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REVIEW

Heart failure in older people: causes, diagnosis and treatment

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Abstract

Congestive heart failure affects >5% of those aged 65–75 and 10–20% of those aged >80 in the UK, and levels are likely to rise in the wake of improved therapies for hypertension and myocardial infarction. It is often multifactorial in this group. The most common causes are hypertension and coronary heart disease, with valvular heart disease playing an increasing role. The most common precipitant of pre-existing heart failure is non-compliance with medication or diet; non-steroidal anti-inflammatory drugs are particularly likely to exacerbate the condition. Diagnosis may be difficult since typical signs are often absent or masked in older people, but plasma levels of brain natriuretic peptide appear to be a reliable indicator and may provide diagnostic test in the future. Systolic heart failure is managed by conventional therapy (diuretics, angiotensin-converting enzyme inhibitors and β-blockers). The management of diastolic heart failure is less well defined, but symptoms should be managed, ischaemia prevented and the underlying causes identified and treated. Nurse-directed, multidisciplinary intervention (including education of patient and family, social support, review of medication, dietary modification and weight monitoring) have resulted in improvements in event-free survival and quality of life.

Keywords: *congestive heart failure, diastolic cardiac dysfunction, systolic cardiac dysfunction*

Introduction

Congestive heart failure (CHF) is among the most common discharge diagnoses in elderly hospitalized patients [1, 2]. It affects 1–2% of the population of the United Kingdom, and its prevalence rises from <1% in those aged <65 to >5% among those aged 65–79 and 10–20% among the over-80s [3, 4]. Annual mortality as high as 50% has been reported in patients classified into classes III and IV of the New York Heart Association [5].

Improved therapies for hypertension and myocardial infarction are allowing patients with these disorders to survive, only to develop CHF at a later point [6]. In the UK, heart failure accounts for 1–2% of National Health Service expenditure [7]. Older people are more likely to need hospital admission, as well as more input from social services and family carers than younger people; indeed, patients aged over 70 probably account for two-thirds of National Health Service spending on CHF [8].

Aetiology and precipitating factors

In general, the aetiology of CHF is similar in older and younger patients, but in older individuals it is more often multifactorial. Hypertension and coronary heart disease are the most common causes in elderly patients, accounting for >70% of cases [9]. Valvular heart disease is an increasingly common cause of CHF at older age. Calcific aortic stenosis is now the most common cause of valve disease requiring surgical intervention [10].

The most common precipitant in older patients with pre-existing CHF is non-compliance with medication or diet, which may contribute to two-thirds of CHF exacerbations [11]. In hospitalized patients, iatrogenic volume overload is also an important precipitant [12]. Older patients have limited cardiovascular reserve: as a result, heart failure is often precipitated by acute or worsening non-cardiac conditions. Patients with acute respiratory disorders, such as pneumonia, or an exacerbation of chronic obstructive airway disease,

are particularly prone to decompensation in cardiac function.

Many drugs may contribute to exacerbations of CHF. Non-steroidal anti-inflammatory drugs impair renal sodium and water excretion and may, therefore, contribute to intra-vascular volume overload [13]. In addition, non-steroidal anti-inflammatory drugs antagonize the effects of angiotensin-converting enzyme inhibitors, thereby limiting the efficacy of these agents [14]. A recent study found that this group of drugs were responsible for about 19% of hospital admissions of elderly patients with CHF [15].

Diagnosis

The lack of a universally accepted definition of CHF represents a problem in diagnosis.

Although CHF is commonly defined as inability of the heart to pump blood at a rate suffient to meet metabolic demands or to do so only at an elevated filling pressure [16], clinicians require a more practical description.

The European Society of Cardiology diagnostic criteria [17], listed in Table 1, represent a pragmatic approach which requires subjective symptoms supported by objective evidence of cardiac dysfunction and, when necessary, response to treatment.

However, in older patients the clinical diagnosis of heart failure may be difficult because of the absence of typical symptoms and signs. Many older patients may not have dyspnoea on exertion because of their sedentary lifestyle. When they do become mildly symptomatic with exertion, they tend to decrease their exertional activities and become relatively asymptomatic. Nonspecific complaints of generalized weakness, anorexia and fatigue often predominate. Insomnia may be a feature. Some studies have reported that heart failure is the most frequent precipitating cause of delirium in older patients [18]. When classical symptoms of pulmonary and peripheral oedema do occur in older heart failure patients, the underlying disease process is usually far advanced.

Table 1. The European Society of Cardiology diagnostic criteria for congestive heart failure

1. Symptoms of heart failure (at rest or during exercise):
   dyspnoea;
   exercise intolerance;
   orthopnoea;
   oedema
2. Objective evidence of cardiac dysfunction (at rest) and (in cases where the diagnosis is in doubt)
3. Response to appropriate treatment

From Cleland *et al.* (1995) [17].

Some older patients will experience more typical symptoms of heart failure but, due to the presence of concomitant diseases, these symptoms are frequently misdiagnosed. For example, a dry cough or mild shortness of breath may be mistakenly attributed to chronic pulmonary disease. Easy fatiguability and generalized weakness may be wrongly thought merely to reflect changes associated with ageing. The physical signs that are obvious in younger patients with heart failure may be more subtle and even obscure in older patients. This difference in physical findings may be related partly to the superimposition of ageing changes and/or the presence of other diseases that mask and obscure the typical findings seen in younger patients. Pulmonary crackles or wheezes may be misinterpreted as being related to lung disease. Peripheral oedema is an unhelpful sign as it is common in older patients without heart failure.

Biochemical diagnosis

Given the difficulties in diagnosing heart failure on clinical grounds alone, and current limited access to echocardiography in some places, the possibility of using a blood test to diagnose heart failure is appealing, especially in general practice.

Determining plasma concentrations of brain natriuretic peptide, a hormone found at an increased level in patients with left ventricular systolic dysfunction, may be one option. A normal brain natriuretic peptide concentration virtually excludes left ventricular systolic dysfunction, but a high concentration merely indicates the presence of some cardiac problem, which requires further investigation [19]. Brain natriuretic peptide can also help discriminate between those with breathlessness caused by heart failure from those with breathlessness from other causes [20]. Such a test has the potential to identify patients in whom heart failure is extremely unlikely and those in whom the probability of heart failure is high; for example, in patients with suspected heart failure who have low plasma concentrations of brain natriuretic peptide, the heart is unlikely to be the cause of the symptoms, whereas those who have higher concentrations warrant further assessment.

Work in Glasgow from the Monitoring Trends and Determinants in Cardiovascular Disease (MONICA) study also suggests that brain natriuretic peptide measurement might be a useful method of identifying individuals with important but asymptomatic left ventricular systolic dysfunction in the general population [21]. A small study from Glasgow has suggested that titration of angiotensin-converting enzyme inhibitor treatment with the aid of serial brain natriuretic peptide measurements leads to a greater inhibition of the renin–angiotensin–aldosterone system and a clinically significant fall in heart rate compared with empiric treatment [22]. The benefits of this hormone-guided approach over the conventional strategy in terms of

reducing hospitalization and death have been confirmed in a recent study where brain natriuretic peptide-guided treatment of heart failure reduced total cardiovascular events compared with intensive clinically guided treatment [23].

Although more work is necessary, brain natriuretic peptide is rapidly moving from being a research tool to being a clinically useful test. In the future, brain natriuretic peptide may be used by primary-care physicians to help confirm diagnosis of heart failure in elderly patients and indicate the need for further cardiac assessment [24]. Perhaps it will be of benefit in monitoring patients with heart failure, using serial measurements to fine-tune treatment in a more scientific way than at present.

Systolic *versus* diastolic dysfunction

In 1984 Dougherty [25] and Soufer [26] demonstrated that a subset of patients with clinical heart failure have normal contracting hearts: the problem is not systolic, but ventricular diastolic dysfunction. In the Olmsted County study, carried out in Minnesota, USA, 43% of patients with CHF had a left ventricular ejection fraction of $\geqslant 50\%$ [27]. Similarly, the Framingham investigators found that 51% of their cohort with heart failure had a left ventricular ejection fraction of $\geqslant 50\%$ [28].

The prevalence increases with age, and some investigators consider heart failure secondary to diastolic dysfunction to be mainly a disorder of older patients. In a retrospective study of patients hospitalized for heart failure, Wong and associates [29] found only 6% of patients aged 60 years or younger had normal systolic function, compared with 21% of patients aged 61–70 years and 41% of patients older than 70 years. In a study of 247 older (mean age 84 years) nursing-home residents with heart failure, Aronow *et al.* [30] reported normal systolic ventricular function (left ventricular ejection fraction $\geqslant 50\%$) in 47% of the patients.

However, in a recent prospective study in general practice, in which 159 patients were referred for a suspected diagnosis of heart failure, 109 (69%) of the participants had suspected diastolic heart failure. Of these, 40 were either obese or very obese, 54 had a reduction in forced expiratory volume in 1 s to $\leqslant 70\%$, and 97 had a peak expiratory flow rate $\leqslant 70\%$ of normal. Thirty-one patients had a history of angina, 12 had had a myocardial infarction and seven had undergone a coronary artery bypass graft. Only seven lacked a recognized explanation for their symptoms. The authors concluded that in most patients with a diagnosis of diastolic heart failure there is an alternative explanation for their symptoms—obesity, lung disease or myocardial ischaemia [31].

Unfortunately, the signs and symptoms of diastolic heart failure do not differ from those of CHF secondary to systolic dysfunction [32, 33]. Although the differentiation is difficult, clues to the type of ventricular dysfunction may be obtained from the electrocardiogram and chest x-ray. Q waves in the electrocardiogram or an enlarged heart on the chest x-ray usually suggest systolic dysfunction, whereas left ventricular hypertrophy, left atrial hypertrophy or a small heart are seen more often in patients with diastolic dysfunction. In other words, loss of muscle mass with ventricular dilatation suggests systolic failure whereas muscle hypertrophy and a normal sized heart suggest diastolic dysfunction.

Echocardiographic diagnosis of diastolic heart failure is problematic and technically difficult, and remains largely one of exclusion. One should first exclude all non-cardiac causes of dyspnoea (such as pulmonary disease, fluid retention in renal insufficiency or iatrogenic volume overload) before attributing the patient's symptoms to heart failure [31].

Treatment

Systolic heart failure

Systolic heart failure is managed as in younger people by the conventional therapy of diuretics, angiotensin-converting enzyme inhibitors and β-blockers. There are no specific clinical trials on management of CHF in elderly people and most of the data are extrapolated from trials in younger groups. Over the past decade, the results of numerous randomized controlled clinical trials have demonstrated that β-blockers both improve the symptoms of systolic heart failure and, importantly, impede disease progression when added to conventional therapy [34–38]. They also reduce the incidence of hospitalization and mortality in patients with a broad range of clinical symptoms [39–44]. Some of the trials have included people up to the age of 80 [40].

The benefits of angiotensin-converting enzyme inhibitors include reduction in morbidity and mortality in patients with CHF due to systolic dysfunction [45–50]. Angiotensin-converting enzyme inhibitors are not, however, always tolerated in older people because of adverse side effects such as hypotension, impairment of renal function and persistent cough. Angiotensin receptor blockers may be an alternative. Several clinical studies [51–53] have documented a beneficial effect of angiotensin receptor blockers on haemodynamic and neurohumoral factors in older patients with CHF.

In the 48-week Evaluation of Losartan in the Elderly (ELITE) I study [53], losartan was tolerated better than captopril and seemed to achieve a greater reduction in overall mortality—primarily because of a decrease in sudden death. However, the ELITE II study [54] found no difference in morbidity and mortality between captopril and losartan in patients with CHF. Since the

study was not powered to demonstrate equal efficacy, the authors concluded that angiotensin-converting enzyme inhibitors remained the drug of choice for CHF.

A few clinical studies [55–57] have shown that the addition of an angiotensin receptor blocker to an angiotensin-converting enzyme inhibitor produces a more effective result than that achieved by either drug alone. However, in none of these studies was the upper dose limit of one drug class clearly defined and fully explored before the other drug was added.

In the Randomized Evaluation of Strategies for Left Ventricular Dysfunction (RESOLVD) study, 601 patients were randomized to candesartan, enalapril or a combination of the two. There was no significant difference in the mortality or rate of hospitalization between the three groups at 43 weeks of follow-up [58]. There are ongoing trials to further evaluate the role of angiotensin receptor blockers in CHF [59–61].

The Valsartan in Heart Failure (Val-HeFT) [60] randomized trial is designed to assess the effects of valsartan and an angiotensin-converting enzyme inhibitor on morbidity and mortality in CHF. Preliminary results at 1 year, based on the primary endpoint of all-cause mortality (time to death), demonstrated no significant reduction in the valsartan group compared with the placebo group. However, for a combined mortality and morbidity (time to an event) endpoint, valsartan showed significant improvement (28.8% compared with 32.1%), with a relative risk reduction of 13% ($P=0.009$). In the active group, only 13.9% of patients had a first admission to hospital, compared with 18.5% in the placebo group (relative risk reduction 27%; $P=0.0001$). Ejection fraction on echocardiography also significantly improved in those taking valsartan (unpublished data). At present the role of angiotensin receptor blockers appears to be second-line, in patients who cannot tolerate angiotensin-converting enzyme inhibitors or β-blockers.

The combined use of aldosterone antagonists (for example, spironolactone) and angiotensin-converting enzyme inhibitors has been proposed as a strategy to optimize long-term benefit of suppression of the renin–angiotensin–aldosterone system. The Randomised Aldactone Evaluation Study investigators [62] reported that, in patients with heart failure being treated with an angiotensin-converting enzyme inhibitor and loop diuretic, the addition of spironolactone at a dose of 25–50 mg/day for 24 months decreased hospitalizations, symptomatology and mortality.

The Digoxin Investigators Group study has evaluated the use of digoxin in heart failure in the absence of atrial fibrillation [63]. Their 37-month follow-up study showed a significant 28% reduction in hospital admissions for heart failure but no effect on all-cause mortality. Overall, it seems reasonable to continue to use digoxin to improve the clinical status of patients with heart failure, especially those whose symptoms persist after receiving the drugs (angiotensin-converting enzyme inhibitors and β-blockers) that have proven efficacy in reducing mortality.

Diastolic heart failure

The management of diastolic heart failure is currently undefined. Because there are no well-controlled, randomized, large-scale trials, treatment strategies are based on empirical data. Symptoms of congestion should be managed, ischaemia prevented and, if possible, the underlying causes identified and treated [64].

Most patients will require diuretics to control congestive symptoms. Patients with diastolic heart failure are sensitive to intra-vascular volume change. Excessive diuretic therapy may result in profound hypotension because they need a high filling pressure to ensure that the left ventricle fills adequately. On the other hand, overloading the stiff, non-compliant ventricle can result in pulmonary oedema.

Angiotensin-converting enzyme inhibitors induce regression of left ventricular hypertrophy and in theory may have some independent effects on diastolic function; they should be considered if an antihypertensive agent is needed.

Control of ischaemia with β-blockers, nitrates and calcium channel blockers or revascularization is appropriate in patients whose diastolic dysfunction is related to coronary artery disease. Calcium channel antagonists (diltiazem, verapamil) and β-blockers improve diastolic function through their effects on hypertensive left ventricular hypertrophy [65, 66], prolonging diastole, reducing exercise heart rate and myocardial oxygen demand. In patients with diastolic heart failure, loss of the atrial contribution to left ventricular filling may be less tolerated, and thus every effort should be made to maintain sinus rhythm and control exercise heart rate. Digoxin is indicated for controlling ventricular rate in patients with atrial fibrillation but is inappropriate for patients in sinus rhythm with diastolic heart failure.

The angiotensin receptor blocker valsartan can improve left ventricular filling in patients with mild or moderate essential hypertension and impaired diastolic function [67]. However the role of the renin–angiotensin–aldosterone system in the treatment of left ventricular diastolic dysfunction is currently under investigation [59]. Although diastolic heart failure has a more favourable prognosis (8% mortality rate compared with 19% in systolic heart failure) [68, 69], the mortality risk in older patients may not differ [70].

The need for a multidisciplinary approach

A nurse-directed, multidisciplinary intervention including comprehensive education of the patient and family, social support, a review of medication, dietary modification, assessment of concordance with medication

and weight monitoring results in superior rates of event-free survival and quality of life for elderly patients with CHF [71, 72].

Stewart *et al.* compared 100 patients with CHF who received conventional care with the same number of patients receiving multidisciplinary care, including a home visit by a cardiac nurse 7–14 days after hospital discharge. The combined frequency of unplanned hospital admission plus out-of-hospital mortality was lower in patients receiving nurse visits (77 events) than in those receiving only conventional care (129 events) during 6 months of follow-up ($P=0.02$). In this study, nurse intervention also reduced the number of days of hospitalisation associated with unplanned admission ($P=0.02$) [73]. Other studies also support a role for nurse intervention. For example, Jaarsma and co-workers' study of 179 patients with a mean age of 73 years [74] showed that an intensive, systematic, nurse-based educational programme produced greater improvements in self-care in this group than in patients not receiving systematic education.

The report by Gattis *et al.* [75] describing how the inclusion of pharmacists in a multidisciplinary heart failure team can improve the outcome of heart failure provides an example of the multidisciplinary approach to chronic diseases. Studies involving the participation of pharmacists in the treatment of hypertension have shown that 55% of patients with uncontrolled hypertension at baseline achieved their goal blood pressure (<140/90 mmHg) after 6 months in the intervention arm compared with 20% in the control arm [76]. The involvement of pharmacists on multidisciplinary teams could improve the outcome because of the increased follow-up can result in earlier therapeutic interventions.

Exercise programmes, which can be conducted by a physiotherapist, might also improve the outcome of heart failure. In a study in patients with stable CHF, exercise training was associated with reduction of peripheral resistance and resulted in a small but a significant improvement in stroke volume and reduction in cardiomegaly [77].

Most studies of congestive heart failure in older patients have focused on survival. We need a holistic multidisciplinary approach with a focus on therapy, which not only prolongs life but also maintains a reasonable quality of life [78].

Key points

- Congestive heart failure affects >5% of those aged 65–75 and 10–20% of those aged >80 in the UK; improved therapies for and survival from hypertension and myocardial infarction will cause these levels to rise.
- The most common causes are hypertension and coronary heart disease; the most common precipitant of pre-existing heart failure is non-compliance with medication or diet.
- Systolic heart failure in older people is managed by conventional therapy; for diastolic heart failure, symptoms should be managed, ischaemia prevented and the underlying causes identified and treated.
- Nurse-directed, multidisciplinary intervention (including education of patient and family, social support, review of medication, dietary modification and weight monitoring) have resulted in improvements in event-free survival and quality of life.

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