Heart Failure Diagnosis and Investigation

Heart failure is a clinical syndrome characterised by:

- Typical symptoms: breathlessness, fatigue, ankle swelling.
- Typical signs: tachycardia, tachypnoea, pulmonary rales, pleural effusion, raised jugular venous pressure (JVP), peripheral oedema, hepatomegaly.
- Objective evidence of a structural or functional abnormality of the heart at rest: cardiomegaly, third heart sound, cardiac murmurs, echocardiogram abnormalities, raised natriuretic peptide concentration.

The European Society of Cardiology (ESC) guidelines require that there be symptoms, signs and objective evidence present before a diagnosis of heart failure can be made. Heart failure has a number of different aetiologies (see 'Aetiology', below) - always try to determine the cause. Heart failure should never be the only diagnosis, as it is a syndrome occurring as a result of other diagnostic entities.

Classification

Acute and chronic
Heart failure has traditionally been described as acute or chronic but this can be confusing and should be used to describe time, rather than severity. Acute heart failure can present as new-onset heart failure in people without known cardiac dysfunction, or as acute decompensation of chronic heart failure.

Systolic and diastolic
Similarly, a distinction is frequently made between systolic and diastolic heart failure. This is somewhat arbitrary and many patients with heart failure have evidence of both. Left ventricular systolic dysfunction (LVSD) is usually defined as an LV ejection fraction <40% on echocardiography. Some symptomatic patients have a normal ejection fraction and no obvious cause for increased myocardial demand. This condition has been variously termed diastolic heart failure, heart failure with preserved LV function, heart failure with a normal ejection fraction or heart failure with preserved systolic function.

High and low output
Certain medical conditions (see 'Aetiology', below) increase demands on cardiac output, causing a clinical picture of heart failure and this is known as high-output cardiac failure. The primary abnormality is not the heart and the heart failure is reversible with treatment. In low-output failure, cardiac output is inadequate to perfuse the body (ie ejection fraction <40%), or can only be adequate with high filling pressures.

Epidemiology

- Prevalence of asymptomatic ventricular dysfunction is approximately 4%.
- 1-2% of the adult population in developed countries have heart failure, with the prevalence rising to 10% in patients 70 years of age or older. [1]
- Community estimates of prevalence vary from 1.6 to 4.6 cases per 1,000 in men aged 45-74 years and from 0.9 to 2.2 cases per 1,000 in women. [4]
- In younger age groups, heart failure is more common in men because of the earlier onset of coronary heart disease (CHD).
- Prevalence is increasing due to ageing populations and improved survival after coronary events and secondary prevention.

A UK survey of heart failure management in general practice found an average prevalence of 8.3 per 1,000 population. [5]

Aetiology

Coronary heart disease and hypertension are the most common causes of heart failure in the UK.

- Valve heart disease - approximately 10% of cases:
  - Aortic stenosis can cause left ventricular hypertrophy (LVH) due to chronic excessive afterload.
  - Aortic or mitral regurgitation, atrial septal defect (ASD), ventricular septal defect (VSD) and tricuspid incompetence cause excessive preload.
Heart failure secondary to myocardial disease:
- Coronary heart disease (myocardial infarction (MI) and ischaemia, arrhythmias - eg, atrial fibrillation (AF), heart block).
- Hypertension (increased vascular resistance, often with LVH but preserved ejection fraction).
- Cardiomyopathies.
- Drugs - eg, beta-blockers, calcium antagonists, anti-arrhythmics, cytotoxics.
- Toxins - eg, alcohol, cocaine, mercury, cobalt, arsenic.
- Endocrine - eg, diabetes, hypothyroidism, hyperthyroidism, Cushing's syndrome, adrenal insufficiency, excessive growth hormone, phaeochromocytoma.
- Nutritional - eg, deficiencies of thiamine, selenium, carnitine, and obesity, cachexia.
- Infiltrative - eg, sarcoidosis, amyloidosis, haemochromatosis, Löffler's eosinophilia, connective tissue disease.
- Infective - eg, Chagas' disease, HIV.

High-output failure - this occurs when cardiac output is normal or increased in the face of much increased needs. It can occur with a normal heart but even earlier if there is heart disease. Causes include:
- Anaemia.
- Pregnancy.
- Hyperthyroidism.
- Paget's disease of bone.
- Arteriovenous malformations.
- Beriberi.

Presentation
In general, the heart fails as a whole. However, sometimes a disproportionate burden falls on one ventricle and this influences the pattern of symptoms and signs. There is no symptom or sign that is both sensitive and specific for chronic heart failure.[6]

Symptoms
Patients do not necessarily have all and some may be predominant at times. In addition, patients may be depressed or complain of drug-related side-effects.
- Dyspnoea and fatigue (may limit exercise tolerance).
- Fluid retention (may cause pulmonary or peripheral oedema).
- Orthopnoea.
- Paroxysmal nocturnal dyspnoea (PND).
- Nocturnal cough (± pink frothy sputum) or wheeze.
- Nocturia, cold peripheries, weight loss and muscle wasting.
- Right ventricular failure (RVF): peripheral oedema (up to thighs, sacrum, abdominal wall), abdominal distension (ascites), nausea, anorexia, facial engorgement, pulsation in the neck and face (tricuspid regurgitation), epistaxis.
Signs
The patient may look ill and exhausted, with tachypnoea, cool peripheries, peripheral ± central cyanosis.[7]

- There may be a tachycardia at rest, low systolic blood pressure (BP), a displaced apex (LV dilatation) or RV heave (pulmonary hypertension), a narrow pulse pressure or pulsus alternans (alternating large and small pulse pressures) and a raised JVP.
- There may be a gallop rhythm due to presence of S3 (see also the separate Heart Auscultation article) or murmurs of mitral or aortic valve disease.
- Bilateral basal end-inspiratory crackles ± wheeze ('cardiac asthma').
- Pleural effusions.
- Tender hepatomegaly - pulsatile in tricuspid regurgitation, with ascites.
- Often extensive peripheral oedema.

The peak expiratory flow rate may be reduced but, if it is <150 litres/minute, suspect chronic obstructive pulmonary disease (COPD) or asthma.

Investigations
The National Institute for Health and Care Excellence (NICE) makes the pathway for diagnosis explicit after detailed history and examination.[8]

If there has been a previous MI
The patient should be referred for specialist assessment and Doppler echocardiography within two weeks. Echocardiography is the key test to provide a semi-objective assessment of cardiac function. It enables an assessment of:

- Overall LV systolic function.
- Diastolic function (necessary to diagnose heart failure with preserved ejection fraction).
- LV wall thickness.
- Valvular disease.
- Estimation of pulmonary artery systolic pressure.

If an abnormality is found consistent with heart failure, the severity, aetiology, precipitating factors, type of cardiac dysfunction and treatable causes are assessed. If there is no obvious abnormality, consider measuring serum natriuretic peptides (if not already done). If the levels are raised, there may be heart failure with a preserved ejection fraction or another diagnosis. If the levels are normal, the diagnosis is unlikely to be heart failure.

No previous MI
B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) are released into the blood when the myocardium is stressed.[9] Measure serum natriuretic peptides:

- If levels are high (BNP ≥400 pg/ml), the patient should be referred for specialist assessment and Doppler echocardiography within two weeks - as before.
- If levels are raised (BNP ≥100-400 pg/ml), the referral and assessment may be made within six weeks.
- If the levels are normal (BNP ≤100 pg/ml), heart failure is unlikely and other diagnoses should be considered.

Very high levels of serum natriuretic peptides (≥400 pg/ml) carry a poor prognosis. Natriuretic peptide levels may also be elevated in other conditions such as chronic hypoxaemia, renal dysfunction, advanced age, liver cirrhosis and sepsis.

The European Society of Cardiology (ESC) guidelines place almost equal emphasis on the role of the 12-lead ECG.[1] This not only identifies potential aetiological factors but is also necessary for treatment decisions - eg, rate control and anticoagulation for AF or pacing for bradycardia. A normal ECG makes LVSD unlikely (negative predictive value of 98%).

Echocardiography is recommended in patients with suspected heart failure who have either a raised BNP or NT-proBNP level or abnormal ECG, in order to confirm the diagnosis and establish the underlying cause.[8] Assessing the aetiology may also involve[10]

- Blood tests: FBC, U&E and creatinine, LFTs, glucose, fasting lipids, TFTs; consider cardiac enzymes if an undiagnosed MI is possible in the preceding few days.
- CXR - provides supportive evidence for heart failure and helps to exclude other potential causes of breathlessness. Typical findings in heart failure include:
  - Cardiomegaly (cardiothoracic ratio >50%).
  - Ventricular hypertrophy.
  - Prominent upper lobe veins (upper lobe diversion).
  - Peribronchial cuffing.
  - Diffuse interstitial or alveolar shadowing - classical perihilar 'bat's wings' or nodular (especially with pre-existing COPD).
  - Fluid in the fissures.
  - Pleural effusions.
  - Kerley B lines.

Apart from pulmonary congestion, CXR findings are only predictive of heart failure where there are co-existing typical signs and symptoms.
- Urinalysis.
- Lung function tests (peak flow or spirometry).
- Cardiac magnetic resonance imaging - the gold standard for assessing ventricular volumes, mass and wall motion. It can be used with contrast to identify inflammation, infiltration and scarring of the myocardium. Its use is limited by availability and cost.[1]
- Exercise testing, CT angiography or coronary angiography is not part of routine diagnosis of heart failure but may be considered where CHD is suspected.
- Radionuclide imaging may be helpful to assess global ventricular function when echocardiography is not possible.

Staging

The New York Heart Association's (NYHA) Classification of Heart Failure has provided a clinically useful, functional classification, outlined below:[7]

**NYHA Heart Failure Severity Classification**

- Class I: no symptoms on ordinary physical activity.
- Class II: slight limitation of physical activity by symptoms.
- Class III: less than ordinary activity leads to symptoms.
- Class IV: inability to carry out any activity without symptoms.

Indications for specialist referral[7]

For people with suspected heart failure:

**Refer urgently (within two weeks) for specialist assessment and echocardiography**

- People who have had a previous MI.
- People without a history of MI who have high levels of natriuretic peptide - BNP level above 400 pg/mL (116 pmol/L) or NT-proBNP level above 2000 pg/mL (236 pmol/L).
- People with severe symptoms (if admission is not indicated).
- Women who are pregnant.
Refer within six weeks

- People without a history of MI who have a BNP level between 100-400 pg/mL (29-116 pmol/L) or an NT-proBNP level between 400-2000 pg/mL (47-236 pmol/L).

If natriuretic peptide levels are normal (BNP level less than 100 pg/mL (29 pmol/L) or NT-proBNP less than 400 pg/mL (47 pmol/L)), a diagnosis of heart failure is unlikely. However, referral may still be needed if:

- Clinical suspicion of heart failure persists and the person is obese or taking drugs which lower natriuretic peptide levels (diuretics, angiotensin-converting enzyme inhibitors, angiotensin-II receptor antagonists, beta-blockers or aldosterone antagonists).
- Another condition is suspected, which requires referral to a specialist.

If it is not possible to measure natriuretic peptide levels, refer if:

- The ECG is abnormal.
- The ECG is normal but there is still a strong suspicion of heart failure.

Prognosis

- Prognosis is poor on the whole, with approximately 50% of people with heart failure dying within four years of diagnosis. [7]
- The mortality rate in the UK appears to be improving. A UK study found that the six-month mortality rate for people with heart failure had improved from 26% in 1995 to 14% in 2005. [11]
- The prognosis for people with heart failure and preserved LV ejection fraction is a little better than the prognosis for people with heart failure and reduced ejection fraction.

Further reading & references

- Acute heart failure: diagnosis and management in adults; NICE Quality Standard, December 2015
- Acute and Chronic Heart Failure; European Society of Cardiology (2012)
- Diagnosing and managing acute heart failure in adults; NICE Clinical Guidelines (Oct 2014)
- Management of chronic heart failure; Scottish Intercollegiate Guidelines Network - SIGN (2016)
- Heart failure - chronic; NICE CKS, May 2015 (UK access only)
- Chronic heart failure: Management of chronic heart failure in adults in primary and secondary care; NICE Clinical Guideline (August 2010)

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