Heart failure in older patients – the same only different

in association with the British Geriatric Society Cardiovascular Section

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This conference has been approved by the RCN Accreditation Unit. It has been awarded 10 study hours and the event reference is 3881.

CPD accreditation has been sought.

Please note that photography, video and audio recording of the sessions and slides of this meeting is strictly prohibited.
Programme – Day One

FRIDAY 24 NOVEMBER 2006

09.00 Registration – Tea/coffee

09.15–09.40 BSH Annual General Meeting (BSH Members only)

DAY ONE Programme director: Jackie Taylor (Glasgow)

Session 1: Understanding heart failure in older patients
Co-Chairs: John Cleland (Kingston-upon-Hull) & Chakravarthi Rajkumar (Brighton)

09.45 What changes as we age?
David J Stott (Glasgow)

10.05 What is changing in the population?
Chakravarthi Rajkumar (Brighton)

10.25 What are the different aetiologies of heart failure in older patients?
Theresa McDonagh (London)

10.50 What are the diagnostic challenges in older patients?
John Baxter (Sunderland)

11.05 Panel discussion

11.00 Tea/coffee

Session 2: Management of heart failure in older patients: what is the evidence?
Co-Chairs: Hugh McIntyre (Hastings) & Iain Squire (Leicester)

11.40 Managing older patients with heart failure and LVSD
John McMurray (Glasgow)

12.00 Managing older patients with heart failure and preserved systolic function
John Cleland (Kingston-upon-Hull)

12.20 Managing older patients with CHF and valvular disease
Mark Cheesman (Bristol)

12.40 Panel discussion

12.45 Lunch
Programme – Day One

Session 3: Coping with co-morbidity
Co-Chairs: Steve Parry (Newcastle) & Jackie Taylor (Glasgow)

13.45 Co-morbidity in heart failure: therapeutic and prognostic impact
Marion ET McMurdo (Dundee)

Illustrative cases
14.00 Failing kidneys Philip Kalra (Manchester)
14.15 Failing lungs Sundeep Puri (Manchester)
14.30 Failing generally Andrew T Elder (Edinburgh)

Chair: Henry Dargie (Glasgow)

Debate: “This house believes that, for patients with heart failure, quality is more important than quantity of life”
14.45 The case ‘For’
John McMurray (Glasgow)
14.55 The case ‘Against’
Andrew Clark (Hull)
15.05 Voting

15.10 Tea/Coffee

Session 4: Heart failure rehabilitation: Should we? Dare we? How to?
Programme director: Fiona Lough (London)
Co-Chairs: Bernie Downey (Belfast) & Suzanna Hardman (London)

15.40 BACR/BSH heart failure rehabilitation group: an update
Fiona Lough (London)

15.50 Conventional models of risk stratification in heart failure rehabilitation: help or hindrance?
Martin Cowie (London)

16.10 Five-year follow-up findings from a RCT of cardiac rehabilitation for heart failure in Gwent
Jacky Austin (Abergavenny)

16.30 Evidence-based exercise rehabilitation: from clinical trials to day-to-day practice
Rod Taylor (Exeter)

16.50 Report from National Heart Failure Audit
Henry Dargie (Glasgow) and an analyst from the CCAD

17.20–18.50 Poster presentations and reception with exhibitors

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Programme – Day Two

Continued from page 3

SATURDAY 25 NOVEMBER 2006
09.00 Registration – Tea/coffee

DAY TWO Programme directors: John Cleland (Kingston-upon-Hull), Peter Cowburn (Southampton) and Nigel Rowell (Middlesbrough)

Session 1: Failure to recognise heart failure
Co-Chairs: John Cleland (Kingston-upon-Hull) & Nigel Rowell (Middlesbrough)
09.15 The primary care perspective
Nigel Rowell (Middlesbrough)
09.30 The hospital perspective
Martin Cowie (London)
09.45 Should heart failure be a ‘one-key’ (natriuretic peptides or echo) or ‘two-key’ (natriuretic peptides + echo) diagnosis?
Kevin Goode (Hull)
10.00 Barriers to the implementation of new technologies within the NHS
Tom Quinn (Coventry)
10.15 Tea/coffee

Session 2: Cardiac resynchronisation therapy: some answers, more questions
Co-chairs: Peter Cowburn (Southampton) & Suzanna Hardman (London)
10.50 Patient selection for CRT: who is most likely to respond?
John Cleland (Kingston-upon-Hull)
11.05 Management of the non-responder
Rakesh Sharma (London)
11.20 Should most patients who need CRT get an ICD?
Peter J Cowburn (Southampton)
11.35 Panel discussion

Session 3: Hot topics in heart failure
Co-chairs: Martin Cowie (London) & Henry Dargie (Glasgow)
11.50 ABCD
John Cleland (Kingston-upon-Hull)
12.00 The perindopril in elderly people with chronic heart failure (PEP-CHF) study
John Cleland (Kingston-upon-Hull)
12.10 Erythropoietin analogues
Kenneth McDonald (Dublin)
12.20 IMPROVE-CHD
Andrew Clark (Hull)
12.40 Ultrafiltration
Andrew Clark (Hull)
12.50 Close
Ageing is associated with a gradual reduction in cardiac reserve. Key factors that contribute to this include the intrinsic ageing of cells, tissues and organs, a gradual reduction in physical activity (with ‘detraining’), adverse environmental and social factors, and a greatly increased prevalence of chronic cardiac disease.

Early studies of cardiac function and ageing were confounded by the inclusion of older individuals with ischaemic heart disease. Clinically significant coronary artery disease is present in as many as two-thirds of the over 65s in the developed world. However, only a minority of these individuals have classic symptoms and, therefore, the condition is not generally diagnosed. This high burden of ischaemic vascular disease is a major contributor to reduced cardiac reserve in the older population.

However, intrinsic ageing also has an effect. There is attenuation of the rise in heart rate during aerobic exercise in older people, due to a reduction in both the number and sensitivity of cardiac beta-receptors. During submaximal aerobic exercise, the older heart relies on cardiac dilatation and increased stroke volume to maintain cardiac output. However, this physiological compensation has limits, and contributes to the reduction in maximal cardiac output and decreased cardiac reserve seen in older people. In addition, left ventricular diastolic function deteriorates with ageing, associated with an increase in left ventricular mass and increased wall rigidity. The early phase of diastolic filling becomes less effective, and the elderly heart relies more on atrial contraction during late diastole for effective ventricular filling. As a result, if atrial contraction is lost, such as in atrial fibrillation, this has marked deleterious effects on diastolic filling with an associated reduction in maximal cardiac output.

In addition to the reduction in maximum cardiac output with ageing, there is reduced efficiency of aerobic exercise, with increased oxygen costs at the same work-rates in older individuals. This is likely to be due mainly to reduced biomechanical efficiency, including joint and muscle stiffness, and alterations in the cerebral control of movement (with reduced step length). There are also major changes in the structure and function of skeletal muscle with advancing age, with preferential loss of type 2 (fast-twitch) fibres. These changes can be attenuated but not prevented by regular exercise.

There are, therefore, are many factors contributing to the age-associated reduction in maximal exercise capacity and cardiac reserve. They include intrinsic ageing, adverse environmental influences, a high prevalence of disease (particularly ischaemic vascular disease) and physical inactivity. This complex aetiology gives many opportunities for intervention and improving cardiovascular function in older people.
What is changing in the population?

Chakravarthi Rajkumar

Brighton & Sussex Medical School, Royal Sussex County Hospital, Brighton

It is well known that life expectancy has increased in the past 100 years. As a result, the proportion of people in the UK over the age of 65 years continues to increase. The incidence of heart failure, which is a problem in this age group, is also increasing. With recent advances in treatment, a greater proportion of elderly patients with heart failure live longer. However, this group of patients have a number of other co-morbidities. This has a major impact on service delivery to this group. This talk will focus on the changes occurring in this population and how co-morbidities will affect the management of these patients.
What are the different aetiologies of heart failure in older patients?

Theresa McDonagh
Department of Cardiology, Royal Brompton Hospital, London

As previous speakers will have alluded to, heart failure is essentially a cardiogeriatric syndrome that most health care systems are struggling to cope with due to the changing demographics of the population.

Given that both the prevalence and incidence of heart failure are said to be increasing in the population, not just due to the greater proportion of elderly patients at risk, but also to greater survival of cardiovascular disease earlier in life, it is not a surprise that the causes of heart failure in the elderly are the same as those in younger patients. However, the relative importance and population-attributable risks of the more common causes, such as coronary heart disease (CHD), hypertension and valvular heart disease, differ.

The most common difference in the underlying pathology is the greater proportion of older patients who have heart failure in the presence of preserved systolic function.

Hypertension and CHD account for up to 80% of heart failure cases in the elderly. Whereas hypertension is the most common aetiology of heart failure in elderly women, CHD remains the most important cause in older men. Valvular heart disease, especially aortic stenosis and mitral incompetence, are important causes of heart failure in older patients. Of the cardiomyopathies involved in the elderly, restrictive ones associated with amyloid are relatively more common than in the young.

In addition to the actual aetiology, it is important to remember the many precipitants of decompensation of heart failure in the elderly, which include the development of arrhythmias, anaemia, and renal and thyroid disease. Also of note is that heart failure in the elderly virtually never occurs alone – the frequency of a myriad of co-morbid conditions and their respective treatments also increases.
What are the diagnostic challenges in older patients?

John Baxter
Sunderland Royal Hospital, Sunderland

The gold standard for diagnosing congestive heart failure (CHF) is based on guidelines produced by the European Society of Cardiology. However, the triad of symptoms and signs of heart failure, investigational evidence of ventricular dysfunction and symptomatic improvement with treatment is more difficult to establish in older patients with suspected CHF and hence the diagnostic challenge is greater.

The three main reasons for this are:
1. The difficulty in obtaining a classic history and examination.
2. The under-investigation and difficulty in interpreting investigational findings in older persons.
3. The presence of co-morbidities that may confound the diagnosis.

A number of factors contribute to the difficulty and delay in obtaining a classic history in older persons with suspected CHF. Cognitive impairment and delirium confound history taking. Older persons are more likely to be sedentary and hence may not complain of classic dyspnoea. Atypical features, such as weight loss, anorexia and generalised fatigue, may be the sole presenting features.

If CHF is suspected, recent evidence suggests that older patients are less likely than younger patients to undergo a transthoracic echocardiogram during an index admission as a newly diagnosed heart failure patient.

Investigational findings may also be more difficult to interpret. This is particularly the case for patients with suspected CHF who have relatively preserved left ventricular systolic function. Many of the features of diastolic heart failure also occur in normal ageing hearts and there are no universally accepted reference ranges for elderly heart failure patients with suspected diastolic heart failure. Also, B-type natriuretic peptide appears to be less specific as age increases.

Co-morbidities are a significant confounding factor in attempts to establish a diagnosis of CHF. Spirometry evidence of severe chronic obstructive pulmonary disease and echo evidence of severe left ventricular systolic dysfunction are often found in the same patient. Dependent oedema, hypoalbuminaemia, pulmonary fibrosis and obesity are all common confounding factors in attempts to establish heart failure in older persons.

Poly-pharmacy may also contribute to the difficulties in diagnosing heart failure in older patients and is more common in this group.

It is for the reasons described above that physicians interested in managing patients with heart failure should encourage referral for all patients with suspected CHF, particularly elderly patients, so that expert opinion can accurately confirm or refute the diagnosis.
Managing older patients with heart failure and LVSD

John McMurray
Department of Cardiology, Western Infirmary, Glasgow

Although it is frequently said that we do not have a good evidence base for the treatment of older patients with heart failure, the contrary is true, especially for newer pharmacological therapies.

For example, in a meta-analysis of more than 12,000 patients in large randomised trials, Dulin et al. found that the relative risk of death in 4617 elderly patients was 0.76 (95% CI 0.64, 0.90) compared with 0.66 (0.52, 0.85) in the non-elderly (no statistically significant difference).\(^1\) Furthermore, in the Study of the Effects of Nebivolol Intervention on Outcomes and Rehospitalisation in Seniors with Heart Failure (SENIORS), compared with placebo, nebivolol reduced the risk of the primary composite endpoint of death or cardiovascular hospitalisation by 14% (p=0.039) in the 2128 patients aged ≥70 years (mean age 76 years) who were randomised to the study.\(^2\)

Just as the evidence for the use of angiotensin-converting enzyme (ACE) inhibitors is less than that for beta-blockers in heart failure overall, so it is the case for the elderly with heart failure. It is worth remembering, however, that the mean age of patients in the first study to show a reduction in death with an ACE inhibitor, the Cooperative North Scandinavian Survival Study (CONSENSUS), was 70 years.\(^3\) Unfortunately, no good meta-analyses of the effect of ACE inhibitors in the elderly with heart failure have been conducted. Garg and Yusuf examined the effect of an ACE inhibitor in patients aged >60 years compared with those aged ≤60 years and found no difference in treatment benefit.\(^4\) Flather et al. looked at patients aged >75 years (1066 in total) in a number of acute infarction trials, including the Studies of Left Ventricular Dysfunction (however, the meta-analysis excluded CONSENSUS and other trials in heart failure).\(^5\) Again, there was no statistically significant heterogeneity of treatment effect according to age.

Turning to angiotensin receptor blockers (ARBs), 2409 patients aged 70–79 years and 610 patients aged ≥80 years were included in the Candesartan in Heart Failure: Assessment of Reduction in Mortality and Morbidity (CHARM) programme.\(^6\) The candesartan/placebo hazard ratios for the primary endpoint of cardiovascular (CV) death or heart failure hospitalisation were 0.81 (0.71, 0.92) and 0.79 (0.63, 0.98), respectively, in these two age bands compared with 0.84 (0.77, 0.91) in the overall programme (p<0.0001). Of note, the risk of CV death or heart failure hospitalisation over the average follow-up period of 38 months was 46.3% in those aged ≥80 years, 38.4% in those aged 70–79 years, 30.2% in those aged 60–69%, 22.7% in those aged 50–59 years and 23.8% in those <50 years of age. In other words, because elderly patients were at much higher risk, but had the same relative reduction in risk with treatment, the absolute benefit was much greater.

The benefit of other treatments in the elderly with heart failure is less certain. This is particularly true of spironolactone, although the effect of this treatment was similar in patients above and below the median age (67 years) in the Randomized Aldactone Evaluation Study (RALES).\(^7\) Similarly, in the Cardiac Resynchronization – Heart Failure (CARE-HF) trial, the benefit of treatment was similar in those above and below the median age of 66.4 years.\(^8\) At the time of writing, I am not aware of any further analysis of the effect of these treatments by age.

My focus so far has been on ‘efficacy’ as opposed to ‘tolerability’. It seems clear that adverse event and discontinuation rates have been higher in elderly patients in both the active therapy and placebo groups in randomized trials, although the relative increase in discontinuation rates (active therapy versus placebo) seems to be relatively constant across the age spectrum. The limited available evidence also shows that the target dose of therapy achieved is lower in elderly than in younger patients. It is worth pointing out, however, that this makes it all the more impressive that older patients obtain the same relative and greater absolute risk reductions than younger ones.

Of course, the nay-sayers and therapeutic nihilists will counter with claims that the older patients in trials are super-selected and not really very elderly. I think even my granny could see that the evidence overwhelmingly shows she would be better off trying the treatments mentioned!

References

Managing older patients with heart failure and preserved systolic function

John Cleland
Department of Cardiology, Castle Hill Hospital, University of Hull, Kingston-upon-Hull

Many older people who appear to have heart failure do not have obvious left ventricular systolic dysfunction (LVSD). This reflects inadequate left ventricular assessment in some and, in others, the presence of long-axis systolic dysfunction or a problem other than heart disease mimicking heart failure. Measurement of natriuretic peptides may be a better diagnostic method than imaging in order to identify patients whose symptoms are more likely to respond to anti-failure therapy and who are at increased risk of heart failure events.

So far, substantial trials of digoxin, candesartan and perindopril have been reported in this population and partially reported data exist on nebivolol and propranolol. The completion of trials of irbesartan and spironolactone are awaited. None of the trials reported so far has been conclusive, although perindopril has been shown to improve symptoms and functional capacity, as well as reduce heart failure hospitalisation over the first year of treatment.

Many patients with heart failure who do not have LVSD will have atrial fibrillation, hypertension and ischaemic heart disease as potential causes of symptoms. Good control of these problems may improve patient well-being.

Further reading
Old people with congestive heart failure (CHF) often have abnormal heart valves, although these may not be the underlying cause of the CHF. Stethoscopes have inadequate sensitivity and specificity for accurate assessment in elderly patients with CHF, and every older person with heart failure needs an echocardiogram at some point during their clinical course.

Aortic stenosis is common, and must not be missed or underestimated: palliation with rate control is helpful in those unfit for surgery. Mitral regurgitation is much more frequent than suggested clinically, and systemic vasodilation with angiotensin-converting enzyme inhibitors commonly produces good symptom relief. Tricuspid regurgitation can be awkward to control medically.

The opportunity to replace or repair a culprit valve requires earlier diagnosis than has often been the case in elderly patients. Minimal-access surgery may offer elderly patients with severe mitral regurgitation or aortic stenosis considerable benefits in the near future.
Co-morbidity in heart failure: therapeutic and prognostic impact

Marion ET McMurdo
University of Dundee, Dundee

Non-cardiac co-morbidities are common in older heart failure patients and include chronic obstructive pulmonary disease, osteoarthritis, lower respiratory and thyroid disease, anxiety and depression, dementia, chronic renal impairment, asthma and osteoporosis. Such conditions may easily be neglected if clinicians are unable to resist an exclusive focus on heart failure management. This phenomenon has been described for a number of diseases in which the presence of one chronic condition is likely to lead to over-attention to that condition, but at the direct expense of under-treatment of other co-morbid diseases. This matters because older heart failure patients with multiple co-morbidities are at high risk of adverse events, including hospital admission, around half of which are potentially preventable.

There may be under-use of effective therapies when co-morbid conditions are present because of safety concerns; older people may have low expectations of healthcare services and delay seeking attention if symptoms recur; the co-ordination and planning of discharge and post-discharge may be poor, and the greater burden of chronic illness may worsen the decline in homeostatic resilience that occurs as part of the ageing process. Co-morbidity is almost inevitably associated with being in receipt of multiple medicines and, unfortunately for older people, there is strong correlation between number of drugs prescribed and the risk of suffering an adverse drug reaction. What and whether to prescribe to older heart failure patients is currently based on an individual assessment of biological and chronological age, and a judicious weighing of potential risks and benefits. It is a matter of great regret that the current dearth of research in older heart failure patients with co-morbidities makes such Solomon-like judgements necessary.

The presence of co-morbid disease is one of the hallmarks of ageing, and heart failure in very old people virtually never occurs in isolation. Research is needed on the most effective strategies for managing heart failure in the context of multiple illnesses and medications. There is an urgent need for heart failure physicians to broaden their management focus beyond simply the cardiac problems, and for closer working with clinicians who relish the challenge of tackling co-morbid diseases – those specialising in the medical care of older people.

Further reading

Failing kidneys

Philip Kalra
Department of Renal Medicine, Hope Hospital Salford, Manchester

Abstract not available at time of going to press.
Heart failure itself can cause abnormalities of lung function. In the acute phase with pulmonary oedema, an obstructive picture can be seen. In stable chronic heart failure (CHF), reductions in long volumes and transfer factor are seen. These abnormalities have been shown to correlate well with functional status.

The most common cause of heart failure is ischaemic heart disease, and many patients with heart failure have a history of smoking. CHF and chronic obstructive pulmonary disease (COPD) are notoriously under-diagnosed in the community as well as in hospitals. Moreover, distinguishing between the two as well as between heart failure in the presence of COPD is difficult clinically. In a hospital-based study, only 31% of patients with a diagnosis of COPD had confirmatory pulmonary function tests. In patients with CHF, 78% had a confirmatory echocardiogram. Patients who had both COPD and CHF as a concomitant diagnosis had limited investigations (only 34% underwent an echocardiogram, 16% spirometry and only 2% had both investigations). Of the patients with COPD who underwent spirometry, 30% had either a restrictive picture or normal spirometry (i.e. had been misdiagnosed).

In a series of almost 55,000 patients with a history of myocardial infarction (MI), approximately 20% had COPD. Several studies in patients with COPD (both in a hospital setting and in primary care) have suggested that 20% of patients with a diagnosis of COPD also have concomitant heart failure. It is therefore apparent that many patients with COPD may have undiagnosed heart failure (or a misdiagnosis), and are therefore not receiving appropriate medical treatment such as angiotensin-converting enzyme (ACE) inhibitors.

However, even when a correct diagnosis has been made there is a marked reluctance to use beta-blockers in patients with confirmed COPD or asthma. A Cochrane review of the use of beta-blockers in COPD showed the use of cardioselective beta-blockers to have no significant effect on spirometry (studies were short ranging, from 2 days to 12 weeks). In patients with asthma, beta-blockers without intrinsic sympathomimetic activity showed no adverse effects on spirometry in the short term, although there did appear to be an increase in bronchial re-activity.

There are very few studies in the literature on the use of beta-blocker therapy in patients with both COPD and heart failure. Kotlyar et al. looked at 487 patients receiving carvedilol. Nine per cent of patients had confirmed lung disease on lung function tests (31 had COPD and 12 had asthma). Carvedilol was tolerated in 84% of patients with COPD but in only 50% of asthmatic patients.

Summary
Abnormalities of lung function are common in heart failure. Approximately 20% of patients with COPD may have undiagnosed concomitant heart failure. The evidence suggests that cardioselective beta-blockers are safe to use in COPD. In asthma, the evidence is less clear and it appears that beta-blockers are tolerated less well.

Further reading
The management of co-morbidity is a significant issue in the care of older people with heart failure. In addition to common physical problems such as chronic obstructive pulmonary disease (COPD), osteoarthritis and renal impairment, psychiatric problems such as cognitive impairment secondary to dementia can complicate assessment, treatment and care. In addition, frailty, which may be a consequence of heart failure, co-morbidity, the ageing process per se, or the combination of these, is increasingly recognised as a distinct clinical syndrome.

Management and care strategies for older people with heart failure must therefore take these changes into account. Co-ordinated multiprofessional geriatric assessment provides an evidence-based method in the hospital or community by which such multiple issues can be identified and managed. All inpatient units managing heart failure patients should have good access to specialist elderly medicine assessment from appropriately trained doctors, nurses and therapists, and seek to develop good links with such specialist inpatient services, social services and the wider community services. Good discharge planning leads to a reduction in re-admission and can only occur if a multiprofessional approach with clearly defined lines of communication and responsibility is adopted.

During assessment, functional deficits in the ability to undertake personal or domestic care should be identified, documented and treated if possible. Mobility must be appraised, and simple correctible factors, such as pain, treated. Incontinence should be accurately assessed and a management strategy devised. The occurrence of falls should be actively investigated and the risk of consequent harm minimised or further events reduced. Cognitive impairment should be identified and correctible precipitants sought.

It is vital to appreciate that the combined effect of small improvements in the multiple and, individually, relatively minor functional deficits or symptoms that may be identified during assessment may have a large impact on the quality of life and function of an older person.

Specific heart failure management plans should take into account the individual needs and views of the older person. A strategy that includes the addition or uptitration of drugs designed to improve prognosis rather than symptoms or quality of life may not be appropriate for, or desirable to, all older patients.

Community care of heart failure patients with multiple co-morbidity and functional deficits should include a form of follow-up and monitoring that addresses their multiple needs. If ongoing therapy input is required in the community, day hospital attendance or domiciliary input may be useful. The needs of carers should be addressed and respite provision actively considered.

Further reading


BACR/BSH heart failure rehabilitation group: an update

Fiona Lough
University College Hospital London NHS Trust, Cardiac Rehabilitation Department, The Hatter Institute, University College Hospitals, London

Abstract not available at time of going to press.
Conventional models of risk stratification in heart failure rehabilitation: help or hindrance?

Martin Cowie
National Heart & Lung Institute, Imperial College, London & Royal Brompton Hospital, London

Abstract not available at time of going to press.
Five-year follow-up findings from a RCT of cardiac rehabilitation for heart failure in Gwent

Jacky Austin
Gwent Healthcare NHS Trust, Nevill Hall Hospital, Abergavenny

W Robert Williams, Linda Ross (School of Care Services, University of Glamorgan, Pontypridd) and Stephen Hutchison (Gwent Healthcare NHS Trust, Nevill Hall Hospital, Abergavenny) also participated in this study

Multidisciplinary specialist follow-up and exercise training are two well-endorsed treatment modalities for patients with chronic heart failure. As a care strategy, cardiac rehabilitation incorporates both these modalities and requires evaluation.

Aim
To report on the 5-year status of surviving patients (n=179 at 6 months) in a 24-week randomised controlled trial comparing cardiac rehabilitation with a heart failure outpatient clinic (standard care) in patients over 60 years of age with chronic stable heart failure.¹

Methods
In the original study, 200 patients (aged 60–89 years, 132 male) with New York Heart Association (NYHA) class II–III heart failure confirmed by echocardiography were randomised between 2000 and 2001. At 5-year follow-up, the initial trial measures (6-min walk test, Minnesota Living with Heart Failure [MLHF] and EuroQol quality of life scores, and routine biochemistry) were repeated if the patient was in a satisfactory condition. Data on deaths and admissions were obtained from the medical records department.

Results
Two-thirds of the sample (n=112, 66.5%) were alive at 5 years (mean age 75.2 years) and most (94%) attended clinic for assessment. There were no statistically significant differences in clinical variables, although the intervention group showed better trends for survival, quality of life and walking distance, and reduced time in hospital. More patients in the intervention group were taking regular exercise (71% versus 51%; p<0.05, n=102). In general, NYHA class and walking distance had deteriorated in surviving patients compared with baseline measurements, although MLHF scores were better than those at baseline. In comparison with deceased patients, survivors showed significant differences in renal function, physiology and quality of life parameters at 6 months. Deaths increased with age, higher NYHA class and left ventricular dysfunction.

Conclusion
A 24-week cardiac rehabilitation programme for patients with stable heart failure had a limited effect on patients at 5 years. The intervention group showed positive trends for survival and reduced time in hospital. Differences in the mean values of most functional and quality of life measures were evident to the advantage of the intervention group, which also showed a better exercise profile.

Reference
Evidence-based exercise rehabilitation: from clinical trials to day-to-day practice

Rod Taylor
Peninsula Medical School, University of Exeter, Exeter

Meta-analyses of randomised controlled trials undertaken over the past 20–30 years show that exercise training following a myocardial infarction or revascularisation can reduce the mortality, improve the morbidity and may also enhance the health-related quality of life of patients. Nevertheless, the direct applicability of this evidence base to current UK cardiac rehabilitation practice has recently been called into question.¹

During the past decade, exercise training has also increasingly been used in the management and rehabilitation of patients with heart failure. This presentation will:

- assess the evidence for the use of exercise-based rehabilitation in heart failure
- review the characteristics of the patients and the nature of the exercise intervention used in these studies
- explore the applicability of this evidence base to current UK rehabilitative practice.

Reference

Report from National Heart Failure Audit

Henry Dargie
University of Glasgow, Glasgow
and
Analyst from the CCAD

Abstract not available at time of going to press.
The primary care perspective

Nigel Rowell
James Cook University Hospital, Middlesbrough

Heart failure remains a diagnosis that will catch physicians by surprise when it is found, and the converse is true – that some patients are on inappropriate treatment for a false diagnosis.

- So in whom should we suspect this diagnosis?
- Are there any clues in the history or the past?

I hope to enlighten these issues using audits in primary care and clinical cases. As General Practice Quality and Outcomes Framework data become ‘cleaner’, the demographics of heart failure will become clearer. Much of the data hitherto has been based on medication searches and is, as such, possibly inaccurate.

Finally, could the screening of target groups – patients with ischaemic heart disease, diabetes and hypertension in primary care – be the way forward and if so, how?
The hospital perspective

Martin Cowie
National Heart & Lung Institute, Imperial College, London & Royal Brompton Hospital, London

Abstract not available at time of going to press.
Should heart failure be a ‘one-key’ (natriuretic peptides or echo) or ‘two-key’ (natriuretic peptides + echo) diagnosis?

Kevin Goode
Hull Royal Infirmary, Hull

Abstract not available at time of going to press.
Barriers to the implementation of new technologies within the NHS

Tom Quinn
Coventry University, Coventry

Abstract not available at time of going to press.
SATURDAY 25 NOVEMBER 2006 - DAY TWO - SESSION TWO

Patient selection for CRT: who is most likely to respond?

John Cleland
Department of Cardiology, Castle Hill Hospital, University of Hull, Kingston-upon-Hull

The CARE-HF study showed that cardiac resynchronisation therapy (CRT) could improve ventricular function, symptoms, and morbidity and mortality (reduced by 40%) over 3 years of follow-up. It is the only large, long-term trial that collected detailed baseline data on dyssynchrony and the ventricular response to CRT.

Although all patients do not obtain equal benefit from CRT in terms of improved cardiac function, symptoms, morbidity or mortality, a simplistic division of patients into responders and non-responders is inappropriate for several reasons.

1. There is a continuous spectrum of response, which will extend from deterioration in some patients right through to a response resembling a cure for heart failure in others. CRT devices can be reprogrammed in patients who appear to worsen.
2. When judging response, the underlying natural history must be taken into account. Patients who are destined to do well anyway will probably do well with CRT.
3. Different criteria may be applied with which to judge response and these may be poorly related. For instance, an improvement in ventricular function or symptoms may not be a good guide to effects on morbidity and mortality.

Analysis of the CARE-HF study, which had a control group that could be used to show whether treatment changed the natural history of the disease, has shown some surprising results. No measured variable accurately predicted the ability of CRT to reduce morbidity and mortality. Greater inter-ventricular mechanical delay (IVMD) and lower systolic blood pressure both predicted a slightly greater response to CRT, but symptoms, N-terminal prohormone brain natriuretic peptide (NT-proBNP) and QRS duration did not, although they did predict overall prognosis, regardless of assigned group. Patients with ischaemic heart disease had a smaller increase in left ventricular ejection fraction with CRT but at least as great a mortality benefit as those that did not. Patients with greater IVMD had a better prognosis in the control group, suggesting that the better outcome observed in observational studies in patients with more dyssynchrony treated with CRT may just reflect their intrinsically better prognosis.

The CARE-HF study entry criteria should be used to select patients for CRT. These are New York Heart Association III–IV (current or recent), left ventricular ejection fraction ≤35%, QRS >120 msec and, only for those with QRS 1201–149 msec, an imaging test showing dyssynchrony, of which IVMD is the best proven. Imaging assessment for dyssynchrony is unnecessary and perhaps unwise for those with QRS ≥150 msec. Patients with atrial fibrillation were excluded from CARE-HF but it is not clear that this is appropriate. On the other hand, for patients referred for a defibrillator who have a depressed ejection fraction, there are few reasons why routine upgrade to CRT with an implantable defibrillator (CRT-D) should not be considered. Indeed, if a patient with low ejection fraction is not a candidate for CRT they should probably not receive a defibrillator either.

Further reading
Management of the non-responder

Rakesh Sharma
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In clinical trials of cardiac resynchronisation therapy (CRT), approximately one-third of patients did not appear to gain benefit. In everyday clinical practice, this figure may be even higher. Considering that CRT has important implications in terms of potential complications and cost, it is crucial that we are better able to identify the predictors of responder status. Furthermore, patients who do not appear to benefit (or actually worsen) following device implantation require careful and systematic evaluation.

Non-responder status may be defined in several ways: worsening symptoms (New York Heart Association class, exercise capacity), increased hospitalisation rates for decompensated heart failure or the objective measure of the lack of reverse left ventricular remodelling (plasma B-type natriuretic peptide levels, left ventricular volumes).

The management of the apparent non-responder requires the adoption of a clinical problem-solving approach. The fluid status of the patient needs close monitoring following CRT. Over-diuresis is relatively common in the first few days after successful biventricular pacing due to an improvement in the patient’s haemodynamic profile leading to a reduction in diuretic requirements. Failure to recognise and correct this may result in a deterioration in renal function and masking of the potential benefits from CRT.

Interrogation of the device can also yield useful information and should be performed routinely. Pacemaker diagnostics can determine the presence and frequency of new-onset atrial tachyarrhythmias, which can reduce the percentage of time biventricular pacing is effectively delivered. The device parameters should also be reprogrammed under echocardiographic guidance to ensure that optimal haemodynamics are obtained for the individual. Even though the presence of cardiac ischaemia should have been evaluated prior to device implantation, this may need further consideration if symptoms worsen in a patient who is being paced.

The management of the heart failure patient who requires CRT needs close collaboration between heart failure physicians, electrophysiologists and echocardiographers in order to achieve the lowest non-responder rate possible.
Should most patients who need CRT get an ICD?

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Cardiac resynchronisation therapy (CRT) improves symptoms and exercise tolerance, reduces hospitalisation and decreases mortality in patients with moderate/severe chronic heart failure and a prolonged QRS.\(^1\,^2\) CRT can be delivered as a CRT pacemaker (CRT-P) or with a defibrillator (CRT-D). Both CRT-P and CRT-D have been shown to improve symptoms and mortality. CRT-D is considerably more complex than CRT-P, and comes at significantly greater cost. What we don’t know is who should receive which type of device.

Patients with chronic heart failure die predominantly as a result of pump failure or sudden cardiac death. CRT-P reduces heart failure deaths and offers protection against bradycardic death, whereas CRT-D offers additional protection against tachyarrhythmic death. The simple logic, therefore, should be that all patients should receive CRT-D. However, an implantable cardioverter defibrillator (ICD) has not always been shown to reduce mortality in patients with chronic heart failure. The benefit of an ICD depends upon competing risks (e.g. the Defibrillator in Acute Myocardial Infarction Trial [DINAMIT]\(^3\)). In a subgroup analysis of the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT), the benefit of an ICD appeared to be predominantly in patients with class II heart failure.\(^4\) No evidence currently exists that an ICD reduces mortality in class IV heart failure. As such, there may be subgroups of patients that would do as well with CRT-P as with CRT-D.

The major driving force behind this debate is cost. Whereas both CRT-D and CRT-P are cost-effective when compared with medical therapy, the additional benefit from CRT-D comes at much greater cost.\(^5\) The arguments for and against CRT-D will be discussed.

References
ABCD

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Data not available at time of going to press.
The perindopril in elderly people with chronic heart failure (PEP-CHF) study

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Aims
Many patients who receive a diagnosis of heart failure have neither a low left ventricular ejection fraction nor valve disease. Few substantial randomised controlled trials have been conducted in this population; none has focused on patients with evidence of diastolic dysfunction and none has shown a clear benefit on symptoms, morbidity or mortality.

Methods and results
This was a randomised, double-blind trial comparing placebo with perindopril 4 mg/day in patients aged ≥70 years with a diagnosis of heart failure treated with diuretics and an echocardiogram suggesting diastolic dysfunction and excluding substantial left ventricular systolic dysfunction or valve disease. The primary endpoint was a composite of all-cause mortality and unplanned heart failure-related hospitalisation, with a minimum follow-up of 1 year. A total of 850 patients were randomised. Their mean age was 76 (SD 5) years and 55% were women. Median follow-up was 2.1 (IQR 1.5–2.8) years. Enrolment and event rates were lower than anticipated, reducing the power of the study to show a difference in the primary endpoint to 35%. Many patients withdrew from perindopril (28%) and placebo (26%) after 1 year and started taking open-label angiotensin-converting enzyme (ACE) inhibitors. Overall, 107 patients assigned to placebo and 100 assigned to perindopril reached the primary endpoint (hazard ratio [HR] 0.919: 95% CI 0.700–1.208; p=0.545). By 1 year, reductions in the primary outcome (HR 0.692: 95% CI 0.474–1.010; p=0.055) and hospitalisation for heart failure (HR 0.628: 95% CI 0.408–0.966; p=0.033) were observed, and functional class (p=0.030) and 6-min corridor walk distance (p=0.011) had improved in those assigned to perindopril.

Conclusion
Uncertainty remains about the effects of perindopril on long-term morbidity and mortality in this clinical setting since this study had insufficient power for its primary endpoint. However, improved symptoms and exercise capacity and fewer hospitalisations for heart failure in the first year were observed in those receiving perindopril, during which most patients were on assigned therapy, suggesting that it may be of benefit in this patient population.

Further reading
The link between anaemia and cardiovascular disease has been well established for years, with extensive information from the dialysis literature. More recently, there has been increasing awareness of the importance of anaemia in heart failure. Several databases, from both community and clinical trial heart failure populations, have closely linked level of haemoglobin with outcomes in this condition, independently of other well-recognised predictors. Furthermore, anaemia has been associated with other important endpoints in heart failure, including New York Heart Association functional class, peak oxygen consumption, heart failure hospitalisation and renal dysfunction.

The precise pathophysiological effect of anaemia in heart failure is uncertain but probably relates to reduced delivery of oxygen to tissues. There are many possible explanations for the development of low haemoglobin in this syndrome, including chronic disease, bone marrow suppression from cytokines, malnutrition, renal insufficiency and a possible influence of drugs. In addition, the high prevalence of use of antiplatelet and anticoagulant therapies in this population may contribute to anaemia. Silverberg and colleagues were the first to demonstrate benefits of intervention in anaemia of heart failure, with an improvement in functional classification and ventricular function, as well as a possible mortality benefit with erythropoietin and iron. Mancini and colleagues subsequently showed an improvement in peak oxygen consumption. Subsequently, there have been three phase II clinical trials examining the safety and efficacy of erythropoietin in patients with low ejection fraction heart failure and anaemia. While not achieving statistical significance in any of the primary trial endpoints, the data demonstrated trends towards improvements in the measurement of functional capacity, including peak oxygen consumption, 6-min walk tests and quality of life assessments.

The above data suggest that there may be benefit from intervention in patients with anaemia of heart failure. However, conclusive evidence on this issue awaits large-scale clinical trials such as the RED-HF Trial, which is designed to assess the impact of darbopoietin-alpha on the combined endpoint of death and heart failure hospitalisation in 3400 patients with heart failure with systolic dysfunction and anaemia.
IMPROVE-CHD

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Data not available at time of going to press.
Ultrafiltration

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A common clinical problem for patients with chronic heart failure is fluid retention leading to hospital admission, often for a prolonged period. The traditional approach to fluid removal in heart failure is with intravenous diuretics. Difficulties with diuretic therapy for fluid retention arise once patients are hospitalised for a number of reasons.

• By the time that patients present, they often have very gross fluid retention (at least 5 L overload needed before oedema is clinically apparent and up to 15 L overload is not rare).
• It can take the clinicians a considerable time to identify the required regimen, with the risks of both over- and under-treatment.
• Oral diuretics are unreliable and often need to be replaced with intravenous ones.
• A sufficiently high dose of loop diuretic needs to be used.
• Continuous infusion is probably more efficacious than repeated boluses,
• but increases complexity and access-site complications.
• There is a general appreciation that fluid should be removed at a rate of no more than approximately 2 L/24 h.
• The need for prolonged bed rest during diuresis, with attendant risks for mobility and thromboembolic complications.
• Side effects:
  – fall in renal perfusion and worsening renal function
  – hyponatraemia
  – gout
  – dehydration.
• Diuretic resistance.

Diuretic resistance occurs as increasing doses of loop diuretics have less and less effect. Methods for circumventing the problem include:

• using intravenous infusions
• using ‘sequential nephron blockade’ by adding a thiazide diuretic and/or spironolactone to the diuretic regimen
• the use of intense angiotensin II suppression, which may increase frusemide efficacy
• stopping drugs causing fluid retention (e.g. non-steroidal anti-inflammatory drugs)
• the addition of dopamine as renal vasodilator.

An alternative strategy is to use mechanical means to remove fluid. Ultrafiltration can be used to remove large quantities of fluid in a much shorter period of time, and is less dependent than diuresis upon the patient’s renal function for its effect. The urine produced by diuresis is hypotonic, whereas fluid removed by ultrafiltration is isotonic – that is, ultrafiltration empties more salt from the body for every litre of removed fluid.

In the UNLOAD trial, an open-label study, 200 patients with a mean age of 63 years and mean New York Heart Association class of III–IV were enrolled. Patients were randomised to receive ultrafiltration using a commercially available system (CHF Solutions, Brooklyn Park, MN, USA) or intravenous diuretic therapy (either bolus or continuous infusion) within 24 h of admission.

Ultrafiltration caused more rapid fluid loss, but had no effect on dyspnoea score at 48 h. There was a marked reduction in rehospitalisation at 90 days (from 32% to 18%) after filtration, with similar reductions in number of days in hospital and number of unscheduled office visits.

The UNLOAD study is by no means definitive and a lot more work needs to be done to establish its role in routine clinical practice. Ultrafiltration needs to be tested against more aggressive diuretic regimens, in larger groups of patients and in patients who have become resistant to conventional therapy. However, it does offer a new way forward for patients with marked fluid retention. Although set-up costs around €20,000 (and the filters used for each session cost in the order of €900), it perhaps offers management sight of the Holy Grail of reducing the average length of hospital stay of heart failure patients from the present 11 days.

References
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1. Changes in plasma concentrations of novel peptide, apelin, in patients with established anterior myocardial infarction

KS Chong, RA Weir, RS Gardner, JJ Morton (Department of Cardiology, Western Infirmary, Glasgow), EA Ashley (Stanford University School of Medicine, California, USA), TA McDonagh (Royal Brompton Hospital, London) and H Dargie (Department of Cardiology, Western Infirmary, Glasgow)

Presenting author: KS Chong

Key points:

The novel peptide, apelin, an endogenous ligand for the APJ receptor, has been shown in animal studies to have positive inotropic, vasodilatory and antidiuretic properties, indicating that apelin might play an important role in cardiovascular homeostasis. Plasma concentrations of apelin have been shown to be significantly reduced in patients with chronic heart failure regardless of aetiology. This is a pilot study aiming to investigate the changes in plasma apelin concentrations in 20 patients with established anterior myocardial infarction during admission and after 6 months of follow-up.

2. Patients’ experiences of severe breathlessness in heart failure

Barbara Flowers (Southport & Ormskirk NHS Trust, Merseyside)

Presenting author: Barbara Flowers

Key points:

• Symptom relief at the advanced stages of heart failure.
• Outcomes of a qualitative study exploring the experience of severe breathlessness in six patients at the end stages of heart failure.
• Identifying coping strategies developed by the patients, formulated into a plan of care.
• Suggested holistic response to distressing symptoms for patients and family.
3.

**Does estimation of B-type natriuretic peptide immediately after exercise treadmill testing aid in early detection of left ventricular dysfunction?**

*Sujatha Kesavan (Royal United Hospital, Bath) and P Boreham (North Bristol NHS Trust, Bristol)*

Presenting author: Sujatha Kesavan

**Key points:**

- B-type natriuretic peptide (BNP) has an excellent negative predictive value. A value of 100 pg/ml and below excludes the diagnosis of heart failure.
- BNP levels above 100 pg/ml in patients with positive exercise treadmill testing may help to predict future adverse cardiac events, diagnose left ventricular dysfunction early and will help to identify high-risk patients.
- BNP analysis is important for risk stratification and confers prognostic information.
- BNP can be measured rapidly and accurately at a reasonable cost and serves as an important cardiac biomarker providing diagnostic and prognostic information.

4.

**Cost-effectiveness of B-type natriuretic peptide in diagnosing and managing heart failure**

*Sujatha Kesavan (Royal United Hospital, Bath) and P Boreham (North Bristol NHS Trust, Bristol)*

Presenting author: Sujatha Kesavan

**Key points:**

- B-type natriuretic peptide (BNP) has an excellent negative predictive value. A value of 100 pg/ml and below excludes the diagnosis of heart failure.
- BNP analysis is a simple, rapid, cost-effective and an inexpensive test that can be used in the community to streamline decision making and for the prompt diagnosis of heart failure.
- Serial estimation of BNP helps to optimise anti-heart failure therapy and obviates the need for repeated trans-thoracic echocardiograms to assess left ventricular function.
- BNP analysis in the community prevents recurrent hospital admissions due to heart failure and reduces in-hospital stay, thereby saving time, money and demand on the health-care system.
5. Renal dysfunction, as measured by the modification of diet in renal disease (MDRD) equations, and outcome in patients with advanced heart failure

Roy S Gardner (Royal Infirmary, Glasgow), Kwok S Chong (Western Infirmary, Glasgow), Eileen O’Meara (Montreal Heart Institute, Canada), Alan Jardine (Western Infirmary, Glasgow), Ian Ford (University of Glasgow, Glasgow), Theresa A McDonagh (Royal Brompton Hospital, London)

Presenting author: Theresa A McDonagh

Key points:
- Renal dysfunction is common in advanced heart failure.
- Renal dysfunction is associated with an adverse prognosis.
- N-terminal pro-brain natriuretic peptide (NT-proBNP) appears superior to glomerular filtration rate (GFR) estimated by MDRD equations in patients with advanced chronic heart failure.
- NT-proBNP was able to identify patients with a poor prognosis whose GFR was already low.

6. Evaluation of a community based heart function clinic for older adults

JA McManus and J Taylor (Department of Medicine for the Elderly, Glasgow Royal Infirmary, Glasgow)

Presenting author: JA McManus

Key points:
The management of chronic heart failure (CHF) in older adults can be challenging. We have specifically developed a heart function clinic to manage this patient group. Retrospective analysis of clinic data revealed that the population attending have significant symptoms, multiple co-morbidities and many require further multidisciplinary input. Many were prescribed appropriate evidence-based therapy, though polypharmacy was evident. Our results suggest that a specialised clinic for older adults with CHF can be successful in implementing evidence-based treatment and highlights the importance of a multidisciplinary approach to management. In the future we hope to develop the clinic within a day hospital.
7. Llandough Day Hospital – a multidisciplinary service for titration and monitoring of cardiac failure patients

Maria Oliver, Victor Sim, Bun Little and Joanne Davies (Cardiff and Vale NHS Trust)

Presenting author: Maria Oliver

Key points:
We are a multidisciplinary team working in a day hospital setting. The aim of the service is to provide a structured multidisciplinary programme of care for patients with heart failure and their families throughout the patients' journeys. Since 2002, we have been providing weekly service for the titration, review and monitoring of heart failure patients, with consultant input. There is a growing amount of evidence within the literature that a multidisciplinary heart failure service can reduce mortality, readmission rates and length of stay (Stewart 1998, Rich 1999, Stewart 2000, Stewart 2001, Stewart 2001, Blue 2001, Doughty 2002).

8. Anaemia and chronic kidney disease in patients with chronic heart failure – service and cost implications?

Victor Sim, Steve Riley, Zaheer Yousef and Sinead O’Mahony (Care of Elderly/Medicine, Nephrology and Cardiology Department, Llandough Hospital and University Hospital of Wales of Cardiff and Vale NHS Trust)

Presenting author: Victor Sim

Key points:
Both anaemia and chronic kidney disease (CKD) are common in elderly patients with chronic heart failure. In our study of 86 heart failure patients over the age of 65 (mean age 80 years), 40% have anaemia as defined by European Best Practice (haemoglobin level <12 g/dl in men and <11.5 g/dl in women). 80% of patients have CKD stage III, defined as having estimated glomerular filtration rate <60 ml/min/1.73 cm². At the haemoglobin threshold value of 11.0 g/dl for the treatment of anaemia using a combination of intravenous iron and erythropoietin, as adopted by previous investigators, 24% of patients will require treatment. This translates to 967 heart failure patients with CKD over the age of 65 years in the Cardiff and Vale NHS Trust serving a population of 424,654 people. The estimated cost of erythropoietin alone would be over two million pounds. Further research studying the effectiveness and cost-effectiveness of treatment of anaemia in heart failure patients with CKD is urgently needed.
9. **Preserved cognition in heat failure with preserved systolic function? Insights from the PEP-CHF study**

J Taylor (Glasgow Royal Infirmary, Glasgow), JGF Cleland (University of Hull, Kingston-upon-Hull), D O’Mahoney (Cork University Hospital, Cork, Ireland), C Gray (Sunderland Royal Hospital, Sunderland), N Freemantle (University of Birmingham, Birmingham), J Adamus (Department of National Defence, Warsaw, Poland), L Polonski (Silesian School of Medicine, Zabrze, Poland), M Tendera (Silesian School of Medicine, Katowice, Poland)

**Presenting author: J Taylor**

**Key points:**
- Studies have suggested that chronic heart failure (CHF) is independently associated with cognitive impairment and that these patients have poorer outcomes.
- This was a prospective study of cognitive function in a cohort of older patients with CHF and preserved systolic function.
- A subgroup of patients enrolled in PEP-CHF (n=215; mean age 80.1 years; 109 receiving perindopril and 106 receiving placebo) completed the Mini-Mental State Examination (MMSE) at baseline and after 1 year of follow-up.
- MMSE score fell by a mean of 0.1 (SD 3.3) in those assigned to perindopril and by 0.2 (SD 3.3) in those receiving placebo (ns).
- This study showed no evidence of a rapid decline in cognition in a cohort of elderly patients with preserved systolic function.

10. **A functional exercise programme for older heart failure patients – tolerability and acceptability**

Miles D Witham, Anne R Daykin, Marion ET McMurdo
(University of Dundee)

**Presenting author: Miles D Witham**

**Key points:**
Exercise training programmes for older heart failure patients have not been well studied. We developed a twice-weekly group exercise programme for older heart failure patients incorporating resistance, aerobic and everyday functional exercises. The 12-week programme was tested on 17 heart failure patients, mean age 81.6 years. The programme required minimal equipment, and was safe and well attended (83% of sessions attended). Encouraging improvements were seen in the 6-min walk test (+19 m, p=0.14) and in the Functional Limitation Profile score (~82 points, p=0.02). 50% of informal carers attended at least one session, and no increase in measured carer strain was noted.
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