

Symptoms of heart failure

GENERAL

HF has many causes, as described in Chapter 1. A high index of suspicion is necessary in patients with predisposing conditions, such as a previous history of MI, diabetes, or hypertension. It is important not to discount the diagnosis of HF in young patients, as dilated cardiomyopathy can occur at any age. If the diagnosis is not considered, such patients may be readily misdiagnosed as having asthma, for example.

Lethargy and general malaise

For many patients with chronic HF, the most debilitating symptom is tiredness. Patients may have difficulty sleeping due to other symptoms of HF, but lethargy is itself a feature of HF, and has a profound effect on patients' exercise capacity and quality of life, over and above that caused by breathlessness and other symptoms. Patients with HF also typically report increased muscle fatigue on exertion, contributing to reduced exercise capacity. Skeletal muscle metabolism has been shown to be abnormal in HF (Table 10). Some patients with HF may present only with feelings of general malaise, and careful questioning may be necessary to detect other symptoms of HF.

Loss of appetite

Patients with heart failure may complain of loss of appetite, which may be due to bowel oedema. Weight loss due to such anorexia may be masked by fluid accumulation, and become apparent only when diuretic therapy is instituted. Poor nutrition due to anorexia combined with reduced absorption from the oedematous bowel eventually results in cardiac cachexia, and the resulting reduction in muscle bulk compounds lethargy and breathlessness. Patients may also complain of right upper quadrant discomfort or pain, caused by liver congestion and capsular stretch.

Table 10 Muscle abnormalities in heart failure

- ✧ Wasting
- ✧ Impaired resting blood flow
- ✧ Limited capacity to enhance blood flow on exertion
- ✧ Increased fatigue
- ✧ Abnormal metabolism
- ✧ Early anaerobic metabolism
- ✧ Early intracellular acidosis

(From Harlan WR, et al. *Ann. Intern. Med.* 1977;86:133–138.)

BREATHLESSNESS

Shortness of breath is the most well known symptom of HF, but is a common symptom of many conditions, and therefore is not very specific for HF. Classically, patients complain of orthopnoea (breathlessness when lying flat), which may be quantified by asking how many pillows the patient uses in bed. Paroxysmal nocturnal dyspnoea (PND; episodic breathlessness waking the patient from sleep) is another classic symptom of HF and, again, the frequency of such episodes can be useful in quantifying severity. Not all patients complain of these symptoms, however, and their absence does not rule out the possibility of a diagnosis of HF.

Acute HF most often presents with extreme breathlessness of very sudden onset, such that the patient is often too breathless to talk. Diagnosis in this circumstance relies on clinical examination and investigation, although witnesses may be able to provide some clues. HF should always be considered in the acutely breathless patient. Where there is diagnostic doubt, a trial of a diuretic may result in rapid improvement in the patient with HF.

Patients with more insidious onset of HF may describe gradually worsening shortness of breath, and decreasing exercise tolerance over weeks or months. Cough may be an early symptom of HF, and some patients may complain of wheeze, caused by oedema of the bronchial tree, both of which may initially suggest the diagnosis of asthma.

PERIPHERAL OEDEMA

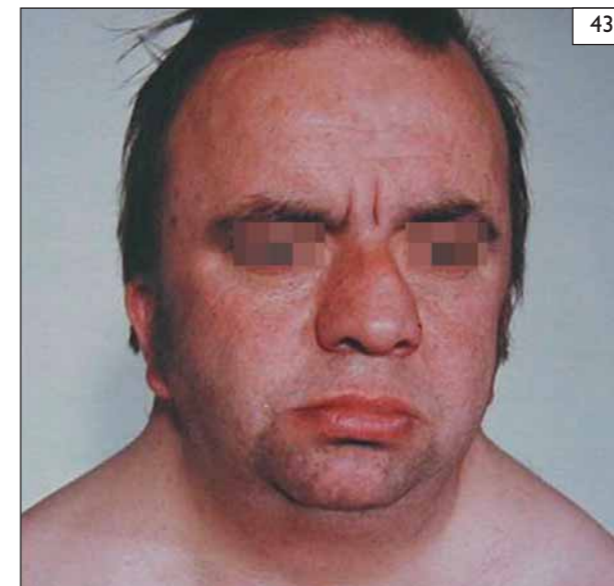
In some patients, the first symptom of HF may be development of peripheral oedema. The patient's usual posture determines the location of oedema: ambulant patients typically initially develop oedema of the ankles which resolves overnight; patients who are immobile may develop oedema predominantly over the sacrum. Oedema has many causes, particularly gravitational in immobile patients. Other causes of oedema may be mistaken for, or coexist with, HF (Table 11). Oedema is classically a sign of right-sided HF, for example in the patient with chronic obstructive pulmonary disease (cor pulmonale), but even in patients with predominant left-sided HF, oedema may be the main presenting feature with breathlessness less apparent.

Signs of heart failure

In some patients, clinical examination may make the diagnosis clear, but some patients with early HF may have few or no apparent clinical signs. In addition, the signs associated with HF have other

Table 11 Causes of oedema

- ✧ Heart failure
- ✧ Gravitational oedema
- ✧ Hypoproteinaemia
- ✧ Liver disease
- ✧ Nephrotic syndrome
- ✧ Lymphoedema
- ✧ Medications – calcium channel blockers, e.g. amlodipine



43 Noonan's syndrome – short webbed neck, low-set ears, high nasal bridge; may be subtle and present in adulthood.

Table 12 Diagnostic sensitivity and specificity of clinical features of heart failure

Clinical features	Sensitivity (%)	Specificity (%)
Breathlessness	66	52
Orthopnoea	21	81
PND	33	76
Oedema (history)	23	80
Tachycardia	7	99
Pulmonary crackles	13	91
Oedema (examination)	10	93
Third heart sound	31	95
Raised JVP	10	97

PND: paroxysmal nocturnal dyspnoea; JVP: jugular venous pulsation

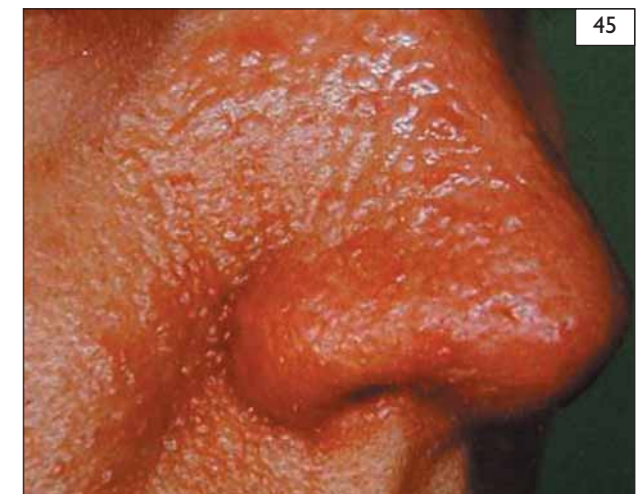


44 Atrial septal defect associated with Noonan's syndrome.

causes which must be considered. Table 12 presents the diagnostic sensitivity and specificity of clinical features of heart failure.

GENERAL EXAMINATION

Careful general examination may reveal useful information. Although HF is most commonly caused by ischaemic heart disease or hypertension, rarer causes must not be forgotten. Characteristic facies may be present. For example, Noonan's syndrome, which is associated with congenital heart disease (43, 44), or tuberose sclerosis (45) which may be associated with cardiac rhabdomyoma, which can be present as HF in



45 Tuberose sclerosis – facial angiofibroma.

childhood. Systemic disorders such as gout (46, 47), dyslipidaemia (48), scleroderma (49, 50), connective tissue disorders (e.g. Ehlers–Danlos syndrome [51, 52] or Marfan syndrome [53]), both associated with mitral

valve prolapse), or neurofibromatosis (Von Recklinghausen syndrome [54–57, 76]) may provide clues. Hypothyroidism is associated with HF and the typical ‘myxoedema facies’ should not be missed (58–60).



46 Chronic tophaceous gout.



47 Chronic tophaceous gout.



48 Cutaneous xanthomata in a patient with dyslipidaemia.



49 Scleroderma – tight, shiny skin over fingers, loss of finger pulp.



50 Skin in scleroderma showing telangiectasia.



51, 52 Ehlers–Danlos syndrome – demonstration of skin laxity.



53 High arched palate in a patient with Marfan syndrome.



54–56 Von Recklinghausen syndrome – café-au-lait spots.



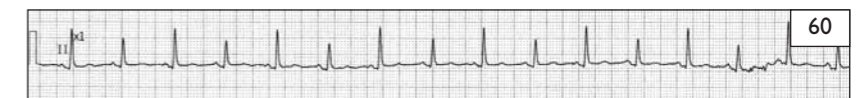
57 Von Recklinghausen's disease showing multiple neurofibromata.



58 Myxoedema facies.

59 2D echocardiogram showing pericardial effusion due to hypothyroidism (arrow).

60 Electrical alternans in a patient with a large pericardial effusion. Alternating large and small QRS complexes caused by the heart swinging within the pericardial fluid.



Carcinoid syndrome is associated with right-sided valvular abnormalities (61–63).

Patients with chronic HF are often anaemic (64). Anaemia may be due to poor nutrition, or alternatively due to anaemia of chronic disease. Anaemia may be sufficiently severe as to be apparent clinically. Recognition of anaemia is important, as correction may lead to an improvement in functional status.

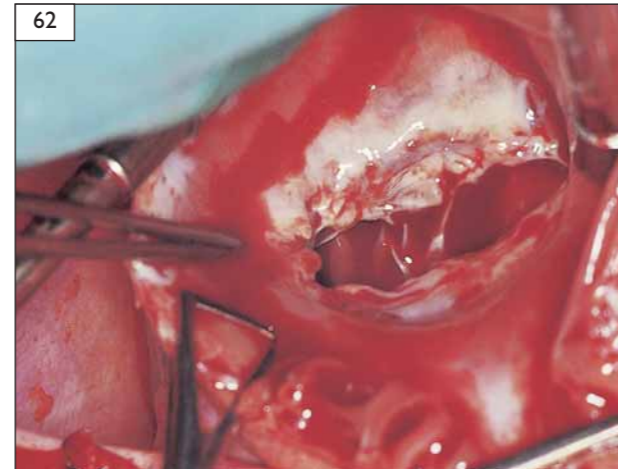
Cachexia may occur in chronic HF (65, 66). Weight loss may be masked by accumulation of oedema, but loss of muscle bulk in the face and

upper body may be clearly visible. Cachexia is a poor prognostic sign, and indicates severe HF.

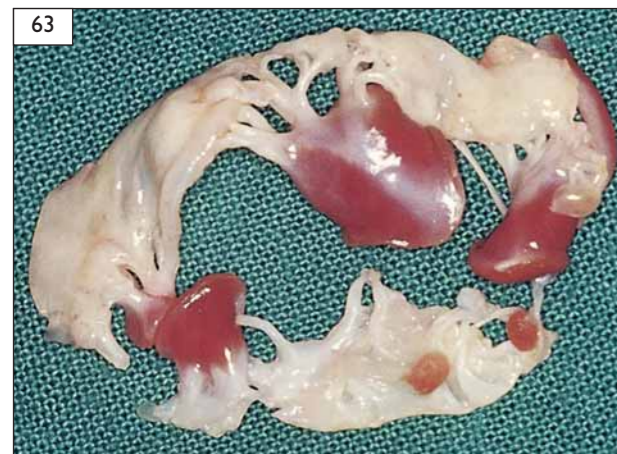
Oedema may be the most obvious clinical sign of heart failure. It may range from subtle pitting to pressure over the tibia (67, 68), to gross oedema extending up to the abdomen and genitalia (69). Patients treated with diuretics may become dehydrated even in the presence of persistent oedema. Reduction of skin turgor and reduced urine output may accompany dehydration. During therapy, fluid status can be monitored by weighing.



61 Facial telangiectasia in carcinoid syndrome.



62 Fibrotic tricuspid valve in carcinoid syndrome.



63 Excised carcinoid tricuspid valve.



64 Patient with heart failure and clinical anaemia.



65 Patient with advanced heart failure: note pitting oedema, ascites, and cachexia.

66 Cachexia – note severe loss of upper arm musculature.



67, 68 Pitting peripheral oedema.



69 All body oedema.

Cardiovascular drugs, for example long-term therapy with amiodarone, may induce certain clinical features (70). Certain cardiac conditions may provide features on clinical examination, for example infective endocarditis (71–74).



70 Amiodarone facial discoloration (slate-grey appearance).

CARDIOVASCULAR EXAMINATION

Cardiovascular examination is obviously important in the detection of HF, and may provide additional information concerning the underlying cause. Full cardiovascular examination may be difficult in the



71 Digital gangrene in infective endocarditis.



72, 73 Vasculitic spots (Janeway lesion) in infective endocarditis.



74 Splinter haemorrhages in infective endocarditis.

patient with acute breathlessness; in this situation a limited initial examination may be followed by detailed examination once therapy has commenced and symptoms have started to improve.

Pulse

Examination of the pulse often reveals tachycardia (compensating for reduced stroke volume), even in patients who are asymptomatic at rest. In acute HF, the radial pulse may be weak, and tachycardia may be extreme. Atrial fibrillation (AF) is common in HF, and the ventricular rate may be very rapid. Recognition and early treatment of AF may rapidly produce improvement in clinical status.

Jugular venous pulsation

The jugular venous pulsation (JVP) is regarded as a barometer of right atrial pressure, and may be raised in HF (75). It should be assessed with the patient lying at 45 degrees. It is useful to differentiate HF clinically from other causes of peripheral oedema (such as hypoproteinaemia, where the JVP is likely to be low), but it has low sensitivity for HF. It may not be visible in the obese patient, or the patient with acute HF and severe breathlessness. The absence of a raised JVP should not rule out the possibility of heart failure.

Palpation

Valve lesions may be associated with palpable thrills. A right ventricular heave may be present in patients with pulmonary hypertension and right-sided HF (for example, cor pulmonale). Palpation of the cardiac apex may show lateral displacement from the normal position (5th intercostal space, midclavicular line). The character of the apex beat may also be abnormal. A sustained, heaving apex may occur. All

75 Raised jugular venous pulsation.



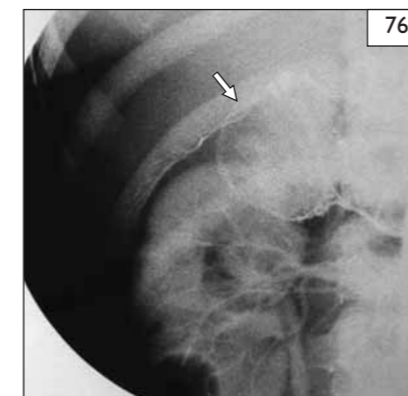
of these signs take time to develop, and may well be absent in patients with HF of acute onset.

Auscultation

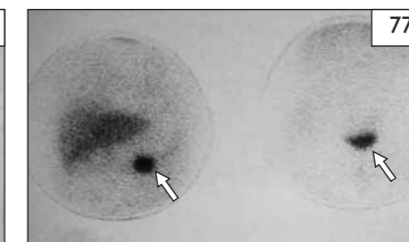
Auscultation may be difficult in the breathless patient. Tachycardia may also mask signs. There may be a third or fourth heart sound (due to rapid ventricular filling or atrial contraction against a stiff left ventricle, respectively), or murmurs of underlying valve disease (or functional mitral regurgitation). The presence of a third heart sound is specific for HF, but has a high interobserver variability.

Blood pressure

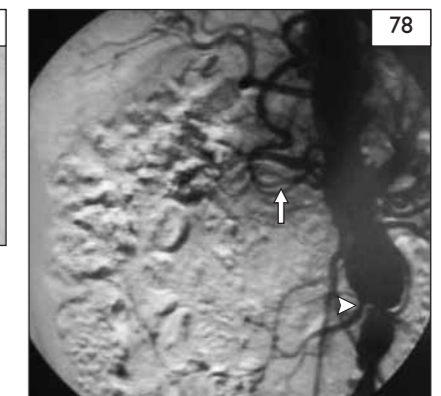
Blood pressure measurement should be part of cardiovascular examination. High blood pressure should be detected, treated, and controlled. Rarely, unusual causes of hypertension, such as pheochromocytoma (76, 77) and renal artery stenosis (78) should be considered as causes of HF.



76 Renal angiogram showing phaeochromocytoma (arrow) (here associated with Von Recklinghausen syndrome).



77 MIBG (meta-iodobenzyl guanidine) scan of phaeochromocytoma (arrows).



78 Renal artery stenosis (arrow) and distal aortic stenosis (arrowhead).