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Anatomy

Coronary Arteries

- Aortic sinuses kept away from valves by eddy currents
- Orifices patent throughout cardiac cycle
- RCA & LCA arise immediately above the cusps of AV (aortic root)
- LCA supplies:
  - L circumflex - L & R atrium, lat wall of LV, SAN (45%) (I, aVL)
  - LAD - walls of L & R vent incl septum (V3-V5)
- RCA supplies - SAN (55%), AVN (90%), HIS (90%), IVS, RA & RV (II, III, aVF)
  - R marginal - R vent
  - post descending art - walls L & R vent, IVS
- dominant circulation named after which one supplies AVN:
  - 80% people R dominant - via PDA
  - 10% co-dominant
  - 10% L dominant - ∴ LMS occlusion ⇒ absolute no flow to LV

- Venous blood return:
  - Coronary sinus RA – 95% of blood
  - Ant coronary vein RA
  - Thesbian veins various cardiac chambers
    - some drain into LV contribute to physiological shunt
      - ie contributing to normal $D[A-a]O_2$
  - Others:
    - Arteriosinusoidal vessels – connect arterioles to chambers
    - Arterioluminal vessels – arteries straight into chambers
• Diagram of artery supply

**Valves**
• ant leaflet of mitral valve closely related to aorta

**Cardiac Pressures**

![Diagram of artery supply]

**Pulmonary Pressures**
• mean pressure gradients for pHTN (mmHg):
  ‣ mild = 25-40
  ‣ mod = 40-55
  ‣ severe = >55

**Chambers**

**LV**
• thick high pressure
• posterior in heart
• pap muscles
• chordae

**RV**
• thin, anterior
• moderator band
• separate chordae

**Atria**
• L & R atrial appendages

**TOE view**
• transgastric mid pap view shows all vessel territories =
Physiology

Basics
• CBF to LV occurs predominantly in diastole
• ↑HR ⇒ ↓diastolic time & no change in systolic time

Myocardial O2 supply
• Depends on:
  • O2 content =
    - Hb
    - SaO2
    - PaO2 (insignificant)
  • Myocardial blood flow =
    - DBP:
      • SBP
      • arterial compliance ie SVR
      • aortic valve competence
      • HR
    - diastolic length
    - blood viscosity - ↓ed on CPB
    - coronary vasc resistance -
      → variable vascular tone vs fixed artheromatous lesions
    - LVEDP = ↑ed pressure ⇒ ↓flow

Myocardial O2 demand
• depend on:
  • myocardial wall tension = SBP
  • HR:
    - physiological HR = optimal flow
    - ↓HR ⇒
      • longer diastolic time which ⇒ ↑CBF & fewer contractions BUT also ⇒ ↓ing diastolic pressure during prolonged diastole ⇒ ↓CBF in late diastole
    - ↑HR ⇒
      • ↑mean diastolic pressure ⇒ ↑coronary perfusion pressure BUT also ⇒ ↓time for flow to occur & ↑no of contractions
  • Blood pressure:
    - ↑bp: ↑ed disatolic pressures ⇒ ↑CBF BUT also ↑systolic pressure ⇒ ↑O2 consumption
    - ↓bp: requires ↓ed myocardial wall tension to achieve ↓O2 consumption BUT also ↓ed diastolic pressure ⇒ ↓CBF
PreAssessment

General PreAx Visit

Basics
• Standard preAx questions

Surgery
• ?artery harvesting ?side
• ?re-do

Systems R/V
• CVS:
  • IHD:
     - Angina - Canadian scale I-IV:
       • 1 = bothersome only - only during v strenuous/prolonged activity
       • 2 = mild limitation - only during vigorous activity
       • 3 = moderate limitation - symptoms everyday living
       • 4 = total limitation - angina @rest/unable to perform any activity without angina
     - ACS
     - Anti-coags
     - Heart failure symptoms
     - Anti-HTN Rx
     - Exercise tolerance -
       • METs
         • NYHA I-IV:
           • no limitation: cardiac disease but no limitation ie no SOB up stairs
           • mild: mild SOB during normal activity
           • marked limitation: comfortable only at rest. Symptoms walking short distances
           • severe limitation: Symptoms at rest. Mostly bed bound
  • Valves:
    • Which valves
    • ?pHTN
    • Warfarin
  • Ventricles/Pulmon:
    • LV:
      • EF - (aware of overestimation in MR)
      • Size - dilated or LVH
    • RV function
    • diastolic function ↑ filling pressures
      ▪ very impt - 50% pts with acute heart failure have normal EF
      ▪ EF = (SV/EDV) * 100
  • Vascular:
    • Carotids:
      • previous stroke ?CUSS
      • ?bruits
    • PVD:
      • AAA
      • ?bypass surgery
      • palpate all arteries
  • Resp:
    • Standard
• PFTs
• Renal:
  • Creatinine
• Endocrine:
  • DM ?HbA1C
  • ?end-organ damage
• GI:
  • aspiration
  • TOE contraindications:
    - ?obstructive problems in past eg ‘?food sticking ?vomiting blood ?oesophageal problems’
      • absolute CIs: stricure, web, tumour
      • relative CIs: varices, diverticula, prev GI bleed, prev surgery or radiation

Investigation
• vitals
• ECG: ?LBBB - relevant if PAC planned
• Blds incl TFTs
• PFTs - bedside spiro done on admission. May need to request PFTs
• Angio
• ECHO

Medications
• antiHTNs:
  • BBBlockers (cont ?↓dose by 50% if brady)
• Anticoags & platelets - stop, if needs bridging (eg unstable ACS) use heparin
• Sedatives: useful overnight!

Risk Scoring
• need way to calculate predicted operative mortality
• many scores eg EuroSCORE II - use calculators to ascertain based on categories:
  • patient related
  • cardiac related
  • operative related
• most impt factors:
  • emergency surgery
  • re-do
  • ↑age
  • poor LV function
  • renal dysfunction
General Operating Procedure

Generalised Induction

• aims:
  ‣ maintain CBF
  ‣ prevent tachycardia
• in critical patients surgeon should be ready at induction
• standard full monitoring + BIS
• BIS:
  ‣ no method to prevent awareness during CPB
  ‣ useful to monitor cortex during deep hypothermic arrest

Induction Agents

• Fentanyl 5-15mcg/kg
  ‣ Good:
    - ↓ or no induction agent required
    - myocardially stable esp in ↓LVF
    - analgesia
  ‣ Bad:
    - no amnesia - ?use midaz
    - ?prolong postop time
    - chest wall rigidity
• Ketamine (0-1mg/kg ie 0-100mg):
  ‣ Good:
    - haemodynamically stable - central SNS stimulation but direct -ve inotropic effect
    - good in hypovolaemic shock & tamponade
    - analgesia
  ‣ Bad:
    - ↑ed -ve inotropic effect if ↓LVf & α blockade
    - central SNS stim ⇒ ↑myocardial O2 demand
• Propofol:
  ‣ Good:
    - resets baroreceptor reflex ⇒ no ↑HR
    - fast on & off
  ‣ Bad:
    - ↓SVR ⇒ 15-40% ↓ bp at 2mg/kg (may be good for regurg lesions)
    - some -ve inotropy
• Etomidate:
  ‣ Good:
    - less CVS depression ~10-15%
    - in normovolaemia see unchanged: SV, LVEDP, contractility
    - useful in heart transplants
    - fast onset
  ‣ Bad:
    - ↓Adrenal activity
    - ↑epileptiform activity
    - ↓bp if combined with opioids
• Midazolam:
  ‣ Good:
- ↑ amnesia
- CVS stable

• Bad:
  - no analgesia
  - needs hepatic metabolism
Equipment/Procedures

Cardiopulmonary Bypass
• replaces heart & lungs to allow bloodless, stable surgical field

Gas Exchange
• Membrane oxygenators used most commonly
  • minute hollow fibres ⇒
    • diffusion distance 200um [lung 10um]
    • large surface area (2-2.5m²) [lung 70-100m²]
• Gas exchange down concentration gradients:
  • ↑gas flow ⇒ ↑CO₂ removal
  • ↑FiO₂ ⇒ ↑O₂ supply
• includes heater

Blood Pumps
• blood flow rate = P/R
• non pulsatile flow
• types of pump:
  • roller =
    - ADV: predictable low, linear CO in l/min
    - BAD: risk of pumping air, pipe burst, damage cells, micro plastic particles from tube ⇒ embolisation (spallation)
  • centrifugal pumps: flow = mass x radius x angular velocity
    - afterload dependant ie ↓flow if ↑afterload - can be a safety feature
    - ADV:
      • prevent circuit rupture
      • unable to pump air ⇒ disaster
      • ↓trauma to cells

Venous Reservoir
• 2 types:
  • hard shell (open) = allow passive removal of entrained air, can apply suction to encourage venous drainage
  • soft shell (closed) = less volume :. less blood contact with foreign material ⇒ less inflammation

Arterial Filter
• mesh of polyester fibres filer arterial blood of contaminants & leucocytes

Starting Bypass
• bypass circuit primed with
  • crystalloid
  • heparin
  • +/- mannitol & HCO₃-
• cannula:
  • arterial: 24Fr into distal ascending aorta (can use fem or axillary)
  • venous:
    - R atrial appendage with tip in IVC
    - drainage port also drains from SVC & coronary sinus
  • retrograde cardioplegia line:
    - 15F into coronary sinus - watch with TOE & observe pressure trace
Cardioplegia - delivered via a secondary circuit
- 2 delivery methods:
  - anterograde = into aortic root
  - retrograde = via coronary sinus
- intermittent 20mins
- K 20mmol = diastolic arrest
- induction = cold solution
- reperfusion = warm solution
- ventilator turned off after bypass established

Steps
1. Baseline ACT, ABG, TEG
2. Heparinise with 300IU/kg via central line - action to augment antithrombin III
   ⇐ if heparin resistance use FFP (contains antithrombin III) or recombinant antithrombin III
3. Prior to cannula: ↓ systolic bp to 80-100mmHg = ↓’s risk of dissection
4. Cannula: arterial > venous > retrograde cardioplegia lines
5. Final check ACT
6. Test arterial input - look for swing & infuseability
7. Release venous clamp ⇒ VR to CPB commences ⇒ ↓ CVP & ↓ PAP ⇒ heart empties
8. Inspect heart ⇒ confirm low CVP & head not congested
9. Flow set 2-3l/min/m2
10. Pressure 50-80mmHg. Consider higher range if carotid/renal disease
11. aortic cross clamp to isolate heart
12. cardioplegia ⇒ diastolic arrest
13. cool - typically to 32-34

TIVA or Volatile
- TIVA started or volatile given via pump
- All agents in dose dependant manner:
  - ↑VD
  - cardiac depression
  - ↓HR
- isoflurane:
  - animal models show coronary steal phenomenon:
    - ischaemia normally ⇒ targeted dilation of relevant arterioles to maximise blood flow
    - potent vasoDs like isoflurane may ⇒ non-specific vasoD in all arterioles
    - if fixed flow means loss of local autoregulation ⇒ ↑ed ischaemia
      BUT not be shown to be imp in humans
  - Iso may also be ischaemia protective via ATP sensitive K channels
- desflurane:
  - similar cardiac profile to isoflurane but slightly ↑ed SNS outflow

On Bypass
- tranexamic acid used (max 30-40mg/kg):
  - given at:
    - at start of bypass
    - infusion through the procedure
  - caution as risks of
    - epilepsy

Targets
- bypass machine delivers 2.4L/min/m2 ie typical cardiac index
• MAP target = 50-80mmHg
  → achieved by altering SVR with
    • vasoconstrictors
    • vasodilators eg GTN, phentolamine

• Blood gases & ACT checked every 30mins
  • ACT -
    - measure every 30mins
    - >480
    - beware heparin resistance if pts been on heparin preop
  • HCT target 20-30%:
    - add fluid to pump reservoir
    - remove fluid by ultrafiltration
  • Normal PaO2 & PaCO2
  • Gluc <10
  • BE < -2.5

• ECG:
  • VF should be terminated unless planned
  • electrical activity ≈ repeat cardioplegia
  • VF during rewarming needs defib
  • ectopics are common - dont panic

• Urine - aim >1ml/kg/hr

**Cardioplegia:**

• Administered diff ways:
  • anterograde = via coronary arteries
  • retrograde = via coronary sinus
    → monitor infusion pressure & make sure it doesnt rise

• Can be blood or crystalloid:
  • Based on Ringers solution - contains:
    - K
    - Mg
    - procaine
  • Blood -
    - assumption that it contains O2 ↓ might ↓ischaemia
    - reperfusion warm blood cardioplegia may be used towards end of bypass to wash out metabolites
    → advantages seem to be theoretical only

• 1litre renders heart asystolic
  → further doses rpted 20mins or if electrical activity

• 4deg cold solution protects heart against ischaemia

**Temperature:**

• Diff options depending on surgery:
  • 30-32 deg target - short operations
  • 26-28 typical
  • 16-22 deep hypothermic arrest

• Lower temps offer better cerebral protection but longer time on CPB to rewarm
• Lower temp generally reserved for complex cases

**Cross Clamping & Fibrillation**

• 2 options for CABG:
  • cardioplegia (asystole)
  • intermittent cross clamping with fibrillation
- method:
  • aorta clamped
  • fibrillation pad placed underneath heart to induce VF
  • graft bottom end (post lesion) is sutured
  • cross clamp removed ⇒ heart converted to SR
  • graft top end (pre lesion) is completed
- advs:
  • no cardioplegia = good because:
    ‣ ↓ incidence of complete heart block
    ‣ ECG can be inspected for resolution of ischaemia
- disadv: no ischaemia protection ⇒ surgical time must be quick <10mins

**Hypotension on Bypass**

- Differentials:
  ‣ haemodilution - from ↑ circuit length
  ‣ ↓ SVR, ↓ viscosity
  ‣ dilution of catecholamines
  ‣ vasoactive inflammation due to circuit contact
  ‣ drugs
  ‣ check drainage/flows

**Coming Off Bypass**

- team effort between surgeon, anaesthetist & perfusionist
- TRAVVEL checklist for weaning:
  ‣ T = temp
  ‣ R = rate & rhythm
  ‣ A = air
  ‣ V = ventilation
  ‣ V = aortic root venting ↓ ed & matched by arterial pump
  ‣ E = electrolytes
  ‣ L = level table & pressure transducers
- preconditions for stopping bypass:
  ‣ body temp >36.5
  ‣ K = 4.5-5mmol
  ‣ HCT >20%
  ‣ Normal pH
  ‣ HR 70-100 (+/- pacing, defibrillation, other drugs)

or…..

- A irway - check in position
- B - Lungs:
  ‣ re-establish ventilation: 100% O2
  ‣ re-inflate ensure basal expansion under direct vision
  ‣ use sustained ~30cmH2O breath. watch grafts esp IMA
- C-
  ‣ components of CO:
    - preload return blood to R side of heart,
    - afterload (vasopressors),
    - rate (target 80, check PMs), rhythm (SR or PM),
    - contractility (temp, electrolytes etc)
  ‣ Heart de-airing:
- impt in valve surgery
- various manoeuvres
- check with TOE

- D:
  - analgesia - ?given
  - anaesthesia
- E:
  - temp
- F:
  - fluids input/output
- G - aetric
- H - aematology
  - heparin - protamine
- I nfection
  - cephazolin 2nd dose 4 hrs post loading, then 8hours
- M etabolic
  - blood gas

- Final check:
  - infusions ready & able
  - PM leads checked and working
  - defib present & ready

- Venous bypass line slowly clamped & heart allowed to fill & eject
- Usual practise to come off pump with heart relatively underfilled
  \[\rightarrow\] prevents overdistension in already impaired ventricular function

- perfusionist
  - transfuse 100ml fluid boluses
  - Need to assess heart performance & filling
  - commence inotropic support if required
- Protamine -
  - only to be drawn up after off CPB
  - administer slowly peripherally
  - 1mg/100units heparin used
- SE’s:
  - hypotension - may need rapid IVF boluses
  - pulmonary HTN
  - anaphylaxis

**Problems Coming off Bypass**
- check
  - fluid/volume status esp R heart
  - pacing
  - inotropes
- Consider:
  - IABP
  - VAD/ECM

**Post Bypass**
- ensure adequate anaesthesia & pain relief
- MAP (>65) & SBP (<140) kept under close targets
• Maintain:
  - Normal electrolytes
  - Normal coagulation - TEG repeated

**Complications**
• minimal in 1st 24hrs but include:
  - haemolysis
  - platelet damage
  - Bleeding - multifactorial reasons:
    (significant = >400ml in 1st hour, >300ml each hr thereafter)
    - preop anticoags
    - hypothermia - rewarming post bypass can be difficult to sustain. Easy to lose heat to peripheral compartment with ↓temp at core
    - intraop anticoags:
      - incomplete heparin reversal
      - hypothermia
      - poor clotting factors in pump blood
      - protamine itself in the wrong dose = anticoagulant
  - CPB insult:
    - endothelial dysfunction ⇒ activation of coagulation ⇒ fibrinolysis ⇒ inflam & consumption of factors
    - thrombocytopenia = dilution, consumption
    - platelet dysfunction - ↓temp, contact on foreign substances, heparin, protamine
    - fibrinolysis:
      - primary - endothelial plasminogen activators
      - secondary - fibrin formation
  - Cell saver blood = rbcs only
    ↳ everything else washed out

▷ strategies to minimise:
  - minimise CPB time
  - warmth
  - ACT & TEG to guide protamine
  - TXA
  - transfuse based on TEG & coag screen

• other problems:
  - ↓venous drainage
  - occlusion/dislodgement of cannulae
  - aortic dissection
  - gas embolisation
  - inflammation/SIRS:
    - humoral ⇒ activation of endothelium ⇒ neutrophil adhesion, aggregation & activation
    - cellular immunity ⇒ complement, fibrinolytic, coag systems
  • cerebral problems - ↑ed risk if >2hrs CPB:
    - Stroke -
      • 1-5% risk
      • non fatal or fatal
      • Factors ↑risk=
        • ↑age
        • HTN
        • aortic atheroma
        • previous stroke
DM

surgery type eg aortic arch > valve > CABG

aetiology = hypoperfusion & emboli

emboli sources:
  - proximal aorta atheroma (most sig site) - ultrasound can help avoid sites
  - other sources: air, FB, cellular aggregates, fat, calcium

strategies to avoid=
  - optimum perfusion pressures
  - normal BSL
  - pH control (see later):
    - mild hypothermia - use a stat
    - deep hypothermia - use pH stat
  - careful de-airing of heart prior to coming off CPB
  - careful temperature control - target 33-35

  - coma
  - encephalopathy
  - POCD - short (up to 80%) or long term (up to 5-20%)
  - delirium
**Intra-Aortic Balloon Pump**

- Inserted percutaneously via femoral artery
- Sized on height of patient
- Can use for 5 to 7 days
- Most important affects achieved:
  - ↓afterload
  - ↑coronary perfusion
  

\[ \therefore \text{only} \] ↑CO significantly if LV is limited by ischaemia or high afterload

<table>
<thead>
<tr>
<th>Aortic pressure</th>
<th>Cardiac</th>
<th>Blood flow</th>
<th>LV</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ systolic</td>
<td>↓ afterload</td>
<td>↑ coronary BF</td>
<td>↓ volume</td>
</tr>
<tr>
<td>↑ diastolic</td>
<td>↓ preload</td>
<td>↑ CO</td>
<td>↓ wall tension</td>
</tr>
<tr>
<td>↑ Windkessel effect</td>
<td>↑ RBF (up to 25% by ↑ CO)</td>
<td>↓ systolic pressure</td>
<td></td>
</tr>
</tbody>
</table>

- Pump stroke is triggered by either:
  - Arterial pressure trace:
    - inflation - just following dicrotic notch (just after AV closure) (beginning of diastole)
    - deflation: just prior to ejection on upstroke of arterial trace (just before AV opening) (end of diastole)
  - ECG - more preferable
    - inflation - middle of T wave

- \( A = \) systolic pressure
- \( B = \) dicrotic notch
- \( C = \) augmented bp
- \( D = \) ↓ed end diastolic pressure
- deflation = start at peak of R wave

- helium used to inflate/deflate balloon
- inflation (beginning of diastole) causes:
  - ↑ed diastolic pressure
  - ↑CBF & O2 delivery
  - no ↑ in O2 demand
- (end of diastole) immediately prior to systole ⇒ deflation:
  - ↓ afterload
  - offloads LV ⇒ ↑ LV ejection
- can set 1:1; 1:2; 1:4
- required heparinisation
- must check position of pump on CXR (tip 3-5cm below L subclavian artery)
  - not occluding major arteries off aortic arch ⇒ check periph pulses

- indications:
  - ongoing myocardial ischaemia
  - cardiogenic shock
  - weaning from CPB
  - MR eg acute waiting surgery
  - VSD
  - stunned myocardium
  - bridge to transplant
- IABP less effective if:
  - ↑ HR
  - irregular rhythm
  - ↑ aortic compliance or ↓ SVR
- contraindications:
  - absolute:
    - AR > mild
    - severe aortic atheroma
    - HOCM/SAM
    - aortic dissection
    - end stage with no anticipation of recovery
  - relative:
    - AAA
    - tachyarrhythmia
    - severe PVD
    - sepsis
- Complications:
  - vascular:
    - arterial injury
    - aortic injury
    - thromboembolism
    - ischaemia - limb or visceral
    - compartment syndrome
  - balloon:
    - rupture
    - gas embolism
    - cardiac tamponade
    - incorrect position
  - other:
- haemorrhage
- infection
- haemolysis
- thrombocytopenia

By A Hollingworth

A = too early inflation ⇒ ↑afterload
B = too late inflation ⇒ failed diastolic augmentation
C = too early deflation ⇒ can see flow reversal in cornaries
D = too late deflation ⇒ high afterload decr cardiac output

Ventricular Assist Devices
- Diff types available to support ventricles or both: RVAD, LVAD, BIVAD
- Position:
  - LVAD - LA to ascending aorta
  - RVAD - RA to main pulmonary artery
- require sternotomies
Indications

- Acute heart failure:
  - cardiogenic shock - bridge to recovery
    - e.g. MI, post cardiotomy, viral cardiomyopathy, post donor
- chronic heart failure:
  - post ischaemia & limited heart transplant
  - bridge to transplant

Patient Selection

- arrhythmias:
  - atrial not a concern
  - ventricular must be Rxed
- AR: any can be problematic as a VAD \( \Rightarrow \) ↑gradient between MAP and LVEDP \( \Rightarrow \) ↑regurg
- MS - needs to be corrected
- septal defects - corrected to prevent R to L shunt

Ongoing Treatment

- lifelong warfarin
- aspirin
- battery care & technical follow up

Anaesthesia for VAD Patient

PreOp
- preoptimise as any cardiac patient

IntraOp
- strict asepsis
- anticoagulation continued as long as possible
- invasive monitoring
- maintain normal preload

Extracorporeal Membrane Oxygenation (ECMO)

- advantage of supporting heart & lung without need for sternotomy
- needs big resources, specialist team with constant monitoring, not for long term
- membrane oxygenators allow better CO2 removal than O2 addition

Types

- VA:
  - allows gas exchange & haemodynamic support
  - centrifugal external pump
  - bypasses heart - part or all of blood
- VV:
  - facilitates gas exchange
  - no haemodynamic support
  - centrifugal external pump
- AV:
  - facilitates gas exchange by using pts own arterial pressure to pump blood
  - generally femoral \( \Rightarrow \) femoral
  - dependant on cardiac output
VA ECMO
- drainage commonly from IVC or RA
- return via ascending aorta or femoral artery
- indications:
  - refractory reversible cardiogenic shock
  - bridge to VAD or cardiac transplant
  - salvage during cardiac arrest - after 10mins of adequate but unsuccessful CPR
- advs:
  - ↓ cardiac work \(\Rightarrow\) ↓ cardiac O2 consumption
  - allows proportion of blood \(\Rightarrow\) lungs \(\Rightarrow\) final content of blood depends on mixture
- disadv:
  - if using peripheral VA ECMO head & coronaries receive proportionally low O2 blood compared to LLs

VV ECMO
- used when cardiac function is preserved
- used to provide oxygenation & rest lungs
- 2 methods:
  - x2 cannula: IJ (oxy) +/- femoral (de-oxy)
  - x1 cannula new dual chamber cannula - overcomes recirculation problem & bleeding problems
- indications:
  - ALI/ARDS
  - graft dysfunction post lung transplant
- adv:
  - ↓ ITP \(\Rightarrow\) off load R heart
  - lower risk of thromboembolic problems
  - allows full endocrine functions of lung

AV ECMO
- pumpless connection via oxygenator
- cannula in fem artery & vein
- must have CI >2.5l/min/m2
- adv:
  - simpler system which is smaller & requires no battery for transport
- disadv:
  - mortality high in ARDS
- indications:
  - severe hypercapnia, resp acidosis & only moderate hypoxia

Management
- often marked improvement in haemodynamics
- can extracorporeal haemofiltrate off ECMO circuit to remove fluid
- anticoagulation impt:
  - heparin \(\Rightarrow\) ACT 1.5x normal
- thrombocytopaenia a problem \(\Rightarrow\) regular platelet transfusions

Contraindications
- irreversible organ damage
- multiorgan failure
- unable to anticogulate
- VA ECMO = severe AR or aortic dissection

Complications
- haemorrhage - higher risk in arterial cannulation
• thrombosis - in VA ⇒ stroke
• infection
• circuit failure catastrophic
Pulmonary Artery Catheters

Indications
• risk stratify pts to decide on need for PAC:
  ‣ low risk = no PAC:
    - good LVF
    - surgery will not ↑load on LV eg CABG, AVR for AS
  ‣ intermediate risk = +/- PAC:
    - ↓LVF
    - surgery with ↑ed loading on LV expected eg double surgery, MVR for MR, double valve, AVR, complex aortic cases
    - if unsure PA sheath may be compromise
  ‣ high risk = PAC:
    - as intermediate but also:
      • pHTN +/- ↓RVF

Contraindications
• absolute: mechanical tricuspid valve
• relative:
  ‣ VSD, ASD
  ‣ LBBB, severe vent arrhythmia
  ‣ bioprosthetic tricuspid valve, tricuspid endocarditis
  ‣ pneumonectomy
  ‣ vent pacing wire dependant

Advantages
• early detection of ischaemia ⇒ see diastolic dysfunction ⇒ if LV ⇒ ↑pulmonary pressures
• RV vs LV failure
• ongoing monitoring in ICU

Insertion
• insert sheath
• flush all lines & put 3 way taps on lumens
• connect PAC to pressure transducer
• insert with curve directed medially ⇒ advance 10cm & look for trace
• floating:
  ‣ inflate balloon & advance
    - 20-30cm = RV trace
    - 35-50cm = PA trace
    - Advance another 5-10cm until wedge pressure appears
  ‣ deflate balloon
    ➔ continuous wedging may ⇒ PA rupture
• lock in place
• initiating CPB - safest to withdraw catheter 5cm
• if you need to withdraw catheter ALWAYS deflate balloon first

Complications
• Arrhythmia:
  • if irritable heart or intolerable of arrhythmias: safer to wait for heart to be exposed prior to floating PA catheter
    ➔ eg ectopics, stenotic lesions
always be ready to defib

Knotting ⇒
- surgical fix - direct exploration & cutting line then removal via open heart surgery
- interventional radiology -

Damage to R side valves
PA rupture: highest in elderly, pHTN. Unlikely need to wedge - do very gently!!!
RBBB: if has pre-existing LBBB ⇒ complete HB . need to be ready to pace or CPB
misinterpretation of data -
  - abnormal wave form
  - poor wedging
  - if give fluid bolus need to wait for response as numbers are averaged over time

HITT - heparin & latex coated lines

TOE

Advantages over TTE:
  - don't have to image through chest wall/lungs
  - Better images of post structures eg aorta & LA
  - probe left in place to allow temporal comparison
  - can see tamponade sitting posteriorly

Basics

- 4-7Mhz
- Piezo-electric: time = distance
- speed of sound = 1540m/s
- omniplane probe:
  - 0 deg = beam perpendicular
  - 90 deg = in line with probe ie vertical
  - 180 = beam opposite to 0 deg
Indications

- general for intra-op:
  - all open heart ops & thoracic aortic surgeries
  - some CABGs
  - non-cardiac with suspected CVS pathology & will be useful
  - guiding Rx of catheter based intracardic procedures

- indications above TTE:
  - eval of structures in far field ie aorta & L atrial appendage
  - eval of prosthetic heart valves
  - eval of valvular abscesses
  - vent’ed pts
  - chest wall injuries
  - ↑↑BMI
  - pts unable to move into L lat decubitus
  - critically ill pts in who TTE vies unobtainable & TOE will alter Rx

Contraindications

- Absolute:
  - perf hollow viscus
  - active GI bleeding
  - strictures/obstructions/tumours/scleroderma

- relative:
  - Cx spine inj
  - prev oesophageal: surgery, varices, barretts, diverticulum
  - severe coagulopathy
  - HH

- Complications:
  - Injury: sore throat, teeth damage, mucosa, oesophageal bleeding & tearing
  - ETT displacement
  - ischaemia from probe pressure
  - thermal injury - turn off when on CPB
  - resp difficulty - if not vent’ed
  - CVS problems - ↓MAP, ↓HR

Complications

- 1-3% complication:
  - sore throat
  - airway obstructions
  - dislodgement of ETT
  - trauma - teeth, oesophagus
  - SNS stim
  - infection

Views

- Mid oesophageal point = 20cm from incisors
  - MV parallel to this

- Order to imaging:
  - LA - ?PFO use bubble saline injection
  - LV - dilatation & hypertrophy
  - R side of heart

- Standard views are now 28
- Essential views:
• ME AV SAX - 30deg
• ME AV LAX - 100 deg
• ME 4Ch - 0 deg
• ME 2Ch
• TG SAX - 0 deg with flexion probe

Assessment of Function
• 3 regions to assess: basal, mid, apical
• Ventricular Function Methods:
  ‣ fractional shortening = EDD-ESD/EDD
    \[ \Rightarrow \text{may not be homogenous .: can be unreliable} \]
  ‣ fractional area change = trace 1 slice diameter at EDD & ESD
  ‣ 2Ch ME (ant & inf wall) & 4 Ch ME (lat & ant septal)
• Diastolic Dysfunction:
  ‣ PW doppler through MV tips & Ax velocity
  ‣ Waves:
    - E = early filling once MV opens
    - A = atrial contraction
    - E<A = normal
  ‣ Need to check for pseudonormalisation:
    - check LA size
    - PV flow
    - interatrial septum movement
• Cause of hypotension:
  ‣ ↓preload - kissing ventricles
  ‣ ↓contraction - RWMA or global dyskinesia
  ‣ ↓SVR - heart fills well & empties more than expected \(\Rightarrow\) kissing ventricle

<table>
<thead>
<tr>
<th>Table 4 Evaluation of hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection fraction</td>
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<tr>
<td>--------------------</td>
</tr>
<tr>
<td>Hypovolaemia</td>
</tr>
<tr>
<td>Ventricular failure</td>
</tr>
<tr>
<td>Reduced systemic vascular resistance</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>LVED</th>
<th>Men</th>
<th>Fem</th>
</tr>
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<tbody>
<tr>
<td>Norm</td>
<td>42-59</td>
<td>39-53</td>
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<tr>
<td>Mild</td>
<td>60-63</td>
<td>54-57</td>
</tr>
<tr>
<td>Mod</td>
<td>64-68</td>
<td>58-61</td>
</tr>
<tr>
<td>Sev</td>
<td>&gt;69</td>
<td>&gt;62</td>
</tr>
</tbody>
</table>

ACT
• 1ml blood place in tube containing magnetic rod & activator - usually celite or kaolin
• tube warmed to 37 deg & rotated \(\Rightarrow\) clot.
• resistance to movement of magnetic rod stops the timer
• normal = 100-140s
• crude test with 10% error spread
• Other effects on ACT:
  ‣ ↓temp \(\Rightarrow\) prolongs time
  ‣ haemodilution \(\Rightarrow\) prolongs time
• Post protamine ACT aim is <10% variance from baseline ACT
TEG

- using TEG guided algorithm shown to ⇒ ↓ consumption of products & transfusion related complications post CABG

**Method**

- blood placed in cuvette which is rotated through 4deg in 6cycles/min
  - imitate sluggish venous flow
- pin sensor is inserted into sample ⇒ clot formation between cup & pin
- speed & strength of clot is measured which tests:
  - platelet function
  - fibrinolysis
  - coagulation cascade

![Coagulation and Fibrinolysis Diagram](image)

- main variables determined:
  - **R time**:
    - = time until see first evidence of clot
    - norm = 4-8min
    - causes:
      - long >10min = anticoags, factor deficient, ↓ fibrinogen ⇒ **FFP (or protamine)**
      - give protamine 0.5mg/kg if >3mins diff between K TEG & KH TEG
      - short = enzymatic hypercoagulability ⇒ **anticoags**
  - **K value**:
    - = time from end of R until clot reaches 20mm := speed of clot formation
    - 1-4mins
    - causes:
      - long = anticoags, ↓ fibrinogen, ↓ platelets ⇒ **give cryo**
  - **α angle** (functionally similar to K value)
    - = angle of curve made as K is reached
    - faster rate of fibrin generation ⇒ ↑ pin oscillation amplitude ⇒ larger angle
    - norm = 47-74deg
- causes as K value \( \implies \angle < 45^\circ \text{ give cryo} \)
- MA (max amplitude)
  - = reflects clot strength 80-90% platelets, 10-20% fibrinogen
  - 55-73mm
  - causes:
    - ↓ed size = ↓plts, poor platelet function, ↓fibrinogen \( \Rightarrow \angle < 50\text{mm} \text{ give platelets} \)
    - ↑ed size = hypercoagulable, ↑plts \( \Rightarrow \text{ give anti platelet agent} \)
- A30 (amplitude at 30min)
  - = decrease in clot size 30mins after MA
  - TEG often not allowed to run out that far
  - depends on fibrinolysis
  - norm <7.5%
  - ↓’ed size causes =
    - primary = hyperfibrinolysis \( \Rightarrow \text{ give TXA} \)
    - secondary = DIC = support coag products as required

**Dosing Summary**
- TEG to monitor coagulopathy:
  - \( \alpha \) angle < 45deg - give 1unit/30kg cryo
  - MA <50mm - give platelets (40-50mm = 1 pool; <40mm = 2 pools)
  - R time >10mins - give FFP (11-15 = 1unit; 15-20 = 2units; >20=4units)

**Advantages over Coag Screen**
- PT & APTT:
  - poorly represent cell based model of haemostasis
  - show time to initiation of clot formation - no further info thereafter
- TEG shows:
  - speed to clot formation
  - strength of clot
  - whether excessive clot being formed
- other TEG advs:
  - can see clot trace evolve in near pt testing (coag screen up to 45min in lab)
  - more reliable in liver disease

**Deficiencies of TEG**
- None will assess function:
  - specific deficiencies: Factor 13, Alpha2 antiplasmin deficiency, vWF deficiency
  - antiplatelet agent effects ie aspirin & clopidogrel
- always risk of excessive bleeding
- is a functional reserve in concentration of clotting factors:
  - haemophilia A = no symptoms until factor 8 level <5%
- to determine specific cause for defective clotting need to do
  - specific factor assays
  - tests for anti-factor antibodies
Coag Screen
- targets in ICU
  - INR 1.5-2 ⇒ give 2 FFP; >2 4 FFP
  - fibrinogen <1.5 ⇒ give 1 unit cryo /30kg of body weight
  - platelet transfusion if any of:
    - <50
    - antiplatelet agent in last 5 days
    - high bleeding

Myocardial Protection
- pathophysiology of myocardial injury:
  - ischaemia:
- depletion of high energy phosphates
- intracellular acidosis
- altered calcium homeostasis
- direct myocellular damage

• re-perfusion:
  - intracellular Ca overload
  - generation free O2 radicals
  - complement activation
  - endothelial cell-leucocyte interactions
  - myocellular oedema

• 3 states of myocardial cells:
  • stunning:
    - = post acute ischaemia impairment
    - viable myocardium remains
    - if no further injury ⇒ complete recovery
  • hibernation:
    - chronic underperfusion ⇒ down regulation of contractile elements
    - can be improved with revascularisation
  • myocardial necrosis:
    - = irreversible

Strategies of Protection

• include:
  • Cardioplegia
  • hypothermia
  • LV venting : avoids distension ⇒ ↓wall tension Use TOE
  • de-airing - RCA esp vulnerable to air emboli
  • pre-conditioning
  • glycaemic control

• work by ↑O2 delivery & ↓O2 requirement

Cardioplegia

• antegrade:
  • normal pathway
  • may miss stenotic areas

• retrograde:
  • into coronary sinus
  • drains via coronary ostia into aortic root ⇒ drained via aortic root cannula
  • may miss RV

Hypothermia

• causes:
  • ↓electromechanical activity
  • inhibits apoptosis
  • ↓O2 consumption

• effect is controversial

Preconditioning

• =phenomenon where tissue exposed to brief non lethal ischaemia becomes relatively resistant to damage from subsequent prolong ischaemia

• Advantages:
  • ↓infarct size
  • ↓contractile dysfunction
  • ↓arrhythmias
leucocyte adhesion

- types:
  - early =
    - minutes & lasts 1-3hrs
    - causes: local mediators
  - late =
    - 12-24hrs ⇒ lasts 2-4days
    - causes: altered gene expression

- unknown MOA - theory:
  - up regulation of PKC ⇒ phosphorylation of K-ATP channels ⇒ ↓ed hyperpolarisation ⇒ ↓Ca influx ⇒ ↓phase II of AP
  - K-ATP channels see in mitochondria & cell membrane

- drugs which may trigger =
  - volatiles
  - morphine
  - adenosine
  - ACEI
  - β agonists
  - CaCl

- antagonists of preconditioning:
  - ↓temp
  - ↑BSL
  - methylxanthines
  - sulphonylureas

- Strategies:
  - pre-op ACEI
  - volatiles: 0.5MAC pre CPB; >1MAC post
  - morphine 0.25-0.5mg/kg
  - mild hypothermia only
  - normoglycaemia
  - stop sulphonylureas

**Neuroprotection**

- post CPB:
  - injury or stroke:
    - CABG = 2-6%
    - Valve surgery = 4-13%
  - subtle changes seen in 60% pts in CPB
  - some dysfunction = 35% @ 5yrs
  - POCD 83% pts in 1st week postop

**Risk Factors**

- age >75
- HTN
- carotid stenosis
- DM
- prior stroke
- post by pass ↓bp, arrhythmias
- complex procedures & long CPB time (>2hrs)

**Etiology**

- emboli - gaseous, particulate from calcifications, air, atheroma
• hypoperfusion
• loss autoregulation: DM, ↓ temp, DHCA, prev stroke
• DHCA

**IntraOp Cerebral Monitoring**
• see neurosurgery notes

**Prevention Strategies**
• Surgical strategies:
  • minimise CPB time
• Embolism:
  • TOE - to check for aortic plaques prior to cannulation/clamping
  • minimise no of clampings
  • cell saver for blood
  • filter use
  • de-airing techniques
• Hypoperfusion:
  • pre-op CUSS in high risk patients
  • NIRS
  • CVP & MAP
• Hypocapnia:
  • ⇒ cerebral vasoconstriction ⇒ esp bad with ↓MAP & anaemia
• Inflammation:
  • minimal volume CPB circuit
  • minimise blood transfusions
• other:
  • stable haemodynamics
  • BIS - ?avoid deep anaesthesia
  • control arrhythmias
  • glucose control
  • α-stat pH management = better cerebral autoregulation
  • close temp control
• pharmacological (no drug proven):
  • EEG burst suppression ⇒ 50% ↓ CMRO2
    - thio/propofol/isoflurane
    - ketamine = found to be neuroprotective following cardiac arrest
  ↩ general plan is to ensure EEG isoelectric before on pump & ↓CPB time
• DHCA:
  • hypothermia 15-20C with paralysis
  • anterograde & retrograde perfusion ⇒ cerebral ischaemia tolerance

**Pacemakers**
• routine insertion of epicardial pacing wires
  • ventricle
  • +/- atria

**Indications**
• asystole
• sinus brady with inadequate CO
• heart blocks
• suppression of arrhythmias
Codes

<table>
<thead>
<tr>
<th>Chamber paced</th>
<th>Chamber sensed</th>
<th>Response to sensing</th>
<th>Extra options</th>
<th>Cardioverting options</th>
</tr>
</thead>
<tbody>
<tr>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td>I = inhibited</td>
<td>p = programmable</td>
<td>p = pacing</td>
</tr>
<tr>
<td>A = atrium</td>
<td>A = atrium</td>
<td>T = triggered</td>
<td>m = multiprogramme</td>
<td>s = shocking</td>
</tr>
<tr>
<td>D = dual</td>
<td>D = dual</td>
<td>D = dual</td>
<td>c = communicating</td>
<td>D = dual</td>
</tr>
<tr>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>r = rate adaptation</td>
<td>O = none</td>
</tr>
</tbody>
</table>

- Epicardial pacing ⇒ first 3 functions relevant

**DDD**
- may inhibit or pace each chamber with preserved R-R interval
- if SAN & AVN working ok then will do nothing
- if failure of either node PM will take over
  - benefit by synchronised A & V with atrial kick
- useful unless pt in AF as don’t see synchronised atrial contraction
  - used if competitive underlying rhythm & inadequate AV conduction

**AAI**
- Atrial demand inhibitory mode
- it inhibits itself if senses pts own p waves
- use if not in AF & no significant AV delay
  - used if competitive underlying rhythm & good AV conduction

**VVI**
- Vent demand inhibitor mode
- inhibits itself if senses pts R wave in ventricle
- asynchronous pacing
  - used if competitive underlying rhythm is AF

**DOO**
- dual chamber pacing (A-V sequentially) with no sensing
- good intraop as surgical stim & diathermy will not be mi-sensed
- is a risk of R on T phenomenon with no sensing ⇒ VF
  - used if PM dependant & risk of diathermy

**VOO**
- asynchronous vent pacing
  - risk of R on T when starting mode
  - used if PM dependant & risk with diathermy & AF

**AOO**
- asynchronous atrial pacing
  - used if PM dependant, diathermy & adequate AV conduction
Settings

dynamic depending on pt & lead position:
- rate 80-90/min
- atrial amp = 5v (default 10v on our boxes)
- vent amp = 8V (default 10v on our boxes)
- atrial sensitivity = 0.5mV
- vent sensitivity = 2mV
- AV interval 150ms
- atrial refractory period = 400ms

Practical Issues

- Should be seen for PM check at least <1yr prior to op (ideally within 3months)
- need to know:
  - reason for PM insertion
  - battery status
  - base line rate/rhythm
  - magnet setting - not a modern strategy as diff PMs have different magnet settings
- Concern intraop =
  - electromagnetic interference ⇒ inappropriate pacing/reprogramming/damage
  - pacing wires can act as conduits for heat ⇒ damage endocardium
- diathermy is dangerous, bipolar is safe
  - can minimise risk by placing pad away from PM (>20cm) & using short sharp bursts
- emergency:
  - asynchronous vent pacing = VOO
    - (theoretical risk of VF on initialising VOO but modern machines have safety mechanism to avoid R on T)
- surgeries:
  - MRI - avoid
  - lithotripsy - avoid path of sound
  - ECT - asynchronous
- **disable** ICDs preop if diathermy to be used in case ⇒ shock
  - if need to defib then should place pads away from PM (or better AP pads)
  - avoid sux if possible - fasciculations can effect sensing modes in PM
  - send back for PM check post op

Implantable Cardiac Defibrillators

Indications:
- VF or sudden cardiac death survivors
- VT causing syncope no caused by MI or other correctable cause
- minimally symptomatic VT with EF <35%
- previous MI, LVEF <35% and sustained VF/VT on EPS
- long QT with syncope or FHx of sudden cardiac death
- Brugada syndrome with VT/VF
- arrhythmogenic right ventricular hyperplasia with VF/VF
- HCOM with VF/VT

ICD Code
1. **Shock chamber** (0 = none, A = atrium, V = ventricle, D = dual)
2. **Chamber to which anti-tachycardia pacing is delivered** (0 = none, A = atrium, V = ventricle, D = dual)
3. **Means of detection of tachyarrhythmia** (E = intracardiac electogram, H = haemodynamic means)
4. **Pacemaker code**
- recent counter shock episodes
- effects of magnet interference - many different modes on machines so should assume magnet mode = emergency mode
- continue anti-arrhythmic and anti-failure medications
- CXR and ECG
- deactivation of defibrillation and anti-tachycardia function pre-operatively so diathermy isn't interpreted as VF or VT (very important in lithotripsy and ECT) -> 'monitor only' mode

**Intraoperative**
- ideally have the cardiac technician present to enable the defibrillation function
- have defibrillator close @ hand (attach prior to surgery)
- place pads @ cardiac apex and below right clavicle

**Postoperative**
- nurse in HDU with resuscitation equipment close by
- cardiac technician interrogation and reprogramming post surgery

**α Stat & pH Stat Strategies**

- **pH stat:**
  - benefit = ↓seizure, ↓ICU length of stay, ↓mortality
  - addition of CO2 to pump circuit
  - this corrects for ↑solubility of CO2 at lower temps:
    - PCO2 37deg = 40mmhg ⇒ 20deg = 16mmHg
    - if uncorrected this would obviously ⇒ ↑pH from 7.4 to 7.8
  - ∴ addition of CO2 results in:
    - total body CO2 is ↑ed
    - pH remains the same at lower temp
  - better in DHCA ⇒ improved clinical outcomes

- **α stat:**
  - as temp ↓s H+ & OH- dissociation constant increases ⇒ ↓H & ↑pH
  - CO2 is NOT added to blood
  - ∴ total body CO2 remains same but at low temp pH ↑s
  - at mild-mod hypothermia α stat maintains cerebral blood flow metabolism coupling
    - pH stat obtunds autoregulation
Drugs in Cardiac

Heparin

Heparin Resistance

- causes:
  - hypercoag states:
    - ATIII deficiency
    - septicaemia
    - arteriosclerotic disease, unstable angina, DVT, PE
  - Drugs: heparin, GTN
- Treatment:
  - give more heparin
  - if >600units/kg been given then consider giving supplemental ATIII
  - ATIII found in FFP so give 2-4 units
  - bivalirudin
    - direct thrombin inhibitor
    - rapid & reversible effect (half life 25mins)
    - no specific reversal available
    - clearance unaffected by renal dysfunction
    - monitored with ACT & TEG
- Alternatives to heparin eg in HIT:
  - bivalirudin
  - tirofiban

Adverse Reactions

- excessive bleeding:
  - ↓ed risk with LMWHs
  - ↑ed risk if vit K deficiency or concurrent anti-plt Rx
- hypersensitivity reactions:
  - type 1 IgE mediated eg bronchospasm/anaphylaxis
  - anaphylactoid reaction – involves alternative complement pathway
- thrombocytopaenia:
  - HITTS:
    - = heparin induced thrombocytopaenia
    - if concurrent thrombosis = HITTS
    - 1-6% incidence (much less with LMWH)
    - more frequent with bovine lung heparin
    - 2 types:
      - type 1:
        - onset 1-4 days post exposure
        - transient/self limiting ↓platelets to ~50
        - = direct heparin induced plt agglutination ie non immune mechanism
      - type 2:
        - present 4-14 days after 2nd exposure to heparin
        - platelet ↓ to ~10 & assoc with thromboembolic phenomena
        - immune mediated plt aggregation by PAF 4, IgG & IgM antibodies
        - development of antibodies to platelets following 1st heparin exposure. ie occurs on next exposure
        - = type II hypersensitivity reaction
• usually resolves rapidly on stopping heparin (can last for 2/12)
• must avoid UFH forever, but can use LMWH (with caution)

• thrombosis:
  • prolonged Rx
    • ↓ AT-3 activity
    • ↓ plasmin activity
  • ↑ K:
    o ↑ risk if have problems with K homeostasis
    o MOA:
      • inhibition of final step in pathway for production of aldosterone
      • ↓ number and affinity for AT II receptors ↓ aldosterone secretion
• chronic use of heparin >1 month (e.g., pregnancy):
  o ↓ bone density in 30% pts
  o MOA - ?↓ osteoblasts ↑ osteoclasts
  LMWH less risk

**Protamine**

• administration of protamine on CPB ⇒ death
• only drawn up when needed
• cannot neutralise heparin bound to plasma proteins/endothelial cells
  ⇐ ∴ repeat small boli useful
• dose = 1mg/100 units of heparin used
• infuse over 10 mins
• side effects:
  • histamine induced ↓ bp
  • pulmon vasoC
  • anaphylaxis
  • anticoagulant effect - impairs platelet activation, transient thrombocytopenia

**Tranexamic Acid**

• = antifibrinolytic
• useful if given before CPB
• bolus:
  • 10-25mg/kg bolus
  • 1-5mg/kg/hr (need to ↓ in renal dysfunction)
• side effects:
  • ↓ seizure threshold
• note if critical LMS stenosis may consider giving after heparin

**Aprotinin**

• No longer used
• BART trial
  • = aprotinin vs lysine analogues
  • danger in aprotinin with ↑ ed:
    - cardiogenic shock
    - CHF
    - MI
    - trend to massive bleeding
rFVIIa

- off label use
- no good RCT in cardiac surgery
- but observed trend towards thromboembolic complications
By Surgery

on Pump CABG

• = bypass stenosis in coronary artery with arterial or venous graft

Preoperative

• common medical co-morbidities:
  • HTN
  • COPD/smoking
  • DM
  • stroke
  • CRF
  • Hx of angina/ACS

Cardiac Assessment

• Assess LV:
  • failure signs eg PND, orthopnoea
  • exercise ECG
  • angiography - within 12months
  • ECHO - TOE or TTE
  • Exercise tolerance

Premed

• anxiolysis & stress can lead to comorbidity
• drugs:
  • night time sedation eg zopiclone
  • morning: benzodiazepine
• continue all meds except:
  • antiplatelet agents
  • ACEIs

Perioperative

MAP 65-70mmHg, HR 65-90, have GTN ready

• Induction
  • drugs:
    - fentanyl 10-15mcg/kg
    - propofol uncommonly used
  • A line awake, CVL asleep
  • monitoring:
    - 5 lead ECG- II for rhythm, V5 for ischaemia

• Maintenance
  • anticipate surgical steps:
    - sternotomy is painful (more fentanyl) & must temporarily stop ventilation (empty lungs)
    - low SBP for aortic cannulation
    - heparin prior to bypass

• End of case:
  • optimal filling vital at end due to:
    - bleeding
    - diuresis - mannitol
    - vasodilation due to re-warming
  • if cardioplegia used - temporary pacing wires generally inserted
Postop
- ICU
- check bloods & TEG/clotting
- CXR
- Monitor drain output
- WWW
- SBP <120-140mmHg

Special Points
- If severe L mainstem disease:
  - must maintain diastolic pressures & normal HR to preserve perfusion
- If unstable angina:
  - use PAC
  - intra-aortic balloon pump
- thoracic epidurals are used in some centres:
  - adv = excellent pain relief & haemodynamic stability
  - disadv = risk of epidural haematoma & paraplegia
- if arterial grafts used eg internal mammary or radial:
  - prone to early spasm
  - use GTN infusion post op

Off Pump CABG
- ↓ed risks & complications compared to on pump:
  - mortality 2.9 ⇒ 2.3%
  - complication rate 12% ⇒ 8%
- advs may include less:
  - stroke
  - dissection
  - fluid
  - K given
  - expense
- disadv:
  - suboptimal revascularisation
  - ↑graft failure
- as for on pump but use a ‘stabiliser’ to keep heart as still as possible
- pt usually heparinised in case of urgent need for CPB
- 1-10% will need to go on CPB
- keep pt warm & well filled
- avoid ↑HR - β blockers or CCBs
- ↑risk of arrhythmia with handling - keep K & Mg high/normal
- ↓MAP common when surgeon manipulates heart:
  - heart tilted to vertical position - blood has to flow upwards into ventricle
  - A/V regurg common
  - ↑preload, Trendelenburg, IVF & vasopressors, Rx tachycardia
- for R/post descending coronary artery grafts: pt placed in Trendelenburg to ↑VR

Emergency CABG (Failed PCI)

Preoperative
- pt may be peri-arrest with need to correct ischaemia
- good arterial access should remain from cath lab ie femoral lines
- aim for CVS stability with inotropes/vasopressors
• IABP if time can help:
  • ↑diastolic pressure ⇒ ↑CBF
  • augment LV ejection ⇒ ↑LV function
• consider CVL preop
• pts may have been given multiple antiplatelet agents ∴ will require platelets post bypass

**Perioperative**
• Induction
  • PAC if time able
  • CVS stable induction
  • adrenaline as required

**Postop**
• restart IABP
• no urgency to extubate
• high risk of renal dysfunction

**Aortic Valve Replacement: Stenosis**
(if mixed stenotic & regurgitant lesions then should manage dominant lesion)
• commonest valve replacement (severe AS 2% 65-85; 4% >85)
• Stenosis ⇒ effects on the LV:
  • myocardial hypertrophy with no ↑in LV volume (concentric)
  • poor diastolic function ie relaxation (stiff non compliant)
  • high wall tension
  ▸ ⇒ ↑O2 demand & higher filling pressures
  • if long standing severe AS can ⇒ LV failure:
    - ↑ing LVEDP ⇒ MR ⇒ ↑pulmon art pressure ⇒ potential RV failure
• type of valve used:
  • younger pts = mechanical valves as longer lasting but need warfarin
  • older pts = homograft (biological tissue) - anticoag not required but last ~15yrs
• Aortic stenosis = always slow

**Preoperative**
• change in rhythm eg AF can ⇒ LV failure
• Ax LV function & CBF = ECHO & angiography

**Parameters of Assessment**
• significant obstruction to LV outflow =
  • LV-aorta gradient >40mmHg
  • aortic orifice <0.8cm
• Surgery indicated if gradient:
  • (good LV) >70mmHg
  • (poor LV) >50mmHg
• If known gradient (with small area) is ↓ing ≈ LV failure

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Grading of AS in adults: BSA, body surface area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Aortic jet velocity (m s⁻¹)</td>
<td>&lt;2</td>
</tr>
<tr>
<td>Peak gradient (mm Hg)</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Mean gradient (mm Hg)</td>
<td>&lt;5</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
<td>3–4</td>
</tr>
<tr>
<td>Valve area indexed (cm² m⁻² BSA)</td>
<td>&gt;0.85</td>
</tr>
</tbody>
</table>

Cardiac - 41
Perioperative

• Induction
  • HR = avoid extremes - esp tachycardia
    \[\downarrow \text{diastolic time} \Rightarrow \downarrow \text{CBF} \& \uparrow \text{O2 demand}\]
  • SR gives atrial kick which can contribute ~20-40% of filling into stiff ventricular
  • Preload:
    - maintain to aid LV filling
    - beware vasodilators
  • SVR:
    - maintain with \(\alpha_1\) agonists eg metaraminol or norad
    - any \(\downarrow\) in diastolic pressure \(\Rightarrow\) critical \(\downarrow\) CBF
  • Contraction - stiff/thick ventricle may need adrenaline

• Maintenance
• End of case:
  • TOE to assess LV function & valve performance

Post-Bypass

• AV node damage may require pacing
• preload:
  • volume remains essential - ventricle still stiff
  • use Dual pacing to gain coordinated atrial contraction
• PAC may help to guide filling

Special Points
• good myocardial protection by cardioplegia \(\Rightarrow\) good outcomes

Transcatheter Aortic Valve Implantation

• commonly used in frail elderly requiring AVR
• other indications:
  • porcelain aorta
  • severe kyphoscoliosis
  • sig cirrhosis
• mediastinal radiotherapy
• can be done
  • transapically - mini-thoracotomy
    - requires more analgesia
    - blood ready in angio suite for apical ventriculotomy
    - consider cell salvage
  • transfemorally - percutaneous
    - may be done under LA if TOE not required

Preoperative
• Set up as for CPB

Perioperative
• Procedure involves rapid intermittent ventricular pacing - allows
  • valvotomy
  • placement of prosthetic valve
• Prior to atrial pacing:
  • SBP >100mmHg
  • adrenaline ready in case of stunning
• stop ventilating when valve is being positioned to stabilise field
• Post valve insertion may see marked ↑bp
  → have esmolol, GTN ready

Postop
• early extubation is possible with TAVI
• complications:
  • cardiac - any incl tamponade
  • vascular - eg rupture, tearing
  • neurological
  • valve regurgitation - minor paravalvular regurg occurs in >50%

Aortic Valve Replacement: Regurgitation
• usually assoc with
  • aortic root dilation or dissection
  • +/- Hx of endocarditis
    → generally not septic when operated on
• cardiac physiology in AR:
  • LV overload
  • LV dilatation
  • ↑SNS drive ⇒
    • ↑HR
    • ↑contractility
    • ↑periph vasoC
    • fluid retention ⇒ ↑preload

Preoperative
• surgery indicated if symptomatic
  • angina = end stage symptom

Perioperative
• regurg lesion = classic: full, fast & forward
• consider:
  • HR:
- fast HR \(\Rightarrow\) ↓ diastolic time \(\Rightarrow\) ↓ regurg
- CO is rate dependant . aim for ~90/min
- ↓ DBP \(\Rightarrow\) ↓ perfusion pressure so ↑ HR \(\Rightarrow\) ↑ DBP \(\Rightarrow\) ↑ CBF

**Preload:**
- LV stiff & large . need adequate filling
- SR is useful but pts often in AF
- consider Dual pacing

**SVR:**
- anaesthesia \(\Rightarrow\) ↓ SVR \(\Rightarrow\) ↓ regurg fraction \(\Rightarrow\) forward flow
- vasodilators are similar but may also \(\Rightarrow\) ↓ VR/preload so need caution
- too much ↓ SVR may need to \(\Rightarrow\) ↓ DBP \(\Rightarrow\) ↓ CBF \(\Rightarrow\) caution

**contraction:**
- if LV function poor \(\Rightarrow\) inotropic/inodilator support

---

### Special Points

**IABP:**
- contraindicated in AR
- may be useful with replacement valve post CBP

**careful bp control post CPB to prevent rupture of root:**
- SBP<120
- MAP>65

### HOCM /SAM

- HOCM = bulging of septum \(\Rightarrow\) occlusion of LVOT \(\Rightarrow\) ↑ vent pressures \(\Rightarrow\) vent dilatation \(\Rightarrow\) MR \(\Rightarrow\) ↑ LA size \(\Rightarrow\) malignant arrhythmia
- SAM - ant mitral valve leaflet occludes LVOT in systole
  \(\downarrow\) can co-exist

**Perioperative**

need to keep ventricle open . if ↓ MAP \(\Rightarrow\) IVF & α agonists

**goals:**
- preload: keep up = diastolic dysfunction
- SVR: keep up
- contractility:
  - avoid increasing
  - -ve inotropes are useful eg βblocker, Ca channel antagonists, volatiles
- HR: 60-80 to allow diastolic filling
- rhythm: SR critical, AF common

**post correction:**
- same goals
Mitral Valve Replacement: Stenosis

(stenotic lesion = slow & steady)

- prosthetic mitral valves often mechanical
- most pts anticoag’ed due to chronic AF

Preoperative

- classic presentation (pregnancy in exam):
  - frail
  - flushed
  - AF on warfarin
  - fixed cardiac output
  - +/- Pulmon HTN
- almost always 2nd to rheumatic heart disease
  \[\rightarrow\] may have been asymptomatic for >20yrs

- indications for surgery:
  - SOB on mild exertion/rest

- medications:
  - cont anti-arrhythmics
  - convert warf to heparin

- Investigations:
  - ECHO -
    - need to Ax pulmon artery pressures
    - vent function
  - angio

- numbers:
  - valve areas similar to AS (normal is a bit bigger) 4...2...1

<table>
<thead>
<tr>
<th></th>
<th>Norm</th>
<th>Mild</th>
<th>Mod</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Gradient</td>
<td>&lt;5</td>
<td>5-10</td>
<td>&gt;10</td>
<td></td>
</tr>
<tr>
<td>Valve Area cm²</td>
<td>4-6</td>
<td>&gt;1.5</td>
<td>1.5-1</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Peak Pressure</td>
<td>&lt;30</td>
<td>30-50</td>
<td>&gt;50</td>
<td></td>
</tr>
</tbody>
</table>

Perioperative

- cardiac physiology:
  - HR
    - mitral flow is relatively fixed
    - keep HR <100
    - SR if possible
    \[\rightarrow\] allows max diastolic time & CBF
  - Preload:
    - does not need to be augmented pre CPB
    - too much preload may \(\Rightarrow\) RV failure
    - too little preload \(\Rightarrow\) ↓↓bp
  - SVR:
    - VERY afterload dependant
    - chronic fixed CO usually \(\Rightarrow\) compensatory ↑SVR to maintain MAP
    - avoid ↓ in SVR otherwise \(\Rightarrow\) ↓DBP \(\Rightarrow\) ↓CBF
- avoid venodilation otherwise \( \Rightarrow \) ↓VR \( \Rightarrow \) ↓CO \( \Rightarrow \) heart unable to compensate

- PVR - pulmon HTN 2nd to ↑PVR & ↑Pulmon art pressure may be partially reversible
  - ↓consider techniques to ↑pulmon vasoD:
    - maintain filling pressures
    - avoid ↓PaO2 & ↑PaCO2 (more impt than concern about ↑ITP)
      \( \Rightarrow \) ↑IPPV \( \Rightarrow \) ↑ITP \( \Rightarrow \) ↑PVR but vasodilation more impt
    - avoid acidosis - check ABGs regularly
  - pulmonary vasodilators:
    - use if PAP > \( \frac{2}{3} \) systemic pressure
    - choices:
      - Nitric oxide
      - inhaled prostacyclin
      - PDE inhibitors eg sildenafil, milrinone

- contraction:
  - severe MS \( \Rightarrow \) pulmon HTN \( \Rightarrow \) RV failure
  - LV normally unaffected until end stage disease
  - inodilator (adrenaline) may be required if RV is failing

- HR:
  - surgery may disrupt conducting pathways
  - pacing +/- chronotropic agents may be required

**Induction**

- full monitoring
- PAC - helps to Ax:
  - PAP
  - PVR
  - filling
  - inotropic requirement

**Post CBP**

- TOE following valve insertion
- targets:
  - preload:
    - obstruction to flow has been removed so keep well filled
  - SVR:
    - ↓SVR will now encourage forward flow
  - PVR: maintain pulmon vasoD \( \Rightarrow \) ↑ed RV & LV function
  - Contraction:
    - RV support via inotropic agents eg adrenaline

**Special Points**

- PAPs take days/weeks to ↓:
  - continue good pulmon vasoD care as above

**Mitral Valve Replacement: Regurgitation**

*(fast & full & forward for a regurgitant lesion)*

- causes incl:
  - primary = myxomatous degen, rheumatic disease, endocarditis, MV prolapse
  - secondary = ischaemia to papillary mms, dilated LV

**Preoperative**

- comorbidities:
  - IHD \( \Rightarrow \) ACS \( \Rightarrow \) papillary mm rupture
  - acute regurg \( \Rightarrow \) pulmon oedema
- AF - in 75%
- R heart failure: MR ⇒ ↑PAP ⇒ RVF

**ECHO**
- LV function often over-estimated preop (EF <60% = severe LVF)
  - pulmon circulation provides low pressure release system for poor LV
  - regurgitant fraction:
    - **severe** MR = regurg jet fills LA >8cm²
    - **mild** MR = regurg jet fills LA <4cm²
- severity relates:
  - PAP
    - chronic significant MR = ↑ed PAP

---

**Perioperative**

**Cardiac Physiology**

- **HR**:
  - HR >70 ⇒ minor effect on systole length
  - less imp than in AR but encourages forward flow

- preload: filled
- SVR:
  - ↑SVR ⇒ ↑regurgitation ⇒ avoid vasoconstrictors
  - if ↓bp ⇒ give IVF
- PVR:
  - avoid pulmon vasoC due to R heart failure. (see MVR:stenosis for vaso D care)
- Contractility:
  - rarely needed pre bypass
  - if acute MR: IABP ⇒ ↓afterload & ↑LVF function

**Induction**

- PAC always indicated to Ax PAP

**Post Bypass**

- LV function:
  - replacement valve ⇒ LV has to work harder (no pressure release to pulmon circulation)
  - risk of LVF & need for inotropes/inodilators

- Preload:
  - adequate filling
- SVR:
  - ↓SVR will benefit forward flow & CO
- PVR:
  - vasculature often reactive & can spasm vasoC ⇒ good vasoD care impt
  - adequate filling will prevent pulmon vessel collapse ⇒ ↑PVR
- Contraction:
  - inotropic support likely

**Special Points**

- IABP may be helpful in short term for failing LV
- Pacing required if conduction system damaged
- Repair instead of replacement is ↑ing in frequency: Anaesthetic Rx is same
Cardiac Tamponade

- key questions:
  - local (eg around RA) or global
  - acute or chronic
- signs:
  - pulsus paradoxus + electrical alternans
- ideally drain while awake
- use ketamine
- keep spont breathing if possible until surgeon ready

Perioperative

- targets:
  - preload = ↑ give volume
  - afterload = unchanged
  - contractility = ↑ i.e. avoid -ve inotropes
  - HR = ↑ as CO is rate dependant
- Post correction can deepen anaesthesia

Induction

- classically keep spont venting if practically possible
- prep pt pre-op before starting

Thoracic Artery Surgery

- replacement of ascending aorta with tubular graft
- may need deep hypothermic circulatory arrest
- 2 main pathologies:
  - aneurysm
  - dissection
- causes of aneurysm:
  - HTN
  - hereditary conditions eg Marfans syndrome
- comorbidites:
  - IHD - 65%
  - dilation of root with AR
- aneurysms classified:
  - type A = ascending aorta to brachiocephalic artery
    - surgical Rx
  - type B = arch & descending aorta
    - medical Rx
- if arch Rx is planned - need deep hypothermic circulatory arrest
- emergency or planned operations
- if aortic root involved may need:
  - AV replacement
  - re-implantation of coronary arteries

Type A Corrections

- high mortality 11-30%
- may require
  - femoral CPB pre-induction/sternotomy
  - DHCA
Preoperative
• Emergency dissections Rx:
  • control bp & HR:
    - SBP <120, HR <100
  • bleeding
    - consider entering a PA sheath or RIC line
    - X match 6 units urgently
    - warn lab of potential for MTP
  • fluid resus - guarded. permissive hypotension is ok if pt mentating & conscious

Perioperative
Induction
• must avoid ↑SBP otherwise may ⇒ rupture
• once stable should Rx:
  • HR - avoid ↓ & ↑HR
  • SVR - reduce
  • preload - keep filled
  • contractility - avoid inotropes otherwise may ⇒ rupture of dissection extension
• dissection/surgical clamps may interfere with invasive arterial monitoring
• TXA should be used
• femoral artery cannulation usually required:
  • done because ascending aorta is to be resected
  • prior to cannulation: heparin (300IU/kg) & ACT >400s

Post Bypass
• bleeding & control of arterial pressure v impt
  • SBP must be kept <120
• dissection may have involved renal & mesenteric vessels ⇒ monitor kidney/gut perfusion
• steep head down tilt used to allow air out of aortic graft

Deep Hypothermic Circulatory Arrest (DHCA)
• If aortic arch is to be operated on it is likely required
• 8-15% mortality; stroke 7-11%
• key points:
  • protection of CNS
  • not possible to perfuse cerebral vessels reliably on bypass
• ↓temp effects:
  • ↓CMRO2 - 6-7% for every 1deg C
  • protects cerebral integrity during reperfusion
• max safe duration of deep hypothermic circulatory arrest (DHCA) @ 18degC
  • adults ~ 45min
  • neonates ~ 60mins
• cerebral protection in DHCA:
  • head packed in ice
  • careful cooling & warming
  • acid base management - pH stat used as better outcomes in DHCA
  • haemodilution - to HCT of 20%
  • glycaemic control
  • drugs added to pump prime:
    - barbituate eg thiopental 7mg/kg
    - steroids eg methylprednisolone 15mg/kg - need to be given ~6-8hrs prior to procedure
    - mannitol 0.5g/kg
  • surgical procedures to perfuse brain:
- retrograde cerebral perfusion - cold oxygenated blood sent into SVC
- anterograde perfusion - via catheters into both common carotid arts
  → allow slightly less cold temps to be used eg 22-25degC
• shorter DHCA the better as post op neuro problems is proportional to time of DHCA
• DHCA initiation:
  • vasodilator (eg GTN) used - aids rapid cooling & prevents reflex cerebral vasoC
  • circulation is arrested - all infusions & pumps stopped
  • measure 2 temps:
    - core (bladder) - must reach <20degC
    - nasopharyngeal temp = cerebral temp
• DHCA rewarming:
  • remove head ice packs
  • switch on warming blankets - set <10degC above pt temp to avoid burns
  • start propofol infusion - as tolerated
  • check coags - frequently require products
  • vasodilator eg GTN can be used to maintain vasoD & help rewarming
  • mannitol (0.5g/kg) may be given to encourage diuresis
  • core temp @35degC ⟹ start inotrope to ↑ cardiac function
• coming off bypass:
  • takes time to rewarm
  • temp goals to allow attempts to come off CPB:
    - skin = 33degC
    - core >37degC
• Complications:
  • stroke
  • other neurological impairment
  • coagulopathy

**Pulmonary Thromboembolectomy**
• =use of CPB to allow embolism retrieval

**Preoperative**
• pt often collapsed with resuscitation in progress
• a healthy heart requires 50-80% of pulmonary trunk to be obstructed before RV failure
• pt may have received thrombolysis
• urgent CPB is essential for chance of survival

**Perioperative**
• if decision made to attempt surgery move with no delay

**Induction**
• rapid induction
• inotropes as required
• heparin 300IU/kg
• start CPB asap

**Maintenance**
• massive airway haemorrhage may be a problem with difficult ventilation:
  • frequent suctioning
  • DLT

**Post Bypass**
• HR - normal
• Preload - keep well filled
• Contractility - Inotropic support likely
SVR - ↓SVR via vasodilators can be useful

Pulmonary pressures:
  - likely ↑reactivity
  - good pulmon vasodilators care imp
  - adrenaline to support RV function may be required

**Postop**
- Delay heparinisation post CPB for at least 24hrs to ↓surgical bleeding

**Special Points**
- ↑↑R sided pressures may ⇒ opening of foramen ovale ⇒ R to L shunt ⇒
  - hypoxia
  - ?stroke if emboli showering
- Capnography monitoring is not reliable on induction:
  - caused by ↓↓pulmonary blood flow
  - following successful embolectomy ⇒ dramatic improvement in EtCO2

## Redo Cardiac Surgery

### Risks
- RV laceration ⇒ severe bleeding
- arrhythmia
- damage to prev coronary grafts

### Preoperative
- often poor LV function

### Perioperative

#### Induction
- standard monitoring
- risk of torrential bleeding - RV may be stuck to underside of sternum
  - wide bore access essential eg PA sheath
  - have blood in theatre
  - femoral cannulation prior to starting with CPB team on standby for urgent on bypass

#### Maintenance
- risk of VF at sternotomy/dissection of adhesions - have defib pads on patient
  - use of diathermy may obscure your ECG monitoring - look at A line trace
- damage to prev coronary graft during dissection possible - look at ECG
- coagulopathy is common -
  - TXA
  - monitor TEG

### Postop
- ↑ed risk of post op bleeding
- poor LV function may be problematic

## Cardioversion

### Preoperative
- commonest rhythm = AF
- ensure normal K & Mg prior to procedure
- if AF >24hr:
  - LA must be checked for clot with TOE prior to cardioversion
    - can use propofol sedation
• if clot ⇒  
  - delay for 3 weeks of anticoagulation  
  - continue anti-coag for 4 weeks post cardioversion

**Perioperative**

**Induction**
• guarded induction - low doses required  
• use FM unless aspiration risj  
• obese pts with difficult airway can be defib’ed laterally  
• reduced doses of energy needed  
• ensure synchronisation

**Implantable Defibrillators**
• used for pts at risk of malignant arrhythmias  
• procedure complexity varies widely:  
  - simple = 2 venous wires placed transvenously to R heart - sensing & shock  
  - complex =  
    - replacement PM  
    - coronary sinus catheterised to gain access to LV myocardium  
• during procedure VF induced to test device  
  ↳ pt should be sedated  
• usual access is via L cephalic vein & fluoroscopy

**Preoperative**
• Assess functional cardiac reserve:  
  - LA & sedation if pt compromised  
• check if on anticoagulants

**Perioperative**
• remote anaesthesia

**Induction**
• A line if ↓ contractility  
• prophylactic Abx’s used  
• sedation plan:  
  - propofol TCI  
  - midaz & fentanyl  
  ↳ deepen prior to VF testing  
• if defib unit is to be placed sub-muscular - may need period of deeper sedation

**Post Op**
• location to recover pt must be considered

**Special Points**
• post VF/defib - may see periods of myocardial stunning & ↓bp ⇒ support as required  
• existing PM & diathermy being used ⇒ may lose PM function

**Transcatheter Ablation Procedures**
• broadly same concerns as for implantable defib  
• long term success ~70%  
• technique:  
  - pulmonary vein ostial segmental disconnection (OSD)  
  - L atrial circumferential ablation (LACA) - need to pass wires through foramen ovale  
• procedures take >3hrs  
• plan:
- light GA +/- ETT depending on need for TOE
- TCI prop & remi:
  - volatiles may suppress AVNRTs
- full monitoring
- keep warm

Practicalities
- non stimulating long procedure
- if converting AF -
  - must exclude atrial thrombus first with TOE ∴ need ETT
  - septal puncture requires heparin to ACT 25-300
- pt must not move: remi or NDNMBs
  - systems adjust for resp movement but low VTs often used
- if hypotension must always consider aortic puncture, pericardial effusion/tamponade
- positioning:
  - on table is difficult
  - watch for brachial injury if arms above head
- NG tube:
  - locates oesophagus
  - can get tubes with rapidly reacting thermistors ⇒ warning of heat injury to oesophagus
  - withdraw TOE probe into prox oesophagus during ablation

Complications
- worldwide mortality of 1:1000
- must always consider pericardial bleeding & exclude
- Cryo -balloon -
  - phrenix nerve close to R upper pulmon vein
  - phrenic nerve is paced & diaphragm action is monitored
    - ∴ must not be paralysed at this point
- risk factors:
  - >75yr
  - heart failure

Percutaneous Closure Atrial Septal Defects
- 2 indications for septal closure:
  - ASD for shunt lesion:
    - causes:
      - ostium secundum = only one amenable to percutaneous closure
      - ostium primum
      - sinus venosus
      - unroofed coronary sinus
    - ECG: RBBB & axis deviation (direction depending on type)
  - PFO for cryptogenic stroke
    - failure of primum & secundum to adhere at one edge of fossa ovalis
    - PFO seen in up to 27% of people
      - L to R flow - usually limited by a flap
      - R to L flow - limited by ↑L atrial pressures
    - ECHO done after stroke/TIA to exclude PFO
    - closure can ↓risk of further stroke 33% to 7%
Preoperative
• standard

Perioperative
Induction
• TOE essential ⊙ ETT
(intracardiac ECHO is evolving ⇒ inserted via fem vein meaning sedation is sufficient

Maintenance
• heparin required

End of case

Postop
• Pt to remain supine for 2hours until venous sheath removed
• antiplatelets prescribed post op

Special Points
• Complications:
  • Air embolism
  • arrhythmias - AF & flutter common
  • thrombus on device
  • embolisation of device
  • pericardial haemorrhage & tamponade

Anaesthesia for patients with A Cardiac Transplant
• 1yr survival = 90%; 10 yr = 50%

Complications of Cardiac Transplant
• include:
  • donor coronary artery disease
  • rejection
  • immunosupression
  • associated disease eg DM, epilepsy, HTN (2nd to ciclosporin), pre-eclampsia
  • atypical infections eg CMV, listeria, toxoplasma gondii

Donor Coronary artery Disease
• =commonest cause of death >1yr post transplant
• is immunologically mediated ie not from pre-existing atherosclerosis
• pts presentation:
  • will not get chest pain - as heart not innervated
    ↩ although long term may see some sympathetic re-innervation ⇒ CP
  • symptoms of LV dysfunction
  • arrhythmias
• must maintain CPP during anaesthesia

Rejection
• acute rejection - pt in 1st yr
• avoid cannulating RIJ if possible - site of biopsy screening
• S&S of rejection:
  • unexplained weight gain / fluid retention
  • fever
  • ↓function on ECHO

Immunosupression
• triple Rx:
- azathioprine
- cyclosporin - nephrotoxic, multiple interactions
- prednisone
- common side effects:
  - infection
  - malignancy
  - mSK problems
  - chronic renal impairment

**Physiology**

**Atria & SAN**
- donor atria are sutured to remnants of recipient atria
- recipient atria remain electrically active but limited impact on ECG
- donor heart dependant on its own sinus node
- loss of blood supply to SAN ⇒ persistent brady cardia requiring PM
- RBBB occurs in 10% of donor hearts

**Innervation & HR**
- donor heart has no autonomic innervation
- resting HR 90-100 due to no resting vagal tone
- see no reflex HR changes with stimulating activity eg laryngoscopy
- see rapid swings in bp: no intrinsic rapid homeostatic adjustments in HR to changes in drug induced ↓↑SVR
  \[ \Rightarrow \vdots \text{ must maintain good preload} \]
- exercise response:
  - blunted response with gradual HR changes only
  - from endogenous catecholamine response

**Innervation & Drug Response**

**Table 2 Pharmacology after cardiac transplantation**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effect in recipient</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>4-fold increase in sinus and atrioventricular nodal blocking effect</td>
<td>Denervation supersensitivity</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Minimal delay in atrioventricular nodal conduction</td>
<td>Denervation</td>
</tr>
<tr>
<td>Atropine</td>
<td>No effect on heart rate</td>
<td>Denervation</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Increased contractility and chronotropy</td>
<td>Denervation supersensitivity</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Increased contractility and chronotropy</td>
<td>Denervation supersensitivity</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>Normal chronotropic effect</td>
<td>Denervation</td>
</tr>
<tr>
<td>Glyceryl trinitrate</td>
<td>No reflex uchycardia</td>
<td>Baroreflex disruption</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>No tachycardia</td>
<td>Denervation</td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>No bradycardia</td>
<td>Denervation</td>
</tr>
<tr>
<td>Neostigmine</td>
<td>No bradycardia</td>
<td>Denervation</td>
</tr>
</tbody>
</table>

- loss of action of anticholinergics eg atropine
- no action of dig as vagal activity no longer influences HR

**Anaesthesia**
- keep simple, maintain preload, use direct acting chronotropics (atropine doesnt work)

**Preoperative**
- seek follow up program results from transplant coordinator:
  - ECHO
  - rejection status - biopsy
  - coronary diseases - angio
  - CMV status
• seek advice about periop immunosupression ?given IV

**Perioperative**
- practically impt issues:
  - blood transfusion - must receive CMV -ve blood
  - strict asepsis for all procedures

**Induction**
- normovolaemia vital to maintain preload
- monitoring:
  - PAC - if no PM
  - intraop TOE - preload & contractility

**Maintenance**
- if hypotension:
  - IVF
  - vasocontractors
- direct acting chronotropic agents ready eg adrenaline & isoprenaline
- ext pacing machine also required

**Postop**
- invasive lines should be removed asap ⇒ ↓risk of infection

---

**Correction of Congenital Heart Disease**

**Preoperative**
- psychological preparation for pt & parents
- key areas:
  - History:
    - Risk factors for respiratory adverse events
  - Bloods -
    - any sign of infection may cause problems post CPB
  - Cardiac failure -
    - pressure or volume overload?
      - poor feeding
      - failure to gain weight
      - sweating
      - ↑HR, ↑RR, hepatomegaly
  - pulmonary HTN = PAP>25mmHg at rest?
    - x8 more likely to have complications
    - risk in pts with:
      • L to R shunt
      • obstructed venous drainage
      • ↑LAP
    - common in AV septal defects, tricuspid atresia, trisomy 21
    - need careful pulmon vasc care on cessation of CPB
  - cyanosis?
    - very high risk group as often have concurrent failure, pHTN, arrhythmias
    - causes ↑risk of
      - bleeding
      - hyperviscosity - can lead to intracerebral thrombosis

↓ these children have poor cardiac reserve, will tolerate ↓SVR very poorly
↓ often have large L to R shunt ⇒ high FiO2 will ↑shunt flow rather than systemic perfusion
- RFs:
  - <5yr ⇒ cerebral vein & sinus thrombosis
  - dehydration
  - fever
  - IDA
- use fluids to ↓Hb if >180
- consider TXA & ready blood products

- Arrhythmias:
  - all should have preop ECG
  - RBBB common but benign
  - high risk:
    - Vent ectopics worrisome - ~30% die suddenly
    - single ventricle circulation ~30% death arrhythmias
  - low risk: ventriculotomy or RV to PA conduit with normal ECG

- potential sites for vasc access
- other congenital syndromes?
  - Di George - need irradiated blood products
  - CHARGE syndrome -
    - choanal atresia ⇒ no nasal intubation
    - midface hypoplasia & micrognathia ⇒ difficult oral intubation

Perioperative

Induction Drugs
- circulation times longer - wait
- ketamine IV induction agent of choice:
  - no effect on SVR
  - ↑MAP
  - well tolerated in pHTN
- propofol not liked:
  - worsen cyanosis in children with R to L shunt as ↓↓SVR
- neonates have rate dependant CO:
  - pancuronium liked as ⇒ THR
- fentanyl good - high doses may delay extubation

CPB
- priming volume of pump = >x2 child total blood volume ⇒ risk of haemodilution
  - often add blood usually added to prime
- target MAPs vary by age:
  - neonate = 30mmHg
  - young adult = 50mmHg
- decision on α stat & pH stat:

Post CPB
- modified ultra filtration (MUF):
  - action:
    - removes excess body water ⇒ ↑haematocrit
    - removes some inflam mediators
  - shown to ↑cardiac output & ↓SVR

Postop Complications
- divided into:
  - arrhythmias
  - significant bleeding:
    - >5ml/kg in first 2 hours
- 1ml/kg in subsequent hours
  - blood loss >10ml/kg ⇒ surgical r/v
  - sudden ↓ in drain output ⇒ suspect tamponade
- SIRS -
  - peaks 8-12 hrs post op
  - MUF & steroids
- pHTN:
  - ↑RV afterload ⇒ ↓LV preload ⇒ ↓CO
  - ↓lung compliance ⇒ ↑WOB
  - start pHTN care
- low cardiac output syndrome

### Table 2: Specific complications associated with specific lesions

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Complication</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVSD</td>
<td>Atrioventricular valve regurgitation</td>
<td>Minimize overload or stretch on the repaired valve using afterload reduction</td>
</tr>
<tr>
<td></td>
<td>Arhythmias</td>
<td>(milrinone, SNP)</td>
</tr>
<tr>
<td></td>
<td>Pulmonary hypertension</td>
<td>Maintain normothermia, correct electrolyte abnormalities</td>
</tr>
<tr>
<td></td>
<td>RV outflow tract obstruction</td>
<td>Avoid hypoxia, hypercapnia, acidosis</td>
</tr>
<tr>
<td>TGA</td>
<td>Corona ischaemia</td>
<td>Avoid overtension of heart, use small (5 ml kg⁻¹) fluid bolus’s</td>
</tr>
<tr>
<td></td>
<td>LV dysfunction</td>
<td>Afterload reduction</td>
</tr>
<tr>
<td></td>
<td>Arhythmias</td>
<td>Maintain normothermia, correct electrolyte abnormalities</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>RV dysfunction, associated with RV failure and LCOS</td>
<td>RV afterload reduction; maintain high CVP, reduce PVR, reduce LAF (improve contractility). Milrinone reduces PVR and improves diastolic function</td>
</tr>
<tr>
<td></td>
<td>Arhythmias</td>
<td>Maintain normothermia, correct electrolyte abnormalities</td>
</tr>
<tr>
<td>Single-ventricle repair, e.g. Fontan</td>
<td>Cardiac output is dependent on PBF, where PBF=(CVP–LAP)/PVR</td>
<td>Keep CVP high (head-up and elevate legs); LAP low (maximize contractility, maintain sinus rhythm, consider milrinone); PVR low (good oxygenation and analgesia; early spontaneous ventilation; avoid amebactia)</td>
</tr>
<tr>
<td></td>
<td>Pleural effusions and liver dysfunction</td>
<td>Monitor and treat accordingly</td>
</tr>
</tbody>
</table>

### Special Points
- Some surgery can be done without CPB:
  - coarctation of aorta
  - PA banding - done to limit excess pulmon blood flow as temporising procedure
  - shunt procedures - augments pulmonary blood flow

### Fontan Circulation
- used in patients with congen heart disease & 1 ventricle
- procedure diverts all venous blood into the pulmonary vasculature without passing into the heart
- systemic & pulmonary systems in parallel & survive due to shunts
- palliative procedure

### Indications
- all congen heart defects where biventricular repair not possible eg
  - tricuspid atresia
  - pulmonary atresia with intact vent septum
  - hypoplastic L heart syndrome
  - double inlet LV or double outlet RV
  - complete AV septal defect
• preconditions:
  • SR
  • good vent function
  • good sized pulmon arteries

**Surgical Technique**
• multi staged process not done in neonates due to high PVR
• interstage mortality 5-30%
• step 1 = systemic-pulmonary shunt (eg Blalock Tausig Shunt) (A-A):
  • remove systemic obstruction
  • provide pulmonary blood flow just sufficient to oxygenate blood
  • usually see conduit placed between subclavian artery to pulmonary artery
  → highest risk step
• step 2 = Sup cavo-pulmonary connection (eg bidirectional Glen shunt) (V-A):
  • done after PAs have grown ⇒ ↓ed PVR
  • stage 1 ligated
  • bidirectional shunt
  • SVC sutured onto pulmon artery
  • ventricle receives:
    - deoxyHb from IVC (via atrial septal defect)
    - oxyHb from pulmon veins
• step 3 = completion:
  - performed at 1-5yrs when PA's even larger ⇒ ↓↓PVR
  - IVC directed into pulmonary artery - usually by extracardiac conduit

**Complications of Fontan Circulation**
• ↓ventricular function ⇒↓exercise tolerance:
  • fixed HR
  • unable to vary SV - impaired VF & fixed preload
• arrhythmias - mostly atrial
• shunts:
  • volume overload
  • chronic desaturation
• protein losing enteropathy:
  • protein loss into gut
  • causes:
    - ↓ed thoracic duct drainage due to ↑SVC pressures
    - mesenteric vasc inflamation
• developmental deficit:
  • multiple bypass, thrombotic events, chronic hypoxaemia
• thromboembolism:
  • atrial scaring
  • all pts need anticoagulation - warf or anti-platelet

**Anaesthetic Management**
• principles maintain:
  • SVR
  • PVR
  • valve function
  • rhythm
  • contractility
• preload = atrial pressure - CVP
Preoperative
• high risk paradoxical air embolism
• standard detailed pre op Ax

Perioperative
Induction
• mandatory A line & CVP trend
  ➔ demonstrates pulmonary pressures
• avoid -ve inotropes eg thiopentone
• ketamine ideal - as no change SVR

Maintenance
• low conc of volatiles + remifent good combination
• target SpO2 95% only
• good PVR care
• guided fluid administration eg with CVP
• spont venting ideal - if can avoid hypercarbia
  ➔ IPPV ➔ ↑ITP ➔ ↓VR ➔ ↓CO
  ➦ use low RR, rapid insp time, low tidal volumes

Postop
• ICU for careful fluid monitoring

Special Points
• Pregnancy:
  • ↑fluid ➔ distension of atria ➔ arrhythmias
  • should be fully anticoagulated to prevent VTE
    ➦ convert to heparin
  • elective procedure
  • early epidural with careful topups to
    - prevent loss of preload due to vasoD
    - prevent 2nd stage pain
  • avoid excessive pushing eg forceps assisted delivery
• laproscopic surgery:
  • complications:
    - ↑CO2
    - gas embolism
  • keep pressures <10mmHg to avoid IVC compression & get max VR benefits

Children With Congen Heart Disease for Non Cardiac Surgery

Overview of Circulations
Normal or Series
• separate systemic & pulmon circulations
• = most common type of CHD
• may be pure series or mixed with ASDs & VSDs mixing blood

Parallel or Balance Circulation
• circulations communicate with each other & function as being in parallel
• flow into each circuit depends on relative resistance:
  • ↑PBF ➔ pulmon oedema, ↓systemic perfusion
  • ↓PBF ➔ profound cyanosis
• eg large A-V septal defects or VSDs with L to R shunt:
  - ↑O2 ⇒ ↑PBF ⇒ ↓systemic perfusion & pHTN
  - large induction doses ⇒ ↓SVR ⇒ R to L shunt ⇒ cyanosis

**Single Ventricle Circulation**
- palliative procedure ⇒ blood flow passing down pressure gradient from VR ⇒ PA ⇒ lungs ⇒ LA
- see Fontan circulation
- Variables which compromise PBF = CVP, PVR, ITP

**Risk Stratification**

- need to assess:
  - physiological status
  - High risk complex heart disease =
    - single ventricle
    - balance circulation
    - cardiomyopathy
  - AS
  - High risk surgery = intraperitoneal, intrathoracic, vasc reconstructive surgery
  - age
- where is best place to perform surgery:
  - High risk = transfer to specialist centre
  - Low risk = perform locally
  - Intermediate = discuss with specialist centre

**Physiological Status**
- see history section for repair CHD

**Cardiac Failure**
- volume overload eg residual shunts, incompetent valves eg post Fallot repair
- pressure overload - residual outflow obstruction
- severe failure very bad sign

**pHTN**
- Rx:
  - 100% O2
  - inhaled nitric oxide
  - IV prostacyclin
- inotropic support of RV
- support resp tract infections

**Arrhythmias**
- all children with CHD repair must have preop ECG.
- beware of vent ectopics

**Cyanosis**
- common feature of unrepaired or partial repair CHD
- high risk group

**Management**
- balance emergency vs elective & need to transfer child
- anaesthesia plan based on cardiac physiology:
  - septal defects & shunt
  - pHTN
  - type of circulation
  - concurrent medical problems