HEART FAILURE

The diagnosis of heart failure

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Heart failure is a difficult disease to define. It is easy to recognise heart failure in its moderate/severe version where the patient has pronounced symptoms and signs accompanied by echocardiographic evidence of left ventricular (LV) systolic dysfunction.[1] However, the problem of defining heart failure arises in its milder forms where patients may complain of dyspnoea but do not have echocardiographic evidence of LV systolic dysfunction. The complexities of what represents heart failure are illustrated in fig 1 but space precludes a detailed discussion of the definition of heart failure.

To overcome the various difficulties in defining heart failure, the European Society of Cardiology (ESC) has developed guidelines for the diagnosis of heart failure.[2] However, like all statements which are meant to define the undefinable, there is a certain deliberate vagueness about them. For example, they do not specify what they mean by cardiac dysfunction. Does an elderly lady whose echocardiogram meets criteria for "diastolic dysfunction" and who has swollen ankles have heart failure, even if she has no breathlessness or fatigue? Despite this caveat, the ESC guidelines have clarified the situation, even if one can still point to isolated patients who remain ambiguous with the ESC definition.

At a more pragmatic level, the clinician who is faced with a patient with suspected heart failure should try to answer two major questions:

1. Are the patient's symptoms cardiac in origin?

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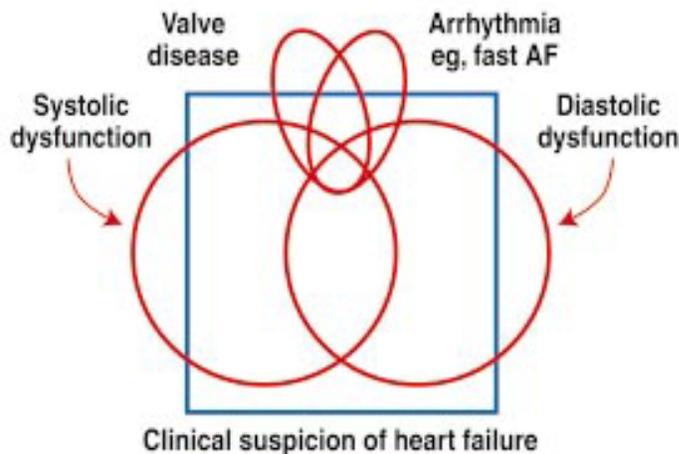

Figure 1. The various subcategories of heart failure.

> **European Society of Cardiology guidelines for the diagnosis of heart failure**
>
> - Essential features
>   1. Symptoms of heart failure (for example, breathlessness, fatigue, ankle swelling)
>   and
>   2. Objective evidence of cardiac dysfunction (at rest)
>
> - Non-essential features
>   In cases where the diagnosis is in doubt, there is a response to treatment directed towards heart failure

2. If so, what kind of cardiac disease is producing these symptoms?

In order to answer these questions, the clinician goes through the standard process of assessing the patient's symptoms, then the patient's signs, and finally arranging appropriate investigations. Obtaining the answer to the above questions is much more complex than it is with some other disease. For example, absolute levels of blood glucose make or break the diagnosis of diabetes mellitus. Similarly, absolute levels of blood pressure decide whether a patient has hypertension or not (even if the cutoff values for blood pressure and blood glucose do change repeatedly). Heart failure is much more difficult because it is not definable by an absolute level of any one parameter. Even if one could define a set of echocardiographic criteria to make the diagnosis, experts would never agree on the cutoff values and, even if they did, echocardiography is much more subject to interobserver bias than is a blood glucose. In order to set the scene, it is also worth saying how poor clinicians are at diagnosing mild heart failure using purely symptoms and signs. This information comes from the many open access echo services which have been set up throughout the country. In these, general practitioners are asked to send up all patients whom they suspect may have heart failure. To the surprise of many, it was found that only 25% of those sent up had LV systolic dysfunction on their echocardiogram—that is, 75% had normal LV systolic function.[3 4] This is not to criticise doctor's skills. Rather this says how non-specific are the symptoms and signs which classically lead us to suspect heart failure (table 1).[5]

Clinical history

The most classic symptom of heart failure is exertional dyspnoea. Unfortunately, exertional dyspnoea is also a common symptom in the general population. Indeed, with sufficient exercise, normal individuals will experience dyspnoea. Furthermore, in the general population, an individual patients' level of "fitness" will determine at what stage during exercise he or she experiences dyspnoea. Another problem is that there are a whole host of other non-cardiac diseases which also produce dyspnoea. Obvious examples are respiratory dis-

Table 1 Predictive values of clinical features[5]

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Past history of myocardial infarction	59	86	44	92
Ingesting diuretic	73	41	19	89
Dyspnoea on exertion	100	17	18	100
Orthopnoea	22	74	14	83
Paroxysmal nocturnal dyspnoea	39	80	27	87
Oedema in history	49	47	15	83
Jugular venous pressure distension	17	98	64	86
Crackles	29	77	19	85
Gallop rhythm	24	99	77	87
Oedema on examination	20	86	21	85

PPV, positive predictive value; NPV, negative predictive value.

eases, anaemia, and simple obesity. All these factors make exertional dyspnoea a very non-specific symptom.

There are more extreme versions of dyspnoea which are more useful diagnostically. Orthopnoea is certainly more specific since it does not occur in normal individuals and probably not in respiratory disease. However, heart failure patients have to be quite severe before orthopnoea occurs, and diuretics are usually instituted long before orthopnoea becomes a problem. Indeed, if orthopnoea occurs, it is likely that premonitory symptoms have been ignored (either by the patient or the doctor). Thus, orthopnoea is a specific symptom but it has low sensitivity. Another extreme version of dyspnoea is paroxysmal nocturnal dyspnoea (PND). This results from nocturnal fluid redistribution which causes an increased LV filling pressure. Like orthopnoea, PND is a specific but insensitive symptom because again it signifies fairly severe heart failure which should have been noted and treated beforehand. Another small practical point is that in some patients, a very careful history is required to differentiate the PND of heart failure from the nocturnal dyspnoea and wheezing of asthma.

Fatigue and lethargy are common problems in heart failure, but they are probably even harder to define and assess than dyspnoea. Fatigue is an intermittent symptom in normal individuals as well as being a recognised symptom of nearly every disease that exists. Fatigue is therefore so unspecific as a symptom as to render it virtually worthless in guiding you to a diagnosis of heart failure or not.

A history of ankle oedema is another common presenting feature, but there are many alternative causes for it. Cor pulmonale (right heart failure caused by lung disease) also causes ankle oedema although the lung disease is usually so extreme as to be clinically obvious. Ankle oedema is much more helpful in diagnosing heart failure in men than in women. Elderly ladies commonly have ankle oedema which is not caused by heart failure: its precise cause is unknown although venous insufficiency accompanied by pelvic obstruction to venous blood flow are commonly blamed. In fact, it is elderly ladies with swollen ankles who most commonly cause the "false positives" which are seen in open access echo referrals.

Perhaps the best clue from the history is not a symptom which the patient complains of, but a past history of a previous myocardial infarction. Less useful but also worth noting is a past history of hypertension. It should be noted, however, that many elderly patients experience clinically "silent" myocardial infarctions. Other useful features in the history are excessive alcohol intake, a past history of rheumatic fever making valve disease possible, and the use of a drug such as a non-steroidal anti-inflammatory (NSAID) which might precipitate heart failure.

Physical signs

As with the symptoms, the physical signs fall into two categories. Firstly, there are physical signs which occur in so many other diseases that they are non-specific and hence of low predictive value. These are tachycardia, pulmonary crepitations, and peripheral oedema. On the other hand, there are physical signs which are relatively specific to heart failure but they are also insensitive because they only occur once heart failure has become quite severe. These latter physical signs are elevation of the jugular venous pressure, gallop rhythm, and displacement of the apex beat. There is a further problem with these latter physical signs in that the ability of doctors to detect these more specific signs is variable.

Thus, it can be seen that few symptoms or signs are valuable on their own. However, clinical medicine is not about using single parameters to make diagnoses. As a clinician, one assesses the overall probability of a diagnosis based on the whole history and examination. When one does that, it becomes clear that heart failure is not as hard to diagnose correctly as the above discussion would imply. For example, a man with a past history of a myocardial infarction who now develops ankle oedema is very likely to have heart failure. On the other hand, heart failure is unlikely in a patient who complains of fatigue with no relevant past history and no other symptoms/signs of heart failure.

Investigations

The main purpose of investigations is to confirm or refute the diagnosis of heart failure *and* to define the precise underlying cardiac cause. The latter is especially important as treatment is based on the underlying cause and not simply on the existence of heart failure. For example, heart failure can be caused by valve disease, where the correct treatment may be surgery, or by LV systolic dysfunction, where the correct treatment will be diuretics, angiotensin converting enzyme (ACE) inhibitors, β blockers, and spironolactone.

Blood analysis

It goes without saying that all patients with suspected heart failure should have blood sent for haematological and biochemical analysis (including thyroid function and cholesterol). These

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tests will not only exclude anaemia and hypothyroidism but will also detect other entities which might need treatment, such as cholesterol.

Chest *x* ray

The chest *x* ray is routinely performed and can produce useful information. Cardiac enlargement (cardiothoracic ratio > 50%) implies cardiomegaly and if this is present it is a good guide to heart failure. However, many heart failure patients will not exhibit cardiomegaly so that it tends to be a specific but insensitive test which identifies severe heart failure only. The other helpful findings at chest *x* ray are pulmonary oedema (bat's wing appearance), upper lobe diversion, fluid in the horizontal fissures, and Kerley B lines in the costophrenic angles. In extreme cases, pleural effusions may be present although clearly there are alternative explanations for pleural effusions such as bronchial carcinoma, pneumonia or pulmonary emboli. Clearly the chest *x* ray can also reveal other clues as to non-cardiac disease which might cause the dyspnoea. A lung tumour might be obvious, and emphysema might also be present. Nevertheless, the chest *x* ray should be seen as a whole—for example, the finding of cardiomegaly plus bilateral pleural effusions with no other parenchymal lung disease makes heart failure extremely likely (although it should still be confirmed by echocardiography).

Electrocardiogram

The 12 lead ECG should be routinely performed. The situation with the ECG is very different for heart failure than it is for angina—that is, a completely normal resting ECG does not by any means exclude the diagnosis of angina whereas it does virtually exclude the presence of heart failure. Put another way, LV systolic dysfunction is rare in the presence of a completely normal 12 lead ECG. In practice, what this means is that if a patient has dyspnoea and a completely normal ECG, then the doctor should first consider alternative diagnoses for the patient's dyspnoea, rather than heart failure. On the other hand, an abnormal resting ECG will often occur in the absence of LV systolic dysfunction.[6] Overall therefore, an abnormal resting ECG is sensitive (94%) with excellent negative predictive value (98%) but it is much less specific (61%) and has poor positive predictive value (35%). Although most studies support this finding, in general, there are contrary data including one paper where 27% of cases were missed with this approach.[7] Therefore, if in a certain area the availability of echocardiography is limited, then a possible strategy is to limit echocardiography to those with abnormal ECGs as well as those with cardiac murmurs.

Echocardiography

The real gold standard investigation in suspected heart failure is echocardiography. It is clearly the most valuable single investigation. A qualitative assessment can be made to decide whether LV systolic function is normal or impaired (hypokinetic). This may well be all

Ability of a normal ECG to exclude LV systolic dysfunction?[6]	
● Sensitivity	94%
● Specificity	61%
● Positive predictive value	35%
● Negative predictive value	98%

that is necessary in routine clinical use. However, it can be quantified by measuring various parameters—for example, fractional shortening, LV ejection fraction (LVEF) or wall motion index. Fractional shortening is the quickest and, for clinical purposes, is usually sufficient. When it comes to research, more accurate quantification is preferable which is why the LVEF or the wall motion index are more usually used in research studies. Another useful measure is whether the left ventricle is dilated or not. Again, this can be described qualitatively or quantitatively by LV diameters or LV volume indices. LV dilatation and LV systolic dysfunction usually accompany each other, but occasionally the left ventricle is dilated even although its systolic function is normal. LV dilatation implies impending LV systolic dysfunction and should probably be treated as such.

In true systolic dysfunction, secondary mitral incompetence is often seen—that is, the mitral ring becomes stretched owing to LV dilatation. It is crucial to distinguish this from primary mitral valve disease, where surgery is the treatment of choice. In secondary mitral incompetence caused by LV systolic dysfunction, surgery used not to be indicated since optimised medical treatment reduces the LV dilatation and can hence reduce the secondary mitral incompetence. However, this situation may be changing as mitral valve replacement is now thought to be of benefit in selected cases of secondary mitral incompetence.

Clearly, echocardiography will also be able to assess valve structure and function and identify patients with aortic stenosis, etc, who would benefit from surgery.

Echocardiography will also be able to assess diastolic function although this is a contentious issue. The problem is that the left ventricle (like the aorta) becomes stiffer as it ages and it is difficult to define when this stiff left ventricle constitutes diastolic dysfunction. No echocardiographic criteria are fully accepted as measures of diastolic dysfunction. It should be recognised, however, that there is one extreme version of diastolic dysfunction which can cause severe pulmonary oedema. This is when the left ventricle is so stiff that left atrial pressure increases and leads to fast atrial fibrillation with profound dyspnoea. The short term treatment here is to reverse the abnormal rhythm, but longer term strategies to prevent LV stiffness which leads to atrial fibrillation should be explored. As yet, no established treatment exists for diastolic dysfunction per se.

Value of BNP as a screening tool in two important situations

- Ability of a high BNP concentration to identify heart failure in symptomatic patients in primary care[8]

  Sensitivity   97%
  Specificity   84%
  Positive predictive value   70%
  Negative predictive value   98%

- Ability of BNP to identify LV systolic dysfunction in a cross section of the community[9]

  Sensitivity   76%
  Specificity   87%
  Positive predictive value   16%
  Negative predictive value   98%

In a small proportion of patients, echocardiographic windows are poor and in that situation radionuclide ventriculography is often used instead to assess LV systolic function. Although it does so very reliably, unfortunately it does not give useful structural information about valves or LV mass.

Open access echocardiography

Because of the key role of echocardiography in assessing such patients, the concept of direct access or open access echocardiography has arisen. Francis and colleagues showed the true value of such a service.[4] Among the referred patients who were already being treated for heart failure, only 36% of men and 18% of women had definitely impaired LV systolic function. In fact, the echo result led to a change in treatment for 70% of those patients who were already taking diuretics for suspected heart failure—this change was often the cessation of unnecessary treatment. However, open access echocardiography is not without its problems. These problems relate to guidelines on whom to refer, interpretation of the results, and on the maintenance of echocardiographic standards. General practitioners should ideally receive guidelines on whom to refer. In addition, echocardiography can be difficult to interpret in some cases without a fuller assessment of the individual patient. The main difficulty here is that many symptomatic patients will not have systolic dysfunction but may have "diastolic dysfunction". Whether such diastolic dysfunction seen on an echocardiogram is the cause of the patient's symptoms or not is often a difficult judgement even for an experienced cardiologist. Hence the lack of systolic dysfunction in an open access echo service could be falsely reassuring to the general practitioner. In fact it may be better referring this kind of difficult patient to a hospital consultant who can then assess the echo result as part of the whole clinical picture.

Despite this important caveat, open access echo services continue to flourish and expand. Dialogue between referring general practitioners and consultants should help to iron out dif-

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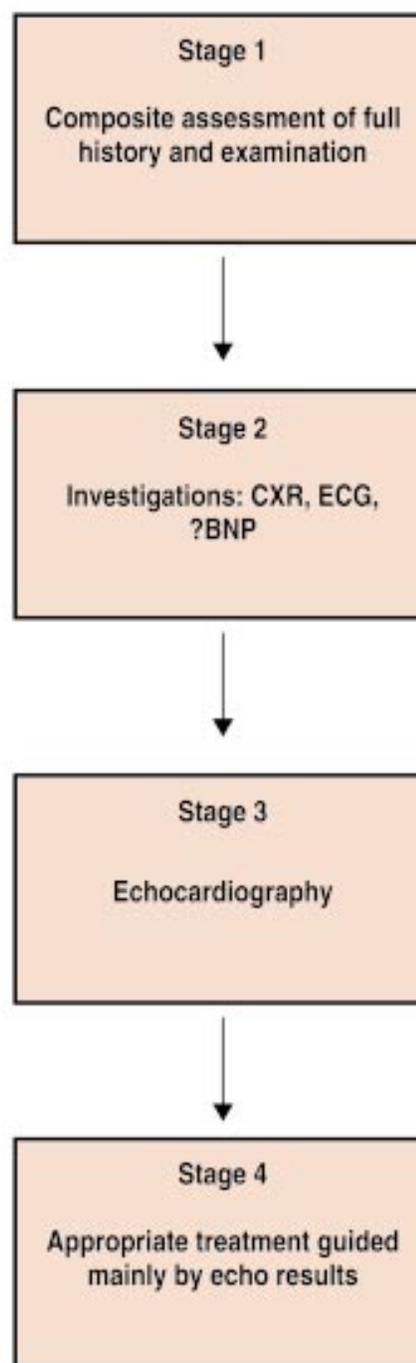
Figure 2. Diagnosing heart failure in practice. CXR, chest x ray; BNP, B-type natriuretic peptide.

ficulties and to identify two separate groups: those patients who only need a factual echo report, and those who require further interpretation of the echo with knowledge of the whole clinical picture.

Natriuretic peptides for prescreening patients for echocardiography

General practitioners would still like more guidance as to whom to refer for echocardiography, especially since they are aware that only

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25% of those referred for echo turn out to have LV systolic dysfunction. They are aware that a completely normal ECG can be used to select patients for echocardiography, although this does miss some cases of LV systolic dysfunction. In addition, general practitioners are unsure about their ability to differentiate between ECGs with minor abnormalities and totally normal ECGs. Therefore, there arose the idea that plasma concentrations of natriuretic peptides could help identify who to refer for echocardiography. The best measures are B-type natriuretic peptide (BNP) or N-terminal BNP (N-BNP). Although many papers have addressed this question, most of them were based on hospital patients and suffer from the weakness that the blood was taken from patients already on diuretics, which distort the relation between BNP and LV function since they reduce the BNP concentration but do not alter the echo. In one study set in general practice, BNP was 97% sensitive and 84% specific at identifying heart failure.[8] In another study set in the community, BNP was 76% sensitive and 87% specific in identifying LV systolic dysfunction.[9]

Despite most of the data being very positive, the use of BNP or N-BNP has not yet entered routine clinical practice. The reasons for this are that there are virtually no studies yet of general practitioners using BNP/N-BNP in routine practice and before diuretic treatment or referral; in addition, the cost-effectiveness of such a strategy still needs to be established. Future studies should clarify these important issues which may lead to general practitioners using BNP/N-BNP to preselect symptomatic patients for echocardiography.

Conclusions

The diagnosis of heart failure has three stages (fig 2). Firstly, the clinical history and examination have many pointers, none of which should be used in isolation, but when put together lead to a relatively accurate assessment of the likelihood of the patient having heart failure. The next stages are the preliminary investigations of chest *x* ray, ECG, and in the future perhaps BNP.[10] This second stage is mainly to preselect patients for the next stage which is the gold standard investigation of echocardiography—that is, if the second stage excludes heart failure then echocardiography would then become unnecessary and alternatively diagnosis should be considered. The third stage is the definitive investigation of echocardiography which would not only confirm heart failure definitely but even more importantly classify the cause of the heart failure into systolic dysfunction, diastolic dysfunction or valve disease. This differentiation is essential since the treatment is very different for each subcategory of heart failure.

Summary

- Symptoms which suggest heart failure tend to be either sensitive (for example, dyspnoea) or specific (PND, orthopnoea), but no symptom is both.

- Physical signs which suggest heart failure tend to be specific (raised jugular venous pressure, gallop rhythm), but none of them are sensitive.

- Echocardiography is essential to diagnose heart failure in suspected cases. Open access echo services seem popular.

- The ECG can be used as a prescreen to select patients for echocardiography, but some cases of LV systolic dysfunction (2–27% in different reports) are missed by this approach.

- Measurement of natriuretic peptides (BNP and N-BNP) might become an alternative way to prescreen suspected patients for echocardiography, but more work is required to establish feasibility and accuracy.

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