Cardiovascular
The Elements

- General Inspection
- The Hands
- The Pulse
- The JVP
- Feel the preaordium
- Auscultate

Use the **POW** formula

Hands and pulse

JVP

Feel

Listen
The Key

- To know how to put them all together
Symptoms of the chest:

- Apex beat
- Inhalation
- Auscultation

4 REASONS OF DIED:

- Left desaturation
- RV failure
- PFT abnormalities
- Aspiration

ACUTE DIASTOLIC: RV failure

- RV failure
- Constriction

MAJOR STELLAR:

- Major STELLAR response
- Aspiration

- Auscultation
General Appearance

- Is there SOB
- Malar Flush- dusky flush of cheeks in mitral stenosis
- Visible chest scars
- Syndromes
  - Tall and thin- MARFAN'S (AORTIC REGURGITATION)
  - Trisomy 21- CONGENITAL HEART DISEASE
  - TURNER'S SYNDROME – COARCTATION OF AORTA
The Hands

- **Nails**
  - Clubbing
  - Splinter haemorrhages

- **Fingers**
  - Peripheral cyanosis
  - Osler’s Nodes

- **Hands**
  - Janeway lesions
  - Palmar palor
  - Tendon xanthomata

Unless it is an inpatient you will probably not see Osler’s nodes and Janeway lesions.
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Osler’s go ‘OUCH’
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  o **Tendon xanthomata**
What do we know so far?

• Clubbing indicates that there may be some heart disease
• Cyanosis indicates some potential heart disease
• IS THERE ANYTHING ELSE?
The PULSE

Now the important stuff begins
Rate

• Is it regular/irregular?
  o If irregular i.e., atrial fibrillation
    • Think MITRAL VALVE disease with left atrial enlargement
  o Is it bigeminy?

• Is it fast or slow?
Radial-Radial Delay

- Atherosclerotic plaque
- Aneurysm
- Subclavian artery stenosis
- Thoracic aorta dissection
Radial-Femoral Delay

• Coarctation of Aorta
  o Congenital
  o Narrowing of aorta distal to origin of left subclavian artery
  o Hypertension in arms but not in legs
  o Femoral pulses are weak
  o Upper body may be better developed than the lower
  o mid-systolic murmur over praecordium and back.
Pulse character and vol

- Use the brachial or carotid arteries for this.
- Collapsing pulse of aortic regurgitation
- Pulsus alternans
What do we know so far?

- Rate of pulse?
- Is it regular?
  - Is it AF? - think of mitral valve disease
- Are there ectopics?
- Is it collapsing?
  - Think AR
- Is it alternating?
  - Think LVF
- Is it a good volume?
  - Small vol = AS
The Neck

JVP and Arterial Pulse
• Small volume (anacrotic)
  o Aortic Stenosis
  o Pericardial Effusion
• Collapsing
  o Aortic regurg
  o Patent Ductus Arteriosus
  o Hyperdynamic Circulation
• Alternans
  o Left Ventricular Failure
• Bisferiens (small volume + collapsing)
  o AS + AR
JVP

- Pt at 45°
  - Sternal angle at base of neck
  - Zero point
- Complex waveform
- Decreased on inspiration
- >3cm = raised
- ‘a’
  - Right atrial systole
  - Coincides with first heart sound
  - PRECEDES carotid pulse
- ‘v’
  - Atrial filling whilst tricuspid remains closed during ventricular systole
  - After ‘v’ tricuspid opens and there is rapid ventricular filling
‘a’ wave is pre-systole

‘v’ wave coincides more with systole
• Raised JVP
  o Anything that raised right pressures
    • RVF
    • Tricuspid Stenosis
    • Pericardial effusion

• Big ‘a’ waves (↑ R atrial pressure)
  o Tricuspid stenosis
  o Raised pulmonary pressures
  o Complete heart block
    • ‘canon’
      o Explosive
      o Complete heart block
        • Atrium contracts against closed tricuspid

• Big ‘v’ waves
  o Tricuspid regurgitation
  o Occur during each ventricular systole

• Hepatojugular (abdominojugular) Reflex
  o Pressure over middle of abdomen 10 sec
  o Increases venous return to right atrium
  o There should be transient rise in JVP
  o If remains elevated >4cm for whole time of compression
    • Right ventricular failure OR
    • Elevated left atrial pressures (LV failure)
What do we know so far?

• We’ve felt the pulse (including carotid)
  o We know if the patient is in sinus rhythm
    • If in AF we start to think of mitral valve stenosis and L atrial enlargement
      o ‘a’ wave is usually lost
    • It may be heart block
  o If it is a small volume pulse then we think of aortic stenosis
  o If it is a collapsing pulse we think of aortic regurgitation

• We’ve looked at the JVP
  o Is there evidence or right heart failure?
  o Are there explosive (canon) ‘a’ waves which put together with the irregular pulse- it is a complete heart block
  o Is there a large ‘v’ wave which may indicate TR?
The Praecordium

Show me the money!!!!
Observe

- **Scars and lumps**
  - Median sternotomy
  - Pacemaker

- **Shape of the chest**
  - Pectus excavatum - no effect really
  - Kyphoscoliosis
    - May be associated with Marfan’s

- **Is the apex impulse visible?**
  - Due to heart recoil as blood is expelled.
**APEX BEAT**
- Only palpable in about 50%
- Normally in 5\textsuperscript{th} ic space, 1cm medial to mid-clavicular line
  - Displacement may mean enlargement of the heart
- What is the character?
  - Is it forceful and sustained? (\textit{pressure loaded})
    - AS, HT
  - Is it thrusting and diffuse? (\textit{volume loaded})
    - MR, dilated Cardiomyopathy
  - Is it uncoordinated? (\textit{dyskinetic})
    - LV dysfunction
  - Is there a double impulse? (\textit{double impulse beat})
    - Hypertrophic cardiomyopathy
  - Is there a tap? (\textit{tapping apex beat})
    - Coincides with 1\textsuperscript{st} heart sound and is due to valve closure MS/TS

**A THRILL** – \textit{turbulent blood flow (murmur) that is palpable}
- Parasternal Impulse (palm to left of the sternum)
  - Usually RV enlargement or severe left atrial enlargement will cause
- Base of the heart
  - Aortic or pulmonary
What do we know so far?
Auscultation

• Heart Sounds
• Murmurs

• Start at MITRAL area
  o BELL(low-pitched sounds)- diastolic murmurs (MS) or S3
  o Diaphragm(high pitched sounds) (MR) or S4

• Then tricuspid
• Then pulmonary
• Then aortic
Heart Sounds

• Use the carotid pulse to time the murmur

• S1- mitral closes slightly before tricuspid
  o It is the beginning of ventricular systole

• S2- Usually pulmonary closes after aortic
  o The lower pressure in pulmonary circulation compared to aorta results in flow continuing in the pulmonary artery after the end of RV systole.
  o Marks beginning of diastole
  o Best heard in the pulmonary area
  o With inspiration there is increased venous return to the right ventricle, so that splitting of the second heart sound is increased further.

• Both S1 and S2 are high pitched and best heard with diaphragm
S1

- Loudness determined by mitral valve speed of closure, which really depends on the left ventricular contraction strength.
- Loudest at apex
- It can be LOUD
  - Mild MS
  - Short PR- because the LV pressure is greater earlier
- It can be soft
  - Long PR
  - Severe MS
  - LBBB
- It can vary
  - 3rd° block
  - AF
S2

- Best heard at upper sternal border
- Increased split in inspiration
- Wide split
  - Anything that delays RV contraction, or premature LV contraction
    - RBBB
    - Pulm HT – can also have a very loud P2
    - Pre-excitation of LV
    - Severe MR
- Fixed Split ie no change with inspiration
  - ASD
  - Severe RVF
- Reversed split- split in expiration not inspiration
  - It is the opposite of wide split
  - LBBB
  - Pre-excitation of RV
  - AS/AR
  - LV outflow obstruction
• Can occur in young, but in >40yo – pathological
  o LV failure
  o Due to blood hitting a non-compliant left ventricular wall
• Early diastolic
• Best heard at apex with patient on left
S4

- Pre-systolic
- Always pathological
- Caused by atrial contraction into stiff non-compliant ventricle
‘Clicks’

- **Aortic**
  - Early systole

- **Mitral Valve Prolapse**
  - Mitral valve leaflet prolapse
  - Occurs in mid-systole

- **Mechanical valves**
Murmurs
Systolic Murmurs

- **Pansystolic**
  - All of systole
  - MR / TR / ASD

- **Ejection Systolic (crescendo-decrescendo)**
  - Mid-systole
  - AS/ PS
  - HOCM

- **Late Systolic**
  - Starts later in systole and continues until S2
    - MVP
    - Papillary muscle dysfunction
Diastolic Murmurs

- These are usually softer
- A *loud murmur is unlikely to be diastolic*

**Early diastolic**
- Begin at the end of S2
- Are decrescendo and end within diastole
- AR / PR

**Mid-diastolic**
- Late in diastole
- May end early or continue until S1
- MS / TS
- In severe AR, the regurgitant jet can cause the mitral valve to shudder, producing a diastolic murmur

**Presystolic**
- MS / TS
Continuous murmurs

- Occur throughout systole and diastole
  - PDA
  - AV fistula
  - Aortopulmonary connection
HOCM

- Hypertrophy in left /right or both outflow tracts
- There is obstruction to flow late in systole when the hypertrophied area contracts- but can be a mid-systolic murmur
- The mitral valve may also be displaced into the LV outflow tract in systole, causing MR
- Prominent ‘a’ wave
- Late systolic murmur and pansystolic murmur at apex, plus S4
- Outflow murmur increased by valsalva and decreased by squatting and isometric
Dynamic Manoeuvres

We are differentiating between murmurs

Pansystolic
MR
TR
VSD

Early Diastolic
AR
PR

Mid diastolic
MS
TS

Pre systolic
MS
TS

Mid systolic
AS
PS
HOCM

End systolic
MVP
Dynamic Manoeuvres

• **Respiration**
  - Right sided murmurs are louder on inspiration, left sided are unchanged or become softer

• **Deep Expiration**
  - Lean the patient forward and listen at the base
  - Good for AR

• **Valsalva (forceful expiration against closed glottis)**
  - Makes HOCM louder and everything else softer

• **Isometric (20 seconds)**
  - Increases systemic arterial resistance and BP
  - Most murmurs become louder EXCEPT HOCM (softer) and MVP (delayed)

• **Standing to squatting**
  - There is increase in venous return and systemic arterial resistance
  - Decreased intensity of HOCM

• **Squatting to standing**
  - Increased HOCM murmur
Dynamic Manoeuvres

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Which ones?
Auscultation of Neck

• The bruit of aortic stenosis is heard in the neck
• There may be a soft bruit heard in mitral regurgitation or pulmonary stenosis.
Back, Belly and both legs

- **Listen to lungs**
  - Crackles at the lung bases in heart failure, or dullness of pleural effusion
- **Feel for pitting oedema of the sacrum**
- **Palpate the abdomen**
  - Enlarged tender liver in RHF
  - Pulsatile liver in TR
  - Hepatojugular reflex
  - Ascites in severe RHF
  - Splenomegaly in IE
  - Feel abdominal aorta
- **The legs**
  - Feel for pitting oedema
  - Achilles tendon xanthomata
  - Clubbing
    - Can occur only in toes in PDA
  - Palpate for pulses
  - Capillary return - If slow perform Berger’s test
    - Elevate legs to 45° - if pallor is rapid, then poor arterial supply
    - Then lower them in the dependent position - cyanosis occurs in poor arterial supply
    - In the normal leg, colour doesn’t change
  - Ankle-Brachial reflex
    - Systolic Blood pressure of ankle (dorsalis pedis or post tibial)/SBP in arm
      - If <0.9 = severe arterial disease
      - 0.4 – 0.9 = claudication
      - <0.4 = critical ischaemia
      - >1.3 = calcified artery
How severe is that?
Mitral Valve

• Normal area 4-6 cm$^2$

• Mitral Stenosis
  o $\leq$ half this will cause significant obstruction to LV filling
  o SEVERITY
    • Clinically
      o PND, orthopnoea, fatigue
      o Small pulse pressure, long diastolic murmur
    • Echo - severe is $< 1$ cm$^2$
      o Causes- rheumatic fever, congenital parachute wave

• Mitral Regurgitation
  • Clinically
    o Dyspnoea, fatigue
    o Small pulse vol, S3, LVF
  o Causes- mitral valve prolapse, rheumatic, papillary muscle dysfunction, cardiomyopathy, Marfan’s.
Aortic

• Normal area is $>2cm^2$

• **Aortic Stenosis**
  o Clinical
    • Exertional chest pain, dyspnoea and exertional syncope
    • Signs of plateau pulse, aortic thrill, length of murmur shw disease is present but do not distinguish moderate to severe disease
  o Echo – Severe if Valve area of $< 1cm^2$, or valve gradient $> 50mmHg$
  o Causes- Calcific

• **Aortic Regurgitation**
  o Blood regurgitates into aorta from LV during diastole
  o Clinical
    • Exertional dyspnoea and fatigue
    • Marfan's Syndrome, ankylosing spondylitis (sero –ve arthropathies)
    • Collapsing pulse (water hammer), wide pulse pressure, S3
  o Causes- Rheumatic, aortic root dilatation, IE
Tricuspid

• Tricuspid Stenosis
  o Very rare
  o Clinical
    • Giant ‘a’ waves
  o Causes
    • Rheumatic

• **Tricuspid Regurgitation**
  o Clinical
    • Large ‘v’ waves, pulsatile liver
  o Causes – rheumatic, IE, prolapse, trauma, Ebstein’s anomaly
Pulmonary

• Pulmonary Stenosis
  o Clinical
    • Peripheral cyanosis, giant ‘a’ waves
    • S4, right ventricular failure
  o Causes – congenital

• Pulmonary Regurgitation
  o Uncommon and trivial regurgitation is considered physiological
  o Clinical
    • Decrescendo diastolic murmur
  o Causes – Pulm HT, IE, congenital abscence