CARDIOLOGY

Chest Hx Cardiovascular & Resp History Taking

1. HPS CADSPIF

Chest Pain	SOCRATES • NB: Continuous prolonged pain = **GORD, MSK, Resp.**
Ankle/Leg swelling	• Pitting vs non-pitting oedema • Symmetrical oedema (RVF) • Drug induced (e.g. Ca channel blocker – verapamil, -dipines)

Dyspnoea /SOB	• Exercise tolerance (what makes it stop? – be careful of fit individuals)
	• **MI** = mitral regurg., pulmonary oedema, rupture of chordae tendinae, papillary muscle infarction
	  ○ **Drug induced:** cocaine, amphetamine
	• **LVF = Orthopnoea (breathing difficulty when lying down)** → *# of pillows*
	  ○ *redistribution of interstitial oedema to fill upper zones of lung decreasing overall blood oxygenation (when sitting up → oedema in lower lobes = less effect)*
	• **LVF = Paroxysmal Nocturnal Dyspnoea (PND)**: Waking from sleep (exc. OSA)
	• Diurnal or seasonal variation
	• Wheezing?

Syncope [*transient LOC due to cerebral anoxia*]	• **Vasovagal (sweaty/nausea/Dizzy)** → *Emotional stress (e.g. seeing blood, crowded hot places)* • **Aortic stenosis** (on exertion) • **Arrythmia (***chest pain before syncope, anti-arrhythmics, or heart block***) ➔ rapid recovery unlike seizures** • ***Stokes Adams attack*** = *recurrent sudden syncope due to bradycardia* • **Tussive** (on cough) • **Micturition** (passing urine) • **Hypoglyceamic episode** • **Postural hypotension (**CaBs) • **Prolonged QT interval (**antiarrhythmics, gastric motility promotor, Antibiotics, antipsychotics) • **Bradycardia (**BBs, CaBs, Digoxin**)** • **Seizures (**post-ictal confusion)
Palpitations	• Regularity/Rhythm → Rate [Tap out for me!] • When did it start? Triggers?
Intermittent claudication	• Max claudication distance → Cramping pain? • **6 P's → pain, pallor, paresthesia, perishingly cold, pulselessness, paralysed** ○ Lumbar spinal stenosis, popliteal artery entrapment in young men
Fatigue	• Lack of energy or lack of motivation? • **Sleeping issue (OSA) →** Daytime somnolescence, **reduced CO (poor vascularization, aneamia)**

Palpitations

Type	Onset	Rhythm	Feature
SVT	Sudden	Regular	Pounding sensation in neck – CHECK CAFFIENE USAGE (DDx: idiopathic premature v. ectopics) Relived on Valsalva, carotid massage, cold ice cube
AF	Sudden	Irregular	Lethargy, exertional dyspnoea, orthopnoea, postural hypotension, light-headed
STEMI	Sudden		Pain or chest tightness
ST	Gradual	Regular	Rapid pounding
Ectopics	Sudden	Irregular	Pounding/fluttering/skipped beats followed by heavy beat
VT → VF	Sudden	Regular	Syncope + rapid → nausea, light-headed, exertional dyspnoea, central chest tightness

Cough	Productive vs non-productive → Triggers (nocturnal, exercise, Risk factors, ACEi usage)
Sputum	Volume → Frequency → Colour/Blood → Consistency
Heamoptysis	Volume → Frequency → Fresh red? Mixed in as streaks? coffee ground (vomit – upper GI bleed)

2. Past MHx

- *Previous* **cardiac problems**? **Operations/stents ? ECGs/ angiograms**?
- **MDT**? (e.g ED assoc. with ischemic heart disease)
- Birthplace + early life (childhood resp. dx)
- Home Oxygen
- Inhalers (freq + dosage)

Modifiable Risk Factors:

- Diabetes, HTN, IV drug use, HC (high LDL >5.2 mM),
- **CKD [highest risk for CAD]**, ED (linked to end-organ damage),
- chronic inflammatory disorder (e.g. RA, psoriasis, HIV),
- obese/sedentary, smoking, gout,

Non-modifiable:

- Male, Age
- 1st degree relatives Hx of premature IHD,
- past Hx of IHD or vascular HD

- Co-morbidities: **Asthma | COPD | ILD**
  - When diagnosed? How?
  - Spacer and reliever usage (Freq. and technique)
  - Compliance + hospital Ax
- **Other drugs causing lung toxicity**
  - COCP, MTX, NSAID, ACEi
  - Cocaine, thiazide, tryptophan, timolol (eye drops)

- **Allergies**: Ectopic Triad (food allergy + hayfever + eczema) → epipen? Anaphylaxis or rash?
- **Vaccinations:** fluvax, pneumococcal?

3. Social Hx [LOST]

- **Living** situation → carers, apartment/stairs, mobility aids
- **Occupation** (esp. for resp.) → birds or pets / miners / asbestos / IT office
- **Smoking** + alcohol
- **Travel** | **Mood** | **Hobbies** (spas/jacuzzi = non-TB bacterial infection) |

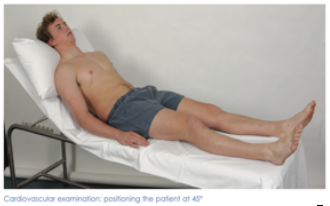
4. Family Hx

- Family CV diseases (Esp. Hx of CAD < 60 in 1st deg relative) → IHD, HTN, HC
- Marfan's syndrome or Ehler Danlos
- FHx of lung cancer, CF
- A1-anti-trypsin deficiency (↑emphysema, liver disease)

CV exam

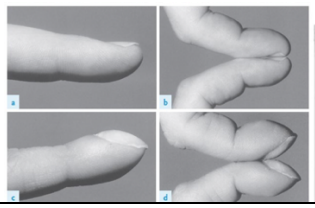
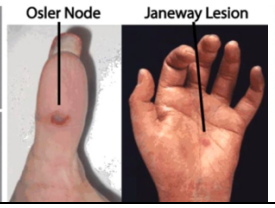
General inspection

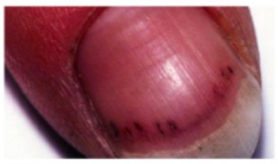
- (45° with chest/neck fully exposed)
- **Syndromes: Marfan's (***AR,/MR/Mitral Prolapse***), Turner's (***AS***), Down syndromes (***congenital heart disease***)**
- **General**: Pallor, cyanosis, comfort, SOB
- **Praecordium Scars**: pacemakers/ metallic valve sounds
- **Vital Signs**


Cardiovascular examination: positioning the patient at 45°

Hands

Perfusion	• Capillary refill, , peripheral cyanosis • Temperature
Nails	• *Clubbing (cyanotic CHD, IE), splinter haemorrhages (IE or mechanic occupation)* • Nicotine staining,
Dorsum	• Extensor tendon Xanthomata (irregular nodules overlying tendon – HC)
Palms	• Janeway Lesions | Osler's nodes (**P**ainful **p**urple **p**alpules**)** (IE) • Sweaty palms

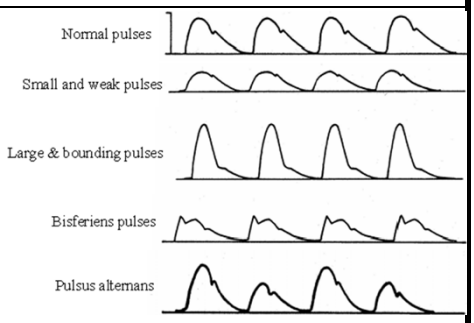

Splinter hemorrhages

Arms

Pulse -Rate & Rhythm	• Irregularly irregular = **AF** • Regularly irregular = **2nd deg heart block**

Pulse - character	• **Weak or low volume** = aortic stenosis + shock/hypovolaemia
	• **Bounding** = sepsis, AR, T2 respiratory failure
	• **Collapsing: (sudden drop in PP when blood returns into ventricle)**→ *CHECK THERE is **NO** shoulder pain → feel radial pulse with right hand → quickly lift arm with left hand → feel for any stronger tapping when arm is elevated*
	  ○ AR, *patent ductus arteriosus, high-output states e.g. anaemia, thyrotoxicosis, fever, pregnancy)*
	• ***Pulsus paradoxus** = asthma, PE, pericarditis, MI*
	• ***Pulsus alterans (sig. beat-beat variation)** = severe L) ventricular failure*
	• ***Jerky** = **HOCM***
	• ***Radio-radio delay** (aortic dissection/aneurysm or proximal coarctation)*
	*Only ask → ***Radio-femoral delay** = aortic coarctation (i.e. narrowing of aorta) → signs include*
	  • **Pre-repair:** *Severe HTN, weal left radial pulse, systolic vascular murmur*
	  • **Post-repair:** left lateral thoracotomy scar

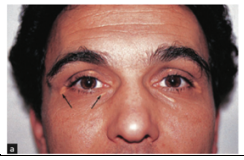
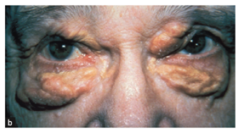
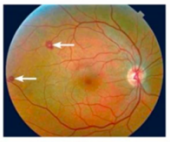
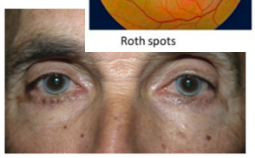
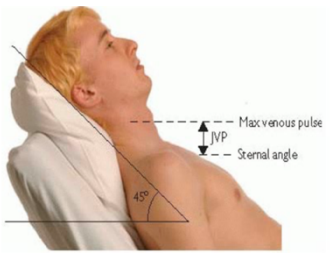
BP	• **Wide pulse pressure (AR), narrow pulse pressure (AS)**



Face & Neck

Face
- Malar flush (MS, pulmonary stenosis)

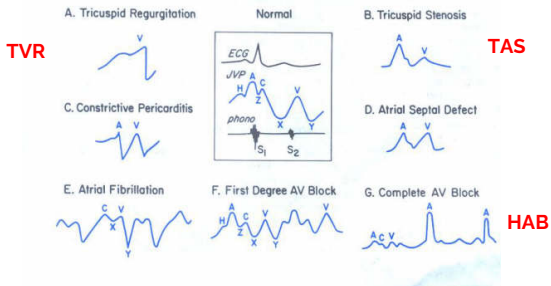
Eyes
- Conjunctival pallor (aneamia) | heamorrhages (IE) |
- **Corneal arc**us = HTN, HC, atherosclerosis
- **Roth spots = IE**, but also HTN, diabetes, Hypoxia
- **Xanthelasma (HC)**



(a) and (b) Xanthelasmata

Roth spots

Arcus senilis


Mouth
- Central cyanosis
- **Petechiae on mucosa &** Poor dentition (IE)
- **Palate** (high arched—Marfan's syndrome) → "LIFT tongue to roof of mouth"

Neck (at 45°)
- **PQRST = Elevated JVP** (**>3cm above sternal angle** –double pulsation of internal jugular vein)
  - **P**ulmonary HTN/PE/PS/Pericardial effusion
  - **Q**ty of fluid (e.g. overload)
    - **R**VF
    - **S**VC obstruction
    - **T**amponade/TR
- **Hepatojugular Reflux test** (apply pressure in RUQ → observe JVP → sustained rise = RVF)
- **Carotid pulse** (char + vol.)
  - *Slow-rising low volume (i.e. anacrotic plateau pulse)*= **AS**
  - *Bounding/collapsing* = **AR or patent ductus arteriosus**

Jugular pulsation	Carotid pulsation
2 peaks /double waveform	1 peak
Impalpable	Palpable
Obliterated by pressure @ base of neck	Hard to obliterate
Moves with respiration to increase VR	Little movement with respiration



	Pathology
A waves [atrial contraction]	• Dominant = TS, RVH → PS, Pulmonary HTN • Cannon = Complete Heart Block, VT
V wave [passive atrial filling]	Dominant = TR (very common)
X descent [atrial relaxation]	• Absent = AF • Exaggerated = constrictive pericarditis, cardiac tamponade
Y descent [tricuspid valve opens]	• Sharp = severe TR • Slow = TS,

Praecordium

Inspect

- **Chest Deformity** (e.g. pectus excavatum/carinatum)
- **Visible apex beat**, distended veins (SVC obstruction)
- **Scars** (pacemaker, ICD)
  - **Sternotomy** (midline) → ?CABG, thoracic aortic aneurysm open repair
  - **valvotomy** (mitral), **lateral thoracotomy, under L clavicle**, **saphenous vein graft**

Palpate

Apex beat position	• Palpate with whole hand → 5th IC space (1cm medial to midclavicular line) • **Impalpable** (**DOPE**): **D**extrocardia, **O**bese, **P**ericardial effusion, **E**mphysema • **Displaced**: LV dilation (MR, AR), cardiomegaly, RV enlargement
Apex beat character	• **Heaving**: High pressure pulsation in LVH → **AS, systemic HTN** • **Thrusting**: Large area/vol. pulsation in volume overload → **(MR, AR)** • **Tapping beat**: palpable S1 in MS *Cannot feel → patient roll to left and expire
Parasternal heave	Heel of right hand over left lower parasternal edge with straight elbow • Heave = **RVH** due to pulmonary HTN or PS
Thrills	Palpate over valve areas with pads of fingers • **AS** = Most common • **Pulmonary HTN** = Palpable S2 over pulmonary area

Auscultate [*all heart valves with diaphragm*]

	Procedure
Mitral valve	• Lying at 45° → Feel apex then auscultate → listen **left axilla for radiation (MR)** • **Roll to LEFT side → listen using BELL over apex on expiration → accentuates MS low tones**
Tricuspid valve	
Pulmonary valve	• Loud pulmonary S2 (pulmonary HTN)
Aortic valve	• Lying at 45° → listen over right carotid artery while holding breath for RADIATION (AS) • Sit patient forward and listen at Erb's point (3rd IC space, left sternal edge) on expiration → accentuates AR

Murmurs:	• Right valves = heard better on full inspiration
	• Left valves = heart better on full Expiration
	• **Systolic** murmurs = radiate (AS, MR, PS, TR)
	  ○ S1 = regurgitation [leakage across 'closed' valve]
	  ○ S2 =stenosis [obstruction to flow]
	  ○ **Innocent murmurs** = systolic ejection murmur (minor turbulence in blood flow in children)
	  ○ ***Always perform dynamic auscultation (i.e. Valsalva manoevre) if systolic murmur present (emphasises HCM and LVF)**
	• **Diastolic** murmurs= need dynamic manoeuvre to accentuate (AR, MS, PR, TS)
	  ○ S1 = stenosis
	  ○ S2 = regurgitation

Peripheral (*signs of fluid overload*)

• **Lungs**	• Percuss back to exclude **pleural effusion (stony dull percussion)** • Auscultate lung bases for: ○ **coarse crackles** while patient sitting (pulmonary oedema secondary to **LVF**)
• **Abdo + Lower LImb**	• Palpate sacrum, tibia and medial malleolus for 10s → feel for any indent (RVF, hypoalbuminemia, AR assoc. with ankylosing spondylitis) • Palpate for pulsatile hepatomegaly (?AR, RVF)

In Summary

"Today I performed a CV exam on_____"
"On general inspection, there was ____ peripheral stigmata of CV disease? With a regular/irregular pulse?
"On palpation, there were yes/no displaced apex / heaves/ thrills"
"On auscultation, 1st and 2nd heart sounds were present/absent with added sounds present/absent"

To complete

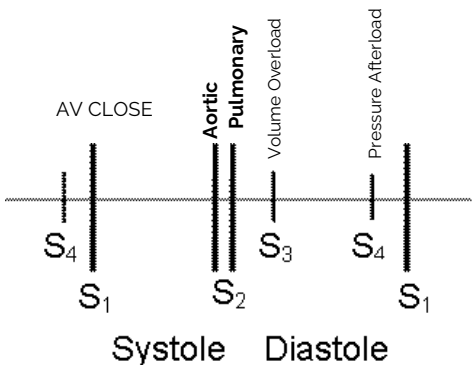
- Check peripheral pulses, perform an ECG,
- Palpate for hepatomegaly (RVF)
- **Review observation charts [fever in IE]**
- **24 hr holter monitor = "flutter", AF paroxysmal**
- **Urinalysis: dipstick the urine** (heamaturia in IE)
- **Bloods = FBC, EUC, LFT, CRP, BNP, TROPONIN**
- **SPECIFIC BLOODS** = TFT, Fe, B12, folate
- **Fundoscopy**: Roth spots (IE) & Hypertensive changes (e.g. papilloedema)

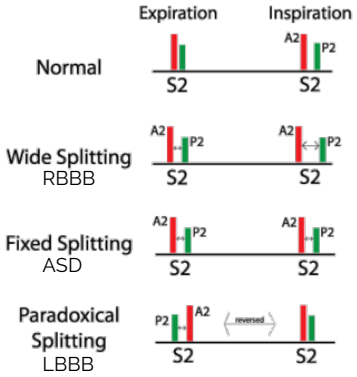
	TAMPONADE	CONSTRICTIVE PERICARDITIS
DEFINE	Fluid in pericardiac sac	Thick right inflamed pericardium
ONSET	Chronic	Acute
SX	HypoTN (best to differentiate) Muffled HS Raised JVP	Kussmaul breathing
RX	Pericardiocentesis	Meds – NSAID, aspirin +/- pred

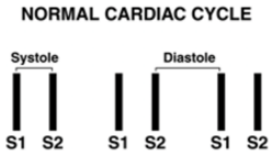
Added/Split Heart Sounds

Split = on inspiration

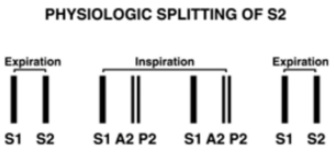
Single = on expiration







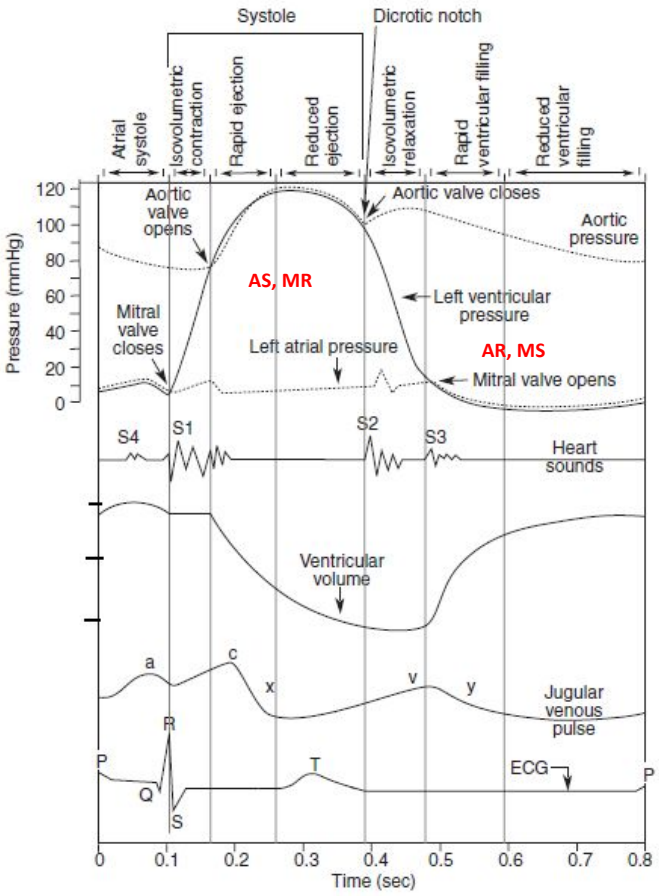
Loud S1 + S2 = SEVERE anaemia (systolic ejection murmur)



Heart Sound	Character		Pathophysiology	Cause
S4 **[atrial gallop]**	Before S1 ***[ALWAYS pathological!]*** • *Soft, low pitched* • *Disappears with AF* • *Heard every beat (late diastole)*		• Pressure overload: atrial contraction into stiff hypertrophied ventricle • ↑Afterload	• **Left ventricular S4**: IHD, HCM Systemic HTN, AS, LVH • **Right ventricular S4**: Pulmonary HTN, PS
S1 **[AV valves close]**	Loud		• AV valves close with higher velocity (as wide open at end of diastole)	• High atrial pressure (MS, AF) • Short diastole (short PR interval, tachycardia)
	Soft		• AV valves close with reduced velocity	• Valves don't close (MR) • Reduced contraction pressure (HF) • 1st degree heart block (Prolonged PR interval) → valves partially closed at end of diastole because atrial relaxation occurs before LV contraction
	Split S1		• Asynchronous AV valve closure	• RBBB • ASD (quite systolic flow murmur)
S2 **[Aortic/pulmonary valves close]**	**Volume**	Loud	• Valves close with higher velocity due to upstream pressure	• Pulmonary HTN (loud **Pulmonary -** P2) • Systemic HTN (loud aortic – A2)
		Soft	• Reduced aortic. Pulmonary valve mobility	• Calcified aortic valve, AS, PS
	Splitting *check on left sternal edge*	Split S2 on inspiration	• Aortic valve closes before pulmonary valve since increased blood return to right heart due to negative intrathoracic pressure	• **Normal** (↓RV afterload during inspiration) (↑transmural pressure across pulmonary vessels + dilatation of vessels)
		Wide split *R side*	• Exaggerated split → increases during inspiration *aortic valve closes before pulmonary valve*)	• RBBB, • **Increased resistance** to RV ejection (pulmonary HTN or stenosis, ILD
		Reverse Split (paradoxical)	• split increases during expiration (*pulmonary valve closes before aortic valve*)	• LBBB, • Large patent PDA • Increased resistance to LV ejection (systemic HTN or AS)
		Fixed split	• No change with respiration	• ASD (↑PE risk)
S3 **[ventricular gallop]**	**After S2** ***[may be normal]*** • *Soft, low pitched* • ***Persists*** *with AF* • *Heard every 3rd/ 4th beat*		• **Vol. overload** → high vol. of blood from atrium rapidly fills ventricle during passive filling phase • **Congestive cardiac failure**	• Physiologically normal until 30 • Hyperdynamic states (e.g. athlete, anaemia, fever, thyrotoxicosis) • **Left ventricular S3**: LVF, AR, MR, pregnant, thyrotoxicosis • **Right ventricular S3**: RVF, Constrictive pericarditis

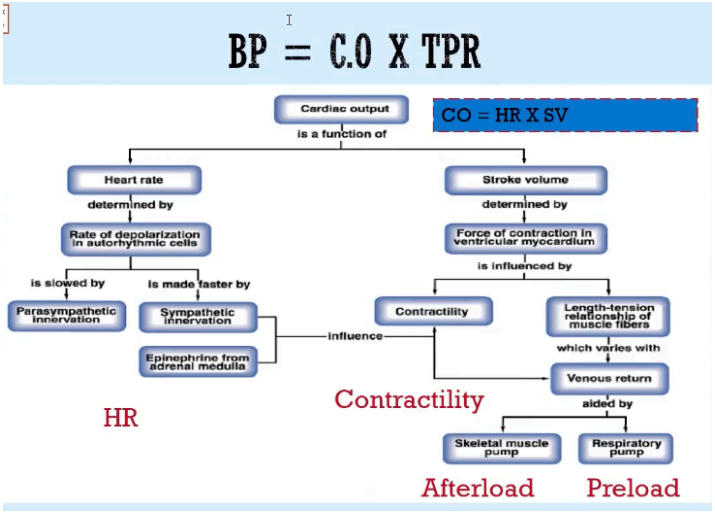
CARDIOVASCULAR PHYSIOLOGY

WIGGER'S DIAGRAM



Key Points:

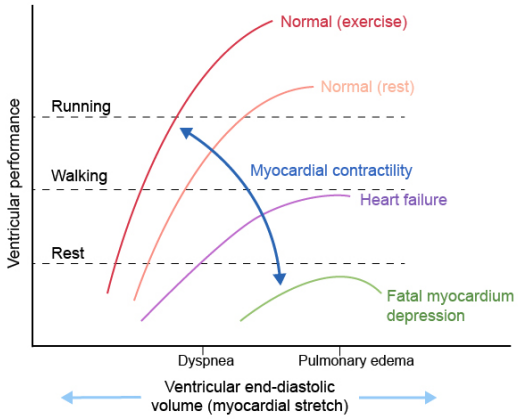
- AR = both LV and aortic pressure ↑
- AS = ONLY aortic pressure ↑



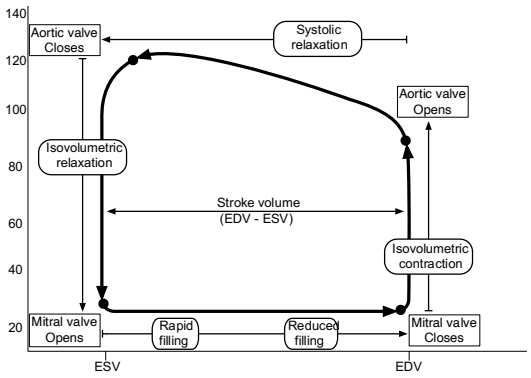
DETERMINANTS OF CARDIAC OUTPUT

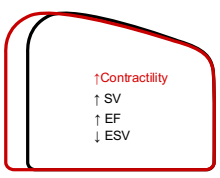
	Decrease	increased
HR	PSNS = opiate	SNS = Shock, stress
	Inotropes	BB
Preload (CVP = RAP) = *heart muscle tension or pressure filling ventricle*	Volume loss = bleeding	↑ vol. to heart = ↑RA =↑EDV (e.g. pregnancy)
	IV fluid or MTP	Diuresis
Afterload (≈TPR or intrathoracic pressure) = force needed to pump against aorta (aortic pressure)	MR Vasodilating (e.g. Anaphylaxis)	Viscous blood L-side (systemic HTN, AR, MR) R side (PHTN)
	↑TPR	Anti-coags,
Contractility = *affected by HR, preload, afterload*	Ischaemia	SNS
	Inotropes	BB, CaB
Venous return		Resp. pump SKM pump SNS systemic veins Abdo compression reflex

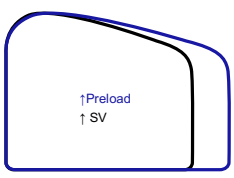
Frank-Starling Relationship

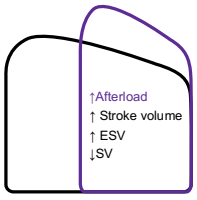


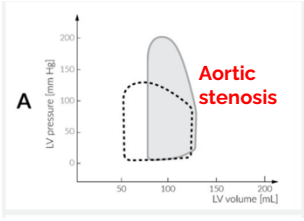
Pressure-volume Loop Diagrams

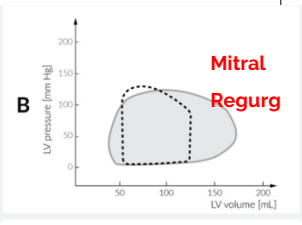


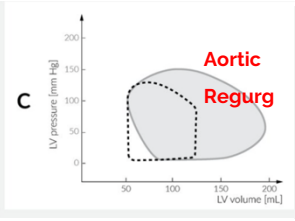


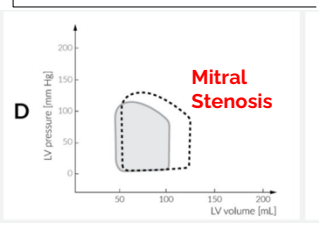


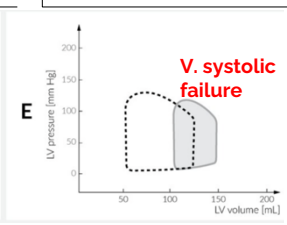


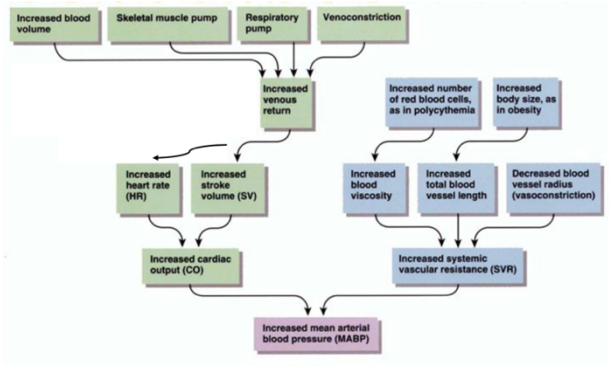






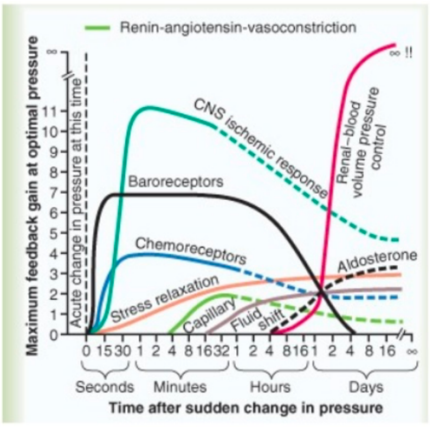






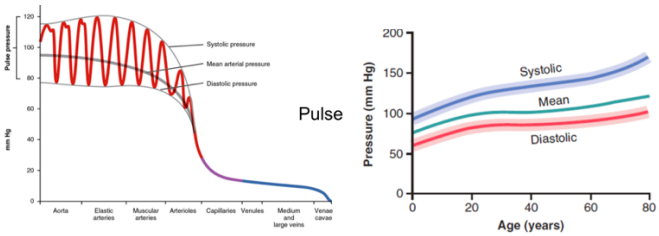
MAP = CO x TPR

Blood pressure control systems



Why does SBP increase with age?

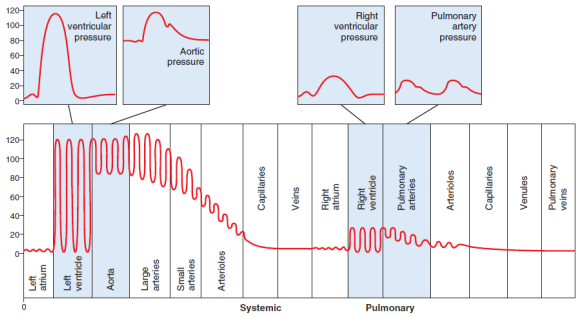
- Mainly due to atherosclerotic plaques in arteries generating greater afterload, hence more pressure
- Isolated systolic hypertension, (only when SP is elevated, DP normal) → most prevalent type of hypertension in those aged 50 or over (mostly due to calcification of large artery)



Both SP and DP increases with age

Why is pressure lower in the right side?

- Right side of heart only needs to pump to pulmonary circulation as shorter distance, compared to left side which pumps to entire systemic circulation
- Damping of pulse pressure across the vascular tree is proportional to resistance of smaller vessels and compliance of larger ones



Static vs dynamic exercise. What are the differences?

Dynamic Exercise	Rhythmic Contraction (Moving Whole Body)	• More energy for rhythmic contraction • DP does not change during aerobic exercise

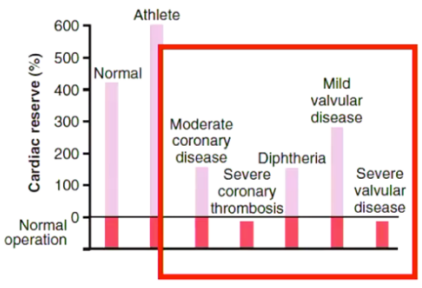
Static Exercise	Muscle Contracted And Remains In Contracted State	• ↑↑ **HR much LESS** compared to dynamic aerobic exercise
		• BUT **arterial BP** (both systolic and diastolic) for static exercise has a significantly <u>larger increase</u> DUE to difference in muscle mass
		• Engages smaller vol. of muscle mass (less energy required) and thus:
		  ○ ↓CO
		  ○ ↓vasodilation in exercising SKM (compared w/ aerobic) → ↓ TPR due to ↓ MAP since ↓ SNS → exaggerated ↑↑↑ MAP

*Both experience **<u>exercise pressor reflex</u>**

What changes would you expect to see in a patient with chronic heart failure if they exercise?

- Chronic heart failure = cardiac output cannot meet demands of vital tissues even at rest

1. *Inadequate blood supply to exercising muscles*
   - Suboptimal increase in SV, hence minimal increase in CO
   - Impaired muscular vasodilation (reduced blood flow to exercising muscles and thus cannot meet demand of the exercise)
2. *Decreased exercise tolerance*
   - Immediate SOB → Extreme and early muscle fatigue
3. *Exaggerated exercise pressor response*
   - Excessive increase in HR, BP and vasoconstriction
   - Initiated as a compensatory mechanism



*Healthy individuals have reserve in CO. Diseased individuals do not have a reserve to compensate.

ATHEROSCLEROSIS

Definition

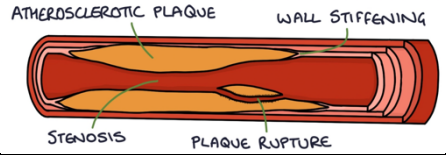
- *Chronic inflammation and activation of artery wall causing lipid deposition and fibrous plaque formation*
  - **Atheromas** (fatty deposits in artery walls) +
  - **Sclerosis** (hardening/stiffening of vessel walls)
- affects **medium** and **large** BVs

Risk factors on Hx

Non-modifiable RF	Modifiable RF	Medical RFs
• OLD age • FHX • Male	• Smoking + EtOH • High trans-fats and sugar diet • Low exercise • Obesity • Poor sleep • Stress	• T2DM • HTN • CKD • Inflammation (e.g. RA) • Atypical anti-psychotics

Comp.

- **Stiffened artery walls → HTN**
- **Stenosis of blood flow → angina AND peripheral artery disease**
- **Plaque rupture →** thrombus blocking distal vessel → ischaemia **(e.g. ACS / TIA or stroke / mesenteric ischaemia)**
  - a. Thrombus Composed of mostly platelets
  - b. **Hence antiplatelets are the best treatment**



Ix

- Regular BP check on visits
- CT Ca score

Px

Primary Prevention	Secondary prevention (4 A's)	*When to start statin – atorvastatin 20mg?*
• ***Assess QRISK 3 score = risk of MI in next 10 years*** • Wt loss • Mediterranean Diet • , exercise • Stop smoking • Stop drinking alcohol • Rx co-morbidities (DM)	• **ACEi** • **Aspirin** 75mg o.d. PO +/- clopidogrel • **Atorvastatin** 80mg o.d. PO - check LFTs before starting • **Atenolol** (BB)	• ***QRISK score > 10%*** • ***CKD*** • ***T1DM for > 10 years*** *What are the major AE of statins?* • Myopathy (check CK) • T2DM • Haemorrhagic strokes (rare)

HYPERTENSION

Def

- High blood pressure.
- BP above 140/90 in clinic or 135/85 with ambulatory or home readings.
  - **Correct cuff size**
  - **Average ≥ 2 readings on ≥2 occasions**
- Educate 1/3rd adults > 18 have HTN

Causes

Primary	Secondary	Medical RFs
• Essential (95%) - idiopathic	• RAS (bilateral), CKD • Endocrine (Conn's (2.5%), acromegaly, thytoxicosis, pheo, cushing's) • Drugs (cocaine, sympathomimetics, STEROIDS) • Aortic Coarctation (?Turner's) • Pregnancy (pre-eclampsia) • OSA / Obesity	• T2DM • HC • Obesity • CKD

Comp.

1) **Thromboembolic event (***ACS, TIA, Stroke***)**
2) **HTN retinopathy**
3) **HTN nephropathy**
4) **Heart Failure**

BLOOD PRESSURE CATEGORY	SYSTOLIC mm Hg (upper number)		DIASTOLIC mm Hg (lower number)
NORMAL	LESS THAN 120	and	LESS THAN 80
ELEVATED	120 – 129	and	LESS THAN 80
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 1	130 – 139	or	80 – 89
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 2	140 OR HIGHER	or	90 OR HIGHER
HYPERTENSIVE CRISIS (consult your doctor immediately)	HIGHER THAN 180	and/or	HIGHER THAN 120

Ix

- Vitals, BMI
- ECG
- FBC, EUC, LFT,
- B-HCG

Endocrine screen:

- **Renin-aldosterone ratio** (if ↑ renin) RAS (young female – inherited) conn (adrenal adenoma) - ↑ aldo:renin
- **TFT,**
- **24 HR urine and plasma ACTH**
- **IGF-1 assay + OGTT**
- **plasma and urinary metanephrines**

HTN nephropathy or RAS

- **Urine Albumin:creatinine ratio** (proteinuria) and dipstick (microscopic haematuria) – check for kidney damage (gn, T2DM)
- **Renal artery doppler**
- **CT adrenals – "beaded" angio for RAS**

HTN retinopathy

- **Fundus Exam** (HTN retinopathy)
  Papilloedema* = *↑ICP

Mx

Non-pharm

- ***Confirm Dx***
- ***Ix for causes and end-organ damage***
- ***Home BP measurements***

Lifestyle mod:

- Lose wt =
  - a. Low salt diet
  - b. Regular PA
- Stop smoking, EtOH,
- Reduce caffiene, drugs

Pharm

Indications:

- **All patients with stage 2 hypertension**
- **All patients < 80 yo with stage 1 hypertension** or Q-risk score of 10% or more, T2DM, renal disease, CVD or end organ damage.
- **Check compliance**

Medications

- **A – ACEi** (e.g. ramipril 1.25mg up to 10mg od)
- **B - BB** (e.g. bisoprolol 5mg up to 20mg od)
- **C – CaB** (e.g. amlodipine 5mg up to 10mg od)
- **D – Thiazide-like diuretic** (e.g. indapamide 2.5mg od)

Key considerations

Alternatives:

- ARB used – if ACEi not tolerated or African-Caribbean descent

K+ balance

- **K+ spraing diuretic = if serum K+ < 4.5mM**
- A-blocker or B-blocker = if **serum K + > 5.5mM**

Rx targets

Age	Systolic Target	Diastolic Target
< 80 years	< 140	< 90
> 80 years	< 150	< 90

Hypertnesion VS Hypercholesterolemia GP APPROACH

Red Flags

HYPERTENSION
Symptomatic HTN (> 180/110) + end-organ dysfunction
- IHD = chest pain, palpitations, diaphoresis, N/V
- TIA/ Stroke = frontal headache, visual disturbance (photophobia, blurred), FND (slurred speech)
- CKD = Proteinuria,

DYSLIPIDEMIA
Abnormal Tg and LDL
- Triglycerides > 10mM
- Total cholesterol > 8mM

URGENT REFRRAL TO LIPID CLINIC

RF

HYPERTENSION
- ***Modifiable*** = T2DM, HC, low PA, high caloric diet, high BMI, smoking, EtOH
- ***Non-modifiable*** = genetic
- ***FHx:*** *HTN, Dyslipidemia, stroke, diabetes, early coronary artery disease, CKD (ADPKD)*

CV RISK Calc (5-10 year risk of ACS/stroke)
- Age (> 45)
- Sex (M > F)
- SBP > 160
- SMOKING
- ECG = LVH
- T2DM
- ++ Total TC, HDL > 7.5

DYSLIPIDEMIA
Automatic high risk
- *DM + > 60yo*
- *DM + albumin > 30*
- *eGFR < 45*
- *FHx of dyslipidaemia (familial HC)*
- Grade 3 HTN (>180/110)
- Hyperlipidaemia(TC > 7.5)

S+S / Exam

HYPERTENSION
- Chest pain
- palpitations
- Headache
- Oedema
- Claudication
- HR, rhythm, character
- JVP
- Obesity (BMI, WHR)
- ABPI difference

Assess for organ failure
- **Cardiac enlarged** (displaced apex beat, S3/4)
- **LHF** (bibasal crackles, oedema, pulsatile liver)
- **Enlarged kidneys** (ADPKD) - ?bruits
- **Eye abnormalities** (e.g. retinal haem, AV nipping, HTN retinopathy
- **Idiopathic intracranial HTN**
- **Thyrotoxicosis**
- **Pulsatile abdo mass (AAA)**

DYSLIPIDEMIA
Screen based on CV risk category for 45-75 yo or 30-75 ATSI

Low CV risk	<10% CVD abs risk in next 5 years	**Every 2 years lipids check** • **Lifestyle advice**
Moderate CV risk	10-15% CVD abs risk in next 5 years	**Every years lipids check** • **Lifestyle + statins + anti-HTN**
High CV risk	>15% CVD abs risk in next 5 years	**Every 6/12 lipids check** • **statins + anti-HTN**

DDX

HYPERTENSION
- **Idiopathic – primary/essential HTN**
  - White-coat HTN
- **Secondary HTN: (RED CAP OR)**
  - Renal (RAS, Renal vein thrombosis, CKD, AKI)
  - Endo (cushing, conn's, pheo, acromegaly, T2DM)
  - Drugs (*cocaine, ecstasy, thyroxine)*
  - Coarctation of aorta
  - Pregnancy
  - OSA ➔ PSG ➔ CPAP
  - Raised ICP (Cushing's triad ➔ irregular RR, HTN and bradycardia)

DYSLIPIDEMIA
Rule out secondary causes of HC
- Hypothyroidism (↑ LDL)
- Nephrotic syndrome
- Cholestatic biliary disease OR acute hepatitis
- Hyperparathyroidism
- AN
- Porphyria
- Familial HC
- Meds (e.g. thiazides, BB, oral E2, protease inhibitors, EtOH)

Comp.

HYPERTENSION
- IHD = chest pain, palpitations, diaphoresis, N/V
- TIA/ Stroke = frontal headache, visual disturbance (photophobia, blurred), FND (slurred speech)
- CKD = Proteinuria,

DYSLIPIDEMIA
- IHD = chest pain, palpitations, diaphoresis, N/V
- TIA/ Stroke
- Chronic liver disease - MAFLD
- CKD = Proteinuria,

Ix

HYPERTENSION
- **FBC, EUC, LFT**
- **Fasting lipids, BSL (HbA1C)**
- **Urine dipstick (ACR, MSU)**
- **12-lead ECG + ECHO**
- **2x ave seated BP 1-2mins apart** (after resting for 5 mins)
- **Repeat BP on 2x separate occasions** (at least 1 week apart)
- **Ambulatory or Home BP monitoring**

Heart foundation classification

Optimal	**< 120/80**
Normal	> 120/80
High normal	> 130/85
Grade 1 (mild)	> 140/90
Grade 2 (mod)	> 160/ 100
Grade 3 (severe)	> 180/110

DYSLIPIDEMIA
Rule out secondary causes of HC
- BMI + Vitals = BP, HR, RR, Sats
- FBC, EUC, LFT
  - Check CK if using statins (stop if CK > 1000)
- Fasting BSL and HbA1C
- Fasting lipids
- TFT, PTH, CMP and Vit D
- Urine dipstick → proteinuria

Mx

HYPERTENSION
Patient-specific APPROACH TOWARDS PREVENTION + Mx

Educate
- Ongoing education ➔ **Info booklets – GP healthcare pathway**

Goal:
- <130/90 (in all w/ diabetes)
- Urine ACR < 2.5 mg/mM

Non-pharm (lifestyle)
- 5-10% of Wt loss (BMI < 25, reduce WHR < 94 M, < 80 F)
- Na restricted diet (↓processed food intake)
- Nutrition (dietician)- ↓ caffeine, ↑Ca, ↑Mg
- Exercise (30 min regular PA x5/week)
- Avoid alcohol, smoking
- Tranquil – reduce stress

Pharm

Diuretic + ACEi Ai

1st line = ACEi/ARB	*cough, angioedema, hyperK* (avoid pregnant) • Check EUC before and after ACEi • Check then every 6/12
2nd line = BB	*postural HypoTN, X-asthma*
2nd line = CaB (DHP)	**peripheral vasodilatation** (*peripheral oedema, flushing, hypoTN, reflex tachy* *for diabetes mellitus and CAD*
2nd line = CaB (N-DHP)	**↓vent. Contraction** ➔ *AV block, HF, constipation, bradycardia*
3rd line = thiazide	↑HyperGLUC, ↓K, ↓Na, postural HypoTN (avoid GOUT) **(for** *for stroke, HF)*

*__AVOID__ triple whammy (ACE/ARB, NSAID, diuretic)

ALWAYS check compliance **before adding new anti-HTN

DYSLIPIDEMIA
Patient-specific APPROACH TOWARDS PREVENTION + Mx

Educate
- Ongoing education ➔ knowledge, attitudes (info)

Goal:
- TC < 4, HDL > 1, LDL < 1.8, Tg , 1.7

Non-pharm (IF LOW HDL or high Tg)

Primary prevention = 3-6/12 of lifestyle interventions -start slow and steady [NEAT]
- ↓wt loss (↓WHR)
  - ↑PA -regular 30 min 5x/week
  - Intermittentfasting or DASH diet (low salt, trans fats + increase oily fish consumption)
- Stop smoke,
- Low risk ➔ lifestyle + routine screen
- Mod risk ➔ lifestyle + recheck 6/12 (LFT, Fasting lipids)
- High risk ➔ lifestyle, medications and recheck 6/12

Pharm [If high LDL]]

Secondary prevention (recheck every 3 mths until controlled)

1st line = Statins *80mg atorvastatin PO od* *Check EUC and LFT* ***before*** *starting*	*HMG-CoA reductase inhib* (• Once daily at night → forever • **A/E** = N/V, diarrhea, raised ALT • Must stop 3/12 before conception (teratogenic)
***2nd line** = fibrates*	*↑VLDL breakdown via lipase*
***3rd line** = Bile acid binding resin*	**Cholestyramine (adjunct to statins)** ➢ **A/E** = constipation
3rd line = PCSK9 inhibitors (IM)	*3/12 injections of alirocumab (Parluent) or Evolocumab (Repatha) for Familial HC*

****If dark urine + muscle pain + myalgia ➔ rhabdo ➔ seek urgent medical advice***

****Avoid statins in low-risk CVD + pregnancy***

FU

HYPERTENSION
- ED (ambulance) ➔ > 220/140 ➔ Hypertensive ED ➔ immediate Rx (aim < 160/110) (**avoid rapid fall in BP as this will increase risk of ischaemic stroke**)
  - 5-10mg Amlodipine Oral
  - 10-20mg Nifedipine MR oral
  - 10-20mg Methyldopa oral
  - 0.5mg Prazosin oral
- **Dietician + exercise physiologist**
- **Non-urgent cardiology + ophthalmology referral** (if CV risk is high)
- Refer to **Nephrology / endocrinology** = medication resistant HTN after 3/12

DYSLIPIDEMIA
- Dietician + exercise physiologist
- MH psychologist
- Non-urgent cardiology referral (if CV risk is high)

For aged > 75 ➔ consider:
- Co-morbidities
- Polypharmacy
- Risks and benefits
- Life expectancy

Urgent lipid clinic referral if:
- Fasting Tg > 10mM
- CK > 1000 (after statin therapy

Familial HC pathway:
- Total Chol > 8 or >6 (in FHx of premature IHD)

Legal/ SCREEN

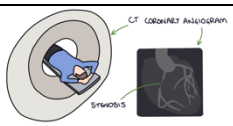
HYPERTENSION
- **Check EUC, LFT, lipids and BSL every 3-6/12**
- **General Screen** = FOBT (Bowel), MMG (BC), CST (Cervical), Skin check (melanoma)
- **OSA – sleep study, CT ca score (atherosclerosis of CAD)**
- **2-yearly total CVD risk assessment** from 45-75 yo or 30-75 yo (ATSI)
- **IUTD** (flu, pneumococcal, COVID-19)

DYSLIPIDEMIA
- **Check lipids every 6/12 – monitor for myalgia and side effects**
- **Screen** = FOBT (Bowel), MMG (BC), CST (Cervical), Skin check (melanoma)
- **OSA – sleep study, CT ca score (atherosclerosis of CAD)**
- **2-yearly total CVD risk assessment** from 45-75 yo or 30-75 yo (ATSI)
- **IUTD** (flu, pneumococcal, COVID-19)

CORONARY ARTERY DISEASE

	Stable Angina	Acute Coronary Syndrome (UA, NSTEMI, STEMI)
Def	Narrowed coronary artery supplying mycoardium	• **Type 1 MI (classical)** THROMBUS from atherosclerotic plaque • **Type 2 MI:** secondary to increased demand or reduced supply of oxygen (e.g. severe anaemia, tachycardia or HypoTN) • **Type 3 MI:** Sudden cardiac death • **Type 4 MI:** assoc. with PCI / coronary stunting / CABG • **Type 5 MI:** assoc. after surgery

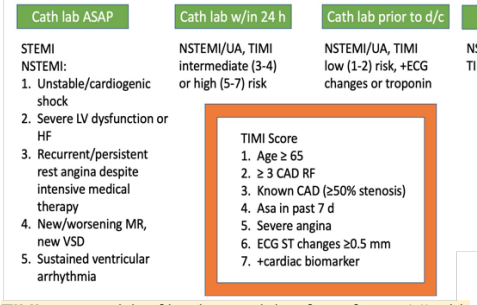
Comp.	Future ACS
	HF - Reduced functional capacity
		• **D –** Death
		• **R** – Rupture of the:
		  ○ **heart septum** (TAMPONADE) or
		  ○ **papillary muscles** (MITRAL STENOSIS)
		• **E –** "Edema" (Heart Failure)
		• **A** – TachyArrhythmia (VT/VF) and Aneurysm
		• **D** – Dressler's Syndrome (post-mI syndrome) – 2-3 wks post MI causing localised immune response causing pericarditis (pleuritc chest pain and pericardial rub)
		  ○ **↑↑CRP/ESR**
		  ○ **ECG = widespread ST elevation + T wave inbversion**
		  ○ **ECHO** = Pericardialc effusion
		  ○ Rx: **NSAID** → **steroids** (prednisolone) → **pericardiocentesis** (if needed)

H+E	Pain on exertion Relieved w/ rest and GTN	• **Classic MI signs** (nausea, diaphoesis, rad chest pain to jaw) • Unrelieved with rest or GTN
Ix	• HF signs, BMI • ECG • FBC (anaemia) • EUC (ACEi, other meds) • LFTs (prior to statins) • Troponin • Fasting lipids + BSL + HbA1C • TFTs • **CT coronary angiogram (gold std)** 	• ECG • FBC (anaemia) • EUC (ACEi, other meds) • LFTs (prior to statins) • Troponin • Fasting lipids + BSL + HbA1C • TFTs **PLUS**: ➢ CXR (?APO, pneumonia) ➢ TTE (?functional damage) ➢ **CT coronary angiogram** (CAD) **What ALSO causes raised troponins?** 1) CKD or chronic HTN 2) Sepsis / infection 3) Myocarditis 4) Aortic dissection 5) PE
Mx	**R**efer to • cardiology (urgently if unstable) **A –** Advise – safety net • pt about dx, Mx and when to call 000 **M –** Medical treatment **ASAP** GTN spray (repeat every 5 mins) **Long-term** BB (5mg bisoprolol PO o.d.) or CaB (5mg amolidipine PO o.d.) **P –** Procedural/surgical Mx **When Ind.?** PCI: Proximal or extensive disease on CT cardio angio; CABG: Severe stenosis **How?** 2x scars PCI: • Feeding Catheter into **brachial** or **femoral artery** w/ XR guidance • Balloon dilitaiton → stent insert; CABG: • **Midline sternotomy** • **Graft vein** (usu. great saphenous vein) – inner calf scar **Disadv.** PCI: Required IVR training; CABG: Slower recovery + higher comp. rate	**Unstable Angina** • Normal • Stratify risk using **TIMI score** **NSTEMI** • ↑Troponin (within 12 hrs) • ST depression + deep T wave inv. • Path. Q waves (deep infarct – late sign) • Stratify risk using **TIMI score** BATMAN-O • **BB** (unless CI) • **Aspirin** 300mg STAT • **Ticargrelor 180mg STAT (or clopidogrel 300mg)** • **Mopphine** • **Anti-coag** (LMWH) • **Nitrates** (relieve coronary spasm) • **O2** (only if sats < 95%) **STEMI** • ST elevation + ↑Troponin • New LBBB 1) **Primary PCI** (< 2hrs of STEMI)) 2) **Thrombolysis** (> 2hrs of STEMI) using streptokinase, alteplase
Px	See atherosclerosis Px **Additional Ix:** Stress ECHO PET (best if obese or extensive infarcts) Cardiac MRI (? Dead or viable myocardium)	• **Aspirin** 75mg PO od +/- antiplatelet e.g. clopidogrel for 12 months (**DAPT**) + **anti-coags** (LMWH) • **Atorvastatin** 80mg PO od (↓ Atherosclerosis) • **ACEi** (10mg ramipril PO od) (↓ Remodelling) • **Atenolol** or 25mg metoprolol PO qid (↓ VT/VF risk) • **Aldosterone** (50mg eplerenone PO od) – if heart failure

STEMI progression		Heart Area	Which part?	ECG Leads
1)	**Left Coronary Artery**	Anterolateral		I, aVL, V3-6
2)	**LAD**	Anterior	LV + septum	V1-4
3)	**Right Coronary Artery**	Inferior	RA + LA SA/AV nodal block	II, III, aVF
4)	**Left Circumflex**	Lateral	LA + posterior LV (major infarct)	I, aVL, V5-6
5)	**Posterior desc. Artery**	Posterior	RA + LA	V1-v6 (ST depress)

The HEART Score for Chest Pain Patients in the ED		
History	• Highly Suspicious • Moderately Suspicious • Slightly or Non-Suspicious	• 2 points • 1 point • 0 points
ECG	• Significant ST-Depression • Nonspecific Repolarization • Normal	• 2 points • 1 point • 0 points
Age	• ≥ 65 years • > 45 - < 65 years • ≤ 45 years	• 2 points • 1 point • 0 points
Risk Factors	• ≥ 3 Risk Factors or History of CAD • 1 or 2 Risk Factors • No Risk Factors	• 2 points • 1 point • 0 points
Troponin	• ≥ 3 x Normal Limit • > 1 - < 3 x Normal Limit • ≤ Normal Limit	• 2 points • 1 point • 0 points
Risk Factors: DM, current or recent (<one month) smoker, HTN, HLP, family history of CAD, & obesity		
Score 0 – 3: 1.7% MACE over next 6 weeks; Score 4 – 6: 16.6% MACE over next 6 weeks; Score 7 – 10: 50.1% MACE over next 6 weeks		

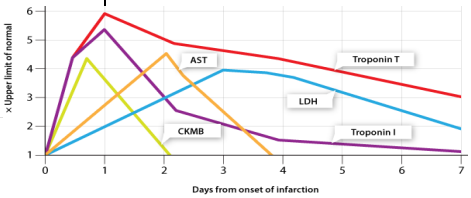
HEART score = stratify low, intermiedate or high risk of future ACS in next 6 weeks



TIMI score = risk of having or dying from future MI with UA/NSTEMI

Beware of atypical MI:
1) Silent MI (T2DM)
2) Vasculitis
3) Prinzmental angina (coronary spasm)

Elevated cardiac markers



*Troponin rises 4-8 hrs post Sx (peaks at 12-24 hrs)

CHRONIC HEART FAILURE

Definition & Types

- Supply demand mismatch - inadequate oxygen delivery to tissues

Framingham criteria (2 major OR 1 major + 2 minor)

- **Major** = PND, orthopnoea, JVP distension (or distended neck veins), APO signs, 3rd heart sound (on inspitation),
- **Minor** = SOBOE, ankle odema, tachycardia (? 120bpm) , nocturnal cough, + HM, pleural effusion,

Types

	Class	EF	Cause	Treatment
Diastolic Dysfunction • Restricted cardiomyopathy • parallel sarcomere • uncontrolled HTN → ↑ afterload • concentric hypertrophy = ↑ stiffness ↓ compliance = ↓ relaxation = ↓ LVEDV	HFpEF	≥50%	• HOCM (LVH) – S3 • Aortic Stenosis • **HTN (most common cause) -S4** • **Amyloidosis** → AL → MM • **Sarcoidosis** • **Haemochromatosis** = *symmetrical arthropathy 2nd, 3rd MCP + fatigue, abdo pain + hepatomegaly*	**Limited Rx:** • manage fluid status • avoid exacerbators (e.g. AF)
	HFpEF [borderline]	41-49%		
	HFpEF [improved]	> 40%		
Systolic HF • Dilated cardiomyopathy • sarcomeres in series • ↑ compliance = ↓ contractility • ↓CO = ↑ LVEDV = ↑ PCWP = ↑ TPR	HFrEF	≤40%	• **Toxins** (alcohol, doxuribicin) • **Infection** (Chagas, Coxsackie B) • **Malnutrition** (B1 def.) • **STEMI** ➔ cardio shock ➔ ↓ CO ➔ ↑ LVEDV ➔ ↑ PCWP	**Yes** • **↑ prognosis** = BB, ACEi, aldo antags, hydralazine • **↑ symptoms** = diuretics, digoxin +/- nitrates

Causes

Pathophysiology:

- ↓ contractility = ↓ CO = ↓ map
  - Decompensation with ↑ LVEDV
- RAAS activation = ↑ Ang II = ↑ Aldosterone = ↑ systemic vasoconstriction
- ↑Na reabsorption + ↑ eGFR (due to ↑ efferent arterioles constriction/tone)

HIGH *output states*

- **Sepsis**
- **Shunt/Fistulas** (shunts from high pressure to low pressure = ↓ TPR = ↑ HR = ↑CO **BUT** ↓ TPR= ↓ organ perfusion = HF)
- **Anaemia**- treat Fe deficiency in people with CHF
- **Thyrotoxicosis/hyperthyroidism**
- **Vit def. (e.**g. Beri Beri - thiamine)

LOW *output states*

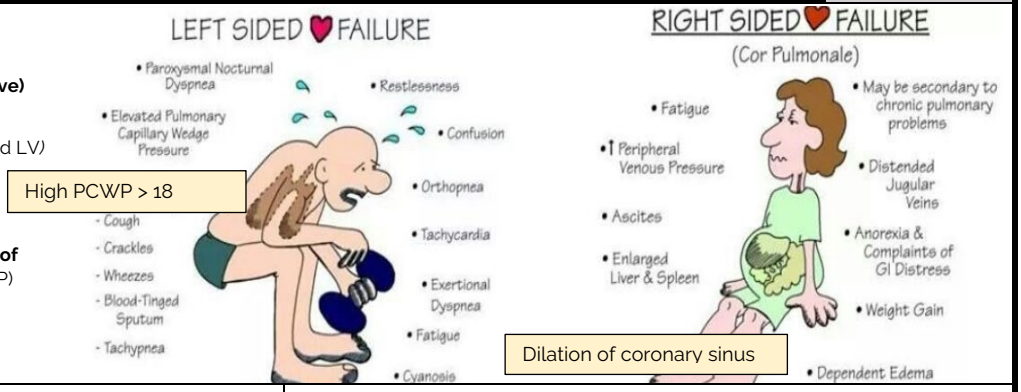
- Valvular disease
- Structural disease (e.g. **Cardiomyopathy)**
- Acromegaly
- Hypothyroidism

Classify [NYHA]

Class 1:	Normal	Asymptomatic LV dysfunction
Class 2:	*Moderate* Exertion causes SOB, angina pectoris	**Mild** CHF
Class 3:	*Small* exertion causes symptoms	**Moderate** CHF
Class 4:	*Any* exertion causes symptoms	**Severe** CHF

Signs

- **Raised JVP (94%)**
  Peripheral oedema + weight gain
- **Displaced apex beat (most Sensitive)**
- ***S4 (atrial gallop)*** = *diastolic HF*
- ***S3*** = **↑ diastolic filling** due to dilated LV) → *MR, AR, LVF*
- **Heamodynamic issues** (e.g. HTN, arrhythmia)
- **Pulm. Crepitations = transudation of plasma into alveoli (due to** ↑ PCWP)
  Inspiratory basal crackles = Left HF



Ix for suspected CHF

General tests for heart failure:

1. **FLUID STATUS**
2. **FBC, EUC** = repeat 6/12 in patients with stable CHF
   a. **Aneamia**
   b. **LFT: hypoalbuminemia (***malnutrion, cirrhosis, nephrotic syn.***)**
3. **ECG [NON-SPECIFIC changes]** = adds to Dx (e.g. MI, LBBB, AF, VT) but cannot rule in HF
4. **CXR** = **Rule in** CHF [NB: normal CXR does NOT rule out CHF]
   a. ***Alveolar and Interstitial shadowing (oedema)***
   b. ***Kerley B lines –*** *lymphatics filled up* ***Bat-wing appearance***
   c. ***Cardiomegaly*** *(cardio-thoracic ratio > 50%)*
   d. ***Pulm venous changes (H****ilar opacities w/ upper lobe diversion)*
   e. ***Pleural effusion (****blunting of costophrenic angles)*
5. **ECHO [MOST USEFUL]**= assess cardiac structure/fn (confirm systolic LV dysfunction) → However, do simpler tests 1st Bloods, ECG, CXR

POOR PROGNOSTIC INDICATORS:

1) Low BP
2) Low VO2
3) HypoNa
4) High BNP

General order of tests:

BLOODS → ECG → CXR → ECHO → CTA→ STRESS TEST or Plasma BNP

Plasma BNP → released BY ventricles when stretched DURING filling

- **BNP Vasodilates through ↑↑cGMP = ↓ PCWP**
- **< 100 pg/mL** = rules **out** dyspnoea caused by CHF (NB: also due to *obesity, RAAS drugs, diuretics, stunning*)
- **> 400 pg/mL = confirms CHF ➔ nitrates, diuretics** (Elevated also due to *female, old age, PE, MR, AF*)

- **Troponin →** *non-specific for STEMI* (also due to PE, myocarditis, arrhythmias)
- **Stress test** = unexplained SOB to exc. ischeamic cause of CHF
- **Endomyocardial biopsy** = *Prussian blue stain -> Leaked RBC due to cap rupture → excess Fe broken down into* ***hemosiderin*** *by alveolar macrophages [****feature of Left HF = hemosiderin-laden marcophages****]*
- ***Spirometry*** = *exc. COPD, asthma* (before CABG)
- **TFT** = exc. thyrotoxicosis, hypothyroid
- **LFT** = exc. congestive hepatomegaly
- **EPG + LIGHT CHAIN**
- **A-GALACTOSIDASE = ?FABRY's**
- **Sputum M/C/S**

Rx (non-pharm)

NUTRITION	• Low GI, trans-fats diet • Reduced processed foods • Salt restriction (<2g/day)	**Control /Optimise**	• HTN, HC • T2DM • Reduce weight (> 10%)
EXERCISE	• 30mins 5x day/week (regular + doable)	**Prevention**	• IUTD = flu, COVID, Pneumococcal • Rx triggers = arrythmias, hypoK, anaemia, MI, ADRs, infection, thyrotoxicosis, fluid overload
AVOID	• Limit coffee (<1-2x/day) • Smoking, • EtOH *< 4 max on 1 setting, < 10/week) • Illicit drugs	**FU (Every 3/12)**	• FBC, EUC, LFT • Fasting Lipids, BSL, HbA1C • TFT + urinalysis • Viral serology (viral myocarditis) • Coronary Angio + ECHO (Every 2 years)
TRANQUIL	• Reduce stress	**Referral**	• **GPMP –** cardiologist, respiratory physician (OSA)., endocrinologist • PT/OR, rehab program, dietician, psychologist

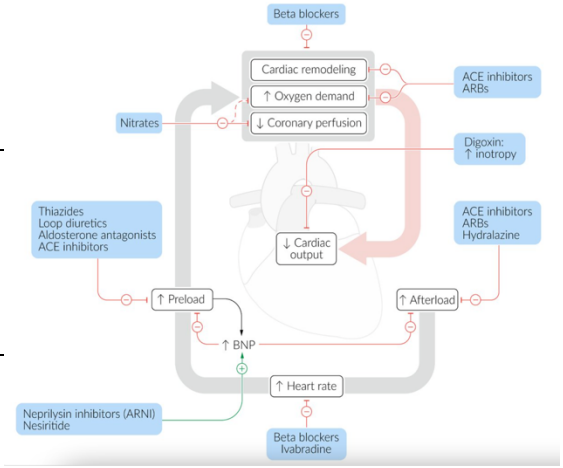
Rx (pharm)

1) ACEi/ARB
rampril, candesartan (up to 32mg)

- **1st line = ACEi > ARBs in HFrEF patients** = Vasodilation to reduce cardiac afterload by inhibiting RAAS. **(↓ preload + ↓ afterload, cardiac remodelling)**
- Monitor EUC closely
  - **A/E** = cough, hyperK (>5mM), postural HypoTN
  - **CI** = ARB > ACEi in pregnancy (teratogenic) & elderly (avoid angioedema) | Avoid BOTH in bilateral renal artery stenosis

2) BB
[nebivolol, metoprolol, bisoprolol best]

- **For HFrEF (systolic HF) (↓ preload + ↓ afterload)** → minimise chronic NA/A exposure = minimise systemic HTN + myocardial remodelling
  - **Also for: stable IHD (angina) to** ↑ exercise tolerance + **↓ angina episodes**
- **AE = hypotension, fatigue, ED** → AVOID in ***acute decompensated HF*** (due to -ve ionotropic effect)



3) Diuretics
Aldosterone antagonist

- Reduced EF unresponsive to ACEi/ARB or BB
- Monitor EUC closely
- **AE = hypokalemia, met. alkalosis, hypoCa, hypoMg, ototoxic**

4) Ivabradine

- **For severe HFrEF <35% [systolic HF] or if BB ineffective** [**NOT** for ***AF, paced, acute MI, shock, long QT, bradyacardia***]
- Inhibits "funny" channels in SA node = reduce automaticity
- **ONLY -ve chronotrope** (↓HR) → ↑ LV output = Nil effect on BP

- **AVOID CaB** = -ve ionotrope = ↑ risk of CHF due to bradycardia and **↓ contractility**
- **AVOID Triple Whammy – ACEi + Diuretic + NSAID**

5) Vasodilators

If ACEi/ARB not working

- Nitrates **(↓ preload) = venodilates (relax coronary vessels)**
- **Hydralazine (↓ afterload) – A**/E = flush, headache, lupus-like Sx

6) Entresto

ARB + neprilysin inhibitor → **inhibits** breakdown of BNP→ increased Na/water loss

7) Ionotropes

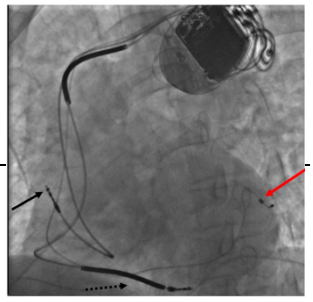
Rescues therapy for HF BUT Bad for myocardium → ↑ mortality post-discharge

- **Dobutamine** = pure B agonist to ↑contractility
- **Levosimendan** = ↑ calcium sensitivity in cardiomyocytes by binding troponin C
- **Digoxin = block Na/K → hypoK, bradycardia, ST depression, reverse tick, visual disturbance, confusion, GI upset**

Rx (surgery)

Current devices

- **Pacemakers (alternative)** → bradycardia, AV block, sinus node issue (e.g. sick sinus syndrome)
- **AICD (automatic implantable cardioverter defib.)** → **Indications for CHF patients** with:
  - 1° prevention = **LVEF ≤ 35% + NYHA class II-III** [*not class IV = too ill*]
  - 2° prevention (happened already)
    - Hx of cardiac arrest (due to VF or VT)
    - Spontaneous sustained VT + structural CHD + Brugada syndrome
    - LVEF ≤30% when stabilised post MI or revascularisation



CRT
Cardiac resynchronization therapy

- **Biventricular pacing Criteria**
  - CHF [class III–IV] - very ILL
  - LVEF ≤ 35%
  - QRS interval ≥ 120 ms (i.e. LBBB + Cardiomyopathy)
  - sinus rhythm

MDT approach

- **HF specialist units** → Reduce admission rates → improve patient outcomes
  - 1st week nurse consult ➔ med compliance/ symptom control before next cardiologist appointment
  - Minimise hospitalisation due to VF/VT-induced cardiac arrest

Referral When?

- Dx is uncertain
- **Complex Mx** needed
- Revascularisation, device implantation or heart +/- lung transplantation **NEEDED**
- < 65 years of age

Advanced treatment options in HF patients

- Left ventricular assist devices (LVADs)
- Hemofiltration
- Cardiac transplant [St Vincent's]

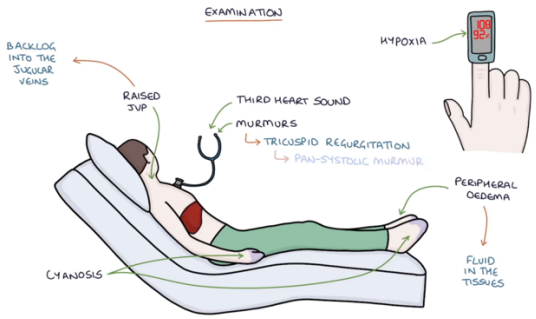
Transplant Indication

- **NYHA class 3-4,** unfixable HF or congenital HD, unresolvable **ventricular** arrythmias, **HCM, HFrEF** – systolic HF]
- **CI:** *>70, obese, sig. co morbidity (eg malignancy, diabetes + renal failure), psychological issues, non-compliance*

ACUTE LVF & PULMONARY OEDEMA vs COR PULMONALE

	COR PULMONALE	ACUTE LVF
Def	• Right-sided RF casued by respiratory disease • Backfkwif of blood into RA, vena cava and systemic venous system	1) LV dysfunction causing backflow of blood into LA, pulmonary veins and lungs 2) ↑ Intravascular pressure = interstitial fluid leakage into alveoli → Pulmonary oedema 3) Impaired gas Xchange = SOB, ↓ desats
RF.	**COPD (most common cause)** **PE** **ILD** **Cystic fibrosis** **Primary PHTN**	**Iatrogenic** (aggressive IVF in frail elderly pts w/ impaired LV function) **Sepsis** (fever) **MI** **Arrythmias** (palpitations)
H+E	Asymptomatic SOB + /- chest pain (hard to delineate w/ resp/ disease) Peripheral oedema Syncope (dizziness and fainting) --- Hypoxia, cyanosis, Raised JVP, 3rd Heart sound Murmur (pan-systolic = TR) Pulsatile Hepatomegaly (> 15cm liver span)	Acute onset SOB – worse supine Frothy white/pink sputum cough --- 3rd Heart Sound Bilateral bibasal crackles (sounds "Wet")

Ix	• **Vitals** (↑HR ↑RR, ↓sats)
	• ECG
	• FBC (anaemia)
	• EUC (ACEi, other meds)
	• LFTs (prior to statins)
	• Troponin
	• Lung function test
	• CTPA or V/Q scan
	• R heart catherisation
		• **Vitals** (↑HR ↑RR, ↓sats)
		• **ECG (ischaemia or arrythmias)**
		• **FBC (infection)**
		• **EUC (kidney function)**
		• **ABG -** Type 1 Respiratory Failure
		• **BNP (***high sensitivity for HF***)**
		  ○ *Released from heart ventricles when myocardium stretched beyond normal range*
		  ○ *BNP → SMC relax in BV → reduce systemic TPR + diuresis via kidneys*
		  ○ **May also be raised in sepsis, tachycardia, PE, AKI, COPD**
		• **Troponin**
		Imaging:
		• **CXR (***check APO***)**
		  ○ **Alveolar shadowing**
		  ○ **Kerley B lines (fluid in septal lines)**
		  ○ **Cardiomegaly**
		  ○ **Upper lobe venous diversion**
		  ○ **Pleural effusion**
		• **TTE (***check EF***)**

Mx	1) Rx symptoms and underlying cause
	2) Long-term O2 therapy
	3) Poor prognosis unless able to reverse underlying cause
	 
		<u>Check fluid status & Vitals (S-LMNOP)</u>
		➢ **Stop IVF**
		➢ **Lasix** (IV 40mg Furosemide STAT)
		➢ **Morphine**
		➢ **Nitrates**
		➢ **Oxygen** (if sats < 95%)
		➢ **Position upright** (clears fluid from upper lobe for gas exchange)
		
		<u>Additional Mx</u>:
		➢ ICU transfer
		➢ **CPAP** or **I•V** – splint open airways to improve gas exchange
		➢ **Inotropes** (e.g. dobutamine = pure B agonist to improve contractility but worse prognosis)
		
		<u>Long-term Mx:</u>
		➢ Fluid balance = measure intake, UO, daily wts
		➢ Regular bloods – FBC, EUC,
		➢ Fluid restriction 1L/day
		➢ Low salt diet
		➢ F/U ECHO + cardiologist
		➢ *<u>Meds</u>*:
		  ○ **Aldosterone** (take AM to avoid nocturia)
		  ○ **BB** (metoprolol – easier to titrate)
		  ○ **ACEi** (day 4-5)
		<u>Common scenario:</u>
		➢ Frail 85yo lady w/ CKD and AS given 2L fluid over 4 hrs has sudden sats drop
		➢ Rx: IV 40mg Furosemide

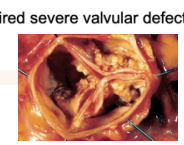
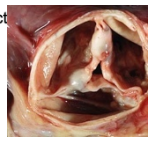
VALVULAR HEART DISEASE/ MURMURS

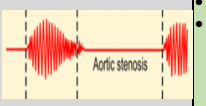
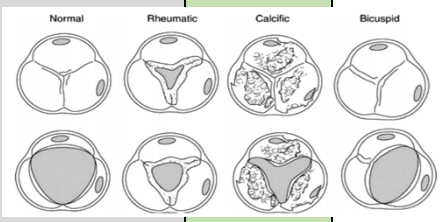
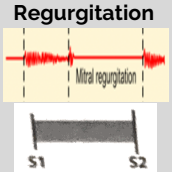
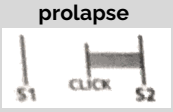
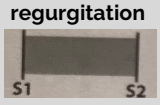
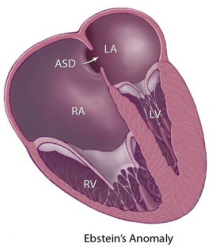
Main causes = (1) advanced age, (2) congenital (CT disorder), (3) acquired (infection, PHTN, ACS)

What is the commonest congenital valvular heart disease lesion
- A. Congenital tricuspid regurgitation
- B. Bicuspid aortic Valve
- C. Pulmonary stenosis
- D. Ebsteins anomaly

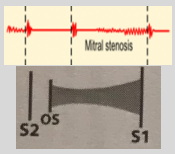
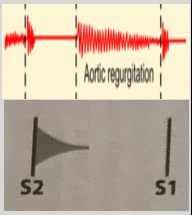
Which of the following is a major criteria for infective endocarditis
A. Predisposition / predisposing heart condition, or IVDU
B. Fever, temperature >38C
C. Two Blood cultures positive with typical organisms
D. Vascular phenomena:
E. Immunological phenomena:

What is the commonest acquired severe valvular defect
A. Severe mitral stenosis
B. Severe Aortic regurgitation
C. Severe Aortic Stenosis
D. Severe Mitral regurgitation

Valve disease	Murmur character	Best heard?	PP	Symptoms	Signs	Causes	Rx
Systolic (radiate)							
Aortic stenosis **MOST COMMON**  	**Ejection systolic** *DDx:* • MR • Aortic sclerosis (no radiation)	Upper RSE (→ carotids and apex) Outflow tract (Left lower SE) **AMPLIFY:** [expiration, squatting]	*Effects of severe AS:* • **"Stiff" noncompliant LV** = Chronic LV overload = ↓ CO = LVH • **Well tolerated until LV fails = CHF** • **If AF** = ↓ atrial kick = ↑ HR = APO + HypoTN *3 types:* • **Valvular** (most common) • **Sub-valvular** (subaortic) = HOCM (left outflow tract) • **Supravalvular** (Williams syndrome → affects asc. aorta)	***Mild AS*** = *ASYMPT +easy fatigue* ***TRIAD - Mod As***: 1. Angina/chest pain (**coronary** perfusion impaired) 2. CHF - **Exertional SOB** 3. **Syncope** on exertion ***Severe AS:*** • Sudden death • Severe L HF • Weak pulse Can lead to *aortic aneurysms*	• Slow rising pulse • Narrow pulse pressure • **LV thrust apex beat** • **Soft or absent S2** (severe) • **Systolic thrill** at aortic area (R. sternal edge) • **Later the murmur peak = more severe** • Reverse split S2 *Imaging:* • **ECG**: LVH [MAINLY] + AF • **CXR**: • *Cardiomegaly* • *Dilated asc. Aorta* • *LVH* • **ECHO**: narrowed valve	• **Degenerative calcification . >70 + most common)** • **Bicuspid aortic valve** (<70) (usu. **Congenital (Aut. Dom.) = Turner's= young**) → assoc. w/ coarctation, ectasia & aneurysms • **Rheumatic fever** (3rd world) → valves fused together ***Severe degenerative AS***: • Insidious onset • long latency period • Rapid decline • High mortality → better if w/ *angina rather than syncope* ***Normal AS*** = *2.5-3cm²* ***Severe AS:*** *<1cm² or >40mmHg* ***Critical AS:*** *< 0.7cm² or >75mmHg* ↑ HR = ↓ Diastole = ↑ AV block	***Medical Rx*** • *Mild AS*: Activities OK ○ AVOID dehydration & vasodilation (i.e. overheat) • *Severe AS*: NO sports or diuretics → possible sudden death • ***Rx of hyperlipidaemia does NOT slow progression*** • ***Risks = >70****, CAD, Long-term Warfarin, CKD* ***Surgical Rx*** • Balloon valvuloplasty (BAV) *(for critical AS) → buy time for replacement* • **SAVR: Surgical Aortic Valve replacement** [BEST] (severe AS) • **TAVI** (transcatheter aortic valve implantation) → if high risk using open-heart SAVR ○ Post-op anti-coags ○ Complete HB/LBBB → Rx w dual-chambered pacemaker ○ 6-12 mth follow up
Aortic sclerosis 	**Ejection systolic**	Upper RSE (no RAD)	• Rigid Valve → Turbulence (thickening NOT narrowing) → Local sound only	• None	*Differentiate from AS* • No abnormal signs • normal pulse, apex and S2	• Senile calcification (most) • Turner's syndrome (webbed neck)	
HOCM	**Ejection systolic**	Lower LSE (no RAD)	• **LVH** "asymmetric LVH and thickened IVS" • **Q wave on septal lead**s (lead I, aVF, V5, V6)	• Syncope • Early cardiac death	• murmur intensity decreases on squatting • increases in Valsalva or standing (opp. of AS *standing reduces preload = worsens LV outflow ➔ increase murmur intensity	• idiopathic • Frederich's ataxia • Genetic (B-myosin heavy chain)	Aim to miniimise risk of ventricular arrythmias: • BB • CaB • ICD ECHO = THICK ivs + diastolic dysfn
Mitral Regurgitation 	**Pan-systolic**	Apex (→ left axilla) **AMPLIFY:** [expiration]	• Regurgitation to LA • LA dilation → LV dilation and failure IF **acute (EMERGENCY)**: • LA pressure increases → pulmonary oedema	• SOB • Fatigue • Palpitations (AF) • Comp. of acute MI, trauma, surgery	• **AF (due to LA dilation)** → *fatigue + low CO* • **Displaced thrusting apex** (vol. overload) ➔ **CARDIOMEGALY** • **Soft S1** • **LVF** (S3, **Pulm oedema***) • **Pulm HTN** (RV heave, loud P2)	**COMMONEST ACQUIRED** • Idiopathic weakening with age • **IHD → Papillary muscle dysfunction** (post-MI) • **Infective endocarditis** • **Dilated cardiomyopathy** (*LV dysfunction*) • **Rheumatic heart disease** • **Congenital (Marfan', Ehler's** • Prosthetic valve leakage	***Medical Rx*** • Vasodilator • Diuretic • **Anti-coags (ONLY if AF)** ***Surgical Rx*** • MV replacement/CLIP/ring • Improves long-term LV fn *For *MVP, papillary rupture, IE*
Mitral valve prolapse 	**(LATE) Mid-systolic click** *(DDx from MR by normal S1 then gap before murmur)*	Apex (→ left axilla and back)	• Weak CT in MV • During systole → mitral valve leaflet prolapses to LA • ↑ EDV → dilates atrium	• Atypical chest pain	• Murmur only	**Associations**: • **CT disorders** (Marfan's, PKD, SLE, DMD, Ehlers-Danlos) • **1º congenital (ADPKD)** • congestive cardiomyopathy, • myocarditis, • osteogenesis imperfecta,	
Ventricular septal defect (VSD)	**Pan-systolic**	Lower LSE (loud → whole precordium)	During systole some blood from LV leaks into RV	• Often none *if small*	• Loud P2 • Confirm with ECHO	Congenital Post-MI (free-wall rupture)	***Complications*** → raised JVP , peripheral oedema (RHF), pulm. HTN
Tricuspid regurgitation 	**Pan-systolic** (Louder on inspiration unlike MR)	Lower LSE (4th ICP) **AMPLIFY:** [inspiration]	***RV dilatation*** due to Regurgitation to RA and systemic backflow	• Fatigue • Hepatic pain on exertion • Ascites • oedema	• **Giant V waves in JVP** (without RVF = TR) • **Pulsatile hepatomegaly** • Parasternal heave = severe	• **RV dilation in pulm HTN (most**; e.g. due to chronic lung disease or left heart/valve disease) • Rheumatic • IE (IV drug user) • **Ebstein's anomaly** (apical displacement of septal and posterior TR leaflets → smaller functional EDV) ○ Dilated RA ○ ASD ○ Small RV ○ Displaced tricuspid ○ split S1 and S2)	 Ebstein's Anomaly
Pulmonary Stenosis *"bicuspid or 2-leaflet fusion"*	**Split S2 w/ Ejection systolic click** (during inspiration) *DDx: **ASD** = fixed split mid-systolic*	Upper LSE (→ back)	Obstructed Pulm. Valve or pulm. Outflow: • RVH → • RV failure	• SOB, fatigue, oedema, ascites • Cyanosis + HF (if severe)	• Dysmorphic face • RV heave • **Prominent A wave (JVP)** • Split S2 → systolic murmur	**Congenital (most)** • **Tetralogy of Fallot** • Noonan's syndrome • Valve dysplasia • *Mod. Stenosis 30-60mmHg*	***Medical Rx*** • Mild= None ***Surgical Rx (if severe)*** • Balloon mitral valvuloplasty

Diastolic (need to be accentuated → valsalvre maneuver)

Mitral stenosis 	**Low rumbling mid-diastolic** w/ **opening snap** (if heavily scarred) "parachute valve" **Similar to cardiac myxodema in LA**	Apex → 5th IC space on MCL (roll on left side) → use **BELL**	1. High LA pressure 2. Pulmonary HTN 3. RV hypertrophy 4. TR → Right heart failure (late)	• Thin females • HypoTN • SOB • Fatigue • Haemoptysis • Chest pain • **Ascites**	• **Malar flush (low CO)** • AF + P mitrale (biphasic) → triple • Tapping apex (palpable S1) • Loud S1 • Pulmonary HTN (RV heave, loud P2) • Oedema + tachypnoea	• **Rheumatic fever (60%) – esp. chronic → antibody X-react** *Rare:* • Congenital • Prosthetic valve stenosis via pannus growth • mitral annular calcification ***Normal MVA*** = 4-6cm² ***Mild MS*** = 2-4cm² or 5-10mmHg ***Severe MS*** < 1cm² or >10mmHg **Mean pressure gradient between LV/aorta (AS)**	***Medical Rx (Class I, II)*** • HR control (digoxin, BB) • **Anti-Coags** if: ○ AF + > 40y ○ LA enlarged, MR ○ Prior embolic event ***Surgical Rx (Class III, IV)*** • Balloon mitral valvuloplasty (not if MV is heavily calcified or regurgitation) • MV surgery/ replacement
Aortic regurgitation 	**Early-mid diastolic** ("fast blowing")	**Lower LSE** **AMPLIFY:** [lean forward + full expiration]	Systemic backflow	• Fatigue • SOB • Palpitations	• **Collapsing BOUNDING** pulse (water-hammer) • **Wide pulse pressure** (< 70 mmHg) e.g. 170/60 • Very displaced apex • Low diastolic (< 60mmHg) • *Backflow signs*: ○ **Corrigan's** (visible carotid pulsation) ○ **de Musset's** (head nodding pulse) ○ **Quincke's** (red colour pulsation in nails) • **±Austin Flint murmur** (apical diastolic rumble)	**Acute causes** • Infective endocarditis • Aortic dissection **Chronic causes** • Congenital = Biscuspid (most common) • **CT disorders (e.g. Marfan's, Ank. Spond. Ehler's))** • Rheumatic • Luetic heart disease (syphilis) • Long standing HTN	• Mild = anti-coags (E.g. DOAC) • Severe = Open-heart surgery
Tricuspid stenosis	**Early diastolic**	**Lower LSE**	Systemic congestion and R atrial dilation	• Fatigue • Ascites • oedema	Raised JVP • Giant A wave • Slow Y descent	• Rheumatic (most) • Congenital atresia • carcinoid	
Pulmonary regurgitation	Decrescendo murmur in **early diastole**	**Upper LSE**	Pulmonary backflow	Often none	RV hypertrophy	• Any cause of pulmonary HTN	

*LV hypertrophy (**due to stenosis on left side OR HOCM**) = *non-displaced* heaving apex beat

** **LV dilation** = LVF (**due to regurgitation on left side**) = *displaced* thrusting apex beat

***Aortic coarctation = systolic murmur below Left clavicle → amplified w/ valsalvre (↑ VR)

NB: ergot derived drugs (e.g. cabergoline for PD) can cause valvular disorders

Infective Endocarditis

Risk Factors

Core-cardiac

- Previous or Degenerative Valvular heart disease
- Cardiac interventions (e.g. *pacemaker*)
- Congenital HD (e.g. *VSD, patent ductus arteriosus*)

Non-cardiac

- IVDU → R-sided IE = ↑ PE risk, TR, leucocytosis
- Dental procedures
- GI and GU instrumentation (IV lines, catheters etc.)

Dx [*duke's criteria*] BE - TIMER

High pre-test probability
➔ **Dx confirmed if:**

- 2 major **OR**
- 1 major + 3 minor **OR**
- 5 minor criteria.

Blood culture
ECHO

Temp
Immunologic
Microbio
Embolic
RF

Major Criteria

Positive blood cultures
Typical pathogens from at least two separate cultures

Evidence of endocardial involvement by echocardiography
Endocardial vegetation, perivalvular abscess, new partial dehiscence of prosthetic valve, new vavular regurgitation

Minor Criteria

Predisposition	Fever
Heart condition or IV drug use	Greater than or equal to 38°C
Microbiologic evidence	**Vascular phenomena**
Single positive blood culture (except for coagulase-negative staphylococcus or an organism that does not cause endocarditis)	Arterial emboli, mycotic aneurysm, septic pulmonary infarcts, conjunctival hemorrhages, Janeway lesions

Echocardiographic findings
Consistent with endocarditis, but does not meet major criteria

Exam

- **High fever** (90%) + *prolonged UWL, anorexia polyarthralgia*
- **Heart murmur** (85%) – usu Mitral stenosis

L-*sided IE*

- **Septal emboli** (25%) = renal infection + stroke
- **Splenomegaly (30-40%)**
- **Non-blanching petechiae rash (40-50%) +/- haematuria**
- **Skin lesions (***Osler nodes, Janeway lesions, splinter haemorrhages***) (5%)**
- **Eyes** = roth spots + conjunctival **haemorrhages (5%)**
- **Poor dentition**

Labs

- FBC
- **3x sets** of blood cultures (different times)

Image

- **1st TTE (higher specificity –99%) → see vegetation = IE**
- **2nd TOE (higher sensitivity) > TTE → CLEARER IMAGES as closer to the heart → Dx** prosthetic endocarditis
- TTE/TOE dx easier for aortic abscesses > mitral abscesses

Complications of IE:

- **MAIN = MITRAL stenosis**
- **Other: TR, glomerulonephritis [**immune complex deposition]

Rx

- **4 - 6weeks IV Abs** = DEPENDS ON organism
  - **Viridians ➔** benzylpenicillin (**+ve**) + gentamicin (**-ve**)
  - **S. Aureus ➔** flucloxacillin + vancomycin (**MRSA**)

If sig. valvular damage / HF / uncontrolled infection (e.g. fungal growth) / embolism risk → surgical replacement **INDICATED**

- **St Jude Medical = Metallic heart valve- life-time anti-coags** (ONLY warfarin) = longer lifespan FOR YOUNG
  - **INR target = 2.0-3.0**
  - **S1 click = Mitral valve, S2 click = Aortic valve**
  - **Complications = (1) Infective endocarditis, (2) haemolytic anaemia (shear stress), (3) thrombus formation**
- **(TAVI) Tissue valve for AORTIC STENOSIS– short lifespan** (*older patients **OR** Anti-COAG CI e.g. females trying to fall pregnant*)
  - **Inserted via femoral artery via XR guidance**
  - **Risk of infective endocarditis (usu. gram +ve)**

Cause

Staph Aureus (60%)		• IVDU, prosthetic valve • Macroangiopathic hemolytic anemia • worse prognosis
Strep viridians (30%) **[MITRAL VALVE]**	Subacute	• affects native valve • Dental, , prosthetic valves **(≥60 days** post-surgery)
Staph epidermidis	Acute IE	venous catheters, prosthetic valves **(≤ 60 days** post-cardiac surgery)
HACEK	**Culture -ve**	Haemophilus, actinobacillus, cardiobacterium ,eikenella, Kingella
Prior Abx given		**Strep Bovis (bladder Cancer)**
Other		Fungi, Q fever (Coxiella), legionella, whipplei

NON-SHOCKABLE = ASYSTOLE + PEA (4H's + 4T's)

NO PULSE
NO DEFIB

- hypothermia
- hypovolaemia
- hypoxia
- hyp o/k, hyper K
- tamponade
- thrombosis
- tension pneumothorax
- toxins

Remember
1. arrest → infarct
2. BROAD COMPLEX TACHY → VT
3. BRADYCARDIA → HB

- BB — AF, V. Arrhythmia (↑V. Filling time)
- LIDOCAINE = V. Arrhythmia
- FLECAINIDE + AMIODARONE = rhythm — VT, (AF+BBB)
- VALSAVRE = transient AV block (SVT)

Adult tachycardia (with pulse) algorithm

- Assess using the ABCDE approach
- Give oxygen if appropriate and obtain IV access
- Monitor ECG, BP, SpO_2, record 12-lead ECG
- Identify and treat reversible causes (e.g. electrolyte abnormalities)

SHOCKABLE PATHWAY (VT, VF) inc. tachyarrhythmia w/ adverse signs (e.g. hypoTN)

Adverse features?
- Shock → hypoTN, clammy, dizzy
- Syncope
- Myocardial ischaemia → chest pain
- Heart failure → oedema, SOB

Yes/Unstable → ① **Synchronized DC Shock** Up to 3 attempts → ② Amiodarone 300 mg IV over 10-20 min and repeat shock; followed by: Amiodarone 900 mg over 24 h

No/Stable (NON-SHOCKABLE)

* Class III = K^+ channel blocker (phase 3) = ↑AP and QT interval

* AVOID AMIODARONE in TORSADES SINCE IT PROLONGS QT

Is QRS narrow (< 0.12 s)?

Broad

Broad QRS Is rhythm regular?

Irregular → **Seek expert help**

Possibilities include:
- **AF with bundle branch block** treat as for narrow complex
- **Pre-excited AF** consider amiodarone
- **Polymorphic VT** (e.g. torsade de pointes - give magnesium 2 g over 10 min)

Regular → If **ventricular tachycardia** (or uncertain rhythm):
- Amiodarone 300 mg IV over 20-60 min; then 900 mg over 24 h (implantable defib)

If previously confirmed **SVT with bundle branch block:**
- Give adenosine as for regular narrow complex tachycardia

Narrow

Narrow QRS Is rhythm regular?

Regular → SVT?
- Use vagal manoeuvres (transient AV block)
- Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg.
- Monitor ECG continuously

Sinus rhythm restored?

Yes → Probable **re-entry paroxysmal SVT:**
- Record 12-lead ECG in sinus rhythm
- If recurs, give adenosine again & consider choice of anti-arrhythmic prophylaxis

* Adenosine = CI for asthma (vasodilation of coronary, ↓SA, AV node activity, ↓ β2) — low dose β2 = ↓BP; high dose α1 = ↑TPR
↳ use verapamil instead

No → **Seek expert help**

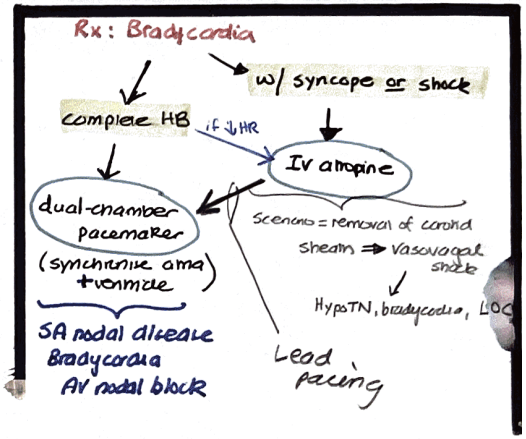
Possible **atrial flutter**
- Control rate (e.g. β-Blocker)

Irregular → **Irregular Narrow Complex Tachycardia** ↓HR, ↓BP = ↓O_2 demand

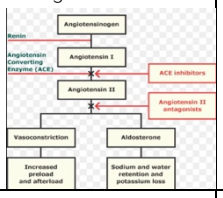
Probable atrial fibrillation

Control rate with: (UNCONTROLLED AF)
- β-Blocker or diltiazem (Class V = AV node blocker)
- Consider digoxin or amiodarone if evidence of heart failure (CCF, LHF)

• CARDIOVERT = AF + FAST V. RATE + UNSTABLE HAEMODYNAMICS

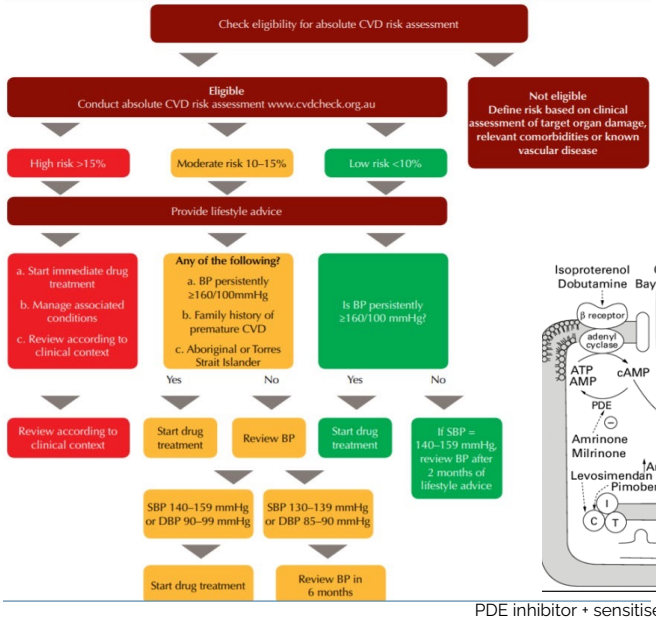


Antihypertensives, Inotropes and Vasopressors

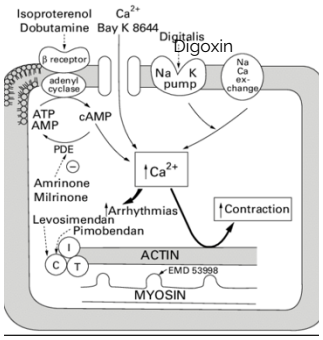
	ACEi (-prils)/ ARB (-sartans)	Diuretics	CaBs	BB (-lols)	Alpha-blockers (-ozins)	Central alpha-agonists	Nitrates & vasodilators
MoA	• ACEi = inhibit ACE • ARB = block action of AngII 	• **Thiazide** (Mainly) → inhibit Na+/Cl- symporter in DCT • **Amiloride** = inhibit Na/H exchanger in cortical collecting duct (no effect on aldosterone)	• **Dihydropyridines** (i.e. amlodipine) → SMC relaxation = peripheral vasodilatation • ***Non-dihydro (e.g.** verapamil) → control palpitations/ arrhythmia (class IV)	***Block*** beta adrenergic receptors: • ↓ HR (B1) • ↓ contractility (B1) • Broncho-constriction (B2)	***Block*** a1-adrenoceptors → inhibit peripheral vasoconstriction	• **Clonidine** (old) → activates central a2 receptors → inhibits central SNS (stop A release) • **Moxonidine** (newer) → wean slowly esp. if used with BB	• Nitrates (GTN) for HTN emergencies • **Hydralazine** = direct acting vasodilators (relax SMC) → reduces afterload • **Minoxidil (↑ K-ATPase)**
Ind	• **Renoprotective for diabetic**s (via efferent renal arteriole → CKD vasoconstriction) • **ARB > ACEi** (improve CVS mortality)	Combined with ACEi or ARB *Spironalactone* = best for Conn's	• **Dihydropyridines** **2nd line** after diuretics • Use CaB 1st line if > 55 or black	**1ST line = stable angina** **3rd line** → esp. for hypertensive crisis (e.g. labetalol) • Arrythmia • Migraine • Anxiety • hyperthyroidism	Adjunct or if CI for other drugs • *also helps with prostatism = bladder neck obstruction*	**Methyldopa** = pregnancy induced HTN → heamolytic anaemia (due to crystals in RBC)	**Hydralazine** • **Safe in pregnancy → pre-eclampsia** • Combined with nitrates
A/E	• Renal issue (**hypoNa**) • **Cough** (5-20% - bradykinin buildup) • Angioedema (elderly) • Postural HypoTN • Birth defect (pregnancy) **Avoid combined ACEi/ARB = ↑ risk of renal failure**	• HypoNa • Met. Alkalosis • **HyperGLUC:** ○ Glycemia ○ Lipid ○ Uriceamia ○ Calcemia Furosemide: • hypoK • Gout • hypoTN	• Peripheral oedema • Postural HypoTN • Reflex tachycardia (palpitations) *old agents = nifidepine have increased CVS	• Fatigue • Depression • Impotence • Nightmares • Raynaud's • Hide hypoBSL (Care w/ diabetics)	• Tolerance (long-term use) • Dry mouth • **Postural HypoTN** (esp. 1st dose → advise to take at night)	• Drowsiness • Sedation • Dry mouth • Refractory HTN → if sudden withdrawn • ED, • Depression • Postural hypoTN (clonidine)	• Peripheral oedema (leg swelling) • Induced cutaneous SLE (lupus) • **IV Na nitroprusside (ICU)** → light sensitive need to monitor cyanide levels

Antihypertensive treatment

1. Risk of CVS morbidity x2 for every 20mmHg increase in SBP > 120 mmHg
2. **Mild HTN in low-risk (<10%)** = commence drug Rx **ONLY** if systolic BP > 160 on ≥2 occasions
3. Pts with **CVS, DM, renal failure** = Aggressive Rx
4. **Combine drugs (usu. ≥2) w/ lifestyle measures**
5. **High-dose IV Magnesium = pre-eclamptic women is well tolerated*
6. Loop diuretics, acetazolamide = not useful as anti-hypertensives



HypoTN effect:
- Headache
- Flushing
- Dizziness
- falls



PDE inhibitor + sensitise troponin C to calcium

	Vasopressin (ADH)	Adrenaline	Noradrenaline	Dobutamine	Milronone	Digoxin	Isoprenaline
Class	**Vasopressor**	**Vasopressor**	**Vasopressor**	***+ve inotrope*** *weak chronotrope*	***+ve inotrope***	*weak inotrope*	***+ve inotrope*** ***+ve chronotrope***
MoA	• **V1** = vascular SMC constriction • **V2** = ↑ aquaporin = ↑ H_2O resorption = Hypervoleamia	**SNS amine → high affinity for:** • **B1, B2 (small dose) → ↑↑↑ HR / contractility** • a1 (high dose)	**SNS amine** • **potent a1 → vasoconstrict** • modest B1, B2	**Synthetic catecholamine** • **B1, B2 (potent) → ↑↑↑ HR / contractility** • Net mild vasodilation (due to B2 > a1)	• **Phosphodiesterase inhibitor** → inhibit cAMP breakdown • **↑ contractility** + Vasodilation (↓preload & TPR)	• Block NA/K ATPase pump • AV node block	Pure/non-selective B-adrenergic agonist • **↑ HR / contractility** • ↓ TPR = ↓ dBP
Ind	• **Shock (cardiogenic / vasodilatory)** • Cardiac arrest → when all has failed	• **Shock (cardiogenic / vasodilatory)** E.g. LVF, large anterior MI • Cardiac arrest • Anaphylaxis • Bradycardia	• **Shock (cardiogenic / vasodilatory** • LOW CO w/ low TPR	• Low CO (decompensated HF, **cardiogenic shock**, sepsis-induced MI) • Peri-op cardiac surgery	ICU = enhance cardiac function • no effect on adrenoceptors • *slight ↑ **HR due to vasodilation***	• ICU • CCF pts w/ AF • Hypokalemia • Bradycardia • ST depression • Reverse tick	• Heart block • brady arrythmias
A/E	• **Poor perfusion → peripheral ischeamia** ○ Ischemia Worsened by increased myocardial O_2 demand • **Lactic acidosis** (esp. adrenaline) • **Proarrhythmic** (esp. dobutamine)→ increased risk of ventricular arrhythmias [esp. using digoxin in young pts = death] • Vasopressors/inotropes → **Increased mortality** in HF patients (esp. post-discharge) • Metaramniol – for acute hypoTN → can be given IV peripherally to acta as LA vasoconstrictor (unlike NA)						

*These meds if given via a ***central line*** → can cause ***tissue necrosis*** if they

Anti-hypertensive drugs – Contraindications

Drug class	Contraindications	
	Compelling	Possible
ACE inhibitors or ARBs	Pregnancy Angioedema Hyperkalaemia Bilateral renal artery stenosis RAS	Women with child bearing potential
Calcium channel blockers (dihydropyridines)		Heart failure
Diuretics (low-dose thiazide)	Gout Age*	~~Glucose intolerance~~ Metabolic syndrome Hypercalcaemia Hypokalaemia
Beta-blockers Not first-line therapy†	Asthma Bradycardia A-V block (grade 2 or 3) Uncontrolled heart failure	Type 1 or 2 diabetes Metabolic syndrome Glucose intolerance Athletes and active patients Chronic obstructive pulmonary disease (except for vasodilator beta-blockers) Depression

→ Do not give at young age (risk of diabetes)

Activity 1: HYPERTENSION TREATMENT

Case Study 1:

A patient presents with high blood pressure (BP) which is sustained on repeated readings. The doctor initiates treatment with perindopril (an ACEi) and although this lowers BP slightly, it is still unsatisfactory. The doctor then adds in a second drug, HCT (thiazide diuretic) which again achieves some lowering of blood pressure, but this remains unsatisfactory. The doctor adds in a third drug, amlodipine. Satisfactory blood pressure is achieved on this.

Discuss if this is an accepted way of managing high BP? Consider:

- **Drug class of amlodipine** — Dihydropyridine – calcium channel blocker
- **Time between interventions** — If target has not reached after 3 months, however, earlier intervention can be considered if severe hypertension. Maximum effect of drug likely to be seen in 4-6 weeks → hence do not add new drug as would not be able to see actual effect of drugs
- **Patient age** — HCT should be avoided at young age → risk of diabetes assoc. with long term use
- **Adherence** — If therapy is not working, consider adherence of treatment as *possible cause*
- **Why not a beta-blocker?** — Beta blockers are NO longer a 1st line therapy for hypertension *(as not as effective as current drugs in preventing stroke and HT + contraindication for diabetes in asthmatics)*

Activity 2: WHY DOESN'T IT WORK?

Case Study 2:

- A 65-year-old man gets chest pain on exertion. This has been diagnosed as angina. What can be taken as required (PRN) during acute attack?
- He was prescribed GTN tablets. One week later, he had another episode and took the tablet with a glass of water. But there was no effect, why not?

1) Short acting nitrates such as glyceryl trinitrate (GTN) → usually used as a patch

2) GTN is a administered as a sublingual tablet (on tongue) → hence when taken orally with glass of water there was a has high 1st pass metabolism leading to negligible bioavailability = low levels in bloodstream (as oppose to using sublingually)

→ faster absorption than oral tablets + higher BA generally (as bypasses 1st pass liver metabolism)