# ANZCA Introductory Training

## Anaesthetic Machine Check

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Anaesthetic Machine Check

[1. Basics]
• Anaesthetic machine is connected to electric supply and working
• Check service labels
• Check bulk flow panel on wall

Take manifold apart
Leak test on vent common outlet inner port
Take O2 sensor off
Check monitor analyser system - no water droplets in container
Check alarm settings on monitor (low O2 18%)
• Check frequency of bp measurement

Put filter and sampling line on common gas outlet - watch analysis of O2 and air

• Remove manifold cover
  • Check RA 21%
  • Check 100% O2
  • Check 50% nitrous with O2 mix 2 litres
  • Check anti hypoxia 1:3 ratio and O2 not dropping below 21%
  • Stop O2 check that nitrous stops flowing prior to loss of O2
  • Disconnect piped gas
Check low O2 alarm audible and visual alarm on vent

[3. Gases]
• Reserve Cylinder:
  • Pull out O2 leads
  • Empty machine
  • Switch to O2 cylinder
  \(\Rightarrow\) pressure gauges should be 400-500kPa
Check nitrous cylinder

• Reconnect machine then Tug test

[4. Gas flows]
  • Bulk gas pressure for air and O2 (4bar)
  • Check O2 and air appropriate analyser range
  • Bobbin freely and through range
  • Check emergency bypass O2 button with test lung. Should fill <5seconds 2 litre bag

[5. Vaporiser]
• Check adequately filled
• Check filling port closed
• Turn on each vaporiser, check O2 does not fall <1 litre
• Each vaporiser correctly seated on back bar

[6. Breathing circuit]
• Inspect configuration of circuit with test lung on
By Adam Hollingworth

- Vaporiser leak test open common outlet port
  - Use squeeze suction device to check no leak
  - Turn vaporizer on, re-remove air from system & check leak
- Remove scavenger from system
- APL valve closed up, O2 flush until circuit pressures to 30
- Then put scavenger back in and check for leak
- Open APL: check bags not emptying (overactive scavenger)
- Squeeze bag then test lung - check unidirectional valves
- Squeeze bag and test lungs till empty

[7. Vent settings]

**turn machine off, with empty belows. ****

- Fill belows with O2 then check for leak

***turn machine on again with vent on***

- Run at 6 litres: Wait for atmospheric pressure reading (<5 pmax) with O2 running at 6 litres
  - turn vent off and on again
- Standard settings: VT 500, RR 12 check getting to target
- High pressure test
- Low pressure test: remove test lung. Await alarm low paw
- Reset vent panel

- Check ambi bag, aux O2 port

[8. Gas Scavenging System:]
- Check CO2 scrubber working correctly
  - Good colour, gauge in green
  - Port closed on side

[9. Ancillary Equip:]
- Laryngoscope, bougie, forceps, OPA
- Suction working high and variable pressure with occluded (thumb over end suction holds itself above ground)
- Check Yanker tube suction
- Bed/operating trolley working
2.1.1 Airway Management

1.1 Basic Structure of Upper Airway incl Larynx
Nerves of the Larynx

- Superior laryngeal nerve
  - Divides into:
    - Internal branch –
      - Sensory to:
        - Ipsilateral larynx from sup boundary to true cords
        - Pyriform sinus
        - Epiplottis
    - External branch –
      - Motor
      - Cricothyroid muscle
      - Sensory:
        - Ant infraglottic larynx cricothyroid membrane
          - Unilateral paralysis ⇒ failure of ipsilateral cord closure event with intact RLNs

- Recurrent (inf) laryngeal nerve:
  - Motor:
    - All intrinsic mm of larynx on same side except cricothyroid mm (ext laryngeal from Vagus)
  - Sensory:
    - Ipsilateral mucosa below true cords
      - L RLN longer course, turning around aortic arch; R RLN turns around subclavian artery
      - Paralysis of RLN ⇒ paramedian vocal cord position due to adduction action of SLN (cricothyroid)
1.2 Airway Assessment

**Intro**

- Impt - 30% anaesthetic deaths caused by failure of airway management
- Most catastrophe due to unexpected difficult airway
- Prediction of difficult BMV or LMA as important as ETT placement
- Intubation =
  - difficult in 1:50
  - Impossible 1:2000 - ↑'ed to 1:200 for emergencies
- BMV =
  - Difficult 1:20
  - Impossible 1:1500
- Both can't CICO: 1:10000
- Rescue techniques fail 1:20
- Rfs for hypoxemia are important:
  - Pregnancy
  - Obesity
  - Children

**History**

- Congenital airway difficulties:
  - Pierre Robin = micrognathia, small tongue, cleft palate
  - Klippel Feil = congen fusion of >2 Cx vertebrae
    - head displaced ant and inferiorly
  - Down Syndrome =
    - Very large tongue
    - Laryngomalacia = inward collapsing of tissues at laryngeal inlet
    - Tracheomalacia
    - Tracheal bronchus = bronchi come from trachea level
    - Bronchomalacia = collapsing of airways
- Inflammatory:
  - RA -
    - atlanto-axial subluxation (25%) due to deg of transverse ligament
      - types: ➔ with risk spinal cord damage
        - Anterior - (80%)
          - C1 moves forward on C2 causing risk spinal cord compression by peg
          - Lat C spine - atlas to peg distance: >44yrs old = >4mm, <44yrs old = >3mm
        - Posterior (5%)
          - Lat extension views
          - Peg is destroyed
        - Vertical (10-20%)
          - Destruction of lat mass of C1 ➔ peg through foramen magnum & compression cervico-medullary junction
          - Lat or rotatory = degen changes in C1/C2 facet joints ➔ spinal nerve & vertebral artery compression
  - Subaxial subluxation - uncommon. Occurs below C2
  - Cricothytenoid joint involvement:
    - Dyspnoea, stridor, hoarseness, severe ➔ upper airway obstruction
    - Laryngeal amyloidosis & rheumatoid nodules ➔ obstruction of larynx
- TMJ joint involvement ⇒ difficult mouth opening
  • Stills disease = (juvenile or adult onset)
    - polyarthritis with sore throat & high spiking fever & salmon pink rash
  • Anky Spond
  • Scleroderma - tight skin & mouth
• Infectious:
  - Epiglottitis
  - Submandibular abscesses or Ludwig’s Angina
    ⇒ cellulitis of submandibular tissues
  • Retropharyngeal abscesses
• Endocrine
  - Acromegaly - hypertrophy of upper airway soft tissues
  - DM - generalised joint and cartilage damage
  - obesity
• Pregnancy
  - Upper airway oedema
  - Incr aspiration risk
• Trauma
  - Foreign bodies
  - Facial or neck trauma
• Iatrogenic Problems:
  - TMJ surgery
  - Cervical fusion
  - Oral/pharyngeal radiotherapy
  - Laryngeal.trachel surgery
• Reported previous anaesthetic problems - check notes, med alerts, databases

**Examination**
• Unusual anatomy:
  - Small mouth
  - Receding chin
  - High arched palate
  - Large tongue
  - Bull neck
  - Morbid obesity
  - Large breasts
• Acquired problems:
  - Head/neck burns
  - Tumours
  - Abscesses
  - Radiotherapy
  - Scars
• Mechanical limitation
  - ↓mouth opening
  - ↓Ant TMJ movement (protrusion)
  - Poor Cx movement
  - Poor dentition
  - External equipment ie halo traction, C collar, dental wiring
  - Unpatent nasal passages - for nasal intubation
Radiology
- Recent CT/MRI helpful
- Occipito-atlanto-axial disease is more predictive of difficult laryngoscopy than disease below C2
- Plain XRs not that useful:
  - Flex/ext views in RA may be helpful but poor correlation with risk

Predictive Tests for Intubation
- For intubation need:
  - Mouth opening
  - Ext upper Cx spine
  - Ability to create submandibular space
- Tests have statistical problems:
  - Low specificity & PPV ie large no of false +ves
  - Sensitivity ≈ 50%. Tests quoted high often in specific populations, not in routine practice
  - Combination of tests ⇒ ↑ specificity (↓’ed false positives) BUT ↓’es sensitivity (miss more truly difficult airways)

Laryngeal View Grades
- Restricted = need bougie
- Difficult = advanced techniques

Interincisor Gap
- Distance between incisors with max open mouth
- Affected by TMJ & upper Cx spine mobility
  - <3cm ≈ difficult intubation
  - <2.5cm ≈ LMA insertion difficult

Protrusion of Mandible
- Class A = lower incisors can protrude beyond upper
- Class B = lower reach margin of uppers
- Class C = lowers cannot reach uppers
  - class B & C ≈ difficulty

Mallampati Test
- Patient sitting upright, from opposite patient, open mouth maximally and protrude tongue without phonating
- gradings:
  - Class 1 = faucial pillars, soft palate & uvula visible
  - Class 2 = uvula tip masked by bass of tongue
  - Class 3 = soft palate only
  - Class 4 = soft palate not visible
- Class 3 & 4 ≈ difficult intubation BUT:
  - Interobsever variation
  - Sensitivity 50%
  - Low specificity and positive predictive value - 90% false positive rate

Extension of Upper Cx spine
- <90 ≈ difficulty
• Methods:
  • 1:
    - Fully flex head on neck
    - Immobilisin lower Cx spine with one hand, then fully extend head
    - A pointer on the forehead allows angle to be estimated
  • 2:
    - One finger on chin and one on occipital protuberance & extend head max
    \[\text{norm} = \text{chin finger higher}; \text{mod limitation} = \text{level fingers}\]

**Thromental distance (Patil test)**
• Neck fully extended, mouth closed: distance tip thyroid cartilage to tip of mandible
• Score:
  • Normal >7cm
  • <6cm ≈ 75% of diff laryngoscopies
• Patil & mallampati tests combined (<7cm & gd 3-4) = specificity 97%, sensitivity 81%

**Sternomental Distance (Savva Test)**
• Neck fully extended, mouth closed: Upper border of manubrium to tip mandible
• <12.5cm ≈ difficulty (PPV 82%)

**Wilson Score**
• 5 factors:
  • Weight
  • Upper Cx mobility
  • Jaw movement
  • Receding mandible
  • Buck teeth
• Each gets subjective score 0-2
• Score 2 or >2 ≈ 75% difficult intubations 12% false positives

**Predictive Tests for Difficult BMV**
• Age >55
• BMI > 26
• Snoring Hx
• Beards
• No teeth
\[\text{if have 2 of above} >70\% \text{ sensitivity & specificity}\]
• Facial abnormality
• OSA
• Receding or marked prognathism
  \[\text{marked jaw protrusion relative to skull}\]

**Predictors of Problems with Back Up Techniques**

**LMA**
• Inability to open mouth >2.5cm
  \[\text{impossible if} <2cm\]
• Intraoral/pharyngeal masses

**Direct Tracheal Access**
• If contemplating need for tracheal access:
  • Position of larynx & trachea
  • Accessibility of cricothyroid membrane & trachea
• Risk factors:
  • Obesity
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- Goitre
- Other ant neck masses
- Deviated trachea
- Fixed neck flexion
- Prev radiotherapy
- Surg collar or ext fixator

1.3 Perioperative Fasting Requirements & Aspiration Risk

- Aspiration causes:
  - chemical pneumonitis
  - FB obstruction
  - Atelectasis
- 30-40mls of gastric contents ⇒ sig mortality and morbidity

**Gastric Physiology**

- Clear fluids - emptied from stomach in exponential manner half life around 10-20mins
  ➔ Thus complete clearance 2hrs

- Solids:
  - High fats/meats 8hrs+
  - Light meals eg toast approx 4hrs
  - Milk = solid as congeals with gastric juice.
    - Cows milk clears approx 5hrs
    - Human milk less fat & protein so clears quicker

**Elective Surgery Times**

- Adult:
  - Clear liquids 2hrs(<200mls)
  - Light meals 6hrs

**Risk Factors**

- Full stomach/delayed emptying
  - Causes delayed emptying:
    - Metabolic eg DM, renal failure, sepsis
    - Decr gastric motility eg head injury
    - Pyloric obstruction eg stenosis
  - Delayed emptying of fluids only in very advanced stages
- Known reflux - effects solids not liquids
- Raised intragastric pressure ie obstruction, pregnancy, laproscopic surgery
- Recent trauma
- Peri-op opioids - marked delays
- DM
- Topically anaesthetised airway
  ➔ anxiety has not been shown to effect gastric emptying

**Premeds**

- Premeds 1hr prior to surg have no effect on gastric volume of induction anaesthesia
- Oral midaz 30mins prior to surg no link to aspiration risk
**Gastric Acidity**
- Antacids to decr gastric pH in high risk eg pregnancy
  - sodium citrate commonly used
- H2 blocker/PPI:
  - Give evening before, and 2hrs prior
- Gastric motility agents: metoclopramide (better IV) can incr speed of emptying in healthy.
  - ?benefit in trauma patients

**Pregnancy**
- Elective C section:
  - Ranitidine 150mg evening before (7am on afternoon list) AND 2hr preop
- High risk pt in labour: 150mg 6 hourly
- Emergency case: 50mg IV ranitidine earliest opportunity
  - all should also have 30ml sodium citrate

**Management of Aspiration**

**Diagnosis**
- Clinical:
  - ↑RR, ↑HR, ↓lung compliance, ↓SpO2
  - Ausc: wheeze & creps
  - Tracheal aspirate may be acidic - negative finding does not exclude aspiration
- CXR: diffuse infiltrative pattern esp in R lower lobe
  - late sign

**Differential Diagnosis**
- Pulmonary oedema
- PE
- ARDS

**Management**
- 100% O2, minimise further aspiration risk
- Situational Rx:
  - awake or nearly awake: suction in recovery position
  - Unconscious & spont breathing:
    - Apply cricoid pressure (don’t if active vomit as risk oesophageal rupture)
    - Place L lat head down position
    - Intubate if tracheal suction & vent indicated
  - Unconscious & apnoeic: intubate immed & ventilate
- Minimise pure vent until airway secured and all aspirates suctioned
- NG tube
- CXR : look for oedema, collapse/consolidation
- Spo2 90-95% try CPAP & chest physio
- Spo2 <90% despite FiO2 1 ≈ food bolus obstructing bronchial tree & consider bronchoscopy
  - should be ICU ref

**1.4 Choosing an Airway Strategy**

- Procedure:
  - Elective
  - Emergency
- Patient:
  - Age
- Cooperation
- Surgery needed
- Position of patient
- Trauma
- Comorbidities
- Full airway Assessment
  - Predictive tests
  - Fasting

- Own skills
- Equipment & resources available
- Drugs available

**Rapid Sequence Induction**
- = rapid IV induction mm relaxation to aid tracheal intubation combined with cricoid pressure to ↓ risk of pulmonary aspiration
- Mask ventilation relatively contraindicated
- Consider other techniques if intubation predicted to be difficult ie AFOI

**Plan**
- 2 laryngoscopes
- Ventilator of anaesthetic machine incl suction
- tipping trolley bed
- Monitoring
- Positioning - tragus of ear above sternum
- Reliable cannulation
- Drugs:
  - Induction agents:
    - Thiopentone 2-5mg/kg
    - Propofol 1-3mg/kg
    - Etomidate 0.3mg/kg
  - Sux 1-1.5mg/kg
  - Emerg drugs
- Equipment for failed intubation (difficult 1:50, impossible 1:200 in emergency)
  - LMA different sizes - with gastric port if possible
  - BM
  - Emerg cricoid kit
  - Video laryngoscopes

**Procedure**
- Suction on and under pillow
- Preoxygenate until ETO₂ >90% or at least 4 vital capacity breaths
- (risks for quick desaturation:
  - Pregnant
  - Obese
  - Septic
  - Anaemic
  - Paediatric
  - Resp disease)
- Apply cricoid pressure 10N
- Administer induction agent, then rapid sux
- At loss of consciousness incr cricoid to 30N
Problems
- Haemodynamic instability:
  - Excessive induction agent ≈ circulatory collapse esp if hypovolaemic
  - Airway instrumentation ≈ tacy, HTN
    ↳ alfentanil 10-30mcg/kg 1min prior may be helpful
- Cricoid pressure:
  - Cartilage held between thumb & finger and pushed post by index finger
  - Poor tolerance eg children
  - Too much pressure makes intubation difficult
  - BURP +/- helpful
  - If vomit with cricoid before loss of consciousness should be released.
    ↳ once unconscious vomiting does not occur
  - Unknown force in paeds

1.5 Manual Inline Stabilisation & Implications for Intubation

Indications
- Proven or suspected neck #
- Major mechanism of injury
- Multi trauma unconscious patient

Implications
- Increased difficulty in obtaining intubation
- Increasing failed intubation rate
- Increasing need for adjuncts eg bougie
1.6 Can’t Intubate, Can’t Oxygenate

Unanticipated difficult tracheal intubation during routine induction of anaesthesia in an adult patient

Direct laryngoscopy → Any problems → Call for help

**Plan A:** Initial tracheal intubation plan

- Direct laryngoscopy - check:
  - Neck flexion and head extension
  - Laryngoscope technique and vector
  - External laryngeal manipulation - by laryngoscopist
  - Vocal cords open and immobile

- If poor view: Introducer (bougie) - seek clicks or hold-up and/or Alternative laryngoscope

**Plan B:** Secondary tracheal intubation plan

- ILMA™ or LMA™
  - Not more than 2 insertions
  - Oxygenate and ventilate

- Oxygenation (e.g. \(\text{SpO}_2 < 90\% \text{ with } \text{FiO}_2 1.0\))

**Plan C:** Maintenance of oxygenation, ventilation, postponement of surgery and awakening

- Revert to face mask
  - Oxygenate and ventilate
  - Reverse non-depolarising relaxant
  - 1 or 2 person mask technique (with oral ± nasal airway)

**Plan D:** Rescue techniques for “can’t intubate, can’t ventilate” situation

- Confirm: ventilation, oxygenation, anaesthesia, CVS stability and muscle relaxation - then fiberoptic tracheal intubation through ILMA™ or LMA™ - 1 attempt
  - If LMA™, consider long flexometallic, nasal RAE or microlaryngeal tube

- Verify intubation and proceed with surgery

Not more than 4 attempts, maintaining:
- (1) oxygenation with face mask and (2) anaesthesia

Verify tracheal intubation
- (1) Visual, if possible
- (2) Capnograph
- (3) Oesophageal detector
  - “If in doubt, take it out”


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Unanticipated difficult tracheal intubation - during rapid sequence induction of anaesthesia in non-obstetric adult patient

Plan A: Initial tracheal intubation plan

Pre-oxygenate
- Cricoid force: 10N awake → 30N anaesthetised
- Direct laryngoscopy - check:
  - Neck flexion and head extension
  - Laryngoscopy technique and vector
  - By laryngoscopist
  - Vocal cords open and immobile

If poor view:
- Reduce cricoid force
- Introducer (bougie) - seek clicks or hold-up and/or Alternative laryngoscope

Plan B not appropriate for this scenario

Failed intubation and difficult ventilation (other than laryngospasm)

Failed intubation, increasing hypoxaemia and difficult ventilation in the paralysed anaesthetised patient: Rescue techniques for the "can't intubate, can't ventilate" situation

Plan C: Maintenance of oxygenation, ventilation, postponement of surgery and awakening

Use face mask, oxygenate and ventilate (1 or 2 person mask technique (with oral ± nasal airway)) Consider reducing cricoid force if ventilation difficult

Plan D: Rescue techniques for "can't intubate, can't ventilate" situation

Difficult Airway Society guidelines Flow-chart 2004 (use with DAS guidelines paper)

Notes:
1. These techniques can have serious complications - use only in life-threatening situations
2. Convert to definitive airway as soon as possible
3. Postoperative management - see other difficult airway guidelines and flow-charts
4. 4mm cannula with low-pressure ventilation may be successful in patient breathing spontaneously

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Intro Training - 17
Plan A: Initial tracheal intubation plan
- Direct laryngoscopy
  - succeed: Tracheal intubation
  - failed intubation

Plan B: Secondary tracheal intubation plan
- ILMA\textsuperscript{TM} or LMA\textsuperscript{TM}
  - succeed
  - failed oxygenation: Revert to face mask, Oxygenate & ventilate
  - succeed
  - failed intubation: Confirm - then fibroptic tracheal intubation through ILMA\textsuperscript{TM} or LMA\textsuperscript{TM}

Plan C: Maintenance of oxygenation, ventilation, postponement of surgery and awakening
- Revert to face mask, Oxygenate & ventilate
  - succeed: Postpone surgery, Awaken patient
  - failed oxygenation

Plan D: Rescue techniques for "can't intubate, can't ventilate" situation
- LMA\textsuperscript{TM}
  - improved oxygenation
    - fail: Cannula cricothyroidotomy
    - succeed: Surgical cricothyroidotomy
1.8 Common complications of Intubation

- Airway related complications in 4%:
  - Aspiration
  - Oesophageal intubation
  - Dental injury
  - Pneumothorax
  - Laryngospasm
  - Perf trachea or oesophagus
  - # or dislocation Cx spine/TMJ/arytenoid cartilages
  - Vocal cord damage

1.9 Preoxygenation & Physiology

- Breathing 100% O2 from close fitting mask for 3-5mins (or 4 vital capacity breaths)
- Aim is to denitrogenate lungs ➞ oxygenation of FRC >1800mls O2
- ↑ time to desaturation of 7-8mins
- Best way to measure effectiveness of preoxygenation is measure ET O2 fraction (FEO2)
  - FEO2 ≈ FAO2 (alveolar O2 fraction)
  - Use alveolar gas equation to understand % of O2 in lung:
    - 149 - 40/0.8 = 100mmHg
    - 100mMg as percentage of atmosphere (760mmHg) = 100/760 x 100 = 13%
- Typical FRC volume = 2.2 litres which in RA contains 13% O2 = 270mls O2
- In norm adult with complete preoxygenation (FAO2 >0.9) lungs should contain around 2000ml O2
- Total body oxygen consumption ≈ 250mls/min
  - Apnoea with norm store takes ~1min (270/250)
- If FRC preoxygenated with FiO2 1:
  - 760 - 47 - (40/0.8) = 663
  - 663/760 x 100 = 0.87
  - 2200*0.87 = 1914mls
  - 1914/250 = 7.65mins

Total Ventilation

- Vt = 500ml & RR 15/min:
- Total ventilation: = Vt x RR
- 500 x 15 = 7500ml/min
- Volume of air entering is slightly greater as more O2 is taken in than Co2 is given out

Alveolar ventilation:

- Vt – dead space x RR
- Amount getting to respiratory zone
- Anatomic dead space = 150mls ➞ alveolar vent = 500 – 150 x 15 = 5250ml/min

Partial Pressure of Gas

- Partial pressure of gas = concentration x total pressure
  - Eg dry air had 20.93% O2
    - @ sea level pressure = 760mmHg ➞ Po2 @ sea level = 20.93/100 x 760 = 159mmHg
- When air inhaled it is warmed & moistened
  - Water vapour pressure = 47mmHg ➞ total dry gas pressure = 760 – 47 = 713
- P1O2 inspired air = 20.93/100 x 713 = 149mmHg
Alveolar Gas Equation
Allows relationship between fall in PO2 & rise in PCO2 which occurs in hypovent can be calculated

\[ P_{A\text{O}2} = \frac{P_{I\text{O}2} - P_{ACO2}}{R} + F \]

Functional Residual Capacity
- FRC = major oxygen store within body
- FRC = balance between tendency of chest wall to spring outwards and tendency of lung to collapse
- Volume changes by many factors
- Decreasing factors:
  - Age
  - Posture - supine
  - Anaesthesia - mm relaxants - diaphragm tone will ↓ pull away from lungs
  - Pregnancy - ↑ abdo pressure
  - Surgery - laprasaic
  - Pulmon fibrosis
  - Pulmon oedema
  - Obesity
  - Abdo swelling
- Increasing factors:
  - ↑ing height
  - Erect position
  - Emphysema - less elastic recoil of lungs
  - Asthma - air trapping

1.10 Ventilatory Strategies in Elective and Emergency Patients

IPPV Indications
- Indications:
  - Where neuromuscular blockade is required
  - Abdo or thoracic operations
  - Close control of arterial CO2 is required eg Neuro
  - Resp disease
  - Gross obesity
**Ventilators**

- Reservoir bag:
  - Hand squeeze $\Rightarrow$ positive pressure in circuit $\Rightarrow$ gas forced into lung under positive pressure
- Bag squeezers
  - $\leftarrow$ flow generator ventilator
  - Bellows are squeezed - usually by intermittently pressurising bellows in fixed jar
  - Gas in circuit are kept separate from compressing air
  - Aka bag in bottle type
  - Deliver constant flow but can create very high airway pressures
- Other flow generators:
  - Fluid logic to divide pressurised gas into smaller volumes
  - Volumes to patient or drive anaesthetic gas from reservoir to patient
  - Often used in paeds or transport vents
- Jet ventilation
  - Used during:
    - Rigid bronchoscopy
    - Upper airway surgery
    - Emerg cricothyrotomy
  - Works on Bernoulli principle: high pressure o2 passed out of narrow tube $\Rightarrow$ entrainment of air at an area of low pressure around opening
  - Jet of o2 applied intermittently
  - Risk of barotrauma is very high
- Minute volume dividers
  - $\leftarrow$ constant pressure ventilator
  - Eg Manley series
  - Minute volume of gas is taken from machine and passed under low pressure to ventilator
  - Gas flow divided up into tidal volumes by bellows/lever mechanism and pressuried by small weight
  - Low pressure system causes problems in people with low lung compliance or high airway resistance ie inadequate air flows

**Delivering IPPV**

- Patients physiological resp drive can be overcome by:
  - Mm relaxants
  - Sedation/anaesthesia
  - Opiates
  - hyperventilation
- Tidal Volume:
  - Without pre-existing lung disease & children: 12ml/kg, 12/min
  - With chronic resp disease: 10ml/kg, 10/min
  - ARDS: 6-8ml/kg & high PEEP upto 15
- I:E ratio:
  - Start with 1:2
  - ↑ inspiration - good with large shunts
  - ↑ expiration - bronchospasm obstruction

**Hyperventilation**

$\text{H}_2\text{O} + \text{CO}_2 \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{H}^+ + \text{HCO}_3^-$

- Hyperventilation drives equation to L causing
Respiratory alkalosis via decreased available H+ ions

Consequences of hyperventilation:
- ↑ risk of ventricular dysrhythmias
- Hypokalaemia
- ↓ ionic Ca → neuromuscular irritability
- Cerebral VC →
  - ↓ ICP (limited to approx 24hrs)
  - ↑ risk of regional ischaemia

**Physiological Consequences of IPPV & PEEP**

**CVS**
- ↑ intrathoracic pressure:
  - ↓ filling R heart → ↓ CO → ↓ bp
- ↑ Pulmonary vascular resistance → ↓ R ventricular outflow → RV distension → bulging of septum → ↓ LV compliance

**Renal**
- ↓ renal perfusion 2nd to hypoperfusion from ↓ CO
- Humeral effects:
  - ↓ ANP secretion
  - Stim renin-angiotensin axis
  - ↑ vasopressin production
  "all" → ↓ urine output & sodium & water retention

**Resp**
- IPPV much less efficient in maintaining VQ ratio:
  - Atelectasis
  - ↓ FRC → shunting
  - ↑ alveolar & anatomical dead space
- Risk of barotrauma
- Long term complications (ie ICU):
  - Bronchopulmonary dysplasia
  - Oxygen induced lung injury
  - Tracheal stenosis
  - Nosocomial lung infection

**Other**
- Consequences of ↓ VR to heart:
  - ↑ ICP
  - Liver dysfunction from hepatic congestion
1.12 Peri-operative Upper Airway Obstruction

- Approach changes based on:
  - Urgency
  - Level of obstruction
  - General condition of patient
- Any airway obstruction always likely to get worse during anaesthesia or airway manipulation:
  - Loss of airway tone
  - Reflex airway responses
  - Trauma
  - Bleeding
- Life threatening complications:
  - Complete obstruction on induction
  - Intra-airway haemorrhage
  - Swelling
  → always have a back up plan

Assessment

- Preop tests can be useful if have time:
  - Nasoendoscopy
  - CT/MRI
  - PFTs with flow volume loops
  - ECHO - if pulmon vessel suspected
- To consider:
  - What level is it?
    - Oral
    - Supraglottic
    - Laryngeal ≈ insp stridor & voice change
    - Mid tracheal
    - Lower tracheal ≈ exp stridor/wheeze
    → several levels may be effected by 1 pathology
  - Severity:
    - Resp distress & acc muscle use
    - Stridor
    - Hypoxaemia
    - Silent chest
    - Dysphagia
    - Nocturnal panic
  - Lesion - mobile or friable
  - Neck - how easy is it to access trachea as back up plan
  - Effect of positioning
- Management plan for extubation
  → may need to be delayed
- Prolonged instrumentation may cause airway oedema
- Heliox:
  - Premixed helium & O2 21-30%
  - Decr viscocity thus allows improved flow through narrowed tube
  - Note ↓FiO2 BUT can use a Y connector to incr FiO2

Intro Training - 23
Oral, Supraglottic & Laryngeal Lesions

- Eg trauma, burns, tumour, infection
- Semi-elective cases - careful nasoendoscopy may help in predicting difficult cases
- Cricothyroidotomy will only work if lesion is not obscuring access

Options:
  - AFOI
  - Inhalational induction ⇒ direct or fibre optic laryngoscopy
  - Elective awake (with LA):
    - Cricothyroidotomy
    - Tracheostomy
    - Trans-tracheal ventilation catheter - back up oxygenation plan

- LMA may be helpful if unexpected obstruction
- if concerns:
  - IV induction with no back up plan should never be done

Mid Tracheal

- Eg tumour or retrosternal goitre
  - may expand suddenly with haemorrhage
- Site of lesion may prevent emerg cric or trachy if needed
- Inhalational induction may be very slow if severe narrowing
- AFOI:
  - Coughing & distress may ⇒ ↑ obstruction & cyclic of decline
  - Tube through narrowing may prevent spont vent (= cork in bottle)
  - Need to pass through narrowing:
    - ET tube
    - Endobronchial tube
    - Hollow intubation bougie or Cook airway exchange catheter
  - Ideal of tube and cuff can sit below obstruction but above corina
  - RSI with rigid bronchoscope
  - May need TIVA

Lower Tracheal/Bronchial Lesions

- Eg tumour, trauma, mediastinal masses
- Best managed by in tertiary centre
- Cardiopulmonary bypass sometimes necessary eg pulmo artery compression
- RSI, rigid bronchoscope may be life saving

1.15 Oesophageal intubation

Direct confirmatory techniques:
  - Fibreoptic bronchoscopy with visualisation of tracheal rings through ETT
  - Visualisation of ETT passing through vocal cords
    - commonly mistaken

Indirect markers:
  - Auscultation of chest & epigastrium - fail to identify 1:40 oesophageal intubations
  - Condensation on tube: BUT 42:60 oesophageal intubations fogged tube
  - Spo2 - but can have delay of hypoxia up to 8mins with OI
  - CXR - only really useful for identifying bronchial intubation
  - Capnography -
    - Sensitivity 93%, specificity 97% = failed recognition OI in 3% cases
    - False readings possible:
• Tube in oesophagus but CO shows in trachea: expired alveolar gas introduced into stomach during
  • BMV
  • Ingestion carbonated beverages
  • Antacids
• Tube in trachea but CO in oesophagus:
  • Low cardiac output (Cardiac arrest, Severe hypotension )
  • Severe pulmon disease
  • PE
• Oesophageal Detector Devices
  - Rely on differences in rigidity of tracheal & oesophageal walls
  - Sensitivity & specificity up to 100% although false +ves:
    ↪ controversial paramedic testing 50% sensitivity
  • Prev air insufflation of GI tract
  • COPD
  • Copious secretions
  • COPD
    • Much more useful in low cardiac output states ie arrest
1.17 - 1.20 Extubation

- Resp complications x3 more likely than intubation (4.6 vs 12.6%)
- Main questions:
  - Prev difficulties with controlling the airway
  - What is risk of pulmon aspiration
- Deep vs awake:
  - General rule: extubate when awake
  - Deep ≈ ↓CVS stim, ↓coughing on tube BUT ↑↑complications regardless of operation

---

**Emergence and Extubation: A systematic approach**

Can this patient be extubated while deeply anesthetized?

- **YES**
  - No residual neuromuscular block
  - Easy mask ventilation
  - Easily intubated
  - Not at increased risk for regurgitation/aspiration
  - Normothermic

- **NO**
  - Difficult mask ventilation
  - Difficult intubation
  - Residual neuromuscular block present
  - Full stomach
    - Pregnant
    - Obese
    - Recent ingestion of food
    - Diabetic
    - Has ascites

Can this patient be extubated immediately following surgery and emergence from general anesthesia?

- **YES**
  - Awake
  - Following commands
  - Breathing spontaneously
    - well oxygenated
    - not excessively hypercarbic (PaCO₂ ≤ 50 mmHg)
  - Fully recovered from neuromuscular blockers
    - sustained head lift
    - strong hand grip
    - strong tongue protrusion

- **NO**
  - Hypoxic (O₂ saturation < 90 mmHg)
  - Excessively hypercarbic (PaCO₂ > 50 mmHg)
  - Hypothermic (<34°C)
  - Residual neuromuscular block present
  - Patient may be unable to protect his or her own airway
    * Airway swelling
      - long surgery in Trendelenburg position
      - airway surgery
      - patient received excessive intravenous fluid volume
    * Impairment of cough/gag reflex
      - brainstem surgery
      - intraop cerebral ischemic events
    * Vocal cord paralysis
      - Inadequate strength
  - Excessively long surgical procedure
  - Airway may be difficult to re-establish
  - Unexplained hemodynamic instability

The patient requires continued intubation and mechanical ventilation
Positioning

- Traditional extubate in L lat, head down position:
  - Tongue moved away from post pharyngeal wall
  - Protects airway from aspiration
  - Laryngoscopy & reintubation favourable if skilled in this position
- Supine sitting up position - controversial:
  - No evidence to show less complications that lat position in standard cases
  - Physiological benefits:
    - Facilitates spont rest & diaphragmatic movement
    - Aids cough
    - ↑FRC
    - Encourages from lymph drainage
    - ↓airway oedema
  - May be easier to reintubate esp if expected diff intubation:
    - Obese
    - Chronic resp disease
- Prone:
  - May be necessary after spinal surg
- Children usually extubated in recovery position

Timing Extubation

- ↑threshold to fire of laryngeal adductor neurons during inspiration
  \[ \rightarrow \quad \text{extubate at end inspiration when glottis fully open} \]

Method

- Suction post pharynx
- Bite block
- 100% O2
- High flows to washout inhalational agents
- Positive pressure breath at extubation to prevent atelectasis

Problems with Extubation

Mechanical

- Failure to deflate cuff
- Trauma to larynx
- Cuff herniation
- Adhesion to tracheal wall
- Surg fixation of tube to adjacent structures

CVS Response

- Extubation ≈ 10-30% ↑ bp & HR lasting 5-15mins
- If coronary artery disease ≈ ↓40-50% EF
- Can use drugs to manage:
  - Esmolol 1.5mg/kg 2-5min before extubation
  - GTN
  - Mg
  - Remi/alfentanil infusion
  - Lignocaine 1mg/kg over 2mins
- Can convert to LMA prior to extubation

Resp Complications

- Cough & sore throat - 38-96%
• Fill cuff with fluid rather than air - less change in pressure via temp & N2O diffusion
• Lignocaine 2% in cuff - 4-6hrs 45-65% diffusion across cuff
• Special ETT which has port for topical LA

Postoperative hypoxaemia:
• Causes:
  - ↓MV
  - Airway obstruction
  - ↑VQ mismatch
  - Diffusion hypoxia
  - Post hyperventilation hypoventilation
  - Shivering
  - Inhibition of hypoxic pulmon VC
  - Mucociliary dysfunction
  - ↓CO
• Prevention:
  - 100% preoxygenation prior to extubation
  - Continuous positive pressure vent
  - High inspired O2 during transfer to PACU

Risk of bronchospasm:
• Smokers
• COPD
• Children upper resp tract infections

Airway Obstruction
• Differential diagnosis of post extubation upper airway obstruction (UAO):
  • Laryngospasm -
    - most common 5% of intubated pts
    - More common kids with upper airway surgery
    - Caused by local irritation of blood/saliva
    - Likely in light planes anaesthesia - no airway reflex or poor cough to clear
    - Leave children in L lat position until they wake up
    - Mg 15mg/kg/20mins or lignocaine 1.5mg/kg over 2mins can help
  • Laryngeal oedema
    - Impet cause in neonates & infants
    = insp stridor within 6hrs of extubation
    - Supraglottic oedema displace epilgotis post blocking glottis on inspiration
    - Retroarytenoidal oedema below cords limits abduction of vocal cords on inspiration
    - Subglottic oedema of 1mm in neonate ≈ ↓laryngeal cross section by 35%
  • Rfs:
    • Tight tube
    • Trauma at intubation
    • Intubation >1hr
    • Cough on tube
    • Change head/neck position during surg
  • Rx:
    • Humidified air
    • Neb adrenaline 1-5mls 1:1000
    • Dex 0.25mg/kg then 0.1mg/kg 6hrly for 24hrs
    • Heliox 60:40 or 80:20 as temporising measure
    • Reintubation with smaller tube if necessary
• Haemorrhage
- Trauma:
  - arytenoid cartilage dislocation - voice change or painful swallowing
- Vocal cord paralysis:
  - Rare
  - Trauma to vagus nerve
  - Unilat paralysis ≈ hoarseness - may recover over weeks depending on aetiology
  - Bilat paralysis ≈ UAO ⇒ reintubation
- Vocal cord dysfunction:
  - Uncommon
  - Young females, recent URTI, emotional stress
  - Stridor or wheeze resistant to treatment
  - Paradoxical vocal cord adduction during inspiration

**Table 1** Structured approach to the management of laryngospasm\(^7\) (the main aim is to rapidly oxygenate the patient)

<table>
<thead>
<tr>
<th>Think of</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway irritation/obstruction</td>
<td>100% oxygen</td>
</tr>
<tr>
<td>Blood/secretions</td>
<td>Visualize and clear pharynx/airway</td>
</tr>
<tr>
<td>Light anaesthesia</td>
<td>Jaw thrust with bilateral digital pressure behind temperomandibular joint, oral/nasal airway</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>Mask CPAP/IPPV</td>
</tr>
<tr>
<td>Deepen anaesthesia with propofol (20% induction dose)</td>
<td></td>
</tr>
<tr>
<td>Succinylcholine 0.5 mg/kg to relieve laryngospasm (1.0–1.5 mg/kg i.v. or 4.0 mg/kg i.m. for intubation). Be aware of contraindications, for example, neuromuscular problems</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intubate and ventilate</td>
</tr>
</tbody>
</table>

**Post Obstructive Pulmonary Oedema**

- Incidence 1:1000
- Most children or young fit adults
- Presentation:
  - Airway obstruction at emergence ⇒ rapid onset distress ⇒ haemoptysis ⇒ bilat CXR changes consistent with pulmonary oedema
  - All features usually resolve at 24hr with no sequelae
- Pathophys uncertain:
  - Negative intra-alveolar pressure
  - ↑ cardiac filling
  - Haemorrhage of pulmonary vessels
  - Hypoxaemia
  - Catecholamine release on alveolar capillaries ⇒ ↑ permeability
- Rx with +ve airways pressure & oxygenation
- Differential = neurogenic pulmonary oedema:
  - Similar but more severe onset
  - From severe CNS insult

**Tracheomalacia**

- Failed extubation with stridor or wheezing may be first signs of tracheomalacia
- Usually erosion of tracheal rings by:
  - Retrosternal thyroid or tumour
• Enlarged thymus
• Vascular malformations
• Prolonged intubation
• Trial deep extubation to avoid coughing
• Maintain CPAP to keep airway patent

**Pulmon Aspiration**
• ½ aspiration occur at extubation
• Swallowing reflex obtunded for approx 4hrs

**Recognising High Risk Patients**
• Severe heart/lung disease
• Airway pathology
• Obese
• OSA
• Severe GORD
• Multiple attempts at intubation
• Surg factors:
  • Recurrent laryngeal nerve damage (10% thyroids)
  • Haematoma
  • Oedema
  • Post fossa surg
  • Inter-maxillary fixation
  • Drainage neck/dental abscesses

**Strategies for Presumed Difficult Extubation**
• LMA:
  • Insert when deep
  • Reverse relaxation
  • LMA removed when spont breathing
• Extubation over flex bronchoscope:
  • Used if ?laryngeal paralysis, tracheomalacia, tube entrapment
  • ETT > LMA
  • Bronch passed and cords visualised +/- ETT re placed
• Tracheal Tube Exchange Catheter
  • Useful if expected difficult to reintubate
  • = long hollow catheters with connectors of manual/jet vent
  • Can be left in place for upto 72hrs post
  • Spont breathing, coughing, talking well tolerated

**Predicting Unsuccessful Extubation**
• Alert test = x4 more likely to succeed:
  • Open eyes
  • Follow with eyes
  • Grasp hand
  • Stick out tongue
• Cuff leak test:
  • Av diff between insp & exp volume after cuff down, 6 consecutive breaths is determined
  • <10% volume difference of delivered Vt ≈ upper airway oedema
2.1.2 General Anaesthesia & Sedation

1.5 Chemical Composition of Fluids and Effects in Volume replacement

<table>
<thead>
<tr>
<th></th>
<th>Normal Saline (0.9%)</th>
<th>Dextrose 4% /Saline (0.18%)</th>
<th>Plasmalyte 148 pH 7.4</th>
<th>Gelofusine</th>
<th>Pentastarch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na (mM/l)</td>
<td>150</td>
<td>30</td>
<td>140</td>
<td>154</td>
<td>154</td>
</tr>
<tr>
<td>K (mM /l)</td>
<td></td>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ca (mM /l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mg (mM /l)</td>
<td></td>
<td></td>
<td>1.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cl (mM /l)</td>
<td>150</td>
<td>30</td>
<td>98</td>
<td>120</td>
<td>154</td>
</tr>
<tr>
<td>Acetate (mM /l)</td>
<td></td>
<td></td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gluconate (mM/l)</td>
<td></td>
<td></td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose (mM /l)</td>
<td></td>
<td>222</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osmolality (mOsm/kg)</td>
<td>300</td>
<td>282</td>
<td>294</td>
<td>274</td>
<td>320</td>
</tr>
<tr>
<td>Energy (Kilojoules/l)</td>
<td>0</td>
<td>638</td>
<td>66</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Molecular Wt (Daltons)</td>
<td>30 K</td>
<td>250 K</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PH</td>
<td>5.0</td>
<td>4 -5</td>
<td>7.4</td>
<td>7.4</td>
<td>5</td>
</tr>
</tbody>
</table>

- NSL & pentastarch are significantly hyperchloreaemic
- Plasmalyte & ringers lactate = bicarbonate equivalent
- Blood cannot be mixed with
  - Ringers lactate - calcium in ringers
  - Dextrose
  - Haemaccel
- Plasmalyte best for replacement of small bowel or colonic loss or sequestration

1.6 IV Fluid Replacement

IntraOperative Fluid Loss

- <3 litres - Norm saline ok
- >3 litres problems with hyperchloreaemic load

Guidelines

- Monitor UO -
By Adam Hollingworth

- 1ml/kg AND absence of pulsus paradoxus
  \[\downarrow = \text{abnormally large (>10mmHg) } \downarrow \text{in SBP during inspiration}\]
- Response to volume trial with CVP monitoring: give 5ml/kg over 10min. Result:
  - \(<2\text{mmHg} = \text{hypovolaemia}\)
  - \(>5\text{mmHg} = \text{hypervolaemia}\)
  - 2-5mmHg = reassess or repeat
  \[\downarrow = 2-5 \text{ rule}\]
- Fasted patients (no surg losses)
  - Maintenance:
    - 1st hr 5ml/kg/hr
    - Thereafter: 2ml/kg/hr
- Surgical losses - best managed to clinical demand - blood but:
  - Routine - 4-6ml/kg/hr NSL or plasmalyte
  - Open cavity - 6-8ml/kg/hr
- Monitor hyperglycaemia & hyponatraemia

1.7 Anxiolytic or Sedative Premedications

Paediatrics
- Routine premed not required.
- Parents usually useful
- Indications:
  - Very upset
  - Prev unpleasant anaesthetic experience
  - Developmental delay
- Preschool child most at need due to separation anxiety from parents
- Complications of excessive anxiety include:
  - Sleep disturbance
  - Nightmares
  - Bed wetting
  - Eating disorders

Drugs
- Options:
  - Midaz 0.5mg/kg PO (max 15mg)
    \[\downarrow \text{(Intranasal 0.2mg/kg an option but burns)}\]
    - Onset in 15-30min
    - IV solution is bitter so dilute in pamol
  - Ketamine (0.5mg/kg PO)
    - Action within 15mins
    - May cause ↑salivation & emergence delirium
    - Option for IM (2-3mg/kg if required)
  - Clonidine (5mcg/kg PO)
    - Good induction conditions, good analgesic
    - BUT causes ↓bp & delayed recovery
- Don’t routinely need to coadminister anticholinergics
- Antisialogues (atropine 40mcg/kg PO but variable absorbed) reserved for:
  - Downs & CP
  - +/- ketamine

Intro Training - 32
Contraindications (relative) to Premedications

- New born <1yr
- Elderly
- Decr GCS
- Intracranial pathology
- Severe pulmonary disease
- hypovolaemia

1.8 Physiology of Pneumoperitoneum

- Insuflation of CO2 to av max 20mmHg
- Once intrabdominal pressure (IAP) exceeds physiological thresholds see organ effects

CVS Effects

- ↑SVR:
  - Mechanical compression of abdo aorta
  - ↑release vasopressin and activation of renin-angiotensin-aldosterone axis
- ↓CO:
  - Compression of IVC ⇒ ↓VR ⇒ ↓preload ⇒ ↓CO
    - especially if hypovolaemic
  - Cephalad displacement of diaphragm ⇒ ↑intrathoracic pressure ⇒
    - ↓VR (as above)
  - Compression pulmonary vasculature ⇒ ↑RV afterload

Resp Effects

- ↑IAP ⇒ ↓diaphragmatic excursion ⇒
  - ↑intrathoracic pressure
  - ↓compliance
  - ↓FRC
  - Atelectasis
  - Altered VQ relationships
  - Hypoxaemia
- Absorbed CO2 ⇒ ↑PCO2 which is worsened by VQ mismatching

GI Effects

- ↓kidney & liver blood flow - especially in mod/severe organ disease states
  - IAP 20mmHg = ↓GFR ≈ 25%
  - Mechanism thought to be ↓afferent flow (2nd to low CO) & ↓efferent flow (high venous pressure)
- IAP persistently >20 = ↓40% blood flow to mesenteric & GI mucosa ⇒ ↑acidosis

Neuro Effects

- ↑ICP:
  - ↑IAP ⇒ ↑intrathoracic pressure ⇒ ↓cerebral venous drainage
    - despite ↑ed mean cerebral arterial pressure
1.9 Physiological Effects of Positioning

Supine

• Resp:
  - ↓FRC - abdo contents encroaching on diaphragm
  - ↑VQ mismatch
  - ↓pulmonary compliance

• CVS:
  - ↑VR from LL vasculature
  - ± heart failure in borderline hearts
  - +/-compression of IVC in obese/pregnant ⇒ ↓↓ CO & ↓↓ bp

• GI:
  - ↑risk regurgitation

• Eye:
  - Risk of corneal drying in 10mins

• Nerve injury:
  - Supraorbital & facial nerve at risk from tube ties & FMs
  - Brachial plexus (esp C8, T1) - ↑ risk of injury when:
    - Arm abducted >90
    - Hand supinated
    - Head turned away
  - Ulnar nerve (>25% all nerve injuries) - in ulnar groove, medial epicondyle
    (↑ x3 males > female)

• MSK:
  - Loss lumbar lordosis ⇒ ↑ chance LBP
  - Pressure sores - heels, occiput, sacrum

Lateral

• VQ mismatch - dependant lung vs non dependant lung
• Greatest amount of ocular complications:
  - Mostly corneal abrasions - either eye

• Nerve damage:
  - Brachial plexus - need good lateral support
  - Saphenous nerve & common peroneal - need padding between legs

Lithotomy

• Very similar to Trendelenburg
• Hands and digits at the side of the patient - must be careful to avoid crush when replacing bottom of table
• Nerve damage - bilat flex of hip joints ≈
  - stretch sciatic & obturator nerves
  - Femoral nerve - direct compression under inguinal ligament

• Calf compression ⇒ VTE risk
• Compartment syndrome - multiple causes of ↓ perfusion pressure:
  - Weight of extremity against support ⇒ ↓ compartment capacity
  - Elevation above heart
    - stirups no better than combined calf support
  - ↓ length of op >5hrs main risk factor
Prone
- Must try and avoid pressure on abdo by good positioning
- Effective positioning can be positive physiologically (approx 70-80% see improvement initially)
  - ↑FRC
  - ↓VQ mismatch
- BUT position assoc with most MSK injuries:
  - Eye & nose
  - UL positions: small ant flex, abducted 90deg and ext rotation

Reverse Trendelenburg
- Beneficial physiological effects:
  - ↑head & neck drainage
  - ↓ICP
  - ↓regurgitation
- Risks:
  - ↓bp
  - ↑risk venous air embolism

Seated
- Venous pooling into LLs & refractory hypotension
- Venous air embolism - esp during craniotomy:
  - Subatmospheric venous pressure & non collapsable dural sinuses

Trendelenburg
- Classic 45deg head down tilt

CVS system
- In healthy little long lasting effect due to quick compensation VD to overcome ↑VR
- No RCT evidence to support trendelenburg position is of benefit in correcting acute ↓bp
- In elderly or comorbidities with impaired vasomotor control may see ↑bp:
  - Capillaries and most of venous blood above heart
  - Incr VR ⇒ ↑preload ⇒ ↑stroke volume ⇒ ↑CO ⇒ ↑bp
    - effect is marked in
  - deep inhalation: -ve pressure vent ⇒ ↑-ve intrathoracic pressure
  - high spinal/anaesthesia - sympathetic blocking ⇒ ↑VD ⇒ ↑VR
- Possibility of ↓bp is also argued:
  - ↓VR 2nd to intraabdo and pelvic organs compressing IVC
- Risk of adverse consequences in people with cormobidities:
  - Obese
  - Compromised RV EF ⇒ R heart failure
  - Pulmonary disorders
  - Head injuries
- Well leg compartment syndrome - combination of:
  - ↓arterial perfusion to raised LLs
  - Compression of leg vessels by SCDs
  - ↓femoral drainage by +/- pneumoperitoneum

Resp system:
- Rased diaphragm with gravity and weight of abdo cavity organs:
  - ↓VC, ↓FRC, ↑risk basal atelectasis
By Adam Hollingworth

\[ \downarrow 20 \text{deg head tilt} = \downarrow \text{VC by 15\%} \]

- Hypercarbia 2nd to shunt
- Incr VQ mismatch: ventilation maximal at bases, perfusion maximal at apex 2nd to gravity
- Endobronchial intubation - northward movement of pt with fixed position of ETT \( \Rightarrow \) relative southwards migration of tip of ETT further into lungs
- Upper airway oedema 2nd to orthostatic forces (prolonged positioning)

- **Airway/Positioning:**
  - Movement of pt with gravity causing soft tissue damage to lips on ETT and tie
  - Danger of patient falling from surg table

- **Digestive system:**
  - Pooling of secretions in dependant part ie nasopharynx \( \Rightarrow \) ↑ risk laryngospasm if not suctionning pre extubation
  - Increased risk of aspiration of gastric contents - if non secured airway

- **Neuro:**
  - Intra and extra cranial venous congestion \( \Rightarrow \) ↑ ICP
  - ↑ risk cerebral oedema
  - Eye - ↑ intraocular pressure

### 1.10 Post Operative Nausea & Vomiting

- 20-30% after GA with volatiles
- Up to 70% in high risk patients
- Morbidity:
  - Pt satisfaction, Delayed d/c, Unexpected admission
  - Wound dehiscence
  - Bleeding
  - Pulmon aspiration
  - Oesophageal rupture
  - Fluid & electrolyte disturbance

**Physiology Of PONV**

- induction of vomiting coordinated response from 2 diff areas:
  - chemoreceptor trigger zone (CTZ) – floor fourth ventricle
  - vomiting/emetic centre – medulla

- emetic centre receives
  - inputs from:
    - CTZ – via neurotransmitters:
      - ACh, 5HT, Histamine, DA
      - vestibular apparatus/cerebellum
      - higher centres – pain/smell/sight
      - organs eg heart, testes, GI tract
  - efferent to:
    - CN 5, 7, 9, 10, 12
    - Spinal nerves to GI tract, diaphragm, abdo muscles

- CTZ activated by:
  - CSF & blood borne emetics eg chem. toxins & drugs (poor bbb in area)
  - 5HT neurotransmitter from afferent nerves from stomach & small intestine receives input from vestibular apparatus
- higher centres – smells, emotions, pain
- ↑ICP
- endocrine disturbances
- radiation & chemotherapy

- CTZ cannot initiate vomiting alone
- CTZ very close physically to resp centre ∴ difficult to full abolish vom without effecting RR
- vomiting action via efferent nerves from emetic centre

### Risk Factors

[use a score predictor]

- **Patient:**
  - **Age:**
    - ↑children:adult
    - >50 = ↓ risk
    - Female = x3 risk
    - Previous PONV or motion sickness = x2-3 risk
    - Smoker = ↓ 0.6% risk
  - Surgical - high risk procedures = breast, strabismus repair, ENT, gynae, laprascopic, laparotomy, craniotomy (post fossa), genitourinary, shoulder surgery
  - Anaesthetic:
    - Premedication:
      - ↓risk = benzo & clonidine
      - ↑ risk = opiates
    - Type - GA x11 than regional
    - TIVA < volatile
    - Intraop drugs:
      - ↑risk =
        - opioids,
        - NO, volatiles,
        - induction agents of ketamine, etomidate, thio
        - Neostigmine - muscarininc effects on GI tract
      - ↓risk =
        - Propofol
        - Adequate IV hydration

### Management

- Multi-modal approach
- Prophylaxis vs treatment is controversial
- High risk patients where PONV >33% ondansetron prophylaxis cost effective
- Combo Rx eg dex & ondansetron
- Look for surgical cause
- Start using different classes:
  - Anticholinergic eg hyoscine or scopoderm
  - Antihistamine - cyclizine
  - Antidopaminergic - prochlorperazine, metoclopramide, droperidol or haloperidol
  - 5HT3 antagonist
  - Steroid - dex
Flow Chart for PONV

1.12 Failure to Wake from Anaesthetic

Causes
1. Pharmacological
2. Metabolic
3. Hypothermia
4. Resp failure
5. Neurological
6. Uncommon

- **Pharmacological:**
  - Benzo’s:
    - Elderly
    - In OD
    - In combo with opiates $\Rightarrow$ ↓resp drive $\Rightarrow$ ↑CO2 $\Rightarrow$ coma
    $\Rightarrow$ NB Midaz & alfentanil metabolised by same P450 iso-enzyme which can prolong action of both
  - Opioids -
    - major side effects from:
      - Resp depression -
        $\Rightarrow$ opioids direct ↓central chemoreceptors to CO2 $\Rightarrow$ ↑CO2
      - Direct sedation via opioid receptors
    - Note combination with other sedatives eg benzo’s
    - Active metabolites esp in renal failure
  - Neuromuscular blockade - mimicks unconsciousness:
- Drug interactions - (as table). Different mechanisms of action:
  - Interfering with Ca - causes Ach release
  - Electolyte disturbances ⇒ cell hyperpolarisation & prolonged block
- Hypothermia ⇒ ↓ metabolism of NMBs
- Acidosis ⇒ donation of proton to tertiary amine ⇒ ↑ affinity of NMB for receptors
- Deficiency of plasma cholinesterases ⇒ prolonged sux action

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<td>Drugs (ecothiopate, ketamine, oral contraceptive pill (OCP), lidocaine, neostigmine, ester local anaesthetics)</td>
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- IV anaesthetic agents:
  - Bolus propofol doses terminated by redistribution
  - TIVA - context sensitive half life = time for effect site to ↓ by 50%
    ⇒ this depends on length of infusion ie context
  - Need 80% reduction effect site conc before emergence
    ⇒ eg 80% reduction in effect site after 2 hrs = 36min (x2 dose = 105min; ½ dose = 10min)
  - Time to wake effected by:
    - Context sensitive half life
    - Amount of drug
    - Other drugs administered
    - Patient factors
- Volatiles:
  - Emergence depends on pulmonary elimination of the drug
  - MACawake = 30% of MAC:
    - Iso 0.39%
    - Des 2.17%
    - Sevo 0.61%
  - Pulmon elimination determined by:
    - Alveolar vent - low vent = longer emergence
    - Blood-gas partition coefficient
      ⇒ low coefficient = quicker
    - Dose (MAC-hours) - higher = longer emergence ie incr context sensitive half life

**Metabolic**
- Hypoglycaemia:
  - BSL <2.2
  - Effect categories:
    - Sympathetic response
- Neuroglycopaenia
  - Confusion/abnormal behaviour
  - Seizure
  - Coma

- Causes:
  - DM
  - Starvation
  - Alcohol consumption - impaired gluconeogenesis in starved pt with poor nutrition & energy reserves

- Hyperglycaemia:
  - Severe can prolong unconsciousness
  - BSL >14 ➞
    - osmotic diuresis & dehydration
    - Hyperosmolality & hyperviscosity ➞ ↑VTE risk
  - DM micro & macrovascular disease ➞ ↑chance intra-operative stroke

- Hyponatraemia:
  - Na level
    - <120 ➞ confusion & irritability
    - <110 ➞ seizure, coma, mortality

- Causes:
  - SIADH - 2nd to:
    - Brain trauma
    - SAH
    - Drugs eg opioids, haloperidol, vasopressin
  - Cerebral salt wasting syndrome -
    - in brain injured pt
    - ANP secretion 2nd to intracranial pathology ➞ salt loss at kidneys
  - TURP syndrome -
    - hypotonic glycine solution absorption
    - Pulmonary oedema
    - Cerebral oedema

- Hypernatraemia:
  - Uncommon postop

- Uraemia

**Hypothermia**
- <35 = confusion
- <30 = unconsciousness
- <24 = apnoea
- <18 = absent cerebral activity

- CVS effects:
  - ↓CO
  - ↑risk arrhythmia’s

**Respiratory Failure**
- Causes:
  - Neurological ie ↓central drive:
    - Drug overdose
    - Intracranial pathology
    - COPD
    - Sleep apnoea
  - Pulmonary disease:
- Dead space
- PE
- Atelectasis
- Obstruction
- Aspiration
- Consolidation
- ARDS
- TRALI
• Musculature:
  - Primary muscle problem
  - Metabolic imbalance
  - Obesity
  - Residual NMB
• Hypoxaemia:
  • ➔ cerebral hypoxia ➔ ↓cerebral function AND ↑ production of:
    - Lactic acid
    - Free radicals
    - Intracellular metabolites
  • ➔ cell death
• Hypercapnia:
  • Central chemoreceptors ➔ ↑resp stim to a point THEN ➔ ↓ing resp stim & ↓RR
  • Hypoventilation ➔ acidosis & ↑ing hypercapnic ➔ cerebral VD ➔ ↑ICP and 2nd brain injury

**Neurological Causes**
- Intraoperative cerebral insult causes are diverse:
  - Ischaemic brain cell death (most common)
    - Inadequate cerebral perfusion 2nd to low MAP
      ➔ (autoreg possible with MAP 60-160)
      ➔ watch for impaired auto reg in hypercapnic/hypoxic/↑metabolism
    - haemorrhage
    - Thrombosis
    - Infarct
  - cerebral hypoxaemia:
    - Prolonged seizure (masked by NMB)
    - Air embolism
  - Intracranial LA toxicity
• Must try and minimise 2nd brain injury by close bp monitoring & strict targets

**Uncommon Causes**
- Central anticholinergic syndrome:
  • Less common with newer agents
  • Central - irritation, delerium, stupor, coma
  • Peripheral - tachy, blurred vision, dry mouth, urinary retention
  • Reversed by a -stigmine which crosses the bbb
  • Caused by any anticholinergic drug
• Dissociative coma:
  • If organic & pharmacological causes excluded dissociative coma shld be considered
  • 2-30 hours
• Thyroid failure:
  • Myxoedema coma -
  • Consider lx thyroid
• LA toxicity
## 1.13 Post Op Cognitive Changes

### Delirium
- = acute onset of disturbed mental function. Often short lived
- features:
  - Alteration of consciousness
  - Hallucinations
  - Fleeting delusions
  - Anxiety & distress
  - Diurnal variation
- Risk factors for development:
  - Age >65
  - Dementia
  - Functional impairment
  - Anaemia
  - Substance abuse
- 3 different motor types:
  - Hyperactive delirium (rare) = restless, irritable, agitated

---

A stepwise approach to the patient with prolonged unconsciousness.
Hypoactive delirium (71%) = lethargy, ↓ activity, unawareness

Mixed (29%)

Diagnosed using scoring systems eg CAM-ICU

Causes & investigations - need thorough workup for reversible causes:
  - Labs - UEs, phosphate, Mg, Ca, VBGs
  - Infection screen
  - Medications:
    - Top 3 = anticholinergics, opioids, benzo’s
    - Others eg dig, diuretics, steroids, warfarin
  - Substance abuse
  - Brain imaging

Treatment:
  - Prevention -
    - optimise all physiological parameters eg CVS stability, O2, acid base status, electrolyte abnormalities
    - Orientation protocol - repeatedly to surroundings
    - Protected night time sleep
    - Early mobilisation
    - senses:
      - Vision - access to glasses/visual aids
      - Hearing - access to hearing devices
    - Avoid dehydration/hypovolaemia
    - Remove non essential lines & catheters eg urinary catheters
  - Drugs:
    - Haloperidol (better than benzo’s & respiridone):
      - Initial: 1-2mg IV/PO/IM
      - Maintenance: 0.25-0.5 IV/PO/IM 4hourly
        ↪ can double doses if severe agitation
  - Specific circumstances:
    - Delirium 2nd to substance withdrawal:
      - Down taper dose rather than stopping
      - Alpha 2 agonist eg clonidine
    - Central anticholinergic syndrome - dramatic delirium (hypo or hyper)
      - Use physostigmine 10-30mcg/kg
Dementia

- Defined as:
  - series of chronic organic brain syndromes with irreversible pathology
  - Global deterioration of cognitive function without clouding of consciousness
- Frequent misdiagnosis of delirium vs dementia. Both can occur together
- Many causes of dementia assoc failure cholinergic transmission
  \[ \rightarrow \] . anticholinesterases can be used to ↑cognitive function

Postop Cognitive Dysfunction

- Definitions:
  - = deterioration in formal neuropsychological testing that would be expected in <3.5% of controls
    \[ \rightarrow \] doesn't define clinical features or severity
  - Disorder of thought processes which effect memory, comprehension, attention
- Difficult trial to do
- 1 study 1200 >60yrs old incidence of POCD:
  - 25% at week 1
  - 10% at 3 months
  - ↑incidence in age: 33% of 80+ group
- Known causes:

<table>
<thead>
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<td>Early POCD</td>
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<td>General rather than regional anaesthesia</td>
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<td>Lower level of education</td>
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<tr>
<td>Re-operation</td>
</tr>
<tr>
<td>Postoperative infection</td>
</tr>
<tr>
<td>Prolonged POCD (months postoperatively)</td>
</tr>
<tr>
<td>Increasing age only</td>
</tr>
</tbody>
</table>

- Theorised causes:
  - multiple emboli - especially following bypass
  - Periop physiological disturbances - eg
- Hyponatraemia
- Hypoxaemia/hypotension - although no evidence to support this
- Pre-existing cog impairment - ↑ risk with pre-existing issues

**Conduct of Anaesthesia to ↓POCD**

- Regional vs GA:
  - POCD incidence in 1st week: regional (12.7%) vs GA (21.2%)
    - but difference does not persist at 3 months
  - Overall no difference in POCD between regional & GA
    - but early differences may have large effect on recovery/length of stay/mobility

---

2.1.3 Pain Medicine

**1.1 Pain Definitions**

- Pain = an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage
- Duration of pain defines acute (<30d - 6months) ⇒ subacute (1-6months) ⇒ chronic (>6-12months)
  - arbitrary lengths

**1.2 Basic Pain History**

- SOCRATES:
  - Site
  - Onset
  - Character
  - Radiation
  - Associations
  - Timing
  - Exacerbating/Relieving factors
  - Severity

**1.3 Multimodal & Pre-emptive Analgesia**

**Multimodal Analgesia**

- use of number of drugs/analgesics/adjuvants in combo to achieve best pain relief possible
- Pain complex construct with sophisticated transmission pathways through nervous system
- Main targets of modulating pain transmission:
  - Peripheral receptors:
    - LA's
    - NSAIDs
  - ascending pathways
    - Opiates
    - NSAIDs
    - NMDA receptor antagonists
    - gabapentinoids
  - Descending pathways
    - Tramadol
    - Clonidine
    - 5HT3 antagonists
Central perception
- Opioids
- paracetamol
• Combination of drugs means can reduce total dose of any one drug

Pre-emptive Analgesia
• Transmission of pain signals evoked by tissue damage leads to sensitisation of complex peripheral & central pain pathways
• Pre-emptive analgesia given before surgery aims to limit this sensitisation
• Theory that preventing cascade of sensitisation will limit subsequent doses of analgesia
• Theory holds for nociceptive stimuli associated with tissue damage
  ➔ this leads to
  • Peripheral (nociceptors) sensitisation - by inflam response - substance P/prostaglandins/
  serotonin/bradykinin/histamine
  • Central sensitisation - by sustained afferent activation & upregulation of transmission ⇒
  ‘pain memory’
• Drugs & evidence:
  • Opioids - no evidence for pre-emptive
  • Ketamine - no evidence
  • Epidural -
    - single shot - some evidence reduction in analgesic demand postop
    - Continuous - no change post op analgesic demand
  • Caudal block - no evidence
  • Peripheral LA’s:
    - Pre-op incisional LA - no evidence compared to post op LA infiltration
    - Nerve blocks - very limited evidence
• Pre-emptive analgesia & chronic pain:
  • 1 trial pre vs post incisional treatment showed sig ↓ chronic pain at 6months
• Summary: limited evidence to support pre-emptive analgesia at all but limited side effects & good scientific rationale

1.4 Analgesic Agents
Opioids
Morphine
MOA
• still not entirely clear
• diff actions at diff levels:
  ➔ spinal cord level:
    - stim opioid receptors ⇒ ↓ release of substance P from dorsal horn neurons ⇒ ↓ afferent transmission of pain
  ➔ supraspinal levels:
    - opioid receptors widely distributed in CNS esp limbic, thalamus, hypothalamus, midbrain
      ➔ altered perception of pain
Opioid Receptors
• receptors where endogenous opioid peptides function (enkephalins & endorphins)
• action at these receptors classified:
  o agonists – natural or synthetic
  o antagonists
  o partial agoists eg buprenorphine – less than max effect at mu receptors
• opioid receptors are GPCRs. Activation ⇒
  o inhibit adenylate cyclase ⇒↓cAMP levels
  o ↑opening K channels ⇒↑K out
  o ↓opening of Ca channels ⇒↓Ca in
  ➔ overall effect ↓neuronal excitability & ↓release of excitatory pain transmitters
• tolerance due to:
  o loss inhibitory functions
  o ↑excitatory signalling
• withdrawal due to rebound ↑cAMP levels via delta opioid receptors
• receptors:
  o µ (mu): (endogenous = B endorphins)
    • strong agonists – morphine & fentanyl
    • partial agonist – buprenorphine
    • weak agonist – pethidine
    • response:
      • supraspinal analgesia & euphoria
      • resp depression & sedation
      • constipation
      • Miosis
      • bradycardia
    • dependance
  o κ (kappa): (endogenous = dynorphins)
    • agonist – morphine
    • little/no activity – methadone, pethidine
    • response:
      • spinal & periph analgesia
      • resp depression & sedation
      • dysphoria
      • miosis
  o δ (delta):
    • agonist – (endogenous enkephalins)
    • response:
      • spinal analgesia
      • resp depression & constipation
      • rebound in withdrawal
  o σ (sigma):
    • stim by partial agonists eg buprenorphine
    • only –ve response: dysphoria, hallucinations, confusion
• receptor summary:
  o analgesia & constipation assoc with all three
  o euphoria = µ
  o dysphoria = κ & δ

**Agonists & Antagonists of Receptors**
• agonist analgesics =
  o morphine, pethidine
  o activate µ & κ
• partial agonists =
buprenorphine
- only activate 1 receptor & minimal effects at others
- may induce undesirable σ receptor
- antagonists – naloxone & naltrexone antagonise all receptors

Pharmacokinetics
- generally not well absorbed
- low & variable bioavailability due to extensive 1st pass metab in liver
- in IV dosing remains variable plasma conc, rates of metab & elim
- morphine protein bound (35%)
- main metabolites of morphine:
  - morphine-6-glucuronide (M6G)
  - morphine-3-glucuronide (M3G)
- wide volume of distribution
- small fraction cross bbb
- excreted:
  - primarily by kidneys
  - 10% enterhepatic circulation ⇒ prolonging half life
- mean elim half life 2-3hrs
- onset of action:
  - morphine = hydrophilic . only slow entry to CNS
  - fentanyl = highly lipophilic . rapid onset & short duration action
- liver damage:
  - may accumulate active drug
  - sensitive to depressant effects of drug
  - pethidine ⇒ toxic metabolite norpethidine may ⇒ seizures
  - methadone may be safer in liver disease
- renal disease:
  - extend half life of opioids excreted in an active form ⇒ resp depression
- eg methadone, pethidine, M6G

Equivalent Dosing
- 30mg oral morphine = 10mg IV

Uses
- CNS effects:
  - analgesia
  - suppression cough reflex
  - suppression resp centre
  - sedation & sleep
  - euphoria
  - dysphoria – hallucinations & nightmares
  - miosis – pinpoint pupils
  - N&V via CTZ
  - prolongation of labour
  - ↓bp & bradycardia in large doses via medulla
  - tolerance & dependence via μ receptors
• PNS effects:
  o GIT effects: \( \downarrow \) motility & \( \uparrow \) smooth mm tone \( \Rightarrow \) constipation
  \( \downarrow \) loperamide = weak opioid
  o spasm of smooth mm \Rightarrow delayed gastric emptying, bilary colic, urinary retention
  o suppression of some spinal reflexes
  o release of histamine \Rightarrow bronchoconstriction & severe itching

**Adverse Reactions**

• main incl:
  o resp depression
  o excessive sedation
  o dysphoria
  o constipation
  o N&V
  o tolerance & dependence
  \( \downarrow \) tolerance to dosing but no tolerance to SEs

**Cautions/Contraindications**

• elderly & infant <1 – dose needs reduced due to \( \uparrow \) CNS sensitivity & \( \downarrow \) ed clearance
• hypovolaemic pts – IM absorb \( \downarrow \) ed
• avoid in:
  o acute resp depression
  o acute alcoholism
  o HI
• caution in:
  o acute asthma
  o COPD
  o elevated ICP – morphine small \( \uparrow \) ICP
  o pancreatitis/bilary colic
• Rx in preg – risk of fetal withdrawal in labour

**Interactions**

• alcohol or other CNS depressants –
  o additive effect on CNS
  o \( \downarrow \) RR
  o \( \downarrow \) bp
• buprenorphine given with full agonist:
  o additive effect on \( \downarrow \) RR if given concurrently with full agonist
  o \( \downarrow \) analgesic effect of full agonist
  o precipitate withdrawal symptoms
• MAOIs:
  o intensify opioid effects – esp tramadol & pethidine
  o risk of serotonin syndrome
• diltiazem, erythromycin, fluconazole:
  o inhibit metab of alfentanil \Rightarrow \( \uparrow \) conc
• rifampicin \Rightarrow \( \uparrow \) metab of morphine, codeine, & alfentanil

**Dose**

standard dose 10mg IV/IM; 30mg oral
Other Opiates

Codeine
- = prodrug of morphine
- metabolised to morphine & norcodeine; metabolites excreted in kidneys
- 5-10% of whites lack enzyme (CYP2D6) to metabolise codeine, so no analgesic effect
  - rapid metaboliser may reach toxic concentrations

Fentanyl
- potent opioid
- short duration of action
- good adverse effect profile, so popular in anaesthetics
  - ↓ ed constipation
- varies preparations of administration, including patch
  - patch:
    - duration of action 3 days: not easily reversed
    - heat ⇒ ↑ uptake of drug from patch
    - rash & itching from site
    - after 3 days patches still contain 50% activity

Methadone
- duration of action 4-6 hours; but with repeated doses may extend to 72 hours

Tramadol
- centrally acting synthetic analgesic, which is not chemically related to opioids
- MOA:
  - agonist of µ receptors (<50% of action)
  - inhibit reuptake of NA & 5HT
  - called opioid-SSRI analgesic
- used for moderate-severe pain & neuropathic pain
- less effective than morphine
- prodrug which requires activation by liver metabolism (CYP2D)
  - multiple interactions especially with drugs affecting serotonin levels

Excreted by kidney
- SEs: nausea, dizziness, HTN, seizures

Pethidine
- only IV/IM
- less effect than morphine on histamine release or to ↑ smooth muscle contraction
  - good for acute asthma, biliary colic/pancreatitis
- has a toxic metabolite = norpethidine:
  - from liver metabolism
  - can accumulate, so drug only suitable for short term
- MAOIs ⇒ severe SEs incl serotonin syndrome
- used highly in drug seekers

Oxycodone
- synthetic opioid x10 more powerful than codeine
- well absorbed rectal mucosa if required

Heroin
- = prodrug rapidly metabolised to morphine on administration
  - is more lipophilic than morphine, so greater CNS penetration ⇒ ‘rush’
Paracetamol

- safer than aspirin because:
  - adverse effects & allergic reactions rare with therapeutic doses
  - low risk gastric upset
  - plasma protein binding negligible \( \therefore \) no displacement & less drug interactions
  - no sig drug interactions eg can take concurrently with anticoagulants
  - safe in children – no Reye’s syndrome
  - safe in preg & lactation

MOA

- inhibition of some COX isoenzymes \( \Rightarrow \) \( \downarrow \) PGs at site of injury
- exact MOA are not clear
- does inhibit COX in some tissues in some species
- \( ?? \)acts as prodrug with one of its active metabolites activating cannabinoid receptors in CNS

Pharmacokinetics

- orally – rapidly absorbed – peak plasma 15-60mins
- elim half life 1-3hrs
- metabolised in liver:
  - norm pathway: metabolised to glucuronide & sulfate derivatives
  - high dose/toxic pathway:
    - saturation of normal pathway
    - metabolised to benzoquinone intermediates (BQI)
    - BQI has 2 pathways of metab depending on available glutathione:
      - enough glutathione \( \Rightarrow \) paracetamol-mercapturic acid derivative (non toxic)
      - depleted glutathione \( \Rightarrow \) formation protein derivatives, lipid peroxidation, oxidative stress
        \( \Rightarrow \) liver cell death

\( \Downarrow \) N acetylcysteine is a synthetic analogue of glutathione

Uses

- effective
  - antipyrexic
  - analgesic
- very limited anti-inflammatory

Adverse Reactions

- rare at normal levels
- nausea & rash have been reported
- overdose can lead to serious liver/renal damage

NSAIDs

MOA

Analgesic

- inhibition of COX isoenzymes \( \Rightarrow \) \( \downarrow \) breakdown of arachidonic acid \( \Rightarrow \) \( \downarrow \) PGs, \( \downarrow \) prostacyclins & \( \downarrow \) Thromboxane A2 at site of injury
- PGs sensitise nociceptors to actions of bradykinin & other pain mediators
- COX1 & COX2 catalyse synthesis of PGs involved in pain
  \( \Downarrow \) also GI side effects of which COX2 shows less of
- analgesic action is peripheral & central
• Opioid sparing effect of 20-40%

**Antipyrexic**
• inhibition of PG synthesis in hypothalamus

**Side Effects**
• GI side effects:
  o due to ↓synthesis of mucoprotective PGs by systemically absorbed NSAIDs
  o incl: dyspepsia, N&V, gastritis, constipation/diarrhoea
• renal damage:
  o ↓ed vasodilator PGs
  o esp in elderly on long acting NSAIDs
• asthma
• skin reaction – urticaria
• Na retention ⇒ heart failure & HTN

**Comparison of NSAIDs and COX-2**

<table>
<thead>
<tr>
<th></th>
<th>NSAIDs</th>
<th>COX-2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Efficacy for</strong></td>
<td>Diclofenac 50mg (2.3)</td>
<td>Celecoxib 200mg (4.5)</td>
</tr>
<tr>
<td><strong>moderate to</strong></td>
<td>Ibuprofen 400mg (2.4)</td>
<td>Parecoxib 20mg (3.0)</td>
</tr>
<tr>
<td><strong>severe acute pain</strong></td>
<td>Ketorolac 10mg (2.6)</td>
<td>Valdecoxib 20mg (1.7)</td>
</tr>
<tr>
<td>(numbers needed to treat—NNT)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Renal function</strong></td>
<td>Can affect renal function</td>
<td>Similar adverse effects</td>
</tr>
<tr>
<td></td>
<td>postoperatively</td>
<td>on renal function</td>
</tr>
<tr>
<td><strong>Gastrointestinal</strong></td>
<td>Acute gastroduodenal damage</td>
<td>Less clinically</td>
</tr>
<tr>
<td></td>
<td>and bleeding can occur. Risk</td>
<td>significant peptic</td>
</tr>
<tr>
<td></td>
<td>increased with higher doses,</td>
<td>ulceration than</td>
</tr>
<tr>
<td></td>
<td>history of GI ulceration,</td>
<td>NSAIDs (VIGOR</td>
</tr>
<tr>
<td></td>
<td>long-term use, and elderly</td>
<td>and CLASS studies)</td>
</tr>
<tr>
<td><strong>Platelet function</strong></td>
<td>Inhibit platelet function but do</td>
<td>Do not impair</td>
</tr>
<tr>
<td></td>
<td>not significantly increase</td>
<td>platelet function</td>
</tr>
<tr>
<td></td>
<td>surgical blood loss in normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>patients. Associated with higher</td>
<td></td>
</tr>
<tr>
<td></td>
<td>incidence of post-tonsillectomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>haemorrhage</td>
<td></td>
</tr>
<tr>
<td><strong>Aspirin-exacerbated</strong></td>
<td>10–15% of asthmatics affected</td>
<td>Do not produce</td>
</tr>
<tr>
<td><strong>respiratory disease</strong></td>
<td>when given aspirin. Cross-</td>
<td>bronchospasm</td>
</tr>
<tr>
<td></td>
<td>sensitivity with NSAIDs</td>
<td></td>
</tr>
<tr>
<td><strong>Bone healing</strong></td>
<td>Impaired in animal models. No</td>
<td>Similar to NSAIDs</td>
</tr>
<tr>
<td></td>
<td>good evidence that clinically</td>
<td></td>
</tr>
<tr>
<td></td>
<td>important</td>
<td></td>
</tr>
</tbody>
</table>

**Ketamine**
• has certain benefits over other GA/analgesic agents:
  o bronchodilator
  o minimal cardiovascular depression
  o minimal resp depression
  o amnesia
MOA
• non competitive NMDA receptor antagonist:
  o receptor opens in response to glutamate
  o ketamine blocks channel ⇒ analgesic effects
• at high doses: also binds to opioid µ (mu) & σ (sigma) receptors
• also effects on other receptors:
  o potent D2 partial agonist
  o dopamine reuptake inhibitor
  o NA reuptake inhibitor
• produces dissociative anaesthesia
  ↓MOA of these hypnotic effects under debate

Pharmacokinetics
• onset of anaesthesia 15-30sec
• recovery time 15-30min
• metab in liver
• frequent dosing ⇒ tolerance due to induction of hepatic enzymes

Uses
• GA – induction & maintenance
• analgesia

Side Effects
• tachycardia & HT
• ↑ICP
• ↑intraocular pressure
• hypersalivation
• laryngospasm
• hallucinations – thus often also give benzodiazepines. Worse in adults
• re-emergence phenomena – disagreeable dreams, hallucination on awakening

Cautions/Contraindications
• caution in:
  o CVS disease- although tends to maintain or ↑CO
• crosses placenta:

Interactions
• additive effect with other sedatives incl benzo’s, barbituates, opiates, alcohol

Dose
• induction dose 1-2mg/kg
• paeds dose for minor procedure 2-2.5mg/kg IM (0.5mg-1mg/kg IV)

1.6 Principles of Acute Pain Management
(PS41)

Principles
• Adverse physiological & psychological effects result from unrelieved severe acute pain
• Effective post op pain relief will:
  • ↓morbidity
  • ↓hosp length of stay
• Chronic pain
• Requiring tailoring of Rx regimes to individual patients
• Requires close liaison with all staff & education of patient & carer
• Effective acute pain Rx depends on formal protocols & guidelines at local institutions & quality assurance programs to evaluate effectiveness of these regimes
• Special pt groups:
  • Children
  • Pregnant
  • Elderly
  • Indigenous peoples
  • OSA pts
  • Liver & renal disease
  • Opioid tolerant pts/substance abuse patients
  • Cognitive behavioural pts

Assessment of Analgesic Efficacy
• Regular assessment of pt needing including checking for side effects
• Patients should be involved in self assessment of their pain including effects of different interventions
• Pain should be assessed at rest & during activity
• Pain which suddenly increasing may signal development of new medical/surg/psych diagnosis
• All side effects & complications should be recorded

Pharmacological Therapies
• Agents to use:
  ‣ Opioids
  ‣ NSAIDs
  ‣ LA’s
  ‣ Adjuvants:
    - Antidepressants
    - Anticonvulsants
    - Membrane stabilisers
• Use careful titration & individualisation of dosing
• Multimodal analgesia (use of diff classes) is good
• Specialist routes require expertise:
  ‣ PCA
  ‣ Epidural & intra-thecal
  ‣ Regional LA’s
  ‣ Continuous infusions opioids/LA’s/ketamine

Non Pharmacological
• Complimentary:
  ‣ Psych interventions
  ‣ Acupuncture
  ‣ TENS
  ‣ Physio

Acute Pain Service Guidelines
• For all patients with complex medical/psych problems:
• Features:
  ‣ Med personal: anaesthetists & specialist nurses
  ‣ Liaison with MDT
- Develop protocols & guidelines for Rx & monitoring
- Review all patients at least daily
- Consultation service for pts with acute/acute on chronic pain
- After hours service
- Discharge analgesia plans
- Research
- Education

1.7 Management of pain in Recovery
• Nurse controlled IV opiate titration

1.8 Pain Management plan for Day Surgery

Summary
• Patient education - starting at pre-assessment
• Written information about analgesics & the regimen eg:
  - Description of drug
  - When taken
  - For how long
  - Side effects
  - Who to contact if problems
• Detailed drug history and allergies:
• Pain assessment:
  - Verbal rating - none, mild, mod, severe
  - Visual analogue - <3cm acceptable
• Peri-operative techniques:
  - Early planning - from pre-assessment
  - Multimodal:
    - NSAIDs - always if able
    - Opiates - use shorter acting if able (if using morphine use <0.1mg/kg)
    - Regional blocks - bupivocaine max 2mg/kg
    - Spinal anaesthesia - time to mobilisation may be increased. Full recovery prior to d/c
• Paeds patients:
  - Pain assessment harder in young children
  - Paracetamol 90mg/kg/day
  - Distraction therapy

1.9 & 1.12 PCA’s & Opioid Infusions
• PCA helps overcome the marked variability in response to post op opioids
• Patients titrate their own plasma opioid concentration into the therapeutic window:
  - > minimum effective analgesic concentration (MEAC)
  - < minimum toxic concentration (MTC)
• Safety of PCA is that if excessive doses of opioid given pt will become sedated and thus stop pressing button

PCA Regimes
• Most common is morphine although greater incidence of pruritis than other fentanyl
• Regime:
• No loading dose - pts should be comfortable before starting PCA
• Bolus - morphine 1g, fentanyl 10mcg, tramadol 10mg
• Concentration - standardised to institution
• Lock out - 5mins
• Background infusion - use with extreme caution
• Dose limit - often not used. Eg 30mg morphine in 4hrs
• Paeds regime - local protocols
• PCA been shown to be effective in as low as 6yr olds

**Complications**

- Equipment malfunction:
  - Battery failure
  - Electricity surges
  - Failure of anti-reflux valve led to resp depression
- Operator error:
  - Programming errors
  - Drug errors
- Side effects to opiates:
  - N&V
  - Pruritis
  - Sedation
  - Resp depression
  - Urinary retention
  - Confusion
  - Constipations
  - Hypotension
- Continuous infusions very dangerous and should be used very sparingly due to context sensitive half live of drugs

**Troubleshooting**

- N&V:
  - Add antiemetic to bag eg ondansetron, cyclizine, haloperidol
  - Prescribe reg antiemetic
  - Change opioid
- Pruritis:
  - Ondansetron
  - Anti-histamine
  - Change opioid
- Breakthrough pain:
  - Multi-modal analgesia
  - Incr bolus dose
  - (consider background infusion)
- Resp depression:
  - Best indicator of resp depression is sedation level
    - 0 = wide awake
    - 1 = easy to rouse (mild drowsy)
    - 2 = easy to rouse (mod drowsy)
    - 3 = difficult to rouse
    - S = asleep but easy to rouse
  - Decrease dosing
  - Use titrated naloxone if required
1.9 Regional Anaesthesia Risks & complications

- Major risks:
  - Direct trauma to nerve - needle/suture/instrument
  - Neurotoxicity of LA's
  - Ischaemia from compression (haematoma/abscess)
  - Infection
  - Unknown cause

**Direct Trauma**

- Good technique & anatomy knowledge
- Use short bevelled needle
- Use ultrasound (although nerve stim though to be no better)
- If severe resistance or pain on injection ≈ stop. Suggests intraneural or intrafascicular injection
- Symptoms within hours = extra/intra-neural haematoma or oedema
- Symptoms within weeks = tissue reaction or scar formation

**LA Toxicity**

- High plasma levels from:
  - Drug overdose
  - Direct IV injection
  - Rapid absorption from highly vascular area
  - Cumulative effect from multiple injections
- Thus consider:
  - Site & vascularity of injection
  - Acidosis, hypoxia, hypercarbia all potentiate =ve ionotropic/chronotropic effects of LA
  - Keep to max doses

Maximum recommended doses of common agents (BNF)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Maximum recommended doses</th>
<th>Maximum recommended doses with vasoconstrictor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bupivacaine</td>
<td>2mg/kg</td>
<td>2mg/kg</td>
</tr>
<tr>
<td>Levobupivacaine</td>
<td>2mg/kg</td>
<td>2mg/kg</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>3mg/kg</td>
<td>3mg/kg</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>3mg/kg</td>
<td>6mg/kg</td>
</tr>
<tr>
<td>Prilocaine</td>
<td>6mg/kg</td>
<td>8mg/kg</td>
</tr>
<tr>
<td>Cocaine</td>
<td>1.5–3mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

**Signs of toxicity**

- Mild:
  - Perioral tingling
  - Metallic taste
  - Tinnitus
  - Visual disturbance
  - Slurred speech
- Moderate:
Altered consciousness
Seizures
Coma

Fatal:
Cardiovascular collapse
Resp arrest

**Treatment of Toxicity**
- Stop injection
- ABC

- Mild symptoms - consider midaz or small doses of propofol to ↑seizure threshold
  ↓ NB hypoventilation & acidosis will worsen toxicity

- Moderate to severe toxicity:
  - Conventional therapies to Rx hypotension/tachy/bradycardia
  - Early use of 20% intralipid:
    - 1.5ml/kg bolus over 1min
    - Start infusion 15ml/kg/hr
    - @5mins: if CVS still unstable
      - repeat bolus (can do total of 3 boluses)
      - Double infusion rate
  - Continue CPR - arrythmias may be very refractory to treatment

- Methaemoglobinaemia Prilocaine toxicity
  - Specific to prilocaine
  - Hb oxidated to metHb by o-toluidine
  - O-toluidine formed by metabolism of prilocaine in liver
    ↓ in high doses >600mg
  - MetHb has ↓O2 carrying capacity ⇒ cyanosis
  - ↓: avoid prilocaine in pregnancy and anaemia
  - Rx: methylene blue 1mg/kg IV

**1.10 Actue Pain patients who are Previously opioid Dependent**
- Tolerance = ↓sensitivity to opioids to same dose
- Dependence = physiological phenomenon characterised by withdrawal reaction when drug is withdrawn or antagonist administrered
- Addiction = pattern of drug abuse characterised by compulsive use to experience a psychological effect & to avoid withdrawal reaction
- Pseudoaddiction = iatrogenic drug seeking behaviour normally due to under-treatment of acute pain by physician
- Signs of withdrawal:
  - Yawning
  - Sweating
  - Anxiety
  - Rhinorrhoea
  - Lacrimation
  - Tachy
  - Hypertension
  - Diarrhoea
  - N&V
  - Abdo pain
Cranps
- Symptoms peak at 36-72hrs
- Aims of treatment:
  - Provide analgesia
  - Prevent opioid withdrawal
  - Manage abnormal behaviour
- PCA settings may need to replace usual opioid dose eg ↑bolus dose or background infusion
- Aim to d/c pt on no more opioid than was on at admission
- Dose reduction of 20-25% every day towards pre-admission opioid will avoid withdrawal
- Oral or s/c clonidine 50mcg tds can be used to Rx opioid withdrawal
- Objective assessment of function ie ability to cough better guide than pain scores
- Use regional techniques wherever possible

1.13 Management of hypotension assoc with a central neuraxial block
- Order of Rx:
  - Volume resuscitation with IV fluids
  - Posture - legs up if possible
  - Consider wide bore access
  - Vasopressors - especially if unresponsive to volume bolus

2.1.4 Perioperative Medicine

PO 1.1 ASA status
- American Society of Anaesthesiologists:
  - 1 = healthy with no systemic disease
  - 2 = Mild to mod systemic disease
  - 3 = severe systemic disease imposing functional limitation on patient
  - 4 = severe disease with constant threat to life
  - 5 = moribund pt who not expected to survive ± operation
  - 6 = brainstem dead pt for organ donation
- Incidence of death in ASA
  - 1 & 2 = 1:100,000
  - 3 & 4 ± emergency surgery = ↑x5-10 risk

PO 1.2 & 1.3 Functional Assessment
- Exercise tolerance (cardiovascular fitness)= major predictor of risk
- Physiological response to major surgery ⇒ ↑o2 demand by 40%
- Fitness defined by metabolic equivalents (METs)
- Scale defined by Duke Activity Status Index:
  - 1-3 METS = light activities:
    - 1 = Watching tv
    - 2 = strolling very slowly
    - 3 = walking at 4k/hr
  - 3 -6 METS = moderate intensity activities
    - 3 = Static bike very slowly
    - >4 = climbing a flight of stairs
- 4 = leisure bicycle <10 mph
- 4 = climbing flights of stairs,
- >6 METS = vigorous activities
  - 7 = jogging
  - 8 = pushups, situps
  - 10 = rope jumping

Cardiopulmonary exercise testing (CPET)

- Risk of survival depend on:
  - Age
  - Sex
  - Organ dysfunction: brain, heart, kidney, periph artery disease
  - Fitness
- Fitness only variable not routinely quantitatively measured and documented
- CPET used to define resp & cardiac variables of pt
- Requirements for CPET:
  - Exercise machine
  - Computer controlled ramped ↑workload
  - Calibrated pneumotachograph to measure gas flow & composition
  - Continuous 12 lead ECG
  - Someone trained to perform and analyse results
- Survival correlates with:
  - Peak O2 consumption
  - Power
  - HR
  - Anaerobic threshold
  - O2 uptake slope
  - Oxygen pulse
  - HR recovery
- Early work showed anaerobic threshold to be most important factor:

<table>
<thead>
<tr>
<th>Anaerobic threshold</th>
<th>Mortality rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test ECG: no ischaemia</td>
</tr>
<tr>
<td>&gt;1ml O2/kg/min</td>
<td>0/107 (0%)</td>
</tr>
<tr>
<td>&lt;1ml O2/kg/min</td>
<td>2/36 (5.5%)</td>
</tr>
<tr>
<td>All</td>
<td>2/143 (1.4%)</td>
</tr>
</tbody>
</table>

- CPET helpful to provide:
  - Individual estimation of survival
  - Informed decision making
  - Peri-op management - HDU/ICU need
  - Risk reduction by guiding interventions
- Used as standard before AAA surgery & heart transplants

PO 1.4 Treatment of life threatening arrhythmias

- ....insert brady & tachy arrhythmia algorithms
PO 1.5 Perioperative Risk & Anaesthetic Implications

Respiratory Infection

Adult
- Current resp tract infections with:
  - Fever AND
  - Cough
  - ± chest signs
  - should not have elective procedure 2nd to ↑ risk post op resp complications
- Adult pts with coryza not at ↑ risk unless:
  - have other chronic resp problems OR
  - Major abdo/thoracic surgery
- Laryngospasm more likely if recent URTI but currently asymptomatic

Paeds
- Pre-school kids 6-8 URTIs/yr
- 25% kids have chronic runny nose
- GA with concurrent URTI assoc ↑ risk of:
  - Excess secretions
  - Airway obstruction
  - Laryngospasm
  - Bronchoconstriction
  - risk x5 with LMA; x10 with intubation
- Children to postpone
  - Productive cough
  - Purulent sputum or nasal secretions
  - Fever
  - Constitutional symptoms eg D&V
- Child with mild URTI borderline decision:
  - Hx: if now post viral, apyrexial, no chest signs & systemically well = prob ok for surg even runny nose
- Length of time to postpone:
  - Significant URTI - postpone 2wks
  - LRTI - 4 weeks
  - Bronchiolitis - 6wks

COPD
- If element of reversibility of airflow obstruction then Rx as asthma
- BiPAP very helpful post op if needed

Preop Ax
- Exercise tolerance eg METs
- Rx all potential reversibility - consider trial oral pred/resp r/v
- Pulmon HTN & R vent failure possibility - optimise heart failure Rx

Ix
- Spirometry
- ABGs - if:
  - difficulty climbing 1 flight stairs
  - Cyanotic
Spo2 <95% on RA
Periph oedema
CXR
ECG

Anaesthesia
- Severe COPD ≈ likely post op NIV needed ≈ elective HDU/ICU admission
- Avoid ETT if able although pts with marked secretions may benefit from endotracheal toilet
- Vigilance for pneumothorax
- Avoid histamine releasing drugs
- Premed B agonists
- ↑ risk bronchospasm - consider potent opioids/LA to cords
- Use short acting potent opioids if post op pain will allow

Post Op
- Exubate in sitting position

OSA
- Sleep apnoea syndrome = cessation of airflow for >10 seconds
- Develop hypoxaemia & resp arrest during REM sleep
- Hypoxia ⇒ restart of resp
- Symptoms:
  - Overweight snorers
  - Disturbed sleep
  - Excessive daytime drowsiness
  - Headache
- 2 types of sleep apnoea syndrome:
  - OSA 85%
  - Central apnoea 10% - loss of central drive
- Long term complications of undiagnosed apnoea’s:
  - Systemic & pulmon HTN
  - RV hypertrophy ⇒ failure
- Pts at ↑ risk peri-op airway obstruction & resp failure post drugs

PreOp Ax
- Undiagnosed in 80%
- Ask about daytime sleepiness & snoring from partner
- Ensure HTN & failure maximally managed
- Consider resp opinion if periph oedema & Spo2 <92%
- Bring own CPAP machine to hosp for op
- All children presenting for adenotonsillectomy should be considered to ± sleep apnoea

Ix
- FBC
- ECG - ?R heart strain ⇒ ECHO
- ABG baseline

Anaesthesia
- Avoid sedative premeds
- Anticipate intubation & BMV may be difficult
- Regionals good
- Short acting opiates if able

Post Op
- Exubate sitting
- ?HDU/ICU
• Few hours of ventilation post op of benefit
• Aim for preop spo2
• Watch for CO2 retention

**Heart Failure**
- Commonest cause of admission to hosp in >65
- 50% 5 yr mortality
- Characteristics:
  - Decreased exercise tolerance & fatigue
  - Orthopnoea
  - SOB
  - Ventricular arrhythmias
- Uncontrolled failure & emergency laparotomy = mortality of 20-30%

**Medical Management**
- Drugs:
  - Diuretics:
    - Spiro & ACEI ↓s mortality if ECF <25%
    - Vasodilators - ACEI, ARB, nitrates
    - Bblockers - ↓ arrhythmia's & ↓ myocardial o2 demand
    - Inotropes - dig useful if concurrent atrial arrhythmia
    - Anticoags - indicated in:
      - Atrial arrhythmia
      - Intracardiac thrombus
      - LV aneurysm
      - Hx of VTE

**PreOp**
- Hx - any decompensating episodes last 6/12
- Optimise med management & continue meds
- Rx metabolic abnormalities
- Aggressive Rx of arrhythmias - esp AF

**ECHO**
- EF:
  - 60-80% = norm
  - 40-50 = mild
  - 30-40 = mod
  - <30 = severe

**Anaesthesia**
- If severe heart failure:
  - Dependant on preload for vent filling
  - Rely on sympathetic tone
  - Poorly tolerant of any change in physiology
  - Use regional techniques if able
- Give all anti-failure meds that day
- ACEI - resume as soon as poss post op. If >3days then resume at lower dose
- Decompensating pts may need inotropes or phosphodiesterase inhibitors
- Watch Uo carefully as renal perfusion and GFR will be borderline
- Good analgesia regime to avoid symp stresses of pain
- Low threshold for ICU admission
Arrhythmias

Sinus Brady
• Causes:
  • Drugs - BB’s, dig, anticholinesterases, sux
  • Cardiogenic - MI, sick sinus
  • ↑ICP, hypothyroid, hypothermia
• Rx:
  • Stop surg stim
  • Antimuscarinic
  • Chronotropes - isoprenaline or adrenaline
    ▶️ also consider glucagon

SVT/Nodal Re-entry
• Sinus massage
• Adenosine
• Bblockers - esmolol/metoprolol
• Ca channel blockers - verapamil can be useful if relapse post adenosine
  ▶️ avoid co-use with BB’s
• Amiodarone
  (avoid dig - facilitate accessory pathway and WPW)

VT
• May be triggered intra-op by:
  • MI, hypoxia, hypotension
  • Fluid overload
  • Electrolyte imbalance
  • Ionotropes
• Rx:
  • Sync shock - 200-360J (approx 100% success)
  • If relapse use lignocaine or amiodarone
  • Lignocaine -
    - bolus 100mg (30-40% success)
    - Maintenance 4mg/min for 30min; 2mg/min 2 hr, then 1mg/min
  • Other drugs:
    - Amiodarone
    - Procainamide

Heart Block
• Bi/tri-fasicular block rarely progress to complete heart block during anaesthesia thus not normal to pace unless episode of syncope:
  • RBBB with L ant hemiblock = V1 RSR AND L axis dev (more common)
  • RBBB with L post hemiblock = R axis dev (non specific)
• 1st deg block ok
• 2nd degree block - consider need for pacing
• Intraoperative heart block:
  • Atropine - rarely effective but try
  • Isoprenaline
  • Transcutaneous/oesophageal/invasive pacing

Pacemakers
• Pacemaker codes:
  • 5 positions - 1st 3 antibrady functions and are always stated
• Position 1 = chamber paced
  - O - none
  - V - ventricle
  - A - atrial
  - D - dual
• Position 2 = sensing chamber - OVAD (as above)
• Position 3 = response to sensing:
  - O - no action in response to sensing. ie will pace no matter what.
  - I - inhibit
  - D - dual
• Position 4 - rate modulation:
  - O - none
  - P - simple program
  - M - multi-program
  - R - rate modulation
• Position 5 - anti-tachy functions
  - O - none
  - P - pacing
  - S - shock
  - D - dual
• emergency mode = DOO
• ideal ICU mode = DDI
• ICD codes:
  - Pos 1 = shock chamber: OAVD
  - Pos 2 = chamber to which antitachycardia pacing is delivered: OAVD
  - Pos 3 = means of detection of tachy:
    - E - intracardiac electrogram
    - H - haemodynamic means
  - Pos 4 = 3-5 letter code for pacemaker function

**Anaesthesia**
- Preop battery check & function
- ECG to confirm function eg AV synchronicity, polarity of pacing, baseline rate
- Concern about electromagnetic interference
  - ↓ diathermy - if a must - plate position so current flows away from pacemaker
- Bipolar diathermy is safe
- In emergency magnet over box ⇒ asynchronous vent pacing (VOO) on next cardiac cycle
  - ↓ note if in severe heart failure loss of A-V synchrony may ⇒ ↓↓ CO
  - ↓ may need technician to help!

**Venous Thromboembolism**
- PE responsible for ≈ 10% hosp deaths
- Without prophylaxis 40-80% high risk pts will develop DVTs
- Incr VTE present 2nd to:
  - Hypercoagulable 2nd to surg/cancer/hormone therapy
  - Venous stasis
  - Interference with VR eg pregnancy, pelvic surg, pneumoperitoneum
  - Dehydration
  - ↓Cardiac output

**Risk Factors**
- Duration and type surg:
• >30mins = high risk
• Surg to abdo/pelvis/joint replacement

• Pts factors:
  • Hypercoagulable RFs eg prev DVT, thrombophilia
  • Obstetric eg preg, OCP
  • >40yrs
  • Obese
  • Vvs

• Assoc diseases:
  • Malignancy
  • Trauma
  • Heart disease
  • Sepsis
  • Haem diseases

plits into low, moderate, high risk

Prophylaxis
• Heparin ↓s incidence of fatal VTE by 66%
• LMWH:
  • Give 1800 so >12hrs prior to surg allowing neuraxial blocks
  • Check renal function and local dosing policy
  • Start post op

• Unfractionated heparin:
  • Bridging heparin in high risk. Local protocol
• Graduated compression stockings:
  • ↓DVT risk, but not PE risk
  • May be better with LMWH
  • Advisable for all laparoscopic procedures
• Intermittent pneumatic compression devices:
  • Compress leg 35-40mmHg for 10secs/min
  • As good as heparin in preventing DVT
• Warfarin - good evidence in ortho ops

Choice of Anaesthetic
• Regional is protective esp in LL joint replacement

OCP use & VTE
• OCP may ↑risk 3-4x VTE periop
• Risk may ↓longer been on OCP
• Progesterone only OCPs do not change risk
• Poor evidence means:
  • Decide on individual basis for people undertaking major operations and other individual RFs
  • Need to stop 4/52 prior to surg

HRT & VTE
• HRT ⇒ ↑risk VTE
• BNF suggests to stop HRT 4-6weeks before major surgery but balanced decision

Electrolyte Abnormalities

Acid-Base Abnormalities
Chronic Renal Impairment

- CRF = multisystem disease
- Renal failure when GFR <35
- Dialysis usually when <15
- ESRF GFR <5
- Main causes:
  - DM 30%
  - HTN 24%
  - GN 17%
  - Unknown 20%

Preoperative

- Check for HTN/DM/anaemia/IHD
  - Consider ECHO - higher risk valve disease & LVF
- Type of dialysis
- Residual UO
- Fluid status: hyper vs hypo-volaemic
- Allow 4-6hr post haemodialysis before surg
- Indications for urgent dialysis:
  - Hyperkalaemia
  - Fluid overload
  - Acute acidosis
  - Symptomatic uraemia
- Plan for ICU

Ix’s

- FBC: aim 80-100
- K - aim <6
- Coags:
  - ↓ platelets - consider cryo or DDAVP

PeriOp Care

- vessels:
  - Avoid fistula arm for all lines/monitoring
  - Cannulate back of hand to save other vessels
  - Use A lines sparingly - and radial only
- Fluids:
  - Aim for normovolaemia
  - Avoid hypotension
  - Use NaCl 0.9%. avoid any fluid with K+
  - Use CVL line if big fluid shifts expected
- Sux - ↑’s serum K by 0.5mmol.
  - ↑K also worsened by acidosis so avoid hypovent & hypercarbia
- Delayed gastric emptying likely. But reserve RSI for normal indications
- Careful aseptic technique for all lines - immunosuppressed
- Universal precautions - Hep B & C are common

Post Op

- Liaise with renal unit for next dialysis
- Close fluid balance - if oliguric:
  - Hourly fluids to replace losses + 30mls/hr for insensible losses
- Avoid nephrotoxics
- Avoid hypotension
**Drugs in CRF**
- Loading doses unchanged, maintenance doses ↓ed
- Hypoalbuminaemia & acidoses ⇒ ↑ active available drugs which norm protein bound eg induction agents
- Drug classes:
  - Analgesics:
    - Fentanyl - inactive metabolites but still may accumulate if prolonged use
    - Remi & alfentanyl fine
    - Tramadol has active metabolites
  - Induction agents: ↓ by 30% dosage
  - Volatiles - no change
  - Muscle relaxants:
    - Sux as above
    - Plasma cholinesterase unchanged
    - Avoid vec & roc infusions
    - Neo/glyco excretion is prolonged
  - La’s:
    - ↓ max dose by 25%
    - Consider ↑ ed risk of spinal haemorrhage & haematoma with neuraxial blocks

**Steroid Dependance**
- Endogenous cortisol (hydrocortisone) = 25-30mg/24 in circadian pattern
- During stress = 75-100mg/day
  - can remain elevated up to 72hr following major surgery
- Pred (vs hydrocortisone):
  - X3-4 more potent in glucocorticoid & anti inflam
  - Much less active mineralocorticoid
  - thus why hydrocort often used peri-op
- Expect HPA suppression if taking >10mg pred daily
- HPA suppression ln’ed by short Synacthen test
- Fludrocortisone:
  - Oral tab only
  - Withhold if being given IV hydrocort

**Rx Regime**
- <10mg - no change
- >10mg:
  - Minor surg (eg hernia)- routine steroid that day or hydrocort 25mg IV @ induction
  - Mod surg (eg hysterectomy) -
    - routine pre op steroid
    - Hydrocort 25mg Iv @ induction AND 6hrly for 24hrs
  - Major surg -
    - Routine preop steroid
    - Hydrocort 25mg @ induction and then 6hrly for 48-72hrs
- High dose immunosupression:
  - Convert usual oral steroid dose to hydrocort, then revert back to oral dose when able
- If taking steroids until <3months ago ⇒ Rx as if on steroids

**TIA/Strokes**
- Causes of death in developed world = heart disease > Ca > stroke
TIA
- Causes by embolism of platelet & fibrin from atherosclerotic plaques
- Risk of stroke post TIA = 5%/yr with mortality 30%/episode
- Ix with doppler studies in defined service
- Should delay all but emerg surgery for workup
- Indications ref for carotid surgery:
  - >80% stenosis
  - Ragged plaque

Stroke
- Assoc with:
  - HTN
  - DM
  - Obesity
  - Smoking
  - ↑age
- Look for renal & herat disease

Timing of operation
- Op within 6 wks of stroke ⇒ ↑x20 risk post-op stroke
- Hemiplegia <6-9months ⇒ ↑ed K response to sux
  ↓: wait 3-6 months before elective surg

PreOp Ax
- Aim for stable bp & BSL
- Bridging LMWH is required
- Document carefully neuro baseline - allows Ax new lesions
- Consider VBI symptoms - can they extend neck without any symptoms

Anaesthesia
- Cont antiHTN's (except ACEIs)
- Maintain normotension:
  - Pressors
  - Opioids/labetalol/esmolol/GTN
- Neutral neck position
- Cover intubation with strong opiod to prevent HTN spikes
- Avoid hypocarbia ⇒ ↓CPP
- Close examination in PACU

Plasma cholinesterase Deficiency
- Aka pseudo-cholinesterase deficiency
- Capable of hydrolysing variety of esters
- No physiological function found for enzyme yet
- Synthesized in liver, half life 5-12d
- Metabolised 70% 100mg sux <1min
- Several variant genes:
  - Atypical -
    - heterozygotes no issue unless concurrent illness
    - Homozygous - 1:3000 - paralyse for 2-3hrs
  - Silent gene:
    - Heterozygote - mild prolongation sux
    - Homozygote - prolonged apnoea - 3-4 hrs but upto 24hrs
  - Flouride resistant gene:
- Homozygote very rare - 1:150000, moderately sensitive to sux
- Other variants also seen with varying effects
- Can lab test for activity
- Also see ↓ plasma cholinesterase activity in:
  - Hepatic/renal disease/burns/malignancy/malnutrition
- Drug interactions:
  - Esmolol, MAOI, MTX - all compete for metabolism \( \Rightarrow \) prolonged sux action
  - Anticholinesterases - inhibit plasma cholinesterase as well
- Pregnancy - ↓ activity by 25%
- Plasmapheresis & bypass

**Diagnosis**
- Unable to sustained head off pillow for 5 secs
- TOF - adductor pollicis
- DBS - more accurate than TOF

  (non depolarising) Post tetanic count:
  - Use when TOF = 0
  - 50Hz tetanic stim applied for 5 secs then single stim every second
  - Reversal possible if count >10

**Anaemia**
- Causes:
  - Blood loss - acute vs chronic
  - Bone marrow failure
  - Megaloblastic anaemias - b12 or folate deficiency
  - Complex anaemias:
    - Renal failure
    - RA
    - Hypothyroid
  - Haemolytic anaemias:
    - Inherited - thallasaemia or sickle cell
    - Acquired - autoimmune, drugs, infections
    - Physical - mechanical valves, DIC
- Always ask about NSAIDs and alcohol

**Ix**
- Pre-op Hb in major surg or those at risk
- Anaemia screen:
  - Iron studies
  - B12/folates
  - TSH
  - Renal/liver function
  - Direct Coombs test

**Rx**
- Check FBC weeks before elective surgery to allow for corrective Rx:
  - IV iron/oral iron
  - B12/folate supplementation

**Periop Transfusion**
- Restrictive approach to transfusion becoming more evidence based
  - If Hb ↓s then CO↑s due to a ↓ in viscosity of blood \( \Rightarrow \) maintenance of O2 delivery
  - Use of HaemoCue - to check Hb intra-op
• Threshold 70-80.
• Consider higher levels if major systemic disease but this is only a historical theory

**Transfusion Reactions**

**Types:**

- **Actue haemolytic transfusion reaction:**
  - ABO incompatibility due to clerical error
  - Recipient antibodies bind to transfused red cell antigens ⇒ haemolysis
  - Shock, ARF ± death

- **Bacterial contamination:**
  - Rapid onset of CVS instability, rigors, collapse
  - Rare but more common with platelets stored at room temp

- **TRALI:**
  - Antibodies in transfusion unit reacting with antigens in recipient
  - 1:5000 to 10,000 of plasma products ie FFP, whole blood
  - Leuco-deplete rbcs is ↓s frequency
  - Should be considered if pt develops APO within 6hrs of transfusion
  - Manage as would ARDS/ALI

- **Acute transfusion reactions (ATR):**
  - Up to 24hrs post transfusion
  - Anaphylaxis to febrile non haemolytic reactions

- **Delayed heamolytic transfusion reactions (DHTR):**
  - >24hrs post transfusion
  - 2nd to development of red cell alloantibodies

- **Transfusion assoc graft vs host disease (TA GvHD):**
  - Usually in immunocomprimsed
  - Engraftment & proliferation of transfused lymphocytes
  - Damage cells with HLA antigens in skin, liver, spleen, bone marrow
  - Fever, skin rash, diarrhoea, dermatitis
  - Usually fatal
  - Leucodepletion also reduced incidence

- **Infections transmissible by transfusion:**
  - Eg HIV/HCV/syphillis, vCJD
**Acute Blood Loss**

- Establish percentage of circulatory loss:

<table>
<thead>
<tr>
<th>Classification of hypovolaemic shock according to blood loss (adult)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
</tr>
<tr>
<td>Blood loss (%)</td>
</tr>
<tr>
<td>Blood loss (ml)</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
</tr>
<tr>
<td>Pulse (bpm)</td>
</tr>
<tr>
<td>Capillary refill</td>
</tr>
<tr>
<td>Respiratory rate</td>
</tr>
<tr>
<td>Urine output (ml/hr)</td>
</tr>
<tr>
<td>Extremities</td>
</tr>
<tr>
<td>Complexion</td>
</tr>
<tr>
<td>Mental state</td>
</tr>
</tbody>
</table>

- If no antibodies on G&S - compatible blood can be electronically issued in 5mins
- If antibodies present then delay up to 2hrs

**Processes for Red Cell or blood Product Transfusion**

- Confirm identity to pt or by wrist band
- Check blood compatibility label with the blood bag
- Check expiry date & unit
- Inspect bag integrity & evidence of red cell clumping
- If blood out of fridge >30min needs to transfused within 4hrs of discarded
- Meticulous documentation

**Thrombocytopenia**

- Platelet count <150
- Spont bleeding uncommon unless <10-20

**Causes:**

- Failure production:
  - Selectively:
    - Hereditary
    - Drugs
    - Alcohol
    - Viral
  - General marrow failure:
• Aplasia
• Cytotoxics
• Infiltration/fibrosis
• Myelodysplasia

- ↑ed consumption:
  - With immune basis:
    • ITP
    • Drugs
    • Viral infections
    • SLE
    • Lymphoproliferative disorder
  - Without immune basis:
    • DIC
    • TTP
    • Bypass
- Dilution - massive transfusions
- Splenic pooling - hypersplenism

• If unexpected results - rpt sample

Preop Preparation
• Ifx unexpected thrombocytopenia preop
• Bone marrow biopsy can be done without platelet cover
• Acceptable counts:
  - >50=
    - Major procedures eg laparotomy
    - CVLs
  - >100
    - LP/epidurals
    - Special ops eg brain/eye
• If ITP - reserve platelet T/Fs for major surgery & use high dose steroids

Post Op
• If microvascular oozing despite platelets >50 = DIC
  ↑ if so give cryo & FFP

• Avoid all IM injections
• Desmopressin 0.3mcg/kg in 100mls NAcL/30min may help in certain situations:
  • ARF/CRF
  • Haemophilia
  • vWF disease

Coagulation Disorders
• Extrinsic & intrinsic pathways now thought only in vitro
• Now common pathway:
  • TF release from vascular beds
  • TF combines with VIIa ⇒ activation IX, X ⇒ generation IIa (thrombin)
  • Process amplified causing activation V & VIII ⇒ massive amounts of thrombin ⇒ fibrin
• Causes coagulation disturbance:
  • Acquired:
    - Lack synthesis of factors
    - Consumption of factors eg DIC
    - Massive blood loss
  • Hereditary:
    - Haemophilia A -
• X linked defect in VIII activity
• Levels:
  • <2% = severe - spont bleeding
  • 5-30% = mild - bleed after trauma
• Elective cases get level day prior to surg & aim for 50-100% levels AND for 2/7 post op
• Avoid all drugs effecting platelet function
  - Haemophilia B - sex linked recessive IX
  - vWD -
    • autosomal dominant
    • 3 subtypes
    • Desompressin trial can be undertaken to see if a responder
    • Responders can have desompressin for surg (prophylactically or bleeding)
    • Non responders - give
      • VIII concentrate - includes vWF
      • cryo
• Concurrent medical problems may be relevant:
  • Liver disease
  • Malabsorption - vit K deficiency
  • Infection
  • Malignancy (DIC)
  • Autoimmune disease - RA/SLE
  • Medications - NSAIDs

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Platelet count</th>
<th>INR</th>
<th>APTT</th>
<th>TT</th>
<th>Fibrinogen</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemophilia A</td>
<td>Normal</td>
<td>Normal</td>
<td>†</td>
<td>Normal</td>
<td>Normal</td>
<td>↓ VIII</td>
</tr>
<tr>
<td>Haemophilia B</td>
<td>Normal</td>
<td>Normal</td>
<td>†</td>
<td>Normal</td>
<td>Normal</td>
<td>↓ IX</td>
</tr>
<tr>
<td>von Willebrand's disease</td>
<td>Normal (usually)</td>
<td>Normal</td>
<td>†</td>
<td>Normal</td>
<td>Normal</td>
<td>↓ VIII, vWF, † bleeding time</td>
</tr>
<tr>
<td>Liver disease</td>
<td>Normal or ↓</td>
<td>↑</td>
<td>↑</td>
<td>Normal or ↓</td>
<td>Normal</td>
<td>↓ V</td>
</tr>
<tr>
<td>Vitamin K deficiency</td>
<td>Normal</td>
<td>↑</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>↑ II, VII, IX, X</td>
</tr>
<tr>
<td>DIC</td>
<td>Normal or ↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑ FDPs, d-dimers, ↓ II, V, VIII</td>
</tr>
<tr>
<td>Massive transfusion</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>Normal or ↑</td>
<td>Normal or ↑</td>
<td>Normal FDPs</td>
</tr>
<tr>
<td>Heparin (unfractionated)</td>
<td>Normal (rarely ↓)</td>
<td>Normal or ↑</td>
<td>↑</td>
<td>↑</td>
<td>Normal</td>
<td>↑ anti-Xa</td>
</tr>
<tr>
<td>Heparin (LMWH)</td>
<td>Normal (rarely ↓)</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>↑ anti-Xa</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Normal</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>Normal</td>
<td>↓ II, VII, IX, X</td>
</tr>
<tr>
<td>Lupus anticoagulant</td>
<td>Normal</td>
<td>Normal or ↑</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>DRVVT +ve, cardiolipin antibody</td>
</tr>
</tbody>
</table>

**AntiCoagulants**

**Warfarin**
• Interferes with vit K metabolism ⇒ liver produces non functioning factors (II, VII, IX, X, protein C &S)
• Reversal:
  • Need depends on INR - >5 consider reversing
  • Vit K - oral vs IV
    ⇔ if emergency always give as adjunct
  • FFP
Prothrombin complex
- Surgery INR threshold:
  - <1.5 - norm surgery
  - <1.2 high risk surg
    ⇨ once <2 consider need for bridging anticoagulation
- Need to stop warfarin for operation controversial - can be based on scoring system
  ⇨ eg CHADS (heart failure, HTN, >75yrs, DM, stroke x2 points) 0-2 low, 3-4 mod, >5 high)
- 10% peri-op major bleeding risk if don’t stop warf (33% of them need blood transfusion)
- Risks without anticoagulation of VTE:
  - Mechanical heart valve = annual 17% or 0.4% for 8day periop period
  - AF = 8day perip op = 1%
  - Previous VTE = embolic stroke significant neuro deficit in 70% of cases & fatal in 4-9%
    ⇨ bridging VTE effective for VTE
- Warf should be stopped 5d prior to surgery
- Bridging:
  - Unfractionated heparin - by protocol. Stop 6hr prior to surg
  - LMWH
  - IVC filter - very high risk
- Restart warf 12-24hrs post op

Heparin
- Potentiates antithrombin
- Unfractionated heparin monitored by APTT
- Half life is 1-2hrs but complex pharmacokinetics and narrow therapeutic window mean strict protocols are important
- Stop 6 hours prior to surgery
- Protamine reversal - give slowly to avoid hypotension.
- Complications of heparin:
  - HIT ⇒ serious venous & arterial thrombosis
    ⇨ less of a problem with LMWH
- LMWH renally excreted

Anti-platelet Agents
- Decrease platelet aggregation
- May inhibit thrombus formation in arterial circulation
  ⇨ anticoagulants have little effect

Aspirin
- Irreversible binding to platelets ⇒ ↓ thromboxane A2 production
- Need new platelets to reverse effect (7-9days)
- Aspirin use peri-op In
  - CABGs:
    - ↑peri-op bleeding
    - ↑graft patency
  - TURPs - significant ↑peri-op bleeding
- Defo need to stop if:
  - Retinal surgery
  - Intracranial surgery
  - TURP

Dipyridamole
- Needs to be stopped at least 7d prior to surgery
- Less clinically significant effect than aspirin
Clopidogrel
- Binds irreversibly with ADP receptor on platelets
- prodrug
- Stop 7d prior surg
- Can try platelet transfusion but should be ≥24hr after last dosing

**Immunosuppressed patient**
- 3 classes of drug:
  - Immunophilin binding drugs - prevent cytokine mediated T cell activation & proliferation
    - eg ciclosporin A, tacrolimus
  - Nucleic acid synthesis inhibitors - block lymphocyte proliferation
    - eg azathioprine
  - Steroids -
    - block production inflam cytokines
    - Lyse T lymphocytes
    - Alter function remaining lymphocytes
- Ciclosporin -
  - associations:
    - Renal dysfunction - Often causes HTN
    - Prolongs non depolarising muscle relaxants
  - Ca channel blockers ⇒ ↑ciclosporin levels ∴ ⇒ ↓dosing regimes
- Tacrolimus:
  - Renal dysfunction
- Steroids - supplementation may be required. See above
- Must use strict asepsis in all invasive procedures

**Rheumatoid Arthritis**
- =chronic systemic inflam disorder involving mainly joints but with extra articular effects
- Peak onset 30-55
- Higher than av mortality due to both disease & concurrent disorders
- Stills disease in children

**PreOp Ax**
- See airway Ax
- Non articular:
  - CVS:
    - Assoc IHD
    - Vasculitis & raynauds
    - Pericarditis & pericardial effusions common
    - Aortic incompetence & endocarditis rare
  - Resp:
    - Costco-chondral disease gives ↓ed chest wall compliance
    - Fibrosing alveolitis or acute pneumonitis
    - Pleural effusions
  - Anaemia:
    - NSAID assoc blood loss or anaemia of chronic disease
    - DMARD assoc bone marrow suppression
    - Felty’s syndrom = splenomegaly, neutropaenia, anaeamia & thrombocytopaenia
  - Nervous system:
    - Periph & compression neuropathies
    - Cx cord compression
• Infections - common 2nd to disease or iatrogenic
• Renal & Hepatic -
  - Iatrogenic CRF
  - ↓albumin, ↑fibrinogen & ↑a - acid glycoprotein

Ix
• Routine blood tests
• Cx spine XRs - flex & ext views:
  ↩ only mandatory if neuro signs or symptoms or persistent neck pain
  ↩ MRI better test
  ↩ consider inline stabilisation/AFOI

Peri-Op
• Drugs:
  • Steroid supplement if required
  • NSAIDs- only stop if:
    - Bleeding risk
    - Hypotension
    - ↓ing renal function
  • DMARDs - little evidence effects risk of wound infection thus continue
  • TNF-a blockers - suggestions of potential ↑post op infection risk but no consensus whether to stop
  • Use gastro prophylaxis esp if on NSAIDs
• Good positioning on the table
• Regional techniques may be difficult because of pain while remaining immobile
• Norothermia
• Strict asepsis techniques

PostOp
• PCA may be difficult due to hand function
• Early mobilisation
• Maintain fluids
• Restart DMARDs early

Smoking
• Contains nicotine and at least 43 known carcinogenic compounds
• Long term assoc ⇒ ↑risk:
  • COPD
  • Lung cancer
  • IHD
  • Vascular disorders
• Effects of smoking:
  • ↑resp tract mucus
  • ↓mucociliary clearance
  • ↑anaesthesia susceptibility :
    - resp events:
      • Post op atelectasis
      • Desat during induction
      • Post op pneumonia
        ↩ these risk specifically ↑ed with abdo/thoracic surgery or obesity
    - ↑ed airway irritability:
      • Coughing
• Laryngospasm
  ➔ can avoid by using less irritant volatile eg sevo & deepening anaesthesia slowly
  ➔ if spont breathing required may have to LA vocal cords or use high dose opioids
• COHb may be up to 15% in heavy smokers
  ➔ falsely reassuring Spo2 readings

**Risk Reduction**
• Total abstinence from smoking for 8 weeks ➔ ↓ morbidity from resp complications to non smoking level
• If stop for 12 hrs prior to surg still get benefit
  ➔ ↓ ed nicotine activated ↑ coronary vasc resistance (via symp system) AND ↓ COHb levels

## 2.15 Regional & Local Anaesthesia

**RA 1.1 College Document on Major Regional Analgesia**

• Informed consent should include discussion of risks including:
  • Nerve injury
  • Drug toxicity
  • Haemodynamic changes
  • Bleeding or bruising
  • Infection
  • Failure of technique
  • Post dural puncture headache
• Problems with informed consent in labour ward of PACU understood
• Should have qualified help when doing technique - tech or midwife
• preparation:
  • Need full infection control
  • Skin prep must be dried to avoid contaminating equipment or drugs
  • Coagulation status must be assessed before all blocks
  • IV access prior & maintained during duration of technique
• Monitoring:
  • During insertion:
    - ECG, SPo2, RR, conscious state, frequent bp
    - Continue that level until 30 mins after vitals stable
  • Person doing block must be around to assess satisfaction of block or until immediate complications have passed
  • May then delegate responsibility to other MDT members eg pain team
• Full record keeping incl prescription charting
• Equipment:
  • Catheters & giving sets must be well labelled and specifically a diff colour
  • Dedicated pumps with set protocols to avoid OD
• Post procedure r/v:
  • Local protocols to r/v for complications, effectiveness, side effects, timing of removal
  • Daily r/v
MRI preferred to CT for nerve injury
Remove catheters if suspected infection and send for culture
Late complications of neuraxial analgesia:
- Postdural puncture headache
- Epidural abscess
- Epidural haematoma
- Spinal cord or nerve root compression

Neuraxial Anatomy
- Spinal cord terminates L1 adults (L3 infants)
- Iliac crests = Tuffers line = L4 level
Subarachnoid space
- ends S2 in adults (lower in children)
- Extends laterally along nerve roots to dorsal root ganglia
Subdural space = potential space inbetween dura & arachnoid mater
Epidural space =
- lies between walls of vertebral canal & ligamentum flavum & spinal dura mater
- Low pressure area occupied areolar tissues, loose fat & internal vertebral venous plexus
- Ligamentum flavum maximal thickness in Lx region 2-5mm

Technique
- Midline
- Paramedian:
  - 1-cm lateral to upper border of spinous process
  - Insert needle perpendicular to contact lamina of vertebra
  - Withdraw slightly reinserting 15 deg medial, 30deg cephalad to pass over lamina through interlamina space until pop through dura

Coagulation Disorders & Regional Techniques
- Haemorrhage can be brisk ⇒ haematoma ⇒ nerve compression
  \[\text{in/around spinal cord} \Rightarrow \text{permanent paralysis}\]
- Coagulopathy relative contraindication depending considerably on context
- Numbers:
  - Platelets >80
  - INR <1.5

Epidural Analgesia
- Can provide complete analgesia for 3-5days

Benefits
- Efficacious
- ↓ed atelectasis & pulmon infection, better cough
- ↓post op ACS:
  - ↓sympathetic stress thus ↓myocardial oxygen requirement
  - ↓hypercoagulable states & fibrinolytic function is improved
  \[\text{proven benefit in graft survival in vascular surgery}\]
- Quicker post op mobility ⇒ ↓post op DVT
• ↑gut action by ↓pain & ↓opiate need
• Intraop epidural ↓s post op blood transfusions
  \(\Rightarrow\) BUT no ↑ survival benefit in high risk patients

**Contraindications**

- Patient refusal
- Untrained staff
- Contraindications to needle placement:
  - Local or general sepsis
  - Hypovolaemia
  - Coag disorders:
    - Platelets <80
    - INR >1.5
  - Concurrent anticoag drugs
  - Central neurological diseases

**Tips**

- Breakthrough pain:
  - Add oral paracetamol or NSAID
  - Bolus dose 3-5ml then ↑ infusion rate
  - Check all connections and infusion site
  - Check block - if patchy withdraw catheter to 2cm in space
  - Bolus fentanyl 50-100mcg only
- Pruritis:
  - Give naloxone 50-100mcg & consider adding 300mcg to infusion fluids
  - Remove opioid from infusion
  - Try antihistamines or ondansetron
- Hypotension:
  - Check fluid status
  - Check block height \(\Rightarrow\) ↓ infusion rate
  - Ephedrine/metaraminol
- Motor block -
  - ↓ infusion rate
  - ↓ LA concentration
Complications

Complications of epidural anaesthesia\(^4\) (see also pp746–51)

<table>
<thead>
<tr>
<th>Complication</th>
<th>Incidence (%)</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dural puncture</td>
<td>0.16–1.3</td>
<td>Bed rest, analgesia, hydration, blood patch (see p748)</td>
</tr>
<tr>
<td>Headache</td>
<td>16–86</td>
<td>Bed rest, analgesia, hydration, suspect dural puncture</td>
</tr>
<tr>
<td>Nerve or spinal cord injury</td>
<td>0.016–0.56</td>
<td>Immediate neurological assessment (see p22 and p1178)</td>
</tr>
<tr>
<td>Catheter migration</td>
<td>0.15–0.18</td>
<td>Remove catheter and resite if appropriate</td>
</tr>
<tr>
<td>Epidural haematoma</td>
<td>0.0004–0.03</td>
<td>MRI or CT scan. Immediate neurosurgical assessment. Antibiotics (see also p1105 and p1171)</td>
</tr>
<tr>
<td>Epidural abscess</td>
<td>0.01–0.05</td>
<td></td>
</tr>
<tr>
<td>Respiratory depression</td>
<td>0.13–0.4</td>
<td>Decrease in opioid concentration may be required</td>
</tr>
<tr>
<td>Hypotension</td>
<td>3–30</td>
<td>IV fluids ± vasopressors. Temporarily reduce or stop infusion</td>
</tr>
<tr>
<td>Pruritus</td>
<td>10</td>
<td>Naloxone IV (50–100µg) ± antihistamine</td>
</tr>
<tr>
<td>Urinary retention</td>
<td>10–30 (in males)</td>
<td>Catheterisation</td>
</tr>
<tr>
<td>Motor block</td>
<td>3</td>
<td>Check for catheter migration. Temporarily cease infusion. Consider epidural haematoma (p1171 and p1174)</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>Possible increased risk of anastomotic leakage after bowel surgery. No evidence to support this</td>
</tr>
</tbody>
</table>

- Spinal infection:
  - Classic triad of epidural abscess (only seen together in 13%):
    - Fever (66% on own)
    - Backache (75% on own)
    - Neurological signs (very late sign)
  - Normal bloods mean nothing
  - If suspect should remove immediately and send line tip to lab
  - 90% infections are bacterial (mostly staph aureus)
  - MRI early before neurology develops
  - Once muscle weakness develops:
    - only 20% will regain full function even after surgery
    - Better prognosis: <36hrs, extent compression, younger
  - Mortality 10%
  - Needs percutaneous abscess & Abx

Drugs in Epidural

- Standard protocols used in different institutions:
  - Light mix - bupivacaine 0.125% & fentanyl 5mcg/ml
- Infusion rates:
  - 8-15ml/hr adult
  - 4-8ml/hr >70yr olds
Spinal Anaesthesia

Dosing

- Older & pregnant need less
- 2.5 - 3mls of hyperbaric will reach T6-T10 in most non pregnant young if placed in lying shortly after injection
- If isobaric LA given dose needs to be higher
- Lignocaine not used
- Ropivocaine not licensed for intrathecal use
- Hyperbaric solutions:
  - Used to get higher block
  - More hypotension
- Isobaric:
  - Produce lower lock height
  - Less hypotension

Contraindications

- Absolute:
  - Local sepsis
  - Refusal
  - Anticoagulation (see epidural)
- Relative:
  - Aortic or mitral stenosis
  - Hypovolaemia/hypotension
  - Prev back surgery - possibly technically difficult
  - Neurological disease
  - Systemic sepsis - ↑ed risk of meningitis/epidural abscess

Complications

- Hypotension
- Bradycardia -
  - block into mid thoracic region
  - Can progress to cardiac arrest
- High block ⇒ compromised breathing ⇒ total spinal
- Urinary retention
- Nerve damage -
  - permanent injury 1:25,000 to 1:50,000
  - Paraplegia or death 1:50,000 to 1:140,000
- Post dural puncture headache
- Infection
- Bleeding

RA 1.6 & 1.11 Complications of Neuraxial Block

Hypotension

- Avoid aortocaval occlusion (pregnancy) ⇒ move to full lateral position
  - measure bp on dependant arm
- IV fluid bolus
• Vasopressor/inotrope - ephedrine vs metaraminol

**Subdural block**

• When epidural catheter placed between dura mater & arachnoid mater
• Less than 1:1000 BUT may be indistinguishable from epidural placement
• Definitive diagnosis is radiological
• Characteristics of subdural block:
  ‣ Slow onset 20-30min which is much more extensive than volume should dictate
    ↦ may extend to Cx dermatomes with Horners syndrome
  ‣ Patchy & asymmetrical block with sparing of motor fibres to LLs
  ‣ Total spinal with top up dose
    ↦ due to ↑volume ⇒ rupture of arachnoid mater
• Rx by stopping infusion and re-siting catheter

**Total Spinal**

• If initial plan is epidural incidence = 1:5,000 - 1:50,000
• Features:
  ‣ Rapid onset BUT can be delay upto 30mins
    ↦ change maternal position or migration of catheter
  ‣ Rapid rising block
  ‣ Impaired coughing
  ‣ Loss hand/arm strength
  ‣ Difficulty talking, breathing & swallowing
  ‣ Cardiovascular depression ⇒ resp paralysis ⇒ unconsciousness ⇒ fixed dilated pupils
• Rx:
  ‣ Maintain airway & ventilation
    ↦ may need intubation if if not fully unconscious in order to protect airway
  ‣ Avoid aortocaval compression (pregnant)
  ‣ Ventilation for 1-2hours may be required

**IV injection of LA**

• IV or partial IV catheter positioning occurs in at least 5% epidurals
• Every dose is a test dose
• Strategies to reduce risk:
  ‣ Always check for blood in catheter
  ‣ Always think of LA poisoning with ever dose even if prev had no issues
  ‣ Divide all large LA doses into smaller aliquots
  ‣ Use low toxicity LAs
  ‣ LA toxicity algorithm

**IT 1.120 Plan B for a Regional technique**

• Steps:
  ‣ Consider technique - US vs periph nerve stim
  ‣ Reattempt if dosing allows
  ‣ Get help, another operator
  ‣ If partial:
    ‣ Co-sedation an option - midaz/propofol/remi
  ‣ GA
  ‣ Postpone surgery
  ‣ Consider placing indwelling catheter - epidural/periph nerve catheter/indwelling intrathecal catheter
IT RT 1.1 Systematic Approach to Identifying Problems

C - circulation, capnograph & colour
O - oxygen supply & oxygen analyser
V - ventilation & vaporisers
E - ETT, & eliminate the machine
R - review monitors & equipment
A - airway
B - breathing
C - circulation
D - drugs

SWIFT CHECK - of patient, surgeon, process, & responses

Four levels of intensity:
S - scan (every 5mins)
C - check (when not going to plan)
A/R - alert/ready
E - emergency

Severe Hypoxia

• Causes:
  • Gas mixture:
    - Incorrect flowmeter settings
    - Second gas effect - NO (especially on extubation)
    - O2 failure
    - Machine error
  • Failure to ventilate:
    - Vent depression or narcosis
    - Inadequate IPPV
    - Disconnection
    - Misplaced ETT - oesophageal/endobronchial
    - Airway obstruction - patient to machine
    - ↑airway resistance eg bronchospasm/laryngospasm
    - ↓FRC - PtX, ↑intra-abdominal pressure, morbid obesity
  • Shunt:
    - Atelectasis
    - Airway secretions
    - ↓hypoxic pulmonary VC
    - Heart failure & APO
    - Gastric aspiration
    - Pre-existing pathology - VSD/ASD
  • Poor O2 delivery in body:
    - Systemic hypoperfusion - hypovolaemia/sepsis
    - Embolus
Regional problems - Raynauds/vascular problems
- \( \uparrow \text{O}_2 \) demand -
  - Sepsis
  - Malignant hyperthermia

**Rx:**
- 100\% \text{O}_2
- Check Fio2
- Expose pt & check for central cyanosis
- Check vent bilaterally
- Hand ventilate on simple system - 4 large breaths for recruitment
- Secure airway
- Endotracheal suction
- Initially remove PEEP (consider brief disconnection of circuit) then trial more
- Adrenaline if losing pulses

**Hypocarbia**

**Causes:**
- shock:
  - Cardiogenic shock
  - Ischaemia
  - emboli
  - Distributive - septic
  - Anaphylactic
  - Hypovolaemic
- \( \uparrow \text{Ventilation} \)
  - Pain
  - Too much IPPV

**Check ABC**

**Hypoventilation/Hypercarbia**

**Causes:**
- Anaesthesia:
  - Coughing/breath holding/light anaesthesia \( \Rightarrow \) rapidly deepen with IV agent (10\%-20\% dose)
- Airway obstruction
- Position:
  - Lithotomy/trendelenburg
- Surgical factors:
  - Distended abdo
  - Loss integrity chest wall or diaphragm
- CNS depression - drugs eg opioids or sedatives
- Drugs:
  - High spinal
  - relaxants
- Muscle weakness
- Pre-existing conditions:
  - Primary - myopathies
  - Secondary - drugs/electrolytes
  - Trauma/neuropathy/stroke
- Equipment problems -
  - Disconnection/leaks/obstructions

**Signs:**
• Desat
• Hypercarbia
• Tachy/bradycardia

• Rx:
  • Rx primary cause
  • Control airway and ventilate lungs

**High Airway Pressures**

• Causes:
  • Misplaced ETT - listen to chest
  • Obstruction to airway/filter/mount/circuit ⇒ isolate with ambi bag
  • ↑ airway resistance - listen to chest
    - Laryngospasm
    - Bronchospasm
    - Anaphylaxis
    - Pulmonary oedema
    - Airway secretions
    - Aspiration gastric contents
  • ↓ FRC:
    - Morbid obesity
    - ↑ intraabdominal pressure - check with surgeon

**Bradycardia**

• Causes:
  • Vagal stimuli:
    - Peritoneal tension
    - Abdo distension
    - Visceral retraction
    - Airway stim
    - Extraocular muscle retraction
  • airway:
    - Severe hypoxia/hypoventilation
  • Primary cardiac problems:
    - Rhythm ie Av blockade
    - Ischaemia
  • Electrolytes:
    - hypokalaemia
  • Drugs:
    - Neostigmine
    - Propofol
    - Volatiles
    - Sux
    - Vasopressors
    - phenytoin
    - High Neuraxial LA blockade

• Signs - obvious

• Rx:
  • Stop all vagal stimuli
  • If cardiovascular unstable:
    - Atropine in 500mcg boluses up to 3mg
    - Adrenaline/isoprenaline/glucagon/glyco
- Transcutaneous pacing

• Be concerned if:
  • Recent asystole
  • Mobitz II/type 3
  • Vent pauses >3secs

**Tachycardia**

• If sinus tachy ⇒ consider hypotension and Rx
• If tachy arrhythmia choose Rx based on severity of hypotension:
  • Severe ⇒ sync shock
  • Mild ⇒ drugs

• Reversible causes:
  • Hypovolaemia:
    - Dehydration
    - Diuresis
    - Sepsis
    - Blood loss
  • Drugs:
    - Anaesthetic agents
    - Atropine
    - LA toxicity
  • Airway:
    - Hypoventilation/hypoxia
  • Anaphylaxis
  • Reflex stim
    - Pain!
  • Cardiopulmonary problems:
    - Obstructive lesions:
      • Tension
      • Tamponade
      • Massive haemothorax
    - Sepsis
    - Embolism - gas/amniotic/thrombus
    - Myocardial irritability - drugs/ischaemia/trauma

• Rx - based on diagnosis
• Rx of arrhythmias based on:
  • Pulse ⇒ no ⇒ ALS
    - Narrow
      • Patient stable ⇒ no ⇒ ALS (DC shock)
      • Regular or irreguler
        • Regular:
          - Vagal
          - Adenosine
        • Irregular:
          - AF - onset <48hrs:
            • Rate control
            • Rhythm control
    - Broad:
      • Stable ⇒ no ⇒ ALS (DC shock)
      • Regular:
        • Pulsatile VT ⇒ drugs vs shock
Severe Hypotension

• Causes:
  • Patient:
    - Hypovolaemia - HR >100, RR >20, ↑CRT, narrow pulse pressure, swing arm line
    - Obstructed venous return
    - Raised intrathoracic pressure eg tension Ptx - examine chest
    - Anaphylaxis
    - Embolism -
      • Suspect if pre-existing low CVP & open venous bed
    • Signs:
      - Sudden ↓ETCO2
      - ↓Spo2
      - Cardiovascular collapse ⇒ PEA
    • Pump failure -
      - Ischaemia
      - Failure - worsening Spo2 with fluid challenge, distended neck veins
      - Arrhythmia
    • Sepsis - warm peripheries
  • Technique
    - Measurement error - check pulse when cuff up
    - Excessive depth anaesthesia
    - High spinal block:
      • Horner's syndrome - small pupil/ptosis/anhydrosis/stuffy nose)
    • Drug error eg LA toxicity, barbituates

• Rx:
  • ABC
  • Optimise preload -
    - Fluid challenge with pressure infusion
    - Lift legs - very acute temporising measure (↑preload & afterload)
  • ↑contractility - ephedrine, adrenaline, Ca
  • ↑SVR - vasopressors

Severe Hypertension

• Causes:
  • Inadequate depth of anaesthesia - check TIVA/volatiles
  • Inadequate analgesia - trial alfentanil 10-20mcg/kg
  • Measurement error - palpate pulse/check transducer height
  • Hypoxia/hypercapnia
  • Drug error
  • Pre-eclampsia - >20wks, check platelets, proteinuria, LFTs, clotting
  • Raised ICP - Cushings
  • Thyroid storm
  • Phaeochromocytoma
  • Surgical techniques:
    - Aortic x clamp

• Rx:
· ABC
· Vasodilators (may cause tachycardias)
  - ↑ volatiles
  - GTN infusion
  - MgSo4 bolus 10mmol then infusion 5mmol/hr
  - clonidine
· B blockade (↑HR or dysrhythmia):
  - Esmolol
  - Labetalol - b:a blockade 7:1
· a blockade (normal or ↓HR):
  - Phentolamine

**Oliguria/Anuria**

- Causes:
  - Surgical factors
  - Hypovolaemia/hypotension
  - Fluid status
  - Cardiovascular/renal perfusion

**IT RT 1.2 Management of Life Threatening Conditions**

**Cardiac & Respiratory Arrest**

- ALS protocols
- Consider naloxone post op
- ABCD
- Post arrest care:
  - Optimise oxygenation
  - Ventilate to normalise CO2
  - Correct electrolytes
  - Keep BSL <10

**Shock**

- Hypovolaemic - fluids, blood, stop bleeding
- Distributive - fluids, adrenaline or other vasopressors
- Cardiogenic -
  - normalise cardiovascular parameters
  - Consider anti-thrombotics
  - Consider intubation if appropriate
  - Monitoring especially post op
- Obstructive - Rx underlying cause

**Cardiac Tamponade**

- Diagnose with ultrasound
- Classically:
  - Muffled heart sounds
  - Distended neck veins
  - Hypotension
- Rx with pericardiocentesis
- Call CT surgeon!
**Acute Myocardial Ischaemia**

**Perioperative MI:**
- Usually day 3-4 post op
- Causes:
  - acute plaque rupture (50%)
  - Oxygen supply/demand imbalance
- Rx:
  - Move to CCU/HDU
  - Aspirin/morphine/GTN
  - Consider B blockers to decr myocardial o2 demand
  - Angio relatively contraindicated - D/W cardiologist
  - Rx APO

**Acute Pulmonary Oedema**
- Causes:
  - ↑ hydrostatic pressure
  - ↑vascular permeability
  - ↓colloid pressure
  - -ve interstitial pressure
  - Obstructed lymph drainage
- Presents:
  - Frothy sputum
  - ↑HR
  - ↑RR
  - ↓SPo2
  - ↑CVP
- Rx:
  - 100% o2
  - If awake:
    - Sit up right if able - ↑s FRC and offloads pulmon vasculature
    - CPAP
  - If intubated:
    - ↑PEEP to at least >5cmH20
    - 15deg head up - ↓s atelectasis & improves FRC
    - Aspirate free fluid from trachea
  - Use GTN via spray/infusion or patch in either

**Aortic Dissection**
- Aim to reduce:
  - HR - to 60-70
  - Bp SBP 100-120 mmHg
- Use labetalol initially as has alpha action
- Cautious GTN as can cause reflex tachy

**Aspiration of Gastric Contents**
See pg 19

**Bronchospasm**
- Identify causes:
  - APO
  - Analphylaxis
Asthma

ETT obstruction

Rx:
- Suction or place bougie down ETT
- ↑ volatile - sevoflurane least irritant. Considering stopping dex
- IV salbutamol
- Inhaled salbutamol:
  - Aerosol spray in 50ml syringe with fine bore tubing fed directly down ETT
- Ketamine
- Aminophylline - 250mg (max 5mg/kg) slow IV injection

Tension Pneumo

Decompress

Massive Haemoptysis

- Early cardiothoracic involvement
- 100% o2
- Place pt in recovery position with bleeding lung down
- Plan RSI
- Consider need for double lumen tube to isolate bleeding lung
- IV access ⇒ CVL
- Flexible bronchoscopy later

Raised ICP

- Normal ICP 5-12mmHg
- Initial compensatory mechanisms by ↓ing volume of CSF & ↓ blood volume
- Then after marked ↑ in ICP per intracranial volume
- Causes of raised ICP:
  - ↑ed brain substance
  - ↑ed CSF volume
  - ↑ed blood volume
  - ↑ed ECF
- Autoregulation maintains cBF between MAP 50-140mmHg
  \[ \rightarrow \text{if chronic HTN then all limits increase} \]
- Rx:
  - Avoid ↑ing CBF further by avoiding:
    - hypercarbia
    - Hypoxia
    - HTN
    - Hyperthermia
  - Good anaesthetic depth
  - Good Analgesia
- Avoid ↑venous pressure - tube ties, head 30deg up, avoid coughing on tube
- Avoid hypotonic fluids
- Maintain CPP - avoid hypotension. Aim CPP >70mmHg
- Specific measures to ↓ICP:
  - Diuretics - mannitol 0.25-1g/kg over 15mins
  - Aim PaCO2 30-35 mmHg- effect for 24hours
  - Dexamethasone (if NOT trauma)
  - CSF drainage
  - Head up
Conc NaCL 25% 20ml boluses

Prolonged Seizures
• Benzo’s - lorazepam (0.1mg/kg) or midazolam (0.2mg/kg)
• Phenytoin 15-18mg/kg load
• Sodium valproate 20mg/kg slow push
• Clonazepam
• Intubate & sedate - thiopentone better than propofol

LA Toxicity
- see earlier

Anaphylaxis
• Adrenaline 50-100mcg boluses IV or 500mcg IM
• IVF
• Anti Hs and steroids later
• Refer for testing
• Reverse roc & vec with suggamadex

Malignant Hyperthermia
Aetiology
• = pharmacogenetic disease of skeletal mm
• Induced by exposure to:
  - Volatile agents
  - Depolarising mm relaxant ie sux
• Inherited autosomal dominant condition
• Caused by loss normal Ca homeostasis within excitation-contraction coupling process on exposure to trigger
• Any defect along complex process can trigger MH
• Most likely site:
  - Junction between T tubules
  - Voltage sensor of dihydropyridine receptor (DHPR) & Ryanodine receptor (RYR)
    \[\leftrightarrow\] efflux Ca channel in sarcoplasmic reticulum
    \[\leftrightarrow\] 70% families RYR1 gene linkage

Epidemiology
• Rare 1:10,000. All races
• Mortality fallen from 70-80% to 2-3% due to awareness & dantrolene
• Young adults; males>females
• Previous uneventful anaesthetic does not prevent occurrence

Signs & Symptoms
• Varied presentation:
  - Florid & life threatening vs insidious onset
  - Acutely vs 2-3d postop with massive myoglobinuria & rhabdomyolysis
• Signs:
  - ↑metabolism:
    - Tachy/Arrhythmia \[\Rightarrow\]
    - ↑ed CO2 production \[\Rightarrow\] most important early sign
    - Met acidosis
    - Fever (late) - ↑temp 2 deg/hr
    - DIC
  - Muscle signs:
- Masseter muscle spasm (MMS) after sux
  - = spasm impeding intubation persisting for around 2mins
  - 30% pts with MMS alone & otherwise normal anaesthetic ⇒ MH susceptible
  - If present:
    - Abandon surgery - possible OR
    - TIVA - volatile free surgery
    - Consider A line
  - Investigations:
    - Initial and 24hr CK
    - First void urinary myoglobinuria
  - Consider neurological opinion
    - Generalised rigidity
    - ↑K
    - High CK
    - Myoglobinuria ⇒ renal failure

**Differential**
- Rebreathing
- Sepsis
- Awareness
- Neuroleptic malignant syndrome
- Ectasy
- Thyroid storm

**Treatment**
- ABC. Stop volatiles
- Hyperventilate - 100% O2 to flush volatiles from system
- Declare problem to team and get help
- Use fresh breathing circuit machine if able
- Dantrolene 2-3mg/kg IV (20mg ampoules - so about 4)
  - up to 10mg/kg
- Stop surgery or use TIVA
- Reduce core temp:
  - Ice to groin & axilla
  - Cold fluid into
    - bladder via catheter
    - Veins
    - Stomach via NG tube
- ABG - correct acidosis & potassium
  - beware bicarb as will produce more CO2
- Call for surg team help to conclude operation as quickly as possible

**Peri-MH Treatment**
- Invasive monitoring
- Clotting screen & CK
- Urine samples
- Monitor renal function ⇒ diuretics and IVF

**Post Episode Care**
- Ref to MH investigation unit for mm biopsy & testing
- Warn pt & family
- Pt & family should be offered screening
Anaesthesia for known MH
• MH safe technique - TIVA with no sux may be safe - but balance risks
• All LA's are safe
• Dantrolene should not be given prophylactically
• Standard monitoring
• Baseline temp recorded 2hr preop & temp monitored for 4 hrs post op
• Use vapour free machine
  ➔ if unable: remove soda lime, vaporisers and purge for 30mins with O2

Anaesthesia for suspected FHx
• Establish goof Fhx and d/w MH centre for contact tracing & diagnoses
• If case urgent then proceed with MH safe technique

General Anaesthesia
• GA drug = produces reversible state of unconsciousness with absence of pain sensation over entire body
• drugs need rapid onset of action and to be reversible
• usually
  o induced by injection of anaesthetic agent eg propofol or thiopentone
  o maintained by inhalational of a gas (nitrous oxide) mixed with volatile liquid eg halothane/sevoflurane

Stages of Anaesthesia
• 4 stages:
  o 1-2 = induction
    ➔ stage 2 dangerous . rapid induction to stage 3, with maintenance there
  o 3 = surgical anaesthesia
  o 4 = medullary paralysis

Stage 1 Analgesia
• beings with onset of anaesthetic administration
• lasts until LOC
• order of effects:
  o smell & pain ↓ ed first
  o auditory or visual hallucinations
  o speech difficult
  o hearing last sense lost

Stage 2 Excitement
• varies greatly individuals
• depends on
  o amount & type of premeds
  o anaesthetic agent
  o levelof external stimuli
• most reflexs still present & exaggerated esp noise
• swallowing risk abolished ⇒ risk aspiration
• signs:
  o increase in:
    * autonomic activity
• mm tone
• eye movement
• dilation of pupils
  o irreg breathing – uneven inhalation of anaesthetic
  o vomiting

**Stage 3 Surg Anaesthesia**
• surgery generally done in plane 2 – upper plane 3
• subdivided into 4 planes:
  o plane 1:
    • resp incr shallow & rapid until paralysis & requires assisted ventilation
  o plane 2:
    • loss of reflexs in cephalocaudal direction
    • conjunctival reflex lost
    • pupil constrict ⇒ reaction to light lost ⇒ dilate
    • gag & laryngeal reflexs lost
  o plane 3:
    • ↓mm tone – need flaccid abdo wall for surgery
    • ↓body temp: skin cold, wet & pale
  o plane 4: ↓ing bp & weaker pulse

**Stage 4 – Medullary Paralysis**
• toxic stage
• impending overdose, resp arrest & vasomotor collapse
• artificial resp required to reverse this stage

**Mechanisms of Action of GA’s**
• assumed no one anaesthetic receptor
• potency of anaesthetic effect strongly correlated with lipid solubility
  \[\text{very lipid soluble} \Rightarrow \text{very potent}\]
• MAC =
  o minimal alveolar concentration to prevent movement to standardised surg stimuli in 50% of people breathing 100% oxygen
  o inverse correlation between lipid solubility and dose (MAC)
• Awake MAC = concentration in alveolar which permits voluntary response to command in 50% of patients
  \[\downarrow \text{approximately 1/3 MAC}\]
• any GA has narrow band of conc at which LOC

**Factors Effecting MAC**
• Increasing MAC:
  o Young
  o Chronic alcohol abuse
• Factors decreasing MAC:
  o Elderly
  o N20, sedatives, analgesics
  o ↓bp
  o ↓temp
  o low brain sodium
  o pregnancy
Membrane Theory

- an anaesthetic agent dissolves into hydrophobic sites on the CNS nerve cell membrane & expands these sites
- \( \Rightarrow \) ↓ nerve conduction by physical disruption of channels permitting ion transport across membrane
- anaesthesia depends on concurrent list of factors
  - membrane site sufficiently expanded
  - no. of molecules of agent in membrane
  - partial pressure of anaesthetic in tissues
  - p.p. of anaesthetic in blood
  - alveolar p.p. of anaesthetic
- alveolar p.p. of anaesthetic determines the CNS p.p. & onset of anaesthesia

Targets for GA Actions

- theories
- are protein targets which are important
- best theory of GA action is modulate transmitter gated ion channels
- 3 main targets:
  - GABA\(_A\) receptors –
    - at synapses & extrasynaptic receptors
    - GA binding \( \Rightarrow \) opening Cl channels \( \Rightarrow \) ↑ depressant action of GABA
  - K channels – 2 pore domain channels
    - opening of these mediates effects of some volatile GAs
  - NMDA receptors
    - mediate slow components of synaptic transmission
    - inhibited by most inhalational GAs
- other possible targets:
  - glycine receptors
  - cyclic nucleotide-gated cation channels
  - presynaptic Na channels
- overall effect of GAs is LOC by
  - ↓ ing excitatory neurotransmitters:
    - ACh – nicotinic
    - 5HT
    - glutamate
    - NMDA
  - ↑ ing inhibitory neurotransmitters:
    - GABA
    - glycine
- interacting with
  - peptidergic transmission:
  - opioid receptors
  - NO-cGMP transduction pathway
  - ROS
- sensitive areas of CNS:
  - sensory pathways thalamus - cortex \( \Rightarrow \) potentiation of sleep & LOC
  - hippocampus \( \Rightarrow \) amnesia of GA
  - multiple molecular targets in spinal cord \( \Rightarrow \) immobility
Pharmacokinetics

- conc of anaesthetic in lung/blood needs to rapidly equilibrate with CNS levels
  \[\therefore\ \text{depth of anaesthetic depends on partial pressure or conc of drug in brain}\]
- variables involve:
  - high inspired anaesthetic concentration
  - high alveolar ventilation
  - oil-gas partition coefficient = solubility of agent in lipids
  - low blood-gas partition coefficient = solubility of agent in blood & tissues
  - low cardiac output
  - 2^nd gas effect with N2O
- general rules of GA pharmacokinetics:
  - high lipid solubility \(\Rightarrow\) ↑potency
  - high lipid solubility delays recovery:
    - agent forms depot in fat tissues = 2 compartment pharmacokinetic model
    - take hrs to be cleared – hangover effect
  - high blood-gas partition coefficient & high CO \(\approx\) longer time for equilibrium of gas to tissues
    - if agent is highly soluble and large CO \(\Rightarrow\) agent washed away from alveolar \(\Rightarrow\) longer time for alveolar partial pressure of agent to build \(\because\) tissues would be receiving a lot of anaesthetic but at a low partial pressure
  - low blood-gas partition coefficient \(\approx\) faster equilibration of agent \(\therefore\) quick onset and recovery time
  - alveolar ventilation = most impt factor in equilibration of gas agent into blood
    \[\text{esp if have high blood solubility}\]
  - low blood flow to fatty tissues \(\Rightarrow\) slow equilibration of drugs to them
  \[\therefore\ \text{optimal agent = low blood & tissue solubility with high lipid solubility (potency)}\]
  \[\text{eg sevoflurane}\]
  - NO = rapid but weak (low blood solubility but less lipid solubility). Cannot produce anaesthesia alone except in hyperbaric conditions
  - ether = slow but potent (high blood solubility but very lipid soluble)
Elimination
- routes of elimination:
  - exhalation (most)– esp for agents low blood-gas partition coefficient
    - eg desflurane faster than sevo
    - NO not metabolised at all
  - Hepatic metabolism – halothane (20%), des 0.02%

Halothane Hepatitis
- Mild form =
  - Common
  - Direct hepatocellular damage
  - Norm of no clinical consequence
- Fulminant form:
  - Immune reaction to reactive metabolite of halothane via reductive pathway
  - Risk factors:
    - Repeated halothane exposure
    - Hypoxia
    - Obesity
    - Concomitant drugs inducing liver enzymes
- Other inhalational agents can also cause but halothane most severe

Systemic Effects of Inhalational Agents

Cardiovascular
- All [except halothane]:
  - myocardial depression
  - vasodilation
- halothane:
  - tachycardia in face of decreased vascular resistance
    - due to sensitising myocardium to catecholamines ⇒ fatal ventricular arrhythmias (esp with hypoxia & light anaesthesia)
  - desflurane – pungency of smell ⇒ sympathetic stimulation

Respiratory
- All:
  - ↓ Vt
  - ↑RR
  - ↓ response to hypoxia and ↑CO2
  - bronchodilators
- desflurane:
  - airway irritant
  - if high concentrations too early ⇒ laryngospm & bronchospasm

CNS
- all [except halothane]:
  - dose dependant depression EEG
  - ↓cerebral vascular resistance
  - ↓cerebral metabolic rate of O2 consumption
  - ↑cerebral blood flow
  - ↑ICP
• enflurane & sevoflurane = assoc with epileptiform activity
  ↓ should not be used in epileptics

**Other**
• all:
  o mm relaxation
  o potentiate neuromuscular blockers
  o N&V 1:4
  o Uterine muscle relaxation

**Adverse Effects & toxicity of GA**
• common SEs:
  o post op convulsions
  o headache
  o N&V
  o kidney/liver toxicity
  o hepatotoxicity – esp with chloroform & halothane
  o malignant hyperthermia

**Drug Interactions**
• anticoags eg hep/warf: stopped 6/24hrs prior to surg
• CNS depressants eg alcohol, antiHs, antianxiety, opioids, sedatives:
  o all ↑ CVS, resp & CNS depressant effects of GA
  o reduce GA dose as required
• antiarrhythmics: may ↑ CVS depression & hypotension from GA
• Ca & β blockers: ↑ CVS depression & ↑ arrhythmias. ↓ GA
• chronic steroids: adrenal suppression ⇒ ↓ bp during surg due to lack stress response. ↑ steroids
• inhibitors of CYP3A4 eg azole antifungals, protease inhibitors, macrolides:
  o inhibit metab of midazolam ⇒ ↓ midaz dose
• drgs which affect bp or HR: interact with ketamine which ↑ s bp & HR

**Special Considerations**
• young:
  o halothane & NO commonly used as incidence of hepatitis low in kids
  o neonates more sensitive to non-depolarising mm relaxing agents
• old:
  o ↑ ed and longer drug effect
• preg & childbirth:
  o lipid solubility means drugs will cross placenta
  o careful monitoring of drugs
  o avoid GA if possible
  o epidural with lignocaine & fentanyl
• obesity:
  o obtaining desired depth anaesthesia & mm relaxation may be difficult
  o highly fat soluble anaesthetics should be avoided
• smoke: post op complications x 6 more common
• high alcohol:
  o liver/stomach/pancreas problems
Premedication

- no longer essential as less use of ester & chloroform
- some uses still:
  - ↓ anxiety ⇒ ↓ GA doses needed eg opiates, benzos
  - ↓ secretions eg salivary, gastric, bronchial eg anticholinergics atropine
  - ↓ post op vomiting eg phenothiazines ie prochlorperazine, promethazine
  - prophylactic analgesia & sedation eg opioids, benzodiazepines, phenothiazines

Inhalational Anaesthetics

- gases or volatile liquids
- rapid reach conc in blood & brain
- following chars:
  - complete anaesthesia ⇒ abolish superficial & deep reflexes
  - controllable anaesthesia – depth can be varied quickly
  - lung function critical to administration & excretion
  - may not have analgesic action
  - rapid recovery with removal of drug
  - allergic reactions uncommon

Volatile Liquid Anaesthetics

- ether & chloroform first used
- halothane – assoc with hepatic failure
- now use halogenated hydrocarbon series eg sevoflurane

Nitrous Oxide

- simple inorganic molecule N₂O
  - NB not NO (nitric oxide)
- at room temp = vapour, >36.5 = gas

MOA

- 2 main actions:
  - analgesic action similar to opioids ? mediated by opioid receptors
  - anxiolytic action: ⇒ enhanced GABA mediated CNS depression

Pharmacokinetics

- inhaled & absorb by lungs
- 2nd gas effect: rapid uptake into blood from alveoli ⇒ ↑ concentration of other agent in alveoli ⇒ ↑ more rapid onset anaesthesia
- low solubility in blood & tissues ⇒ rapid onset and offset
- 100% excreted unchanged via lungs

Uses

- powerful analgesic
- useful anxiolytic
- weak anaesthetic – MAC value 105
  - often used as carrier gas with O₂ for other volatile anaesthetics to enhance effects in major surgery
- eg entonox 50:50 O₂:N₂O
**Adverse Reactions**
- non irritant with no odour
- Primary probs
  - Mild ↑CBF & cerebral O2 consumption ⇒ ?avoid in neuroanaesthesia
  - Diffusion hypoxia:
    - at termination of gas administration rapid movement of N2O from circ into lungs
    - occurs faster than N2 can move back into blood
    - may dilute O2 in lung
    - avoid by 3-5mins 100% O2 cover this period
  - Expansion air filled spaces:
    - N2O is x40 more soluble than N2
    - If N2O containing blood perfuses tissue adjacent to air filled space ⇒ N2O diffusion faster than N2 returning from air ⇒ expansion
    - Problem in bowel & PTx & head injuries
- Haematopoetic System
  - N2O alters valency of central cobalt atom of vit B12
  - Prolonged exposure N2O ⇒
    - altered DNA synthesis,
    - megaloblastic & suppressed bone marrow,
    - subacute combined degen of spinal cord
- Pollution:
  - N2O + UV light ⇒ free radicals ⇒ ozone break down
  - Chronic effects on health care workers:
    - Fatigue, malaise
    - abortions, marrow suppression, teratogenicity
- post op nausea & vomit
- safe in pregnancy

**Cautions/Contraindications**
- altered mental state
- recent scuba
- v cold conditions (<-6deg)
  - gases may separate
- Severe pulmonary disease may alter elimination of NO

**Interactions**
- nil

**Dose**
- GA:
  - induction 70:30 N2O:O2
  - maintenance 30:70 N2O:O2
- obstetrics: entonox 50:50
- dental procedures 25:75% mixture

**Sevoflurane**

**Compound A**
- = vinyl ether produced by degredation of sevo
- in rats shown to produce ATN
- debate about effect in humans but likely little clinical effect
• compound A production
  o directly related to
    • sevo concentrations
    • absorbent temp
  o inversely related to fresh gas flow rate (FGF)
• manufacture recommends sevo not used in FGF <1L/min and for no longer than 2 MAC hours
• for anaesthetic >2hrs FGF should be at least 2L/min

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<th>Characteristics that influence the choice of an agent</th>
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<td><strong>Agent</strong></td>
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* Although hepatitis and coronary steal are classically associated with halothane and isoflurane, the problems are described with all inhalational agents.
Intravenous Induction Anaesthetics

- major gps:
  - ultrashort acting:
    - barbituates – thiopentone
    - non-barbituates – propofol & ketamine
  - midazolam – actually a benzo but has benefits & common adjunct
- benefits of IV anaesthetics:
  - rapid onset
  - controllable
  - amnesic effects
  - ↓ amount of inhalational agent required
  - prompt recovery with small doses
  - no risk of explosion
- disadvantages of IV anaesthetics:
  - minimal mm relaxation & analgesic properties
  - subject to liver & renal excretion
  - common hypersensitivity reactions
  - tissue reactions if extravasation
  - hypotension/laryngospasm & resp failure a risk

Pharmacokinetics

- high lipid solubility ⇒ high potency & rapid onset
- short duration of action as drug quickly redistributed into fat deposits
- 2 compartment distribution of drug:
  - obese people have shorter effect of single IV dose
  - saturation of fat ⇒ prolonged action of drug as drug slow release back into circulation

Pharmaceutics

- 2 problems:
  - need high lipid solubility ⇒ to cross bbb
  - water soluble to be formulated as a solution for safe IV injection
- formulating as oil in water emulsions (milk)
- propofol in soya oil/egg lecithin/glycerol emulsion

Total IV Anaesthesia

- GA using only IV anaesthesia & no inhalational drugs
- bolus dose then maintenance
Ultrashort Barbituate – Thiopentone

- CNS depressant produces hypnosis & anaesthesia without analgesia
- combine with mm relaxant & analgesia

MOA
- suppression of RAS

Pharmacokinetics
- high lipid soluble ⇒ rapid onset
- slow metabolism as moves out of adipose tissue slowly

Uses
- good emerg anaesthesia drug:
  - anticonvulsant properties
  - ↓ICP

Adverse Reactions
- serious:
  - ↓CO (↓SV) & ↑venous capacitance ⇒ ↓bp
  - cardiac arrhythmias
  - emergence delirium – excitability, confusion, hallucinations
  - resp depression
  - allergy
- during recovery:
  - shivering & trembling
- prolonged fatigue & headache

Non- Barbituates: Propofol

MOA
- rapidly acting non barbiturate hypnotic
- formulated in an emulsion for IV use
- no analgesic properties
- MOA not known- ?CNS depression via GABA receptors

Pharmacokinetics
- rapid onset of action – 40seconds
- duration of effect 3-5mins
- majority liver metab +/- extrahepatic metabolism
- almost completely metabolised to glucuronide
  \(^{\text{inactive metab; half life 3-8hrs}}\)

Uses
- induction & maintenance of GA
- PSA

Adverse Reactions
- resp & CVS depressant:
  - apnoea
  - bradycardia & ↓bp
- N&V
- involuntary mm movement common

Cautions/Contraindications
- pain & thrombophlebitis on injection
potentially for abuse

Interactions
- sedative effects of other CNS depressants ↑
- no other sig interactions

Dose
- IV dose 2-2.5mg/kg
- PSA 0.5-2mg/kg

Somatic NS
- aka voluntary ns
- primary motor area of cerebral cortex initiate voluntary movement
- impulse through UMN which decussate in medulla oblongata
- UMN terminate in ant grey horn of spinal cord at each spinal segment
- often interneurons which then connect to LMN
- LMN = final common pathway which connects CNS to skeletal mm

Targets to Block Neuromuscular Transmission
- incl:
  - block AP generation in motor neuron
  - inhibition of release of Ach
    - eg botox
  - inhibition of breakdown of Ach
  - blockade of postsynaptic receptors

Neuromuscular Junction
- @NMJ motor neuron divides into cluster of synaptic end bulbs containing Ach
- Ach released on arrival AP ⇒ diffuses cleft ⇒ postsynaptic nicotinic receptors on end plate
- NMJ norm in centre of mm fibres
- impulses radiate out from NMJ over mm
- action of Ach rapidly terminated by AChE (acetylcholinesterase) which attached to collagen fibres

Motor End Plate Nicotinic Receptors
- receptor 5 subunits with ion channel in centre:
  - α x2
  - β
  - δ
  - ε epsilon
- bulk of receptor faces extracellularly
- 2 molecules of Ach bind onto each α subunit ⇒ channel opens ⇒ Na flow through ⇒ depolarisation end plate ⇒ contraction
Neuromuscular Blocking Drugs

- 2 types:
  - competitive or non depolarising drugs:
    - block action of Ach at
      - postsynaptic nicotinic
      - presynaptic nicotinic ⇒ blocks normal feedback loop which ⇒ ↑Ach under conditions of enhanced stimulation
    - action can be reversed by anticholinesterase
      - eg pancuronium, curare
  - depolarising drugs:
    - nicotinic receptor agonists ⇒ maintain depolarised state of motor end plate .⇒ no further APs
      - eg suxamethonium

Non Depolarising Blocking Drugs

- rapid blockade with motor weakness ⇒ total flaccid paralysis
- small muscles (eye,jaw) ⇒ limbs ⇒ trunk ⇒ diaphragm
- recovery is generally opposite order
- can cause histamine release from mast cells:
  - flushing & rash ⇒ anaphylactic reaction
  - not due to receptor action but acidic nature of drug
  - risk varies inbetween drugs

Pancuronium

MOA
- non depolarising competitive blockade of nicotinic receptors at motor end plate
- interruption requires >70% of N receptors; blockade >95%

Pharmacokinetics
- wide volume of distribution within 5mins post injection
- highly water soluble .⇒ urinary excretion begins immed
- clearances:
  - 25% renal unchanged
  - rest hepatic metab ⇒ bilary excretion
- half life 30mins

Uses
- adjunct to GA for surgery/ICU

Adverse Reactions
- slight ↑HR, ↑CO, ↑bop
- ↑intragastric pressure ⇒ risk of vomiting
- anaphylactoid reaction small risk (1 in 10,000)
- histamine release

Cautions/Contraindications
- care in:
  - HTN
  - liver/kidney failure

Interactions
- additive effect with:
  - inhalational anaesthetics
By Adam Hollingworth

- sux
- aminoglycosides (also cause blockade themselves)
- benzo’s
- Ca channel blockers
- lithium
- propanolol
- ↓ effect with:
  - adrenaline
  - carbamazepine
  - anticholinesterase agents eg neostigmine
  - high dose steroids
  - Ca, Na, K salts

Dose
- initial 0.04-0.15mg/kg in adults & children >1month
- maintenance dose 0.01-0.02mg/kg

Post Op Reversal
- sugammadex
  - modified cyclodextrin
  - forms a complex with neuromuscular blocker ⇒ ↓ binding to nicotinic receptors
  - rapid effect within 5mins (compared to 50min effect of neostigmine
  - SEs: taste sensations & allergic reactions
  - interacts with some drugs – fluclox, progesterones (take extra contraceptive precautions)

Depolarising Blocking Drugs
Suxamethonium

MOA
- agonist of N receptors on motor end plate
- ⇒ persistent stim & maintenance of depolarisation of MEP
- Na channels remain open .∴ no further response to elec stimulus
- during onset of action see mm fasciculation's:
  - as each MEP is depolarised ⇒ local AP to motor units without total mm contraction
  - x1 fasciculation/Motor unit then blockade
- short acting mm relaxant
- reversal by anticholinesterase not possible:
  - will prolong depolarisation

Pharmacokinetics
- rapid onset of action
- half life 2-4mins
- blockade persists for ~10mins
- hydrolysed by butyrylcholinesterase (aka pseudo-cholinesterase) to
  - choline
  - succinyl monocholine ⇒ hydrolysed to choline & succinic acid
  - if atypical pseudo-cholinesterase see extended blockade

Uses
- brief mm relaxation eg
  - ECT
tracheal intubation
surg procedures

Adverse Reactions
(M myalgia
A apnoea
R raised ICP & IOP
K hyperkalaemia
E vent Ectopics & bradys
T MH
& ↑gastric pressure and ↑salivation)

- profound & complex effects on CVS system:
  - bradycardia
  - tachy/arrhythmia’s
  - HTN
  - cardiac arrest
- ↑ICP
- ↑Intra-ocular pressure – avoid in eye surg if anterior chamber needs to be opened
- ↑gastric pressure ⇒ vomit risk
- ↑serum K:
  - release of K from MEP
  - caution in burns & massive trauma
- malignant hyperthermia – mm spasm & rapid rise in body temp
- low pseudo-cholinesterase levels ⇒ prolonged mm paralysis
  - seen with liver disease
  - anticholinesterase drugs inhibit pseudo-C action
- anticholinergic effects ⇒ excessive salivation:
  - muscarinic like action of sux
  - prevented by atropine

Cautions/Contraindications
- care if:
  - electrolyte disturbance
  - low pseudo-C levels
  - renal disease
  - digoxin
- contraindicated:
  - malignant hyperthermia or FH
  - extensive burns or multiple trauma

Interactions
- additive effect (many):
  - lignocaine
  - non penicillin Abx
  - βblockers
  - lithium
  - steroids
  - metoclopramide - ↓s inactivation of sux ⇒ prolonged NMJ blockade

Dose
- 1mg/kg loading; maintenance 0.5mg/kg
- IV or IM
- not to conscious person
Anticholinesterase Agents

- AChE (acetylcholinesterase) hydrolses Ach ⇒ choline & acetate
- enzyme bound to postsynaptic membrane
- active site of enzyme contains 3 amino acids:
  - serine
  - histidine
  - glutamate = anionic site
- 2 broad categories of drugs:
  - short acting eg donepezil
    - bind reversibly to anionic site
    - eg alzhemiers
  - medium acting eg neostigmine, pyridostigmine:
    - bind to both anionic & esteratic sites
    - hydrolysed more slowly
    - eg myasthenia & alzheimers
  - irreversible:
    - bind to esteratic
    - eg pesticides & chem. warfare

Neostigmine

MOA
- reversible inhibitor of AChE
- forms a carbamylated enzyme at active site
- complex slowly hydrolysed by AChE over 3-4hours

Pharmacokinetics
- poorly absorbed from GI tract
- doesn’t cross bbb
- plasma half life 0.5-1.5 hours
- excretion:
  - faeces >50%
  - urine 30%
- metab by plasma cholinesterases
  - liver disease has no effect on drug

Uses
- used for reversal of non depolarising competitive NMJ blockers eg pancuronium
- Rx myasthenia gravis

Adverse Reactions
- best seen in overdose situation which ⇒ cholinergic crisis ie ↑↑Ach action at synapses:
  - NMJ – fasciculation's, weakness, paralysis, depressed vent
  - postganglionic parasympathetic synapses:
    - salivation, tears, ↑Gi & bronchial secretions, ↑bowel activity
    - bronchoconstriction
    - brady & hypotension
    - constricted pupils
    - D&V & urination
- CNS –
  - stim ⇒ depression with larger doses
  - irritability
  - ataxia, fatigue, amnesia
  - ↓GCS & resp depression
- CVS –
  - reflex ganglionic & postganglionic effects of Ach accumulation
    - initial – excitation
    - later – ganglionic blockade through persistent depolarisation . inhibitory
  - ↑parasymp vagal tone ⇒
    - bradycardia
    - ↑refractory period & conduction time SAN/AVN

Cautions/Contraindications
- care in
  - asthma
  - heart disease
  - ↓bp
  - peptic ulceration

Interactions
- ↓effect:
  - steroids
- any drugs with anticholinergic activity will ↓effect of neostigmine & vice versa

Dose
- reversal of NMJ blockade 50-70mcg/kg to max 5mg over 1min
  - give after or with atropine 0.6-1.2mg

Local Anaesthesia
- ideal LA
  - target sensory nerves only
  - rapid reversible
  - non toxic
  - rapid painless onset
- 2 commonest used =
  - lignocaine
  - bupivacaine
- rapid evaporation ⇒ cooling can provide similar LA effect
  - eg ethyl chloride
- = membrane stabilisers or ion channel modulators

Chemistry of LAs
- generally have
  - aromatic (phenyl gp) at one end:
    - makes this end lipid soluble
  - amine (nitrogen containing) gp at other end
    - makes this end hydrophilic
  - joined by intermediate chain of carbons
- different solubilities at either end of molecule allows chemical to align & act in nerve cell membranes
intermediate carbon chain contains a link which defines subgroup of molecules; either:
  • ester link CO-O
    • = ester LAs - cocaine, procaine, amethocaine
    • metab’ed rapidly by plasma esterase enzymes ⇒ PABA metabolites
    • PABA metab’s responsible for allergic reactions
    • less common to use this gp
  • amide link CO-N
    • eg lignocaine, prilocaine, bupivacaine
    • not metab’ed to PABA metabolites . ∴ allergy less common

all LAs are amines ∴ can exist as either: (R=any radical)
  • uncharged amine form (NR₃)
  • charged quaternary amine form (N⁺R₃H)

move in equilibrium: H⁺ + NR₃ ⇔ N⁺R₃H

balance of equilibrium depends on:
  • chemistry of individual LA drug
  • pH of solution

extent of ionisation determined by pH of environment:
  • strength of acid = tendency to dissociate into H⁺ & anions
  • dissociation defined by pKa:
    ⇔ = pH at which half the chemical is in its ionised form (pH – pKa = 0)
  • degree of unionised depends on
    ⇔ whether drug is
      • acid: if pKa < physiological pH (7.4) = <50% unionised
      • base: if pKa < 7.4 = >50% unionised (all LAs are bases)

curves drawn differently for acid/base drugs— for all LA’s:
MOA

- enter cell by diffusion through membranes
- bind to modulatory site in voltage dependant Na channel ⇒ block it by preventing transient opening
- ∴ threshold potential not reached ⇒ not depolarisation & no AP
- LAs effect all membranes eg ANS, motor nerves, mm cells, CNS neurons
- Susceptibility of nerve to LA depends on (better):
  - fibre diameter & myelination – order of block (first to last):
    - small myelinated
    - unmyelinated (C)
    - large myelinated (A delta)
  - tissue pH (physiological – alkaline)
  - length nerve fibre
- ∴ autononmic & sensory fibres effected first – thinner & unmyelinated
  - motor fibre can be effected with big enough dose
- sequence of anaesthesia:
  - loss pain
  - loss temp sens
  - loss proprioception
  - loss touch/pressure

Pharmacokinetics

- injection ⇒ local dispersion
- onset of action determined by movement into nerve cells which determined by:
  - lipid solubility which depends on
    - pH tissue
    - degree ionisation of LA molecules (function of pKa of drug)
- protein binding & vasoconstrictor in solution help to retain drug in tissue for longer
- local action terminated by:
  - diffusion away
  - dilution & uptake into vessels
  - determined by lipid solubility & %VC present in solution
  - bupivocaine have a longer duration of action#
  - as ↑ed lipid solubility & ↑protein bound

Comparisons

- short acting: (30-60mins)
  - procaine:
    - least toxic LA
    - low lipid solubility ∴ slow onset
    - potency 0.5
  - intermediate acting (30mins- 4hrs)
    - lignocaine:
      - potency 1
      - more cardiotoxic than prilocaine
    - prilocaine:
      - products of liver metab may ⇒ methaemoglobinuria
• EMLA (lignocaine/prilocaine)
  • local irritation
  • toxic if swallowed
  • <6months risk of metharmoglobinemia
• long acting (3-10hrs):
  o bupivacaine:
    • potency 4
    • ↑cardiototoxic than lignocaine
    • slow onset
    • less motor blockade
  o amethocaine (tetracaine):
    • topical LA
    • potency 5
    • slow onset

Vasoconstrictors
• most LAs ⇒ VD by
  o direct action on blood vessels
  o action on sympathetic VC nerve fibres ⇒ VD
• risk of rapid systemic absorb: if absorb>rate of elimination then toxicity
• ∴ adrenaline or phenylephrine ⇒ VC ⇒ drug stays local longer
  ←α adrenoceptor agonist in nasal spray with lignocaine

Toxicity
• order of toxicity (less > more):
  o procaine > prilocaine > lignocaine > bupivocaine > amethocaine > cocaine
• reactions:
  o specific to drug eg prilocaine = metHb ⇒ cyanosis
  o allergies eg bronchospasm & anaphylaxis (more common with esters)
  o systemic effects of LA:
    • numb tongue
    • CNS stim: tremor, visual disturbance, convulsions
    • CNS depression: relax smooth mm & skel mm; CVS/resp depression, ↓bp

Reversal
• recovery of sens can be accelerated with phentolamine:
  o α receptor antagonist
  o infiltrate into same site as LA ⇒ VD ⇒ ↑clearance of lignocaine
  ←good in dental surg

Types of Block
• epidural:
  o injection into extradural space between dura & lig flavum
  o space filled with loose adipose & lymph & blood vessels
  o injection C7-T10
  o injection stays local to level
  o post op urinary retention common 2nd to block of parasymp nerves
• spinal anaesthesia
  o injection into CSF in subarach space
below spinal cord level ie >L2
onset action 1-2mins
duration 1-3hrs
specific gravity of LA & position of pt is important to prevent LA rising though spinal cord
SEs:
  • include hypotension; ↓CO; resp depression 2nd to depression symp pathways & medullary centres
  • Rx with sympatomimetics eg ephedrine & metaraminol

Lignocaine
MOA
• amide –type LA
• blockade of Na channel ⇒ prevents initiation & propogation of nerve impulses
  →also stabilises all potentially excitable membranes incl heart
Pharmacokinetics
• rapid onset action 5-10mins
• Peak blood level usually occur 10-25min post injection
  →when toxicity most likely to occur
• duration blockade 1-1.5hrs
• once absorb into gen circulation rapid redistribution to all tissues esp heart
• large 1st pass metab in liver – CP450 hydrolyses amide link
  →why cant take orally
• excretion via kidneys - <10% unchanged
• half life 90-120 mins
Uses
• LAf
• Rx or prevent ventricular arrhythmias
•
Adverse Reactions
• toxic depressant effects in CNS/ANS/PNS & CVS & resp systems if:
  • ↑↑dose
  • rapid absorb
  • delayed elimination
• allergy rare
Cautions/Contraindications
• ↓dose: children, elderly, CVS, Neuro, hepat-renal disease
• contraindicated if:
  • infection at site injection
  • severe shock
  • hypotension
  • SVTs
Interactions
• other anti-arrhythmics/phenytoin/alcohol ⇒ ↑CVS effects of lignocaine
• ↓clearance of lignocaine:
  • β-blockers
  • cimetidine
  • erythromycin
Dose
• lowest effective dose
• Max safe dose 3mg/kg (adults & child) (6mg/kg with adrenaline)
  
  Thus 70kg man = 210mg.  1% contains 10mg/ml thus 20mls contains 200mg
  2% contains 20mg/ml thus 10mls contains 200mg

• anti-arrhythmic dose: not more 300mg/1hr