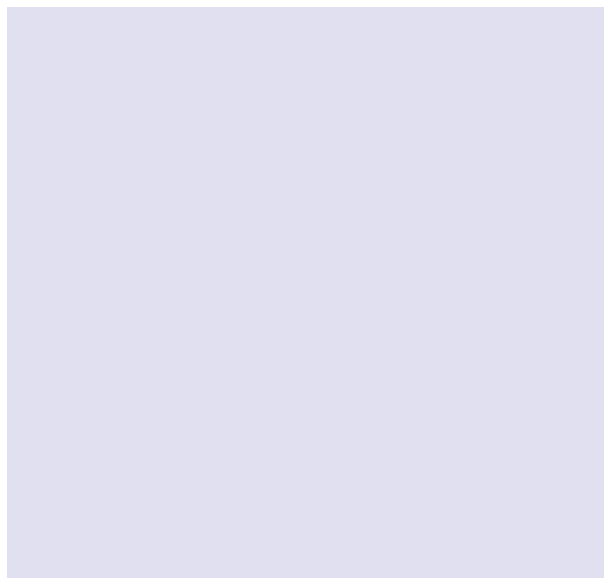


CHRONIC HEART FAILURE

— diagnosis of the disease

By MOJGAN SANI, DPHARM, MBA, MRPHARMS

Chronic heart failure affects more than 10 per cent of those over the age of 80 and is a major cause of sudden death. The first article in this month's special feature reviews the epidemiology, pathophysiology, clinical features, investigation and prognosis of the disease



Coloured X-ray of the chest showing left ventricular failure. The shadow of the heart (yellow) is partly obscured by fluid (appears opaque) which has leaked into the lungs because of increased pressure in pulmonary veins

Chronic heart failure is a major cause of morbidity and mortality, and is responsible for about 5 per cent of all admissions to hospital. Heart failure occurs when the cardiac output is insufficient to meet the body's needs. The European Society of Cardiology task force on heart failure bases diagnosis on both the presence of symptoms and objective evidence for cardiac dysfunction.¹ The degree of limitation the patient perceives is classified according to the New York Heart Association (NYHA) system, and is shown in Table 1. Chronic heart failure develops as a result of cardiac disease and is therefore associated with a number of signs and symptoms due to circulatory and neurohormonal reactions (Table 2, p88).

EPIDEMIOLOGY

Life expectancy of chronic heart failure patients is significantly reduced making the disease a major challenge to the health service. The majority of individuals developing heart failure are aged over 70 years and

the most common cause is coronary artery disease and hypertension.

The prevalence of chronic heart failure increases steeply with age from 1–2 per cent in 50–60 year olds to over 10 per cent in those aged 80 years and over. There is a poor prognosis with 30 per cent mortality within one year, increasing to 60–70 per cent mortality after five years.²

The Framingham Heart Study showed that 50 per cent of all deaths were sudden.³ It was also shown that the age adjusted annual incidence of chronic heart failure was 0.14 per cent in women and 0.23 per cent in men, with better survival rates in women than men.

In the UK General Practice Morbidity Survey, the prevalence of chronic heart failure increased from 0.1 per 1,000 population in those aged 25–44 years, to 140.3 per 1,000 in those aged ≥85 years.⁴ However, these data may lack accuracy because the diagnosis was not made in specialist centres.

PATHOPHYSIOLOGY

Chronic heart failure may occur as a result of damage to the coronary artery, valve disease, hypertension, alcohol misuse or viral infections. It is considered to be a neurohormonal imbalance. Chronic activation of neurohormonal pathways such as the

Table 1: NYHA classification

Class I	Patients with cardiac disease but no limitation during normal physical activity
Class II	Slight limitation, symptoms occur on moderate exertion such as walking and climbing stairs
Class III	Marked limitation, symptoms occur on minimal exertion such as walking on flat ground
Class IV	Breathlessness at rest

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renin-angiotensin-aldosterone and the sympathetic nervous system leads to sodium and water retention, renal dysfunction and progression of heart failure disease.

The natriuretic peptide system consists of three peptides with similar structures circulating in the plasma. Atrial natriuretic peptide (ANP), mainly secreted from the cardiac atria, has natriuretic, diuretic and vasodilatory properties. Brain natriuretic peptide (BNP), which was originally discovered in porcine brain, is predominantly secreted from the ventricles of the human heart and has similar actions to ANP. The third type is the C-type natriuretic peptide (CNP) which is present mainly in the central nervous system and appears to have limited natriuretic and vasodilatory properties.

In patients with heart failure the plasma levels of ANP and BNP are markedly elevated, although this is not the case for CNP.⁵ There has been considerable interest in the use of natriuretic peptides as indicators of left ventricular systolic performance. This has traditionally been used as the “gold standard” for assessing prognosis in patients with heart failure. Most cardiologists agree that ideally all patients with suspected heart failure should be investigated with an echocardiogram, but this may be incompatible with the current level of provision of echocardiography services in the UK and elsewhere. This has led to an increased use of BNP investigations.

It is also well established that the activation of the renin-angiotensin-aldosterone system (RAAS) correlates with mortality in chronic heart failure.⁶ The harmful effects of aldosterone are being appreciated more in addition to the better understood adverse effects of angiotensin-II. The harmful effects of angiotensin-II and aldosterone may be described as sodium retention, potassium loss and increased blood pressure. However, aldosterone is also now known to cause magnesium loss, myocardial fibrosis, sympathetic activation, parasympathetic inhibition and ultimately ventricular arrhythmias.

Neurohormonal activation Neurohormonal mechanisms are activated so that they compensate for myocardial cell dysfunction to normalise output and function. The sympathetic nervous system is activated in response to an increase in pre-load. This will work to increase both the force and frequency of contraction. This activation of the sympathetic nervous system occurs early in the disease process leading to the stimulation of the renin-angiotensin system as the disease progresses and symptoms develop. In addition, locally active vasoconstricting factors such as endothelin are released.

Neurohormonal activation leads to sodium and water retention. Prolonged activation of the sympathetic nervous system and renin-angiotensin system exert adverse

Table 2: Signs and symptoms of chronic heart failure

SIGNS	SYMPTOMS
Breathlessness	Breathlessness
Wheeze	Swollen ankles
Tachycardia	Tired
Third or fourth heart sound	Need to sit out of bed
Basal crackles	Need to open windows for air
Jugular venous pressure raised	Fatigue
Ankle oedema	Wheezing
Cardiomegaly	Cough at night
Murmurs	Painful abdomen
Tender large liver	Swollen abdomen
Ascites	

effects on the heart independent of their haemodynamic action.

CLINICAL FEATURES

Patients with chronic heart failure pass through a phase of asymptomatic left ventricular dysfunction to develop exercise intolerance, shortness of breath, fatigue, oedema, ascites and ultimately skeletal muscle wasting. Chronic heart failure is a clinical syndrome with identifiable causes, with the heart usually enlarged and becoming more spherical over time.

Chronic heart failure is a condition reflected in symptoms and signs by the effects of low cardiac output with retention of sodium and water. The degree of limitation is graded by the NYHA system as has previously been discussed.

Dyspnoea Breathlessness is the most common complaint of a pre-load problem. This symptom may be worse at night when lying flat (orthopnoea) and can sometimes wake the patient. Paroxysmal nocturnal dyspnoea reflects nocturnal absorption of fluid during sleep and can lead to gasping, coughing and wheezing.

Fatigue and lethargy This is related to abnormalities in skeletal muscle with impaired muscle blood flow. Reduced cerebral blood flow, accompanied by abnormal sleep patterns, may lead to insomnia and confusion in severe chronic heart failure.

Oedema Swollen ankles are what patients most often complain of and this symptom is worse at the end of the day. Older people often do not mention the oedema until it is severe, even above the knees. An increase in weight may be associated with fluid retention.

INVESTIGATIONS

A summary of the NICE guidance on the diagnosis of heart failure is presented in Figure 1 (p90).

Chest X-ray The chest X-ray is a routine examination in patients with suspected heart failure and can also be used when monitoring the response to treatment. Cardiomegaly (enlarged heart) may be seen, but this is dependent on the severity and duration of haemodynamic disturbance and,



Pitting oedema associated with chronic heart failure

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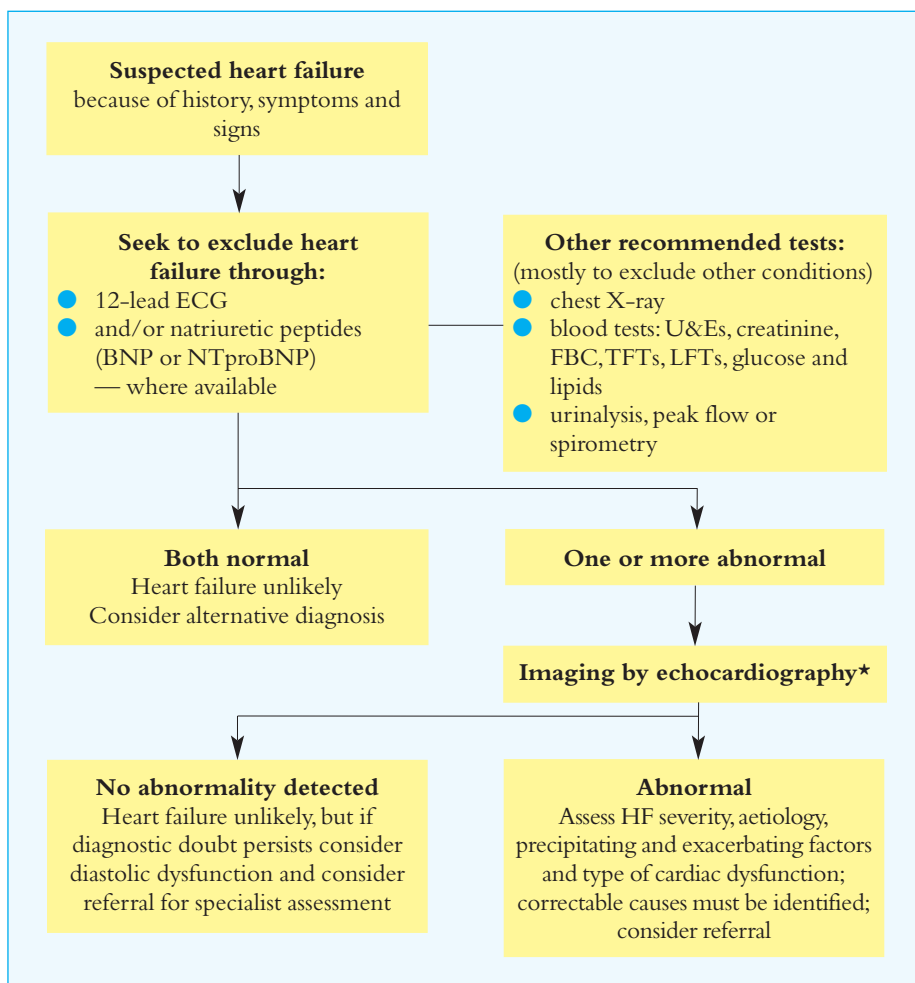


Figure 1: Algorithm summarising recommendations for the diagnosis of heart failure. * Alternative methods of imaging the heart should be considered when a poor image is produced by transthoracic doppler 2D-echocardiography — alternatives include transoesophageal echocardiography, radionuclide imaging or cardiac magnetic resonance imaging. BNP=B-type natriuretic peptide; ECG=electrocardiogram; FBC=full blood count; LFTs=liver function tests; NTproBNP=N-terminal pro-B-type natriuretic peptide; TFTs=thyroid function tests; U&Es=Urea & electrolytes. (Reproduced by permission of the Royal College of Physicians)⁸

used alone, is not an accurate means of detecting left ventricular dysfunction.⁷

Initially, when pulmonary venous congestion develops, blood flow is diverted to the upper zones of the lungs (upper lobe diversion) so that the calibre of the upper zone vessels equal or exceed that of the lower zone vessels.⁹ Further congestion results in secretion of fluid into the lung interstitium and septa. Normally these areas cannot be detected on a chest X-ray but when oedema is present the engorged vasculature becomes visible as short thin lines which are known as Kerley lines. Alveolar oedema is sometimes present in more severe pulmonary congestion (pressures above 25mmHg) and this may give rise to a “bats wings” appearance on the X-ray.⁹

Electrocardiography Most patients with chronic heart failure show abnormalities on a 12-lead electrocardiogram (ECG) tracing. It is a quick, simple and inexpensive test to perform and used together with a clinical assessment and chest X-ray it can provide an

initial diagnosis of heart failure. An ECG may reveal abnormalities involving the Q wave, T wave, ST segment, left ventricular hypertrophy, bundle branch block and atrial fibrillation.⁷

An ECG can be taken over 24 hours using a Holter monitor. This is useful in detecting arrhythmias in patients with symptoms such as palpitations or dizziness.

Echocardiography An echocardiograph is a non-invasive test which provides valuable information about the structure and function of the heart and should be conducted in all patients with suspected heart failure. A test may reveal the presence of systolic and/or diastolic dysfunction and may also reveal the cause, eg, cardiomyopathy, valve disease, and intracardiac thrombus and embolism.

Left ventricular dysfunction can be quantified by measuring the ejection fraction which is the stroke volume expressed as a percentage of the left ventricular end diastolic volume. An ejection fraction of less

than 45 per cent is generally accepted as evidence of systolic dysfunction.¹⁰

Regional abnormalities are usually observed visually and are described as hypokinetic (reduced systolic contraction), akinetic (no systolic contraction) and dyskinesic (abnormalities of direction or timing of contraction).⁷

Haematology and biochemistry Blood analysis in heart failure patients may involve a full blood count, measurement of electrolytes and renal function, and tests for metabolic abnormalities (eg, liver function tests, thyroid function tests). These can be used to identify the underlying cause of heart failure such as thyrotoxicosis or anaemia and for the monitoring of progression of the condition.

Baseline renal function tests to measure urea and creatinine are usually taken before starting treatment. Renal function often deteriorates with severe chronic heart failure. This can be due to reduced renal perfusion and some treatments including angiotensin-converting enzyme (ACE) inhibitors and high doses of diuretics can result in increased creatinine concentrations.

Electrolyte levels, in particular sodium and potassium, are monitored regularly in all chronic heart failure patients. In mild and moderate chronic heart failure electrolyte levels are usually normal but in severe chronic heart failure hyponatraemia may be present because of an inability to excrete water. This occurs for several reasons including reduced renal blood flow, sodium restriction, the use of high doses of diuretics and the activation of the neurohormonal systems.

Hypokalaemia can occur with the use of diuretics without either potassium supplements or potassium sparing diuretics. Hyperkalaemia can occur with the use of ACE inhibitors and potassium sparing diuretics particularly in patients with reduced renal blood flow. Both hypo and hyperkalaemia can result in cardiac arrhythmias.

Abnormal liver function tests (serum bilirubin, aspartate aminotransferase, lactate dehydrogenase) may indicate hepatic congestion which is seen in the later stages of heart failure. Thyroid function tests are useful in excluding thyrotoxicosis as a cause of heart failure.

Radionuclide studies Radionuclide imaging can be used to measure the ejection fraction, wall motion abnormalities, and for assessing left ventricular cavity size. Radionuclide imaging exposes the patient to radiation and tends to be used when echocardiography is not possible.

Cardiopulmonary exercise tests Exercise tests are used to differentiate between the grades of chronic heart failure and are a good indicator of the prognosis of the condition. The most commonly used test is a measure of the maximal rate of oxygen

uptake (VO_2 max) during progressive exercise to exhaustion.

PROGNOSIS

Determining the prognosis for patients with chronic heart failure is extremely important because both patients and their relatives usually want to know so that they can plan their lives. Additionally, understanding the prognosis can help with plans for transplantation and long-term management. Prognostic factors predicting worsening of survival may include: ischaemic aetiology, long disease duration, clinical instability, history of syncope, high NYHA class, abnormal heart rate recovery and inability to walk short distances.

Sudden death often occurs unexpectedly in seemingly stable patients. The ability to predict high-risk patients remains, therefore, poor.

SUMMARY

Patients with chronic heart failure are part of a growing group. Chronic heart failure morbidity and mortality are increasing. However, therapy is improving and the development of innovative technological and pharmacological interventions is continuing.

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Chronic heart failure national guidelines

The full version of “Chronic heart failure — national clinical guideline for diagnosis and management in primary and secondary care” is available from the publications department at the Royal College of Physicians (www.rcplondon.ac.uk)
Price UK £25.00; overseas £28.00 (ISBN 1 86016 188X)