 ↓

IS THE OPERATION **URGENT** AND MUST **CONTINUE**?

 ↓

**IF YES: IS TUBE ESSENTIAL?**

**-YES 🡪** LMA + CRICOID PRESSURE 🡪 Intubating LMA and place tube through it.

 🡪 Tracheostomy

**- NO** 🡪 Mask; LMA; Nasopharyngeal airway

 **IF NO: WAKE THE PATIENT UP:** Local, spinal, epidural, fibreoptic laryngoscope, better anaesthetist.

1. **CAPNOGRAM CHANGES**

**A F**

**B G**

**C H**

****

**D I**

**E**

1. **Sudden Arrest F) Rebreathing CO**
2. **Hyperventilation G) Cardiac Oscillations**
3. **Pressure Effect eg Pregnant Uterus H) Spontaneous Breathing (curare clefts)**
4. **Embolism eg FAT/AIR I) Malignant Hyperthermia/hypoventilation**
5. **Bronchospasm**
6. **PEP**

**Phenylephrine** is a selective [α1-adrenergic receptor](http://en.wikipedia.org/wiki/Alpha-1_adrenergic_receptor) [agonist](http://en.wikipedia.org/wiki/Agonist). Primary effect is constriction of smooth muscle. It is used as a vasopressor to increase blood pressure by causing peripheral arterial vasoconstriction. It is especially useful in counteracting the hypotension caused by epidural/spinal anaesthesia. It has no inotropic or chronotropic effects on the heart and so increases the BP without increasing the heartrate or contractility. A reflex bradycardia may result due to the increased blood pressure. 50ug boluses to ↑ BP. Infusion: 0.5-1ug/kg/min. Dilute 10mg/ml vial into 200ml N/S ---- 50ug/ml.

1. **Post-Tonsillectomy Bleed:**

Divided into **primary** (within 24 hours post-op) and **secondary** ( >24 hours post-op).

**3 considerations:**

* Hypovolaemia (blood loss)
* Full stomach with aspiration (swallowed blood)
* Difficult Airway – blood, clots, swelling

**Management:**

* + 1. **Resuscitate** pt: Give fluids and order blood
		2. Proper **preparation** of theatre – suction MUST be working well (large amount of blood)
		3. Surgeon ready and scrubbed to assist with **surgical airway** if necessary
		4. **Prevent** **aspiration** by anaesthetising in one of following ways:
* **EASIEST IF INEXPERIENCED- Inhalational induction with cricoid pressure-** Left lateral position, head down to prevent aspiration of blood.
* **SAFEST IF EXPERIENCED- Rapid Sequence Induction and cricoid pressure –** Need to be skilled to intubate fast after muscle relaxant is given.
* **AWAKE INTUBATION –** In severely shocked patient.
	+ 1. Extubate ONLY when awake and well resuscitated.
1. **VAPOUR TABLE – BRIEF**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  | **MAC (%)** | **BGPC** | **VAPOUR PRESSURE (20°)** | **COLOUR CODING** | **SODA LIME** |
| **HALOTHANE** | 0.77 | 2.4 | 244mmHg | Red | Fosgene Gas, not NB |
| **ISOFLURANE** | 1.2 | 1.4 | 238mmHg | Purple | Carbon Monoxide ? |
| **DESFLURANE** | 6 | 0.42 | 672mmHg | Blue | Carbon Monoxide |
| **ENFLURANE** | 1.7 | 1.9 | 172mmHg | Orange | Nephrotoxic |
| **SEVOFLURANE** | 2 | 0.69 | **157mmHg** | Yellow | Compound A |
| **N2O** | 105 | 0.47 | 39 000mmHg | Blue |  |

1. **How do you know your sodalime is depleted?**

-↓pH changes colour of the granules to violet/purple when CO2 absorption capacity nearing maximum.

-Rebreathing of CO2 not absorbed causes the capnogram to never baseline (0).

1. **Advantages of circle system**

-Economical (↓FGF/inhalants). ↓ theatre pollution. Heated & humidified gases rebreathed.

-Inspired fraction of gases known

1. **Anaesthetic management of ruptured ectopic, BP 60/30, pulse 120.**
* The patient should be resuscitated as soon as possible. Although the resuscitation per se involves stopping the bleeding asap she should be given crystalloids or colloids IV via two large-bore drips while blood products are ordered on the way to theatre.
* Considering the patient is haemodynamically unstable with a MAP of 40mmHg, a neuraxial/regional technique is contraindicated as the resultant peripheral vasodilation would result in haemodynamic collapse and circulatory failure.
* A general anaesthetic is thus indicated. The patient is probably not NPO and thus requires a rapid sequence induction. I would use Ketamine TIVA for the operation as it is the perfect anaesthetic agent for a shocked patient. It increases the heart rate and cardiac output while simultaneously providing analgesia. Etomidate can also be used as it is CVS stable and will not drop the BP further.
1. **Tabulate differences of PEP, Ephedrine, Adrenaline**

|  |  |  |  |
| --- | --- | --- | --- |
|  | **PEP** | **ADRENALINE** | **EPHEDRINE** |
| **RECEPTOR** | -Selective α1- agonist | nonselective agonist α1; α2; β1; β2; β3 | ↑NA at α and β receptors |
| **EFFECTS** | -Vasoconstriction-↑BP by centralising blood volume | -↑HR, ↑ contractility,vasoconstriction, broncodilation, lipolysis, glycogenolysis | ↑activity of NA on adrenergic receptors(Indirect stimulation)Tachycardia, vasoconstrictionBronchodilatorWeight loss by thermogenesis |
| **DOSE FOR HYPOTENSION IN ADULTS** | -50-100ug boluses(1-2ml of amp diluted in 200ml N/S) | 5-10ug/min infusion(Usually 1ml bolus of 1:100 000) | 5-10mg boluses |

1. **How to dilute adrenaline:**

-Draw up 1ml of 1:1000 adrenaline. (1mg/ml). Dilute with 9ml N/S – now a 1: 10 000 solution (0.1mg/ml). Then take 1 ml of this and dilute to 9ml N/S – now a 1:100 000 solution

1. **Hypoxia versus Hypoxaemia**

-Hypoxia is inadequate oxygen supply to body tissues either in general or locally.

-Hypoxaeamia is a lowered partial pressure of arterial oxygen – usually defined as lower than 60mmHg or at a partial pressure that results in < 90% Hb saturation. Hypoxaemia may result in hypoxia.

1. **Anaesthetic for C/S**
* If no contraindication (see above) exists to neuraxial anaesthesia I would prefer to do a spinal. This avoids the complications of GA such as higher aspiration risk in pregnant women, transfer of drugs from mother-to-fetus as well as allows for bonding post-delivery.
1. **Dose for a spinal**

Depends on the level of anaesthesia you desire

Normally +- 12mg bupivicaine (2.5ml of 0.5% bupivicaine)

1. **Drugs to avoid in Renal Disease**

-**Muscle relaxants** – Scoline is CI if the potassium level is > 5. Avoid pancuronium and alcuronium(rather use atracurium/cis-atracurium). Vecuronium and mivacurium are relatively safe.

-**Induction agents** Their effect is terminated by redistribution. All of these agents are myocardial depressants and should be administered cautiously in patients with heart disease. Propofol is safe in renal impairment

**-Opioids** Morphine is metabolised in the liver to morphine-6-glucuronide which has about half the sedative effect of morphine with a markedly prolonged half life. Pethidine is partially metabolised to norpethidine which is less analgesic and has excitatory and convulsant properties. Both of these metabolites may accumulate in renal failure after repeated doses or with infusions. Standard intraoperative use will not usually cause problems. When available, morphine is preferable to pethidine. Fentanyl and alfentanil can be used as normal.

**-Benzodiazepines** can be used in renal failure.

**-Inhalational agents** There is decreased elimination of the fluoride ions which are significant metabolites of enflurane, sevoflurane which can worsen renal function, so these inhalational agents should be avoided especially if used at low flows. Use desflurane if possible.

**-Non steroidal anti inflammatory agents (NSAIDS)** should be avoided as all decrease renal blood flow and may precipitate complete renal failure.

**THEREFORE:**  avoid scoline, pancuronium, morphine and pethidine, sevoflurane and enflurane, NSAIDS

**What we got in our test:**

* 20kg 6 year old child coming in for tonsillectomy. Child not yet in theatre. What is the complete preparation of the theatre including drugs, equipment and special considerations. 10 marks
* 10 side effects of Scoline
* 5 marks: who is at risk for PONV
* 5 marks: how do you manage/prevent PONV
* 10 marks: complications of massive blood transfusion
* Renal failure pt: 20 year old for appendectomy. 5 marks: describe induction and maintenance in order, which drugs to give/avoid. 5 marks: other considerations in Renal failure.
* Compare PEP, adrenaline, Ephedrine, why does a spinal drop BP to 80/40, mechanism and how would you RX. What position will assist in improving the situation? 10 marks.
* OSCE: know all the values and what they mean on anaesthetic machine. Know different laryngoscope blade names and when you use which one and why.
* Know what circuit is low FGF values and why.
* Penile block: which anaesthetic to use, which to avoid (adrenaline).
* Know oculocardiac reflex, treatment and mechanism.
* Different colour cylinders and what they contain.

**Examples of other questions:**

The written and osce included the following: Adrenalin-PEP-Eph table, Spinal in pregnant women (exact dose, CI etc), dysrrhythmias (risks for lethal and mx), Sux (phase II block, TOF, SE and CI), pictures of ALL the different airways, malignant hyperthermia, mallampati, sternomental and thyromental, vapours (10 x 1mark Q's), Sodalime, NPO periods, Draw normal annotated capnograph, abn capnograph interpretation and mx of the abnormality, Increase in PaCO2 - PETCO2, Prayer sign, Opioid S/E, picture of monitor to give dx and mx, advantages of circle system, dilution of adrenaline, myocardial oxygen supply-demand balance. That's what I can remember from our test. We had almost nothing on induction agents. Other things that I think is important is all the anaesthetic emergencies and things that are specific to anaesthesia eg. RSI, confirmation of correct 2 tube placement, ASA classification, unanticipated failed intub, reversal of muscle relaxants, LA toxicity, Bier's block, oxygen flux, hypoK, hyperK, Hb-dissoc curve, risks for aspiration and prophylaxis, differences in pregnancy and paeds, paeds tube and weight calculations, advantages of Jackson-Reese, delayed emergence, blood gas interpretation, mx of bronchospasm and laryngospasm. Drugs and dosages are important, but don't spend all your time on it . The following questions were a few we got from our counter group (from a previous test): Q1 - draw anaesthetic circuit, how do you know sodalime is depleted, advantages of closed system. Q2 - define MAC, table of N2O, Iso, Sevo, Des for MAC, BGPC, Vapour pressure (sevo), colour, harmful compounds with sodalime. Q3 - Ruptured ectopic with BP 60/30. How would anaesthetize her? PEP/adr/eph mech of action, dilute adrenaline. Q4 - define hypoxia and hypoxaemia, types of hypoxia, C/S - how would you anaesth and why? If you do spinal, which drug and dosage. PEP - how does it work and dosage. Q5 - name 5 reasons why a pt won't wake up and how would you dx/confirm it. Q6 - oxygen flux - what does all stand for and what does it mean? Q7 - CPR MCQ's x 5 (don't have the Q's)

1. **Down syndrome and paediatrics**
2. **Phase Blocks?**
3. **Distinguish between Myaesthenia Gravis and Lambert Eaton syndrome**