

# Guidelines

## Guidelines for the diagnosis of heart failure

THE TASK FORCE ON HEART FAILURE OF THE EUROPEAN SOCIETY OF CARDIOLOGY

### Introduction

The epidemiology of heart failure in Europe is poorly described. The presentation and aetiology are heterogeneous and little is known about differences between countries.

Estimates of the prevalence of heart failure in the general population range from 0.4% to 2%<sup>[1–4]</sup>. The prevalence of heart failure increases rapidly with age<sup>[1]</sup> and, as the proportion of the population that is elderly is increasing, this partly accounts for the rising prevalence of heart failure<sup>[5,6]</sup>. Unlike other common cardiovascular diseases the age-adjusted mortality attributed to heart failure also appears to be increasing<sup>[7]</sup>. The European Society of Cardiology represents countries with a total population of over 500 million, suggesting that there are at least 2 million and possibly in excess of 10 million patients with heart failure in Europe. The prognosis of heart failure is uniformly poor if the underlying problem cannot be rectified. Half of patients carrying a diagnosis of heart failure will die within 4 years and in patients with severe heart failure half will die within one year<sup>[6,7]</sup>.

Recent studies show that the accuracy of diagnosis by clinical means alone is often inadequate<sup>[4,8]</sup> particularly in women, the elderly and the obese. In order to study the epidemiology and prognosis and to optimise the treatment of heart failure the uncertainty relating to the diagnosis must first be minimised or avoided.

The aim of this report is to provide practical guidelines for the diagnosis and assessment of heart failure for use in clinical practice, for epidemiological surveys and for clinical trials. The guidelines have been designed primarily to address the basic minimum requirements for diagnosis of heart failure for all medical and allied staff but also to give guidance for more advanced aspects of diagnosis for the cardiology specialist.

The guidelines on diagnosis and assessment of heart failure are the first of a series. Guidelines for the treatment of heart failure are being prepared currently. Future position papers will cover the epidemiology of heart failure in Europe and guidelines for the assessment of prognosis.

### Methodology

This report was prepared by a drafting committee (see appendix for composition). The initial document was

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considered by the nucleus of the Task Force on Heart Failure of the European Society of Cardiology (see appendix), revised, and circulated to all 170 members of the Task Force. The document was re-drafted in the light of the comments received and was then circulated to the Board of The European Society of Cardiology and chairpersons of nine Working Groups (see appendix). The document was subsequently modified in the light of the comments received from them and the Board of the European Society of Cardiology before being produced in its final version.

### Descriptive terms in heart failure

#### ACUTE VERSUS CHRONIC HEART FAILURE

*Chronic heart failure*, often punctuated by acute exacerbations, is the most common form of heart failure. A definition of chronic heart failure is given below.

The term *acute heart failure* is often used, exclusively, to mean acute (cardiogenic) pulmonary oedema. However, acute heart failure could also apply to cardiogenic shock, a syndrome characterised by a low arterial pressure, oliguria and a cool periphery, that needs to be distinguished from pulmonary oedema. It is advisable not to use the term acute heart failure but the more precise terms acute pulmonary oedema and cardiogenic shock.

#### SYSTOLIC VERSUS DIASTOLIC HEART FAILURE

As ischaemic heart disease is the commonest cause of heart failure in industrialised societies most heart failure is associated with evidence of left ventricular systolic dysfunction, although diastolic impairment at rest is a common if not universal accompaniment. Diastolic heart failure is often diagnosed when symptoms and/or signs of heart failure occur in the presence of a normal ejection fraction at rest. Predominant diastolic dysfunction is relatively uncommon in younger patients but increases in importance in the elderly, in whom hypertension, myocardial hypertrophy and fibrosis make a greater contribution to cardiac dysfunction. Most patients with heart failure and impairment of diastolic function also have impaired systolic function. Conclusive evidence that most elderly patients with a diagnostic label of heart failure but with normal systolic function at rest do indeed have heart failure is lacking.

Table 1 Definition of heart failure. Criteria 1 and 2 should be fulfilled in all cases

1. Symptoms of heart failure (at rest or during exercise) and
2. Objective evidence of cardiac dysfunction (at rest) and
3. Response to treatment directed towards heart failure (in cases where the diagnosis is in doubt)

OTHER DESCRIPTIVE TERMS IN HEART FAILURE

Right and left heart failure refer to syndromes presenting predominantly with congestion of the systemic or pulmonary veins respectively. The terms do not necessarily indicate which ventricle is most severely damaged. High and low-output, forward and backward, overt, treated, congestive and undulating are other descriptive terms still in occasional use; the clinical utility of these terms have yet to be determined.

DEFINITION OF CHRONIC HEART FAILURE

Many definitions of chronic heart failure exist<sup>[9-12]</sup> but highlight only selective features of this complex syndrome. None is entirely satisfactory.

A simple objective definition of chronic heart failure is currently impossible as there is no cut-off value of cardiac or ventricular dysfunction or change in flow, pressure, dimension or volume that can be used reliably to identify patients with heart failure. The diagnosis of heart failure relies on clinical judgement based on a history, physical examination and appropriate investigations.

The Task Force took the view that the essential components of heart failure were that the patients should have the following features; symptoms of heart

failure, typically breathlessness or fatigue, either at rest or during exertion, or ankle swelling and objective evidence of major cardiac dysfunction at rest (Table 1). A clinical response to treatment directed at heart failure alone is not sufficient for diagnosis, although the patient should generally demonstrate some improvement in symptoms and/or signs in response to treatment, with a diuretic, digitalis glycosides or an ACE inhibitor. It should also be recognised that treatment may obscure a diagnosis of heart failure by relieving the patients symptoms. The distinctions between cardiac dysfunction, heart failure, heart failure that has been rendered asymptomatic by therapy and transient heart failure are outlined in Fig. 1. It is important to note that exercise-induced ventricular dysfunction, usually due to myocardial ischaemia, may cause a rise in ventricular filling pressure and a fall in cardiac output and induce symptoms of heart failure such as breathlessness. However, as the treatment of this condition is generally different from that of heart failure secondary to chronic ventricular dysfunction it is not clinically useful to classify such patients as having chronic heart failure.

Aetiology of heart failure in Europe

Heart failure should never be the final diagnosis. The aetiology of heart failure and the presence of exacerbating factors or other diseases that may have an important influence on management should be carefully considered in all cases. The extent to which the cause of heart failure should be pursued by further investigation will depend on the resources available and the likelihood that diagnosis will influence management.

The rationale for a basic minimum set of investigations is outlined in Section 1 (vide infra). Additional investigations shown in Section 2 (vide infra) should be undertaken if a specific reversible cause for heart failure is suspected.

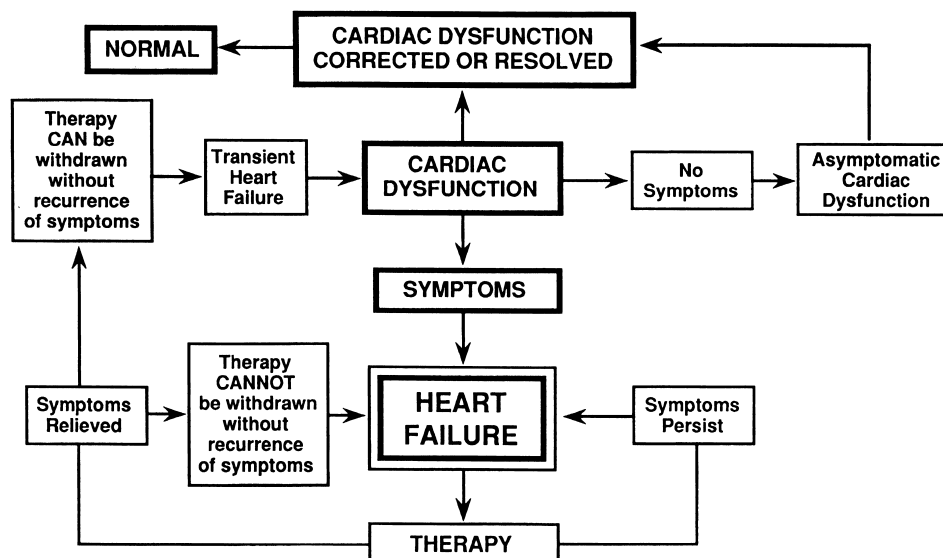


Figure 1 Cardiac dysfunction, heart failure, and heart failure rendered asymptomatic.

Chronic heart failure may be due to myocardial dysfunction, arrhythmias, valve abnormalities or pericardial disease. Anaemia, renal or thyroid dysfunction and cardio-depressant drugs may exacerbate, or more rarely cause, heart failure. Acute pulmonary oedema and cardiogenic shock have a similar aetiological spectrum to chronic heart failure, though pulmonary oedema is rarely due to pericardial disease. Standard textbooks of cardiology should be consulted for a more extensive list of the causes of heart failure<sup>[10,12]</sup>. In Europe myocardial dysfunction secondary to coronary artery disease, usually as a consequence of myocardial infarction, is the most common cause of heart failure among patients under the age of 75 years<sup>[2]</sup> and clear abnormalities in systolic function are usually present. Among elderly patients, who are often less intensively investigated, accurate diagnosis of the presence and the aetiology of heart failure is more difficult and obscured by multiple other diagnoses. Hypertension, hypertrophy, cell loss and fibrosis may be more important causes of heart failure in the elderly and may be more likely to manifest predominantly as abnormalities of diastolic function. The aetiology of heart failure will also depend on ethnic origin, socio-economic status and geographic location.

#### IMPORTANCE OF IDENTIFYING POTENTIALLY REVERSIBLE EXACERBATING FACTORS

Chronic heart failure, pulmonary oedema and shock may be caused by tachy- and bradyarrhythmias or myocardial ischaemia even in patients without major, permanent cardiac dysfunction. Myocardial ischaemia, pulmonary embolism, infection, arrhythmia, renal dysfunction or renal artery stenosis, side effects of drug therapy and excessive fluid, sodium or alcohol intake may all cause or exacerbate symptoms and/or signs of heart failure in patients with pre-existing cardiac dysfunction. It is important to identify any reversible factors in order to treat heart failure optimally.

#### IMPORTANCE OF THE HOLISTIC APPROACH TO PATIENTS WITH HEART FAILURE

In the elderly population, multiple rather than single diseases are the rule rather than the exception. A proper diagnostic formulation must extend beyond the cardiac problem. For instance, in patients with prostatic hypertrophy a vigorous diuresis may precipitate acute urinary retention. Disease of the peripheral vasculature<sup>[13]</sup> and other organs including the kidney and lungs may have an important influence on diagnosis and the choice of treatment. As elderly patients may be more prone to the side-effects of heart failure treatment, especially if this is inappropriate, young and elderly patients should have equal access to basic diagnostic facilities.

#### Aspects of the pathophysiology of the symptoms of heart failure relevant to diagnosis

The origin of the symptoms of heart failure are not fully understood. Increased pulmonary capillary-

pressure is undoubtedly responsible for pulmonary oedema in part, but studies conducted during exercise in patients with chronic heart failure demonstrate no simple relationship between capillary pressure and exercise performance<sup>[14]</sup>. This suggests either that raised pulmonary capillary pressure is not the only factor responsible for exertional breathlessness or that current techniques to measure true pulmonary capillary pressure may not be adequate. Abnormalities of pulmonary diffusion<sup>[15]</sup>, peripheral or respiratory skeletal muscle<sup>[16]</sup> or general cardiovascular deconditioning<sup>[17]</sup> may contribute importantly to the sensation of breathlessness. The origins of fatigue are even more obscure and compounded by difficulties in quantifying this symptom<sup>[18]</sup>. Peripheral oedema is poorly related to right heart pressures; capillary permeability for fluid and small proteins<sup>[19]</sup> and reduced physical activity being important additional factors.

Although impairment of cardiac function is central to the development of heart failure, altered peripheral blood flow, especially to the kidney and skeletal muscle, is typical and probably of major pathophysiological importance<sup>[20]</sup>. Similarly, activation of a number of neuro-endocrine systems is characteristic of heart failure, especially if treated with diuretics<sup>[21]</sup>. Baro-receptor dysfunction is an important link between vasomotor and neuro-endocrine dysfunction<sup>[22,23]</sup>.

## Section 1: Possible methods for the diagnosis of heart failure in clinical practice

### Symptoms and signs in the diagnosis of heart failure

Breathlessness, ankle swelling and fatigue are the characteristic symptoms of heart failure but may be difficult to interpret particularly among elderly patients, the obese and in women. Inter-observer agreement on the presence or absence of symptoms of heart failure may be low<sup>[24]</sup>, at least in the days following a myocardial infarction. There is no standard questionnaire available for the diagnosis of heart failure. In the context of clinical or epidemiological studies, several scoring systems are available that await proper validation and cannot be recommended for clinical practice at present<sup>[25]</sup>.

Peripheral oedema, a raised venous pressure and hepatomegaly are the characteristic signs of congestion of systemic veins<sup>[26,27]</sup>. Peripheral oedema and hepatomegaly are non-specific, while determination of the jugular venous pressure is often difficult. Peripheral oedema is usually absent in well-treated heart failure, even if severe<sup>[27]</sup>. Although cardiologists attain a high agreement on the presence of an elevated jugular venous pressure under study conditions it is likely that reproducibility is much lower among non-specialists<sup>[26]</sup>. Moreover, many patients, even with well documented heart failure, even if severe, do not have an elevated jugular venous pressure<sup>[27]</sup>.

Table 2 New York Heart Association Classification of Heart Failure

Class I.	No limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations.
Class II.	Slight limitation of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations dyspnoea or angina.
Class III.	Marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms.
Class IV.	Unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest with increased discomfort with any physical activity.

N.B. patients in NYHA class I would have to have objective evidence of cardiac dysfunction, have a past history of heart failure symptoms and be receiving treatment for heart failure in order to fulfil the basic definition of heart failure as set out in 'The Guidelines'.

Tachycardia is non-specific and may be absent even in severe heart failure<sup>[27]</sup>. Other signs of heart failure require considerable expertise for their detection. Percussion of the heart for size has been superseded by simple investigations<sup>[28]</sup>. The apex beat is often difficult to palpate and is not an accurate measure of cardiomegaly<sup>[29,30]</sup>. A third heart sound is usually considered to be present in patients with severe heart failure<sup>[27]</sup>, but is not specific to heart failure<sup>[31]</sup>. Although cardiology specialists may attain a high agreement for the presence of a third heart sound under study conditions<sup>[26]</sup> the inter-observer agreement is less than 50% among non-specialists<sup>[32]</sup> and probably even lower in clinical practice. Pulmonary crepitations are not specific to heart failure and again inter-observer differences in eliciting this sign are high<sup>[33]</sup>.

When multiple signs of heart failure are present, including a displaced apex beat, pitting oedema, a raised venous pressure and when a third heart sound is heard confidently then, in the presence of appropriate symptoms, a clinical diagnosis of heart failure may be made with some confidence. Although a clinical diagnosis reached in this way may be specific it is likely to be insensitive and will fail to identify many patients who might benefit from treatment. The subjective component of the examination and the inability to make a permanent direct record with which to convince others, who have not seen the patient, of the diagnosis of heart failure are further major weaknesses of a diagnosis made on clinical features alone.

In summary, symptoms and signs are important as they alert the observer to the possibility that heart failure exists. The clinical suspicion of heart failure must be confirmed by more objective tests.

#### SYMPTOMS AND THE SEVERITY OF HEART FAILURE

Once a diagnosis of heart failure has been established symptoms may be used to classify the severity of heart failure and should be used to monitor the effects of therapy. The New York Heart Association classification (NYHA) is in widespread use (Table 2). The use of examples such as walking distance or number of stairs climbed is recommended. The value of questionnaires for the measurement of quality of life is still being assessed<sup>[34,35]</sup>.

The severity of symptoms are highly dependent on the efficacy of therapy, patient expectation and medical interpretation. Mild symptoms should not be equated with minor cardiac dysfunction. There is a poor relationship between symptoms and the severity of cardiac dysfunction<sup>[25,36]</sup> and between symptoms and prognosis<sup>[37]</sup>.

#### Non-invasive investigation in the diagnosis of heart failure

##### THE ECG

A normal ECG is rare in patients with heart failure and, if present, suggests that the diagnosis of heart failure should be carefully reviewed. The ECG is crucial in confirming the heart rhythm. ECG abnormalities in patients with heart failure often do not suggest any specific underlying cause. The presence of Q-waves suggests myocardial infarction but in the absence of an appropriate history this should be confirmed by other investigations, such as echocardiography.

##### THE CHEST X-RAY

There is a poor relationship between heart size on X-ray and left ventricular function<sup>[38-41]</sup>. Cardiomegaly is frequently absent in acute heart failure, but a normal sized heart associated with clinical evidence suggesting chronic heart failure indicates that the diagnosis should be carefully reviewed. Cardiomegaly lends support to a diagnosis of heart failure, especially if associated with upper lobe venous dilatation, though the latter is a poor guide to the simultaneous pulmonary capillary wedge pressure<sup>[41-44]</sup>. Scrutiny of the lung fields may also reveal evidence of interstitial or alveolar oedema or pleural effusions. The interobserver agreement in the interpretation of pulmonary congestion on the X-ray<sup>[45]</sup> is only modest and the chest X-ray viewed in isolation fails to distinguish reliably between congestion of cardiac or renal origin<sup>[46]</sup>. The relationship between radiological pulmonary congestion and haemodynamic state may depend on the duration as well as severity of the haemodynamic disturbance<sup>[47]</sup>.

The shape of the cardiac silhouette may suggest a specific diagnosis as may calcification in valves,

myocardium or pericardium. Echocardiography is required to differentiate reliably between dilatation of cardiac chambers, hypertrophy and pericardial effusion<sup>[38,39]</sup>. In patients after a myocardial infarction, assessment of left ventricular ejection fraction has, so far, proved inaccurate using clinical information even when combined with information from the ECG and chest X-ray<sup>[24,28]</sup>.

The chest X-ray is useful in helping to exclude pulmonary disease as a cause for symptoms.

#### HAEMATOLOGY AND BIOCHEMISTRY

Anaemia may exacerbate pre-existing heart failure. A raised haematocrit suggests that breathlessness may be due to pulmonary disease, cyanotic congenital heart disease or a pulmonary arteriovenous malformation.

Measurement of serum urea or creatinine is essential for the differential diagnosis from renal failure, which may induce all the features of heart failure secondary to volume overload, and for subsequent management of heart failure. Untreated heart failure is rarely associated with major electrolyte disturbance. Electrolyte disturbances are more common in patients on diuretics. Hyponatraemia and renal dysfunction in the setting of heart failure indicate a worse prognosis. Liver enzymes may be elevated by hepatic congestion.

Urinalysis is useful in detecting proteinuria and glycosuria, alerting the clinician to the possibility of underlying renal problems or diabetes mellitus, conditions that may contribute to or complicate heart failure.

Heart failure due to thyrotoxicosis is frequently associated with (rapid) atrial fibrillation and may be the presenting feature of thyrotoxicosis in the elderly. Hypothyroidism may also present as heart failure.

#### PULMONARY FUNCTION

Measurements of lung function are useful in excluding respiratory causes of breathlessness, though the presence of pulmonary disease does not exclude co-existent heart failure. Epidemiological studies suggest that there is a strong association between chronic obstructive airways disease and ischaemic heart disease, one of the principal causes of heart failure<sup>[49]</sup>.

Peak expiratory flow rate (PEFR) and forced expiratory volume in one second (FEV1) are reduced in heart failure though not to the same extent as in symptomatic obstructive airways disease. In patients presenting with severe breathlessness and wheeze a peak expiratory flow rate  $<2001 \text{ l} \cdot \text{min}^{-1}$  suggests a diagnosis of asthma rather than acute pulmonary oedema<sup>[50]</sup>.

#### ECHOCARDIOGRAPHY

Echocardiography should be used routinely for the optimal diagnosis of heart failure<sup>[36]</sup>. The test is widely available, simple and safe. The functional integrity of

the valves, chamber dimensions, ventricular hypertrophy and systolic and diastolic ventricular function may all be assessed. Evaluation of dimensions, systolic function and regional wall motion abnormalities is reliable. Doppler echocardiographic techniques in experienced hands provide a quantitative assessment of valve gradients and right ventricular systolic pressure; a guide to the presence of pulmonary hypertension can be calculated if tricuspid insufficiency is present, as is common<sup>[51]</sup>. Transoesophageal echocardiography allows evaluation of structure and function in patients who have an inadequate transthoracic echo window or a mechanical mitral valve prosthesis and may be used to assess the atria, pulmonary veins and mitral valve in greater detail if required. As ejection fraction is based on two rather inaccurate measurements of volume which are prone to errors in calculation reproducibility is only moderate. The interpretation of echocardiographic measures of diastolic ventricular function is complex. No practically useful Doppler echocardiographic guidelines are yet available for diagnosing diastolic heart failure, but this is an active area of research<sup>[52]</sup>. The presence of atrial fibrillation reduces the reliability of all these measurements.

#### NUCLEAR CARDIOLOGY

Nuclear angiography provides a simple assessment of global left and right ventricular systolic function<sup>[23]</sup> and myocardial perfusion. The early phase of left ventricular diastolic filling may be evaluated but further interpretation of left ventricular diastolic dysfunction by nuclear techniques is difficult.

Images may be obtained in patients in whom echocardiography is not possible. Myocardial perfusion imaging, at rest and during or after exercise, allows the presence and extent of ischaemia to be evaluated. The disadvantages of nuclear angiography are that it is less useful for the assessment of valve function, it does not measure ventricular hypertrophy and availability is more restricted than for echocardiography. Nuclear angiography is relatively expensive, reproducibility of ventricular volumes is only moderate and the patient is exposed to radiation. The latter limits the frequency with which studies can be performed.

#### EXERCISE TESTING

The Working Group on Exercise Physiology, Physiopathology and Electrocardiography has recently published guidelines on exercising testing<sup>[53]</sup>. Reduced exercise performance, when the limiting symptoms are breathlessness or fatigue, is characteristic of but not specific to heart failure. Therefore, in clinical practice, exercise testing is of limited value in the diagnosis of heart failure. A normal exercise test, in a patient not receiving treatment for heart failure, excludes heart failure as a diagnosis. Pharmacological treatment and exercise training may improve exercise performance, but rarely restore it to normal, in patients with a definite

diagnosis of heart failure. In patients with an established diagnosis, exercise performance is a useful way of assessing the severity of the condition and possibly in monitoring its progress.

Accurate assessment of functional capacity requires that the patient is familiar with what is required and that the observer has the required expertise. Several exercise protocols are in current use.

A marked fall in arterial oxygen saturation during exercise suggests the presence of pulmonary disease<sup>[54]</sup>, though small falls in arterial oxygen tension during exercise in patients with heart failure without evidence of pulmonary disease have been noted in some laboratories<sup>[55,56]</sup>.

Measurement of oxygen consumption during exercise is a research tool and may be a useful guide to prognosis and the need for transplantation. Clinically, measurement of oxygen consumption can help determine if exercise is limited by cardio-respiratory or other factors. The data, especially for women, relating the severity of heart failure to peak exercise oxygen consumption are inadequate.

Haemodynamic studies during exercise are considered further below.

### Invasive investigation in the diagnosis of heart failure

Invasive investigation is generally not required to establish the presence of chronic heart failure but may be important in elucidating the cause. Heart failure may exist in the presence of a normal cardiac output and filling pressures at rest, at least in treated patients<sup>[25,26]</sup>. Conversely, resting cardiac output may be depressed and filling pressure elevated in patients with cardiac dysfunction who do not have symptoms of heart failure. Reduced cardiac output and elevated pulmonary capillary wedge pressures during exercise may be due to reversible myocardial ischaemia and are not specific to heart failure, but a normal haemodynamic response to maximum exercise excludes heart failure as the cause of symptoms.

Exclusion of diastolic dysfunction by non-invasive means may be difficult and in some patients this may only be resolved by direct measurement of intra-cardiac pressures and volumes<sup>[57,58]</sup>. Invasive measurement of cardiac output and filling pressures may also be helpful in supporting or excluding heart failure in the presence of pulmonary or hepatic disease.

Coronary angiography is required to exclude coronary disease when a diagnosis of dilated cardiomyopathy is being considered. In patients with heart failure and evidence of myocardial ischaemia coronary angiography will be required if revascularization is considered a treatment option.

Endomyocardial biopsy is a useful research tool but of limited clinical utility<sup>[59,60]</sup>. In experienced hands patients with unexplained myocardial dysfunction should be considered for biopsy to exclude infiltrative or inflammatory disease.

*Table 3 Alternative conditions masquerading as or exacerbating heart failure. Suggested tests*

1. Lung disease	Chest X-ray Peak flow or pulmonary function tests Full blood count (for secondary polycythaemia)
2. Anaemia	Full blood count
3. Renal or hepatic disease	Biochemistry Urinalysis
4. Reversible myocardial ischaemia	Stress imaging

### Current developments

The following techniques may become valuable for the diagnosis of heart failure in the future.

#### NEURO-ENDOCRINE EVALUATION

Whereas there are no doubts about the importance of neuro-endocrine mechanisms in the pathogenesis of heart failure the role of neuro-endocrine factors in the diagnosis of heart failure is less clear. In large cohorts of patients, there is good evidence that noradrenaline, renin, angiotensin II and aldosterone are related to the severity and prognosis of heart failure<sup>[61]</sup> but in individual patients these predictors are inaccurate and difficult to interpret. Diuretics, vasodilator agents and ACE inhibitors alter plasma concentrations of neuro-endocrine substances in a complex fashion so that they are of limited diagnostic use. Plasma noradrenaline rises with age and healthy subjects over the age of 75 years may have plasma concentrations in the heart failure range<sup>[62]</sup>.

The best candidates for diagnostic evaluation of heart failure in individual patients are the natriuretic peptides (ANP). ANP and brain natriuretic peptide (BNP) increase early in the course of cardiac dysfunction prior to the onset of symptoms. N-terminal-ANP is an inactive by-product of the prohormone and may reflect the presence and severity of ventricular dysfunction more accurately than ANP<sup>[63,64]</sup>. Few data on BNP or beta-ANP for the diagnosis of heart failure are available<sup>[65]</sup>.

An elevated plasma concentration of ANP associated with appropriate symptoms, and in the absence of renal failure, strongly suggests the presence of heart failure. A normal plasma concentration of ANP in a patient receiving treatment may not refute a diagnosis of heart failure as normal levels may reflect the effects of treatment<sup>[66]</sup>. Plasma concentrations increase to a modest extent with age<sup>[64]</sup>.

#### STRESS IMAGING

Stress imaging, using echocardiography, thallium scintigraphy or positron emission tomography, may be

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**Figure 2 Assessments to be performed routinely to establish the presence and likely cause of heart failure.**

**Figure 3 Tests to be considered when diagnostic doubt persists or a reversible cause of heart failure is suspected.**

useful for detecting reversible ischaemia as a cause of breathlessness and in assessing the viability of non-functioning (hibernating) myocardium<sup>[67–70]</sup>.

Physiological stress may be applied in the form of isometric or dynamic exercise or pharmacological stress in the form of an inotropic (dobutamine), vasoconstrictor (angiotensin II or adrenergic agent) or vasodilator (dipyridamole or adenosine) agent. Echocardiography or nuclear techniques may be used for imaging purposes.

#### AMBULATORY ECG

Heart rate variability is a marker of autonomic balance and is reduced in heart failure. The diagnostic

and prognostic utility of this observation is currently being investigated<sup>[71–74]</sup>. Conventionally interpreted Holter monitoring is of no value in establishing the presence of heart failure, though frequent ventricular and supra-ventricular tachyarrhythmias and bradyarrhythmias are common in patients with heart failure. Holter monitoring may identify paroxysmal arrhythmias causing or exacerbating symptoms of heart failure.

#### ALTERNATIVE IMAGING TECHNIQUES

Magnetic resonance imaging (MRI)<sup>[39]</sup> and computed axial tomography (CAT) scans<sup>[28]</sup> are not widely available and are rarely required if a satisfactory echocardiogram can be obtained. MRI may be useful in identifying

**Figure 4** Tests with potential value for and currently being evaluated for the diagnosis of heart failure.

pericardial thickening and for defining the extent of cardiac tumours. MRI and CAT scans are probably the most accurate method for measuring cardiac volumes and have a valuable role in research.

#### ALTERNATIVE METHODS FOR NON-INVASIVE HAEMODYNAMIC EVALUATION

Plethysmography and Doppler techniques may be used to assess peripheral blood flow but are useful only for physiological studies. Assessment of cardiac function in heart failure by bio-impedance techniques has proved unsatisfactory.

## Section 2: Requirements for the diagnosis of heart failure in clinical practice

To satisfy the definition of heart failure, symptoms of heart failure and objective evidence of cardiac dysfunction must be present (Table 1). The assessment of cardiac function by clinical criteria alone is unsatisfactory. Cardiac dysfunction should be assessed objectively. The echocardiogram is the single most effective tool in widespread clinical use. A diagnosis of heart failure also requires the presence of symptoms and/or signs suggestive of the diagnosis and cannot be made by any single laboratory test. Other conditions may mimic or exacerbate the symptoms and signs of heart failure and need to be excluded (Table 3).

Tests were grouped into three categories. Evaluations in Category 1 (Fig. 2) should be performed routinely in patients with suspected heart failure in order to establish the diagnosis.

Tests in Category 2 (Fig. 3) should be performed or re-evaluated in cases where diagnostic doubt persists or clinical features suggest a reversible cause for heart failure. Coronary artery disease is a common, and probably underdiagnosed, cause of heart failure. If there is reason to believe that the patient will benefit from revascularization then an angiogram should be done.

The conduct and interpretation of tests in Category 3 (Fig. 4) are considered to be evolving and their final role for the diagnosis of the presence and/or cause of heart failure remains to be fully defined.

Fig. 5 represents a simplified plan for the evaluation of a patient presenting with symptoms suggestive of heart failure.

## Conclusion

Diagnosing the presence of heart failure reliably remains the first and most important step in the management of heart failure. The cause of heart failure is frequently elucidated during the investigations required to establish its presence. An adequate diagnosis must not only establish the existence and main underlying cause of heart failure but also subsidiary diagnoses that may exacerbate the condition or complicate its management. An inadequate diagnosis exposes the patient to inappropriate treatment that may be life threatening; an accurate diagnosis is usually not expensive.

Future position papers will address other components of the optimal management of heart failure (Table 4).

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Figure 5 A simplified plan for the diagnosis of heart failure.

Table 4 Management outline

1. Establish that the patient has heart failure
2. Ascertain presenting features:  
pulmonary oedema, exertional breathlessness and/or fatigue, peripheral oedema
3. Determine aetiology of heart failure
4. Identify concomitant disease relevant to heart failure and its management
5. Assess severity of symptoms
6. Predict prognosis
7. Anticipate complications
8. Counsel patients and relatives
9. Choose appropriate management
10. Monitor progress and manage accordingly

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## Appendix

The Members of the Drafting Committee were: Cleland J. G. F., Erdmann E, Ferrari R, Hess O. M., Poole-Wilson P. A., Remme W. J., Riegger G, Soler-Soler J, Swedberg K. B., Tavazzi L, Waagstein F. The Current Members of The Nucleus of the Working

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