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 ± S_3 gallop rhythm
   - ii. preoperative cardiology ***investigations***
     - blood picture - Hb and platelets, WCC
     - coag^{n}. 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 - hypokinesis, akinesis, dyskinesis, aneurysm
       - LVEF < 50%
       - LVEDP > 15 mmHg
       - CI < 2.5 l/min/m^2
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^{++}-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

Determinants of Myocardial Oxygen Supply & Demand	
Decreased O_2 Supply	Increased O_2 Demand
Coronary Blood Flow 1. tachycardia ↓ diastolic perfusion 2. hypotension especially diastolic 3. coronary vascular resistance • increased preload ↑ LVEDP • ↑ wall thickness[1] • ↓ capillary density • coronary artery spasm • hypocapnia • increased viscosity of blood	Wall Tension 1. LV volume ↑ preload 2. LV pressure[2] ↑ afterload 3. 1/wall thickness ***NB:*** Laplace's Law
Decreased O_2 content • anaemia • hypoxaemia	Heart Rate • tachycardia
Decreased tissue O_2 uptake • left shift HbO_2 curve • metabolic poisons, CN^- • sepsis syndrome • myocardial depressant factor(s)	Contractility • ↑ myocardial contractile force

[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*** $\rightarrow$ 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,

   perfusion pressure ~ 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

Classification of LV Function	
Good LV Function	Poor LV Function
Absence of CCF	CCF • ↑ JVP, S_3 gallop • CXR pulmonary oedema, ↑ HS
Hypertension	Ventricular arrhythmia
Normal CI > 2.5 l/min/m^2	CI[1] < 2.5 l/min/m^2
Normal LVEDP < 12 mmHg	LVEDP > 15 mmHg
Normal ventriculogram	Abnormal ventriculogram • akinesis, hypokinesis, dyskinesis • aneurysm
	Recent or multiple infarction

[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

     $\rightarrow$ ***heterometric autoregulation***
   - over-distention increases O_2 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 $\rightarrow$ "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***

Cardiac Catheterisation

Severity	MV Area	Gradient
normal	4-6 cm^2	~ 0 mmHg
mild	1.5-2 cm^2	0-5 mmHg
moderate	1-1.5 cm^2	5-10 mmHg
severe	< 1.0 cm^2	> 10 mmHg

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 $\mathbf{N_2O}$
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
     - S_3 gallop rhythm
     - ↑ JVP, pulmonary oedema, hepatic congestion
     - ***duration*** of the murmur, ***not*** loudness
     - PAH
  b. CXR
     - enlarged LA, LV
     - LVF
  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
   - N_2O
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 QT_C
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 ↑ 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 (↑ compliance)
   - competency of the mitral valve protects the pulmonary circulation until late in the disease, when CO falls and results in ↑ 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 VO_2 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
   - VO_2 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 ~ 40% cf. 15% normally
   - acute onset AF may be associated with LV failure & requires prompt treatment
4. ***heart rate*** ~ 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° 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*** **~ 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 → ***MR* ~ 50%**
  g. pre & post-surgical involvement of the conducting system with ***arrhythmias***

- ***Exacerbating Factors***

  a. ↑ ***contractility*** - sympathomimetics
     - digoxin & (+)'ve inotropes
     - tachycardia
  b. ↓ ***preload*** → reduction in ventricular size
     - hypovolaemia
     - venodilators (GTN)
     - increased PVR, high airway pressures
  c. ↓ ***afterload*** - vasodilators
     - regional sympathectomy

- ***Factors Decreasing Dynamic Obstruction***

  a. ↓ ***contractility*** - β adrenergic blockers
     - Ca^{++} entry blockers
     - volatile anaesthetics
  b. ↑ ***preload*** - hypervolaemia
     - bradycardia
  c. ↑ ***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_2PO_4
   - ix. autoimmune — - RA, PAN, SLE, Kawasaki disease; - scleroderma, dermatomyositis
   - 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/comliance 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 R_X = 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
     - V_5 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 EchocardiagraphyTEE

• 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 patients 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 ó
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. b-***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 O_2 - 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

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 - $Sv'O_2$, oximetry, pacing • ECG - multilead, II + V_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_3^-, Ca^{++}, Mg^{++} • 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 later 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 a as supplement to both IV and volatile agents
• effects upon the myocardium are usually mild → **- SVR / ¯ 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***→ regional ischaemia and dysfunction

• other effects of N_2O 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 F_IO_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 O_2 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

Bubble vs. Membrane Oxygenators		
X = advantageous unit	Bubble	Membrane
Cost	X	
Ease of set-up & operation	X	
Efficiency of oxygenation	X	
Circuit air elimination	X	
Lower priming volume	X	
Oxygenator "pulmonary oedema"	X	
Microembolus production		X
Blood trauma		X
Arterial blood gas adjustment		X

• 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 ***reversable*** with protamine
- clotting of the bypass circuit is the one absolutely fatal complication of CPB

1. usual ***loading dose* ~ 300-400 U/kg** → ~ $25{,}000^U$
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 O_2 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 SvO_2 < 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

Myocardial Preservation

• ***ischaemia*** results in,

i. inhibition of metabolism of glucose, free fatty acids and lactate
ii. anaerobic glycolysis with lactate & H^+ production
iii. eventual inhibition of glycolysis by H^+
iv. 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,

i. abnormal Ca^{++} metabolism
ii. reduced ATP stores and production
iii. O_2 free radical production
iv. 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^{++} 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-Q_{10}
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% /°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*** $\rightarrow$ $\downarrow VO_2 \sim 90\%$
2. ***hypothermia*** to 11°C results in only a further 5% reduction

• Lichtenstein proposed normothermic, kyperkalaemia 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	~ 1	ml/min/100g
b.	arrested, distended	~ 5	ml/min/100g
c.	beating, empty	~ 5	ml/min/100g
d.	normal working myocardium is	~ 10	ml/min/100g
		~ 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 ~ 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/m^2
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 VO_2 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_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_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_2O contraindicated
3. particulate embolisation
   - LA enlargement, chronic AF, aortic valve vegetations, LV aneurysm

Other Organ Systems During Bypass

▪ ***Pulmonary Ventilation***

- maximal 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. $_{FI}O_2$ < 50%
        ii. PEEP ≤ 5 cmH_2O
        iii. P_{aO2} > 60 mmHg
        iv. P_{aCO2} < 50 mmHg
        v. IMV ≤ 4 bpm
        vi. VC ≥ 30 ml/kg
        vii. SRR < 20-30 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***,

$$\delta pH/°C = -0.015$$

• this is due principally to the presence of large quantities of ***imadazole 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 $\delta pH/°C = \mathbf{-0.017}$
• 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*** (a**Im**) of imadazole, show a relatively constant value (~ 0.8) over a wide range of body temperatures, despite variable pH, so called a***-stat*** conditions
• maintaining a temperature corrected pH ~ 7.4, so called ***pH-stat*** conditions, results in a variable αIm, 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 *a-stat* conditions

• ectothermic animals display an *in vivo* $\delta pH/°C$ curve which resembles that of *in vitro* blood
• studies of blood and CSF CO_2 conductance show a strong probability that a constant αIm 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 pN_{H2O} and follows a $\delta pH/°C$ 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
     → NADH cytochrome c reductase - 0.044
     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, $\delta pH/°C = -0.017$

Hibernation

• in contrast, hibernating mammals exhibit a near constant, normothermic pH of 7.4 over a wide range of temperatures, ie. ***pH-stat***
• they demonstrate loading of CO_2 prior to hibernation and hyperventilation during arousal
• they are therefore acidotic and demonstrate a decrease in αIm
• however, both the heart and liver demonstrate ***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^{++} flux

• ***alphastat*** regulation to ~ 25 °C → P_{aCO2} ~ 22 mmHg
• previously CO_2 was added due to the fears of hyocapnia induced decreases in cerebral perfusion
• the low P_{aCO2} values seen with alphastat management produced decreases in CBF ~ 30-50%
• however, CBF is still in excess of $CMRO_2$ and the brain has no O_2 storage capacity
• pH-stat management effectively uncouples $CMRO_2$ & CBF, resulting in relative ***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 °C in dogs,

1. LV VO_2 is ~ 1.8x 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

NB: Kroncke *et al.* (Acrh Surg 1986) looked at the incidence of spontaneous VF prior to AOX clamping during hypothermia to 24 °C → 20% versus 40% for pH-stat

• further, alphastat management requires no calculations, as measurements by standard AGA analysis are performed at 37°C, irrespective of the level of hypothermia

• Bashein *et al.* (Anesth. 1990) randomised controlled trial of alphastat vs. pH-stat,

1. showed ***no difference*** in neurological outcome
2. careful ***surgical technique*** and ***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 $[K^+] \sim 25$ mmol/l, however this is usually less than the amount loss 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. sympathomimmetic 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

Monitoring for Cerebral Ischaemia

Method	Features
Single or Dual lead EEG, or Processed EEG	• mainly global changes seen ? adequate sensitivity • decreased use with profound hypothermia or barbiturates
Multi-lead raw, or Processed EEG	• improved sensitivity with regional changes • cumbersome to apply and monitor
Carotid or Transcranial Doppler	• detection of emboli & surgical feedback • primarily a research tool
Jugular bulb - SjO_2	• global or unilateral changes seen • intermittent, not continuous (optode catheter !)
Regional CBF, or velocity	• research tool
Spinal CSF pressure	• highly invasive • utility unclear

• 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 N_2O 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
   - icthyosis
   - 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 VO_2
   - 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 ∝ ↓ $CMRO_2$ ~ **6-7% / °C**
   ~ similar change cf. whole body VO_2
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 CO_2 drive
4. no change in hypoxic drive
5. impaired hypoxic pulmonary vasoconstriction
6. reduced FRC, increased atelectasis
7. decreased gaseous diffusion capacity
8. ↑ VO_2 with ***shivering*** → ↓ VO_2 ≤ 33°C
9. ↓ O_2 availability ∝ ↑ HbO_2 affinity
10. increased ***gas solubility***
    i. ↑ αCO_2 / ↓ P_{aCO2} → ↑ pH
    ii. anaesthetic gases → ↓ rate of rise of F_A/F_I & elimination
    - halothane $MAC_{27°C}$ ~ 50% $MAC_{37°C}$

- ***Metabolic***

1. ↓ VO_2 ~ 6-7% / °C
2. severe ***acidosis*** → HbO_2 curve shifts to the ***right***
   i. respiratory ↓ CO_2 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 $t_{½\beta}$ ∝ ↓ hepatic blood flow & enzyme reaction rates
   → ***heparin, citrate & lactate***

- ***Renal***

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

- ***Neuromuscular Junction***

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

- ***Haematological***

1. ***coagulopathy***
   i. ↓ coagulation ↓ enzyme activity
   ii. thrombocytopaenia ↑ portal platelet sequestration
   ↑ bleeding time
2. increased blood ***viscosity*** - haemoconcentration & ↑ Hct.
   - dehydration
   - ↓ 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 VO_2
   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***

a. cutaneous thermoreceptors ~ 15% of input
   i. cold receptors < 24°C
   ii. heat receptors > 44°C

b. deep/core thermoreceptors ~ 85% of input
   i. anterior hypothalamus
   ii. spinal cord
   iii. 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
   ↑ BMR ≤ 1000% 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 Hz
   - may ↑ BMR ~ 200-500%
   - may ↑ core temperature ~ 2-3 °C/hr
   - requires ↑ VO_2 ~ 100% / ↑1°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 ~ 25% ↑ BMR
   - ***brown fat*** in neonates ~ 100% ↑ BMR
     ~ 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. ↓ VO_2 ~ 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 + N_2O/O_2 ~ 34-34.5°C
     - isoflurane + N_2O/O_2 ~ 3°C/$ET_{\%ISO}$
     - propofol + N_2O/O_2 ~ 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, ↓ O_2 delivery & tissue hypoxaemia
   - impaired coagulation
4. CVS - ↓ CO & arrhythmias (*AF)
5. postoperative problems
   i. shivering - ↑ VO_2 & 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,
core-shell gradient → better assessment of overall body temperature
→ 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. ***understates*** 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. MBA_{20} - 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,

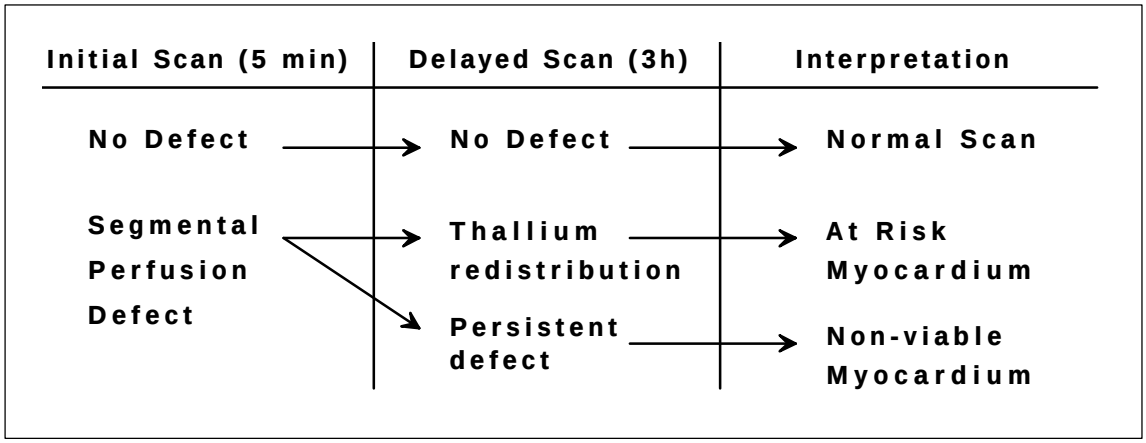
1. 5.7% mortality in 70 CABG's performed on patients with infrarenal aortic aneurysms
2. an incidence of aortic rupture following CABG, prior to repair ~ 2.9%
3. operative mortality for aortic repair ~ 1.8%

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



• 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,

i. AMI
ii. sudden death
iii. CVA
iv. acute aortic dissection
v. ruptured aortic aneurysm
vi. 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,

a. increased basal vasomotor tone & arteriolar hypertrophy
   → vascular "hyperreactivity"
b. reduced intravascular volume
c. LVH - decreased compliance & preload dependence
d. increased capillary leak
e. regional circulatory changes
   i. renal - ↑ RVR > ↑ SVR
      - ↓ RBF ∝ duration & severity of hypertension
   ii. CNS - narrower autoregulatory range & shifted right
   iii. 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,
   i. greater decreases in MAP
   ii. more episodes of hypotension associated with subendocardial ischaemia
   iii. greater ↑ SVR / ↓ CO with hyperventilation
   iv. 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 S_3 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, $[K^+] < 3.0$ mmol/l or > 20% total deficit
   → cancel elective surgery & replace orally over 3-4 days
2. when oral replacement is inappropriate, then, (from Miller)
   i. give ≤ 20 mmol K^+ / hr
   ii. give ≤ 240 mmol K^+ / 24 hrs
   iii. continuously monitor ECG
   iv. measure plasma $[K^+] \leq 4$ hrly ? this seems absurd
   v. allow at least 13 hrs for moderate deficit ~ $[K^+] < 3.0$ mmol/l
   allow 24-48 hours for large deficit ~ $[K^+] < 2.5$ 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. b-***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^{++}-*channel blockers***
   - patients receiving combination β-blockade & CEB therapy may develop profound ***bradycardia*** or asystole during induction with ***fentanyl***
   - exert additive effects on Ca^{++} 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***
   - Odoom (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 intrapumonary 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 hypoglycaemics 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
- R_X 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

• generally accepted goals of management include,

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_{aCO2}
3. enhancing the brains tolerance of ischaemia by pharmacological depression of 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_IO_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 SpO_2
   iii. ? mild $\downarrow P_{aCO2}$
   iv. $\downarrow CMRO_2$ - 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 $\rightarrow$ hypercarbia, hypoxia, hypertension, raised ICP
   iii. avoid hypertension & hypotension - volume status
   - pain relief
   - β-blockade (only if $\uparrow$ 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
   - α_2-agonists (clonidine 4-5 µg/kg po)
5. metoclopramide / ranitidine

Monitoring

- minimum considered adequate (RDM),

a. continuous ECG - II + V_5
   - lead selection as guided by distribution of CAD

b. oesophageal stethoscope & temperature probe

c. F_IO_2, SpO_2, $ETCO_2$, 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, V_5 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 / alfentanyl

• to avoid a hypertensive response to ***intubation***,

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_{EI} (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 $1/6^{th}$ 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

→ small fall in CO < decrease in PAoP and SVR

• studies comparing high dose fentanyl / O_2 / isoflurane showed ***no benefit*** cf. a "conventional" low dose fentanyl / O_2 / N_2O / isoflurane technique
• further, the addition of a volatile agent prevents the hyperdynamic response to AoXC seen with an unsupplemented opioid / O_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 sufentanyl 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/O_2 + relaxant anaesthesia provides the advantages of,

1. a high F_IO_2
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 N_2O 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

Haemodynamic Changes with Aortic Cross-Clamping		
Change = δ%	***Infrarenal***	***Supracoeliac***
MAP mmHg	↑ 7-12%	↑ 35-54%
CO l/min	↓ 16-21%	↓ 29%
SV ml/beat/m^2	↓ 15-20%	↓ 32%
SVR dyne/s/cm^{-5}	↑ 33-36%	↑ 88%
PCWP mmHg	↓ 23 - ↑ 50%	↑ 83%
LVEDA	↑ 9%	↑ 28%
LVESA	↑ 11%	↑ 69%
RWMA's (new)	0%	92%

• 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 supracoeliac clamping

• therapy for haemodynamic alterations following AoXC may include,

1.	<u>hypertension alone</u>		BP > 170/100 mmHg
	i.	deepen anaesthesia	- increase volatile agent - IV supplement (propofol, droperidol, opioid)
	ii.	vasodilatation	- nitroprusside
	iii.	antihypertensives	- esmolol, atenolol, labetalol
2.	<u>LV strain / failure</u>		↑↑ PAoP
	i.	nitroglycerine	~ 0.25-0.75 µg/kg/min
	ii.	dobutamine	~ 2-8 µg/kg/min
	iii.	? amrimone/milrinone	
3.	<u>myocardial ischaemia</u>		↓ 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 $Sv'O_2$

• 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_{aO2}
   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 crystalloidists 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-O_2
     - low dose opioid + STP / propofol
     - high dose opioid
     - non-depolarising relaxant
   - ii. intubation — - when TOF = 0
     - supplemental agents
   - iii. maintenance — - O_2 ± N_2O
     - 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 VO_2
     - cardiorespiratory homeostasis
     - sedation
   - ii. pain relief — - opioid infusion
     - regional anaesthesia
   - iii. fluid management — - PAoP, U/Output
     - CXR, $P_{A\text{-}aO2}$ gradient
     - Hct / Hb and DO_2
     - 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 O_2
2. placement of at least 2 large bore IV cannulae
3. obtain blood for G&M, Hct., FBE, MBA_{20}
   - 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, SpO_2
   ii. defer placement of urinary catheter & N/G tube until after induction
      - risk of induced Valsalva resulting in rupture
6. pre-O_2, 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. N_2O / O_2
   iv. volatile supplement
9. monitoring *post-induction
   i. ECG, IABP & NIBP, CVP ± PA catheter[§§]
   ii. SpO_2, $ETCO_2$, 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 4-5 th 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 2 ° 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 2° 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
   & SpO_2, $ETCO_2$, P_{AW} *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

- b-***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 HCO_3^- is warranted to prevent ***hyperkalaemia*** and arrhythmias,

$$\text{Dose} \sim (0.3 \times \text{Wt} \times \text{BE})/2$$

ISCHAEMIC HEART DISEASE

▪ ***Identification of Patients***

• any of the following may indicate the presence of IHD,

a. a history of "vise-like" chest pain ± radiation to the neck or arm
b. dyspnoea on exertion
c. dyspnoea on exposure to cold, after eating, or after defecation
d. paroxysmal nocturnal dyspnoea
e. orthopnoea
f. past or present, peripheral or pulmonary oedema
g. a history of MI
h. a family history of IHD/MI
i. diagnosis of MI by ECG or cardiac enzymes
j. diagnosis of IHD by ECG, stress testing or Holter monitor
k. 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***

  i. males ~ **3.5** : 1000
  ii. females ~ **1.0** : 1000

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

- ***Aetiology***

  i. ***atherosclerosis*** ~ 99%
  ii. embolism - thrombus, air
  iii. coronary arteritis - polyarteritis nodosa, Kawasaki dis.
  iv. congenital coronary anomalies - eg. LCA from PA
  v. myocardial hypertrophy & aortic stenosis
  vi. aortic dissection 2° - aortitis, syphilis, Marfan's, trauma
  vii. severe trauma
  viii. electrocution
  ix. severe hyperthermic syndromes
  x. prolonged cardiopulmonary bypass
  xi. 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
     i. family history
     ii. obesity
     iii. 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 (LDH_1)
- ↑ 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, occuring at weeks to months
5. rehabilitation

Treatment - General

a. education, explanation and reassurance

b. bed rest

c. analgesia

d. supplemental O_2

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.	b-***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,

i. mild angina
ii. 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,

i. ***left main disease***, or
ii. ***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		
Factor	Streptokinase	tPA
Artery patency at 2 hours	55%	70%
Incidence of re-occlusion	15%	20%
Reduction in mortality **+ *aspirin***	20% ***51%***	26% ?
Cost	$140	$1,350
Hypotension	20%	
Anaphylaxis	0.1%	
Fall in fibrinogen	80%	30%
Cerebral haemorrhage	0.4%	0.5%
Major haemorrhage	0.6%	4%
Plasma half-life	23 min	9 min

PERIOPERATIVE MYOCARDIAL REINFARCTION

Goldman (1977)

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

- ***Independent Variables***

1. S_3 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 3rd 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)

Reinfarction Rate		
Interval	Control Group [1] (n=364 / 1973-76)	**Prospective Group** (n=733 / 1977-82)
< 3 months	36%	**5.8%**
3-6 months	26%	**2.3%**
> 6 months	~ 5% p.a.	**~ 1.5-1.7% p.a.** [2]

[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

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NB: the risk of intraoperative ischaemia, and therefore postoperative MI, is determined primarily by the patients ***native disease severity***, not by perioperative anaesthetic management

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Fleischer & Barash (1992)

• 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***

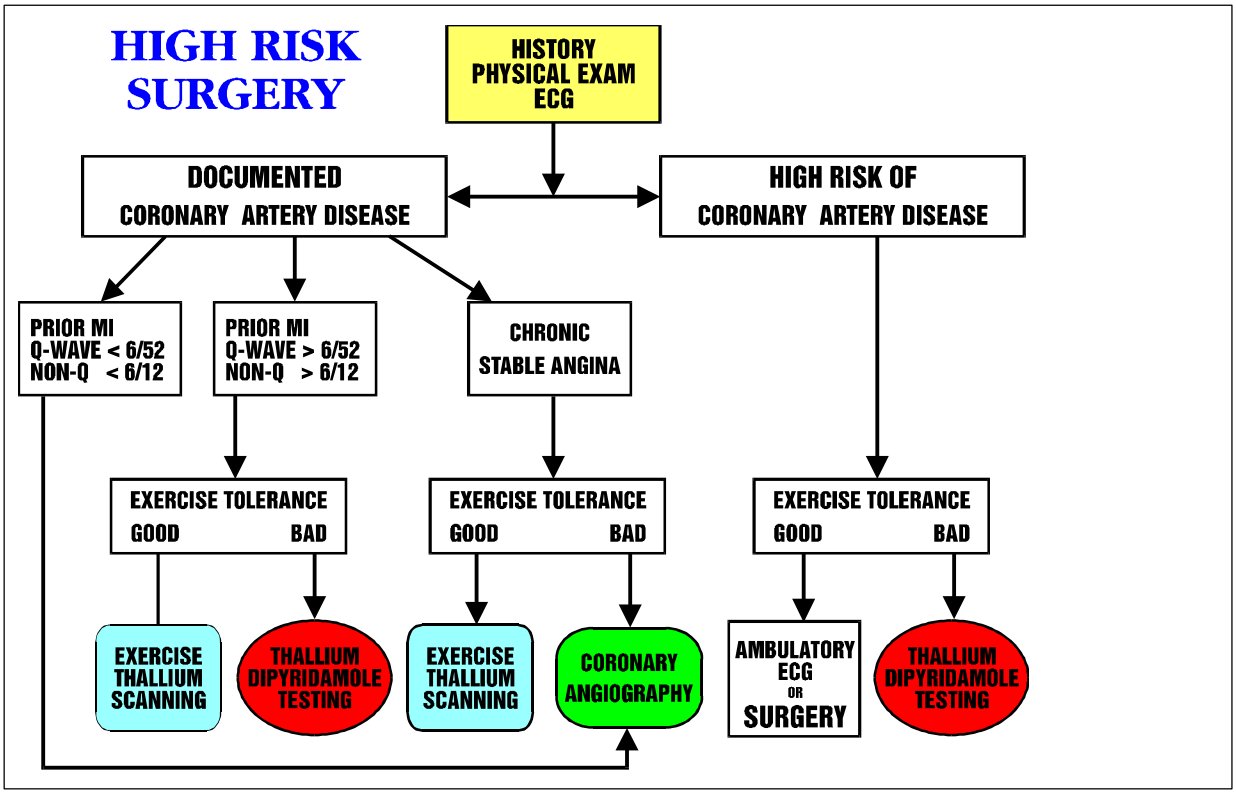
• although distinction between "Q-wave" and "non-Q-wave" infarction* may be relevant, it is important to remember that,

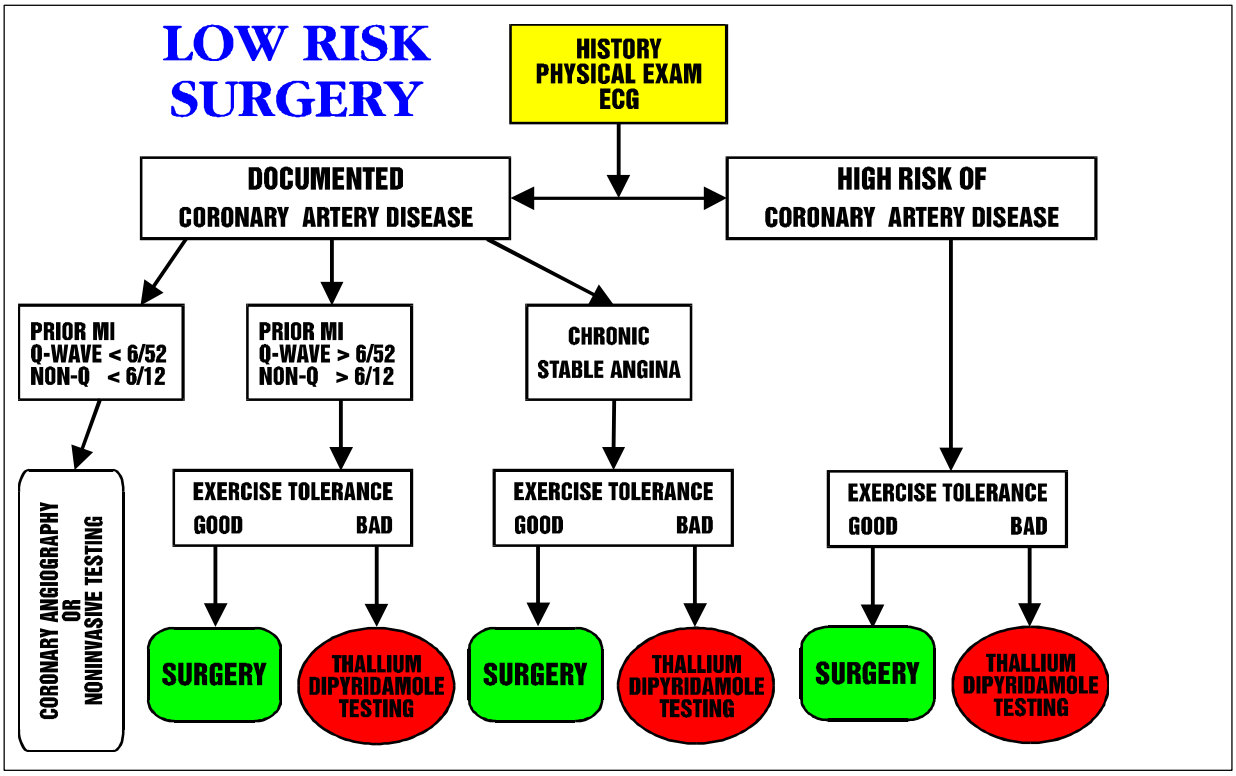
a. ECG classification as such ***does not*** necessarily correlate with transmural and subendocardial infarction
b. there is significant overlap between these groups, especially with the use of thrombolytic therapy

• 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


HIGH RISK SURGERY
HISTORY PHYSICAL EXAM ECG
DOCUMENTED CORONARY ARTERY DISEASE
HIGH RISK OF CORONARY ARTERY DISEASE
PRIOR MI Q-WAVE < 6/52 NON-Q < 6/12
PRIOR MI Q-WAVE > 6/52 NON-Q > 6/12
CHRONIC STABLE ANGINA
EXERCISE TOLERANCE GOOD BAD
EXERCISE TOLERANCE GOOD BAD
EXERCISE TOLERANCE GOOD BAD
EXERCISE THALLIUM SCANNING
THALLIUM DIPYRIDAMOLE TESTING
EXERCISE THALLIUM SCANNING
CORONARY ANGIOGRAPHY
AMBULATORY ECG OR SURGERY
THALLIUM DIPYRIDAMOLE TESTING


LOW RISK SURGERY
HISTORY PHYSICAL EXAM ECG
DOCUMENTED CORONARY ARTERY DISEASE
HIGH RISK OF CORONARY ARTERY DISEASE
PRIOR MI Q-WAVE < 6/52 NON-Q < 6/12
PRIOR MI Q-WAVE > 6/52 NON-Q > 6/12
CHRONIC STABLE ANGINA
CORONARY ANGIOGRAPHY OR NONINVASIVE TESTING
EXERCISE TOLERANCE GOOD BAD
EXERCISE TOLERANCE GOOD BAD
EXERCISE TOLERANCE GOOD BAD
SURGERY
THALLIUM DIPYRIDAMOLE TESTING
SURGERY
THALLIUM DIPYRIDAMOLE TESTING
SURGERY
THALLIUM DIPYRIDAMOLE TESTING

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

Predictive Value of Adverse Cardiac Outcome[1]

Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
Exercise ECG	30.6	83.2	30.6	**83.2**
Ambulatory ECG	43.4	86.3	35	**89.9**
Dipyridamole ^{201}Th	**83.5**	68	37.9	**94.7**
DPT - Cunningham	85-93%	64-80%		

[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 $\geq$ **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

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

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• 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

Lead Placement	Sensitivity (%)[1]	Sensitivity (%)[2]
II alone	**33**	
V_5 alone	**75**	89
II + V_5	**80**	
II + V_4 + V_5	96	
II + aVF + $V_{3\text{-}4\text{-}5\text{-}6}$		100

[1] London, Hollenburg, Wong *et al.* Intraoperative myocardial ischaemia: localisation by continuous 12-lead electrocardiography. Anesthesiol.1988; 69:232-41

[2] 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_5 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_5
• Blackburn found CM_5 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_5
ii. CC_5 more closely approximates true V_5
iii. bipolar leads less affected by noise than a true V_5

• ideally, in high risk cases, anterior, inferior and posterior surfaces of the heart should be monitored
• London showed II + $V_{4\text{-}5}$ had the highest sensitivity, however V_4 & V_5 cannot be monitored simultaneously
• therefore, the best routine combinations are,

i. II + V_5
ii. II + CS_5
iii. **CB_5**

- ***Central Subclavicular*** ***CS_5***

  i. RA → under the right clavicle
  ii. LA → V_5 position
  iii. LL → normal

  NB: **V_5** → 5^{th} 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 → over centre of the right scapula
  ii. LA → V_5 position
  iii. LL → 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 → over the manubrium
  ii. LA → V_5 position
  iii. LL → ? 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 µA 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 (CC_5 & CM_5) 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[n].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,

sensitivity **~ 88%**
specificity **~ 91%**

intraoperative ischaemia in this group was relatively uncommon **~ 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 ~ 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,

i. 28x increase in adverse outcome within 6 months, and
ii. 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	- S_3, ↑ 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 BP_{SYS} > 11,000
4.	long operations	

- ***Intraoperative Findings Uncorrectable***

1.	emergency surgery
2.	major abdominal or thoracic procedures

NYHA Classification of Angina	
Class O	• asymptomatic
Class I	• 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
Class II	• 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
Class III	• 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 • "comfortable at rest"
Class IV	• inability to carry on any physical activity without discomfort • anginal syndrome may be present at rest

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 R_X 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 R_X 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 VO_2 & 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

- ***Perioperative Management ASA E.Miller***

1. patients with a **BP_{DIAS} > 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

Pacemaker Coding[1]		
Chamber ***Paced***	Chamber ***Sensed***	Mode of ***Response***
A = atrium	A = atrium	T = triggered
V = ventricle	**V** = ventricle	**I** = inhibited
D = double	D = double	D = double
	O = none	O = none

[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
   - SpO_2 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 + LPHB*** - 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