ADULT CARDIAC SURGERY

• CABG represents ~ 80% of all adult cardiac surgery (USA)

Patient Factors

1. **anxiety**

2. **haemodynamic status**
   i. functional **history & examination** of the patient
      → **poor** LV function suggested by,
      • dyspnoea - rest, exertional, nocturnal, with angina (NYHA grades)
      • oedema - SOA, pulmonary oedema/infections, hepatomegaly
      • hypoperfusion - CNS confusion
         - oliguria, elevated creatinine & urea
         - fatigue & muscle weakness, anorexia
         - angina, with rest / exertion
         - peripheral cyanosis
      • hypotension + signs of sympathetic stimulation (↑HR, diaphoresis)
      • cardiomegaly ± S₃ gallop rhythm
   ii. preoperative cardiology **investigations**
      • blood picture - Hb and platelets, WCC
      • coag* screen - preheparinisation on CPB
      • biochemistry - electrolytes, Cr & urea, LFT's, BSL
      • AGA's - baseline respiratory function
      • resting ± exercise ECG
      • CXR - cardiomegaly, pulmonary oedema
      • echocardiography
        • angiography - hypokinesia, akinesia, dyskinesia, aneurysm
          - LVEF < 50%
          - LVEDP > 15 mmHg
          - CI < 2.5 l/min/m²

3. **non-cardiac diseases**
   - other vascular disease (esp. cerebral)
   - respiratory function
   - diabetes

4. **chronic drug therapy**
   i. **β**-blockers
      - associated with a lower incidence of adverse outcome
      - discontinuation causes **rebound** hypertension
      - interaction with volatiles & inotropes
   ii. **Ca**²⁺-blockers
      - **no** lower incidence of adverse outcome
      - discontinuation may increase anginal symptoms
      - interact with pressors, inotropes & AV conduction
   iii. antihypertensives
      - rebound hypertension (especially clonidine)
      - **all** should be continued until the morning of surgery
5. previous surgical & diagnostic procedures
   - neck surgery may make central venous cannulation difficult
   - vein stripping surgery may necessitate use of the arm veins for grafting
   - previous **CABG** will make sternotomy more difficult & risky, therefore prolong the pre-bypass period

### Myocardial Oxygen Balance

<table>
<thead>
<tr>
<th>Determinants of Myocardial Oxygen Supply &amp; Demand</th>
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<tbody>
<tr>
<td><strong>Decreased O₂ Supply</strong></td>
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<tr>
<td><strong>Coronary Blood Flow</strong></td>
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<tr>
<td>1. tachycardia ↓ diastolic perfusion</td>
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<tr>
<td>2. hypotension especially diastolic</td>
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<tr>
<td>3. coronary vascular resistance</td>
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<tr>
<td>- increased preload ↑ LVEDP</td>
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<tr>
<td>- ↑ wall thickness¹</td>
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<tr>
<td>- ↓ capillary density</td>
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<tr>
<td>- coronary artery spasm</td>
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<tr>
<td>- hypocapnia</td>
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<tr>
<td>- increased viscosity of blood</td>
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<tr>
<td><strong>Decreased O₂ content</strong></td>
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<tr>
<td>- anaemia</td>
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<tr>
<td>- hypoxaemia</td>
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<tr>
<td><strong>Decreased tissue O₂ uptake</strong></td>
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<tr>
<td>- left shift HbO₂ curve</td>
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<tr>
<td>- metabolic poisons, CN⁻</td>
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<tr>
<td>- sepsis syndrome</td>
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<tr>
<td>- myocardial depressant factor(s)</td>
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</table>

¹ myocardial **perfusion pressure** gradient from epicardium to endocardium → ~ 40-50 mmHg

² **extravascular pressure** gradient from endocardial to epicardial surface → ~ zero subepicardially
Surgical Factors - CABG

1. with a fixed obstruction, little can be done to increase perfusion
   • pressure would have to be increased significantly to compensate for the reduction in lumen diameter & flow
2. HR is crucial, tachycardia → increased \( O_2 \) demand
decreased diastolic perfusion, esp. subendocardial
3. LVEDV is a reflection of preload & major determinant of \( VO_2 \)
4. LVEDP is a reflection of LVEDV, hence preload, but is also a determinant of coronary perfusion pressure, especially in the subendocardium, where,

   \[
   \text{perfusion pressure} \sim \text{mean aortic diastolic pressure - LVEDP}
   \]
5. the incidence of a perioperative myocardial event is greater in those demonstrating intraoperative evidence of myocardial ischaemia (ECG or TEE changes)
   • these have variably been associated with tachycardia, hypertension & hypotension
   • ischaemic episodes occur in the absence of any haemodynamic change
   • adverse cardiac outcomes occur in the absence of ischaemia episodes
   • however, there is a strong correlation between,
     i. both preoperative and intraoperative ischaemia, and
     ii. adverse postoperative cardiac outcome
   • it is therefore recommended to maintain cardiovascular parameters within "normal" ranges for the patient concerned
6. there is no substantial evidence that the choice of anaesthetic technique influences the incidence of perioperative cardiac outcome, this may be due to,
   i. few real outcome studies have been performed & large numbers would be needed
   ii. the measurement of the presence or absence of haemodynamic variation does not allow prediction of adverse cardiac outcome
   iii. few patients are anaesthetised with a sole anaesthetic agent
   iv. monitoring and treatment of potentially adverse haemodynamic changes might improve outcome, and the choice of anaesthetic technique does not do away with the need for such monitoring and treatment
   v. theoretical concerns regarding coronary steal and isoflurane have not been supported clinically
   vi. with respect to non-cardiac surgery, the majority of cardiac events occur in the postoperative, rather than the intraoperative period
   vii. the coincidence of events in time does not establish a cause-effect relationship
The Nature Coronary Artery Disease

1. diffuse versus local obstruction → likelihood of surgical correction
2. distribution supplied distal to the obstruction
   → amount of myocardium "at risk"
3. characteristics of angina
   i. "silent" ischaemia
   ii. "stable" angina
   iii. "unstable" angina
   iv. Prinzmetal's angina - spasm typically at rest or with minimal exertion
      - absence of a fixed obstructive lesion
      - disappearing with exercise (vasodilatation)
4. effects of CAD on pump function
   → "good" or "poor" LV function

<table>
<thead>
<tr>
<th>Classification of LV Function</th>
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<tr>
<td>Good LV Function</td>
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<tr>
<td>Absence of CCF</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Normal CI &gt; 2.5 l/min/m²</td>
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<tr>
<td>Normal LVEDP &lt; 12 mmHg</td>
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<td>Normal ventriculogram</td>
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¹ probably the best single indicator of LV function
Surgical Factors - Valvular Disease

■ **Determinants of Cardiac Output**

1. **rate**
   - increases in HR can compensate for decreases in SV until reduction in diastolic time reduces filling (preload) and/or coronary perfusion (contractility)

2. **rhythm**
   - coordinated ventricular contraction essential for adequate pump function
   - atrial contribution to preload more essential in low compliance states

3. **preload**
   - end-diastolic fibre length determines contractile force as per Frank-Starling
     → **heterometric autoregulation**
   - over-distension increases O₂ demand and impairs contractile force

4. **afterload**
   - represents the impedance to ejection of blood from the LV
   - determines the amount of **work** done during ejection
   - determined predominantly by SVR in the absence of aortic valve disease
   - also by the elastance of the aortic tree & momentum of ejected blood

5. **contractility**
   - the intrinsic property of myofibrils which determines the contractile force developed for a given end-diastolic fibre length → "cardiac function curves"
   - maximal contractility line passes through the end-systolic pressure point
   - dependent upon,
     i. adequate coronary blood flow
     ii. oxygen & nutrient supply
     iii. absence of toxins
     iv. neural and hormonal influences
     v. homeometric autoregulation
       • $\uparrow$ contractility $\propto$ $\uparrow$ pressure load
         **without** changes in end-diastolic fibre length
     vi. interval between beats
       • the increase in contractility seen with moderate increases in heart rate
Compensatory Changes with Disease

NB: acute disease → reflex changes maintaining cardiac performance
    chronic disease → reflex, plus permanent structural alterations

reflex changes are more easily measured, more susceptible to therapeutic intervention, and more readily reversed with correction of the primary pathology

- differing pathologies produce symptoms & signs, the intensity of which may not correlate with the severity of the underlying pathology
- therefore it is necessary to evaluate valvular function and myocardial performance objectively, usually by cardiac catheterisation
- "abnormal values" due to compensatory processes may need to be maintained during anaesthesia and immediately postoperatively for adequate cardiac function
- under such conditions trends are generally more important than absolute values
- similarly, compensatory processes may be detrimental once a faulty valve is replaced by a more normally functioning valve, to the extend that therapy against "compensation" may be required
- "normal" indices of ventricular function may be inappropriate in the presence of valvular malfunction, eg. LVEDP is not a reliable index of preload in the presence of AS, AI or MI
- ejection fraction is probably the best index of ventricular function, except in MR where the impedance to regurgitant flow will also affect ejection
- intraoperatively the determination of ventricular function curves is probably the most efficient means of determining ventricular function

Mitral Stenosis

- usually rheumatic in origin and follows a prolonged course
- atrial contraction may contribute up to 40% of LV filling (cf. 15% normally)
- estimation of the severity of stenosis by the valvular pressure gradient is unreliable
- estimates of mitral valve area provide the most reliable index of severity, ↤ echocardiography

<table>
<thead>
<tr>
<th>Cardiac Catheterisation</th>
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<tr>
<td>Severity</td>
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<tr>
<td>-----------</td>
</tr>
<tr>
<td>normal</td>
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<tr>
<td>mild</td>
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<tr>
<td>moderate</td>
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<tr>
<td>severe</td>
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Additional Information
- PVR and pulmonary hypertension
- LV function
- coronary artery anatomy
- other valvular lesions
Anaesthetic Considerations

NB: full, slow to normal rate and tight

1. SBE prophylaxis
2. heart rate is the primary consideration
   • bradycardia markedly reduces CO as the SV is limited by the stenotic valve and the small size of the LV
   • tachycardia is more detrimental, as it decreases LV filling time, hence preload & cardiac output
   • acute pulmonary oedema may occur if AF with a rapid ventricular response occurs
   • this requires aggressive $R_x \rightarrow$ DCCV, digoxin, verapamil, propranolol
   • digoxin should be continued throughout the operative period in the presence of atrial arrhythmias (? amiodarone)

3. near maximal preload should be maintained
   • within constraints of pulmonary congestion
   • precise monitoring of LAP or PAoP is desirable, however due to the elevated PVR and pulmonary hypertension,
     i. increased risk of PA rupture during balloon inflation
     ii. a PAoP tracing may not obtainable
     iii. the PA diastolic pressure is not a reliable index of either LAP or LVEDP
       • although trends may show similar degrees of change
     iv. floating the catheter into the PA may be difficult
       • therefore, insertion of a LA catheter at the time of surgery may be preferable
       • $\delta P \sim 4-7$ mmHg across the prosthetic valve is normal

4. progression of pulmonary hypertension results in,
   i. increased PVR may limit LA & LV filling
   ii. the RV may fail if its workload is too great
   iii. ventricular interdependence may also limit LV filling with RV failure
     • therefore, factors tending to increase PVR should be avoided,
       ie. hypercarbia, hypoxia and the use of $N_2O$

5. pulmonary hypertension, RVF and tricuspid regurgitation usually improve over the days to weeks following correction of mitral stenosis, however, the structural changes due to longstanding disease limit the extent of long-term improvement
Mitral Regurgitation

**Pathophysiology Acute**

- a. abrupt onset of severe MR into relatively non-compliant LA, ie. high pressure
- b. marked increase in PCWP with large 'v' wave
- c. PVH & PAH → early RVF
- d. compensatory ↑'s in SNS tone → ↑ regurgitant fraction & worsen failure
- e. normal LV function unless infarction & rupture is origin of MR
- f. early onset of clinical heart failure

*NB:* small, high pressure LA & pulmonary circulation reflex compensation is *detrimental* → small heart & florid pulmonary oedema

**Pathophysiology Chronic**

- a. gradual increase in the regurgitant fraction
- b. gradual increase in LA size & compliance
- c. late onset of significant increase in LVEDP & PCWP
- d. irreversible LV dysfunction occurs *before* deterioration of ejection phase and clinical heart failure

*NB:* large, normal pressure LA & pulmonary circulation

**Assessment of Severity**

- a. clinical - heart size, LV heave, diffuse AB
  - S₁ gallop rhythm
  - ↑ JVP, pulmonary oedema, hepatic congestion
  - duration of the murmur, *not* loudness
  - PAH
- b. CXR - enlarged LA, LV
- c. ECG - AF, LVH
- d. Catheter > 0.6 *regurgitant* fraction → CCF

*NB:* BP, pulse, loudness of murmur of *no* significance differentiation of MI vs MS → pulse volume and heart size
Anaesthetic Considerations

NB: full, fast and tight

1. SBE prophylaxis

2. **heart rate** should be maintained at normal to tachycardic levels
   - *bradycardia* → ↑LV volume, ↑regurgitant fraction
     ↓cardiac output

3. factors **decreasing** the regurgitant fraction,
   - decreasing afterload
   - vasodilators
   - regional anaesthesia

4. factors **increasing** the regurgitant fraction,
   - increased afterload
   - increased SNS tone (pain, hypoxia, hypercarbia, acidosis)
   - slow HR
   - N₂O

5. myocardial **contractility** is decreased
   - the myocardium is more sensitive to depressant drugs
   - increasing **preload** → LV dilatation & increased regurgitant flow

6. following valve replacement there is the risk of **ventricular rupture**
   - especially in elderly patients
   - usually transverse & ? due to loss of ventricular support by the valve mechanism
   - measures to reduce the risk include,
     i. continued CPB
     ii. IABP to decrease afterload
     iii. vasodilators & antihypertensive agents
Mitral Valve Prolapse

**Incidence**

i. ~ 17% in 20-30 y.o. females, decreasing with age  
ii. ~ 2-4% in males, little change with age  
iii. ~ 4-5% overall

**Aetiology**

i. ? dominant inheritance in some families - reduced expression  
ii. connective tissue abnormality  
iii. congenital/embryological  
iv. neuroendocrine disease

**Congenital Associations**

i. ostium secundum defects  
ii. IHSS  
iii. Ebstein's anomaly - TI with abnormality of the mitral valve  
iv. long QT syndrome  
v. WPW syndrome *pre-excitation may be present in ~ 50%  
vi. polycystic kidney disease  
vii. von Willebrand's disease  
viii. Marfan's  
ix. Ehler's-Danlos  
x. Turner's syndrome

**Complications**

- usually very low, however, occur more commonly in the presence of,  
  1. symptoms  
  2. dilation of the LV  
     > 5.9 cm male  
     > 5.5 cm females  
  3. an abnormal resting ECG  
  4. increasing age  
     ≥ 40 yrs  
  5. female > male  
  6. syncope  
  7. murmur = MI not MVP  
  8. redundant valve leaflets
Complications

a. **arrhythmias**
   - AE's/VE's ~ 55%
   - bradyarrhythmias ~ 25%
   - VT or SVT ~ 6%

b. sudden death ~ 1.4%

c. thromboembolism

d. mitral regurgitation - may be decreased by increased preload & LV size

e. bacterial endocarditis

f. aortic dissection

g. chordae rupture - acute MI and LVF

**NB:** * these are increased by,

i. increases in SNS tone

ii. administration of catecholamines

iii. prolonged QTc

iv. type I antiarrhythmics

Clinical Presentation

a. chest pain - atypical

b. palpitations / arrhythmias

c. dizziness, syncope

d. rarely progress to MR

e. systemic thromboembolism

f. sudden death

Clinical Findings

a. mid-systolic click

b. mid/late systolic murmur → apex & LSE
   • increased by reducing afterload (valsalva, vasodilators)

c. ECG: ST/T wave changes inferiorly
   arrhythmias

d. Echo: very sensitive → "gold standard"
Aortic Regurgitation

### Aetiology

1. **Acute**
   - SBE
   - aortic dissection
   - traumatic
   - acute LV *volume overload* with elevation of LVEDP & PAoP
   - premature mitral valve closure may produce LVEDP > LAP & PAoP
   - reflex \( \uparrow \) HR & SVR further reduce "forward" flow and LVF progresses rapidly

2. **Chronic**
   - rheumatic
   - syphilis
   - Marfan's
   - SBE
   - RA, psoriasis, Reiter's, UC, Crohn's, ankylosing spondylitis
   - myxomatous degeneration
   - gradually progressing eccentric dilatation & hypertrophy \( \uparrow \) compliance
   - competency of the mitral valve protects the pulmonary circulation until late in the disease, when CO falls and results in \( \uparrow \) SNS tone
   - disease progression is often asymptomatic over 10-20 years, with rapid deterioration once LV failure occurs

### Severity of Incompetence

a. pulse character & BP  - systolic > 140 & diastolic < 60
b. cardiomegaly & LV heave
c. Austin-Flint murmur  * loudness of the murmur is *not* a useful guide
d. ECG  - LVH & strain
e. aortic root angiography *grading*
   i. small amount of contrast enters LV during diastole, clearing in systole
   ii. LV faintly opacified during diastole, but not cleared in systole
   iii. LV progressively opacified
   iv. LV completely opacified during first diastole & remains for several beats
f. assessment of *regurgitant volume*
   i. mild  ~ 1-3 l/min
   ii. moderate  ~ 3-5 l/min
   iii. severe  > 6 l/min
   * volumes up to 25 l/min have been recorded

g. indicators of *severe* chronic AI are the abrupt onset of,
   i. cardiomegaly and CCF
   ii. associated mitral incompetence
Anaesthetic Management

NB: full, dilated and fast

1. SBE prophylaxis
2. Heart rate slightly higher than normal > 80 bpm
   • reduces LV size as less time is available for diastolic regurgitation
   • reduction in size & wall tension offsets ↑ HR, and VO₂ decreases
   • subendocardial flow increases due to higher aortic diastolic pressure and reduced LVEDP
   • therefore, bradycardia should be avoided
3. BP is often labile & very responsive to vasoactive drugs
   • with appropriate monitoring, vasodilators may be used to,
     i. decrease SVR & increase "forward" pump flow
     ii. decrease LV distension, 2° mitral regurgitation & pulmonary pressures
   • diastolic hypotension & reduced coronary blood flow must be avoided
   • avoid excess vasoconstriction due to reverse effects
4. myocardial contractility is usually impaired in both acute & chronic AI
   • VO₂ is increased only moderately as volume loads increase LV work ~ 10-15%
   • LV wall tension is only marginally increased until the later stages of the disease

Aortic Stenosis

Aetiology

a. rheumatic
b. congenital bicuspid valve
c. calcific or degenerative

Pathophysiology

a. chronic pressure overload - concentric LVH & increased LV mass
   - LV failure / decompensation
b. ↓ LVEF and CO
c. fixed low output state
d. LV / aortic root pressure gradient
e. ↑ LVEDP, eventually ↑ LAP
f. ↑ PCWP
g. eventually pulmonary hypertension
Anaesthetic Considerations

**NB:** full, normal rate & tight

1. SBE prophylaxis
2. higher **filling pressures** are required for the non-compliant ventricle
   - these are transmitted into the pulmonary circulation with the risk of pulmonary oedema, therefore monitoring of PAoP may be necessary
   - in the non-compliant ventricle, mean PAoP *underestimates* LVEDP, which more closely approximates the \( a \)-wave of the tracing
3. avoid factors likely to induce **atrial fibrillation**
   - atrial contribution to LV filling may be \( \sim 40\% \) cf. 15% normally
   - acute onset AF may be associated with LV failure & requires prompt treatment
4. **heart rate** \( \sim 70-90 \) bpm is optimal, maintaining **sinus rhythm**
   - avoid tachycardia/bradycardia as these result in decrease coronary perfusion
5. minimise myocardial ischaemia, ie. maintain **coronary perfusion pressure**
   - \( O_2 \) demand is greatly increased \( 2^\circ \) to increased muscle mass & pressure work
   - supply is reduce due to
     - shorter diastolic interval (longer ejection phase)
     - ↓ aortic mean diastolic pressure
     - ↑ LVEDP & ↓ subendocardial perfusion
     - muscle capillary density is decreased
     - coexistent **atherosclerotic disease** \( \sim 50\% \)
   - avoid decreases in **SVR** as these decrease mean aortic diastolic pressure
   - increased sensitivity to myocardial depressant drugs, especially if these also result in peripheral vasodilatation, ie. STP
6. argument regarding advisability of PA catheter insertion
   i. reduced validity of measurements obtained
   ii. risk of inducing AF
   iii. risk of VF, resuscitation from which is almost impossible in the setting of AS
Hypertrophic Cardiomyopathy

- **Features**
  a. hypertrophic cardiomyopathy
  b. marked asymmetrical septal hypertrophy
  c. *autosomal dominant* inheritance ~ 50% are familial, variable expression

- **Pathophysiology**
  a. hypercontractile LV
  b. anatomical septal hypertrophy
  c. dynamic subaortic muscular *stenosis*
  d. markedly reduced LV compliance
  e. increased LAP, frequently with LA dilatation & hypertrophy
  f. systolic anterior motion of anterior MV leaflet $\rightarrow$ **MR** ~ 50%
  g. pre & post-surgical involvement of the conducting system with *arrhythmias*

- **Exacerbating Factors**
  a. $\uparrow$ *contractility*
     - sympathomimetics
     - digoxin & (+)ve inotropes
     - tachycardia
  b. $\downarrow$ *preload*
     $\rightarrow$ reduction in ventricular size
     - hypovolaemia
     - venodilators (GTN)
     - increased PVR, high airway pressures
  c. $\downarrow$ *afterload*
     - vasodilators
     - regional sympathectomy

- **Factors Decreasing Dynamic Obstruction**
  a. $\downarrow$ *contractility*
     - $\beta$ adrenergic blockers
     - Ca$^{++}$ entry blockers
     - volatile anaesthetics
  b. $\uparrow$ *preload*
     - hypervolaemia
     - bradycardia
  c. $\uparrow$ *afterload*
     - vasoconstrictors
     - metaraminol, phenylephrine
**Anaesthetic Considerations**

NB: *full, slow and tight*

1. SBE prophylaxis
2. maintain *filling pressures*
   - the hypertrophied LV is poorly compliant
   - decreased preload decreases LVESV and increases dynamic obstruction
3. maintain a slow *heart rate*
   - tachycardia increases the velocity of contraction, decreases LVESV, decreases diastolic perfusion time & coronary perfusion pressure
   - avoid factors likely to precipitate *atrial fibrillation*, detrimental due to loss of atrial contribution to LV filling and potentially rapid ventricular response
4. maintain *afterload*
   - reductions increasing the LV-aortic pressure gradient & obstruction
   - reductions in mean aortic diastolic pressure required for coronary perfusion
5. avoid increases in *contractility*

NB: the management of MR in the presence of IHSS varies, in that pharmacological interventions affect MR+IHSS in the *opposite* manner to the isolated MR case

**Other Pressure Overload Diseases**

1. chronic systemic arterial hypertension
2. coarctation of the aorta
3. aortic cross-clamping during surgery

**Tricuspid Regurgitation**

- rare as an isolated condition, though, increasing in frequency 2° to IV *drug abuse*
- may be seen 2° to ventricular pacing
- usually 2° to RV failure 2° to aortic or mitral valve disease and is largely corrected by management of the 1° disorder
- produces volume overload of the RV which is usually well tolerated
- management is directed at maintaining a normal-high CVP and reducing PVR

NB: any factor decreasing *RV output*, effectively decreases *LV preload*
Cardiomyopathy & Transplantation

**Def'n:** any and all structural and functional abnormalities of the myocardium

**Aetiology**

1. **dilated congestive cardiomyopathy** *most common form*
   - i. ischaemic
   - ii. idiopathic
   - iii. familial
   - iv. infective - Coxsachie B & A, echoviruses
     - bacterial, fungal, protozoal
   - v. metabolic - hyperthyroidism, ? Addisons
   - vi. peripartum
   - vii. glycogen storage disease - type II
   - viii. nutritional deficiency - thiamine, selenium, ?H$_2$PO$_4$
   - ix. autoimmune - RA, PAN, SLE, Kawasaki disease
   - x. drugs - alcohol
   - adriamycin, daunorubicin, doxorubicin
   - sulphonamides, lithium, phenothiazines
   - sympathomimetics
   - xi. radiation

2. **restrictive cardiomyopathy**
   - i. idiopathic
   - ii. endomyocardial fibrosis
   - iii. eosinophilic endomyocardial disease
   - iv. endocardial fibroelastosis - commonly associated with congenital heart d.
   - v. infiltrations - amyloid, sarcoid
   - - neoplasms
   - vi. glycogen storage disease

3. **hypertrophic cardiomyopathy**
   - i. idiopathic - HOCM, IHSS
   - ii. familial - autosomal dominant
   - iii. Friedrich's ataxia ~ 50%

**NB:**
1. **donor** considerations are supportive prior to organ harvesting
2. **recipient** considerations involve maintaining adequate organ perfusion and function prior to CPB being established
3. postoperatively the heart is *denervated* and the RV frequently has to pump against an *elevated PVR*, which developed 2° to LV failure
Other Disorders - Adult

1. cardiac tamponade or constrictive pericarditis
   - reduced diastolic filling/compliance with limited SV and CO
   - diastolic equalisation of pressures across the heart
   - avoid hypotension, hypoxia, decreased venous return, bradycardia and drugs which decrease contractility
   - avoid excessive IPPV

2. pacemaker insertion
   i. CHB, acquired or congenital
   ii. sick sinus syndrome
   iii. bradycardia with symptoms

3. congenital heart disease
   i. ASD - usually asymptomatic & found incidentally
      - systemic embolisation
      - risk of endocarditis
      - progressive L→R shunt
      - RVH & CCF occur late
   ii. VSD - usually acute MI and ventricular rupture in the adult
      - associated cardiogenic shock
      - 50% operative mortality
      - interim Rx = inotropes + afterload reduction + IABP
      * CO by thermodilution inaccurate

4. arrhythmia surgery
   - epicardial or endocardial electrophysiological mapping with programmed stimulation to precipitate the arrhythmia
   - either excision or cryoablation of the aberrant conduction pathway
   - anaesthetic aims include,
     i. prevent precipitation of the arrhythmia prior to mapping
     ii. avoid drugs which may interfere with mapping
        - anticholinergics - atropine, pancuronium
        - sympathomimetics - ketamine
        - droperidol, which has been shown to prolong the ERP of the accessory pathway in WPW syndrome
     iii. deal with adverse haemodynamic effects of the arrhythmia, plus chronic anti-arrhythmic therapy
        - various anaesthetic techniques have been used and none is superior
        - avoidance of drugs affecting conduction does not preclude the use of local anaesthetic prior to vessel cannulation
        - if a PA catheter is to be inserted prior to CPB, some would not advance beyond CVP until the heart was exposed in case an arrhythmia is generated
**Essential Monitoring**

1. **arterial blood pressure**
   - mean BP during non-pulsatile flow on CPB
   - immediate analysis of the significance of
     - surgical manipulations
     - arrhythmias
     - electrical pacing
     - artificial ventilation
   - waveform analysis for CO, SVR, preload
   - repeated AGA's, coagulation studies & biochemistry

2. **electrocardiogram**
   - detection of arrhythmias and/or ischaemia
   - preferably 2 ECG leads simultaneously for CAD
     - V5 anterolateral
     - II or aVF inferior
   - automated ST segment analysis if available

3. **ventricular filling pressures**
   - either CVP alone, or in combination with either LAP or PAoP
   - estimation of preload, plus administration of irritant drugs
   - LAP or PAoP with significant functional discrepancy between the LV & RV
   - TEE may provide better assessment of filling, especially post-bypass

4. **cardiac output**
   - cf. BP, CO is the only reliable means of rapidly assessing ventricular function
   - postoperatively and monitoring the effects of therapeutic measures
   - inadequate CO will eventually manifest as end-organ dysfunction, however this is
     significantly delayed

5. **urine output**
   - volume & quality are best indicators of renal perfusion
   - however, also altered by
     - vasoactive drugs
     - variations in CO
     - extracorporeal circulation, haemolysis on CPB
     - use of diuretics
   - high U/O during CPB
     - ? ANP release / ADH inhibition

6. **body temperature**
   - indicated with or without deliberate hypothermia due to the likelihood of significant
     temperature loss during surgery
   - gradients between central and peripheral sites indicate the adequacy of rewarming
     post-CPB, and the likelihood of recooling

7. **oesophageal stethoscope**
   - should be inserted prior to the administration of heparin
   - modifications allow
     - measurement of core temperature
     - oesophageal ECG electrode
     - transoesophageal pacing
8. **anticoagulant activity**
   - essential during CPB to minimise the risks of thrombosis and embolism
   - the one absolutely fatal complication is clotting of the bypass circuit
   - **ACT** better than heparin assay, as it measures *activity* not quantity

9. **arterial blood gases**
   - optimisation of ventilation
   - adequacy of peripheral (end-organ) perfusion

10. **serum biochemistry**
    - serum K⁺ requires monitoring due to high content in cardioplegic solutions
    - high urine output during bypass may result in K⁺ depletion and arrhythmias

**Specialised Monitoring**

- **Transoesophageal Echocardiography** (TEE)
  - qualitative & semiquantitative data regarding,
    1. chamber size & filling  *extrapolation from area to volume*
    2. regional wall motion abnormalities & contractility
    3. valvular function & competence
    4. air embolisation
    5. intracardiac shunting
    6. aortic dissection
    7. pericardial effusion
  - as LV compliance & contractility change markedly in the perioperative period, marked changes in LVEDV may be seen without corresponding changes in LVEDP (CVP or PAoP)
  - don't get a good view of the coronary arteries
  - Melbourne course stated extrapolation from area to volume had limitations and there were problems in compring PCWP & LVEDA

- **Electroencephalography**
  - aim is to detect aetiology, duration and significance of cerebral insults
  - as yet no definitive place in monitoring
  - only 1 study, that by Nussmeier *et al.*, has demonstrated cerebral protection using high dose barbiturates
Premedication

*NB:* allay anxiety, produce amnesia, and minimise pain and sympathetic stimulation associated with vascular cannulation in the preanaesthetic period, *without* producing detrimental degrees of cardiac or respiratory depression

- to a large extent will depend upon the patient's baseline cardiac function and the presence of other system disease
- the patient with "good" LV function & no respiratory disease may be heavily premedicated with a combination of benzodiazepine and opioid, eg.,
  i. morphine 0.1 mg/kg & diazepam 0.1-0.2 mg/kg or lorazepam 2-4 mg &
  ii. omnopon 0.3 mg/kg & scopolamine

- clearly lesser doses are required for patients with limited cardiorespiratory reserve

**Regular Medications**

1. **nitrates** - may require supplemental doses in the perioperative period
2. **calcium channel blockers**
   i. if therapy has been successful in controlling angina, arrhythmias or hypertension, then these benefits should be continued into the operative period
   ii. knowledge of the side-effects and interactions with anaesthetic agents allows avoidance / treatment of complications during anaesthesia
   iii. patients who have received large doses of nifedipine for the treatment of angioplasty complications have required larger than usual doses of vasopressors, however no *qualitative* differences have been demonstrated
3. **β-blockers**
   - should not be discontinued, despite theoretical interactions with anaesthetic agents
   - patients with unstable angina but good LV function may benefit from acute administration of a β-blocker with their other premedication
4. **antihypertensives**

**Other Therapy**

1. supplemental O₂ - via nasal canulae during the pre-induction period
2. peptic ulcer / oesophageal reflux *RSI is a difficult proposal
3. IDDM
   - sympathetic-endocrine stress response to CPB and the administration of catecholamines produces insulin resistance and *hyperglycaemia*
   - insulin infusion rates far greater than usual may be required
   - aim for BSL ~ 5-15 mmol/l & check regularly
# Cardiovascular Anaesthesia

## Induction & Maintenance

| Airway Management | • potential for prolonged ventilation  
|                   | • high volume / low pressure cuff |
| Monitoring        | • IABP  
|                   | • CVP  
|                   | • PA catheter  
|                   |   - PAP, PAoP  
|                   |   - thermodilution CO & derived variables  
|                   |   - core temperature  
|                   |   - S\textsubscript{v}O\textsubscript{2}, oximetry, pacing  
|                   | • ECG  
|                   |   - multilead, II + V\textsubscript{5}  
|                   | • EEG  
|                   |   - if indicated  
|                   | • TEE  
|                   |   - if indicated / available |
| Anaesthetic agents | • primary anaesthetic agents  
|                   |   - opioid vs volatile  
|                   | • supplemental agents  
|                   |   - benzodiazepines  
|                   |   - volatile agents  
|                   |   - propofol  
|                   | • neuromuscular agents  
|                   | • nitrous oxide  
|                   |   * not post-CPB |
| Cardiovascular agents | • inotropes  
|                   | • vasopressors  
|                   | • vasodilators  
|                   | • antiarrhythmics  
|                   | • hyper/hypokalaemia  
|                   | • pulmonary hypertension |
| Anticoagulation & Blood Products | • heparin / protamine  
|                   | • platelets, FFP  
|                   | • DDAVP  
|                   | • whole blood or packed cells  
|                   | • NSA-5%  
|                   | • crystalloid |
| Other agents | • HCO\textsubscript{3}, Ca\textsuperscript{++}, Mg\textsuperscript{++}  
|                   | • mannitol, frusemide  
|                   | • antibiotics  
|                   | • steroids |
Volatile Anaesthetic Agents

- **Advantages**
  1. ability to produce all aspects of anaesthesia - amnesia
     - analgesia
     - muscle relaxation
  2. suppression of reflex responses to painful stimuli
  3. rapid recovery enabling early extubation
  4. dose-related decreases in ventricular work & VO\(_2\)

- **Disadvantages**
  1. excessive cardiovascular depression, especially in patients with *poor LV function*
  2. lack of *analgesia* at subanaesthetic concentrations in the recovery phase
  3. postoperative *shivering* and increased VO\(_2\)
  4. exaggerated *heat loss* due to peripheral vasodilatation

- **Isoflurane**
  - extensively studied due to theoretical *coronary steal*, ie. in the presence of a fixed obstruction, parallel and peripheral vessel dilatation may result in decreased perfusion pressure
  - Becker recommended avoiding its use in all patients at risk of CAD, ie. virtually anyone over the age of 50 years
  - this would seem unreasonable as,
    1. steal has *not* been demonstrated under normal clinical conditions
    2. isoflurane has actually been shown to protect against ischaemia induced by electrical pacing in patients with coronary stenosis
    3. isoflurane is seldom used as a sole agent in high concentrations in patients with cardiovascular disease
    4. there is no evidence showing a difference in *outcomes* for CABG surgery in patients anaesthetised primarily with halothane, enflurane, isoflurane or sufentanyl

*NB:* as with the other volatile agents, it is a useful supplement to opioid anaesthesia, allowing brief titration of the anaesthetic depth against noxious stimuli
Opioids

**Advantages**

1. absence of direct effects upon the heart  
   - no effect upon contractility, automaticity, conduction, sensitivity to catecholamines  
   - do result in an increase in *vagal tone*
2. no interference with autonomic or cardiovascular drug action  
3. preservation of blood flow autoregulation in cerebral, myocardial and renal beds  
4. increased patient tolerance of endotracheal intubation and airway manipulation  
5. postoperative analgesia  
6. no organ toxicity

**Disadvantages**

1. *bradycardia* and hypotension during induction  
2. limited ability to produce unconsciousness  
3. *muscular rigidity* during induction and occasionally during emergence  
4. prolonged *recovery time*, especially to spontaneous ventilation

- indirect actions which may have some effect upon the CVS include,
  1. bradycardia mediated through increased *vagal tone*
  2. arterial and venous dilatation due to selective suppression of sympathetic reflexes  
  3. morphine & pethidine release *histamine*

*NB:* these can be prevented or treated readily

- *awareness*, especially in response to noxious stimuli, may occur despite "normal" induction doses of opioids  
- a reliable guide of anaesthetic depth is required; processed EEG is being investigated but its reliability remains to be proven  
- the probability of awareness may be minimised by,
  1. administration of sufficient premed, or intraoperative *supplement*  
     - benzodiazepine, propofol infusion or volatile agent  
  2. titrating induction dose to effect, then maintaining plasma levels by *infusion*,  
     or less effectively by *intermittent bolus*  
  3. using muscle *relaxants* only when required & in minimal doses  
  4. maintaining vigilant observation of the patient
the administration of a purely amnesic drug is inadequate therapy, because,

1. the anaesthetist has a contractual agreement with the patient for general anaesthesia
2. although the patient may not feel pain due to the opioid effects, they do experience anxiety at the time of awareness, even if this is not recalled later
3. even when intraoperative events are not recalled, abnormalities in sleep behaviour and anxiety attacks may occur

Hypnotics & Tranquilisers

- these are frequently used in conjunction with the opioids due to the limited ability of the latter to produce unconsciousness
- the benzodiazepines are most commonly used because of their minimal effects on myocardial function
- their depressive effects on contractility are probably not dose-dependent
- interactions with opioids do however occur,
  1. although the dose of opioid may be reduced, there is a synergistic action, with increased times to,
     i. awakening - depression of consciousness
     ii. spontaneous ventilation - depression of ventilation
  2. administered together at induction the combination may result in hypotension
     - this usually responds readily to volume loading and vasopressors
     - there may be severe hypotension if an opioid is administered after unconsciousness has been obtained with a hypnotic agent

Nitrous Oxide

- used as a supplement to both IV and volatile agents
- effects upon the myocardium are usually mild $\rightarrow \uparrow \text{SVR} / \downarrow \text{CO}$
- however, these may be significant,
  1. in conjunction with opioids
  2. in the presence of poor LV function
  3. in the presence of a critical stenosis $\rightarrow$ regional ischaemia and dysfunction

- other effects of N₂O which require consideration during CPB include,
  1. expansion of air-spaces, especially emboli - bypass circuit - chambers - coronary grafts
  2. potentiation of truncal rigidity produced by opioids
  3. limitation of $\text{FiO}_2$
Muscle Relaxants

- usual *indications* include,
  1. facilitation of endotracheal intubation
  2. counteract rigidity produced by opioids
  3. maintenance of muscle / diaphragmatic paralysis under light levels of anaesthesia
  4. suppress body movement to defibrillation / cardioversion
  5. limit O₂ consumption
  6. prevent shivering associated with hypothermia

*NB:* prolonged paralysis is rarely a problem as these patients are routinely ventilated postoperatively

- the *disadvantages* of continuous paralysis are,
  1. interference with detection of light anaesthesia
  2. decreased muscle tone, increased venous pooling and risk of thrombo-embolic disease
  3. risk of postural injury
  4. inability to use ventilatory assist modes, with subsequently greater respiratory embarrassment
CARDIOPULMONARY BYPASS

*Def’n:* withdrawal of systemic blood as, or before it reaches the heart, the delivery of $O_2$ and removal of $CO_2$ by artificial means, an *oxygenator*, and return of "arterialised" blood distal to the aortic valve

Extracorporeal Systems

<table>
<thead>
<tr>
<th>Bubble vs. Membrane Oxygenators</th>
<th>X = advantageous unit</th>
<th>Bubble</th>
<th>Membrane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cost</td>
<td>X</td>
<td></td>
<td></td>
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<tr>
<td>Ease of set-up &amp; operation</td>
<td>X</td>
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<tr>
<td>Efficiency of oxygenation</td>
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<tr>
<td>Circuit air elimination</td>
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<tr>
<td>Lower priming volume</td>
<td>X</td>
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<tr>
<td>Oxygenator &quot;pulmonary oedema&quot;</td>
<td>X</td>
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<tr>
<td>Microembolus production</td>
<td></td>
<td>X</td>
<td></td>
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<tr>
<td>Blood trauma</td>
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<td>X</td>
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<tr>
<td>Arterial blood gas adjustment</td>
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</tbody>
</table>

- the trauma caused by bubbling and foaming in bubble oxygenators becomes a significant disadvantage with *prolonged bypass times* > 90-120 minutes
- survival is seldom achieved with use of a bubble oxygenator > 6 hours
- because of cell disruption and potential infusion of debris into the circulation, 27-40 µm *micropore filters* should be used on both venous return and arterial limbs
- these cause minimal impedance to blood flow and do not remove platelets
- a non-occlusive roller pump of the DeBakey type is most commonly used
- other types of pumps are being developed, principally to generate *pulsatile flow* and reduce red cell trauma
the most common circuit arrangement is withdrawal of blood from the RA or IVC/SVC and return to the ascending aorta, this may be,

a. **total-CPB**
   - drainage from IVC/SVC with inflow to the RA occluded,
     ie. there is no provision for flow to the RV and lungs in parallel
   - used for open heart procedures or when venous return to the heart causes problems

b. **partial-CPB**
   - either SVC/IVC drainage, or RA drainage without occlusion of inflow to the heart
   - suitable for most CABG or closed heart procedures
   - advantages of use include,
     i. ability to check the bypass circuit before establishment of CPB
     ii. ability to fill the heart to "normal" size, allowing estimation of **graft length**
     iii. allows some pulsatile flow - improved tissue perfusion
          - aids rewarming
     iv. assessment of heart function gradually, prior to "coming-off" bypass
        - preventing rapid distension of the LV acute failure
     v. allows coronary sinus flow to drain to the venae cavae, reducing flow to the pulmonary circulation and LV

c. **LV bypass**
   - venous return from the LA, ie. uses the patients lungs for gas exchange
   - arterial insertion usually into femoral vein
   - suitable for LV or thoracic aortic procedures
   - allows greater afterload regulation cf. AOX clamping in thoracic aneurysms

■ **LV Venting**

- usually by way of a catheter may be necessary to prevent excessive LV distension, due to,
  1. incompetent aortic valve (also prevented by ascending AOX clamping)
  2. coronary sinus and bronchial blood flowing into the pulmonary veins
  3. positioning of the heart such that blood flows into it, or backward flow into the venae cavae is prevented

  **NB:** also reduces the risks associated with elevated PCWP's, ie. **pulmonary oedema**
  monitoring PCWP allows early detection of elevated pressures

- the 3 most serious **complications** requiring an immediate cessation of bypass are,
  1. aortic dissection
  2. superperfusion of a carotid artery
  3. air in the aortic line
• the extracorporeal circuit is usually primed with a heparinised, buffered physiological salt solution, to which may be added,
  1. an osmotically active substance - albumin, hetastarch
  2. an osmotic diuretic - mannitol
  3. antibiotics
  4. electrolyte supplements - K⁺ if hyperkalaemic cardioplegia is not used
  5. packed red cells if the Hb is low
     • prior to haemodilution with the circuit volume
     • during hypothermic CPB aim for Hct ~ 20-25%

Anticoagulation

• heparin anticoagulation is the single most important pre-bypass step
• heparin is used as it has the desired efficacy, a rapid onset and is reversible with protamine
• clotting of the bypass circuit is the one absolutely fatal complication of CPB
  1. usual loading dose ~ 300-400 U/kg → ~ 25,000[
  2. administer into a central vein
     • after verifying intravascular placement, several minutes prior to establishing bypass
  3. monitor anticoagulation during bypass ? check prior to CPB
     i. whole blood heparin concentration
        • usually determined by protamine titration
     ii. activated clotting time, ACT - whole blood
        • preferred as it measures the activity, rather than the concentration of heparin
        • should aim for > 300-400 seconds during bypass
        • following bypass aim for the pre-heparin baseline ~ 90-120 seconds
        • the ACT is markedly prolonged during hypothermia, which may give the false impression of overdosage
        • the ACT will decrease ~ 10-30% on rewarming
        • the elimination of heparin is slowed by hypothermia but rapidly returns to normal on rewarming
  4. signs of inadequate anticoagulation include,
     i. accumulation of fibrin on the walls of the bypass reservoir
     ii. thickening of blood in the pericardial space
     iii. the presence of any clot requires the immediate administration of more heparin

NB: heparin has some deficiencies, the most important being its inability to protect platelets from activation and functional degradation during bypass
Platelet Dysfunction

- the most common cause of inadequate haemostasis following bypass
- this may be achieved with reversible platelet inhibition with use of,

1. prostaglandins - prostacyclin
   - PGE$_1$
   - synthetic prostanoid, iloprost
   - clinical trials of these agents have been disappointing, either because of unacceptable hypotension, or because of insignificant reductions in postoperative blood loss

2. antifibrinolytic agents - EACA
   - tranexamic acid
   - investigated in the presence of heparin anticoagulation
   - potentially inhibit plasmin-mediated digestion of platelet membrane receptors during CPB (GPIb)
   - recent studies have shown decreases in postoperative blood loss

3. aprotinin
   - is a less specific plasmin inhibitor and significantly reduced postoperative losses
   - also inhibits kallikrein, which may further diminish plasma coagulation and fibrinolysis during CPB
Haemodynamic Changes

- SVR initially falls with wash-in of the priming fluid, due to cold, low viscosity, low O₂ content, no humoral vasoconstrictors
- there is no consensus regarding optimal *perfusion pressures*,
  a. the recommended range for most patients ~ 40-70 mmHg
  b. early studies showing cerebral or renal injury 2° MAP < 50 mmHg preceded the era of *haemodilution* to Hct ~ 20-25%
  c. higher perfusion pressures would seem appropriate in the presence of,
     i. untreated chronic hypertension
     ii. symptomatic cerebrovascular disease
     iii. 2° LVH
  d. hypertension in the presence of anticoagulation carries the risk of ICH
- objectives of management should include,
  1. maintenance of tissue perfusion
     - initial pump flows should ~ normal resting CO for the patient
     - use vasopressors/vasodilators to maintain "acceptable" perfusion pressure
     - flow can be reduced proportionately to the degree of *hypothermia*
  2. vasopressors are not without complications,
     i. ↓ renal perfusion, end-organ hypoperfusion & ↑ metabolic acidosis
     ii. ↓ skin perfusion & increased temperature gradients
  3. signs of inadequate *tissue perfusion*
     i. oliguria
     ii. progressive metabolic acidosis
     iii. venous P O₂ < 40 mmHg or SvO₂ < 65%
     iv. wide temperature gradients
        - central - nasopharynx/PA/oesophagus
        - intermediate - rectal, skeletal muscle?
        - peripheral - skin
  4. factors affecting systemic vascular resistance
     i. low Hct. ~ 20-25% at 26-30°C
     ii. hypercarbia, hypoxia, acidosis
     iii. level of anaesthesia - abolition of sympathetic responses
     iv. drug effects - vasoactive agents
        - anaphylactoid / anaphylactic responses
  5. discrepancies between aortic root and radial artery pressure tracings
     - most likely due to marked vasodilatation
     - mean aortic pressure may be better estimated by direct measurement or by using an upper arm cuff and measuring "return to flow" pressure at the radial artery

Cardiovascular Anaesthesia
Myocardial Preservation

- **Ischaemia** results in,
  1. inhibition of metabolism of glucose, free fatty acids and lactate
  2. anaerobic glycolysis with lactate & $H^+$ production
  3. eventual inhibition of glycolysis by $H^+$
  4. structural damage to cellular organelles, especially **mitochondria**
     - as the energy for function and repair is derived from mitochondria, this is believed to be a determinant of **irreversibility**

- in addition, the timing and conditions during **reperfusion** will significantly affect myocardial performance,
  1. abnormal $Ca^{2+}$ metabolism
  2. reduced ATP stores and production
  3. $O_2$ free radical production
  4. damage to the ATP dependent regulation of cellular volume, with oedema and further disruption of cellular function

**NB:** these will affect contractility, conduction and the rate of myocardial repair

- methods of preservation include,
  1. diastolic myocardial arrest by hypothermic hyperkalaemia / hypermagnesaemia
     - **electromechanical arrest** is the basis of all **cardioplegic** techniques
  2. cooling
     - hypothermic CPB
     - epicardial surface cooling with iced solution
     - intracoronary infusion of cold cardioplegia solution
  3. prevention of LV distension and oedema by venting and the inclusion of mannitol in the cardioplegia

- improvements in preservation in the last 20 years has resulted in markedly better myocardial performance coming-off bypass and a reduced need for inotropic support
- potential problems associated with the use of cardioplegic solutions includes,
  1. increased atrioventricular conduction blockade due to **local hyperkalaemia**
     - this usually resolves over several hours and may be managed by temporary pacing
     - recovery may be hastened by insulin ± glucose and $Ca^{2+}$ salts
  2. **ventricular flaccidity** may contribute to transverse rupture following MV replacement

- current controversies in cardioplegia include,
  1. **solutions** - blood versus crystalloid
  2. **additives** - metabolic or non-metabolic
  3. **temperature** - warm versus cold
  4. **reperfusion** - what methods
• **Blood vs. Crystalloid**

  - even the hypothermic, arrested heart has an $O_2$ uptake > 0, therefore blood should be better than crystalloid in providing ,
    1. $O_2$ and metabolic substrate
    2. an appropriate osmotic load
    3. supplementing buffer capacity

  - however, at low temperatures blood may exhibit **rouleaux formation, platelet aggregation** and slugging

  - when oxygenated crystalloids were compared with blood, both hypothermic, the results were conflicting, however both protected better than air-exposed crystalloid solutions

• **Additives: Metabolic**

  - one problem associated with using glucose in non-oxygenated solutions, or low flow ischaemia, is the resultant build-up of $H^+$ may worsen intracellular $Na^+$ and $Ca^{++}$ changes during reperfusion

  - ATP levels after ischaemia may also be reduced because of reductions in amino acids

  - **glutamate & aspartate** are important precursors of the Kreb's cycle and are reduced in ischaemia

  - enriched blood has been compared with normal blood reperfusion in regional ischaemic models, with variable functional, but significant metabolic improvement

• **Additives: Non-Metabolic**

  - Goto *et al.* (CJA 1991), found that,
    a. crystalloid cardioplegia increased **myocardial oedema**, and
    b. water content prior to reperfusion was inversely related to **ventricular function**
    c. the addition of **mannitol** to the solution decreased water slightly,
       but improved LV function dramatically

  - hetastarch alone did not provide an increase in function, but does so if combined with glucose

  - mannitol has been suggested to be a free radical scavenger, and this may be part of its action

• **$O_2$ free radicals** are produced via the **xanthine oxidase** system and may result in myocyte dysfunction and injury

  - animal experiments which reduce free radical production or increase removal and have been shown to decrease reperfusion injury, include,
    i. superoxide dismutase
    ii. catalase
    iii. allopurinol
    iv. desferroxamine
    v. coenzyme-Q10
    vi. mannitol
PMN's may also play a role in reperfusion injury, and inhibition by perfluorochemical perfusion, adenosine, and mechanical or chemical depletion have been investigated. Altered calcium metabolism & handling appears to play an integral role in reperfusion injury. Studies of the CEB's in global and regional ischaemic models have produced mixed results. The negative inotropic effects of these agents may limit their clinical use. Some work has suggested that the increase in \([Ca^{++}]_{ICF}\) in reperfusion is related to ICF acidosis during ischaemia.

1. ICF H\(^+\) is exchanged for ECF Na\(^+\) to limit \([H^{+}]_{ICF}\)
2. Raised ICF Na\(^+\) is then exchanged with Ca\(^{++}\) via an antiport, raising the \([Ca^{++}]_{ICF}\)

**NB:** this work would suggest that efforts should be directed at limiting the increase in \([H^{+}]_{ICF}\) rather than modifying Ca\(^{++}\) flux.

### Warm vs. Cold Cardioplegia

**Hypothermia** reduces myocardial VO\(_2\) \(~7\%/\degree C\)

* however, it has been shown to,
  i. Inactivate the Na\(^+*/K^+\)-ATPase and Ca\(^{++}\)-ATPase of the sarcoplasmic reticulum, leading to loss of cell volume control and **swelling**
  ii. Decreased fluidity of cell membranes and decreased transport functions
  iii. Denature proteins - loss of enzymatic function
  iv. Precipitate ion complexes, resulting in variations in pH
  v. Cause osmotic shifts which may rupture cellular or subcellular membranes

The major reduction in VO\(_2\) with hypothermia comes from the reduction in **heart rate**,

1. **Normothermic electromechanical arrest** \(\rightarrow\) \(\downarrow\) VO\(_2\) \(~90\%\)
2. **Hypothermia** to 11\(^\circ\)C results in only a further 5\% reduction

Lichtenstein proposed normothermic, hyperkalaemia arrest may offer advantages over traditional hypothermic arrest, due to the absence of damage to subcellular organelles. This has yet to be proven by controlled clinical trial.

### Mode of Reperfusion

* VO\(_2\) of myocardium varies,
  a. Arrested, non-distended \(\sim 1\) ml/min/100g
  b. Arrested, distended \(\sim 5\) ml/min/100g
  c. Beating, empty \(\sim 5\) ml/min/100g
  d. Normal working myocardium is \(\sim 10\) ml/min/100g \(\sim 300\) ml/min total blood flow / 280g heart

Reperfusion in an arrested, non-distended ventricle results in better metabolic outcome following regional ischaemia. Allen *et al.* found \(\sim 20\) minutes are required for VO\(_2\) uptake to reach pre-ischaemic levels.
Discontinuation of CPB

- failure to resume **ventilation** is an easy oversight due to disabling of monitoring during bypass
- rewarming increases the incidence of **awareness** and supplemental anaesthetic may be required
- **cardiac function** is the principal limiting factor in separation from bypass, and this is directly related to,
  1. the patient's preoperative LV function
  2. the application of preservation techniques by the surgeon and perfusionist
  3. the effectiveness of the surgical procedure

- factors tending to delay full recovery include,
  1. poor LV function pre-bypass - CI < 2.2 l/min/m²
  2. inadequate bypass graft flow
  3. uncorrected regional ischaemia - ie. diffuse atherosclerotic disease
  4. continued hypothermia - ie. separation attempted too early
  5. VF or other arrhythmias
  6. ventricular distension or hypertrophy

- optimal perfusion pressure during reperfusion is debated, though, immediately following AOX declamping low pressures (~ 30-50 mmHg) are associated with less myocardial injury and oedema
- at some indeterminate point thereafter, higher perfusion pressures (~ 60-80 mmHg) appear to facilitate separation from bypass
- control of **rate & rhythm** facilitate separation and pacing to ~ 70-90 bpm should be used where appropriate to maintain atrioventricular synchrony
- ventricular irritability can be suppressed,
  i. pharmacologically
  ii. electrically - overdrive pacing
  iii. mechanically - repositioning of intra/extracardiac catheters

- optimisation of **preload** requires titration to effect
- LV compliance is reduced immediately post-bypass and relatively high PCWP's may be tolerated without undue increases in chamber diameter
- ventricular **overdistension** should be avoided due to the increased costs in VO₂ and the risks of functional MR
- if this occurs, then acute decompression an extended period of reperfusion on bypass is required
- the use of **inotropes** varies between institutions but is dependent upon the state of the peripheral circulation at the time of separation
  a. those who separate early, when core temperatures first reach 37°C often encounter an elevated SVR and find vasodilatory agents most effective
  b. those who separate later will face a lowered SVR and may require agents with primarily α-agonist action
when usual measures to wean fail, then possible considerations include,

1. unrecognised mechanical problems
   - kinked vein graft
   - prosthetic valve failure or misplacement
   - TEE may provide valuable information in these circumstances

2. extended "payback" may be required
   - continuing bypass for 15-30 minutes may be helpful
   - the "stunned" myocardium may benefit from a higher O\textsubscript{2} carrying capacity and elevation of the Hct to the mid-high 20's may be beneficial

3. creative pharmacology
   - amrinone/milrinone may be beneficial in some patients, due to their inotropic and vasodilatory properties
   - if elevated PAP / PVR is suspected then PGE\textsubscript{1} or isoprenaline may be helpful, however these may have to be combined with α-agonist agents

4. institution of mechanical LV support - usually IABP

Post-Bypass

1. **protamine** administration ~ 3 mg / 300U heparin / kg
   - this should be done slowly due to the risks of hypotension 2° to,
   i. myocardial depression
   ii. histamine release
   iii. systemic vasodilatation - rate dependent effect
   iv. pulmonary vasoconstriction
   v. anaphylactoid & anaphylactic reactions ± bronchospasm & pulmonary oedema
   - LA or intra-aortic administration significantly reduces but does not eliminate the haemodynamic and pulmonary effects
   - the systemic effects appear to be due to the heparin-protamine complex
   - clotting factors and platelet function may be abnormal post-bypass and are included in the differential of excessive bleeding

2. **air embolism**
   i. systemic - open chamber procedures
      - LA enlargement, LV aneurysm, chronic AF
      - CNS & heart consequence
   ii. myocardial - temporary ischaemia and decreased function
      - fine needle aspiration prior to initiation of flow
   iii. N\textsubscript{2}O contraindicated

3. particulate embolisation
   - LA enlargement, chronic AF, aortic valve vegetations, LV aneurysm
Other Organ Systems During Bypass

- **Pulmonary Ventilation**
  - maximal $\text{SaO}_2$ and **normocarbia** are desirable, however attainment of these may be hampered by,
    1. the requirement for small tidal volumes during dissection
    2. actions of vasoactive drugs on the pulmonary vasculature
    3. accumulation of cellular and other debris in pulmonary capillaries
    4. interstitial and/or frank pulmonary oedema
    5. atelectasis
    6. pneumothorax, haemothorax
    7. improper function of the CPB pump-oxygenator
  - RDM suggests checking AGA’s at a minimum,
    i. following intubation
    ii. soon after going-on bypass
    iii. during rewarming
    iv. prior to and after coming-off bypass
  - postoperative ventilation should be maintained until,
    1. the patient is transferred to the intensive care setting
    2. reasonable haemodynamic stability is present
    3. postoperative bleeding is at an acceptably low rate
    4. body rewarming is complete
    5. the patient is conscious and able to respond to commands
    6. the patient meets the desired **criteria for extubation**, 
      i. $\script{P\text{O}_2} < 50\%$
      ii. PEEP $\leq 5 \text{ cmH}_2\text{O}$
      iii. $P_{\text{a}O_2} > 60 \text{ mmHg}$
      iv. $P_{\text{a}CO_2} < 50 \text{ mmHg}$
      v. IMV $\leq 4 \text{ bpm}$
      vi. VC $\geq 30 \text{ ml/kg}$
      vii. SRR $< 20-30 \text{ bpm}$
      viii. a resolving CXR (ie. no new findings)
      ix. no other major organ system failure or instability
• **Acid-Base Regulation**

  - anaerobic **whole blood** follows the correction formula of *Rosenthal*,

    \[ \delta \text{pH} / ^\circ \text{C} = -0.015 \]

  - this is due principally to the presence of large quantities of **imidazole groups** on histidine moieties of plasma proteins
  - pH = 7.0 for pure water only occurs at a temperature of 25 °C, and the pH/temperature slope of **pure water** is not dissimilar \( \delta \text{pH} / ^\circ \text{C} = -0.017 \)
  - thus, blood *in vitro* with a constant CO\(_2\) content maintains a relative constant alkalinity with respect to pure water
  - dissociation curves of the **fractional dissociation** (\(\alpha_{\text{Im}}\)) of imidazole, show a relatively constant value (~ 0.8) over a wide range of body temperatures, despite variable pH, so called *\(\alpha\text{-stat}\)* conditions
  - maintaining a temperature corrected pH ~ 7.4, so called *pH-stat* conditions, results in a variable \(\alpha_{\text{Im}}\), and as this is the principal protein buffer, also results in a variable charge state of other protein groups
  - this contrasts to the relatively constant charge state under *\(\alpha\text{-stat}\)* conditions

  - ectothermic animals display an *in vivo* \(\delta \text{pH} / ^\circ \text{C}\) curve which resembles that of *in vitro* blood
  - studies of blood and CSF CO\(_2\) conductance show a strong probability that a constant \(\alpha_{\text{Im}}\) is maintained systemically by **chemoreceptors** which drive ventilation in response to the CO\(_2\) transport properties of blood
  - ie. alphastat regulation of CSF receptors effectively provides alphastat regulation of body fluids
  - further, the intracellular pH is usually close to the pNH\(_2\) and follows a \(\delta \text{pH} / ^\circ \text{C}\) slope similar to that of the ECF
  - this suggests that ECF alphastat regulation is followed by the ICF

  - other factors supporting the **alphastat** principal include,
    1. maintenance of the Donnan ratio and red blood cell volume
    2. maintenance of the pH gradients across the inner mitochondrial membrane
    3. preservation of enzymatic functions integral to energy production
      i. lactate dehydrogenase
      ii. phosphofructokinase
      iii. citrate synthetase - entry of acetyl-CoA into Kreb's cycle
      iv. pyruvate dehydrogenase
    • although there is a growing list of enzymes which have optimal pH's which are inversely related to temperature, the dissociation slopes vary considerably
      \[ \rightarrow \text{NADH cytochrome c reductase} = 0.044 \]
      \[ \text{acetyl-CoA carboxylase} = 0.027 \]
    • irrespective of these differences, alphastat management will result in considerably greater enzyme function at a given temperature than pH-stat
    4. peak activity relationship for Na\(^+\)/K\(^+\)-ATPase, \(\delta \text{pH} / ^\circ \text{C} = -0.017\)
Hibernation

- in contrast, hibernating mammals exhibit a near constant, normothermic pH of 7.4 over a wide range of temperatures, ie. \textit{pH-stat}
- they demonstrate loading of CO\textsubscript{2} prior to hibernation and hyperventilation during arousal
- they are therefore acidic and demonstrate a decrease in \(\alpha\text{Im}\)
- however, both the heart and liver demonstrate \textit{intracellular alphastat} regulation, with a negative slope of pH with temperature
- this results in an acidosis induced depression of metabolism in all organs except the heart and liver, and is significantly different from pH-stat management of induced hypothermia in humans
- the later results in decreased myocardial contractility and disordered Ca\textsuperscript{2+} flux

\textit{alphastat} regulation to \(\sim 25^\circ\text{C} \rightarrow P_{a\text{CO}_2} \sim 22 \text{ mmHg}\)
- previously CO\textsubscript{2} was added due to the fears of hyocapnia induced decreases in cerebral perfusion
- the low \(P_{a\text{CO}_2}\) values seen with alphastat management produced decreases in CBF \(\sim 30-50\%\)
- however, CBF is still in excess of CMRO\textsubscript{2} and the brain has no O\textsubscript{2} storage capacity
- pH-stat management effectively uncouples CMRO\textsubscript{2} & CBF, resulting in relative \textit{overperfusion}
- the later may therefore exacerbate raised ICP and decrease overall CPP
- further, the excess blood flow has been incriminated in delivery of microemboli to the CNS

- during alphastat regulation to \(28^\circ\text{C}\) in dogs,
  1. LV VO\textsubscript{2} is \(\sim 1.8\)x that during pH-stat
  2. lactate consumption is suppressed during pH-stat regulation and there is a lower peak pressure generated against a standard load (200 vs 330 mmHg alphastat)
  3. under pH-stat conditions there is a decrease in the VF threshold, cf. alphastat regulation and normothermic-normocapnic hearts

\textbf{NB:} Kroncke \textit{et al.} (Arch Surg 1986) looked at the incidence of spontaneous VF prior to AOX clamping during hypothermia to \(24^\circ\text{C} \rightarrow 20\%\) versus 40\% for pH-stat

- further, alphastat management requires no calculations, as measurements by standard AGA analysis are performed at 37\^\circ\text{C}, irrespective of the level of hypothermia

- Bashein \textit{et al.} (Anesth. 1990) randomised controlled trial of alphastat vs. pH-stat,
  1. showed \textit{no difference} in neurological outcome
  2. careful \textbf{surgical technique} and \textbf{arterial filtration} are probably more important for cerebral protection than any monitor, anaesthetic drug, or arterial blood gas management technique!
Renal Function

- during hypothermic CPB and a large volume of dilute urine is usually produced due to,
  1. depression of **tubular function**
  2. **mannitol** used in the priming solution
  3. **haemodilution**
  4. maintenance of renal perfusion pressure

- this is advantageous in the presence of **haemolysis** and the use of $K^+$ in the **cardioplegic** solution
- in the absence of cardioplegia, this may result in $K^+$ loss through the distal tubules requiring supplemental replacement
- cardioplegic solutions typically contain $[K^+] ~ 25$ mmol/l, however this is usually less than the amount lost through the kidneys (dependent upon bypass time)
- should urine output fall < 1 ml/kg/hr then causes of obstruction should be eliminated prior to steps to increase flow being taken
- tubular function readily returns following bypass, providing renal perfusion pressure has been maintained
- body weight gain is typical of postoperative cardiac patients and there is usually a spontaneous diuresis about day 3-4 with return of normal homeostatic mechanisms

Body Temperature & Metabolism

- factors of importance during rewarming include,
  1. recovery of **consciousness** and requirement for additional anaesthetic
  2. avoidance of volatile agents due to their myocardial depressant properties
  3. avoidance of $N_2O$ due to the risk of **air emboli**
  4. increased rate of biotransformation of **heparin** and increased coagulability of blood
  5. cellular uptake of potassium, exacerbating **hypokalaemia**

- ventricular defibrillation and maintenance of a regular rhythm is difficult < 34°C
- recooling of the body can occur unless the CPB rewarming time is sufficient to bring most of the body to "normal" temperature
- persistence of temperature **gradients** is an indication of inadequacy of rewarming
- surface warming is inefficient and cannot be relied upon to maintain body temperature, or correct heat deficits in adult patients
- **pulsatile perfusion** improves rewarming and can be accomplished by **partial bypass** at the end of the procedure
- **vasodilators** may also improve the rate of rewarming
- **persistent hypothermia** contributes to various postoperative problems, including,
  1. coagulopathy
  2. hypertension & tachycardia 2° to sympathetic overactivity
  3. shivering with increased $VO_2$
Diabetes

- in a 1980 study of 340 diabetics vs. 2522 nondiabetics undergoing CABG,
  1. moderate increase in operative mortality ~ 1.8% vs. 0.6%
  2. requirement for inotropic support & IABP ~ 5x ↑

- reasons for these differences include,
  1. more extensive and diffuse CAD
  2. higher incidence of,
     i. preoperative hypertension
     ii. cardiomegaly
     iii. diffuse hypokinesis
     iv. previous MI
  3. IDDM patients with CAD have stiffer LV’s with elevated LVEDP
  4. autonomic dysfunction → ↓ preload regulation
  5. CPB → ↓ responsiveness to insulin due to,
     i. hypothermia
     ii. the stress reaction 2° to CPB
     iii. sympathomimetic amines
       • results in marked hyperglycaemia, even without glucose in the IVT
       • washed cells have been advocated as ACD significantly increases BSL
       • insulin administration has little effect until rewarming
       • lactate containing solutions are gluconeogenic & poorly absorbed
  6. IDDM with poor LV function may have operative mortality ~ 10-15%

- these make management of diabetes difficult
- uncontrolled hyperglycaemia can contribute to the diuresis and is associated with a worse outcome following focal and global CNS ischaemia
CNS Protection

- the CNS is at particular risk from hypoperfusion, especially in elderly patients,
  a. changes in MAP and flow  - hypothermic CPB
    - "hypocarbia"
    - vertebral or carotid artery occlusive disease
    - positioning of the neck
  b. increases in venous pressure
  c. emboli  * air, thrombus, microaggregates/debris

- with the possible exception of multi-lead, processed EEG, no monitoring technique has been prospectively validated as beneficial to neurological outcome
- most centres chose not to monitor the CNS except for,
  i. barbiturate induced coma  ?? benefit
  ii. combined CEA and CABG procedures

- barbiturate prophylaxis probably is not of benefit in closed chamber, hypothermic CABG surgery

<table>
<thead>
<tr>
<th>Monitoring for Cerebral Ischaemia</th>
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<tr>
<td><strong>Method</strong></td>
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<tr>
<td>Single or Dual lead EEG, or</td>
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<td>Processed EEG</td>
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<tr>
<td>Multi-lead raw, or Processed EEG</td>
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<td>Carotid or Transcranial Doppler</td>
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<td>Jugular bulb - SjO₂</td>
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<tr>
<td>Regional CBF, or velocity</td>
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<td>Spinal CSF pressure</td>
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- other preventative measures include,
  1. evaluation ± CEA for symptomatic carotid bruits prior to CABG
  2. maintenance of a slightly higher MAP in the presence of cerebrovascular disease
  3. scrupulous avoidance of air/atheromatous emboli & cessation of N₂O in their presence
  4. prevent hyperglycaemia
  5. good surgical technique & short bypass times
HYPOTHERMIA

*Def'n: core temperature* < 35°C

*homeotherms* and regulate core temperature

~ 36-37.5°C (T.Oh)

~ 37 ± 0.4°C (RDM)

i. mild > 33°C

ii. severe < 33°C

*NB:* demarcation is arbitrary, but effects more pronounced & *loss of compensation*

- **Aetiology**
  a. extremes of *age*
  b. debilitating *illness*
    i. CNS - CVA, head injury, neoplasm
       - progressive mental deterioration
    ii. CVS - CCF, MI, PVD, PTE
    iii. infections - septicaemia, pneumonia
    iv. renal - uraemia
  c. *exposure* - environment
    - IV fluids
    - irrigating fluids
  d. *drugs* - alcohol
    - GA
    - antipyretics
    - vasodilators
    - chlorpromazine
  e. *endocrine* - hypothyroidism
    - panhypopituitarism
    - Addisonian crisis, hypoglycaemia
    - diabetes, hyperosmolar coma, ketoacidosis (20%)
    - protein / calorie malnutrition
  f. *spinal cord trauma*
  g. *skin diseases* - psoriasis
    - ichthyosis
    - erythroderma
  h. *iatrogenic* - induced hypothermia & inadequate rewarming
Clinical Effects

**Cardiovascular**

1. increased sympathetic tone - ↑ plasma NA/AD and FFA's
2. initially → vasoconstriction, tachycardia & increased CO
   
   *later* → bradycardia, hypotension & decreased CO
3. cardiac output - ↓ CO ~ 30-40% at 30°C ∝ decrease in VO₂
   
   - mainly 2° to bradycardia, SV well preserved
   
   - coronary perfusion well maintained
4. ECG changes - exacerbated by acidosis & hyperkalaemia
   
   i. bradycardia
   
   ii. prolonged PR, QRS, QT duration
   
   iii. J point elevation ~ 33°C
   
   iv. AF ~ 25-34°C (commonest arrhythmia)
   
   v. AV block 1° ~ 30°C
      
      3° ~ 20°
   
   vi. VF ~ 28°C
   
   vii. asystole ~ 20°C
5. CPK & LDH levels are elevated
   
   - ? leakage from cells or microinfarction

**Central Nervous System**

• reasonably well preserved to 33°C, below this function deteriorates progressively,

1. initial confusion → coma at ~ 30°C with pupillary dilatation
2. ↓ CBF ∝ ↓ CMRO₂ ~ 6-7% / °C
   
   ~ similar change cf. whole body VO₂
3. progressive brainstem depression → ↓ HR & ↓ RR
4. ↓ temperature regulation → ↓ shivering ~ 33°C
   
   → loss of temperature control ~ 28°C
5. cerebral protection
   
   i. over and above metabolic depression
   
   ii. deep circulatory arrest
   
   iii. recovery from near drowning
**Pulmonary Changes**

1. central depression → ↓ RR ≤ 33°C
   ~ 4 bpm ± respiratory arrest at 25°C
2. impaired cough & gag reflexes → aspiration risk
3. reduced CO₂ drive
4. no change in hypoxic drive
5. impaired hypoxic pulmonary vasoconstriction
6. reduced FRC, increased atelectasis
7. decreased gaseous diffusion capacity
8. ↑ VO₂ with shivering → ↓ VO₂ ≤ 33°C
9. ↓ O₂ availability ∝ ↑ HbO₂ affinity
10. increased gas solubility
   i. ↑ αCO₂ / ↓ P_{4CO₂} → ↑ pH
   ii. anaesthetic gases → ↓ rate of rise of F₄/F₁ & elimination
      - halothane MAC₂⁷°C ~ 50% MAC₃⁷°C

**Metabolic**

1. ↓ VO₂ ~ 6-7% / °C
2. severe acidosis → HbO₂ curve shifts to the right
   i. respiratory ↓ CO₂ elimination due to hypoventilation
   ii. metabolic ↓ tissue perfusion
      ↓ hepatic lactate clearance
      ↓ renal tubular H⁺ excretion
   iii. temperature correction of blood gas values offer no advantage in management
      → δ pH ~ -0.0147/°C
3. hyperkalaemia / hypokalaemia
   • causes for expected rise in K⁺
   i. decreased activity Na⁺/K⁺-ATPase → ↓Na⁺ / ↑K⁺
   ii. cellular hypoxia, membrane damage & acidosis
      • however, hypokalaemia commonly observed
   i. ? 2° diuresis
   ii. ICF shift
4. hyperglycaemia - ↓ insulin secretion & ↓ peripheral glucose utilisation
   - ? mild pancreatitis
   - hypoglycaemia may ensue in longstanding hypothermia
5. ↑ drug t_{½} ∝ ↓ hepatic blood flow & enzyme reaction rates
   → heparin, citrate & lactate
**Renal**

1. decreased GFR $\propto$ ↓ renal blood flow ~ 50% at 30°C
   $\downarrow$ drug clearance
2. decreased tubular function
   i. cold diuresis - volume of urine initially increased or the same
   ii. hypoosmolar urine
   iii. glycosuria, kaluria $\rightarrow$ additional diuresis

**Neuromuscular Junction**

1. shivering occurs ~ 33-36°C
2. increased muscle tone $\rightarrow$ **myoclonus** ~ 26°C
3. increased sensitivity to **both** depolarising & nondepolarising with mild hypothermia

**Haematological**

1. **coagulopathy**
   i. $\downarrow$ coagulation $\downarrow$ enzyme activity
   ii. thrombocytopaenia $\uparrow$ portal platelet sequestration $\uparrow$ bleeding time
2. increased blood **viscosity** - haemoconcentration & $\uparrow$ Hct.
   - dehydration
   - $\downarrow$ microcirculatory blood flow
3. **immunoparesis** - decreased WCC & function
4. marrow hypoplasia

**Immunological**

1. decreased neutrophils, phagocytes, migration, bactericidal activity
2. organ hypoperfusion & increased infection risk
3. diminished gag/cough reflexes
4. atelectasis
Regulation of Body Temperature

*NB:* balance between heat production and heat loss

a. heat production / gain
   i. basal VO₂
   ii. muscular activity
   iii. SDA of food
   iv. non-shivering thermogenesis
   v. gain from the environment

b. heat loss
   i. radiation  ~ 40%
   ii. convection  ~ 30%
   iii. evaporation  ~ 29%
   iv. conduction, feces/urine  ~ 1%

*NB: respiratory losses*  ~ 10%
   i. humidification  ~ 8%
   ii. convection  ~ 2%

### Sensory Systems

- cutaneous thermoreceptors  ~ 15% of input
  - cold receptors  < 24°C
  - heat receptors  > 44°C

- deep/core thermoreceptors  ~ 85% of input
  - anterior hypothalamus
  - spinal cord
  - hollow viscera

### Central Integration

- some processing in the spinal cord, majority in the *posterior hypothalamus*
- "central thermostat" regulated by,
  1. diurnal rhythm, age, sex, hormones
  2. endogenous pyrogens
  3. drugs
  4. neurotransmitters  (? 5HT)
  5. exercise
**Effector Systems**

1. **higher control centres**
   i. posture, avoidance behaviour
   ii. apetite/hunger
   iii. clothing
   iv. level of activity → voluntary muscle metabolism
      \[ \uparrow \text{BMR} \leq 1000\% \text{ with exercise} \]

2. **cutaneous blood flow**
   - especially the extremities
   - may decrease skin blood flow to \(~ 5\% \) of normal & heat loss to \(~ 12\% \)
   - first line of defence activated against heat loss

3. **shivering thermogenesis**
   - involuntary incoordinate muscular activity \(~ 50 \text{ Hz} \)
   - may \[ \uparrow \text{BMR} \sim 200-500\% \]
   - may \[ \uparrow \text{core temperature} \sim 2-3 \degree \text{C/hr} \]
   - requires \[ \uparrow \text{VO}_2 \sim 100\% / \uparrow 1\degree \text{C} \]

4. **nonshivering thermogenesis**
   - increased combustion of FFA's and glucose, regulated by,
     i. sympathoadrenal outflow → fast response - noradrenaline
     ii. thyroid function → slow response - adrenaline & T\(_4\)
   - liver and skeletal muscles in adults \sim 25\% \[ \uparrow \text{BMR} \]
   - *brown fat* in neonates \sim 100\% \[ \uparrow \text{BMR} \]
   - \sim 25\% of total CO

5. **sweating**
   - direct or reflex stimulation of the spinal cord, medulla, hypothalamus or cortex
   - provides only coarse control of temperature

6. **horripilation / piloerection** - minimal effects in man cf. animals

**NB:** usually order of activation,
   i. behavioural modification
   ii. vasoconstriction
   iii. nonshivering thermogenesis
   iv. shivering thermogenesis
Effects of Anaesthesia

- **Unintentional Hypothermia**

1. **↓ heat production**
   - i. **↓ VO₂** ~ 25-30% 
     ~ 1 kcal/kg/hr
   - ii. **↓ muscular activity & shivering**

2. **↑ heat losses**
   - i. **↑ radiation / convection** - undressed in cold theatre 
     - large surgical incisions
   - ii. **↑ evaporation** - cold preparation solutions 
     - from the wound 
     - cold/dry anaesthetic gases 
     - bypassing of upper airway
   - iii. **↑ conduction** 
     - cold IV solutions - 1 kcal/°C/l → ~ 17 kcal / 1000ml / 20-37°C 
     ~ 1% of BMR
     - cold table (minimal) & wet drapes

3. **inhibition of thermoregulation**
   - i. **↓ hypothalamic set point**
     - enflurane/halothane + N₂O/O₂ ~ 34-34.5°C
     - isoflurane + N₂O/O₂ ~ 3°C/ET%ISO
     - propofol + N₂O/O₂ ~ 33°C
   - ii. inhibition of effector responses
     - vasoconstriction & NST only means of heat gain available
     - GA → vasodilatation & redistribution of heat
     decreased core-shell gradient
     responsible for the initial rapid fall ~ 0.5-1.5°C
     - RA → similar initial loss of core-peripheral gradient
     central regulation preserved
     this is the origin of **shivering** with epidural blockade (?? not spinal)

4. **at risk groups**
   - i. neonates - high SA:V ratio, immature thermoregulation, no shivering
   - ii. elderly - low BMR, ↓ body mass
   - iii. prolonged procedures
   - iv. large central incisions
   - v. burns
   - vi. trauma patients, large volume transfusions/blood loss
   - vii. ↓ metabolism - adrenal insufficiency, hypothyroidism, hypopituitarism
Perioperative Effects

1. protection against CNS ischaemia, even with mild hypothermia  
   (Sano et al. 1992)
2. metabolic  
   - acidosis, hyperkalaemia  
   - decreased drug metabolism (citrate, heparin, opioids, relaxants)
3. haematological  
   - ↑ viscosity, ↓ O₂ delivery & tissue hypoxaemia  
   - impaired coagulation
4. CVS  
   - ↓ CO & arrhythmias (*AF)
5. postoperative problems
   i. shivering  
      - ↑ VO₂ & hypoxia if borderline lung function
   ii. marked vasoconstriction  
      - decreased microvascular flow
      - ? graft survival & wound infection
      - haemodynamic instability on rewarming
   iii. impaired drug clearance
   iv. impaired immune function  
      - predisposes to wound infection
   v. impaired conscious level

Intraoperative Management

1. ↑ ambient temperature
   i. adults under cover ~ 21°C
   ii. neonates ≤ 26°C
2. radiant warmers  
   - mainly useful in children (higher SA:V ratio)
   - limited by access
   - potential for burns
3. drapes / coverings  
   - ↓ radiant & convective losses  
     - area more important than type, but must remain dry
     - losses from the head important in neonates/bald adults
     * forced air convective warmers most effective means
4. warming blankets  
   - most effective above patient, minimal losses to table
   - useful when patient < 10 kg
5. respiratory losses < 10% losses through the respiratory tract
   i. heat & moisture exchangers prevent most of this loss
   ii. heater humidifiers will prevent all of this loss
      • however, in adults are unable to significantly raise body heat content
      • studies showing otherwise actually looking at oesophageal probe changes
   iii. heater humidifiers rarely, if ever, indicated in adults
6. blood / IV warmers  
   - especially large volumes given rapidly
Monitoring During Anaesthesia

a. central - lower oesophageal & PA → heart
   - tympanic membrane → brain
b. rectal - intermediate
   - changes lag behind core/shell during cooling & warming
c. shell - skin/peripheral
   - may estimate vasoconstrictor/vasodilator responses

NB: useful to measure both core & shell,

\[ \text{core-shell gradient} \rightarrow \text{better assessment of overall body temperature} \]
\[ \rightarrow \text{adequacy of rewarming & predicts "afterdrop"} \]

Deliberate Hypothermia

Surface Cooling

- principally historical interest, main use currently is in the management of malignant hyperthermia, or severe hyperthermia in septic ICU patients
- cold environment, ice bathing, especially groins & axillae
- problems of slow & uneven effects both during cooling and rewarming,
  a. 2-6°C afterdrop when cooling / rewarming
  b. uneven effects mean some tissues are still "at risk" for ischaemia

Cardiopulmonary Bypass

a. more rapid & even cooling / rewarming
b. more precise temperature regulation
c. maintenance of tissue perfusion despite ↓ CO / arrest
d. combined with haemodilution
  i. offsets the effects on viscosity
  ii. "optimal Hct." ~ 18-22%

Deep Hypothermia & Total Circulatory Arrest

a. allows operation on still & bloodless heart
b. principally for correction of complex CHD
c. current operative times ~ 50-60 minutes at 18-20 °C
d. need for more thorough longterm outcome studies on CNS effects
ANAESTHESIA FOR VASCULAR SURGERY

- factors which distinguish major vascular surgery,
  1. impaired vital organ function by pre-existing vascular disease
     ± intraoperative aortic cross-clamping
  2. major physiological trespass due to,
     i. extensive retroperitoneal dissection
     ii. dissection around the thoracic aorta ± one lung anaesthesia
  3. acute hydraulic stress to the LV by AoX clamping
  4. risk of sudden & profuse perioperative haemorrhage
  5. adverse effects of massive blood transfusion

NB: for elective aortic repair, 30 day mortality ~ 1-6%  (cf. 4-9% 1970-75)
cf. 0.1-0.4% for combined mortality from other types of surgery (? vascular)

- the prevalence of coexisting disease in patients undergoing vascular surgery,
  a. hypertension ~ 40-60%
  b. heart disease ~ 50-70%
     i. previous MI ~ 40-60%
     ii. angina ~ 10-20%
     iii. CCF ~ 5-15%
  c. CAL ~ 25-50%
  d. diabetes mellitus ~ 8-12%
  e. renal insufficiency ~ 5-25%

- Cooperman et al. (1978) showed independent, highly statistical risk factors for adverse cardiac outcome following major vascular surgery,
  i. CCF
  ii. previous MI
  iii. previous CVA
  iv. cardiac arrhythmias
  v. an abnormal ECG

- other investigators have supported these finding, with the addition of angina
- the multifactorial cardiac risk index, developed by Goldman et al. has been applied to major vascular cases,
  a. accurately predicts the trend in adverse outcome
  b. understate the risk by 2-3 fold for vascular cases
     • predominantly Class I & II, classes III & IV are more accurate

NB: major vascular surgery itself is probably a risk factor for adverse cardiac outcome
Risk Factors for Major Vascular Surgery

a. clinical assessment
   i. age > 70 years
   ii. previous MI
   iii. angina pectoris
   iv. CCF
   v. cardiac arrhythmias
   vi. aortic valvular disease
   vii. renal insufficiency
   viii. respiratory insufficiency

b. laboratory assessment
   i. ECG - rhythm other than sinus, ischaemic changes
   ii. CXR - LVF, cardiomegaly
   iii. DPT scan - thallium redistribution, large area of non-viable myocardium
   iv. Echo - LVEF < 0.35, significant RWMA, cardiomegaly, valve disease
   v. MBA₂₀ - renal insufficiency or failure

c. intraoperative / procedure related
   i. operation type - ie. major vascular surgery
   ii. prolonged hypotension - perioperative hypertension
   iii. perioperative myocardial ischaemia
   iv. renal insufficiency or failure

NB: these are common in this population group, many have an asymptomatic period and clinical assessment is an insensitive marker

Cardiovascular Disorders

- Ischaemic Heart Disease
  - the common pathogenesis results in an incidence ~ 50-70%
    1. previous MI ~ 40-60%
    2. angina ~ 10-20%
    3. CCF ~ 5-15%

  NB: further, the incidence of intraoperative myocardial ischaemia ~ 50-70%

  Slogoff & Keats (Anesth.1985) → ↑ perioperative MI

  - MI remains the leading cause of mortality following surgery to the aorta and major branches
    → ~ 40-60% of mortality
    ~ 3-4x other causes
Assessment

1. history
   i. previous MI
   ii. angina - rest / exercise
      - accompanying dyspnoea → ischaemic LV dysfunction
   iii. exercise tolerance, SOBOE
   iv. drug history

2. examination - HR / rhythm / BP / peripheral perfusion
   - signs of CCF, ie. ischaemia induced LV failure

3. investigation
   i. 12 lead ECG / exercise ECG / 24 hr Holter monitor
   ii. echocardiography → RWMA
   iii. DPT scanning
   iv. angiography

- Atherosclerosis develops predominantly in the large epicardial arteries
- Vasodilatation retains perfusion until > 70-80% luminal narrowing
- Despite this, stenoses ~ 40-80% do limit myocardial reserve under conditions of stress
- Coronary angiography is the "gold standard" for assessment of coronary arteries, however is expensive and carries a definite morbidity/mortality

NB: Institutions routinely performing angiography prior to aortic surgery report, that in patients without clinical or ECG evidence of ischaemia, ~ 15-30% have > 70% luminal narrowing in 1 or more vessels

- Recent Cleveland Clinic studies reported,
  1. 5.7% mortality in 70 CABG's performed on patients with infrarenal aortic aneurysms
  2. An incidence of aortic rupture following CABG, prior to repair ~ 2.9%
  3. Operative mortality for aortic repair ~ 1.8%

NB: At that institution, preoperative angiography/CABG was not associated with an improvement in patient outcome, and is no longer practiced

- Therefore, proposed that selective patient screening and angiography may be more cost effective and offer a better improvement in patient outcome, ie. only perform CABG in patients with haemodynamically significant CAD
- Large number of studies addressing the predictive value of preoperative screening tests,

1. Exercise ECG - fair predictive value
   - good negative predictive value
   - technically difficult in patients with severe vascular disease

2. Holter monitor - independent predictor of adverse cardiac outcome
   - very good negative predictive value

3. Nuclear scanning (see table perioperative MI)
data regarding the predictive value of resting LVEF are mixed, recent studies suggesting it is a poor predictor. 
exercise response to LVEF is a better indicator of myocardial reserve, however suffers the same limitations as exercise ECG testing. 
dipyridamole-thallium scanning has been shown to be a more reliable predictor of perioperative myocardial ischaemia than exercise ECG, or any configuration of the known risk factors in this population.
thallium scans are taken 5 minutes following DPT administration, then again at 3 hours after the effects of DP have disappeared.

<table>
<thead>
<tr>
<th>Initial Scan (5 min)</th>
<th>Delayed Scan (3h)</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Defect</td>
<td>No Defect</td>
<td>Normal Scan</td>
</tr>
<tr>
<td>Segmental Perfusion</td>
<td>Thallium redistribution</td>
<td>At Risk Myocardium</td>
</tr>
<tr>
<td>Defect</td>
<td>Persistent defect</td>
<td>Non-viable Myocardium</td>
</tr>
</tbody>
</table>

the value of DPT scanning is it provides a means of determining those patients who should have angiography ± CABG prior to their aortic repair.
Reul et al. (J.Vasc.Surg 1986) showed a decreased mortality in high risk patients with coronary artery disease with CABG performed prior to major vascular repair.

Cunningham divides patients into 4 classes according to risk,

a. Class I
   - patients with no angina, previous MI, CCF, CVD, or diabetes
   - normal resting ECG
   - low surgical risk → proceed straight to surgery
b. Class II
   - abnormalities on routine evaluation, but normal DPT scan
   - low surgical risk → proceed straight to surgery
c. Class III
   - clinical CAD, redistribution on DPT scan
   - high surgical risk → proceed to coronary angiography ± elective CABG
d. Class IV
   - patients with poor LV function, diffuse small vessel CAD
   - inoperative CAD → serial 3 monthly U/sound & operate if ↑↑ size
Hypertension

- Systemic hypertension is the leading cause of LVH & LVF in adults
- Contributes significantly to the mortality from,
  - AMI
  - sudden death
  - CVA
  - acute aortic dissection
  - ruptured aortic aneurysm
  - renal failure

- Between 50-60% of patients for elective aneurysm repair have a history of chronic HT
- Despite treatment, ~40% of patients with aortic occlusive or aneurysmal disease remain hypertensive during the perioperative period, irrespective of treatment
- Pathophysiological factors important to anaesthesia,
  - Increased basal vasomotor tone & arteriolar hypertrophy → vascular "hyperreactivity"
  - Reduced intravascular volume
  - LVH - decreased compliance & preload dependence
  - Increased capillary leak
  - Regional circulatory changes
    - Renal - ↑ RVR > ↑ SVR
    - ↓ RBF ∝ duration & severity of hypertension
    - CNS - narrower autoregulatory range & shifted right
    - CVS - increased extravascular resistance
      - Basal coronary vasodilatation & decreased reserve
      - Increased incidence of ischaemia & SWMA

- Pyrs-Roberts et al. (BJA-1971,1972) found that,
  1. Degree of fall of MAP depended on preoperative MAP, not on treatment
  2. Cf. Patients rendered normotensive, untreated or inadequately treated patients had,
    - Greater decreases in MAP
    - More episodes of hypotension associated with subendocardial ischaemia
    - Greater ↑ SVR / ↓ CO with hyperventilation
    - Greater hypotension associated with epidural anaesthesia ± GA
  3. Treatment does not decrease the exaggerated hypertensive response to intubation
    - Confirmed by larger study by Goldman & Caldera (Anesth.1979)
    - Attenuated but not abolished by β-blockade

NB: All patients should have adequate medical therapy prior to elective operation, all anti-hypertensive medications should be continued to the day of surgery & recommenced as soon as practicable thereafter
Heart Failure

- present in ~ 10-15% of elective patients & complicates recovery in ~ 30%
- studies by Goldman et al. show adult patients undergoing non-cardiac surgery in the presence of decompensated CCF, an S3 gallop or elevated JVP, are associated with ~ 20% incidence of fatal cardiac outcome
- when other factors are removed by multivariate analysis, these remain the strongest indicators of adverse cardiac outcome (> previous MI)
- other factors on univariate analysis associated with adverse cardiac outcome include,
  i. pulmonary oedema ~ 14%
  ii. cardiac related dyspnoea ~ 6%
  iii. orthopnoea ~ 6%
  iv. peripheral oedema ~ 6%
  v. pulmonary rales ~ 5%
  vi. cardiomegaly ~ 5%

NB: a history of CCF, without clinical signs is not associated with increased risk, thus CCF patients appear to fall into 2 distinct risk groups,

i. decompensated CCF ~ 15-20%
ii. compensated CCF ~ 5% → adverse cardiac outcome

- early studies suggested preoperative LVEF was a good predictor of adverse outcome,
  1. Cutler et al. (1981) - LVEF > 56% no patients with adverse outcome
  2. Kazmers et al. (1988) - LVEF > 45% → 14% mortality
     - LVEF < 35% → 50% mortality

NB: more recent studies have indicated resting LVEF is a poor indicator of outcome, exercise LVEF changes may be a more sensitive marker of disease, however, the limitations of exercise ECG apply equally to this test

Electrolyte Abnormalities & Arrhythmias

- in the vascular surgical patient, arrhythmias have been identified as a major risk factor for adverse cardiac outcome
- common causes of electrolyte abnormalities are,
  i. diuretic therapy
  ii. renal insufficiency
  iii. diabetes mellitus

- hypokalaemia is frequently found with diuretic use and results in membrane hyperpolarisation
- this increases cardiac conduction & APD, enhances automaticity, and the increased incidence of supraventricular and ventricular arrhythmias in non-surgical patients is well established
- however, a careful randomised prospective trial failed to show any increased risk of intraoperative arrhythmias, "therefore, efforts to quickly normalise K+ levels should be avoided"
NB: rapid correction is actually more detrimental, being associated with a greater incidence of bradycardia, arrhythmias, conduction abnormalities and cardiac arrest during anaesthesia than no replacement at all.

- guidelines for K+ replacement include,
  1. chronic hypokalaemia, \([K^+] < 3.0 \text{ mmol/l or } > 20\% \text{ total deficit}\) → cancel elective surgery & replace orally over 3-4 days
  2. when oral replacement is inappropriate, then, (from Miller)
     i. give \(\leq 20 \text{ mmol K}^+ / \text{hr}\)
     ii. give \(\leq 240 \text{ mmol K}^+ / 24 \text{ hrs}\)
     iii. continuously monitor ECG
     iv. measure plasma \([K^+] \leq 4 \text{ hrly}\) ? this seems absurd
     v. allow at least 13 hrs for moderate deficit \(\sim [K^+] < 3.0 \text{ mmol/l}\)
        allow 24-48 hours for large deficit \(\sim [K^+] < 2.5 \text{ mmol/l}\)

- rapid correction effectively decreases the \([K^+]_{ICF}/[K^+]_{ECF}\) ratio, resembling acute hyperkalaemia

- **Chronic Medications**

1. **digoxin**
   - prophylactic digitalisation has been recommended for all elderly patients and "at risk" younger patients by some workers
   - however, these agents have narrow therapeutic indices and the conditions for which they are used occur infrequently
   - therefore prophylactic administration is only considered for patients at high risk for SVT's and unable to tolerate the haemodynamic consequences of the arrhythmia,
     i. stenotic valvular heart disease
     ii. elderly patients & upper abdominal / thoracic surgery
     iii. paroxysmal SVT with hypotension or symptoms of CNS insufficiency

2. **β-blockers**
   - resting cardiac performance in patients **without** CCF is not dependent upon β-adrenergic support
   - combination of β-blockade and GA does not produce excessive depression in the non-failing heart
   - actually protect the ischaemia prone myocardium from intraoperative stresses
   - the "propranolol withdrawal syndrome" may occur with acute cessation → ventricular arrhythmias, unstable angina ± AMI

3. **clonidine**
   - selective partial \(α_2\)-agonist \((~ 200:1)\) in both peripheral and CNS
   - results in severe rebound hypertension on withdrawal, (or with naloxone)
   - reduces MAC of inhalational agents & dose requirements of opioids
4. **GTN & "Nitro-preparations"
   - increase blood flow to subendocardial myocardium
   - this area is at greatest risk during haemodynamically mediated ischaemia
   - increase diastolic relaxation, positive lusitropy

5. **Ca\(^{2+}\)-channel blockers**
   - patients receiving combination β-blockade & CEB therapy may develop profound bradycardia or asystole during induction with fentanyl
   - exert additive effects on Ca\(^{2+}\) flux with the volatile agents in myocardial cells,
     i. decreased contractility
     ii. decreased conduction
     iii. peripheral vasodilatation
   - also potentiate the effects of the NMJ blocking agents
   - however, rebound coronary artery spasm has been reported on withdrawal
   - therefore continue but use other agents cautiously

6. **anticoagulants / platelet inhibitors**
   - Odom (1984) showed safety of regional anaesthesia with warfarin
   - Rao & El-Etr (1981) showed safety with intraoperative heparin
   - RDM, therefore perioperative anticoagulation should not preclude regional anaesthesia if there is a strong clinical indication for such
   - situation for antiplatelet agents is unsettled

Respiratory System

- **Chronic Airflow Limitation**
  - many patients are, or were, heavy smokers and there is a high incidence of CAL
  - problems for anaesthesia include,
    i. varying degrees of mucosal inflammation
      - altered secretions
      - increased smooth muscle reactivity
      - tissue destruction and loss of support for terminal airways
    ii. small airways closure & lung hyperinflation
    iii. increased shunt - V/Q mismatch and arterial hypoxaemia
    iv. recurrent pulmonary infections
    v. pulmonary hypertension ± RV hypertrophy & failure
      - R→L intrapulmonary shunt & hypoxaemia
      - increased arrhythmias

- even in normal patients, major abdominal / thoracic surgery results in decreased static lung volumes, smaller \( V_T \) and reduced or absent sighing, weakened cough and impaired gas exchange
- smokers & CAL suffers are at greater risk of atelectasis, pulmonary infection and respiratory failure
• criteria for preoperative *pulmonary function tests* include,
  1. all patients with CAL
  2. obesity
  3. advanced age > 65 years
  4. heavy smoking > 20 pkt/years ± cough

**Obesity**

  1. higher incidence of vascular disease - PVD, CVD & IHD
  2. respiratory embarrassment perioperatively
  3. airway difficulties on induction/recovery
  4. technically more difficult surgery

**Diabetes Mellitus**

  • higher incidence of both large and small vessel disease
  • potential for multiple end-organ dysfunction 2° *microangiopathy*
  • *has not* been identified as an *independent* risk factor for major vascular surgery, ie. patients matched for age, weight and other diseases

  *NB:* however, there is a higher incidence of other disease states which *are* risk factors

• important considerations in the management include,
  1. history & tendency to ketosis
  2. recent control of diabetes
  3. drug management - oral hypoglycaemins vs. insulin
     - recent insulin requirements
  4. presence of 2° organ dysfunction
     i. ischaemic heart disease
     ii. diabetic cardiomyopathy
     iii. renal insufficiency
     iv. autonomic neuropathy - orthostatic hypotension, resting tachycardia
        * disordered oesophageal motility, gastroparesis
        - increased susceptibility to IPPV
     v. peripheral neuropathy - motor/sensory
  5. infections
     i. cutaneous - risk of graft infection
     ii. other - especially UTI, pulmonary
Renal Function

- despite improved techniques, incidence of ARF ~ 0.2-3.0% following elective surgery
- mortality ~ 25% despite aggressive management
- despite stable haemodynamics, infrarenal AoXC → ~ 38% ↓ RBF ~ 75% ↑ renal vascular resistance

- other studies using microspheres have shown no change in the distribution of RBF, providing intravascular volume & CO are maintained
- predisposing factors in development of ARF include,
  a. pre-existing renal insufficiency
  b. function may be exacerbated by angiographic dye studies preoperatively
  c. risks of major haemorrhage & intraoperative hypotension
  d. risks of major transfusion
  e. perfusion alteration during infrarenal / suprarenal AoX clamping
  f. direct renal trauma during retroperitoneal dissection

Clinical Presentation & Management

- 2 principal disease processes,
  1. atherosclerosis
  2. thrombophlebitis - usually superficial / deep veins of lower limbs

■ Cerebrovascular Disease

- commonest lesions are at the,
  i. bifurcation of the common carotid artery, and
  ii. origin of the internal carotid artery

  NB: 75% with cerebrovascular disease have at least 1 surgically accessible lesion, 40% all disease is confined to the extracranial vessels

- these produce ischaemia via 3 mechanisms,
  1. hypoperfusion 2° to major vessel stenosis & inadequate colateral flow
  2. distal embolisation of atheromatous material from an ulcerated plaque
  3. diversion of blood flow away from the brain in a "steal syndrome"

- stroke may be the first presentation of CVD, however this is usually preceded by 1 or more transient ischaemic attacks TIA's, the sudden onset of neurological deficit which subsides over minutes to hours, leaving no residual deficit at 24 hours
• **carotid endarterectomy** is now the most commonly performed non-cardiac major vascular surgical procedure,
  a. **mortality** ~ 1-2%
     • most commonly 2° to MI
  b. **morbidity** ~ 4-10%
     • most commonly perioperative stroke
     • mostly patients with preceding neurological risk factors,
     i. progressing neurological deficit
     ii. neurological deficit < 24 hours duration
     iii. frequent daily TIA’s
     iv. permanent deficit 2° multiple cerebral infarctions

**Peripheral Occlusive Vascular Disease**

• most common sites, in order of frequency are,
  1. the superficial femoral artery in the lower limb
  2. the common iliac arteries
  3. the aortic bifurcation

• these result in claudication, rest pain, ischaemic ulceration and gangrene
• operative procedures include,
  1. femoropopliteal bypass
     • mortality 1-3%
  2. aorto-femoral/bifemoral bypass
  3. aorto-iliac endarterectomy
     • mortality ≤ 4%

**Renal Artery Stenosis**

  1. atherosclerosis ~ 2/3
     • usually confined to the proximal portion
  2. fibromuscular dysplasia ~ 1/3
     • diffuse disease, involving the distal renal artery

• 2 commonly used methods include,
  1. transaortic renal endarterectomy
  2. aortorenal bypass

• these frequently require suprarenal AoX clamping and a variable period of total renal ischaemia
• suprarenal clamping is associated with greater stress on LV function
• operative mortality ranges from 1-6% and is most often 2° acute MI
Aortic Aneurysmal Disease

- ultrasound assessment shows that,
  a. aneurysms > 5 cm diameter occur in ~ 1.5% of patients > 50 years of age
  b. these grow in size ~ 0.4 cm/year
  c. incidence of spontaneous rupture,
     i. aneurysm ≤ 5 cm ~ 10% / year
     ii. aneurysm > 7 cm ~ 76% / year

NB: therefore, elective repair should be performed early, but exact criteria disputed,
elective operative mortality ~ 4-8%, most commonly acute MI

- rupture is usually preceded by acute aneurysmal expansion, presenting with new abdominal or
  back pain in a previously asymptomatic individual
- acute rupture has a ~ 100% mortality unoperated, and most commonly presents with back pain
- in ~ 75% of patients the classic triad is present,
  i. back pain
  ii. hypotension
  iii. pulsatile abdominal mass

- the majority have overt hypovolaemic shock, BP < 80/- & HR > 100 bpm
- many will tamponade bleeding within the retroperitoneal space, providing a "window" during
  which they may be transferred to the operating theatre for resuscitation & operation

Thoracic Aortic Dissection

- usually 2° to hypertension (70-90%), but may occur 2° to collagen vascular disorders
- M:F predominance ~ 2:1, peaking in the 6-7th decades
- frequently associated with acute AI
- dissection may involve,
  i. retrograde into the pericardium
  ii. the origins of the coronary vessels
  iii. the innominate, common carotid and subclavian arteries
  iv. the blood supply to the spinal cord
  v. the mesenteric and renal arteries

- classified as proximal type A, (previously DeBakey types I & II) and distal type B (DeBakey III)
- modern management includes,
  a. conservative - relieve pain
     - Rx hypertension & minimise shear stress
     - thromboembolic prophylaxis later
  b. 1° repair under either,
     i. total circulatory arrest & deep hypothermia
     ii. cardiopulmonary bypass & selective brachiocephalic perfusion
Carotid Endarterectomy

1. ablation of surgical pain
2. provision of a motionless field and a relaxed patient
3. protection of vital organs from ischaemic injury

as noted, perioperative MI is the leading cause of mortality and CVA that of morbidity
chronic hypertension is present in 60-80%
Asiddao et al. (A&A 1982) have shown that the development of new neurological deficits is more common in those patients who develop postoperative hypertension
also, the development of postoperative hypertension is more common in those with poorly controlled preoperative hypertension

all currently used techniques require carotid cross-clamping, during which cerebral perfusion relies upon collateral flow through the circle of Willis, and via anastomoses between the external and internal carotid arteries
anaesthetic techniques to improve perfusion during X-clamping include,

1. maintenance of mild systemic hypertension
2. increasing cerebral / colateral flow by alteration of $P_{\text{aCO}_2}$
3. enhancing the brains tolerance of ischaemia by pharmacological depression of $\text{VO}_2$

NB: the relative merits of these techniques are disputed - see Neuroanaesthesia notes

**Anaesthetic Management**

a. history, examination & investigation
b. optimise medical management of coexisting disease
c. selection of anaesthetic technique
   i. GA
   ii. regional - cervical plexus block, local infiltration
d. premedication - anxiolysis
   - prevent preoperative hypertension or tachycardia
   - routine medications
e. monitoring
   i. routine monitors - $F_1 O_2$, ETCO$_2$, SpO$_2$, spirometry
   ii. CVS
   - NIBP / IABP
   - ECG, leads II + V$_5$ or CM$_5$
   iii. CNS
   - awake patient
   - SSEP’s, processed EEG
   - doppler CBF, ? regional CBF
   - stump pressure (??)
f. cerebral protective measures
i. prevent hypotension, induce mild hypertension
ii. maintain SpO₂
iii. mild ↓ P_{aCO₂}
iv. ↓ CMRO₂ - STP, isoflurane, propofol, etomidate
   * avoid hypotension
v. avoid hyperglycaemia

g. postoperative care
i. avoid coughing / bucking at extubation - lignocaine
   - fentanyl
ii. maintain airway patency
   - loss → hypercarbia, hypoxia, hypertension, raised ICP
iii. avoid hypertension & hypotension - volume status
   - pain relief
   - β-blockade (only if ↓ HR)
iv. prevent / manage myocardial ischaemia - GTN

**NB:** postoperative hypotension / bradycardia is a common finding and appears to relate to baroreceptor dysfunction, which appears to self-adjust over 12-24 hours

### Complications

1. haematoma formation ± upper airway obstruction
2. vocal cord paralysis
3. pneumothorax
4. CCF
5. MI
6. CVA
ABDOMINAL AORTIC SURGERY

- **Management Objectives**
  1. intensive preoperative assessment of risk factors
  2. management & optimisation of coexistent disease states
  3. utilisation of monitoring techniques which allow rapid assessment & management of myocardial function
  4. maintenance of intravascular volume, CO, tissue perfusion and oxygenation
  5. anticipation & prompt management of the haemodynamic & metabolic derangements associated with clamping / declamping
  6. intensive postoperative care

- procedures requiring AoX clamping present similar problems and include,
  a. aneurysmal resection
  b. vaso-occlusive disease
  c. lesions of the coeliac, mesenteric, or renal vessels

- **Premedication**
  - aim is to prevent "stress-induced" increases in HR, BP and SVR
  - especially those associated with placement of "lines" prior to induction
  - myocardial ischaemia associated with stress occurs at lower, or normal, heart rates cf. exercise induced ischaemia
  - increases in BP are also detrimental to aneurysmal disease
  - management should/may include,
    1. an informed preoperative visit by the anaesthetist
    2. a combined benzodiazepine/opioid premed
       - morphine 0.1 mg/kg & diazepam 0.1 mg/kg
       - in those with severe CVS/RS disease doses may have to be reduced
    3. continue usual medications
    4. drugs to reduce CVS "stress" - β-blockers
       - α2-agonists (clonidine 4-5 µg/kg po)
    5. metoclopramide / ranitidine
Monitoring

- minimum considered adequate (RDM),
  a. continuous ECG - II + V₅
     • lead selection as guided by distribution of CAD
  b. oesophageal stethoscope & temperature probe
  c. F₁O₂, SpO₂, ETCO₂, spirometry, disconnect
  d. NIBP & IABP + AGA's
  e. CVP ± PA catheter or TEE
  f. CUD + hourly urine output

- lead II of the ECG is useful for differentiating SVT's, however is relatively poor for ischaemia
- of the commonly used leads, V₅ has been shown to be the best for intraoperative use
- due to the extreme stresses placed upon the LV, monitoring of both of these leads is desirable
- as AoX clamping results in acute LV decompensation in many patients, monitoring of LV filling pressures is desirable
- in patients with good myocardial function, LVEF > 50%, the absolute value and the magnitude of change in CVP correlates well with PAoP
- in patients with unstable angina, previous MI or CCF, CVP does not correlate with PAoP
- TEE provides an accurate measure of the impact of AoXC on LV dimensions & systolic function
- RWMA's provide evidence of myocardial ischaemia and precede changes in PAoP or ECG
- studies have shown a significant disparity between LVEDA (area) and PAoP,
  a. linear regression showed a correlation coefficient < 0.3 in 77% of patients
  b. in 50% of patients, LVEDA & PAoP changed in the opposite directions !!
  c. proposed that changes in LV compliance occur during major aortic surgery

- alteration of LV compliance has been demonstrated in other studies

NB: therefore, all data available from the PA catheter should be used, not just PAoP in isolation,
  trends are of greater value than isolated values
Induction

- the ideal induction agent would provide,
  1. a rapid, smooth, excitement free onset of anaesthesia
  2. minimal inhibition / stimulation of sympathetic or parasympathetic function
  3. stable haemodynamics

- no agent meets all of these criteria
- the particular agent chosen is less important than the manner of administration, agents used,
  1. STP
  2. etomidate
  3. fentanyl / alfentanil

- to avoid a hypertensive response to induction,
  a. intubation should only be attempted once relaxants have taken effect
  b. in patients with good LV function,
     i. supplemental dose of thiopentone
     ii. volatile agent - MAC (endotracheal intubation) ~ 1.3 MAC
     iii. β-blockade - atenolol - esmolol ~ 1.5 mg/kg
  c. in patients with poor LV function,
     i. lignocaine ~ 1.5 mg/kg
     ii. fentanyl ~ 4-5 µg/kg

- patients with poor LV function and low CO will experience slow onset of anaesthesia with IV agents, NMJ blockers included, with a prolonged effect
- induction with the volatile agents will be more rapid
- a primarily opioid induction may be chosen in patients with poor LV function, however, these agents will still result in arterial hypotension
- the avoidance of tachycardia also applies to NMJ blockers
- large doses of pancuronium should be avoided, as the use of pancuronium as the sole relaxant during high dose opioid anaesthesia is associated with a greater incidence of intraoperative myocardial ischaemia (? reference)
- when required, succinylcholine is usually preceded by a small dose of nondepolarising agent to prevent fasciculations and an increase in intra-abdominal pressure
- rapid onset paralysis can also be achieved by high dose atracurium or vecuronium
- the onset for these agents can be further shortened by administration of a priming dose,
  i. atracurium ~ 0.08 mg/kg (0.3-0.5 mg/kg)
  ii. vecuronium ~ 0.015 mg/kg (0.08-0.1 mg/kg)

NB: ie. approximately 1/6th of the intubation dose results in good intubation conditions within 60-90 seconds
**Maintenance**

- despite causing myocardial depression, the volatile agents are frequently used as supplements
- halothane has been used to unload the LV in patients with elevated PAoP & LV failure
- in these patients, the vasodilatory action appears to be greater than the direct depressant effects
  \[ \text{small fall in CO } < \text{ decrease in PAoP and SVR} \]
- studies comparing high dose fentanyl / O\textsubscript{2} / isoflurane showed no benefit cf. a "conventional" low dose fentanyl / O\textsubscript{2} / N\textsubscript{2}O / isoflurane technique
- further, the addition of a volatile agent prevents the hyperdynamic response to AoXC seen with an unsupplemented opioid / O\textsubscript{2} technique
- however, in excessive concentration all volatile agents will result in systemic hypotension and reduced coronary perfusion pressure

- Reiz et al. (Anesth.1983) observed both coronary vasodilatation & myocardial ischaemia during isoflurane induce hypotension in 10/21 patients with IHD undergoing major vascular surgery
- Benefiel et al. (Anesth.1986), studied 100 patients randomly assigned to sufentanil or isoflurane maintenance, the isoflurane group showing,
  1. 4x greater incidence of postoperative renal insufficiency
  2. 3x greater incidence of postoperative cardiac failure

  **NB:** however, the relationship between steal-prone anatomy & these finding is unknown

- Becker (Anesth.1987), in an editorial review of the literature recommended that the use of isoflurane be limited in patients with severe IHD
- RDM therefore suggests that the concentration of isoflurane should be limited to < 0.75%, or its use should be avoided altogether
Slogoff et al. (A&A.1991)

- in a blinded retrospective review failed to show any difference in the occurrence of ischaemia between any of the volatile agents, or sufentanyl
- in a preceding randomised study of the effect of maintenance agent on the outcome after CABG surgery, they failed to find an increased incidence of ischaemia in the isoflurane group
- they then blindly reviewed the angiograms of these 1012 patients, selecting those with "steal-prone" coronary anatomy, ie.

  NB: complete occlusion of one vessel, supply by collateral vessels, 
  *plus* > 50% stenosis in the vessel supplying the collateral flow 
  → ~ 34% of the study group

- this percentage is comparable to the CASS (coronary artery surgery study) group ~ 23%
- a review of this group, > 16,000 patients, also failed to show any increase in ischaemia associated with isoflurane

- Slogoff's group also point out that human data supporting isoflurane steal is limited to a total 64 patients described in 5 studies, 27 of whom developed ischaemia
- however, the majority of these patients were subject to profound hypotension (35-45% ↓MAP)
- they conclude, "these data, when considered together do not document a steal mechanism as a cause of ischaemia during isoflurane anaesthesia"
- additionally they point out that,

  1. ECG ischaemia unrelated to any haemodynamic alteration is common in patients with CAD and occurs spontaneously in,
     i. the ambulatory state
     ii. during hospitalisation
     iii. intraoperatively

  2. *more than 85%* of intraoperative ischaemia is random and occurs unrelated to any haemodynamic abnormality, or to the anaesthetic agent administered

  **NB:** "our data....fail to support any recommendation for the withholding of isoflurane from any patients with any anatomic variant of coronary artery occlusive disease"
certain patients require stimulation of the circulation in order to maintain adequate organ perfusion, these include those with,

1. impaired LV function
2. CCF - valvular heart disease
   - IHD, or cardiomyopathy
3. hypotension 2° to hypovolaemia, or overt shock

opioid/O₂ + relaxant anaesthesia provides the advantages of,

1. a high F₁O₂
2. a low incidence of cardiac arrhythmias
3. minimal depression of contractile function

however, this results in prolonged postoperative respiratory depression, and frequently does not prevent the hypertensive responses to surgical stimulation or AoX clamping

improved control of circulatory responses can be obtained with,

1. low concentrations of N₂O or a volatile agent
2. IV supplements - diazepam, midazolam, droperidol
   - propofol infusion
3. β-adrenergic blockade - esmolol
4. vasodilators - GTN, nitroprusside
5. administration of opioid by continuous infusion

Regional Anaesthesia

Yeager et al. (Anesth.1987) compared GA with epidural + light GA for a number of procedures in a randomised study
postoperative morbidity 2° MI or CCF, major infection, and operative mortality were less in the epidural group
the beneficial effects with respect to ischaemia and CCF have been shown by others and are thought to relate to reduced preload & afterload 2 ° to vasodilatation
other reported benefits include,

1. pain relief without marked respiratory depression
2. decreased sedation
3. better tolerance of chest physiotherapy
4. earlier ambulation
5. increased lung volumes
6. improved arterial oxygenation

the issue of anticoagulation has been settled by the studies of Rao, El-Etr & Odoom et al.
# Aortic Cross Clamping

- factors influencing the haemodynamic response include,
  i. the level of the clamp
  ii. intravascular volume status
  iii. presence of myocardial ischaemia or failure
  iv. the number of collaterals around the point of occlusion
  v. the anaesthetic agents employed
  vi. supplemental agents used prior/during clamping

- physiological problems which may result include,
  i. acute LV strain ± failure or valvular incompetence
  ii. acute LV ischaemia
  iii. ischaemia or hypoperfusion of the kidneys & spinal cord
  iv. accumulation of metabolic byproducts below the level of the clamp

- AoXC produces an immediate rise in all components of aortic ejection impedance

<table>
<thead>
<tr>
<th>Haemodynamic Changes with Aortic Cross-Clamping</th>
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<tbody>
<tr>
<td>Change = $\delta$%</td>
</tr>
<tr>
<td>MAP mmHg</td>
</tr>
<tr>
<td>CO l/min</td>
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<tr>
<td>SV ml/beat/m²</td>
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<tr>
<td>SVR dyne/s/cm⁵</td>
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<tr>
<td>PCWP mmHg</td>
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<tr>
<td>LVEDA</td>
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<tr>
<td>LVESA</td>
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<td>RWMA's (new)</td>
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- the final LVEDV is determined by relative prominence of the decrease in venous return and the increase in LVESV
- in patients with adequate myocardial function, PAoP is usually unchanged or slightly decreased following infrarenal AoXC
- patients appear to fall into 2 distinct groups,
  1. those with good myocardial reserve who tolerate cross clamping well, and
  2. those who decompensate with large decreases in CO and SV, and acute increased PAoP with arrhythmias and/or ECG signs of myocardial ischaemia

- these manifestations are more common following suprarenal or supraceliac clamping
therapy for haemodynamic alterations following AoXC may include,

1. **hypertension alone**  
   - BP > 170/100 mmHg  
   - i. deepen anaesthesia - increase volatile agent  
     - IV supplement (propofol, droperidol, opioid)  
   - ii. vasodilatation - nitroprusside  
   - iii. antihypertensives - esmolol, atenolol, labetalol

2. **LV strain / failure**  
   - ↑↑ PAoP  
   - i. nitroglycerine ~ 0.25-0.75 µg/kg/min  
   - ii. dobutamine ~ 2-8 µg/kg/min  
   - iii. ? amrimone/milrinone

3. **myocardial ischaemia**  
   - ↓ ST ± ↑ PAoP  
   - i. nitroglycerine ~ 0.25-0.75 µg/kg/min  
   - ii. verapamil ~ 2.5 mg boluses (to 10 mg)

- in the presence of failure/ischaemia **GTN** is preferable as it preserves the normal transmural distribution of myocardial blood flow (favouring the subendocardium)
- further, some studies have shown that GTN infusion prevents the progressive decline in LV function and rise in SVR which normally follows AoXC
- when administered to reduce PAoP to pre-clamp levels, SVR usually decreases and CO is raised

- studies have shown that **SNP** may redistribute blood flow away from the subendocardium
- in the group with good LV function, the decrease in CO seen in the absence of significant changes in PAoP or ECG, actually represents a physiological compensation for the reduction in total VO\(_2\)
- administration of vasodilators to this group simply increases SvO\(_2\)

- retroperitoneal dissection, surgical handling and AoXC decrease renal cortical blood flow, GFR and urine output
- AoXC stimulates the release of **renin**, with PRA and aldosterone increased postoperatively
- the exact mechanism of these changes with **infrarenal** clamping is unknown
- obviously there is a period of total renal hypoperfusion associated with suprarenal clamping
- the reduction in RBF & GFR with infrarenal clamping **is not** prevented by epidural anaesthesia with sensory block to T\(_6\) (?? actual level of sympathectomy)
- studies have shown that the degree of intraoperative oliguria does not correlate with postoperative acute renal failure
- however, ARF is unlikely to develop if urine output is maintained > 60 ml/hr
- many anaesthetists administer mannitol 12.5-50g prior to AoXC to induce a brisk diuresis
- mannitol also produces renal vasodilatation and can reverse, or decrease, the decrease in cortical RBF seen with clamping
- further, it has been shown to prevent experimental animal ARF following temporary ischaemia
- the absolute incidence of ARF following aortic surgery has decreased, part of this change being attributed to the widespread use of mannitol (?? 2 ° to volume expansion)
other "corrective" measures used to prevent ARF include frusemide and low-dose dopamine
the efficacy of these agents has been questioned and their place disputed
for dopamine quote review article by G. Duke & A. Bersten (AIC 1992),

a. potential renal benefits
i. increased $O_2$ delivery via a modest rise in CO, and usually a rise in RBF
ii. a potential decrease in renal $VO_2$ through inhibition of Na$^+$ reabsorption

b. potential detrimental effects
i. impairs TGF mechanism which may adversely affect $O_2$ supply/demand
ii. the diuresis is not always associated with an increase in RBF
iii. the diuresis may mask hypovolaemia & renal hypoperfusion
iv. an inappropriate diuresis may result in hypovolaemia

NB: 1. should we be using a diuretic in a potentially hypovolaemic patient
2. similar augmentation of RBF can be achieved with other catechols which do not affect tubular function
3. dopamine produces potentially unwanted cardiorespiratory side-effects

i. tachyarrhythmias
ii. depressed respiratory drive - hypoxic & central
iii. increase intrapulmonary shunt
iv. decreased $P_{aO_2}$
v. increased PAoP
vi. increased LV & RV afterload

Cunningham states, "combinations of mannitol, dopamine and frusemide have been advocated to prevent renal dysfunction during aortic vascular surgery...however, much of the data supporting the proposed benefit of these measures come from animal experiments in which deliberate extracellular fluid expansion was not employed."

he suggests crystalloid infusion preoperatively, to rectify the ECF deficit, & prompt replacement of intraoperative losses, as guided by PAoP, are most likely to be beneficial in renal salvage

rarely AoXC results in ischaemic damage to the spinal cord ~ 0.25% (1:400)
incidence is ~ 10x higher for acute rupture cf. elective cases
minimal incidence with surgery for aortoiliac occlusive disease
produces complete flaccid paralysis & dissociated sensory loss
occurs 2° to interruption of blood flow through,
  i. infrarenal radicular branches
  ii. a low thoracic, or high lumbar radicular artery

NB: in the presence of an anomalous take-off of the radicularis magna

the risk of this is probably increased for any given patient by,
  i. high aortic clamping, and
  ii. prolonged intraoperative hypotension
the inferior mesenteric artery, the primary arterial supply to the descending and sigmoid colon is frequently sacrificed.

- collateral flow from the mid-colic branch of the superior mesenteric artery and the superior haemorrhoidal arteries is usually sufficient.
- the true incidence of ischaemia colitis may be as high as 6%.
- there is also an association with stress ulceration of the stomach, and routine prophylaxis should be employed.

- near total ischaemia of the lower limbs results in the accumulation of metabolic by-products which are released into the circulation following declamping.
- this is accompanied by intense vasodilatation & reactive hyperaemia.

Aortic Declamping

- while SVR and MAP invariably decrease, CO may increase or decrease.
- the change in CO is dependent upon preload which declines with declamping 2° to reactive hyperaemia in the lower extremities.
- the combination of decreased preload and CO, and the increase in blood flow to the lower limbs creates a peripheral vascular steal syndrome.
- this is accompanied by marked hypotension and hypoperfusion of the coronary, mesenteric and renal vascular beds, described in the past as "declamping shock".

- these effects are largely preventable by adequate fluid loading.
- measurement of PAoP and thermodilution CO allows titration of fluid therapy to maximise CO.

NB: in the absence of time to perform serial CO measurements, elevation of the PAoP ~ 4-6 mmHg above baseline values is usually adequate.
**Fluid Therapy**

- Potential for large losses of blood and functional ECF
- Plus large losses to 3rd space compartments, mainly retroperitoneal and bowel lumen
- These changes may occur in patients on chronic diuretic therapy, or with chronic hypertension and a contracted intravascular volume
- Investigations have shown that CVP **does not** correlate with measured blood volume during major vascular surgery
- Therefore, RMD states preferable to use PAoP, urine output and serial Hct
- However, in the same chapter states that TEE shows that PAoP **does not** correlate with LVEDA
- The age old argument of which is better, crystalloid or colloid remains
- The crystalloidsists argue that due to the large loss of functional ECF, replacement with predominantly salt solution restores a more physiological state
- Irrespective of fluid choice, the PAoP should be kept near-baseline ~ 10-15 mmHg
- Immediately prior to declamping fluid loading to elevate PAoP above baseline ~ 4-6 mmHg
- If urine output is < 40 ml/hr then a fluid challenge should be given regardless of the PAoP
- Once LV preload is maximised, if urine output is still low then mannitol may be beneficial
- RDM advocates also trying frusemide / dopamine but these are of questioned benefit
- RDM states blood should be given once operative losses exceed **20%** calculated blood volume
- Assuming a normal preoperative Hct. and adequate volume replacement, this would lower the Hct to ~ 30-35%, and further losses may reduce DO$_2$
- In the average sized adult, 1 unit of blood will raise the Hb ~ 1g/l and the Hct ~ 3%
- All infused fluids should be **warmed** to prevent excessive hypothermia
- The volume required to cause problems of **massive transfusion** varies, but generally requires > 15-20 units in adults
- Methods to reduce the use of homologous blood include,
  1. Toleration of a lower Hct.
  2. Multiple autologous predeposit units
  3. Immediate preoperative phlebotomy, haemodilution & autologous transfusion
  4. Intraoperative red cell salvage & transfusion
- The use of red cell salvage has a number of advantages,
  1. No requirement for cross-matching, with risk of errors or reactions
  2. Warm, fresh red cells
  3. Normal pH and 2,3-DPG content
  4. Longer circulatory viability
- Recent studies have shown up to **50-80%** reduction in homologous transfusion
Summary Guidelines    Abdominal Aortic Surgery

1. **preoperative assessment**
   - concurrent disease

2. **preoperative hydration**
   - maintenance fluids overnight
   - crystalloid preload on the morning of surgery

3. **premedication**
   - BZD ± opioid

4. **monitoring**
   - PAoP vs CVP, ?? TEE
   - lead configuration for ECG

5. **general anaesthesia**
   i. **induction**
      - pre-O2
      - low dose opioid + STP / propofol
      - high dose opioid
      - non-depolarising relaxant
   ii. **intubation**
      - when TOF = 0
      - supplemental agents
   iii. **maintenance**
      - O3 ± N2O
      - supplemental volatile agent
      - supplemental opioid ± infusion
      - ± regional anaesthesia
   iv. **ventilation**
      - controlled normocapnia
   v. **vasodilatation**
      - GTN ~ 1-2 µg/kg/min
      - myocardial ischaemia
      - hypertension > 20% above baseline

6. **intraoperative fluid management**
   i. **crystalloid ± colloid**
      → PAoP ~ 10-15 mmHg
      U/Output > 60 ml/hr
   ii. **blood**
      - losses > 20% estimated blood volume
   iii. **mannitol**
      - PAoP > 15 mmHg & U/Output < 40 ml/hr

7. **postoperative care**
   i. **mechanical ventilation**
      - minimise VO2
      - cardiorespiratory homeostasis
      - sedation
   ii. **pain relief**
      - opioid infusion
      - regional anaesthesia
   iii. **fluid management**
      - PAoP, U/Output
      - CXR, PA-aO2 gradient
      - Hct / Hb and DO2
      - electrolytes
   iv. **temperature homeostasis**
   v. **nutrition**
**Postoperative Care**

1. sedation & pain relief
2. respiratory care
   - primarily a *restrictive deficit*, with decreased FRC and pulmonary compliance
   - factors precluding early extubation include,
     i. prolonged procedure / anaesthesia
     ii. obligatory extended midline incision
     iii. abdominal distension due to fluid sequestration
     iv. bedrest in the supine position
     v. abdominal pain requiring moderate-large doses of opioids
     vi. hypothermia
3. cardiovascular homeostasis
   i. graft patency - s/c heparin for all after 12 hours
   ii. hypertension - pain
      - intraoperative overhydration
      - postoperative hypothermia, hypercarbia, hypoxia
      - rebound following vasodilators
      - preexisting hypertension & vascular hyper-reactivity
   iii. postoperative increases in HR ~ 25-50% are common
   iv. ischaemia is typically *silent*
4. fluid and electrolyte therapy
   i. basal requirements
   ii. preservation of renal function
   iii. nutrition
5. gastrointestinal care
   i. obligatory ileus - N/G tube for all
   ii. ulcer prophylaxis
   iii. antibiotic prophylaxis for wound infection
6. temperature homeostasis
   - forced air convective heating if available
   - "space" blankets
7. regional anaesthesia
   - reduction in volatile / opioid requirements intraoperatively
   - better postoperative pain relief
   - better respiratory function, shorter time to extubation
   - fewer cardiovascular complications postoperatively
   - ?? greater administration of perioperative fluids & decreased LV function cf. general anaesthesia alone (1 study only)
Emergency Aortic Surgery

NB: mortality is directly related to the time interval to control of the proximal aorta, therefore, patients should be transported immediately to theatre and resuscitated.

- immediate preparations should include,
  1. supplemental O₂
  2. placement of at least 2 large bore IV cannulae
  3. obtain blood for G&M, Hct., FBE, MBA
     - G&M 10-12 units - 5 unmatched, type-specific sent to theatre
  4. commence volume replacement sufficient to maintain BP ~ 80-100/? mmHg
     - higher pressures are unwarranted and may result in complete rupture
  5. monitoring pre-induction
     i. ECG, NIBP or IABP, SpO₂
     ii. defer placement of urinary catheter & N/G tube until after induction
        - risk of induced Valsalva resulting in rupture
  6. pre-O₂, prepare & drape patient prior to induction
  7. induce with surgeon ready to start
     i. high dose narcotic - incremental technique
     ii. ketamine
     iii. relaxant (SCh) & intubation - RSI
  8. maintenance supplement according to haemodynamic stability
     i. maintain muscle relaxation
     ii. intermittent opioid / infusion
     iii. N₂O / O₂
     iv. volatile supplement
  9. monitoring post-induction
     i. ECG, IABP & NIBP, CVP ± PA catheter
     ii. SpO₂, ETCO₂, spirometry
     iii. serial Hct & AGA’s
     iv. oesophageal stethoscope & temperature probe
     v. urinary catheter
     vi. N/G tube
  10. postoperative care
     i. delayed extubation
     ii. maintain haemodynamic status & renal function
     iii. pain relief

NB: §§prior to the establishment of haemodynamic stability, volume replacement should take priority over the placement of "lines"
Descending Thoracic Surgery

i. coarctation
ii. dissection - DeBakey type III, type B
iii. aneurysm
iv. traumatic disruption or laceration

- The principal causes of death in a large series were,
  i. haemorrhage ~ 29%
  ii. cardiac ~ 26%
  iii. MOSF ~ 22%

- Most common approach is a left transverse thoracotomy through the 4-5th ribs
- AoXC is usually applied just distal to the left subclavian artery, and a 2nd clamp placed to prevent back-bleeding
- Major decision is the method of LV unloading to be used,
  1. left heart bypass
  2. heparin coated shunts - no requirement for systemic heparinisation
  3. pharmacological

- Optimum anaesthetic management requires,
  1. anticipation of major blood loss & preparation for replacement
  2. intensive haemodynamic monitoring
  3. one lung ventilation
  4. intense vasodilator therapy for AoXC application
  5. correction of the metabolic derangements 2o to temporary tissue ischaemia

- Induction is usually achieved with an incremental high dose opioid technique
- Despite evidence that the volatile agents inhibit hypoxic pulmonary vasoconstriction, none of these agents significantly interfere with oxygenation during clinical anaesthesia
- The use of 1LV not only allows surgical access and prevents retractor trauma but also prevents spillage of blood from the upper lung during dissection of an adherent aneurysm
- Most would insert a left sided DLT, but insertion may be difficult 2o compression of the left mainstem bronchus by the aneurysm
- Monitoring, in addition to the usual for major cases,
  1. PA catheter for virtually all cases ? TEE for acute dilatation & MR
  2. stethoscope for the dependent right lung
 & SpO₂, ETCO₂, Pₐw *detection of malposition of the DLT
  3. right radial IABP ± femoral artery IABP
  4. ?? SSEP's for spinal cord function
AoX clamping results in acute LV strain, a fall in SV & CO and marked hypertension
untreated this may result in,

1. acute dilatation & LV failure
2. mitral valve incompetence
3. florid pulmonary oedema & desaturation
4. breakthrough of cerebral autoregulation
   → raised ICP & cerebral oedema
5. VF & death

blood flow distal to the clamp, kidneys & spinal cord, is reduced ~ 85-94%
the risk of irreversible cord ischaemia increases dramatically with clamp times > 30 minutes
mechanical unloading of the LV does not decrease the incidence of cord ischaemia
therefore many surgeons forego placement of a shunt or establishment of bypass and rely upon
short clamp times and pharmacological decompression of the LV

NB: any of the vasodilators may be used, though some have questioned the use of
arteriolar dilators in the presence of decreased distal aortic pressure & perfusion

β-blockade is useful for treating any tachycardia
renal "preservation" is claimed with the use of mannitol or frusemide prior to clamping
due to the high risk of cord ischaemia, some have tried to correlate SSEP patterns with the
requirement for shunt placement
however, other studies have shown no correlation between SSEP's and cord outcome

AoX declamping results in a significant rise in SV/CO but a fall in preload
there is usually a significant metabolic acidosis and partial correction with HCO₃⁻ is warranted to
prevent hyperkalaemia and arrhythmias,

Dose ~ (0.3 x Wt x BE)/2

Cardiovascular Anaesthesia
ISCHAEMIC HEART DISEASE

- **Identification of Patients**
  - any of the following may indicate the presence of IHD,
    - a history of "vise-like" chest pain ± radiation to the neck or arm
    - dyspnoea on exertion
    - dyspnoea on exposure to cold, after eating, or after defecation
    - paroxysmal nocturnal dyspnoea
    - orthopnoea
    - past or present, peripheral or pulmonary oedema
    - a history of MI
    - a family history of IHD/MI
    - diagnosis of MI by ECG or cardiac enzymes
    - diagnosis of IHD by ECG, stress testing or Holter monitor
    - cardiomegaly
  - other patients who should be suspected of having IHD include,
    1. diabetics
    2. patients with hypertension
    3. patients with hyperlipidaemia (especially smokers)
    4. patients with PVD, or carotid bruits
    5. patients with unexpected dysrhythmias or evidence of CCF
    6. heavy smokers
  - the principal problem is to determine IHD in asymptomatic individuals, or in those with predisposing factors and a normal ECG
  - of the above factors, *history* is the best indicator, most series
    → *sensitivity* in predicting IHD ~ 80% to 91%
  - Hertzer *et al.* found in 1001 vascular surgical patients,
    1. of the 500 with *normal ECGs* → 37% ≥ 70% narrowing ≥ 1 coronary artery
    2. those suspected of IHD → 78% had significant vessel narrowing
      • either a suggestive *history*, or an *abnormal ECG*
    3. up to 15% of patients with *triple vessel disease* have a normal resting ECG
    4. the presence of a *carotid bruit* is highly suggestive,
      perioperative mortality being 15-17%, cf. 2.1% in a control group
ACUTE MYOCARDIAL INFARCTION

- **Incidence**
  1. males ~ 3.5 : 1000
  2. females ~ 1.0 : 1000

  NB: ↑ ~ 5 fold with 2 major risk factors
  ↑ ~ 8 fold with 3 major risk factors

- **Aetiology**
  1. **atherosclerosis** ~ 99%
  2. embolism - thrombus, air
  3. coronary arteritis - polyarteritis nodosa, Kawasaki dis.
  4. congenital coronary anomalies - eg. LCA from PA
  5. myocardial hypertrophy & aortic stenosis
  6. aortic dissection 2° - aortitis, syphilis, Marfan's, trauma
  7. severe trauma
  8. electrocution
  9. severe hyperthermic syndromes
  10. prolonged cardiopulmonary bypass
  11. prolonged hypotension/hypovolaemia

- **Predisposing Factors**
  1. **hypertension***
  2. **hyperlipidaemia*** - high cholesterol/HDL ratio
  3. **smoking*** - ↑ COHb
  - vasoconstriction≈ nicotine
  - accelerated atherosclerosis
  - ↑ lipids and platelet adhesiveness
  - ↑ incidence of sudden death and MI
  4. gender - males > females
  5. increasing age
  6. diabetes mellitus
  7. minor risk factors include
    1. family history
    2. obesity
    3. lifestyle factors - lack of routine exercise, diet
    - ?? psychosocial factors
- **Aggravating Factors**
  a. chronic lung disease
  b. anaemia
  c. surgery, trauma, systemic or respiratory infection
  d. thyroid disease
  e. pulmonary embolism
  f. severe illness, infection

- **Clinical Presentation**
  a. silent AMI ~ 25% in Framingham study
  b. chest pain
  c. atypical pain
  d. syncope / arrhythmias
  e. LV failure / acute pulmonary oedema
  f. peripheral emboli from mural thrombus
  g. hypotension / cardiogenic shock
  h. sudden death ~ 25% of sudden deaths at PM due to acute MI
     - AMI or sudden death → 1st presentation of CAD in ≥ 50%
     - vast majority 2° to VF

- **Time Course of Infarction**

  NB: irreversible myocardial necrosis occurs ~ 60 minutes after "no flow"
  *coronary thrombosis* is demonstrated in ≥ 90% of acute MI

  i. EM changes ~ 15 min
  ii. light microscope changes ~ 6 hrs
  iii. macroscopic changes ~ 24 hrs
  iv. commencement of healing ~ 2 wks
  v. fibrotic scar ~ 6 wks

- **Anatomical Relationships**
  i. RCA - inferior & posterior
     - SA & AV nodes
  ii. LCA - anterior & septum
  iii. circumflex - anterolateral
Diagnosis

a. history and examination - most important
b. ECG
   - ST elevation
   - T wave inversion
   - pathological Q waves
   - LBBB
c. CXR
   - best indicator of degree of LVF
   - not helpful in early diagnosis
d. cardiac enzymes
   i. CK (MB)
      - ↑ 8-24 hrs
      - ↓ 48-72 hrs *except large MI's or delayed excretion
   ii. LDH (LDH₁)
      - ↑ 24-48 hrs
      - ↓ 7-14 days (LDH "flip")
e. echocardiography
   - regional wall motion abnormalities
   - papillary muscle dysfunction, valvular dysfunction
   - ejection fraction
   - pericardial effusions
f. radioisotope scans
   - $^{99m}$Tc hot spots at 1-10 days
   - $^{201}$Th cold spots
g. gated blood pool scan
   - regional wall motion abnormalities
   - papillary muscle dysfunction
   - ejection fraction
h. coronary angiography
   - usually in assessment for CABG
i. non-specific changes
   - rise in BSL
   - rise in WCC (15-20,000/µl)
   - rise in urea & myoglobin

Prognosis - Death

a. prior to hospital ~ 25%
b. within 1 month ~ 10-15%
c. within 1 year ~ 10%
d. each subsequent year ~ 3-5%

NB: not proven to be altered by CCU/ICU's,
prior to the introduction of thrombolytic therapy
LV function is the most powerful predictor of survival
Treatment - Aims

1. relief of symptoms
2. limitation of *infarct size*
3. prevention of *reinfarction*
4. detection and treatment of *complications*
   i. *arrhythmias* - responsible for ~ 40% of post-MI deaths
   ii. CCF - acute pulmonary oedema, hypoxaemia
      - acidaemia, hypoperfusion
   iii. CVA
   iv. cardiac rupture or septal perforation
   v. acute valvular dysfunction
   vi. ventricular aneurysm
   vii. Dressler's syndrome - pericarditis, friction rub, fever ± pneumonitis
      - rare, occurring at weeks to months
5. rehabilitation

Treatment - General

a. education, explanation and reassurance
b. bed rest
c. analgesia
d. supplemental O₂
e. continuous ECG monitoring in CCU for ≥ 48/24
f. *arrhythmia prophylaxis*
   • was recommended by the AHA but *not proven* to decrease the incidence of VF
   • some studies have actually shown decreased survival in lignocaine group
   • now no longer recommended by AHA
g. *anticoagulants*
   i. *low dose heparin* in all patients
      • ↑ survival in unstable angina ~ 50% decrease death & non-fatal MI
   ii. prevention of systemic emboli
      - large anterior infarcts
      - CKMB ≥ 160
      - CPK ≥ 8 times normal
      - presence of AF or ventricular aneurysm
   iii. following thrombolytic therapy
   iv. *warfarin* is better than heparin
Myocardial Salvage

1. Prevention of CAD
2. Limitation of Infarct Size
3. Prevention of Reinfarction

- **Prevention of CAD**
  1. education
  2. treatment/elimination of risk factors
     - risk modification is of benefit *after* the development of CAD
     - Helsinki heart study 1988, 4081 asymptomatic dyslipaemic patients,
       10% reduction in cholesterol → 34% decrease in CAD over 5 years
     - control of hypertension decreases overall mortality (CVA),
       but *does not* alter the incidence of CAD except with β-blockers

- **Limitation of Infarct Size**
  i. streptokinase / TPA  - 50-70% patency
    - 20-25% reduction in early mortality
  ii. GTN
    - reduction in post-infarct angina
    - reduction in CKMB rise
    - may reduce infarct size
    - ?? effect on outcome
  iii. early IV β-blockers
    - small benefit in large AMI
  iv. monitoring/prevention of complications  (?? benefit)

- **Prevention of Reinfarction**
  a. β-blockers
    - 25% reduction in reinfarction & late mortality
    - ISIS I (1986)
    - 15% ↓ mortality with IV β-blockers in *addition* to oral
  b. antiplatelet agents
    - ISIS II, aspirin 100-150 mg/d
    - reduction in mortality at 1 month
  c. warfarin
    - 25% reduction in re-thrombosis following thrombolysis/angioplasty
  d. ACE inhibitors
    - improved survival with LVEF < 45%
  e. Ca⁺⁺-entry blockers
    - no proven benefit
    - oral diltiazem in non-Q-wave infarction
  f. coronary angioplasty
    - 90-95% success in "appropriately selected" patients
    - 33% recurrence in first 3-6 months
  g. CABG
    - LAD or triple vessel disease & depressed LV function
**CASS Study 1983**

- surgically treated patients subjectively better
- no improved survival with,
  1. mild angina
  2. 2 or 3 vessel disease with normal LV function

- patients with severe angina (CHA III or IV) and 2 vessel disease with depressed LV function "probably" benefit in terms of improved prognosis
- improved long-term patency has been demonstrated with *internal mammary arterial conduits*
cf. saphenous vein bypass grafts

  **NB:** improved survival has *only* been demonstrated with,
  1. *left main disease*, or
  2. *triple vessel disease* with depressed LV function

**ISIS II 1988**

- 17,187 patients with suspected MI, within 24 hours on onset of symptoms
- randomised into 4 groups receiving,
  1. oral aspirin ~ 20% ↓ mortality
  2. streptokinase ~ 23% ↓ mortality
  3. streptokinase + aspirin ~ 42% ↓ mortality
  4. neither

  **NB:** the increase in incidence of reinfarction with *streptokinase alone*, possibly due to streptokinase enhancement of *platelet activation* with release of TXA$_2$
Thrombolytic Therapy

a. types
   - streptokinase
   - tPA
   - urokinase
   - ASPAC (anisoylated streptokinase-plasminogen activator complex)

b. indications
   i. clinical & ECG evidence of infarction
   ii. age < 70 yrs
   iii. onset < 4 hrs
   iv. no allergy to streptokinase
   v. no high risk of systemic haemorrhage

c. absolute contraindications
   - allergy to streptokinase
   - streptokinase within 1 year
   - recent streptococcal infection
   - major trauma / surgery within 2 weeks
   - active peptic ulceration within 3 months
   - prolonged CPR
   - systemic coagulopathy
   - pregnancy or post-partum
   - recent CVA ≤ 6 months, or TIA’s

d. relative contraindications
   - age > 70 years
   - recent central venous or arterial puncture
   - risk of systemic emboli (MS, AF, aneurysm)
   - hepatic or renal disease (low clearance)
   - short duration of CPR

e. complications of streptokinase
   - allergy, anaphylaxis
   - febrile reaction
   - haemorrhage
   - reperfusion arrhythmias
   - hypotension, vasodilatation

NB: indications for tPA → indication for streptokinase + allergy
## Comparison of Streptokinase and tPA

<table>
<thead>
<tr>
<th>Factor</th>
<th>Streptokinase</th>
<th>tPA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Artery patency at 2 hours</td>
<td>55%</td>
<td>70%</td>
</tr>
<tr>
<td>Incidence of re-occlusion</td>
<td>15%</td>
<td>20%</td>
</tr>
<tr>
<td>Reduction in mortality + <em>aspirin</em></td>
<td>20%</td>
<td>26%</td>
</tr>
<tr>
<td></td>
<td>51%</td>
<td>?</td>
</tr>
<tr>
<td>Cost</td>
<td>$140</td>
<td>$1,350</td>
</tr>
<tr>
<td>Hypotension</td>
<td>20%</td>
<td></td>
</tr>
<tr>
<td>Anaphylaxis</td>
<td>0.1%</td>
<td></td>
</tr>
<tr>
<td>Fall in fibrinogen</td>
<td>80%</td>
<td>30%</td>
</tr>
<tr>
<td>Cerebral haemorrhage</td>
<td>0.4%</td>
<td>0.5%</td>
</tr>
<tr>
<td>Major haemorrhage</td>
<td>0.6%</td>
<td>4%</td>
</tr>
<tr>
<td>Plasma half-life</td>
<td>23 min</td>
<td>9 min</td>
</tr>
</tbody>
</table>
PERIOPERATIVE MYOCARDIAL REINFARCTION

Goldman (1977)

→ 1001 patients over 40 yrs
surgery = LA, endoscopies, TURP excluded
multivariate discriminant analysis

- **Independent Variables**
  1. S₃ or elevated JVP
  2. AMI in last 6 months
  3. VEB's > 5/min
  4. rhythm other than sinus
  5. aortic stenosis
  6. major abdominal or thoracic procedure
  7. emergency operation
  8. age > 70 yrs
  9. poor general medical condition

- **Insignificant Variables**
  1. smoking
  2. hyperlipidaemia
  3. diabetes
  4. hypertension
  5. PVD
  6. stable angina, ST/T wave changes
  7. old MI > 6 months
  8. RBBB
  9. cardiomegaly
  10. mitral valve disease
  11. controlled CCF
Tarhan (1972)

→ 32,877 patients over 30 yrs at the Mayo Clinic
   422 with previous MI

■ Reinfarction Rate

  a. < 3 months ~ 37%
  b. 3-6 months ~ 16%
  c. > 6 months ~ 4-5%

  NB: most occurred on the 3\textsuperscript{rd} day postoperatively → mortality ~ 54%

Mahar, Steen & Tinker (1978)

→ 148 patients
   226 non-cardiac surgical procedures
   99 with previous CABG

  a. none of the CABG group had an MI
  b. 5% of 49 without prior CABG had an AMI ~ 10.2%
  c. all in AMI group had triple vessel disease

Steen, Tinker & Tarhan (1978 - also at the Mayo Clinic)

→ review of Mayo Clinic practice, comparison to Tarhan's original study
   587 operations 1974-75, all patients with previous AMI
   6.1% reinfarction rate → mortality ~ 69%

  a. < 3 months ~ 27%
  b. 3-6 months ~ 11%
  c. > 6 months ~ 4-5%

■ Other Risk Factors

  a. preoperative hypertension
  b. intraoperative hypotension
  c. thoracic and upper abdominal operations > 3 hrs duration
  d. striking correlation between duration of anaesthesia and reinfarction in all groups
Factors Unrelated to Reinfarction

1. postoperative ICU care
2. diabetes
3. angina
4. age or sex
5. site of the previous MI

Rao, El Etr (Anesth. 1983)

<table>
<thead>
<tr>
<th>Interval</th>
<th>Control Group 1</th>
<th>Prospective Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=364 / 1973-76)</td>
<td>(n=733 / 1977-82)</td>
</tr>
<tr>
<td>&lt; 3 months</td>
<td>36%</td>
<td>5.8%</td>
</tr>
<tr>
<td>3-6 months</td>
<td>26%</td>
<td>2.3%</td>
</tr>
<tr>
<td>&gt; 6 months</td>
<td>~ 5% p.a.</td>
<td>~ 1.5-1.7% p.a.</td>
</tr>
</tbody>
</table>

1. NB: retrospective control group
2. intensive therapy long ceased after 6 months, therefore reinfarction rate should have returned to control rates ~ 4-5% p.a.

• other factors associated with a higher reinfarction rate in both groups,
  i. CCF
  ii. intraoperative hypertension & tachycardia
  iii. intraoperative hypotension

NB: "results suggest that preoperative optimisation of the patient's status, aggressive invasive monitoring of the haemodynamic status, and prompt treatment of any haemodynamic aberration may be associated with decreased perioperative morbidity and mortality in patients with previous myocardial infarction"

• however, Slogoff states, (ASA Lectures 1992)
  1. the original abstract was not peer reviewed, and these claims were subsequently withdrawn in their own peer reviewed article
  2. no other group using intensive postoperative management have been able to approach these figures (including the late figures, making the initial claim suspect)
  3. still quoted by various groups to "support their own opinion", ie. regarding use of PA catheters
Hertzer et al. (1984)

→ 1001 vascular surgical patients scheduled to undergo elective surgery
1. coronary angiography revealed significant CAD in ~ 60%
2. those suspected of IHD → 78% had significant vessel narrowing
3. 500 with normal ECGs → 37% ≥ 70% narrowing ≥ 1 coronary artery
4. up to 15% of patients with triple vessel disease have a normal resting ECG
5. the presence of a carotid bruit is highly suggestive → perioperative mortality being 15-17%, cf. 2.1% in a control group
6. CABG thought indicated in 251,
   i. 216 underwent CABG
      • 12 (5.5%) operative deaths during CABG
      • ? 200 peripheral arterial surgery ~ 1.5% early cardiac deaths
         ~ 12% late cardiac deaths
   ii. 35 without CABG
      • 16 peripheral arterial surgery ~ 12% early cardiac deaths

NB: does not answer question of whether CABG should occur before PVD surgery

Slogoff, Keats (1985)

→ 1023 elective CABG patients
a. ECG ischaemia in ~ 37%, half of these pre-induction
b. postoperative AMI in,
   i. 6.9% with perioperative ischaemia → 3x ↑
   ii. 2.5% without perioperative ischaemia
      • but was independent of when the ischaemia occurred
   c. ischaemia related to tachycardia * not hypo/hypertension
   d. ischaemia occurred frequently in the absence of haemodynamic changes
      • probably due to fluctuations in coronary vascular tone ? spasm

■ Other Associated Factors

a. "Anaesthetist No.7"
b. poor quality anastomosis
c. prolonged ischaemic time

NB: unrelated to patient type, LAD lesion, or EF
therefore, the frequency will relate primarily to perioperative management, rather than patient selection
Foster (1986)

- Coronary Artery Surgery Study (CASS) registry data of 1600 patients undergoing major noncardiac operations between 1978-81, showed an operative mortality of,
  1. controls ~ 0.5%
  2. CAD + CABG ~ 0.9%
  3. CAD ~ 2.4% (p < 0.009)

- however, no differences were noticed between the groups for AMI ?

- supports the use of CABG in patients with significant CAD prior to undergoing major non-cardiac surgery, especially with the following risk factors,
  1. high LV "score"
  2. diabetes
  3. LVH
  4. use of nitrates
  5. males
  6. exertional dyspnoea

Knight, Hollenburg & London et al. (1988)

- incidence of haemodynamically unrelated intraoperative ischaemia is identical to that experienced by the patient in the 2 days preoperatively

**NB:** the risk of intraoperative ischaemia, and therefore postoperative MI, is determined primarily by the patients native disease severity, not by perioperative anaesthetic management
in a review of the literature, suggest that the established data is *inaccurate* for the following reasons,

1. patients have been stratified according to *time* from infarction & *operation type*
2. none of the patient groups were homogenous with regard to the *extent of CAD* and the risk for subsequent infarction
3. no distinction was made between "Q-wave" and "non-Q-wave" infarction*
   i. recent data suggests that survivors of a "non-Q-wave" MI, are at greater risk of a subsequent MI
   ii. although "Q-wave" infarcts are at a lower risk of MI, they are still prone to arrhythmias
4. most of the published data is prior to the widespread use of *thrombolytic therapy*

although distinction between "Q-wave" and "non-Q-wave" infarction* may be relevant, it is important to remember that,

a. ECG classification as such *does not* necessarily correlate with transmural and subendocardial infarction

b. there is significant overlap between these groups, especially with the use of thrombolytic therapy

they suggest a more appropriate approach is *symptom limited exercise testing*, based upon whether the person is about to undergo high, or low risk non-cardiac surgery

this, or cardiac catheterisation, is recommended by the AHA for virtually all patients within 6-8 weeks following a MI

*NB:* irrespective of infarct type, within the first *6 weeks* there will be remodelling and fibrosis, and the myocardium is sensitive to any additional stresses

their approach is therefore limited to "recent infarction", ie. 6 weeks to 6 months

the choice of which test is performed initially depending upon the nature of the patients disease and the extent of the planned surgery
Assessment of Myocardial Reserve

- **Exercise Electrocardiography**
  
  a. patients able to achieve exercise heart rates up to **85%** of predicted maximum
  b. **upsloping** ST-segment depression > 2mm at 0.08s from the J-point
  c. **horizontal** ST-segment depression > 1mm at 0.06s from the J-point
  d. **downsloping** ST-segment depression > 1mm at 0.06s from the J-point
    i. increased mortality cf. upsloping or horizontal changes
    ii. associated with an increased number of diseased vessels
    iii. > 1mm represents severe **transmural ischaemia**
  e. **elevated** ST-segment > 1mm at 0.06s from the J-point
    - in the absence of haemodynamic or rhythm disturbance suggests coronary artery spasm (Prinzmetal's angina)
  
  - a **positive result** represents a high risk, however, may be misleading as,
    a. ischaemia may not occur at the same BP & HR as it would in normal daily life
    b. exercise produces tachycardia with little δBP,
    whereas **anaesthesia** may associated with both a **rate & pressure load**
    c. most ischaemia occurring perioperatively **is not** associated with alteration of haemodynamic variables
    d. ambulatory ECG data shows that individuals suffer ischaemia at different (lower) HR/BP levels to those occurring during exercise
    e. the **critical HR** for the development of ischaemia displays **circadian variation**
      - lowest in the early morning, ∴ morning operations are worse

- **Ambulatory Electrocardiography**
  
  - **silent ischaemia** accounts for at least **75%** of all ischaemic episodes (? higher in diabetics)
  - this correlates with a worse **prognosis**, both in terms of **adverse cardiac outcome & mortality** in,
    1. non-cardiac surgical patients with CAD
    2. patients post-AMI
    3. following CABG surgery

  **NB:** the absence of angina **is not** a reliable indicator of the stability of a patient's CAD, further, angina is not a reliable indicator of myocardial ischaemia


**Exercise Thallium Imaging**

- $^{201}$Th is an analogue of potassium and is actively taken up into the myocardium
- better able to determine the extent and location of the myocardium at risk cf. exercise ECG
- discrimination of fixed versus reversible thallium defects distinguishes between scarred and ischaemic myocardium

- **dipyridamole-thallium scanning** is highly sensitive in predicting perioperative myocardial ischaemia in patients unable to exercise
  
  a. dipyridamole vasodilatation of normal vessels preferentially distributes flow away from an ischaemic area, which appears as a "cold spot"
  
  b. as the vasodilatory effects subside, flow redistributes with reappearance of the ischaemic area, "fixed" defects traditionally thought to represent scar tissue
  
  c. more recent work has shown that fixed defects on standard delayed imaging may occur in the presence of viable myocardium and critical stenosis, being termed **hibernating myocardium**
  
  d. the presence of a redistribution defect is predictive of a postoperative cardiac event, in patients undergoing peripheral vascular surgery
  
  e. the overall sensitivity of DPT scanning is comparable to exercise-thallium scanning

**NB:** in patients with **severe 3 vessel disease** under rare circumstances the DPT scan may appear "normal", as there are no "normal" areas to provide contrast in $^{201}$Th uptake

- other scanning methods presently being evaluated include,
  
  a. stress simulation thallium scanning using adenosine instead of dipyridamole
  
  b. newer $^{99}$Tc isotopes in conjunction with PET

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Positive Predictive Value</th>
<th>Negative Predictive Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise ECG</td>
<td>30.6</td>
<td>83.2</td>
<td>30.6</td>
<td>83.2</td>
</tr>
<tr>
<td>Ambulatory ECG</td>
<td>43.4</td>
<td>86.3</td>
<td>35</td>
<td>89.9</td>
</tr>
<tr>
<td>Dipyridamole $^{201}$Th</td>
<td><strong>83.5</strong></td>
<td>68</td>
<td>37.9</td>
<td><strong>94.7</strong></td>
</tr>
<tr>
<td>DPT - Cunningham</td>
<td>85-93%</td>
<td>64-80%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Mazer-CD: The diagnosis and perioperative management of myocardial ischaemia. Can J Anaes. 1992: 39 (5); R90-R95
Eagle et al. demonstrated that in patients with ≥ 3 clinical risk factors,

1. angina
2. age > 70 years
3. diabetes
4. Q-waves on ECG
5. ventricular ectopy requiring treatment

NB: undergoing peripheral vascular surgery had a 50% chance of a perioperative adverse cardiac outcome, ie.

i. cardiac death
ii. AMI
iii. unstable angina
iv. acute pulmonary oedema

NB: irrespective of the above test results, they recommended cardiac catheterisation as the initial test in these patients

Congestive Heart Failure

- current signs of CCF were shown to be predictive of an adverse outcome by Goldman 1977
- the predictive value obtained by objective measurement of LVEF is less certain
- studies using radionuclide imaging LVEF measurements have been both predictive and non-predictive
- baseline resting LVEF is probably only useful in patients with poor or questionable exercise tolerance, or documented CAD
- more important is the functional response to stress, using either
  a. exercise echocardiography
  b. dipyridamole echocardiography
  c. exercise radionuclide ventriculography
  d. diastolic BP during standard exercise ECG (extremely sensitive marker)

NB: these have been shown in various studies to be predictive of,

i. cardiac death
ii. MI
iii. unstable angina
iv. acute pulmonary oedema (Tischler 1991)
Angina

■ **Stable Angina**

- in the original work by Goldman, chronic stable angina was *not* predictive of perioperative cardiac morbidity
- however, NYHA class IV angina was excluded from the study due to the small number
- Shah *et al.* (1990) found that chronic stable angina *was* a predictive factor, and this is now generally accepted
- patients with either,
  1. frequent anginal symptoms, or
  2. poor exercise tolerance

→ almost a 100% positive result to stress ECG testing, therefore this offers little information, and a negative result is usually false

- scintography may provide useful information,
  1. the *extent* and area of myocardium at risk
  2. whether the patient is likely to benefit from revascularisation
  3. baseline LVEF
  4. the coronary anatomy

- however, if neither percutaneous balloon angioplasty nor CABG are options, and the non-cardiac surgery is required, then this information is superfluous

**NB:** preoperative testing of patients with chronic stable angina should only be performed if the results are likely to alter the perioperative care

- however, even in patients with chronic stable angina, ~ 75% of all ischaemic episodes, as defined by ECG, echocardiography, or nuclear imaging occur in the *absence* of symptoms

■ **Unstable Angina**

1. new onset (< 2 months) of severe angina
2. angina at rest or with minimal activity = NYHA & CCS Class IV
3. recent increase in the frequency, or duration of chronic angina
4. recurrent angina within several days of an AMI, without enzyme changes

**NB:** Shah *et al.* (1990) ~ 28% of those undergoing non-cardiac surgery suffer a perioperative MI or cardiac death

clearly this is a prohibitive risk for anything but emergency anaesthesia
Intraoperative ECG Monitoring

<table>
<thead>
<tr>
<th>Lead Placement</th>
<th>Sensitivity (%)&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Sensitivity (%)&lt;sup&gt;2&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>II alone</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>V&lt;sub&gt;5&lt;/sub&gt; alone</td>
<td>75</td>
<td>89</td>
</tr>
<tr>
<td>II + V&lt;sub&gt;5&lt;/sub&gt;</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>II + V&lt;sub&gt;4&lt;/sub&gt; + V&lt;sub&gt;5&lt;/sub&gt;</td>
<td>96</td>
<td></td>
</tr>
<tr>
<td>II + aVF + V&lt;sub&gt;3-4-5-6&lt;/sub&gt;</td>
<td>100</td>
<td></td>
</tr>
</tbody>
</table>


<sup>2</sup> Blackburn (Ed). Measurements in Exercise Electrocardiography. 1967

**NB:** Blackburn's work is widely published, quoted in RDM etc., but is *exercise* data whereas that obtained by London *et al.* was *intraoperative* ischaemia

- Kaplan & King (1976) using a 5-wire ECG system, monitoring 7 leads, showed ischaemic changes could occur in V<sub>5</sub> in the absence of changes in any other lead
- they recommended that a 5-wire system be used for all cardiac surgery

- most operating rooms have only 3-wire ECG systems, therefore are unable to monitor a true V<sub>5</sub>
- Blackburn found CM<sub>5</sub> the most superior for ischaemia detection and least affected by body weight, electrical frontal plane position and noise
- other studies have found,
  - i. all bipolar leads less sensitive than a true V<sub>5</sub>
  - ii. CC<sub>5</sub> more closely approximates true V<sub>5</sub>
  - iii. bipolar leads less affected by noise than a true V<sub>5</sub>

- ideally, in high risk cases, anterior, inferior and posterior surfaces of the heart should be monitored
- London showed II + V<sub>4-5</sub> had the highest sensitivity, however V<sub>4</sub> & V<sub>5</sub> cannot be monitored simultaneously
- therefore, the best routine combinations are,
  - i. II + V<sub>5</sub>
  - ii. II + CS<sub>5</sub>
  - iii. CB<sub>5</sub>
Central Subclavicular $CS_5$

i. RA $\rightarrow$ under the right clavicle

ii. LA $\rightarrow$ $V_5$ position

iii. LL $\rightarrow$ normal

NB: $V_5$ $\rightarrow$ 5th ICS in anterior axillary line

"lead I" is selected and shows good correlation with true $V_5$ for anterior ischaemia

- advantage is that lead II can be monitored with the same configuration of electrodes,
  a. detection of inferior ischaemia
  b. differentiation of arrhythmias

Central Back $CB_5$

i. RA $\rightarrow$ over centre of the right scapula

ii. LA $\rightarrow$ $V_5$ position

iii. LL $\rightarrow$ normal

"lead I" is selected and monitors the same vector as a true $V_5$ (down, left & anterior)

- however, as origin of vector is behind the RA, cf. true $V_5$, has the advantage of also being useful for assessment of arrhythmias

Central Manubrium $CM_5$

i. RA $\rightarrow$ over the manubrium

ii. LA $\rightarrow$ $V_5$ position

iii. LL $\rightarrow$ ? normal, or any position for ground reference

"lead I" is selected and shows good correlation with true $V_5$ for anterior ischaemia

Oesophageal Electrode

- prominent P-wave and determination of atrial / SVT's
- useful for monitoring posterior wall ischaemia
- consists of an 18F oesophageal stethoscope catheter with 2 leads 13 cm apart
- these are connected to RA & LA, proximal to distal, and "lead I" is monitored
- requires Class A electrical safety features, ie. $< 10 \mu$A leakage current
- also requires attention to adequate grounding for diathermy plates
Studies of Perioperative Ischaemia Research Group (JAMA 1992)

**NB:** series of 7 articles from D. Mangano's group  
almost all data from Veteran's Affairs hospital, therefore older men

- **Predictors of Postoperative MI in Noncardiac Surgery**

  - 474 men scheduled to undergo major noncardiac surgery, entry criteria,
    
    a. **definite CAD**  
       - previous MI  
       - typical angina  
       - atypical angina + positive exercise ECG or DPT scan
    
    b. **high risk of CAD**  
       i. vascular surgery, past or present  
       ii. any 2 of  
           - age > 65  
           - hypertension  
           - smoker  
           - NIDDM / IDDM  
           - high cholesterol

  - 5 major independent preoperative predictors of postoperative ischaemia,
    
    1. LVH by ECG
    2. history of hypertension
    3. diabetes mellitus
    4. definite CAD
    5. use of digoxin

**NB:**  
0 factors → 22%  
1 factor → 31%  
2 factors → 46%  
3 factors → 70%  
4 factors → 77%

- other factors associated with a high incidence were,

  1. **preoperative ischaemia** as detected by holter monitor, and
  2. **intraoperative ischaemia** as detected by 12 lead ECG or holter monitor
**Monitoring for Myocardial Ischaemia in Noncardiac Surgery**

- comparison of TEE or 12-lead ECG, versus 2-lead ECG (CC₅ & CM₅) plus preoperative predictors of ischaemic outcome*
- 332 patients, in whom 285 had technically adequate studies by all 3 techniques
  1. 2 lead ECG* (26%) identified more patients with ischaemia than,
     i. TEE ~ 15%
     ii. 12 lead ECG ~ 14% (this seems incongruous !)
  2. 111 (~ 39%) had intraoperative ischaemia →
     i. ~ 2-3x ↑ in perioperative cardiac outcome
     ii. 63 (19%) had adverse cardiac outcomes, with 11 ischaemic outcomes
  3. using only ischaemic cardiac outcome none of the 3 methods was predictive

**NB:** concluded that, "in comparison with preoperative clinical data and intraoperative monitoring with two-lead ECG,

TEE and 12-lead ECG have little if any incremental value"

- this contrasts Smith *et al.* (Circul*.1985) who assessed TEE during CABG surgery,
  a. TEE ~ 48% versus ECG 12%
  b. all ST changes were in patients with RWMA's

**NB:** generally accepted that TEE is a more sensitive monitor for CABG patients

---

**Ventricular Arrhythmias in Patients Undergoing Noncardiac Surgery**

- major ventricular arrhythmias occurred in 44% of the study group
- more common in,
  1. smokers
  2. history of CCF
  3. ECG evidence of myocardial ischaemia

**NB:** adverse cardiac outcome was not related to the occurrence of arrhythmias

- therefore, when these occur without concomitant signs or symptoms of myocardial ischaemia, they do not require additional monitoring or treatment in the perioperative period
Intraoperative & Postoperative Myocardial Ischaemia in Peripheral Vascular Surgery

- 115 patients (M&F) undergoing elective vascular surgery at the Brigham & Womens hospital
- screened at "low risk" for adverse cardiac outcome,
  1. 35 patients with postoperative ischaemia
  2. 14 of these developed an adverse cardiac outcome
  3. all of these 14 also had preoperative myocardial ischaemia
  4. none of the 15 patients with postoperative ischaemic changes, without preoperative changes, developed an adverse outcome

**NB:** preoperative ischaemia was the single most important predictor of adverse outcome,

  \[ \text{sensitivity} \approx 88\% \]
  \[ \text{specificity} \approx 91\% \]

intraoperative ischaemia in this group was relatively uncommon \( \approx 18\% \)
and was a significant, but much weaker, predictor of adverse outcome, especially in patients at low risk of CAD

Long-Term Cardiac Prognosis Following Noncardiac Surgery

- 444 consecutive patients at high risk for CAD, followed for \( \approx 2 \) years after elective surgery
- 47 (11%) had major CVS complications during the follow-up period,
  1. cardiac death
  2. MI
  3. unstable angina, or new angina requiring hospitalisation
  4. progressive angina requiring CABG or angioplasty

- 5 independent predictors for long-term outcome were identified,
  1. definite CAD
  2. postoperative MI or unstable angina
  3. postoperative ischaemia
  4. history of CCF
  5. history of vascular disease

**NB:** those surviving a postoperative, in-hospital MI had a,

  1. 28x increase in adverse outcome within 6 months, and
  2. 15x increase in adverse outcome at 1 year

- the development of CCF or VT without ischaemia, were not associated with adverse long-term outcome
Summary of Preoperative & Intraoperative Factors

NB: According to Roizen, in Miller 3rd Ed.

- **Preoperative Findings** Correctable
  1. recent MI < 6 months
  2. uncompensated CCF - S₃, ↑ JVP, pulmonary crepitations
  3. severe angina - NYHA Class IV
  4. heart rhythm other than sinus
  5. VEB's > 5/min
  6. BUN > 18 mmol/l
  7. serum [K⁺] < 3.0 mmol/l

- **Preoperative Findings** Uncorrectable
  1. old age > 70 years
  2. significant aortic stenosis
  3. emergency operation
  4. cardiomegaly
  5. history of CCF
  6. angina
  7. ECG evidence of ischaemia - ST, T-wave changes - abnormal QRS complex
  8. significant MR murmur

- **Intraoperative Findings** Correctable
  1. use of vasopressors
  2. hypotension
  3. high rate-pressure product - HR x BPₚₛᵧ > 11,000
  4. long operations

- **Intraoperative Findings** Uncorrectable
  1. emergency surgery
  2. major abdominal or thoracic procedures
<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class O</td>
<td>asymptomatic</td>
</tr>
<tr>
<td>Class I</td>
<td>ordinary physical activity, such as walking or climbing stairs, does not cause angina. Angina with strenuous or rapid prolonged exertion at work or recreation or with sexual relations.</td>
</tr>
<tr>
<td>Class II</td>
<td>slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, or in cold, or in wind, or under emotional stress, or only during a few hours after awakening. Walking more than 2 blocks on the level, or more than 1 flight of stairs at a normal pace and in normal conditions.</td>
</tr>
<tr>
<td>Class III</td>
<td>marked limitation of ordinary physical activity. Walking 1 or 2 blocks on the level and 1 flight of stairs at a normal pace and in normal conditions. &quot;comfortable at rest&quot;</td>
</tr>
<tr>
<td>Class IV</td>
<td>inability to carry on any physical activity without discomfort. Anginal syndrome may be present at rest.</td>
</tr>
</tbody>
</table>
HYPERTENSION

Def’n:  BP > 160 / 95 mmHg

NB:  most studies have focussed upon diastolic hypertension, reflecting PVR, drug therapies aimed at reducing DAP have found good correlation with decreased morbidity & mortality

• although preoperative systolic blood pressure has been found to be a significant predictor of perioperative morbidity, treatment of such hypertension has not been proven to be associated with a reduction in morbidity
• Miller recommends treatment based on the following,
  a.  the patient be educated regarding the necessity for lifelong treatment
  b.  perioperative haemodynamic fluctuations are less frequent in the treated than in the untreated, (Prys-Roberts et al. & Goldman et al.), and
  c.  haemodynamic fluctuations are associated with an increase in morbidity

• such perioperative management should include,
  a.  a search for end-organ disease
    i.  myocardium & coronary arteries
    ii.  kidneys
    iii.  carotid arteries & CNS
    iv.  aorta
    v.  peripheral vascular system
  b.  determination of the "normal range" of pressures for the individual, with the aim of maintaining perioperative pressures within these limits
  c.  all antihypertensive medications should be continued up to the morning of surgery, and the requirement for antihypertensive Rx in the postoperative period addressed

■ Systolic Hypertension

• SAP increases steadily with age 2° loss of elastin, up to age ~ 60, then declines in later life
• previously thought to be "normal" and not indicative of ongoing pathology
• however, the Systolic Hypertension in the Elderly Program (SHEP) found that control of systolic hypertension in the over 60 y.o. group with chlorthalidone decreased the incidence of CVA ~ 35% over 5 years
• therefore likely more elderly patients will be receiving Rx for hypertension
• there is still no general anaesthetic recommendation to treat SAP ~ 180-190 mmHg in elderly patients, except where the surgical procedure necessitates this (eg. CEA)
**Diastolic Hypertension**

1. primary "essential" hypertension ~ 90%
2. secondary hypertension ~ 10%
   i. renal
      - CRF
      - renovascular, high PRA
   ii. endocrine
      - phaeochromocytoma
      - Cushing's disease
      - Conn's syndrome, primary hyperaldosteronism
   iii. drug induced

**Essential Hypertension**

1. CO is normal early but may decrease later with CCF
2. PVR elevated
3. PRA may be low or high
4. LVH - with or without ECG evidence of ↑ volts
5. ↑ IHD - ↑ myocardial VO₂ & accelerated atherosclerosis
   - angina, with or without significant CAD
   - ↑ incidence of AMI
6. 1st functional change is ↓ diastolic relaxation, ie. ↓ compliance
   - therefore more reliant on atrial contribution to filling

- the majority of drug therapy is directed to a reduction of PVR, therefore end-organ perfusion
- first line drug therapy frequently includes diuretics
- since these patients are volume deplete prior to treatment, diuresis is not the mechanism by which these agents reduce pressure
- patients presenting for anaesthesia require volume replacement prior to induction
- other system effects,
  1. vascular integrity
     - endothelial damage, atheroma formation, platelet aggregation, thrombus formation
  2. renal function
     - results in glomerular sclerosis with a ↓ GFR
     - overall RBF may be normal but the distribution is abnormal
     - autoregulation curve is shifted to the right
     - moderate levels of hypotension are more likely to result in postoperative ARF
  3. cerebral function
     - autoregulation curve is shifted to the right
     - ↑ incidence of carotid vascular disease & atherosclerosis
     - ↑ CVA both ischaemic and haemorrhagic
     - TIA's, RIND's, ischaemic atrophy
patients with a BP_{DIA} > 110 mmHg are > 2SD away from the population mean and should have purely elective surgery cancelled

2. isolated systolic hypertension is easily treated acutely and surgery may proceed

3. important to volume load the patient prior to induction

4. use supplemental anaesthetic / hypotensive agents prior to known noxious stimuli
   i. laryngoscopy / intubation
   ii. skin incision, etc.

5. a significant percentage of ischaemia occurs in the post-extubation period in recovery
   - use of supplemental analgesia prior to extubation & adequate pain management
   - avoid - hypothermia & shivering
     - hypoxia, hypercarbia & acidosis
     - bladder distension
   - tachycardia is worse than hypertension in the acute recovery period

PACEMAKERS

<table>
<thead>
<tr>
<th>Chamber Paced</th>
<th>Chamber Sensed</th>
<th>Mode of Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>A = atrium</td>
<td>A = atrium</td>
<td>T = triggered</td>
</tr>
<tr>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td>I = inhibited</td>
</tr>
<tr>
<td>D = double</td>
<td>D = double</td>
<td>D = double</td>
</tr>
<tr>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
</tr>
</tbody>
</table>

1. actually 5 letter code but only 3 commonly used since about 1980
   4 = programmable features
   5 = arrhythmia treatment

- most common type is ventricular R-wave inhibited (demand) type, or VVI
- intraoperative failure may result from,
  i. hyperkalaemia - VT, VF
  ii. hypokalaemia - loss of capture
  iii. other electrolyte abnormalities
  iv. electromagnetic interference
  v. myopotentials
  vi. myocardial ischaemia or infarction
information required about any patient with a pacemaker,

1. the *indication* for insertion and the patient's *default rhythm*
2. pacemaker *type* → pacing, sensing, response
3. location
4. default program mode
5. how to convert the pacemaker to a *fixed mode*,
   • ideally these procedures should be demonstrated to the anaesthetist, and the
     programming unit available in the operating theatre
   i. radiofrequency programmable- programming unit
      • all programmable units have a "panic" setting
      • switching to an output of 5V at 70 bpm in the VVI mode
   ii. nonprogrammable - magnet
6. function with battery failure → increase or decrease in rate

**Electrocautery**

1. use *bipolar* electrocautery if possible
2. use in short bursts & set the electrocautery *current* as low as possible
3. locate the *ground plate* as far as possible from the generator
   ± current axis at 90° to pacing wire
4. the generator should *not* be located between the active electrode & ground plate
5. best monitor is the *peripheral pulse* - manual
   - SpO₂ pleth waveform
6. a *magnet* may be used to reprogram to the asynchronous mode

**Electrocautery Programmable Generators**

1. *do not* reprogram to the asynchronous mode
   • programmable VOO mode can still read EMI as a coded message & reprogram
2. using a *magnet* in the presence of EMI *increases* the likelihood of reprogramming
   • this is in contrast to nonprogrammable generators
3. if a magnet is applied, then the generator will remain in the asynchronous mode, even if
   it reprograms → the new program only manifests when the magnet is *removed*
4. keep the magnet over the generator until a programming unit is available, and
   always remove the magnet under ECG monitoring
5. moving the active electrode over the generator can reprogram the unit
   • electrocautery must be turned-off to avoid interference
• *myopotentials* may be sensed by the unit and result in inhibition
• this may be seen with SCh or postoperative shivering
• *myocardial infarction* may result in loss of capture due to dispersion of current density
• this requires increasing the output from the generator, or relocating the pacing electrode
• inhalational agents *do not* alter pacing thresholds

### Clinical Assessment

1. concurrent disease
2. CXR - lead position and patency
3. native rate < paced rate
   • pacing impulses should appear on the ECG
   • each impulse should correlate with a peripheral pressure pulse
   • absence of a peripheral pulse → cardiology consultation
4. native rate > pacing rate
   • VVI generators will be inhibited
   • vagal (valsalva) manoeuvres will slow native rate & pacing should occur
   • because *sensing* is usually lost before *pacing*, the unit is probably functioning correctly providing,
   i. less than 2 years old
   ii. leads are intact on CXR
   iii. there are no impulses on the ECG

### Temporary Pacing

1. CHB - scheduled for pacemaker insertion
   - emergency surgery & slow ventricular rates
2. 2°HB
   i. Mobitz I - usually associated with inferior AMI
     - no requirement for pacing
     - some would use temporary wire if slow rate & hypotension
   ii. *Mobitz II*
     - may be associated with anterior AMI
     - may rapidly proceed to CHB & *requires pacing*
     *pacing does not* alter 60-70% mortality rate ∝ native disease
3. bifascicular block
   i. RBBB + LAHB - rarely proceed to CHB, pacing not required
   ii. RBBB + LHIB - commonly results in CHB & *requires pacing*
4. SVT - inadequate medical control & emergency surgery
   - requires rapid atrial pacing
5. bradycardia with hypotension, unresponsive to medical therapy, any cause

*NB:* when in doubt, consult the patients cardiologist