

## Introduction

- Most common cause of death in industrialised nations.
- Minor congenital CVS abnormalities in 1:100 live births, more serious abnormalities in about 1:500.
- Mortality decreasing. ?Changing lifestyle. ?Improved Dx & Mx.
- Dx may be delayed due to silent development & limited symptom diversity.

## Anatomy

- Heart posterior to sternum & thymus and anterior to oesophagus & descending aorta.
- Normal heart is <50% of transthoracic diameter on PA CXR
- Coronaries arise from just distal to aortic valve. RCA (inf.) Left coronary → LAD (ant.) & LCX (post.).
- RCA supplies SA node (60%) and AV node (90%). Veins drain into coronary sinus & then right atrium.
- Sympathetic receptors (mainly B1) in muscle fibres & electrical conducting system. Vagus → SA/AV nodes (M2 receptors).
- SA node at junction of SVC & right atrium. AV node is below R atrial endocardium at lower end of interatrial septum.
- Annulus fibrosus prevents direct A-V conduction.
- Conduction: SA → AV node → His bundle → R & L bundles. L bundle → ant. & post. hemibundles. Purkinje network.
- Injury to R or L His bundle → R or L bundle branch block. Injury to a hemibundle → axis deviation.

## Physiology

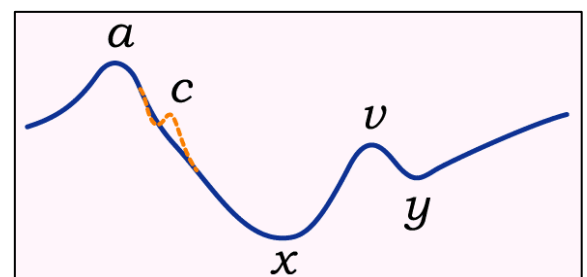
- Cardiac Output = Stroke Volume (SV) x Heart Rate (HR). SV depends on end-diastolic pressure (preload) & TPR (afterload).
- Starling's Law - increased contraction by increasing cardiac muscle stretch (end-diastolic volume)
- Coronary blood flow: constriction (alpha) < dilatation (beta2, some parasympathetic). Other local factors.
- In coronaries: ACh causes dilatation (via NO) or constriction if endothelium damaged (direct effect).
- Systemic blood flow depends on vessel radius to fourth power. Autonomic & local factors (NO, adenosine, PGs, NA, etc)

## Pulse

- Pulse vol depends on cardiac SV and arterial compliance. Small in CF, hypovol, vasoconstriction. Large in vasodilation, pyrexia, anaemia, aortic regurg & AV shunts.
- Pulsus alternans - Uncommon. Alternating small & large volume beats (cardiac failure, coupled ectopics).
- Pulsus paradoxus - exaggeration of drop in SysBP (>10mmHg) with respiration (airways obstruction, tamponade, big PE)
- Pulsus bisferiens - Double peak (ejection & arterial recoil). Seen in aortic stenosis & regurg, Hypertrophic cardiomyopathy.
- Sinus arrhythmia (HR faster on inspiration), AF (irregularly irreg), Ectopics (appears as dropped beat/stronger beat seq.)

## Jugular Venous Pulse

- JVP reflects R atrial pressure. Vertical height between Angle of Louis & top of venous wave. Normally 3-4cms. (<8mmHg)
- JVP peaks - *a* (atrial systole), *v* (atrial filling in ventricular systole), *c* (onset of ventricular contraction)
- JVP decents - *x* (atrial relaxation & downward displacement of tricuspid valve ring), *y* (fall in pressure as tricuspid opens)
- *a* wave lost in AF, large in pulm hyperBP, RVH, tricuspid stenosis (unless pat. In AF). Cannon wave in complete heart block or VT when atrium contracts against closed tricuspid valve. Tricuspid regurg → giant *v* waves.



## Apex Beat

- Apex Beat = most inferior & lateral point of cardiac pulsation. Normally 5icsmcl. Thrusting in aortic/mitral regurg. Sustained with LVH in aortic stenosis. Tapping with no LVH in mitral stenosis. Maybe impalpable in COAD, obesity.
- Parasternal heave at left sternal edge if RVH or rarely left atrial enlargement. Systolic impulse in 2nd ics in pulm. HyperBP.
- Palpable thrills are produced by murmurs e.g. aortic stenosis (systolic), mitral stenosis (diastolic)

## Heart Sounds

- First heart sound - closure of mitral & tricuspid valves. Loud in anaemia, pregnancy, thyrotoxicosis. Soft in CF, mitral regurg or severe calcific mitral stenosis (mitral stenosis usually gives a loud first sound). Intensity varies with variable PR interval.
- Second sound - Closure of aortic & pulmonary valves. Pulm. Valve closes second (longer ventricular emptying & inspiration delays). Wide variable splitting in RBBB, pulm. Stenosis or PE. Wide fixed splitting in ASD with RBBB. Reversed (i.e. splitting in expiration) in LBBB, LVF, aortic stenosis. Second sound is loud in arterial/pulm hypertension and soft in diminished cardiac output (e.g. aortic/pulm stenosis)
- Third sound - Early diastole. Heard best at LSE or apex. Due to vibrations caused by abrupt cessation of ventricular filling. Common in young, pregnancy and other hyperdynamic states (thyrotoxicosis). Also: CF, cardiomyopathy, volume overload of ventricle.
- Fourth sound - Late diastole. Occurs with atrial contraction in presence of stiff ventricle. Always path.
- Clicks - early systolic (aortic/pulm stenosis, prosthetic), midsystolic (valve regurg.), early diastole (mitral stenosis, prosthesis)

## Heart Murmurs

- Murmur: turbulent blood flow. Hyperdynamic circ. (pregnancy, exercise, anaemia), leaky or narrowed valves, septal defects.
- For each murmur elucidate: Systolic or diastolic; where heard best; radiation; intensity (1-6) & thrill; duration; pitch/quality.
- Intensity Grade 1=v. soft, 2=soft, 3=moderate, 4=loud + thrill, 5=very loud, 6=heard without stethoscope.

Murmur	Conditions
Mid-systolic	Aortic stenosis, pulm. Stenosis, hypertrophic myopathy, Fallot's, ASD (pulm. flow)
Pan-systolic	Mitral regurg, tricuspid regurg, VSD
Late-systolic	Mitral valve prolapse, hypertrophic myopathy
Early-diastolic	Aortic regurg, Pulmonary regurg, Graham Steell (pulm regurg due to pulm hyperBP & mitral stenosis)
Mid-diastolic	Mitral stenosis, Austin-Flint (aortic regurg impairing mitral diastolic flow), Tricuspid stenosis
Continuous	PDA, Venous hum (positional), arteriovenous shunts

## Electrocardiography (ECG)

- Used in arrhythmias; conduction defects; myocardial hypertrophy, ischaemia & infarction; and some electrolyte imbalances.
- Std 12-lead ECG: I, II, III, aVR, aVL, aVF (limb leads)-frontal plane & V1 to V6 (chest leads)-horiz. plane
- V1-V2: mainly R ventricle; V3-V4: mainly interventricular septum; V5-V6: left ventricle
- Positive deflection=depol. towards electrode. Scale: 10mm=1mV. Rate:25mm/s. So 5mm square=0.2s, 1mm square=0.04s.
- P wave - atrial depolarisation (not seen in aVR) <0.12s. P-R interval (start of P to start of QRS complex) 0.12 to 0.2s
- QRS complex - ventricular activation. Q waves in lateral leads (I, aVL & V6. Occ III) due to L to R activation of septum