# AWSOME

Anaesthesia Written ShOrt answer & Multiple choice Examination Course

# Cardiothoracic Anaesthesia Masterclass

# Background

- Cardiothoracic Anaesthesia is a Mandatory Unit of Training

- A question on Cardiothoracic Anaesthesia will appear in each SAQ paper

- The RCOA acknowledges that not all exam candidates will have significant experience of this sub-specialty

- Whilst these questions are often rated 'hard' (i.e. pass mark of 10-11 out of 20) – they are often based around basic principles

# Cardiac Questions

- May 1999 – Principles of CPB
- Oct 2001 – Heart transplant patient
- Oct 2002 – CPB complications
- May 2003 – Aortic Stenosis
- Oct 2003 – Thoracic Epidural for CABG
- Oct 2006 – CVS risk evaluation
- May 2007 – ICDs
- Oct 2008 – Congenital Heart Disease
- May 2011 – neurological complications of CABG
- Sept 2012 – Off-Pump CABG
- Sept 2013 – DC Cardioversion and Cardiac Tamponade
- Mar 2014 – ICDs
- Sept 2014 – Cardioplegia solutions and CHD
- Mar 2015 – Anticoagulation for CPB
- Sept 2015 -– neurological complications of CABG
- Mar 2016 – Aortic Stenosis
- Sept 2016 – Cardiac Tamponade

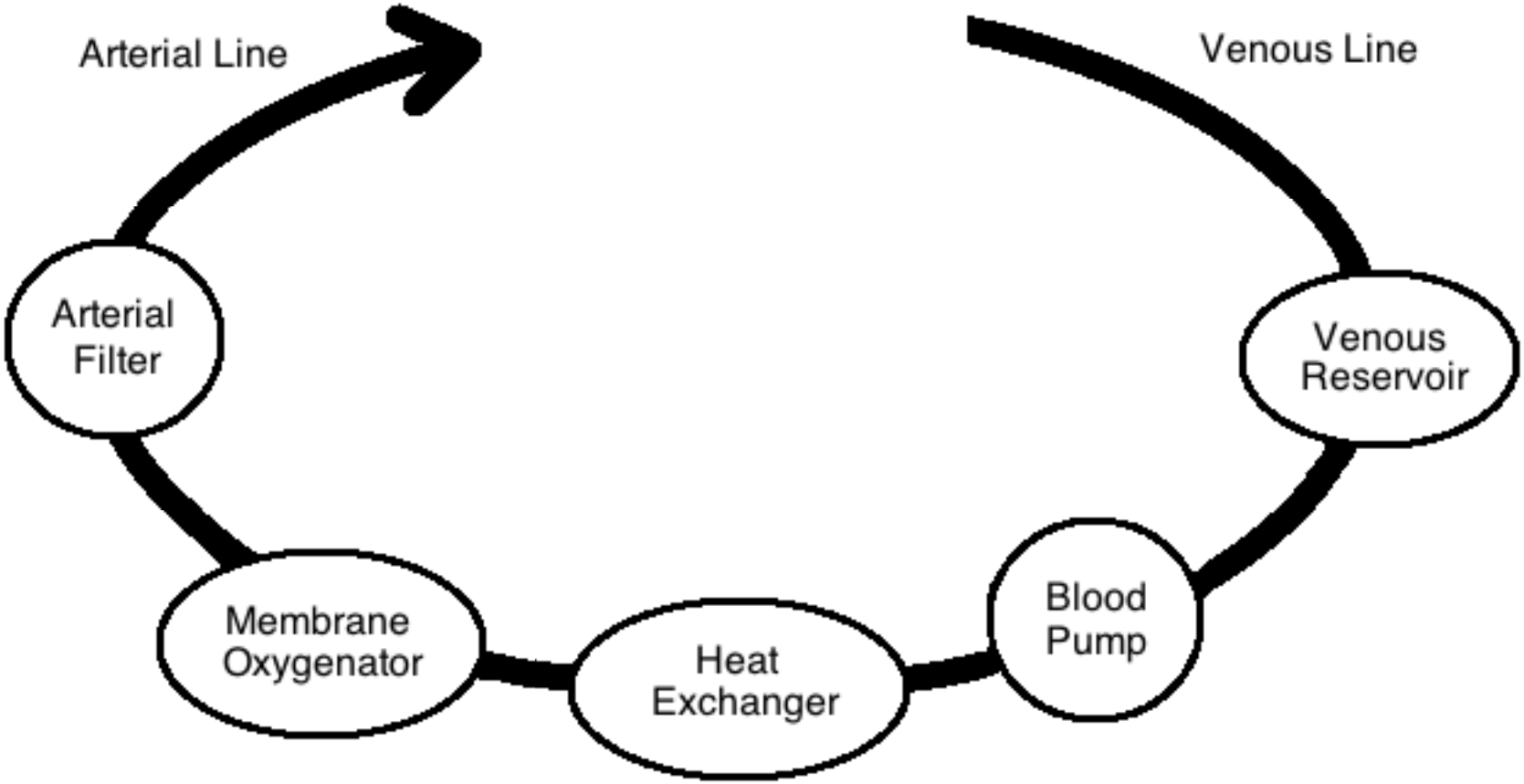
# Thoracic Questions

* Nov 1998 – Bronchoscopy & Inhaled FOB
* May 2004 – Thoraco-abdominal Oesophagectomy
* Oct 2005 – OLV / Pre-op Assessment
* May 2006 – OLV / Pre-op Assessment
* Sept 2009 - OLV / Pre-op Assessment
* Sept 2012 - Endoscopic Thoracic Sympathectomy
* March 2013 - OLV / Pre-op Assessment
* March 2014 – Rigid bronchoscopy & laser tumour resection

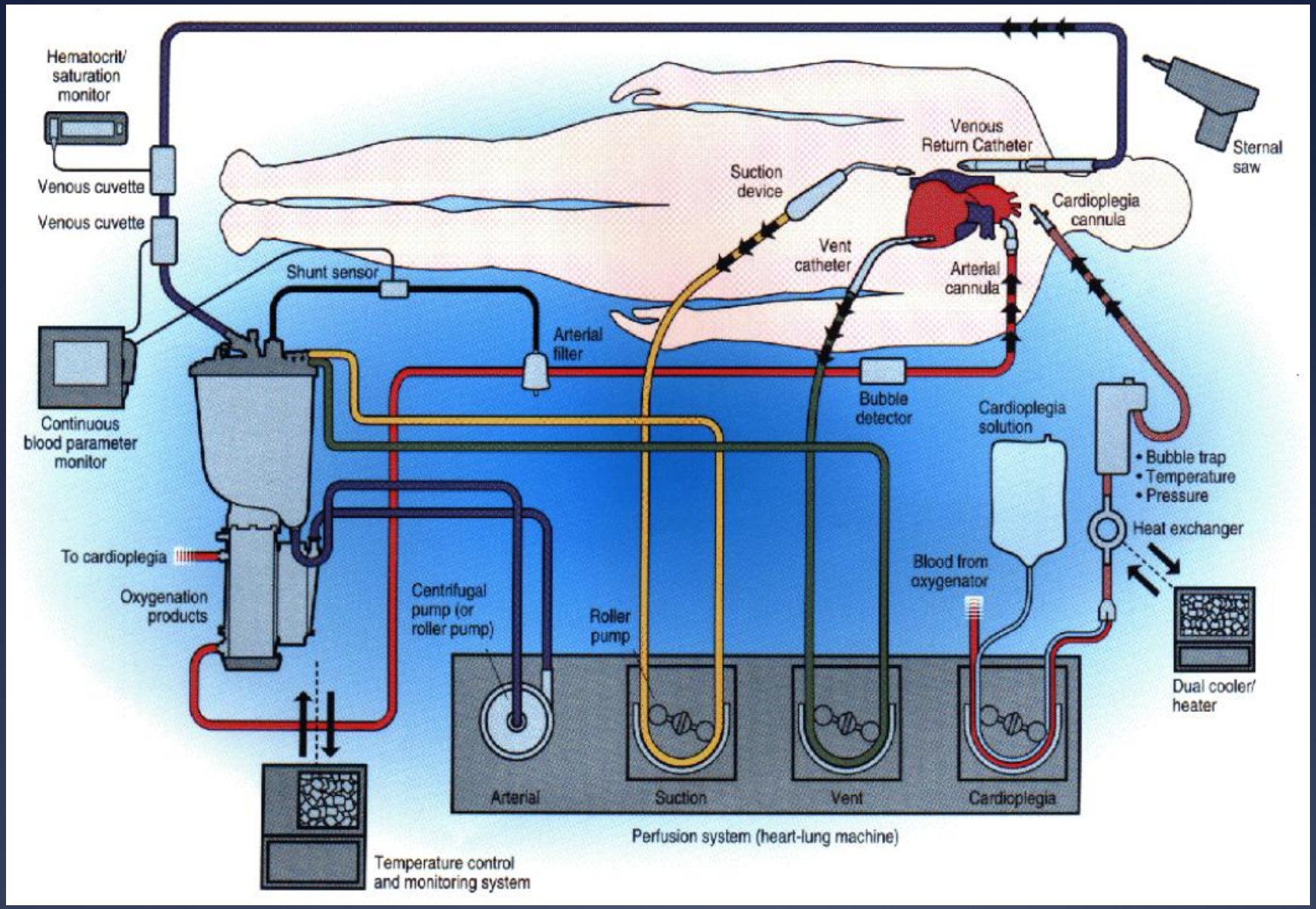
# Topics to Cover

- Cardiopulmonary Bypass
- Off-Pump Surgery
- Thoracic Risk Assessment
- OLV

# Cardiopulmonary Bypass



# Cardiopulmonary Bypass



# Starting on CPB

- Patient needs to be fully anticoagulated
  - Activated Clotting Time of >400Secs needed before aortic cannulation
  - Normal ACT = 105-160secs
  - Heparin Dose = 300IU/Kg
- Process
  - Systolic BP of 80-100mmHg (reduces risk of dissection)
  - Aortic cannulation → blood returned to patient
  - Venous cannulation – RA or VC → blood drained from patient
  - Ventilator turned off
  - Anaesthesia maintained either by IV route or bypass vapouriser
- Bypass Parameters
  - Non-pulsatile flow of 2.4L/min/m2
  - MAP of 50-70mmHg
  - Haematocrit 20-30%
- Perfusionist
  - Checks ABG and ACT every 30mins
- Temperature
  - Allowed to drift to 28-35C
  - Reduces tissue O2 requirements and increases cerebral protection

# Myocardial Oxygen Demand

- Determinants
  - Major = LV wall tension and contractility
  - Minor = HR, Basal metabolism and Ion Homeostasis
- Options
  - Hypothermia
  - Intermittent Cross-Clamping and Fibrillation
    - Cross-clamped → VF induced (tolerated for 10-15mins) → clamps released → shock to sinus
    - Avoids need for cardioplegia and reduces Troponin release
    - Does NOT provide myocardial protection
  - Cardioplegia (e.g. St Thomas' Solution)

# Cardioplegia

- Diastolic arrest – reducing oxygen/energy demand + improved surgical conditions
- Allows for rapid, homogenous cooling & maintains ionic consistency
- Composition
  - K+ = 10-40mmol/L → most important component – makes cardiac cell membrane's resting potential  less negative → inactivating Na+ channels → stopping repolarisation and leaving heart in diastole
  - Others
    - Glucose (or Aspartate/Glutamate)– substrate
    - Local anaesthetic – Procaine or Lidocaine
    - HCO3 (Histidine or THAM) – buffer
    - Mannitol – osmolality / free-radical scavenger
    - Na+, Mg2+, Ca2+
- Risks
  - MI (inadequate distribution) or Arrhythmias (usually CHB)
  - Administration – administration site injury/rupture, Stenosis, air embolus, post-op RV dysfunction
- Two Types - Crystalloid or Blood (theoretical O2 carriage)
- Two delivery sites - Anterograde – coronary arteries & Retrograde – coronary sinus

# Cerebral Injury on CPB

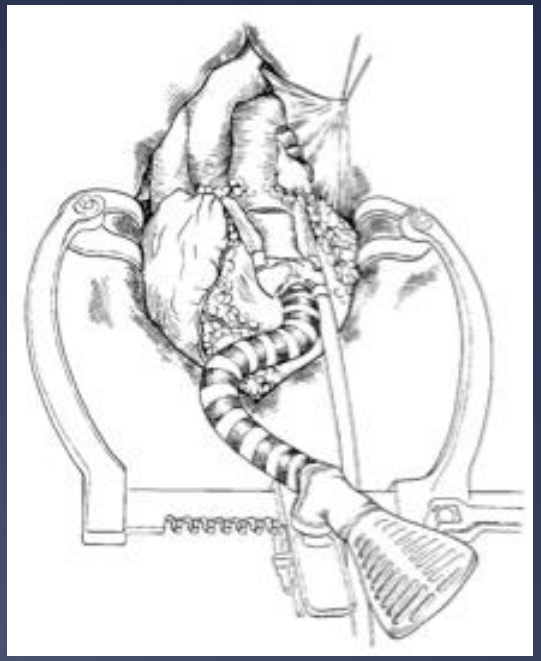
- Injuries are usually due to a) emboli or b) hypoperfusion
- Type of Cerebral Injuries (local or global)
  - Nervous Ischaemia (Cerebral - CVA/TIA, Spinal – paralysis or Peripheral - palsy), POCD or Seizures
- Patient Risk Factors – Age, male, existing heart failure, previous CVA, DM, Genetics
- Procedure Risk factors – embolisation, hypoperfusion, long Bypass time, hyperglycaemia, inadequate hypothermia or rapid rewarming, SIRS, etc
- Determinants of Cerebral Blood Flow on CPB
  - **Temperature** = the most important determinant of cerebral oxygen demand. Cooling reduces CBF
  - **Haematocrit =** ↓Haematocrit → ↓viscosity → ↑ CBF
    - Critical Haematocrit = that beyond which cerebral O2 delivery is inadequate.
  - **Arterial Pressure** - MAP of 50mmHg is considered the minimum
  - **CO2** - reactivity is preserved during CPB, therefore CBF can be altered

# Methods of Limiting Cerebral Injury

- Anti-coagulation – reduces embolisation
- Hypothermia – reduces cerebral metabolic demand
  - Also need to avoid hyperthermia on rewarming
- Operative Technique – lower with Off-pump techniques
- Pump Technology – Rollers cause more damage than centrifugal pumps
- Medical Management – treatment of underlying disease
- Glucose control – Hyperglycaemia → increased lactate production → neurotoxic
- Acid-Base Management – cerebral autoregulation better with a-stat (not temp corrected like ph-stat)
- Limiting SIRS response – e.g. Off-pump, or shorter CPB time
- Neuroprotective Agents

# Off-Pump Cardiac Surgery

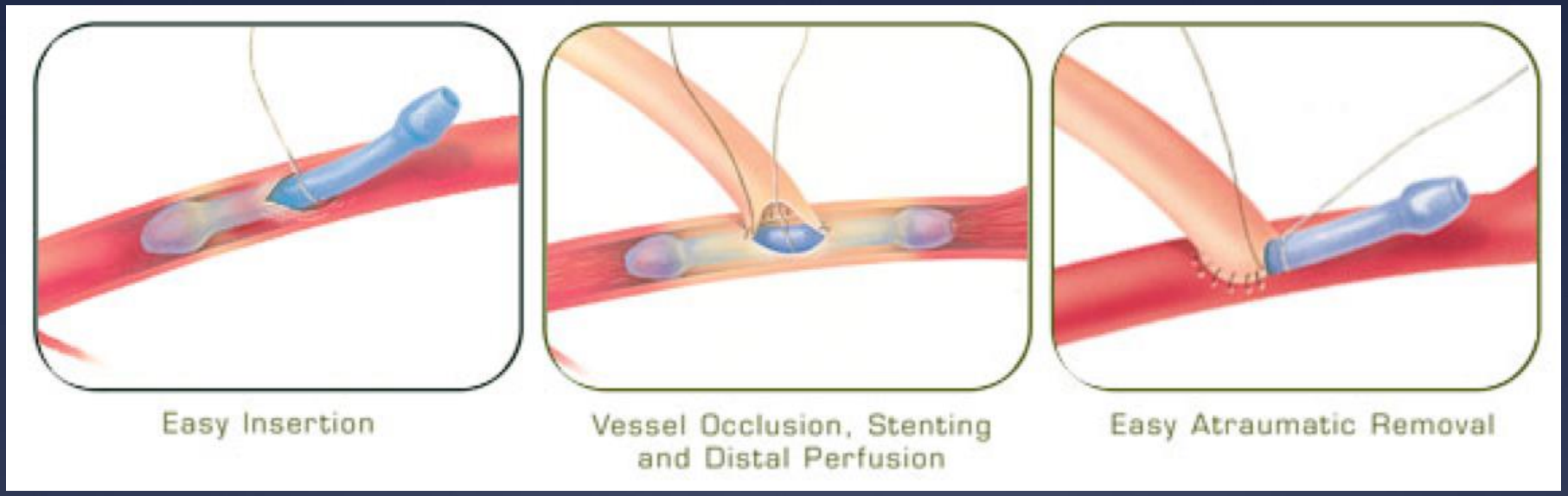
- May be used if a patient will not tolerate CPB
- Advantages – avoids CPB, reduced neurological injury, reduced renal failure, reduced platelet dysfunction
- Disadvantages – still needs anticoagulation, less common technique, 1-10% need to convert to CPB
- Uses a Cardiac Stabiliser
  - Pharmacological – Esmolol, Adenosine or Diltiazem
  - Mechanical
    - E.g. the Octopus Device
    - Pair of suction cups placed parallel to the artery to be grafted → suction applied immobilises the artery



# Haemodynamic Instability Off-Pump

- Causes
  - Displacement of the heart
  - Pressure of retractor on ventricular walls
  - Distortion of Valve annuli
  - Arrhythmias
- Strategies to Minimise
  - Stop surgical manipulation
  - Maintain high perfusion pressure
    - Trendelenburg position, fluids, vasopressors
  - Maintain low myocardial oxygen consumption
    - Avoid excessive tachycardia
  - Other - Avoid electrolyte disturbance, Peri-operative TOE and Intraluminal Shunt Device

# Intra-Luminal Shunt



# Coming off CPB

- = Coordinated effort between anaesthetist, perfusionist and surgeon
- Checklist (Bold = Essential, try to include numbers)
  - Every = Electrolytes normalised (K+ 4.5-5.0)
  - Teenage = Temperature normal again (~37ºC)
  - Boy = Blood products available
  - Dreams = Drugs ready and available
  - ABout = Acid/Base normalised (pH 7.35-7.45)
  - Fondling = Fluids ready and available
  - A = Arterial Transducer at correct level
  - Real = Rhythm, Pacing, Defib available
  - Vagina = Ventilator back on / Adequate PaO2/ PaCO2

# Problems Coming off CPB

- Sometimes weaning off CPB is difficult
- Risk Factors
  - Pre-op
    - Pre-op EF <30%, Drugs – beta-blockers, Ca2+ blockers
  - Intra-op
  - Procedural
    - Prolonged CPB >3hours, Incomplete surgery, Inadequate myocardial protection, prolonged ischaemia
  - Cardiac
    - Preload (too low), Contractility (ischaemia, graft failure, etc), Afterload (too high / too low), Rhythm (brady/tachy)
  - Pulmonary
    - Inadequate FiO2 / Ventilation, Bronchospasm, failure of gas transfer (e.g. pulmonary oedema), Increased pulmonary vascular resistance
  - Metabolic
    - Acidosis, electrolyte abnormality or hypothermia
- Treatment Options
  - Return to CPB
  - Pharmacological – Fluids / Blood / Inotropes +/- Vasodilators/pressors (give examples)
  - Mechanical – Intra-Aortic Balloon Pump, VADs

# Post-CPB

- Anticoagulation must be reversed once off CPB – NOT BEFORE
  - Protamine
- Risks of CPB
  - CVS – hypoperfusion, dissection at cannulation site, embolic events
  - CNS – CVA, POCD, prolonged wakeup
  - Renal - failure
  - Metabolic – hyperglycaemia, hypokalaemia, hypomagnesaemia
  - Haematological – haemolysis, platelet dysfunction, coagulopathy
  - Immunological – increased risk of infection
  - Inflammatory - SIRS

# Thoracic Risk Assessment

**Arterial blood Gas Analysis**

* Resting PaCO2 of >6kPa suggests increased risk of post-op morbidity and ventilatory failure

**Peak Flow**

* If peak flow is <200ml/min coughing is likely to be ineffective

**Predicting Post-op Pulmonary Complications**

* Higher Age / ASA / BMI
* Malignancy

* Smoking within 8weeks of surgery
* COPD
* Abnormal clinical signs / CXR
* Thoracic and upper abdo surgery

**Likelihood of need for post-op ventilation**

- Pre-op DLCO <40% of predicted normal
- Estimated post-op FEV1 <800ml
- Estimated post-op FVC <15ml/kg

**General Criteria for Increased Morbidity / Mortality with Lung Resection**

- FVC < 50% predicted
- FEV1 < 50% predicted or < 2L
- PaCO2 >6kPa

# One-Lung Ventilation I

* Why do it?
  - Allows independent control of each lung
  - Protects dependent lung from contamination
  - Improves surgical access and reduces lung trauma

* Problems
  - More technically difficult and Increased physiological challenge e.g. Shunting

* **Indications**
* Absolute
  * Lung Isolation e.g. infection or haemorrhage
  * Unilateral ventilation e.g. B-P fistula or unilateral bulla
  * Unilateral bronchial lavage e.g. pulmonary alveolar proteinosis

Relative
•Surgical access e.g. lobesctomy or oesophagectomy
•Non-thoracic surgery – thoracic spinal surgery

Pre-Op assessment
•Resting spirometry – FEV1 >2L

# One-Lung Ventilation II

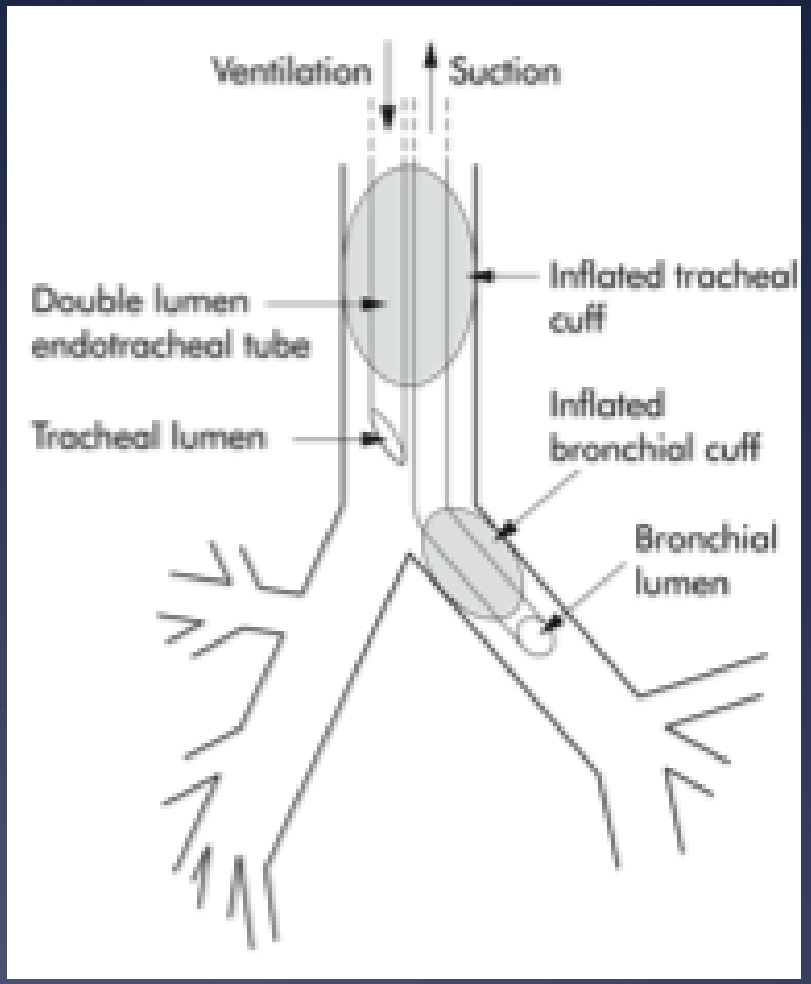
- Physiological Changes
  - Position
    - Altered Compliance (ventilated lung on steep part of curve – receives more ventilation)
    - Altered V/Q relationship in lateral thoracotomy position

| | Dependent Lung | Non-Dependent Lung |
|---|---|---|
| **Ventilation** | Decreased | Increased |
| **Perfusion** | Increased | Decreased |
| **Pulmonary Blood Flow** | 80% | 20% |

- Cardiac Output
  - Usually falls due to GA / positioning → worsening hypoxaemia
  - Remember whilst fluids may help cardiac output they can worsen pulmonary oedema
- Venous Admixture = major cause of hypoxaemia / hypoxia
  - One lung is not being ventilated → true shunt (V/Q =0)
  - Calculated by Shunt Equation
  - Venous admixture – two lung ventilation = 10-20% Vs OLV = 30-40%
- Hypoxic Pulmonary Vasoconstriction
  - Blood is diverted from non-ventilated lung to ventilated lung to maintain oxygenation → reduces venous admixture (but some flow remains)
  - Onset within seconds, max duration = 4hours
  - Is reduced by hypocapnia and volatile anaesthetics

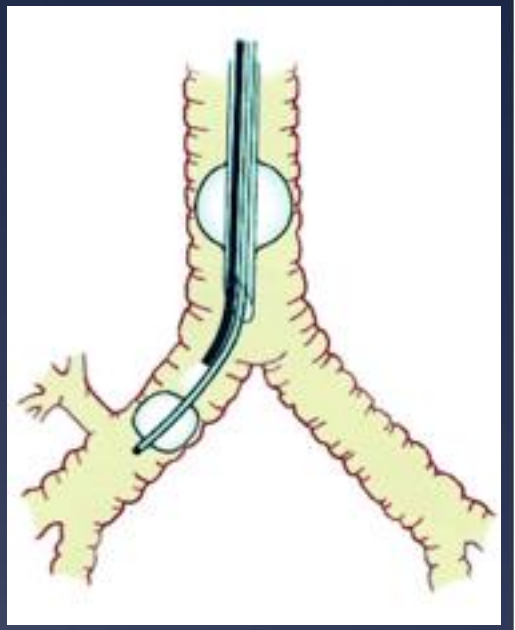
# One-Lung Ventilation III – Double-Lumen Tubes

- Classified by which main bronchus they intubate (i.e. the opposite side to operative side)
- Sizing = Charriere gauge (same as French)
  - Choose largest that will easily pass through glottis
  - Males = 39-41Ch
    Females = 35-37Ch
- Check position after placement / re-positioning
- Two methods
  - Clinically
  - Fibre-optically – gold standard



# One-Lung Ventilation IV – Bronchial Blockers

- = balloon-tipped catheters passed via a standard ETT or tracheostomy
- Two types - Univent or Arndt
- Indications
  - Same as for DLTs but where DLTs are unsuitable or undesirable e.g. permanent tracheostomy
- Insertion
  - Inserted via ETT/tracheostomy under fibre-optic guidance into appropriate bronchus
  - Balloon inflated
  - Gas either a) escapes via central lumen or b) is absorbed → lung collapse



- Reinstituting Two lung ventilation
  - The balloon is deflated and the lung is manually reinflated under direct vision

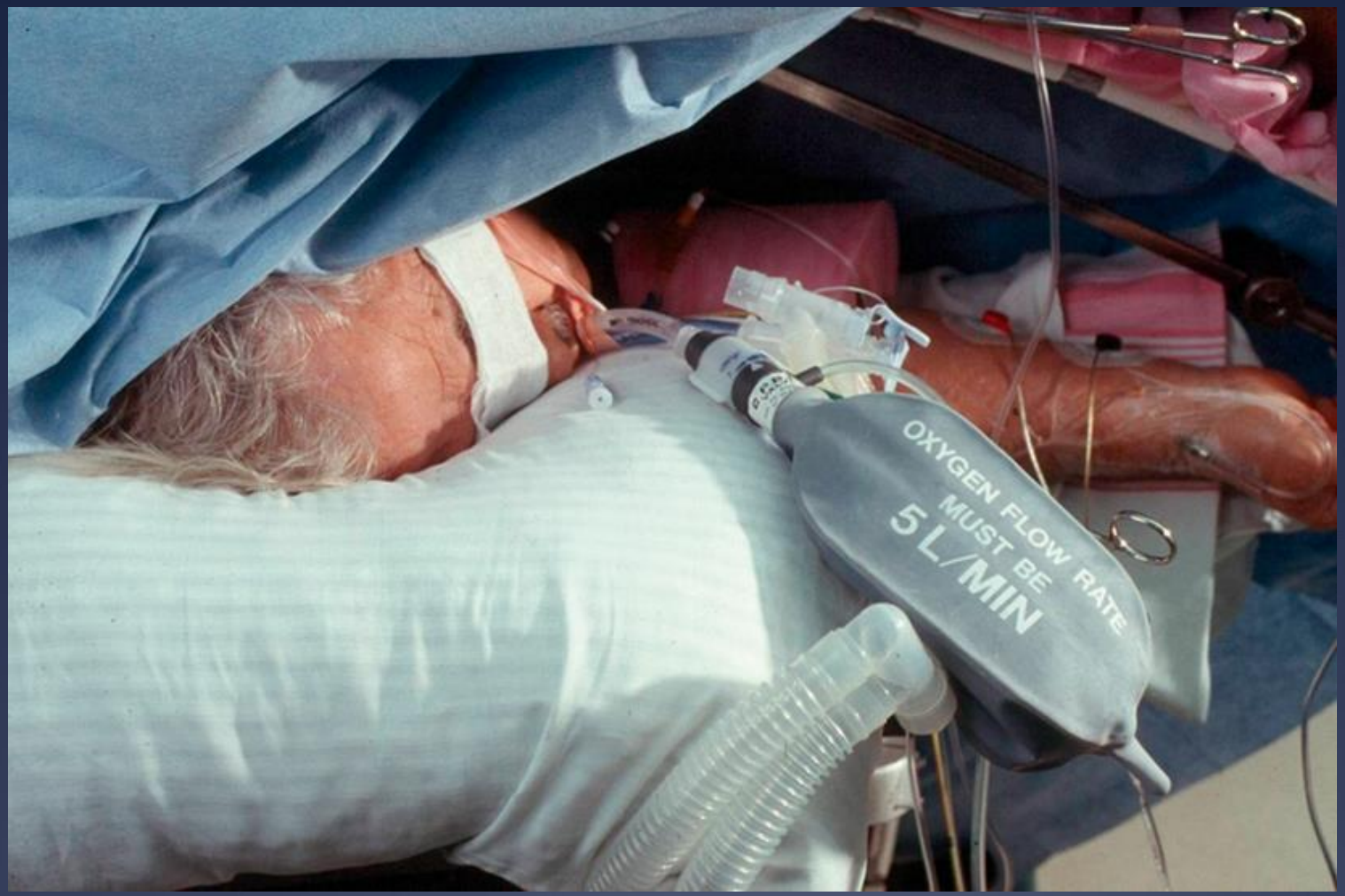
# One-Lung Ventilation IV – Managing OLV

- The overall aim is to minimise the effects of shunt
- Initiating OLV
  - Increase FiO2 and reduce VT
  - Clamp non-dependent lung at Y-connector
  - Open sealing cap and allow gas to escape
  - Expect Airway Pressure to increase (up to 40%)
  - Adjust VT to aim for Paw Pressure of <30cmH20
  - Confirm that lung is deflating
  - Aim to maintain SpO2 and EtCO2 at appropriate levels
- Returning to Two-Lung Ventilation
  - Suction lungs with long DLT suction catheters
  - Close sealing cap on non-ventilated lung
  - Remove clamp from Y-connector
  - Manually re-inflate collapsed lung – confirming reinflation under direct vision – often requires inflation pressures up to 40cmH20
  - Reinstate normal ventilation

# One-Lung Ventilation V

- Hypoxia and OLV
  - Increase FiO2
  - Check circuit and tube for malposition / cuff herniation
  - Bronchoscope position check
  - Ensure adequate cardiac output
  - Insufflate oxygen to non-ventilated lung
  - CPAP to non-ventilated lung
  - PEEP to ventilated lung
  - Clamp to appropriate pulmonary artery
  - Re-inflate collapsed lung with O2 intermittently / permanently
- Increased Airway Pressures and OLV
  - Malposition or Obstruction
  - Dynamic Hyperinflation or Pneumothorax
  - Anaphylaxis or Bronchospasm
  - Pulmonary Oedema

# CPAP to non-ventilated Lung



# Post-op Consideration / Problems

- Most, if not all patients who have received OLV, should be considered for a post-op CXR – to confirm reinflation
- Potential Problems
  - Residual Atelectasis
  - Pneumothorax
  - Respiratory failure
  - Sputum retention
  - Cardiovascular complications e.g. hypotension, blood loss
- Other considerations
  - Analgesia
  - Fluid balance
  - VTE prophylaxis
  - Nutrition – these patients are often malnourished

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# Hypothermia: pH stat management

## Definition

pH-Stat

**During pH-stat acid-base management, the patient's pH is maintained at a constant level by managing pH *at the patient's temperature***. pH-stat pH management *is* temperature-corrected. Compared to alpha-stat, pH stat (which aims for a pCO2 of 40 and pH of 7.40 at the patient's actual temperature) leads to higher pCO2 (*respiratory acidosis*), and *increased cerebral blood flow*. **Often CO2 is deliberately added to maintain a pCO2 of 40 mm Hg during hypothermia.**

## Alpha-Stat

**During alpha-stat acid-base management, the ionization state of histidine is maintained** by managing a standardized pH (measured at 37C). Alpha-stat pH management is *not* temperature-corrected – as the patient's temperature falls, the partial pressure of CO2 decreases (and solubility increases), thus a hypothermic patient with a pH of 7.40 and a pCO2 of 40 (measured at 37C) will, in reality, have a lower pCO2 (because partial pressure of CO2 is lower), and this will manifest as a relative *respiratory alkalosis* coupled with *decreased cerebral blood flow*. During alpha-stat management you have no idea what the patient's pCO2 is, your goal is to maintain a constant dissociation state of histidine.

## Alpha Stat vs. pH Stat

A study by Kiziltan et al., in which 52 patients were randomized to alpha-stat versus pH stat management, showed that **pH stat management led to increased jugular venous oxygen concentrations**, implying increased CBF. A study by Sakamoto et al., comparing pH stat to alpha stat during repair of cyanotic neonatal congenital heart disease, demonstrated that pH stat management led to less pulmonary collateral circulation as well as higher oxyhemoglobin and lower deoxyhemoglobin levels on cerebral near-infrared spectroscopy, suggesting greater cerebral oxygenation through improved oxygen delivery with pH stat. A prior study by Murkin et al. comparing pH stat to alpha stat showed that **during pH stat, CBF and CMRO2 become uncoupled** (CBF is pressure-dependent), whereas **during alpha-stat CBF is related to metabolic needs (CMRO2) and *not* to cerebral perfusion pressure**. **The major concern with pH stat is the potential for increasing the cerebral embolic load**.

**ALPHA STAT VERSUS pH STAT**

This is a dilemma cardiac anaesthetists have grappled with for some time when managing arrested hypothermic cardiac bypass cases

- pH STAT approach: the arterial carbon dioxide tension (paCO2) is maintained at 40 mmHg and the pH is maintained at 7.40 when measured at the actual temperature; it is therfore necessary to add CO2 to the inspired gas
- alpha STAT approach: paCO2 and the pH are maintained at 40 mmHg and 7.40 when measured at +37 C. When a patient is cooled down, the pH-value will increase and the measured pCO2 and the pO2 will decrease with lowering of the temperature if measured at the patient's temperature.

Argument for the pH STAT approach

- adding $CO_2$ to the oxygenator counteracts the increased solubility of $CO_2$ at lower temperatures to keep the normothermic pH the same
- higher $CO_2$ causes cerebral vasodilatation and faster and more homogeneous cooling
- It this counteracts the hypothermic leftward shift of the oxygen dissociation curve, resulting in better oxygen delivery
- may optimise myocardial function

Argument for the alpha STAT approach

- alkaline drift during hypothermia allows cerebral autoregulation to continue and cellular transmembrane pH gradients and protein function to be maintained
- the alkaline pH improves cerebral protection during the ischaemic insult
- by maintaining cerebral autogregulation, avoids potential problems of excess cerebral blood flow such as intracranial hypertension and increased microembolisation

# Thank You … Any Questions ?