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By Disease

Ischaemic Heart Disease

Preoperative

HISTORY

- take pain history
- assess severity
- recent MI's
- previous thrombolysis, stent or CABG
- CHF symptoms
- functional ability (MET's)
- medications

Canadian Cardiovascular Angina Scale

- I – ordinary physical activity doesn't cause angina (> 4 METS)
- II – slight limitation of ordinary activity (2-4 METS)
- III – marked limitation of ordinary activity (1-2 METS)
- IV – inability to carry out any physical activity (angina @ rest)

METS

- Insult to surgery ↑s O₂ demand by up to 40%
- 1 MET = 3.5ml/kg/min O₂ consumption of 40yr old 70kg male
- Guide =
 - ▶ 1-4 = eating, dressing, walking around house
 - ▶ 4-10 = climbing flight of stairs, brief running, golf
 - ▶ >10 = strenuous sports

EXAMINATION

- standard CVS examination
- may be nothing to find
- look for CCF symptoms

RISK STRATIFICATION FOR SURGERY

- need to consider:
 - ▶ patient RFs
 - ▶ Surgery RFs

Box 3.1 Predictors of perioperative risk

Major risk predictors (markers of unstable CAD)

Recent MI (<1 month prior to planned surgery)
 Unstable or severe angina
 Ongoing ischaemia after MI (clinical symptoms or non-invasive testing)
 Decompensated heart failure
 Significant arrhythmias (high-grade AV block, symptomatic arrhythmias, or supraventricular arrhythmias with uncontrolled ventricular rate)
 Severe valvular heart disease (aortic stenosis gradient >40mmHg, valve surface area <1cm²), mitral stenosis)
 CABG/PCI (BMS <6wk, DES <1yr)

Intermediate risk predictors (markers of stable CAD)

History of IHD
 History of compensated or prior heart failure
 History of cerebrovascular disease
 Abnormal renal function creat >180
 Diabetes on insulin

Minor risk predictors (increased probability of heart disease)

Advanced (physiological) age
 Abnormal ECG
 Rhythm other than sinus
 Low functional capacity
 Uncontrolled systemic hypertension

AV, atrioventricular; BMS, bare-metal stent; CABG, coronary artery bypass grafting; CAD, coronary artery disease; DES, drug-eluting stent; ECG, electrocardiogram; IHD, ischaemic heart disease; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Box 3.3 Risks of surgery in ischaemic heart disease**High risk:**

>5% death/non-fatal MI

- Major emergency surgery (especially in elderly)
- Aortic/major vascular surgery
- Prolonged surgery with large fluid shifts

Intermediate risk:

<5% death/non-fatal MI

- Carotid endarterectomy/endovascular aneurysm repair
- Head and neck surgery
- Intraperitoneal and intrathoracic surgery
- Orthopaedic surgery
- Prostatic surgery

Low risk:

<1% death/non-fatal MI

- Minimally invasive endoscopic surgery
- Cataract extraction
- Superficial surgery (including breast)

OTHER RISK STRATIFICATIONS FOR SURGERY

- Lee's criteria for periop CVS risk in non-cardiac surgery (3 day MACE risk):
 - high risk surgery (abdo, thoracic or suprainguinal vasc surgery)
 - Hx IHD
 - Hx stroke/TIA
 - Hx of heart failure
 - chronic renal impairment = creat >177
 - DM on insulin
- ↳ Risk of cardiac events periop based on number of factors:
 - 0 = 0.4%
 - 1 = 1%
 - 2 = 6%
 - ≥3 = 11%

RISK FACTORS FOR IHD

- DM
- HT
- lipids
- family history
- male
- obesity
- previous MI
- hormone replacement for menopause

INVESTIGATIONS

- ask about which investigations the patient has had

NON-INVASIVE12 lead ECG

- those > 60yrs and those with risk factors
- looking for; conduction abnormalities, arrhythmias, LVH, Q waves
- many with IHD will be normal

ETI

- gives an assessment of functional capacity
- looking for; ST depression, hypotension, arrhythmias
- adv: excellent at finding clinically sig vessel disease
- disadv: poor at identifying single vessel disease
- Should have pharmacological stress test if:
 - unable to exercise

- ▶ pre-existing ECG abnormalities eg LBBB, dig effect, LVH, paced

CPET Testing

- bike or hand ergometer
- under exercise O₂ consumption is a linear function of Q and thus LV function
- aerobic threshold of >11mL/min/kg is able to predict survival after major abdominal surgery accurately

Dobutamine Stress Echo

- those that can't exercise
- up to 40mcg/kg/min
- looks @ regional wall motion as an indicator of impaired perfusion
- > 4 new wall motion abnormalities @ <60% of age predicted max HR = high risk
- very operator dependant

Nuclear Medicine Scan – Dipyridamole thallium scintigraphy, SESTAMIBI, SPECT MPI, PET

- coronary vasodilator (dipyridamole) and radio isotope (thallium) which is up taken into perfused myocardium
- impaired perfusion shows up as reversible perfusion defects caused by dipyridamole causing a steal phenomena
- non-perfused areas show up as permanent perfusion defects
- key findings one is looking for = reversible perfusion defects, permanent perfusion defects and cavity dilation
- negative test is very reassuring

CT Coronary Angiogram

- quantification of the amount of Ca²⁺ in the coronary arteries
- massive dose of radiation
- useful when wanting to completely rule out CAD burden

Dobutamine stress MRI

- elegant up and coming form of stress testing
- identifies wall motion abnormalities, R&L ven function, cardiac masses, congen heart disease, suspected ARVD
- highly accurate
- safe

Technique	Sensitivity	Specificity
ETT	70%	80%
Exercise Stress ECHO	80	85
Dobutamine Stress ECHO	80	80
Sestamibi MPI	80	75
SPECT MPI	90	75
PET scan	90	80

INVASIVE

Coronary arteriography

- delineates who needs PCI, CABG or medical management
- considered gold standard
- anatomical nature of lesions
- can stent but has issues relevant to surgery (see AHA 2007 Guidelines)

MANAGEMENT

PreOperative Procedures

- CABG:
 - ▶ may be required prior to high risk non-cardiac surgery
 - ▶ indications same as norm CABG ie
 - >50% L main stem stenosis
 - >70% 2 or 3VD

- severe angina on max medical therapy
- failed angioplasty with ongoing dynamic changes
- life threatening arrhythmias
- ↓LVEf with above stenoses
- ▶ post CABG must wait 3/12 prior to surgery
- ▶ if symptoms stable post CABG then no change to cardiac risk peri-op
- PCI:
 - ▶ rare to be indicated pre elective surgery (only unstable pts)
 - ↳ CARP trial - stable angina no need to re-vascularise as doesn't change outcome
 - ↳ but did exclude certain probs eg LMS disease
 - ▶ recent PCI \approx \uparrow 30d mortality & \uparrow Mi risk
 - ▶ trauma to coronary vessel endothelium \Rightarrow thrombogenic until stent re-endothelialised
 - ▶ \therefore dual antiplatelets post procedure:
 - aspirin for life
 - clopidogrel: 3wk balloon; 6wk BMS; 12month DES
 - ↳ (45% risk of complications within these times if stopped)
 - ▶ DES:
 - ideally no surgery for 12 months:
 - <6/12 = continue aspirin & clopidogrel and accept bleeding risk
 - 6-12 months = consult cardiology - newer DES need shorter DAPT times
 - >12/12 = aspirin only
 - ▶ BMS:
 - <6 weeks - 3 months = aspirin & clopidogrel
 - >6 weeks - 3 months = aspirin only
 - ▶ if need to stop clopidogrel for undelayable surgery and high bleeding risk:
 - stop 7days
 - bridge with short acting IIb/IIIa glycoprotein antagonist ie tirofiban (stop 6hr prior to surgery) + heparin peri-surgery

Practical Actions

- keep most meds going and convert to IV form if oral not an option
- keep betablockers going if on already
- if v high risk patient with inducible ischaemia on stress testing may be benefit if started β blocker >1wk prior to surgery (target HR 60-80)
- clonidine can be used to substitute for beta-blockers (300mcg daily)
- keep nitrates going (can substitute for SL or transdermal)
- keep Ca²⁺ channel blockers going
- ACE I do drop BP but can be continued (debatable about what should be done)
- keep statins going
- high risk (3 or more clinical risk factors) + vascular surgery -> consider non-invasive testing if it will change management

Intraoperative

- invasive monitoring
- CM5 ECG
- avoid tachycardia, hypotension, hypoxia, hypercarbia
- good analgesia
- keep Hb > 70g/L
- avoid SNS surge at extubation:
 - ▶ deep extubation
 - ▶ esmolol

Intra-Op Monitoring to Identify Ischaemia

- PAC
 - ▶ ischaemia \approx \uparrow pressures esp diastolic pressures
 - ▶ but are other causes eg MR, \uparrow SVR, \uparrow CO, \uparrow PVR
 - ▶ also need to consider CI's & risks of insertion
- ECG:
 - ▶ ST depression can be non specific esp if upsloping
 - ▶ not good to localise territory eg abnormal anatomy

- ▶ other causes of changes eg old infarct, pace, drugs
- TOE:
 - ▶ disadv: invasive, op dependant, CI's to insertion, slow onset of signs

Postoperative

- O2 for 3-4 days
- HDU
- high index of suspicion for perioperative MI
- 12 lead ECG post op day 1, 2 and 3
- TNT if high risk day 2 and 3
 - ↳ any rise \approx \uparrow periop mortality risk but need to be careful:
 - no baseline TNT figure eg often \uparrow ed in elderly, renal dysfunction, vascular
 - use serial data
 - not clear what to do then
- keep anti-platelets going
- if develops ST elevation -> urgent angiogram
- treat angina and CHF aggressively
- maintain BP

PeriOperative β Blockers (POISE)

- practical approach:
 - ▶ continue established β blockers
 - ▶ avoid starting perioperatively - POISE
 - ▶ if in pre-Ax >1week prior to surgery \Rightarrow consider starting if no contraindications

ADV's	DISADV's
\downarrow HR	\uparrow periop hypotension \Rightarrow \uparrow NSTEMI
\downarrow afterload	SNS blockade \Rightarrow \downarrow protective response to hypovolaemia
\downarrow myocardial O2 consumption	\uparrow risk of stroke
prevention of periop \uparrow HR \Rightarrow \downarrow NSTEMI	\uparrow risk of bradycardia
prevention of arrhythmias	\uparrow morbidity & mortality
\downarrow bleeding	

PeriOperative Statins

high risk start 30mg/day before surgery \Rightarrow \downarrow mortality 50%

ADV's	DISADV's
\downarrow IHD, \downarrow all cause mortality, \downarrow CVS mortality	rare cause of rhabdomyolysis
Effect additive to β blockers	
NNT to prevent cardiac event <15	
well tolerated	

PeriOperative ACS

- usually occurs d3-4
- 10-20% mortality

- typical clinical symptoms may be masked by residual anaesthetic, analgesia, pain from surgical site
- causes:
 - ▶ type I = plaque rupture - 50%
 - ▶ type II = O2 supply:demand imbalance - 50%
 - ↳ are other types - not relevant peri-op
- TNT's
 - ▶ most sensitive tests
 - ▶ rise within 3hrs; peak at 12-24hr; present for 7-10days
- Standard ACS care
- consider acute thrombolysis & PCI - discuss with surgeon & cardiologist

Left Ventricular Dysfunction

History

- SOB with ↓ ex tolerance
- oedema
- Orthopnoea & PND

Examination

- include:
 - ▶ ↑JVP
 - ▶ pulsus alternans - pulse waveform showing alternating strong then weak beats
 - ↳ note other causes though = pericardial effusion, LVF, asthma
 - ▶ S3 - most predictive
- difficulty in reliability amongst different examiners

Sensitivity and Specificity of Examination Findings for LV dysfunction

Finding	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)
Shortness of breath	66	52	23
Orthopnea	21	81	2
PND	33	76	26
Oedema (history)	23	80	22
Tachycardia (>100/min)	7	99	6
Pulmonary crepitations	13	91	27
Oedema (exam)	10	93	3
Gallop (S3)	31	95	61
Neck vein distension	10	97	2

S3

- can be normal esp <40yrs
- flow into LV
- beginning diastole from rapid vent filling

S4

- always abnormal
- atrial contraction against stiff LV
- end diastole

Basic Investigations

- ECG:
 - ▶ if normal = sensitivity 94%, -ve predictive value 98% for normal vent function
 - ▶ powerful predictors =
 - ant Q waves
 - LBBB
- CXR:
 - ▶ cardiomegaly - sensitivity 51%, specificity 79%
- BNP
 - ▶ sensitivity 73%, specificity 82%
 - ▶ good r/o test for heart failure
 - ▶ optimisation of HF therapy
 - ▶ but one off test usefulness unclear
- ECHO

Heart Failure

- Aim = to determine disease severity and optimization of patient prior to surgery
- 50% mortality at 5yrs
- uncontrolled failure for emergency laparotomy \approx 20-30% mortality

Preoperative

HISTORY

- Current symptoms of heart failure – orthopnoea, PND, ankle swelling, exertional dyspnoea, fatigue
- Frequency of symptoms
- Episode of decompensation - if in last 6/12 = \uparrow risk
- Severity
- Current exercise tolerance
- Recent episodes of chest pain, palpitations

- Circumstances surround diagnosis of heart failure – presentation (MI, valve dysfunction, arrhythmia) and treatments instituted.
- Duration of cardiac failure
- Current control of symptoms
- Recent cardiology opinions
- Whether has systolic or diastolic dysfunction
- Presence of LV aneurysm
- History of mural thrombus ? thromboembolic disease

PMH

- IHD – ischaemic cardiomyopathy
- AF
- OSA with pulmonary hypertension and right sided heart failure
- Chronic respiratory disease
- Congenital heart disease
- Hypothyroidism/Hyperthyroidism
- Renal Failure
- Rheumatic Fever

Meds

- All current medications - look specifically at whether patient is optimised with diuretics, ACE-I, Beta-blockers, nitrates, ARBS and spironolactone.
- Also whether other conditions are optimised – IHD medications, anti-coagulation, digoxin.

- SHx – current smoking history and ET-OH intake (alcoholic cardiomyopathy)

EXAMINATION

- Signs of heart failure –
 - Vitals: tachycardia, BP, arrhythmias
 - Heart: elevated JVP, laterally displaced apex, left or right ventricular heave, murmurs,
 - Lungs: crackles at bases of lungs, pleural effusions, RR
 - Other: enlarged liver, pedal oedema

- Other important signs – ECG: atrial fibrillation (rate controlled or not)

- Walk patient up stairs (unlikely if requiring knee replacement)

INVESTIGATIONS

- FBC – anaemia (cause of high output failure)
- U+E – renal failure from cardiac failure, hyponatraemia from diuretics

- LFTs – liver congestion from right heart failure
- BNP – is sometimes used by cardiologist in outpatient setting to monitor control and response to treatment of anti-heart failure therapy.
- COAGS – if on anti-coagulation for mural thrombosis treatment or AF
- TFT's – hyper/hypothyroidism
- ECG – signs of cardiac dysfunction – dynamic ischaemic changes, degrees of heart block, LBBB, RBBB, arrhythmias
- CXR:
 - ▶ prominent upper lobe veins, engorged peripheral lymphatics (Kerley B lines), alveolar oedema (Bat wing)
 - ▶ pleural effusions
 - ▶ cardiomegaly
- ECHO –
 - ▶ LVEF - impairment: 40-50% = mild, 30-40% = mod, <30% = severe
 - ▶ valve function,
 - ▶ diastolic dysfunction,
 - ▶ RWMA
- CORONARY ANGIOGRAM – if ischaemic cardiomyopathy cause ?candidate for revascularization procedure

MANAGEMENT

- treat cause
- O2
- Medical Rx:
 - ▶ diuretics & aldosterone antagonists
 - ▶ vasodilators: ACE-I & A2RBs or hydralazine + nitrates if intolerant of ACEIs
 - ▶ digoxin
 - ▶ beta-blockers
 - ▶ anti-coagulation if arrhythmia, a LV aneurysm or low EF
 - ▶ anti-HTNs
- biventricular pacing = cardiac resynchronisation therapy
- insitu ICD
- LVADs - bridging

Intraoperative

- lines
- fluid management
- ensure have had all medications - can give nitrates transdermally
- local or regional techniques if possible
- continue dig - if needed can convert to IV (but unusual to require)
- restart ACEIs asap post op (if withheld for >3days then restart at lower dose)
- CVS goals:
 - ▶ maintain preload
 - ▶ minimise -ve inotropy, ↑HR
 - ▶ avoid diastolic ↓pressure and systolic ↑pressure
- useful drugs:
 - ▶ milrinone = 50mcg/kg load then 0.25-0.5 mcg/kg/min
 - ▶ dobutamine 2-20mcg/kg/min

Post-operative

- monitor for overload, MI's and arrhythmias

Hypertension

Preoperative

= systolic BP > 140 and/or diastolic BP > 90 mmHg

- problem = the greater the BP the greater the risk of vascular morbidity & patients who are moderately hypertensive are more labile under anaesthesia.

- primary HTN

- secondary HTN:

- pheochromocytoma
- hyperaldosteronism
- renal parenchyma HTN
- renovascular HTN

- If evidence of end-organ damage from hypertension -> hypertension contributes significantly to bad outcomes.

- there are multiple factors involved in BP control:

- neurogenic control
- rennin-angiotensin-aldosterone
- ANP
- Eicosanoids
- Kallikrein-kinin system
- endothelial mechanisms (NO)
- adrenal steroids
- renomedullary renodepression
- sodium and water excretion

HISTORY

- symptomatic or asymptomatic

- if symptomatic – headache, blurred vision, ringing in ears, blackouts

- assess degree of end-organ disease:

- IHD – chest pain, SOB
- Arrhythmias - palpitations, collapses
- Heart failure - orthopnoea, PND, ankle swelling
- Renal failure – passing urine, fatigue, weight gain, creatinine
- SAH/CVA's - weakness, numbness, balance problems, headache, swallow
- Retinal damage – vision, floaters

- associations: adrenal tumour, renal disease, renal artery stenosis, coarctation, acromegaly, myxoedema

- risk factors: alcohol, obesity, smoking, poor diet, lack of exercise

- medications: diuretics, Ca²⁺ channel blockers, ACE-I, beta-blockers, nitrates

- complications: PVD, visual problems, strokes, aneurysms

EXAMINATION

- CVS – perfusion, P, radio-femoral delay (coarctation), BP in both arms (dissection and coarctation), JVP (signs of failure), HS, murmurs, apex position and right ventricular heave, oedema, liver span (failure), pedal pulses as a measure of PVD

- RESP – RR, tracheal position, chest sounds – crackles at bases (failure), SpO₂ on RA, thyroid examination

- ABDO – AAA present, renal bruits (renal artery stenosis), kidney size (polycystic kidneys as a cause of renal failure)

- NEURO – long tract signs of CVA, papilloedema, retinal vein pulsations and flame haemorrhages

INVESTIGATIONS

- FBC – anaemia in CRF
- U+E – U, K and Cr in CRF
- BSL
- LFT's – hepato-renal syndromes
- Ca, PO – renal failure
- TFTs – r/o hyperthyroidism
- ECG – LVH, dynamic ischaemic changes
- CXR – notching of ribs (coarctation), cardiomegaly (failure), widened mediastinum (dissection), interstitial change (hepato-pulmonary syndrome)
- ABG – hypoxaemia and hypercarbia can produce hypertension
- Renal U/S – blood flow to the kidneys + anatomy + rule out chronic post-obstructive cause of renal failure, rule out AAA
- urinalysis – blood, protein
- 24 hour urine – metadrenaline and normetadrenaline (r/o pheochromocytoma)

MANAGEMENT

- primary or secondary?
- categorise:
 - ▶ Level 1 – up to 160/100
 - ▶ Level 2 – up to 180/110
 - ▶ Level 3 – > 180/110
- levels 1 and 2 with no signs of organ dysfunction -> low risk category (truck on)
- level 3 with no signs of organ dysfunction -> intermediate risk (truck on, but use ↑ed monitoring if **cannot** reasonably delay)
- level 3 with end organ dysfunction -> high risk group (hold you horses and sort out BP)
- evidence of end-organ damage?
 - ▶ IHD,
 - ▶ CVA,
 - ▶ renal dysfunction,
 - ▶ LVH,
 - ▶ heart failure
- length of delay not EBM but suggest 4-6 weeks to allow reset of cerebral autoregulation
- take multiple readings - single number in clinic should not be used to risk stratify
- contact GP to see whether they are controlled
- continue anti-BP medications
- maintain the BP the patient is used to (within 20%)

Intraoperative

- invasive monitoring
- aggressive maintenance of normal perfusion pressure
- consider epidurals & clonidine - but must be careful not to cause ↓bp

Postoperative

- standard

Pulmonary Hypertension

- Pulmonary Hypertension (PHT) = disorder in pulmonary vascular bed -> obstruction to blood flow

- significant impact on mortality

Primary PHT = relatively rare (cause unknown)

Secondary PHT =

- Resp cause - chronic hypoxia
- veno-occlusive disease
- L side heart problems eg valvulopathy
- Intracardiac shunts

- no cure besides treating precipitant
- high risk of morbidity and mortality in the perioperative period

Pathophysiology

Cardiac causes – LA or LV disease -> ↑ LA pressure -> ↑ pulmonary venous pressure -> ↑ PAP -> ↑PVR

Respiratory causes – hypoxic vasoconstriction -> PHT

Which leads to:

- vasoconstriction
- altered vascular endothelium and smooth muscle function
- cellular remodelling
- increased vascular contractility
- lack of relaxation in response to various endogenous vasodilators
- fibrosis of vascular tissue

Diagnosis

- mean pulmonary artery pressure > 25mmHg @ rest and > 30mmHg with exercise
- measured by catheterisation (gold standard)
- CXR sign:
 - pulmonary artery 16mm R hilum
 - pruning central vessels, sparing peripherally

Severity

- Mean pressure measurement (measured at rest):
 - Mild = 25-40mmHg
 - Moderate = 41-55mmHg
 - Severe = > 55mmHg

	Mean	SBP	DBP
Mild	25-40	30-50	20-25
Mod	40-55	50-70	25-35
Severe	>55	70-110	35-45

- Functional assessment (NYHA):
 - I – no limitation on physical activity (no SOB, fatigue, chest pain or syncope)
 - II – minimal limitation of physical activity (comfortable @ rest, but develop symptoms on normal physical activity)
 - III – marked limitation of physical activity (comfortable @ rest but symptoms on less than normal activity)
 - IV – unable to perform any physical activity, RHF, symptoms @ rest

Preoperative

- significant morbidity + mortality with surgery

History

- progressive SOB
- fatigue
- chest pain
- syncope

Examination

- right heart failure – increased JVP, (large V waves), parasternal heave, TR murmur, hepatomegaly, peripheral oedema
- other signs of CHF
- loud P2
- hypoxaemia

Investigations

Minimum:

- ECG – RVH, RAD, right ventricular strain
- CXR – RVH on lateral (loss of retrosternal space), prominent pulmonary vascular
- ECHO –
 - RVH,
 - estimation of PAP:
 - use modified Bernoulli equation to derive RVSP from TR jet
 $\hookrightarrow = 4 \times \text{peak pressure squared}$
 - $\text{PAP} = \text{RVSP} + \text{RAP}$
 - NB RAP unknown and difficult to derive from CVP
- lung function

As indicated:

- ABG
- V/Q scanning
- high resolution CT
- serology testing
- LFT's
- Preoperative therapy to ↓pHTN
 - O2
 - bronchodilators
 - vasodilators
 - inotropes
 - MDT co-ordination (physio's, respiratory nurses, physicians)
 - continue any medical treatment of PHT up until surgery
 - if patient newly diagnosed and presenting for emergency treatment -> sildenafil 50-100mg PO preoperatively

Intraoperative management

- at risk of dramatic haemodynamic changes during induction and positive pressure ventilation

GOALS:

1. optimize PAP
2. optimise RV preload
3. avoid RV ischaemia and failure

Practical Strategies

- choice of anaesthetic doesn't matter -> need to adhere to the goals
- Art Line
- PAC
- CVL
- avoid or treat arrhythmias aggressively (need atrial contraction for adequate preload and cardiac output)
- TOE
- avoid hypoxia (increases PVR)

- controlled ventilation
- high dose narcotics
- minimise volatiles -> decrease SVR, myocardial contractility + potential arrhythmias -> all impair myocardial perfusion and right ventricular cardiac output
- 100% O₂ (decreased PVR)
- aggressively treat hypercarbia, acidosis, hypothermia (all increase PVR)
- use ketamine and N₂O cautiously (increase PVR)
- epidurals should be topped up slowly
- vasopressin + dobutamine will not directly ↑PVR
 - ↳ standard α agonists eg phenylephrine or norad ⇒ ↑PVR

Treatment of Intraoperative PHT

- presents as RV failure and systemic hypotension
- **inhaled nitric oxide** 20-40ppm (good in bypass, doesn't cause systemic hypotension as inactivated when bound to Hb)
- **milrinone** (50mcg/kg bolus -> 0.25 - 0.5 mcg/kg/min)
- **dipyridamole** (0.2-0.6mg/kg IV over 15min bd)
- **inhaled prostacyclin (iloprost)** (increases cAMP) – 50mcg in saline nebulised every hour OR 50ng/kg/min nebulised into inspiratory limb
- **IV prostacyclin** (if inhaled not available) – 4-10ng/kg/min
- **RV assist device**

Factors that have been shown to ↑mortality or morbidity perioperatively:

- RAD
- RVH
- RVSP/SBP ratio > 0.6
- adrenaline or dopamine use
- NYHA class II
- history of PE
- OSA
- high risk surgery
- OT > 3 hours

Post operative management

- ICU admission
- as anaesthetic agents wear off -> monitor of acute PHT -> RV ischaemia
- monitor for: heart failure, arrhythmias, MI

Valvular Heart Disease

[if symptomatic should have valve surgery prior to elective surgery]

[severe AS (even asymptomatic) - should consider balloon valvuloplasty if urgent]

- 4% >65 have valvular heart disease
- indwelling prosthetic valves:
 - ▶ tissue valves require no ongoing anticoagulation
 - ▶ mechanical valves need lifelong anticoag
 - ▶ VTE risk depends on site & type of valve
 - modern bileaflet AVRs:
 - <4% per year risk
 - stop warf for 5d & bridge with low dose (prophylactic) LMWH
 - old AVRs & any mechanical MVR:
 - >4% per yr risk
 - stop warf 5d & bridge with high dose (treatment) LMWH. Consider UFH periop (stop 6hrs prior to surg)

Levine Murmur Classification

[>3 suggest haemodynamic consequences]

- 1 = careful listening for sometime
- 2 = faint but able to hear straight away
- 3 = loud but no thrill
- 4 = loud with thrill
- 5 = (as 4) & can hear with rim of stethoscope
- 6 = (as 4) & can hear without a stethoscope

Undiagnosed Murmurs

- most murmurs do not = disease
- assess
 - ▶ functional capacity ie METS
 - ▶ symptoms
 - ↳ if good capacity & asymptomatic = low risk for surgery esp if not elderly
- elderly aortic systolic murmurs:
 - ▶ sclerosis of aortic leaflets - 15% go onto develop AS within 7yrs
 - ▶ differentiate sclerosis from stenosis:
 - >4METS
 - asymptomatic: no angina/SOB/syncope
 - ECG: no LVH or strain
 - normal pulse pressure (no slow rising pulse)
 - ↳ note volume of murmur does not help
 - ▶ proceed on basis of
 - factors above:
 - all positive then ⇒ proceed no probs
 - any negatives then ⇒ ECHO
 - surgery grade:
 - mild/intermediate = fine
 - major = ECHO

Aortic Stenosis

Preoperative

- congenital (bicuspid 2%), calcification (age) or rheumatic (co-existing mitral disease)
- concentric hypertrophy of LV with decreased compliance -> high risk of myocardial ischaemia
- atrial contraction can contribute up to 40% of preload (normally 20-30%)
- 30% with AS will have normal coronaries & angina due to LV hypertrophy ⇒ subendocardial ischaemia
- >50% mortality in 3yrs if symptomatic AS

Table 1 Grading of AS in adults. BSA, body surface area

	Normal	Mild	Moderate	Severe
Aortic jet velocity (m s^{-1})	<2	<3	3–4	>4
Peak gradient (mm Hg)	<10	<40	40–65	>65
Mean gradient (mm Hg)	<5	<25	25–40	>40
Valve area (cm^2)	3–4	>1.5	1.0–1.5	<1.0
Valve area indexed ($\text{cm}^2 \text{ m}^{-2} \text{ BSA}$)		>0.85	0.6–0.85	<0.6

HISTORY

- angina
- SOB
- syncope
- symptoms don't correlate well with valve area!
- heart failure symptoms
- IHD risk factors

EXAMINATION

- slow rising pulse
- narrow pulse pressure
- ESM loudest in aortic area -> neck
- soft A2, S4
- signs of LVH and LVF eg heave

INVESTIGATIONS

- ECG: LVH + ST changes (strain ie 2 leads of ST-T wave abnormalities)
- CXR: normal unless LV begins to fail, post stenotic dilatation of aorta, calcified annulus
- ECHO:
 - gradient and LV function
 - normal LVEDP may reflect failing LV or hypovolaemia
- ANGIO: estimate gradient across valve + any concurrent coronary disease

MANAGEMENT

- symptomatic patients -> replace
- asymptomatic patients with peak gradient >50mmHg -> replace before major elective surgery,
 - ↳ crack on for intermediate or minor surgery with caution

Intraoperative

- low heart rate
- maintain SR
- full
- high normal SVR (phenylephrine or metaraminol)
 - ↳ will not ↑CO but aiming to ↑coronary perfusion
- strict 10-20% of normal MAP
- invasive monitoring
- balanced induction
- treat arrhythmias aggressively
 - electrolytes
 - cardioversion
 - amiodarone
- good analgesia (caution with neuroaxial anaesthesia c/o hypotension)
- Peripheral regional ideal

Postoperative

- HDU/ICU
- careful fluid balance
- pressors as required

Aortic Regurgitation

Preoperative

- rheumatic, endocarditis, Marfans, syphilis, ankylosing spondylitis, myxomatous
- eccentric hypertrophy
- normally chronically develops
- acute AR -> LVF and APO

HISTORY

- SOB
- palps

EXAMINATION

- wide pulse pressure
- collapsing pulse
- visible neck pulsation (Corrigan's sign)
- head nodding (De Musset sign)
- water hammer pulse
- capillary pulsations in nail bed (Quincke's sign)
- diastolic murmur in aortic area (increased in expiration)
 - ↳ Austin Flint murmur ⇒ low pitched rumbling mid to late diastole

INVESTIGATIONS

- CXR: cardiomegaly, boot shaped heart
- ECG; non specific LVH
- ECHO: quantification of regurgitation

MANAGEMENT

- asymptomatic patients -> tolerate surgery well
- symptomatic patients -> assess for AVR

Intraoperative

- "fast, full and forward"
- high normal heart rate (90/min)
- adequate volume loading
- low SVR
- maintain contractility
- epidural and spinal tolerated well
- treat arrhythmias promptly (cardioversion)
- invasive monitoring

Postoperative

- routine
- watch for pulmon oedema when neuraxial block wears off

Mitral Stenosis

- causes:
 - rheumatic
 - congenital

- myxomatous
- post MV repair
- most have mixed disease (MS and MR)
- LV normal but small and under filled
- LA dilation -> PA dilation -> PHT -> RVH -> RVF -> PR and TR
- poor tolerance to ↑HR due to ↓LV filling time

Preoperative

HISTORY

- SOB:
 - fluid transudate in lungs ⇒ ↓compliance & ↑WOB
 - pulmon oedema
- haemoptysis
- recurrent bronchitis
- fatigue
- palpitations (AF)

EXAMINATION

- mitral facies (malar flush on cheeks)
- peripheral cyanosis
- right heart failure (elevated JVP, hepatomegaly, oedema, ascites)
- heave
- mid-diastolic murmur loudest in mitral area
- signs RVF:
 - loud P2
 - opening snap
 - tapping apex

INVESTIGATIONS

- ECG: p-mitrale, AF
 - RV strain = ST depression or T inversion in V1-V4, II, III, aVF
 - RVH signs =
 - RAD
 - positive R wave V1-3 (norm R wave is to be equiphase in V3)
 - dominant S wave V5 - V6
 - ↳ (& narrow QRS ie not RBBB)
- CXR: valve calcification, large LA, double shadow behind heart on PA, splaying of carina, LVF signs
- ECHO: measures gradient and valve area
- numbers:
 - valve areas similar to AS (normal is a bit bigger) 4...2...1

	Norm	Mild	Mod	Severe
Mean Gradient		<5	5-10	>10
Valve Area cm ²	4-6	>1.5	1.5-1	<1
Peak Pressure		<30	30-50	>50

MANAGEMENT

- symptomatic -> replace
- asymptomatic -> tolerate surgery well

Intraoperative

- similar to AS management
- low normal HR (50-70/min)
- maintain SR if possible:

- if intraop AF should consider avoiding DC cardioversion if possible due to high risk of pre-existing atrial thrombus even when acute arrhythmia \Rightarrow stroke
- adequate preload
- high normal SVR
- avoid increases in PVR (hypercarbia, acidosis, hypoxia)
- invasive monitoring
- avoid spinal/epidurals

Mitral Regurgitation

Preoperative

- endocarditis, rheumatic fever, MVP, acute MI with papillary muscle rupture or LVF
- MR \rightarrow PHT \rightarrow RHF \rightarrow PR + TR
- LVEF becomes supranormal - up to 50% ejected through MV to LA prior to AV opening

HISTORY

- fatigue
- weakness
- SOB

EXAMINATION

- displaced forceful apex + heave
- soft S1, loud S3
- pansystolic murmur \rightarrow axilla
- AF
- heart failure signs

INVESTIGATIONS

- ECG: LA enlargement, AF
- CXR: LA and LV enlargement, mitral annular calcification
- ECHO: degree of regurgitation

MANAGEMENT

- asymptomatic \rightarrow crack on
- symptomatic \rightarrow assess for valve surgery

Intraoperative

- similar to AR
- high normal HR
- adequate preload
- low SVR
- low PVR (avoid hypercarbia, hypoxia, acidosis, high inspiratory pressures)

Postoperative

- routine

Mitral Valve Prolapse

- common - incidental in 5% of population
- generally asymptomatic but \Rightarrow chest pain, palpitations, syncope, emboli
- mid systolic click & late diastolic murmur
- ECHO diagnostic
- anti-arrhythmics impt to continue

Pericardial Disease

Acute Pericarditis

HISTORY

- chest pain that is worsened by leaning forward
- viral infection
- can produce myocarditis

EXAMINATION

- rub

INVESTIGATIONS

- ECG: widespread ST elevation that doesn't fit into a vascular distribution, arrhythmias

MANAGEMENT

- post-pone any elective surgery 6 weeks
- supportive care
- ?steroids

Constrictive Pericarditis

Preoperative

- post infective or secondary to a autoimmune condition (ie. SLE)
- respond well to pericardectomy but can be v high risk operation depending on chronicity
 - ↳ chronic constriction ⇒ severe diastolic dysfunction

HISTORY

- fatigue
- decreased ET
- SOB

EXAMINATION

- pulsus paradoxus = ↑ed fall compared to normal in SBP with inspiration (>10mmHg)

INVESTIGATION

- ECHO:
 - systolic function preserved
 - ↓↓diastolic function

Intraoperative

- avoid ↓ & ↑ HR ie keep normal 60-80
- keep full
- keep intrathoracic pressure low (keep SV if possible)
- inotrope ⇒ milrinone ideal as ↓SVR & ↑contractility
- neuraxial/regional ideal

Cardiomyopathy

- most will have little reserve for surgery/anaesthesia

Hypertrophic Cardiomyopathy

- difficult diagnosis to make without ECHO: if intra-op hypotension and things getting worse despite standard therapy think SAM

Preoperative

- aetiology unknown (>50% autosomal dominant condition)
- hypertrophied septum causes dynamic outflow tract obstruction during systole

- also can get dynamic anterior motion of the mitral valve leaflet towards septum (SAM – systolic anterior motion) -> causing further obstruction
 - ↳ to diagnose: if all things you would normally do for ↓bp is making bp worse ≈ SAM
- this causes a pressure overload of LV and diastolic dysfunction

HISTORY

- SOB
- fatigue
- angina
- palpitations
- sudden death

EXAMINATION

INVESTIGATION

- ECG: LVH
- ECHO: functional obstruction, concentric or asymmetrical hypertrophy of LV, SAM, MR
- angio - abnormal coronary anatomy

MANAGEMENT

- monitoring -> myomectomy

Intraoperative

- goal = maintain a large, slow ventricle
- low normal HR
- adequate volume loading
- high normal SVR
- low ventricular contractility
- avoid inotropes
- use: betablockers, calcium channel blockers
- aggressive management of arrhythmias (may require pacing)
- direct vasopressors
- invasive monitoring

Dilated Cardiomyopathy

Causes

- idiopathic, alcoholic, haemochromatosis, post viral, peri-partum, IVDU, myotonic dystrophies

Preoperative

- cardiac failure + an enlarged poorly contractile heart
- progressive ventricular dilatation -> increased LV end-diastolic volume -> functional MR and TR

HISTORY

- heart failure symptoms
- palpitations (arrhythmias)
- CVA (embolic)

EXAMINATION

- heart failure signs
- neurological examination
- murmurs

INVESTIGATIONS

- FBC: anaemia/polycythaemia
- U+E: electrolyte abnormalities, renal function
- ECG: arrhythmia, conduction abnormalities, ischaemia
- ECHO: LV function, valve pathology
- CXR: signs of LVF

MANAGEMENT

- diuretics
- ACE I
- nitrates
- amiodarone (arrhythmias)
- digoxin (arrhythmias and +ve inotropy)
- beta-blockers (carvedilol)
- anticoagulation
- dual chamber pacing +/- AICD

Intraoperative

- invasive monitoring including cardiac output monitor
- maintain SR
- adequate volume
- normal SVR
- avoid myocardial depression
- inotropes often required (dobutamine or milrinone)

Postoperative

- HDU/ICU monitoring

Restrictive Cardiomyopathy

Preoperative

- rare condition
- R heart failure often prominent
- amyloid infiltration of heart -> stiff ventricle + diastolic dysfunction

HISTORY

- SOB
- fatigue

EXAMINATION

- heart failure (especially RHF)

INVESTIGATIONS

- ECHO: diastolic dysfunction

Intraoperative

- high risk anaesthesia
- maintain SR
- adequate volume loading
- high normal SVR
- avoid myocardial depression
- peripheral vasodilation, myocardial depression, decreased VR \Rightarrow cardiovascular collapse
- SV (if possible)
- ketamine a good agent (increases contractility + peripheral resistance)

Heart Transplant Patient

Criteria for Cardiac Transplant (NZ)

- estimated life expectancy of > 12 months
- age < 60 yrs
- normal or reversible renal and hepatic function
- no infection

- no pulmonary infarction (in preceeding 8 weeks)
- no IDDM
- psychosocial and social support
- low PVR
- no CVA or PVD
- no drug abuse
- no pre-existing malignant disease
- no suppurative lung disease

Preoperative Assessment

HISTORY

- cause of cardiac failure and symptoms patients had, prior functional limitations
- surgery and perioperative course
- complications: rejection, infection, coronary vascular disease, malignancy, hyperlipidaemia and HTN
- currently exercise tolerance
- previous anaesthetic history after transplant
- symptoms of rejection and cardiac performance
 - symptoms of rejection=
 - unexplained weight gain
 - unexplained fever
 - recent cardiac biopsy result – Bilingham grade
- symptoms of heart failure seen or arrhythmia
- donor heart IHD – don't get pain because heart denervated
- ability to work

Medications:

- standard + immunosuppressants

3 classes of drugs used:

1. **Immunophilin binding drugs** (cyclosporin, tacrolimus) – prevent cytokine-mediated T cell activation and proliferation
2. **Nucleic acid synthesis inhibitors** (azathioprine) – block lymphocyte proliferation
3. **Steroids** (prednisone) – block production of inflammatory cytokines, lyse T lymphocytes and alter the function of remaining lymphocytes.

- anaemia, thrombocytopaenia and leukopaenia -> may require treatment
- all can predispose to; infection, malignancy (SCC of skin, lymphoma), OA, CRF
- cyclosporine – associated with HT, renal dysfunction, prolonged effect of NDMBD, calcium antagonists are used to increase cyclosporine levels
- tacrolimus – renal dysfunction
- recent biopsy and angiography results

EXAMINATION

- weight
- BP
- CVS: high HR, no variation, pacemaker, sternotomy, scars over RIJ from biopsies, HS and lungs

INVESTIGATIONS

- ECG: look for second (native p wave), RBBB
- angio
- ECHO: intramural thrombi and ventricular function
- FBC
- U+E
- drug levels
- CXR

MANAGEMENT

- discuss with cardiology regarding optimisation

Intraoperative Care

- strict asepsis
- don't cannulate RIJ as this is where cardiac biopsies are taken from
- no evidence between one technique and another
- be prepared to support BP aggressively if you use a spinal
- heart is denervated (HR around 90/min)
- some patient may have pacemaker (bradyarrhythmias)
- normal autonomic reflexes lost (beat-to-beat variation, Valsava) -> doesn't tolerate acute hypovolaemia well (maintain preload)
- contractility preserved (unless rejection taking place)
- try not to intubate if possible as have a tracheal anastomosis
 - ↳ if you have to: use short tube and check cuff pressure
- use direct acting agents: adrenaline, noradrenaline, isoprenaline and beta-blockers, phenylephrine (doesn't produce reflex bradycardia)
- aggressive maintenance of coronary perfusion pressure because of atherosclerotic burden

Postoperative Care

- cough may be impaired from phrenic and recurrent laryngeal palsies -> increased risk of sputum retention and aspiration
- vigilance regarding screening for infection
- if patient develops severe systemic infection may need increasing of prednisone
- vigilance in screening for cardiac rejection

Congenital Heart Disease

- 8:1000
- over 100 types
- most =

1. ASD
2. VSD
3. PDA
4. TOF
5. PS
6. AS
7. Coarctation of Aorta
8. Transposition of great vessels

- most of rest are palliatively managed by producing a Fontan Circulation

Classification

- **Acyanotic** – VSD, ASD, atrioventricular septal defect, PDA, pulmonary stenosis, coarctation, aortic stenosis
- **Cyanotic** – TOF, transposition of great vessels, hypoplastic left heart, pulmonary atresia, Eisenmengers syndrome (later in life)

Operations

ASD repair

- secundum:
 - ▶ often asymptomatic with L to R shunt
 - ▶ percutaneously or open closure
 - ▶ danger of paradoxical emboli
- primum type:

- endocardial cushion defect ∴ may involve AV valves
- can ⇒ severe heart failure from left to right shunt depending on size and location
- type with Downs incl AVSD ⇒ severe pHTN
- surg repair may ⇒ complete heart block

VSD repair

- commonest form of CHD
- clinical effect depends on size and number
- Rx:
 - prevent air embolism,
 - avoid fluid overload,
 - intubate
 - avoid ↑L to R shunt: avoid hyperventilation & high FiO₂
 - keep SVR and PVR normal,
 - give antibiotics
- if multiple VSD present may required PA banding to protect pulmonary circulation
 - with ↑ing age band ⇒ tightens ⇒ cyanosis ∴ removed when older
 - VSDs often close spontaneously ∴ also removed then
- surgical closure and percutaneous closure options (both are associated with problems with ventricular arrhythmias post op)

PDA

- moderate left to right shunt -> elevation of PVR
- can be closed surgically or using transcatheter approach

Blalock-Taussig shunt placement (stage 1 Fontan)

- palliative care procedure where subclavian artery connected to the pulmonary artery

Bidirectional Glenn Shunt (stage 2 Fontan)

- for hypoplastic left heart syndrome where SVC is anastomosed to the PA (usually used as a staging procedure to a Fontans circulation)

Fontan circulation Completion (stage 3 Fontan)

- palliative care procedure
- group of operations that separates the pulmonary and systemic circulations in patients with a single ventricle
- vena caval blood directly into pulmonary circulation (bypassing right side of heart)
- performed for tricuspid atresia and hypoplastic left heart syndrome
- pulmonary blood flow dependent on systemic venous pressure
- SpO₂ usually normal
- long term effects =
 - elevated systemic venous pressures,
 - liver congestion,
 - protein-losing enteropathy,
 - fluid overload,
 - ascites, pleural and pericardial effusions
- patients anticoagulated
- IPPV can cause a fall in Q
- normovolaemia important

Tetralogy of Fallot Repair:

- pulmonary stenosis, VSD, overriding aorta & RVH
- prior to repair may be treated with
 - beta-blocker or
 - Blalock-Taussig shunt (subclavian to PA)
- ratio of SVR:PVR determines both systemic blood flow and SpO₂
- repaired @ 3-8 months of age
- during anaesthesia:
 - intubate,
 - maintain low PVR,
 - treat cyanosis with:

- hyperventilation,
- IV fluid
- phenylephrine

Eisenmenger's Syndrome

- = high pulmonary vascular resistance with reversed or bidirectional shunt flow
 - ↳ ie L to R flow until \uparrow pHTN becomes higher than SVR \Rightarrow R to L flow \Rightarrow cyanosis
- once irreversible pHTN developed then surgical correction not possible
- associated with significant morbidity and mortality
- can be caused by a number of defects
- definitive treatment = to close defect
- goal when managing is to avoid:
 - ▶ \downarrow SVR
 - ▶ \uparrow PVR
 ↳ otherwise \Rightarrow \uparrow right to left shunt \Rightarrow \uparrow ing cyanosis \Rightarrow death
- increasing SVR or decreasing PVR will \Rightarrow improved SpO₂
- arrhythmias, hypovolaemia and large fluid shifts not tolerated well
- no air bubbles
- invasive monitoring with anticipation and treatment of haemodynamic changes
- treat cyanosis as ToF
- inotropes often used
- ICU post op
- best managed in tertiary level unit

Preoperative

[any abnormal structure or physiology then refer/discuss with tertiary centre]

History

- diagnosis (when, how, age, severity)
- treatment (corrective, palliative or untreated)
- symptoms (cyanosis, heart failure symptoms, pulmonary hypertension symptoms)
- functional capacity
- history of stroke or thrombosis (hyperviscosity)
- often anticoagulated

Examination

- SpO₂
- cyanosis
- CHF signs
- neurological signs

Investigations

- FBC: HCT (high), platelets (low)
- U+E: renal dysfunction from chronic hypoxia
- Coags: coagulation factor deficiencies common in cyanotic heart disease
- ECHO: diastolic dysfunction, decreased ejection fraction, nature and size of lesion, flow reversal
- ECG
- PFT

Factors associated with High Risk

- recent CCF
- recent myocardial ischaemia
- severe hypoxaemia (SpO₂ <75% on RA)
- polycythaemia (Hct >60%)
- unexplained syncope, fever or CVA
- severe aortic/pulmonary stenosis
- uncorrected TOF or Eisenmengers syndrome

- recent onset of arrhythmias

Management

- identification of seriously effected patients with transfer to regional unit
- consult with patients cardiologist
- consider appropriate inotropes or vasodilators
- assess requirement for post operative HDU/ICU
- premedication (decrease O₂ consumption and sympathetic tone)

Intraoperative

- gain an understanding of the anatomy and physiology of defect
- all IV induction agent safe (rate of delivery and dose important not actual drug)
 - ↳ ketamine traditionally useful to maintain balanced resistance but will ↑PVR & SVR in isolation via ↑SNS output
- good analgesia (decrease sympathetic activation)
- 100% FiO₂ can be used in simple cardiac anomalies with left to right shunt (but caution with complex lesions with left to right shunt)
- controlled ventilation
- invasive monitoring
- TOE helpful (PAC not so much)
- capnography underestimates in patient with right to left shunt
- pulmonary hypertension management (to decrease PVR; increase FiO₂, hypocarbia, akalaemia, SV, normal lung volumes, avoid sympathetic stimulation, isoprenaline, milrinone, prostaglandins, NO, sildenafil)
- endocarditis; see guidelines below
- no air in lines

Postoperative

- HDU/ICU
- management of pulmonary hypertension and systemic haemodynamics
- hypoxaemia; from either:
 - inadequate pulmonary blood flow (avoid dehydration, maintain SVR, control PVR, minimise oxygen consumption)
 - pulmonary hyperperfusion (minimise cardiac work)
- HDU/ICU

Approach to Adults with CHD

- discuss anything not straightforward with cardiac centre
- Uncorrected:
 - VSDs/ASDs: small defects present no major issues (except paradoxical emboli)
 - Large L to R shunt ⇒ pHTN ⇒ shunt reversal ie Eisenmengers Syndrome ∴ very high risk
- Corrected disease:
 - Rx as normal
 - exclude assoc congen abnormalities
- Palliated disease:
 - normal anatomy not restored
 - eg Neonatal switch for transposition of great vessels, Fontan for HLHS or pumon atresia
 - T/F to specialist centre
 - Fontan:
 - high CVP ⇒ ↑risk of bleeding from eg T&As or nasal intubation
 - must avoid ↑pHTN

Infective Endocarditis

General

- = infective inflammation of the endocardial lining of the heart and valve
- most commonly effecting the AV (used to be the MV)

Pathophysiology

- originates at site of endothelium damage
- sterile thrombotic vegetation \Rightarrow facilitates bacterial adherence during transient bacteraemia
- platelets & fibrin deposit \Rightarrow +ve cycle
- destructive effects follow on

Classification

By duration

- Acute
 - Fulminant illness over days to weeks
 - More likely due to *S. aureus*
- Subacute
 - Often due to streptococci of low virulence and mild to moderate illness which progresses slowly over weeks and months
- By culture results :
 - Culture-positive
 - Culture-negative
 - Common cause is prior antibiotic administration
- By heart side
 - Right side – 10% usually IVDU
 - ↳ 98% tricuspid valve
 - Left side – patient without IV drug exposures
- By valve type
 - Native-valve endocarditis
 - Prosthetic-valve endocarditis
 - Early (<60 days)
 - Intermediate (60d-1year)
 - Late (>1year)

Duke

- Diagnosis by Duke criteria (2 major or 1 major 3 minor or 5 minor)
 - ↳ low sensitivity & cannot be used if culture -ve or if IE of foreign implant or R side of heart
- Major
 - Positive blood culture with typical organisms
 - Evidence of endocardial involvement with positive echocardiogram
- Minor
 - Predisposing factor: known cardiac lesion, recreational drugs injection
 - Fever >38
 - Evidence of embolism
 - Immunological problems: glomerulonephritis, osler's nodes
 - Positive blood culture (atypical)

History

- nil -> malaise, night sweats, anaemia, weight loss -> crashing cardiogenic shock and sepsis
- haematuria
- CVA

Risk Factors

- rheumatic heart disease
- prosthetic valves
- congenital heart disease (trileaflet MV)
- IVDU
- previous IE
- high risk surgery (dental, respiratory and infective)
- BUT, 50% in normal valves!

Examination

- heart murmurs
- cardiac failure
- valve lesions
- CVA
- splenomegaly
- peripheral stigmata: clubbing, splinter haemorrhages, Osler's nodes, vasculitis rash, retinal changes

Investigations

Organisms

- Majority are gram +ves:
- Strep viridans (~25%)
- Staph aureus (~50)
- Strep pneumoniae
- fungi

- blood cultures
 - +ve in 80%
 - 3 sets from different sites ideal - but volume also impt
- serology
- ECG: look for widening PR interval, p mitrale, TWI
- ECHO: vegetations and valve incompetence
 - TTE - 45-60% sensitivity
 - TOE -
 - 90-100% sensitivity
 - must perform if suspect paravalvular complications eg pt is shocked
 - findings suggestive of IE:
 - vegetation - size >10mm predicts ↑risk of embolic complication
 - peri-annular abscess - TTE esp poor at identifying
 - new dehiscence of valvular prosthesis

Management

- IV antibiotics
 - 4-6 weeks
 - seek advice from microbiologist - common empirical = flucloxacillin & gent
 - curative in 50% of patients
- valve replacement
- prophylaxis

Complications

- embolic (brain, lungs, limbs, organs)
- sepsis (abscess formation)
- death
- valve incompetence
- arrhythmias

Infective Endocarditis Prophylaxis

- more conservative approach as risks of adverse effects from antibiotics higher than risks of developing IE from dental, GI or GU tract procedure
- must have high risk patient and high risk surgery

- high risk patients;

1. any prosthetic material in used in valve repair
2. previous IE
3. congenital heart disease (unrepaired cyanotic, partially repaired, completely repaired within 6 months)

4. cardiac transplant patients with valvulopathy
5. all dental procedures that involve manipulation of gingival tissue or periapical region of teeth or perforation of oral mucosa (thus only check ups and simple fillings that don't involve gingiva don't need antibiotic prophylaxis)
6. Any high risk surgery
 - eg respiratory tract surgery (incision and biopsy, tonsillectomy, adenoidectomy)

Antibiotic Choice

- give a single dose of antibiotic prior to procedure (or within 2 hours of procedure)

1. amoxycillin 50mg/kg PO
2. cephazolin 50mg/kg IV/IM
3. cephalexin 50mg/kg PO (penicillin allergic)
4. clindamycin 20mg/kg PO (penicillin allergic)

- if currently infected with a bug and operation is taking place on that area and the organism is likely to cause IE -> give prophylactic treatment perioperative (consult ID)

Pulmonary Embolism

Diagnosis

- Symptoms:
 - chest pain
 - SOB
- Signs:
 - Resp: ↑RR, haemoptysis, cyanosis
 - CVS: ↑HR, arrhythmias, ↑JVP, ↓bp, collapse, arrest
 - Renal: oliguria
- Investigations:
 - ABG: ↓PaO₂, normal or high PaCO₂, acidosis
 - CXR: prominent PA, oligemic lung fields
 - ECG: Sinus tachy, S1Q3T3, RV strain (normal in 50%)
 - ECHO: RV>LV size
 - VQ scan, CTPA, pulmonary angiography
- Severity Scoring:
 - Minor = <30% obstruction & no RV dysfunction:
 - non specific symptoms
 - anticoag
 - Moderate = 30-50% obstruction, normal bp BUT RV dysfunction
 - ↑HR, ↑RR, haemoptysis
 - thrombolysis
 - Massive = >50% obstruction with severe RV failure +/- haemodynamic collapse:
 - embolectomy or thrombolysis

Long QT Syndrome

= QT > 0.44 seconds -> prolonged ventricular repolarisation -> predisposes malignant ventricular arrhythmias

Causes

- genetic: cardiac ion channel mutation (Na⁺, K⁺)
- acquired: myocardial disease, electrolyte disturbance, drugs, head injury

- myocardial disease: MI, RF, 3rd degree HB, cardiomyopathy
- electrolytes: low Ca²⁺, low K⁺, low Mg²⁺
- drugs: class I and III anti-arrhythmics, phenothiazines, TCA's, SSRI's
- hypothermia

Preoperative

HISTORY

- syncope
- exercised induced syncope
- cardiac arrest
- FHx

EXAMINATION

- may be normal

INVESTIGATIONS

- ECG: QT >440ms
- ECHO: structural heart disease

MANAGEMENT

- beta-blockade
- avoidance of increased sympathetic tone - adequate analgesia/anaesthesia
- cardiac pacing
- treat cause

Intraoperative

- avoid anaesthesia if possible
- full monitoring
- avoid increase in sympathetic tone
- hypothermia cares (can prolong QT)
- MgSO₄, verapamil and phenytoin have been used to treat malignant arrhythmias
↳ Torsades ⇒ MgSO₄ bolus

Drug Concerns

- in essence avoid any drugs which may
 - ▶ ⇒ tachycardia
 - ▶ effect autonomic system
 - ▶ effect cardiac ion channels
- consider checking online resources

Avoid if able	Fine/suggested
glyco/atropine/neo	suggamadex
ketamine	
sux	atrac or roc
volatiles (although sevo only theoretical)	TIVA
sympathomimetics: ephedrine, metaraminol	phenylephrine

Postoperative

- consider HDU/ICU monitoring - ECG monitoring for 24hrs

Rheumatic Fever

Preoperative

CLINICALLY

- autoimmune disease triggered by streptococcus A
- 5-15 years
- 1-3 weeks after a sore throat
- mitral and aortic valve disease

Major Criteria

- carditis
- migratory polyarthritis
- subcutaneous nodules
- erythema marginatum
- chorea

Minor Criteria

- arthralgia
- fever
- previous history of rheumatic fever
- elevated ESR or CRP
- increasing PR interval
- evidence of recent streptococcal A infection (throat swab or titres)

INVESTIGATIONS

- ASOT titres
- ECHO
- CXR

MANAGEMENT

1. antibiotics
2. aspirin & steroids (pain and inflammation)
3. heart failure treatment (digoxin, ACE-I, diuretics, betablockers, O2)
4. antibiotic prophylaxis from IE if indicated
5. chorea (phenobarbitone, haloperidol)

Intraoperative

- management as per specific valve lesion
- consider prophylactic antibiotics (although new AHA/ACC guidelines are more conservative)

Postoperative

- monitoring for IE

ECGs & Arrhythmias

Cardiac Arrhythmias

Classification

- According to site of origin:
 - Supraventricular vs ventricular
 - Narrow vs broad complex
- According to heart rate:
 - Tachy
 - brady

Cardiac Rate

- Normal sinus rhythm – originated in SA node
- Bradycardia –
 - ▶ Causes:
 - Drugs eg β blockers, dig, anticholinesterases, sux
 - MI, sick sinus syndrome
 - \uparrow ICP, \downarrow thyroid, \downarrow temp, vagal
 - ▶ Rx:
 - only correct if haemodynamic compromise - often <40
 - underlying cause - vagal stim
 - Drugs: anticholinergics, isoprenaline, adrenaline
- Tachycardia >100 :
 - ▶ Causes:
 - Inadequate depth anaesthesia, pain
 - fever/sepsis, hypovolaemia, anaemia, heart failure, thyrotoxicosis
 - Drugs eg anticholinergics, catecholamines
 - ▶ Rx: Underlying cause then try β blockers

Delayed After Polarisation

- Caused by inward current assoc with abnormally raised intracellular Ca
- Cause oscillations which ectopic beats

Abnormal pacemakers

- Other parts of conduction system can become pacemakers
- SA node most rapid discharger of conduction system normal pacemaker
- Complete heart block idioventricular rhythm indep of atria
 - due to either:
 - AV nodal block – other AV node takes over $\sim 45/\text{min}$
 - infranodal block in His bundles – new pacemaker 15-35
 ↳ stokes Adams syndrome fainting & cerebral ischaemia
 - caused by:
 - septal MI
 - surgical damage to His
 - congen septal defects

Heart Block

Types:

1st degree – prolongation of PR interval ($>0.2\text{s}$)

2nd degree (Mobitz type I) – progressive lengthening of PR interval with eventual dropped ventricular conduction

2nd degree (Mobitz type II) – intermittent dropping of ventricular conduction with fixed PR interval (can be normal)

3rd degree – complete dissociation between atria and ventricular

RBBB – wide complex QRS & RSR in V1 ('M'), and 'W' in V6 - or just UPRIGHT in V1/2

LBBB – wide complex QRS & septal depolarisation reversed so there is a change in initial direction of QRS (WILLIAM)

Bifascicular block

- RBBB + block of either left anterior or posterior fascicle:
 - RBBB + Left axis Deviation = left anterior fascicle block (more common)
 - RBBB + Right axis deviation = left posterior fascicle block

Trifascicular block – RBBB + R or L axis deviation + prolonged PR interval

Preoperative Management

- 1st degree - nothing unless symptomatic
- 2nd degree (Mobitz type I) – nothing unless symptomatic
- 2nd degree (Mobitz type II) - pacemaker
- 3rd degree – pacemaker
- isolated/bifascicular blocks – nothing unless progresses or symptomatic -> pacemaker
- trifascicular block = very concerning - risk of total heart block intraop ⇒ severe bradycardia - thus must have intraop plan:
 - pharmacological
 - external pacing
 - internal pacing
 - cardiology consult if elective procedure
- ∴ indications for pre-op pacemaker=
 - any symptomatic block of any kind
 - 2nd deg type II, & 3rd block - even asymptomatic
 - symptomatic sinus node disease
- may need temporary pacing wire or external pacing:
- also if rates too slow and unresponsive to drugs -> pace

Intraoperative

- check blood pressure
- atropine 20mcg/kg
- glycopyrulate 5mcg/kg
- isoprenaline 1-10mcg/min
- adrenaline 0.1-1.0mcg/kg/min
- temporary pacing – transthoracic, transoesophageal, transvenous

Ectopic Foci of Excitation

- Can be encouraged by:
 - ↑symp ns activity
 - abnormal electrolytes
- Abnormal conduction ± spont d/c of His/Purkinje system or myocardium
 - = ↑ed automaticity of heart
- Sinus arrhythmia = Extrasystole = Ectopic focus discharges beat before next expected beat
- Paroxysmal tachycardia = repetitive d/c of ectopic focus at rate over SA node

ReEntry

- Common cause of paroxysmal tachyarrhythmia's = defect in conduction permits circus movement
- = transient block on one side of conduction system impulse down good side then back up diseased side then repeat
- in AV node re-entry:
 - retrograde flow back up diseased side atrial depolarisation
 - next beat = echo beat
 - depolarisation then circus back down good side and continue
- non-AV node re-entry:
 - abnormal extra bundle of connecting system connecting atria & vent
= bundle of Kent
 - wave down AV node, retrograde through bundle of kent
- circus movements seen in atrium & vent
- Rx options:
 - ▶ carotid sinus massage:
 - may terminate re-entry SVT
 - may differentiate SVT from flutter & fast AF
 - ▶ adenosine:
 - may terminate re-entry SVT
 - 6mg then 12 mg or 100mcg/kg, then 200mcg/kg, then 300mcg/kg
 - ▶ β blockers or verapamil (5-10mg IV slowly over 2mins)
 - ▶ amiodarone
 - ▶ (avoid digoxin \Rightarrow facilitates WPW syndrome \Rightarrow \uparrow HR)

Atrial Arrhythmias

- **atrial extrasystole :**
 - ECG changes:
 - P wave abnormal
 - QRST normal
 - Extrasystole may depolarisation SA node must repolarise before next normal beat
 \hookrightarrow see pause which allows natural reset of rhythm
 - ▶ generally normal but can be caused by:
 - ischaemia/hypoxia
 - light anaesthesia, sepsis, shock
 - anaesthetic drugs
- **Atrial tachycardia**
 - Rate up to 220/min
 - when:
 - Regular atrial focus d/c
 - Re-entry circus tachy
- **Atrial flutter:**
 - Atrial rate 200-350; ventricular rate commonly \sim 150
 - Anticlockwise circus in R atrium
 - Assoc 2:1 or greater Av block
as AV cannot conduct >230 beats/min
 - \hookrightarrow Rx: if new onset shock. If unknown then same VTE risk as AF:
 - adenosine will expose underlying Atrial saw tooth rhythm
 - drugs as AF

- Sick sinus Syndrome:
 - may coexist with AV nodal disease
 - episodes of AV block, atrial tachycardia, AF, flutter
 - commonest cause =
 - congenital
 - old age
 - asymptomatic or pre/syncope
 - pacing required if symptomatic
- **Atrial Fibrillation:**
 - seen in 25% >75yrs
 - Atrial rate 300-500 irreg disorganised fashion
 - Vent rate 80-160
 - Cause unknown but include:
 - HTN, IHD, cardiomyopathy
 - pericarditis/mediastinitis, thoracic surgery
 - mitral valve disease ie dilated LA
 - electrolyte disturbance - esp ↓K, ↓Mg
 - sepsis, thyrotoxicosis, alcohol esp binge
 - lines in RA/RV
 - idiopathic:
 - Multiple re-entrant circus excitation
 - Ectopic foci – often seen in pulmon veins ~4cm from heart
 - atrial systole contributes 20-30% vent filling
 - blood clots with atria common
 - ↑risk if return to SR >48hr after onset of AF
 - in stable AF risk of stroke = 4% at 75yr
 - ↳ use of anticoagulation ↓risk by 50% (2%/yr)
 - Rx strategies:
 - rate control <100
 1. Beta-blockers
 2. Verapamil (avoid if ↓LVF, ischaemia or on B-blockers) 5-10mg IV
 3. Digoxin - IV loading 500mcg, repeated every 6hrs to max 1.5mg
 - rhythm control
 - if acute <48hrs or anticoagulated for at least 3 weeks:
 1. Amiodarone 300mg IV -> infusion (1hr) then 900mg over 23hrs
 2. Flecainide 2mg/kg (max 150mg) over 30 minutes or 300mg orally - avoid if IHD or ↓LVF
 3. DC Cardioversion
 - **Indications for Emergent Cardioversion**
 - CP
 - hypotension
 - Heart failure
 - uncontrolled chronic AF:
 - aim for <100 bpm prior to theatre
 - options:
 - digoxin oral loading - levels 0.8-2 mcg/L)
 - β blockers
 - amiodarone if ↓LVF

Consequences Atrial Arrhythmias

- If high vent rate ↓diastolic filling heart failure
- Vagal stimulation effects:
 - Ach ↓conduction in AV node & atrial myocardium
 - ↑AV block
- digoxin also blocks AV node

Ventricular Arrhythmias

- differential diagnosis of broad QRS =
 - ▶ VT
 - ▶ SVT with aberrancy (conduction block)
 - ▶ antidromic AVRT
 - ▶ Artefact
 - ▶ Pre-excitation - if slurred delta wave then think WPW
 - ▶ ventricular extrasystole (ectopic)
- ectopic vent focus extrasystole:
 - ECG:
 - abnormal QRS
 - P may be buried in QRS
 - unable to depolarise bundle of His no retrograde flow
 - no resetting of rhythm as in atrial ectopics
 - longer compensatory pause after ectopic
 - SA node continues to fire regularly irrespective of vent activity
 - if underlying sinus rate <50bpm then ectopic may = escape rhythm
 - common & usually benign
 - if early in diastole: may not be strong enough to create pulse at wrist
- Vent tachycardia (VT) =
 - Due to circus movement in ventricles caused by
 - ▶ MI, hypoxia, hypotension
 - ▶ fluid overload
 - ▶ electrolytes - ↓K, ↓Mg
 - ▶ iatrogenic catecholamines
 - Rx:
 - ▶ sync shock - 100% success
 - ▶ drugs:
 - amiodarone
 - lignocaine -100mg bolus then infusion 4mg/min for 30min ⇒ 2mg/min for 2hr
 - sotalol
 - Torsade de pointes:
 - Form of VT with varying QRS morphology
 - Rx with urgent Mg
 - May be assoc with no CO or just ↓CO
 - VF occasional complication of VT
- SVT with conduction block can be diff to differentiate with VT
 - need HBE – VT will not have a H spike
 - unable to differentiate ∴ Rx as VT
 - uncommon ~2%
 - adenosine may expose underlying atrial rhythm but can also cease VT thus not useful discriminator
- VF:
 - Muscle fibres contract in irreg and ineffective way
 - Due to multiple ectopic focus or circus movement
 - Can be induced by defib during vulnerable period

Normal ECG parameters:

- Q wave:
 - ▶ normal in III
 - ▶ look for Q waves in leads either side for confirmation
- R wave:
 - ▶ Normal = equiphasic in V3
 - ▶ should see ↑size through chest leads

- Vulnerable period = midportion of T wave
 - =when ventricular muscle at diff stages of de & repolarisation
great time to start circus movement
- most common cause of death in ACS is VF

Long QT

- long QT \approx ventricular repolarisation is irreg \uparrow incidence of arrhythmia
- QTc > 450ms
- caused by:
 - ischaemia
 - drugs
 - electrolyte abnormalities
 - congenital – genes encoding for Na & K channels mutated

Accelerated AV Conduction

WPW

- additional aberrant connection between atria & vent:
 - types:
 - muscular or
 - nodal tissue ie bundle of Kent
 - conducts more rapidly than AV node one vent excited before other
- ECG change:
 - short PR interval
 - wide QRS
 - normal P axis
 - upstroke slurs of R wave = delta wave
 - normal PJ interval (start P to end QRS)
- arrhythmias start:
 - following atrial extrasystole
 - beat goes 2 ways:
 - through AV node, retrograde through aberrant pathway back to atria
 - through aberrant pathway, retrograde through AV node (less common)
- congenital element:
 - mutation in AMP activated protein kinase
 - ?involved in suppressing development of abnormal pathways in utero
- Rx:
 - need drugs that \downarrow conduction in all pathways (not just AV node)
 - amiodarone & flecannide
 -

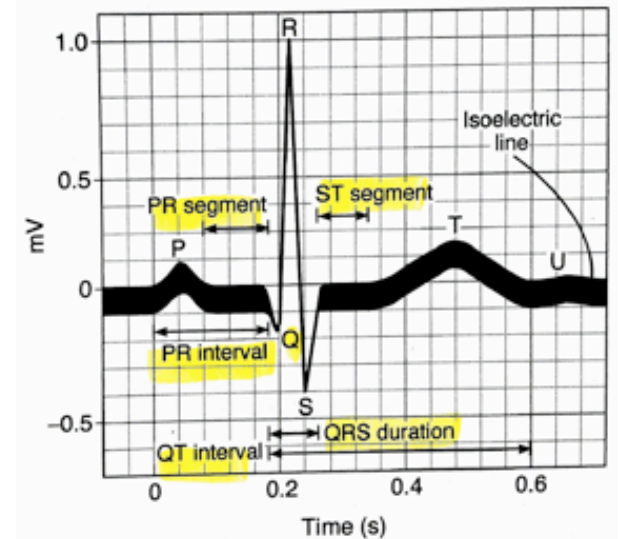
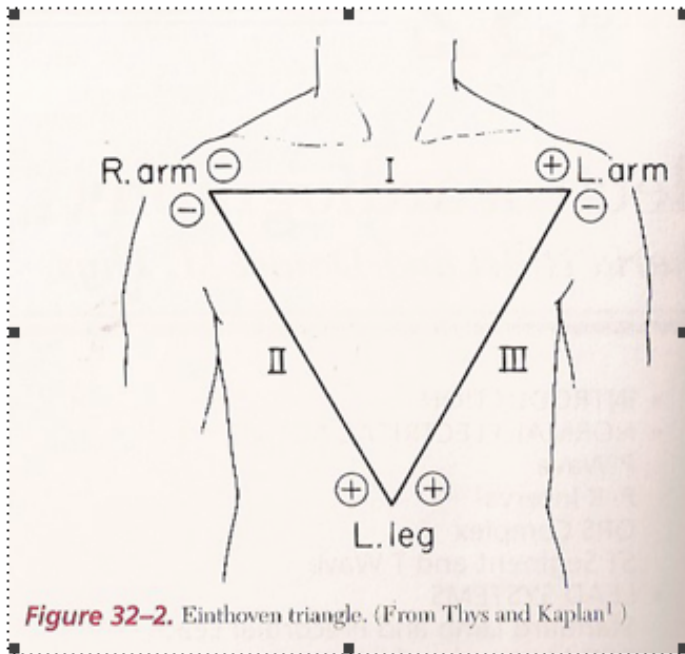
Lown-Ganong-Levine Syndrome

- short PR & normal QRS
- beat bypasses AV node by aberrant conduction pathway but then joins intraventricular conduction system before depolarising vents

ECG

- standard ECG =
 - 25mm/sec 0.04sec/small sq, and 0.2 sec/big square
 - 1mV = 1cm

- Calculate approx rate by
 - dividing 300/number of large squares
 - counting number of big squares in between R waves:
 - 1 = 300
 - 2 = 150
 - 3 = 100
 - 4 = 75
 - 5 = 60
 - 6 = 50



- Einthoven's triangle =
 - heart at centre with 3 limb leads around
 - triangle between shoulders & pubic symphysis
 - electrodes record cardiac electrical activity in vertical plane
 - 3 standard limb leads records electrical activity from 2 corners of triangle:
 - lead I = RA LA
 - lead II = RA LF (left foot)
 - lead III = LA LF
 - (4th electrode acts as an earth)
- Depolarisation towards = upward deflection
- Depolarisation away = downward
- Repolarisation toward = downward
- Repolarisation away = upward
- P = atrial depolarisation
- QRS = vent depolarisation & atrial repolarisation
- T = vent repolarisation
- QT = total duration of vent depolarisation & repolarisation
- U = not always seen – repolarisation of papillary mms
- J point = junction between QRS & ST segment

Lead 2 Example

- Lead 2 lies in axis of heart
- P wave: atrial depolarisation SAN AVN which is down & Left (ie dep toward = upward)
- Small initial Q wave: depolarization starting in IV septum spreading down & right (ie dep away = downward)
- Large R wave = depolarisation spreads endo epicardial & larger bulk of LV means net effect is down & left (ie dep toward = upward)
- Small S wave = activation of remaining ventricle, wave spreading upwards ie (dep away = downwards)
- T wave = ventricular repolarisation moving from epicardial to endocardial (ie repol away = upward)

ECG Intervals

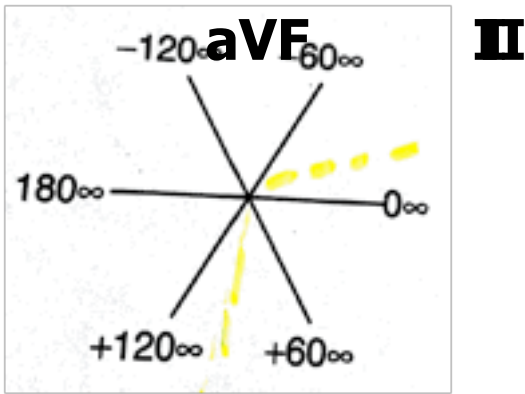
- PR = 0.12-0.2 secs
AV delay (0.1sec) accounts from most of delay
- QRS = <0.12
- QT = 0.3-0.43
varies inversely with HR
- QTc = $\frac{QT}{\sqrt{RR}}$
- ECG Pattern
- aVL & aVF look at ventricles mostly positive
- V1-V2:
 - No Q wave
 - Small initial R wave 2nd to L to R septal depolarisation
 - Large S wave – depolarisation down septum & into ventricle away from electrode
- V4-V6:
 - Small q = initial depolarisation across septum L to R
 - Large R – septal and vent depolarisation
 - Mod S – late depolarisation of vent moving back to atria
- LVH criteria:
 - ▶ S wave in V1 + R wave in V5 or V6 >35mm in size
- LV strain - useless syntax but suggestive:
 - ▶ LVH by voltage
 - ▶ downsloping ST depression
 - ▶ T inversion
- tall R in V1 differential diagnosis:
 - ▶ RBBB
 - ▶ RVH
 - ▶ post MI
 - ▶ dextrocardia/duchenne
 - ▶ WPW

Cardiac Axis

- Mean QRS vector = -30 to +110
- L axis deviation if axis to left (up or <) of -30
- R axis deviation if axis to R (down or >) of +90

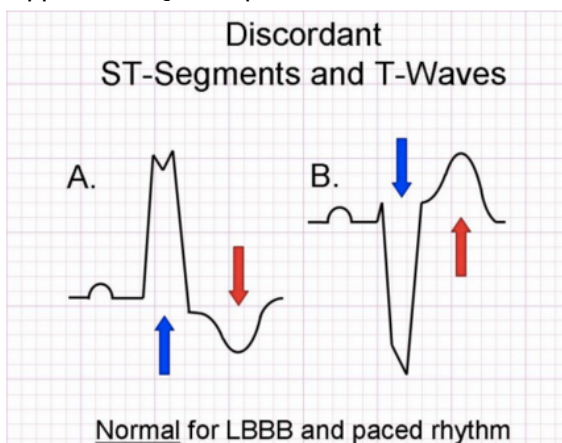
ECG in Ischaemia

- ST elevation differential:
 - ▶ infarct
 - ▶ aneurysm/old infarct

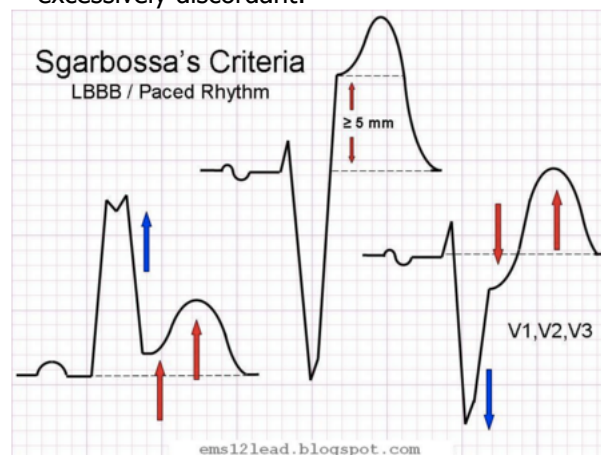


- Conduction abnormality
- LVH
- Pericarditis
- ECG changes:
 - infarction = ST elevation ≥ 2 contiguous leads $> 2\text{mm}$
 - ischaemia = ST depression ≥ 2 contiguous leads $> 1\text{mm}$
 - T flattening or inversion in absence of dig effect
 - hyperacute peaked T waves
 - new onset RBBB with other signs of ischaemia
 - LBBB -
 - not in itself diagnostic but prevents interrogation of ST segment
 - new LBBB is always pathological (of at least something)
 - Sgarbossa Criteria:
 - $\geq 1\text{mm}$ of concordant ST elevation
 - ≥ 1 lead of V1-V3 with $\geq 1\text{mm}$ of concordant ST depression
 - ≥ 1 lead anywhere with $\geq 1\text{mm}$ ST elevation & excessive discordant ST elevation ($\geq 25\%$ of depth of preceding S wave or $\geq 5\text{mm}$)

= normal pattern - discordant
means ST segment should be
opposite to QR complex:



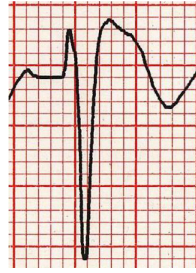
= abnormal pattern concordance
ie ST segment follows QRS **or** is
excessively discordant:



- location of changes allows identification:
 - ▶ septal = V1-2 [ant = LAD; post = PDA]
 - ▶ anterior V2-5 [LAD]
 - ▶ antero-lateral V3-6 + aVL [LCx]
 - ▶ Left main stem = widespread ST depression with ST elevation in aVR > V1
 - ▶ posterior - V1-3 changes: horizontal ST depression, upright T waves, dominant R wave in V2
 - ▶ RV - ST elevation in V1 & lead III > lead II [RCA]
 - ▶ inferior - ST elevation in II, III, aVF & reciprocal depression in aVL

Brugada

- Sodium channel abnormality
- see V1 + V2 ST elevation with partial RBBB abnormality
- in Asian men
- 2 types:
 - type 1 - syncope \Rightarrow needs ICD
 - type 2
- see coved ST segment - ST falls off the cliff



Torsades

- Causes:
 - hereditary
 - TCA overdose
 - \downarrow Mg
- Treatment:
 - Mg load + shock
- Prevent: avoid agents which may prolong QTc eg amiodarone

ECG Monitoring Systems in Anaesthetics

3 Electrode Systems

- ECG observed alone 1 bipolar lead between 2 electrodes
- 3rd electrode = ground
- selector switch allows you to change between electrodes
- simple system to monitor rate & rhythm
- limited info on myocardial ischaemia

5 Electrode System

- allows for recording of all standard 6 limb leads as well as one precordial lead
usually V5 – good for ant ischaemia detection

Modified 3 Lead System

- 3 lead system modified to give better results:
 - maximise P wave height for diagnosing arrhythmias
 - incr sensitivity to detect ant ischaemia
- several modifications exist
- one example = **CS5** lead:
 - = bipolar lead best & easiest alt to true V5 lead for monitoring ischaemia
 - RA electrode placed under R clavicle
 - LA electrode placed in V5 spot
 - LL in usual spot for grounding
 - Leads:
 - Lead 1 = detection ant ischaemia
 - Lead 2 = inferior ischaemia/arrhythmias

Post MI Vent Arrhythmias

- timing:
 - 30mins post – re-entry arrhythmias common
 - 12hrs post – due to \uparrow ed automaticity
 - 3days post – due to reentry
- damage to epicardial regions interrupt sympathetic nerve fibres

- denervation super-sensitivity to catecholamines in area beyond infarct
- damage to endocardial regions interrupt vagal fibres unopposed sympathetic action

Electrolyte Effects on ECG

Na

- ↓Na: low voltage ECG

Hyperkalaemia

- changes in sequence:
 - tall peaked T waves
 - paralysis of atria – loss of P waves
 - prolonged PR
 - prolonged QRS
 - bundle branch blocks
 - sinus brady or slow AF
 - sine wave \Rightarrow terminal rhythm
- resting membrane potential of myocardium ↓'s ↓excitability of myocardium heart stops in diastole

Hypokalaemia

- in sequence:
 - ST segment depression
 - U wave
 - ↑ P wave
 - PR prolongation
 - \pm T inversion
 - apparent long QT (actually fusion of T & U waves)
- not as fatal as hyperkalaemia

Hypercalcaemia

- ↑ed myocardial contractility & ↓ed ability to relax
- in vivo difficult to get plasma Ca levels high enough to effect heart

Hypocalcaemia

- prolongation of ST segment prolongation of QT
- mimicked by drugs incl TCAs, phenothiazines

Defibrillation

- risks:

- myocardial damage
- patient burns
- electrocution of staff
- damage to pt electrical devices ie PMs
- if VT or AF & defib not sync'd \Rightarrow risk of R on T \Rightarrow VF
- VTE risk of cardioversion
- may require repeat defib