- 1st-degree AV block: Most commonly seen in inferior MI. Observe closely as approximately 40% develop higher degrees of AV block (in which case CCB and β-blockers should be stopped)
- Wenckebach phenomenon: (Mobitz type I) Does not require pacing unless poorly tolerated.
- Mobitz type II block: high risk of developing sudden complete AV block; **should be paced**
- Complete AV block: Usually resolves within a few days. Insert **pacemaker** (may not be necessary after inferior MI if narrow QRS, reasonably stable and pulse ≥40–50)
- Bundle branch block: MI complicated by trifascicular block or non-adjacent bifascicular disease (p132) should be paced

Rupture

Ventricular septal defect

Rupture of the interventricular septum usually occurs in the first week and is seen in around 1-2% of patients. Features: acute heart failure associated with a **pan-systolic murmur, ↑JVP**. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed. 50% mortality first week.

Acute mitral regurgitation

More common with infero-posterior infarction and may be due to ischaemia or rupture of the papillary muscle. An early-to-mid systolic murmur is typically heard. Patients are treated with vasodilator therapy but often require emergency surgical repair.

Left ventricular free wall rupture

This is seen in around 3% of MIs and occurs around 1-2 weeks afterwards. Patients present with acute heart failure secondary to **cardiac tamponade** (raised JVP, pulsus paradoxus, diminished heart sounds). Urgent **pericardiocentesis** and **thoracotomy** are required.

Tamponade

Due to left ventricular free wall rupture. Presents with **Beck's Triad** (low BP, ↑JVP, muffled heart sounds) **pulsus paradoxus** (>10mmHg DROP in BP on inspiration) **Kussmaul's sign** (RISE in JVP on inspiration). Diagnosis: **echo**. Treatment: **pericardial aspiration** (provides temporary relief), surgery.

Heart failure

Acute (Right ventricular failure): Presents with low cardiac output and ↑JVP. **Fluid is key**; avoid vasodilators (e.g. nitrates) and **diuretics**. Inotropes are required in some cases.

Chronic (Left ventricular failure): As described above, if the patient survives the acute phase their ventricular myocardium may be dysfunctional resulting in chronic heart failure. Loop **diuretics** such as furosemide will decrease fluid overload. Both ACE-inhibitors and beta-blockers have been shown to improve the long-term prognosis of patients with chronic heart failure

NB: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4225807/

- For **right ventricular failure** *with normal afterload* (i.e. no pulmonary HTN), we want to ↑preload to help RV pump as much as blood as possible into lungs, LV and into systemic circulation ∴ **fluid and avoid diuretics**. We don't care about peripheral oedema, we care about systemic perfusion!
    - However, when RV failure occurs in the setting of increased RV afterload, volume loading can result in displacement of the interventricular septum toward the LV and impaired LV diastolic filling. At the same time, RV dilation increases free wall tension, resulting in increased oxygen demand and decreased RV perfusion. In this setting, intravascular volume may need to be decreased.