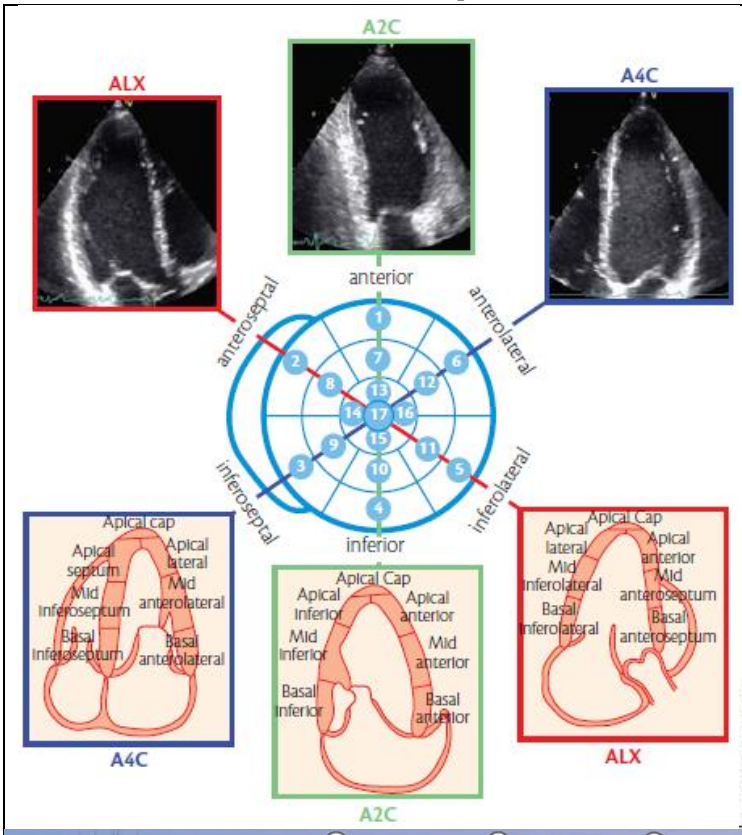
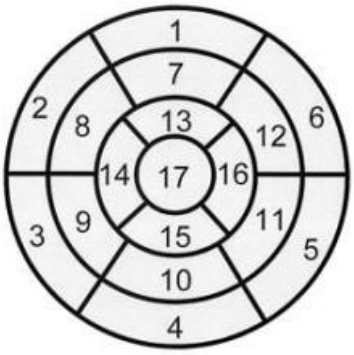
# ECHO FINDINGS IN ISCHEMIC HEART DISEASE

**1. RWMA:** ischaemic heart disease is the commonest cause of RWMAs. In acute ischaemia, akinetic myocardium may indicate stunned or hibernating myocardium that may regain normal contractile function if revascularisation occurs early. In IHD, ventricular dyssynchrony can result from damage to the bundle branch conduction pathways.

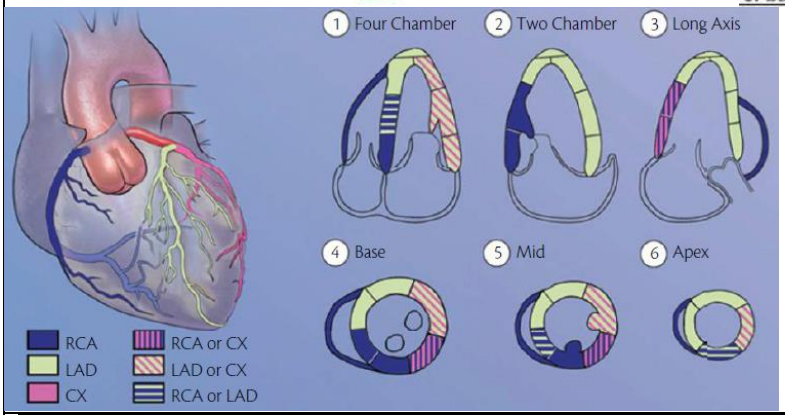
**Coronary territories in the 17 segment model:** (inferoseptal = septal, anterolateral = lateral, inferolateral = posterior)
**LAD: anterior + anteroseptal + apex + apical septal** ± mid inferoseptal ± apical lateral ± apical inferior (± anterolateral)
**RCA: inferior + basal inferoseptal** ± mid inferoseptal (± inferolateral)
**CX: anterolateral + inferolateral** ± apical lateral




## Left Ventricular Segmentation




1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex




**Scoring to assess wall motion**:
Each segment should be analysed individually in multiple views
1 = normal or hyperkinetic
2 = hypokinetic (reduced thickening)
3 = akinetic (absent or negligible thickening)
4 = dyskinetic (systolic thinning or stretching)
**WMSI** ≥ 1.7 indicates a poor prognosis

| | 4C | | 2C | | LA (3C) | |
|---|---|---|---|---|---|---|
| **Apex** | **Apex (L)** | | | | | |
| **Apical** | Septal (L) | Lateral (C/L) | Inferior (L/R) | Anterior (L) | Lateral/inferior | Septal/anterior |
| **Mid** | Inferoseptal (L/R) | Anterolateral (**C**/L) | Inferior (R) | Anterior (L) | Inferolateral (**C**/R) | Anteroseptal (L) |
| **Basal** | Inferoseptal (R) | Anterolateral (**C**/L) | Inferior (R) | Anterior (L) | Inferolateral (**C**/R) | Anteroseptal (L) |

Segments supplied by the LAD or RCA: mid inferoseptal and apical inferior
Segments supplied by the LAD or LCX: basal anterolateral, mid anterolateral and apical lateral
Segments supplied by the RCA or LCX: basal inferolateral and mid inferolateral

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**2. LV aneurysm-** True and false LV aneurysms are both complications of myocardial infarction. Apical aneurysms are by far the commonest type. Incidence of LV aneurysm with first anterior MI = 10–22%. Aneurysm causes a deformity of the LV during ventricular systole and diastole (dyskinesis deforms LV only during ventricular systole)

| True aneurysm | Pseudoaneurysm |
|---|---|
| A bulging of the scarred myocardium. | It is the sequela of myocardial rupture with haemorrhage into the pericardial space. Local tamponade occurs, preventing further haemorrhage into the pericardium. Over time, the intrapericardial thrombus organizes, creating a wall to the pseudoaneurysm |
| Lined by thinned myocardium. Mural thrombus can form as a consequence of ↓wall motion | Lined by organizing thrombus and varying portions of the pericardium |
| Wide neck (at least half the diameter of the aneurysm itself) | Narrow neck |
| More commonly found in the anteroapical wall | Arise more commonly from the base of the inferior and inferolateral wall |

**3. Mural Thrombus-** Chronic thrombus formation is a common complication after MI (up to 40%) and is most common after large anterior myocardial infarction, especially with involvement of the apex. Poor LV function is a substrate for LV thrombus formation in DCM and in ischemia.

**Identification by Echo:**

- LV thrombus density is generally greater than adjacent endocardium (contrast helpful)
- Usually associated with segmental wall motion abnormalities.
- colour Doppler may be useful to demonstrate a ‘filling defect’ in the area of the thrombus (low velocity scale and wall filter)

**4. Mitral Regurgitation-** multiple mechanisms may account for MR complicating acute MI. These include (1) dilation of the mitral valve annulus as a result of LV dilation; (2) ischemic papillary muscle dysfunction; and (3) rupture of the chordae or papillary muscle. Rupture of the chordae or papillary muscle is more common with inferior MI, affecting the posteromedial papillary muscle, resulting in flail posterior leaflet (MR jet is directed anteriorly and medially towards the IVS). This is because the posteromedial papillary muscle receives blood supply solely from the PDA, whereas the anterolateral papillary muscle has dual blood supply from LAD and CX arteries. Similarly, Ischemic papillary muscle dysfunction and associated tethered posterior leaflet is more common with infero-posterior MI (MR jet is strictly posterior and lateral, following the angle of the restricted posterior leaflet). Papillary muscle rupture should be suspected in a patient with acute myocardial infarction who develops a new holosystolic murmur and evidence of congestive heart failure. The differential diagnosis is obviously between papillary muscle rupture and acute VSD. Colour flow Doppler imaging allows clear separation of MR from VSD in most instances.

**5. VSD-** In the pre-interventional era, ventricular septal defect occurred in 3% to 5% of STEMI. There is usually a sudden deterioration in the patient’s condition and a new harsh systolic murmur. It is associated with a high mortality and requires urgent surgical intervention. The defect usually occurs in the apical septum with anterior MI (LAD) and in the basal inferior septum with inferior MI (RCA). However, VSD after acute anterior myocardial infarction is unpredictable in location and can occur anywhere in the ventricular septum.

**6. Ventricular rupture and cardiac tamponade-** Rupture of the ventricular free wall is usually a devastating complication, causing rapid haemorrhage into the pericardium and fatal cardiac tamponade in around 75% of cases. However, sometimes the ventricular rupture can be contained by adhesions or thrombosis, causing a more stable (but nonetheless still extremely dangerous) pseudoaneurysm which can, if time permits, be repaired surgically.

**7. Dressler's syndrome-** Pericardial effusion occurring 2–10 weeks after the myocardial infarction is likely to be due to Dressler's syndrome, a form of pericarditis also known as postmyocardial infarction syndrome. Patients may present with pleuritic chest pain, fever and a pericardial friction rub. Dressler's syndrome is thought to be an autoimmune response, caused by the release of myocardial antigens, and is also seen in some patients after cardiac surgery (post-pericardiotomy syndrome).

**8. Assessment of coronary flow reserve (CFR)** represents the ratio between maximal (stimulated) coronary blood flow, induced by using a coronary vasodilator, and baseline (resting) blood flow. The appropriate setting of the echo scanner is an important prerequisite for CFR assessment. LAD is visualised either with a high-frequency transducer (4–8 MHz) or with transthoracic low-frequency probe (3.5–5 MHz) with a second harmonic capability. PDA is situated more deeply in the chest and a low frequency transducer is needed to assess coronary flow. Colour Doppler PRF should be 15–25 cm/s, wall filters set high and pulse Doppler filters should be low. Pulse wave Doppler sample volume should be 3–4 mm.

- LAD flow (95% feasibility) is evaluated from modified apical 3CV (distal-mid LAD aligned with anteroseptal wall) and in modified (medial) 4CV (distal LAD between LV and RV apex)
- PDA flow (60% feasibility) is evaluated from modified apical 3CV/2CV: PDA aligned with inferolateral wall

Coronary flow is biphasic with diastolic predominance. Maximal flow velocity (averaging three cardiac cycles) at baseline and during hyperaemia is considered, although mean flow velocity could be used as well. It should be emphasised that during administration of a vasodilating agent the probe must be kept in the same position and machine settings must not be changed compared with baseline.
Coronary vasodilatation (hyperaemia) obtained infusing:

- Adenosine (149 mcg/kg/min)
- Dipyridamole (0.84 mg/kg/min)

CFR = vasodilatation to baseline peak diastolic velocity ratio. Abnormal CFR $\leq 2$

**9. RV infarction**
Associated most often with inferior infarction (up to one-third of patients with inferior wall infarction)
Isolated RV infarction is rare (3–5%)
RV dilatation
Paradoxical septal motion
Inferior vena cava dilatation
Bulging of the IAS in the LA
Tricuspid regurgitation with low pulmonary pressures
Pulmonary regurgitation with steep