- Mx: usually need pacing
  - 2:1 or 3:1 (advanced) block
    - Every 2\textsuperscript{nd} or 3\textsuperscript{rd} P wave conducts to the ventricles
    - Either nodal or infranodal level
- Second-degree heart block of any kind may occur following acute MI
  - Inferior MI: close monitoring, may need temporary back-up transcutaneous pacing
  - Anterior MI: high risk of progression, temporary pacing and usually permanent pacing

**Third-degree (complete) AV block**
- All atrial activity fails to conduct to the ventricles
- Can co-exist with any atrial rhythm so P waves could be abnormal or absent
- PC: heart failure, dizziness, falls, LOC
- Life is maintained by a spontaneous escape rhythm
- Narrow complex escape rhythm
  - 50-60bpm and relatively reliable
  - Must originate in His bundle
  - Therefore suggests that the region of block is more proximal in the AV node
  - Management
    - Recent onset due to transient cause: IV atropine, may need temporary pacing
    - Chronic esp. if symptomatic or associated with heart disease: dual chamber permanent pacing
- Broad complex escape rhythm
  - Originates below His bundle so block is more distal in His-Purkinje system
  - 15-40bpm and relatively unreliable
  - Stokes-Adams attacks (dizziness and blackouts)
  - Causes
    - Elderly: Lev’s disease (degenerative fibrosis and calcification of distal conducting system)
    - Young: Lenegre’s disease (proximal progressive cardiac conduction disease due to inflammatory process)
    - Any age: IHD, myocarditis, cardiomyopathy
  - Management
    - Permanent pacing
    - + ICD if severe LV dysfunction (>0.3s)

**Aetiology of complete heart block**
- Congenital
  - Maternal SLE
  - Transposition of the great vessels
- Idiopathic fibrosis
  - Lev’s disease
  - Lenegre’s disease
- IHD
  - Acute MI
May progress to tricuspid regurgitation (pansystolic murmur at left sternal edge)

Indicators of severity
- Proximity of opening snap to S2
- Length of diastolic murmur
- Pulmonary HTN
- Disappearance of S1 and opening snap

Risk factors
- Rheumatic fever (group A beta-haemolytic strep), women > men

Investigation
- CXR: small heart, large LA, pulmonary venous HTN (upper lobe diversion), calcified mitral valve, pulmonary oedema
- ECG: bifid p wave (p mitrale, unless AF) as delayed LA activation, RV hypertrophy (right axis deviation, tall R wave in V1)
- Echo: pressure half time, mean pressure drop, valve area
  - Wilkins score determines suitability for percutaneous valvotomy
- TOE: presence of LA thrombus pre-op
- (Do not use cMRI or catheterisation)

Medical management
- Low dose diuretics for SOB
- Treat AF

Indications for surgical intervention
- Moderate to severe MS on echo
- Pulmonary HTN
- Failure of medical therapy
- Threshold lower due to AF and risk of stroke

Surgical options: trans-septal balloon valvuloplasty, closed valvotomy, open valvotomy or replacement

Complications
- AF (if this occurs get a big deterioration in clinical state)
- Bronchitis
- (Infective endocarditis rare)

Mitral regurgitation

Symptoms
- Often none until late if chronic
- Palpitations (increased stroke volume)
- SOB from pulmonary HTN either from the MR or from LV failure
- Fatigue and lethargy from reduced CO
- Later in disease, right heart failure (congestive)
- If acute, more likely to get pulmonary oedema as LA does not have time to dilate so get an increase in pressure, increased LA v-wave and consequent pulmonary HTN
• SOB  
• Syncope  
• Fatigue

Signs
• Narrow pulse pressure (difficult in context of HTN)  
• Low volume slow rising or plateau pulse (sinus rhythm, difficult in context of HTN)  
• Thrusting non-displaced apex beat (LV hypertrophy) and may feel double impulse of S4  
• Mid-systolic rough low-pitched crescendo-decrescendo ejection murmur loudest in aortic area (may feel thrill)  
• Patient leaning forward in expiration  
• Radiates to carotids (almost always)  
• Early on while valve still mobile get systolic ejection click of valve opening  
• Later on get quiet or absent A2 (calcified and therefore rigid aortic valve)  
• Reversed splitting of S2 (i.e. greater on expiration as A2 comes after P2)  
• S4 (unless coexistent MS)  
• Murmur quite similar to HOCM but differences = radiation to carotids and manoeuvres affecting intensity  
  o Valsalva manoeuvre decreases intensity of murmur  
  o Squatting or passive leg lift increases intensity

Risk factors
• Common  
  o Calcification: age, male, lipids, HTN, DM, smoking  
  o Bicuspid valve: familial, coarctation, root dilatation, aortic dissection  
  o Rheumatic fever  
  o Chronic kidney disease  
  o Paget’s disease of the bone  
  o Radiation  
  o Familial hypercholesterolemia

Indicators of severity
• Longer systolic murmur as longer ejection time needed  
• NB. Intensity of systolic murmur poor indicator as quieter when CO less so in fact may be inaudible in severe AS

Investigations
• CXR: small heart (until heart failure when cardiomegaly), prominent dilated ascending aorta (post-stenotic dilatation), calcified valve  
• ECG: LV hypertrophy, LA delay, LV strain (ST depression, T wave inversion in I, AVL, V5 and V6 i.e. leads orientated to LV), sinus rhythm  
• Echo: peak velocity, pressure drop, valve area  
• Catheterisation to assess for coronary artery disease  
• (Rarely, TOE, cMRI etc)

Medical management
• Stockings etc

Risk factors for bleeding
• Active bleeding
• Acquired bleeding disorder e.g. acute liver failure
• Concurrent use of anticoagulants e.g. warfarin with INR > 2
• LP, epidural or spinal anaesthesia within previous 4h or expected in next 12h
• Acute stroke
• Thrombocytopenia (<75)
• Uncontrolled systolic HTN (>230/120)
• Untreated inherited bleeding disorder e.g. haemophilia or von Willebrand

**Hypertension**

**Definition**
Optimal BP < 120/80

Threshold for HTN is 140/90
Threshold for HTN at home is 135/85
Threshold for HTN on 24h is 125/80

Hypertension (use whichever grade is higher of systolic and diastolic readings)

- Grade 1 (mild) 140-159 and/or 90-99
- Grade 2 (moderate) 160-179 and/or 100-109
- Grade 3 (severe) 180+ and/or 110+

Isolated systolic hypertension

- Grade 1 140-159 and <90
- Grade 2 160+ and <90

**Causes**

**Essential** (80-90%)

- FHx
- Obesity (however may also overestimate BP if cuff is too small)
- Sleep disordered breathing
- Alcohol
- Sodium intake
- Stress
- Metabolic syndrome

**Secondary**

- Endocrine
  - Cushing’s syndrome
  - Acromegaly
  - Thyroid disease
  - Hyperparathyroid disease
- Adrenal
  - Conn’s syndrome
  - Adrenal hyperplasia
Severe HTN: headaches, epistaxis or nocturia
Malignant HTN: severe headaches, visual disturbances, fits, TLOCs or symptoms of heart failure

Phaeo: sweating, headaches, palpitations
Atheromatous renal artery stenosis: angina or other symptoms of peripheral vascular disease

Cardiac failure or LV hypertrophy: breathlessness

**Examination**
Elevated BP usually only abnormality

Signs of underlying cause
- Renal artery bruits (renovascular HTN)
- Radiofemoral delay (aortic coarctation)

Signs of complications
- Cardiac
  - LV hypertrophy
  - Loud A2
  - If failure: sinus tachycardia, S3
- Fundoscopy (Keith-Wagener classification)
  - Grade 1: tortuous retinal arteries with increased reflectiveness (silver wiring)
  - Grade 2: grade 1 plus AV nipping (thickened retinal arteries pass over retinal veins)
  - Grade 3: grade 2 plus flame-shaped hemorrhages and soft (cotton-wool) exudates (small infarcts)
  - Grade 4: grade 3 plus papilloedema (blurring of margins of optic disc)
- Grades 3 and 4 are diagnostic of malignant HTN

Ambulatory BP monitoring
- Exclude white coat HTN
- Monitor treatment response esp. adequacy of 24h control with OD meds
- Loss of nocturnal fall (non-dippers) have worse prognosis

**Investigation**
Aims
- Exclude secondary causes
- Assess target organ damage
- Identify comorbidities that may increase CV risk

Bloods
- U+Es
- Lipids (total and HDL cholesterol – non-fasting initially, only fasting if abnormal triglycerides) and glucose

Urine dip
• Annual review of care
• NB. For statins: must check blood lipids and LFTs 3 months after starting statin, LFTs at 12 months and then annual follow up

Body weight
• BMI 20-25

Aerobic exercise
• >30min brisk walk most days

Diet
• Reduce fat and saturated fat
• Reduce salt (<6g NaCl)
• Limit alcohol (no more than 3 units/day for men, 2 for women)
• 5 portions of fruit and veg per day

Cardiovascular risk reduction
• Stop smoking
• Oily fish

Pharmacological management summary

Young Caucasian patients are more likely to have renin HTN
If a drug from within a pair is not tolerated, try the other drug in that pair
If a drug is not effective, try a drug from the other group
Most patients will need >1 drug
Further diuretic therapy = spironolactone
Do not use diuretic and beta-blocker as both aggravate DM

Specific drugs
Diuretics
• Thiazide diuretics in low dose esp. chlortalidone
  o Indications
    ▪ Stroke secondary prevention
    ▪ >55y
    ▪ Black patients
• Other changes that are highly suggestive of ACS
  o ST depression
  o Pathological Q waves
  o T wave inversion
  o May get transient ST elevation with coronary vasospasm (Prinzmetal’s angina – occurs typically at rest or overnight, link to Raynaud’s)
  o NSTEMIs tend to be more dynamic than STEMIs

• Repeat…
  o Every 15 mins if normal on admission and still pain
  o With any further chest pain
  o Ideally continuous ST segment monitoring

• Progression of ECG changes
  o Hyperacute (tall positive) T waves (very early)
  o ST elevation
  o T wave inversion and Q waves
  o T waves return to normal but Q waves remain

• If an inferior MI is found, must do another ECG with right-sided chest leads to check for RV involvement (ST elevation in V4R)
  o Important as can develop RV failure causing hypotension via reduced LV filling pressure so these patients need fluid resus

Troponin (peaks earliest at 18-24h)
• Not present in normal individuals
• Differentiates NSTEMI and unstable angina
• If initially negative, repeat 6-12 hours after admission
• Relationship between serum troponin I levels and mortality

Creatine-kinase-MB (peaks around same time as troponin)
• Present in normal individuals and those with skeletal muscle damage
• Useful to determine reinfarction as levels return to normal after 36-72 hours

Myoglobin
• Presence in skeletal muscle makes this non-specific
• Useful for rapid diagnosis of ACS as levels become elevated very quickly

Other bloods
• FBC
• U+Es
• Glucose
• Lipids

Echo
• Confirm STEMI (wall motion abnormalities)
• Exclude differentials: aortic dissection, pericarditis or PE

Risk stratification e.g. TIMI
For NSTEMI and UA: 7 factors gaining 1 point each
• Age >65y
• >3 risk factors: HTN, high lipids, FHx, DM, smoking
- Known coronary angiography stenosis >50%
- Aspirin use in last 7 days
- Severe angina (>2 episodes at rest in 24h)
- ST deviation
- Elevated cardiac markers

For STEMI: 11 factors totalling 16 points

**Emergency management of STEMI (inc. LBBB)**
1. Oxygen only if hypoxic (sats < 94%)
2. IV access
3. Bloods: FBC, U+E, glucose, lipids, cardiac enzymes
4. History and examination
5. Aspirin 300mg and ticagrelor (or clopidogrel) all PO
6. Morphine IV + metoclopramide IV
7. GTN sublingually
8. Beta-blockers
9. (CXR pre-thrombolysis if suspected aneurysm e.g. interscapular pain or BP different in each arm)
10. Restore coronary perfusion ASAP
   - EITHER Cath lab +/- primary angioplasty within 90 mins even if this means transferring patient (preferred)
     - If primary PCI, give GP IIb/IIIa inhibitor (abciximab) to reduce short term complications
   - OR Thrombolysis / fibrinolysis
     - Either streptokinase OR t-PA + heparin or enoxaparin
     - Only really useful up to 2h (and either ST elevation or new LBBB)
     - Lots of contraindications
     - If failure at 60-90 mins (30% resolution of ST elevation): rethrombolysis or rescue angioplasty
       - Give aspirin alongside but not clopidogrel or abciximab
     - (CABG only if complications of MI)

**Emergency management of NSTEMI or unstable angina**
1. Admit to CCU and monitor closely
2. Oxygen only if hypoxic (sats < 94%)
3. Morphine IV and metoclopramide IV
4. GTN sublingually
   - Monitor BP
     - May be required for some time, ensure to wean off as tolerance develops
5. Aspirin 300mg and ticagrelor (or clopidogrel) all PO
6. Oral beta blocker e.g. metoprolol or atenolol
   - To decrease myocardial energy consumption
   - Aim for resting HR 50-60
   - Contraindications e.g. asthma, bradycardia, left ventricular failure
   - If CI, give rate-limiting calcium antagonist PO e.g. verapamil or diltiazem
7. Heparin e.g. enoxaparin or dalteparin
8. IV nitrate if pain continues
   - Maintain systolic BP > 100mmHg
- May need intra-aortic ballon pump
- Could use inotropes e.g. dopamine or dobutamine but ischaemic heart won’t respond well

- Arrhythmias
  - May occur with reperfusion
  - Mostly ventricular
    - Bigeminy
    - VT: need IV beta-blockers, lidocaine or amiodarone OR synchronised cardioversion if hypoBP
    - VF
    - IV magnesium can help refractory ventricular arrhythmias
  - AF: beta-blockers, digoxin, (cardioversion)
  - Ensure potassium >4.5 (>4 post-MI in absence of arrhythmias)

- Complete heart block
  - Normally inferior STEMI (right coronary artery supplies the nodes)
    - May recover as may indicate ischaemia c.f. infarct
    - May need atropine
    - However may not need pacing or just temporary transcutaneous
    - Ideally want to avoid pacing an MI due to risk of VF so only pace if persists for >2 weeks
  - Can also get complete heart block with anterior MI due to interruption of conduction pathway
    - Implies very serious MI
    - Definitely need pacing

- LBBB

1-10 days
- Rupture of free wall
- Rupture of IV septum
  - 1-2% of STEMI
  - 92% 1 year mortality without surgery
- Mitral regurgitation
  - 3 mechanisms (may need TOE to differentiate)
    - LV dysfunction and dilatation causing annual dilatation of the valve
    - Dysfunction of the papillary muscle due to infarct of inferior wall
    - Rupture of papillary muscle
- Pericarditis (widespread ST elevation)

7-10 days
- Thromboembolism

Later
- Dressler’s syndrome
  - Secondary pericarditis
  - Sx: fever, pleuritic pain, pericarditis and/or pericardial effusion
  - Rare after MI but common after cardiac surgery
  - Conservative treatment
Unusual risk factors to consider…
- Myeloproliferative neoplasm causing hypercoagulable state (e.g. myelofibrosis or Jak2/PRB)
- Coronary vasospasm
- Coronary inflammation
- Anomalous coronary arteries
- Coronary dissection
- Embolisation

Cardiac Arrest
Emergency management
1. Get help
2. Check for signs of life and if none…
3. Call resus team
4. CPR 30:2 with airway adjuncts (and oxygen)
   - Continuous CPR once advanced airway in place
5. Once someone else arrives, ask them to apply pads and obtain vascular access
6. Stop CPR to assess rhythm
7. Continue below as appropriate

Shockable rhythm (VF or pulseless VT)
1. Charge machine
2. Warn everyone to stand clear except CPR giver and remove oxygen / GTN patches
3. Once charged tell CPR giver to stand clear
4. Give one shock then immediately resume CPR
5. Continue CPR for 2 mins
6. Rhythm and pulse check
7. If VT/VF persists, give a second shock then continue CPR for 2 mins
8. If still persisting, deliver a third shock and then immediately give 1mg IV adrenaline and 300mg IV amiodarone
9. Repeat 2 minute cycles
10. Continue giving further 1mg IV adrenaline after every 2nd cycle
11. May give 150mg more of IV amiodarone if refractory (lidocaine 1mg/kg is an alternative)

If organised electrical activity appears during a rhythm check, look for return of spontaneous circulation (central pulse – if any doubt, continue CPR)
- If ROSC, start post-resuscitation care
- If not ROSC, this is now a non-shockable rhythm (PEA)
If organised electrical activity appears during CPR, do not stop to check for a pulse unless obvious signs of life (e.g. sudden increase in end-tidal CO₂).

2g IV magnesium sulfate (4ml 50% solution) if torsades de pointes or refractory VF if suspected digoxin toxicity or hypomagnesiaemia (e.g. K⁺ losing diuretics)
Treat underlying problems e.g. anaemia

Secondary prevention with risk factor modification
- NB. Different to primary prevention which aims to prevent atherosclerosis development for all those with 10 year risk of fatal event >5% (may be markedly raised single risk factor or a combination)
- Lifestyle
- Statin
- Low dose aspirin
- ACEi indicated if treating any other condition e.g. HTN, HF, chronic kidney disease
- Manage co-existent conditions

Management is mainly symptom control…

**GTN spray**
Repeat after 5mins
SEs: headache and flushing via vasodilation

**Beta-blocker** such as metoprolol, atenolol or bisoprolol
Caution: COPD, acute HR, AV conduction, asthma
SEs: fatigue, peripheral vasoconstriction, bronchospasm and erectile dysfunction

**Ca channel block** e.g. diltiazem or verapamil
CI: severe bradycardia, LV failure with pulmonary congestion, 2nd or 3rd degree heart block
SEs: constipation (V), ankle oedema (D)

**Dihydropyridine Ca channel block** e.g. amlodipine
Not CI in same situations as the others
SEs: ankle oedema, reflex tachycardia

**Long acting nitrate** e.g. isosorbide mononitrate
Must give asymmetrically e.g. 8am and 2pm
SEs: headache, flushing
CI: viagra

**Ivabradine** = funny channel
SEs: bradycardia, phosphenes
CI: sick sinus syndrome, AV block

**Nicorandil** = K channel activator, also nitrate-like properties
• Pallor
• Paraesthesia (when severe)
• Paralysis (when severe)
• Perishingly cold
• Pulseless

Signs
• Tend to be unilateral
• Cold
• Mottling or marbling of skin
• Pulses diminished or absent (6th P!)
• Delayed capillary refill
• Veins empty
• May develop compartment syndrome (pain in calf on compression, bad sign)
• Hardening of muscles, blisters and gangrene spreading proximally

Assessment
• Assess the leg
• Neurological exam focusing on power, sensation and reflexes
• Dopper USS to confirm absence of pulses
• General CV exam
• Examine opposite limb for e.g. pulses, aneurysms etc

Emergency management
• Surgical emergency – need surgery within 4-6 hours
• Give heparin and analgesia whilst awaiting definitive treatment (and heparin afterwards)
• Intra-articular thrombolysis (native or graft)
  • For: embolic or thrombotic disease
    • First choice if graft occlusion
    • May reveal underlying stenosis that can be treated by angioplasty
• Embolectomy
  • For: embolic disease
• Bypass graft or angioplasty
  • For: thrombotic occlusion of popliteal artery (often acute-on-chronic lower limb disease) and trauma
• Amputation for unreconstructable or severe ischaemia
• Intervention may be inappropriate in patients dying from other causes

Reperfusion injury
• Release of toxic metabolites into circulation
• Muscle oedema can cause compartment syndrome (requires fasciotomy)

Consider long-term prevention
• Warfarin if emboli following MI or AF