

Heart Failure in General Practice

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Quality and Safety in Practice Committee

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The guide does not however override the individual responsibility of healthcare professionals to make decisions appropriate to the circumstances of individual patients in consultation with the patient and/or guardian or carer.

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In this document you will see that evidence and recommendations are graded according to levels of evidence (Level 1 – 5) and grades of recommendations (Grades A-C) respectively. This grading system is an adaptation of the revised Oxford Centre 2011 Levels of Evidence.

Levels of Evidence

Level 1: Evidence obtained from systematic review of randomised trials

Level 2: Evidence obtained from at least one randomised trial

Level 3: Evidence obtained from at least one non-randomised controlled cohort/follow-up study

Level 4: Evidence obtained from at least one case-series, case-control or historically controlled study

Level 5: Evidence obtained from mechanism-based reasoning

* Level may be graded down on the basis of study quality, imprecision, indirectness (study PICO does not match questions PICO), because of inconsistency between studies, or because the absolute effect size is very small; Level may be graded up if there is a large or very large effect size. Where possible, systematic review evidence is presented.

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Introduction

Aim of this document

The aim of this document is to give an update of the present knowledge in heart failure for general practitioners. The main section consists of a summary of the European Society of Cardiology Guidelines on Acute and Chronic Heart Failure¹. A number of appendices give practical advice on managing heart failure in general practice. This guide is also accompanied by an infographic which gives a quick summary of the main features for general practitioners.

Definition

Heart failure is defined as a clinical syndrome characterized by typical symptoms (e.g. breathlessness, ankle swelling and fatigue) that may be accompanied by signs (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema) caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intra-cardiac pressures at rest or during stress.

Terminology

Categories of Heart Failure

The main terminology used to describe heart failure is historically based on ejection fraction. The ejection fraction is the percentage of blood pumped out of the heart with each beat. This has been used to categorise heart failure into three main categories in the most recent guidelines. These guide treatments also.

- Heart failure with reduced ejection fraction (HF-rEF) are those patients with symptoms and/or signs of heart failure and an initial ejection fraction less than 40%
- Heart failure with mid-range ejection fraction (HF-mrEF) are those with symptoms and/or signs of heart failure and an initial ejection fraction of 40-49%. In addition, these patient must have elevated levels of natriuretic peptides and evidence of structural heart disease (e.g. left ventricular hypertrophy or left atrial enlargement) or significant diastolic dysfunction.
- Heart failure with preserved ejection fraction (HF-pEF) are those patients with symptoms and/or signs of heart failure and an initial ejection fraction of 50% or more and also must have elevated levels of natriuretic peptides and evidence of structural heart disease (e.g. left ventricular hypertrophy or left atrial enlargement) or significant diastolic dysfunction.

Substantial or complete myocardial recovery can occur in patients with heart failure with reduced ejection fraction following treatment. Patients who present with an initial ejection fraction of less than 40% but have a normal ejection fraction at a later date are termed heart failure with recovered ejection fraction (HFrecEF). They are currently treated based on their initial ejection fraction i.e. same as patients with HF-rEF.

Most guidelines discuss left sided heart failure where the left ventricle is involved. However, it is increasingly recognised that the right ventricle can fail also. Right heart failure most commonly results from left heart failure when pressure is transferred from the left heart failure and causes the right heart to fail. Chronic volume overload from right-sided lesions such as tricuspid regurgitation or lung disease such as pulmonary emboli can also lead to the development of right heart failure. Peripheral oedema is often the most prominent clinical feature in patients with chronic right heart failure. Patients with right heart failure frequently require large diuretic doses because congestion leads to an increased volume of distribution, visceral oedema causing impaired drug absorption and rebound sodium absorption in the hypertrophied distal nephron resulting from chronic loop diuretic use. In contrast to clear guidelines available for the management of HFrEF, less evidence is available to guide therapy of predominant right heart failure syndromes. However, most patients with right heart failure will also have left heart failure and should be managed according to current practice guidelines for the management of chronic HF where advice on left heart failure is given.

Time Course of Heart Failure

New onset heart failure may present as acute pulmonary oedema or more commonly with gradual onset symptoms over weeks to months. Patients who have had heart failure for some time have chronic heart failure. A treated patient with symptoms and signs that have remained generally unchanged for at least one month is said to be 'stable'. If chronic stable HF deteriorates, the patient may be described as 'decompensated' and this may happen suddenly or slowly, which can lead to hospital admission.

Severity of Heart Failure

The NYHA functional classification has been used to describe the severity of symptoms and exercise intolerance.

Table 1: NYHA functional classification

Class	Patient Symptoms
I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea (shortness of breath).
II	Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea (shortness of breath).
III	Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, or dyspnea.
IV	Unable to carry on any physical activity without discomfort. Symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases.

Diagnosis of Heart Failure

For patients presenting with symptoms or signs for the first time, non-urgently in primary care, the probability of HF should first be evaluated based on the patient's prior clinical history [e.g. coronary artery disease (CAD), arterial hypertension, diuretic use], presenting symptoms (e.g. orthopnoea), physical examination (e.g. bilateral oedema, increased jugular venous pressure, displaced apical beat) and resting ECG. If all elements are normal, HF is highly unlikely and other diagnoses need to be considered.

If at least one element is abnormal, plasma natriuretic peptides (NPs) should be measured, if available, to identify those who need echocardiography (an echocardiogram is indicated if the NP level is above the exclusion threshold or if circulating NP levels cannot be assessed)

An algorithm is shown below. Timelines are based on NICE and Canadian guidelines. It is recognised that there is very limited access to investigations, including natriuretic peptides, in general practice in Ireland at present. Therefore, in patients in whom a GP has a high index of suspicion of heart failure it is reasonable to start treatment with loop diuretics and assess response to this intervention while awaiting definitive diagnostics. It is important to optimize management of other cardiovascular risk factors such as hypertension, diabetes and atrial fibrillation while awaiting tests also. Although medications such as ACE inhibitors or beta blockers may be used to optimize management of other conditions such as hypertension or atrial fibrillation while awaiting echocardiography it is not recommended to initiate or titrate medications such as ACE inhibitors or beta blockers for heart failure management alone in this group without a recorded ejection fraction as these medications are only of benefit for heart failure management in those patients with heart failure with reduced ejection fraction.

Heart Failure Suspected (Non-Acute Onset)

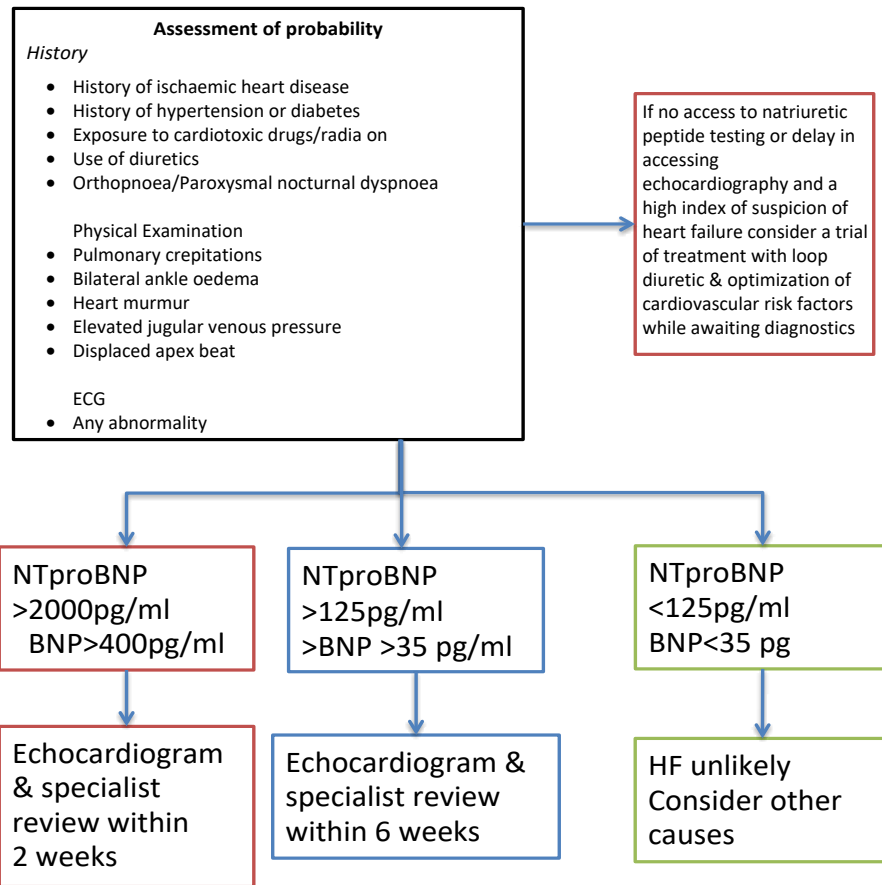


Figure 1: Heart failure suspected (non-acute onset)

Therapy of Heart Failure

Non-Pharmacological Therapy

Management of heart failure should be seen as a shared responsibility between patients, their carers and healthcare professionals. Improved self-care has been associated with improved outcomes in heart failure.

Looking specifically at heart failure self management, early recognition of changes in signs and symptoms is key. Changes can include reduced exercise tolerance, shortness of breath and or weight gain. Being able to evaluate these changes and decide on the appropriate action to take (e.g. taking an extra diuretic dose and or contacting the health care professional) are beneficial. Individuals and their carers need access to structured support programmes for them to gain the knowledge, skill and confidence required to self manage at this level. The website heartfailurematters.org is a useful resource for patients and their carers on heart failure.

Each individual's ability to self-manage will depend, to a large extent, on the actual stage of their condition. The person who has just been diagnosed will have very different support needs to someone who understands and has accepted their condition, or the person who is approaching the end of life stage in their condition.

Table 2: Key topics for Non-Pharmacological Therapy

Topic	Comments
Symptom monitoring and self-care with daily weights	In the case of increasing dyspnoea or oedema or a sudden unexpected weight gain of >2 kg in 3 days, patients may increase their diuretic dose and/or alert their healthcare team. It is important to note also that absence of weight gain does not exclude worsening heart failure in a diagnosed patient.
Immunisation	Immunisations against influenza and pneumococcal disease
Diet and Alcohol	Avoid excessive fluid intake. Monitor body weight and prevent malnutrition Eat healthily, avoid excessive salt intake (>6g/day) and maintain a healthy body weight. Avoid excessive alcohol intake. Normal alcohol guidelines apply (2 units per day in men or 1 unit per day in women). 1 unit is 10 mL of pure alcohol (e.g. 1 glass of wine, ½ pint of beer, 1 measure of spirit). abstain in alcohol induced cardiomyopathy.
Smoking and recreational substance use	Stop smoking and taking recreational substances
Fluid intake	Fluid restriction not normally required but may be considered in patients with severe heart failure. Consider periods when increased fluid intake required e.g. high heat or humidity, nausea or vomiting.
Exercise	Undertake regular exercise to provoke mild/moderate breathlessness.
Travel	Prepare travel plans according to ability. Be aware of times when increased fluid intake needed e.g. hot climates. Be aware of adverse effects with some medications with sun exposure e.g. amiodarone. Take medicine in cabin luggage in plane, carry a list of medications with generic names of drugs

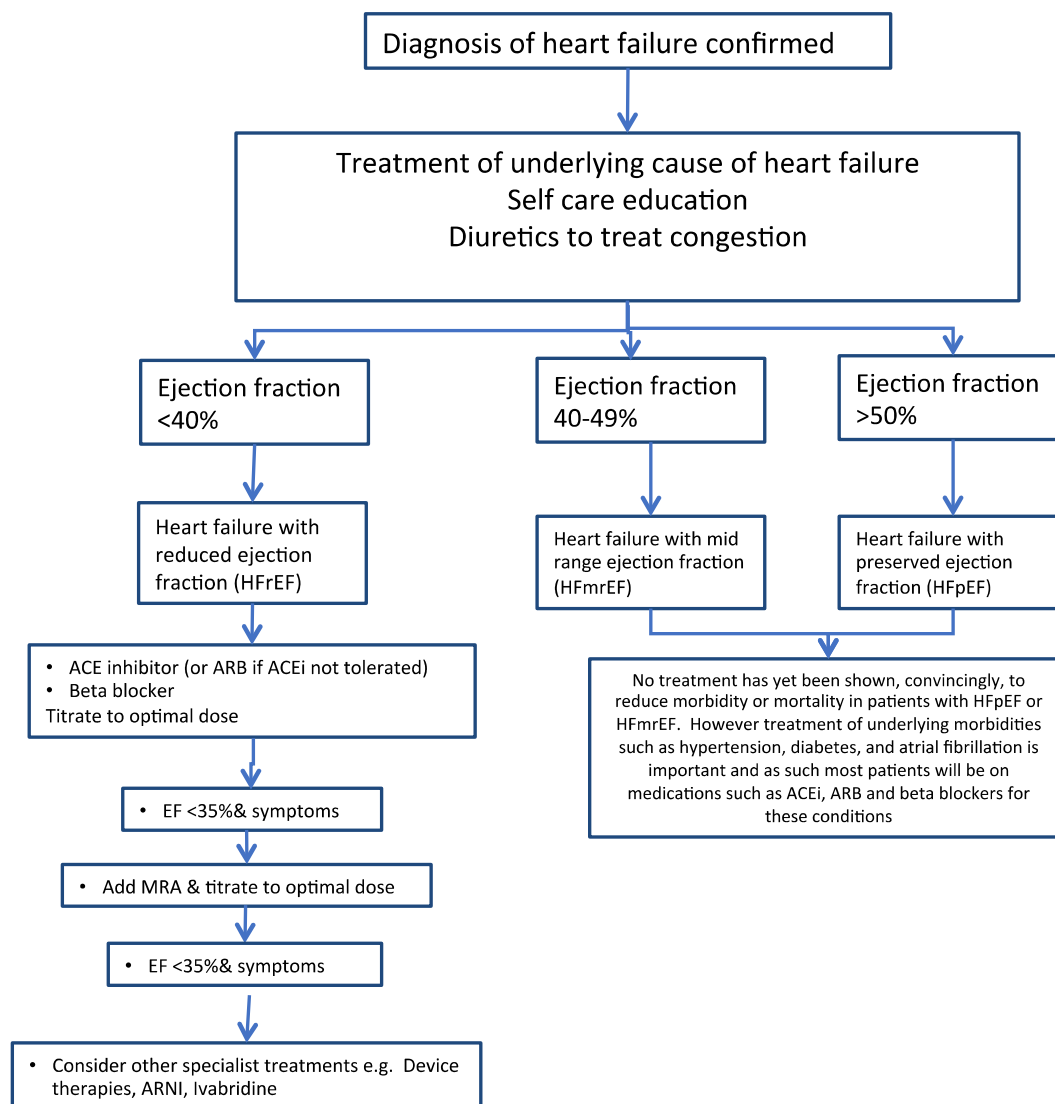


Figure 2: Pharmacological therapy of heart failure

The pharmacological therapy of patients with heart failure has become increasingly more complex as new therapeutic options have become available.

Diuretics

Patients with heart failure and clinical signs or symptoms of fluid overload or congestion should be considered for diuretic therapy with loop diuretics such as frusemide or bumetanide. The dose of diuretic should be individualised to reduce fluid retention without overtreating which may cause dehydration or renal dysfunction and may need adjustment according to individual needs over time.

Loop diuretics produce a more intense and shorter diuresis than thiazides, although they act synergistically and the combination may be used to treat resistant oedema. However, adverse effects are more likely and these combinations should only be used with care (see section on decompensation). The aim of diuretic therapy is to achieve and maintain euvolaemia with the lowest achievable dose. Patients can be trained to self-adjust their diuretic dose based on monitoring of symptoms/signs of congestion and daily weight measurements. Diuretics have been shown to improve symptoms and exercise capacity (Class I Level B) and reduce risks of hospitalization (Class IIa Level B).

Pharmacological Treatment of Heart Failure with Reduced Ejection Fraction

ACE Inhibitors (ACEI)/Angiotensin Receptor Blockers (ARB)

ACEIs have been shown to reduce mortality and morbidity in patients with HFrEF (Class I level A) and should be up-titrated to the maximum tolerated dose in order to achieve adequate inhibition of the renin–angiotensin–aldosterone system (RAAS). ARBs are recommended only as an alternative in patients intolerant of an ACEI (Class I Level B).

Beta Blockers

Beta-blockers reduce mortality and morbidity in symptomatic patients with HFrEF (Class I Level A). Betablockers should be initiated in clinically stable patients at a low dose and gradually up-titrated to the maximum tolerated dose.

Mineralocorticoid Receptor Antagonists (MRA)

MRAs (spironolactone and eplerenone) block receptors that bind aldosterone and, with different degrees of affinity, other steroid hormone (e.g. corticosteroids, androgens) receptors. Spironolactone or eplerenone are recommended in all symptomatic patients (despite treatment with an ACEI and a beta-blocker) with HFrEF and LVEF $\leq 35\%$, to reduce mortality and HF hospitalization (Class I Level A). In this care MRA are being used as a cardioprotective agent rather than as a diuretic and so the doses are often much lower than used for diuretics (e.g. 12.5mg to 25mg daily)

Angiotensin Receptor Neprilysin Inhibitors (ARNI)

A new therapeutic class of agents acting on the RAAS and the neutral endopeptidase system has been developed [angiotensin receptor neprilysin inhibitor (ARNI)]. The first in class is sacubitril/valsartan, which is a molecule that combines the moieties of valsartan and sacubitril (neprilysin inhibitor) in a single substance. It further reduces the risk of HF hospitalization and death in ambulatory patients with HFrEF who remain symptomatic despite optimal treatment with an ACE-I, a beta-blocker and an MRA (Class I level B). They should not be used in combination with an ACE inhibitor or ARB and if the patient is already on an ACE inhibitor a washout period of 36 hours is required after stopping the ACE inhibitor before starting an ARNI.

Ivabridine

Ivabradine slows the heart rate through inhibition of the If channel in the sinus node and therefore should only be used for patients in sinus rhythm. Ivabridine should be considered to reduce the risk of HF hospitalization or cardiovascular death in symptomatic patients with LVEF $\leq 35\%$, in sinus rhythm and a resting heart rate ≥ 70 bpm despite treatment with an evidence-based dose of betablocker (or maximum tolerated dose below that), ACE-I (or ARB), and an MRA (or ARB). (Class IIa Level B).

Pharmacological Therapy of Heart Failure with Preserved Ejection Fraction and Mid-Range Ejection Fraction

No treatment has yet been shown, convincingly, to reduce morbidity or mortality in patients with HFpEF or HFmrEF. However, since these patients are often elderly and highly symptomatic, and often have a poor quality of life, an important aim of therapy may be to alleviate symptoms and improve well-being. Diuretics play an important role in volume management and are used in the same manner as with HF-REF. In clinical practice and clinical trials, compared with HFrEF patients, only slightly fewer patients with HFpEF and HFmrEF currently appear to receive diuretics, beta-blockers, MRAs and ACEIs or ARBs. This may reflect treatment of cardiovascular co-morbidities, such as hypertension, CAD and atrial fibrillation.

Device Therapy

Device Therapy in Heart Failure

Some patients with heart failure with reduced ejection fraction are candidates for device therapy with implantable cardioverter defibrillators (ICD) and cardiac resynchronization therapy (CRT). Eligible patients with severe LV dysfunction may not be candidates for device therapy due to co-morbidities and competing risks. Usually a collaborative, multi-disciplinary approach is best in determining the best device for each patient. The indication for these therapies is constantly developing and therefore only a brief outline is provided below.

Cardiac Resynchronization Therapy

Patients with heart failure may have asynchronous contraction of the ventricles. This leads to inefficient pumping by the heart. This is noted by a prolonged QRS duration as in left bundle branch block. In these patient's ventricular function is improved by pacing the left and right ventricle simultaneously. This ensures that the ventricles contract simultaneously and improves systolic function. This is known as cardiac resynchronization therapy (CRT) or biventricular pacing.

Implantable Cardioverter Defibrillators

Implantable cardioverter defibrillators (ICDs) are devices which deliver a shock to the heart or provide rapid pacing. They do not improve symptoms on their own. They improve mortality by resuscitating patients who sustain a tachyarrhythmia. The mortality benefit of ICD implantation needs to be balanced against the effects of having a device which can deliver painful shocks that are not controllable by the patient. In patients with a prolonged QRS duration the ICD can be combined with biventricular pacing to improve symptoms.

References

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