Managing Heart Failure – Picking up the Pieces

Programme Directors:
Suzanna Hardman and Theresa McDonagh

Friday 25 November 2005
Queen Elizabeth II Conference Centre, London
09.45–18.45
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*This conference has been approved by the RCN Accreditation Unit. It has been awarded 9 hours and the event reference is 3675. CPD accreditation has been sought*
Programme

09.00 Registration – Tea/coffee

09.20–09.40 **BSH Annual General Meeting**
Co-Chairs: *John Cleland (Hull)*
*Theresa McDonagh (London)*

**Session 1:** Acute heart failure: an ever-widening chasm
Co-Chairs: *Suzanna Hardman (London)*
*Theresa McDonagh (London)*

09.45 How far are we from an ideal integrated heart failure service?
*Suzanna Hardman (London)*

10.10 The epidemiology of acute decompensated heart failure
*Martin Cowie (London)*

10.35 Optimising inpatient care and the impact on mortality
*Kenneth McDonald (Dublin)*

11.00 Tea/coffee

**Session 2:** Acute heart failure: recognition of the problem, contemporary audit data and how we move forward
Co-Chairs: *Martin Cowie (London)*
*Iain Squire (Leicester)*

11.30 Audit of patients leaving hospital with a diagnosis of heart failure
*Andrew Clark (Hull)*

11.45 BHF Nurse Programme audit data
*Mary Crawshaw-Ralli (Bradford)*

12.00 Heart failure audit data from a GP with special interest
*Nigel Rowell (Middlesborough)*

12.15 Audit data of discharges from secondary care: view of the COOP physician
*John Baxter (Sunderland)*

12.30 The Glasgow Heart Failure Liaison Service: reflecting audit in practice within a Specialist Nurse Service
*Kirstin Russell (Glasgow)*

12.45 Acute heart failure: a comparison with the rest of Europe
*John Cleland (Hull)*

13.00 How do we move forward?
*Theresa McDonagh (London)*

13.15 Lunch
Programme

Session 3: Heart failure research
Co-Chairs: Henry Dargie (Glasgow)
          Peter Weissberg (London)

14.10–15.30 The natural history of left ventricular dysfunction in the population
Theresa McDonagh (London)

Beta-blockers in heart failure: not just catecholamine antagonists
Sian Harding (London)

Cardiac high-energy phosphate metabolism in heart failure
Stefan Neubauer (Oxford)

Scar extent as a determinant of adverse left ventricular remodelling in
chronic heart failure
Nikolay Nikitin (Hull)

15.30 Tea/coffee

Session 4: HOT topics/Looking to the future
Co-Chairs: Kenneth McDonald (Dublin)
          John Cleland (Hull)

15.55–16.45 MAGIC
William McKenna (London)

BEAUTIFUL
Henry Dargie (Glasgow)

CARE-HF
John Cleland (Hull)

ESSENTIAL
Henry Dargie (Glasgow)

SURVIVE/REVIVE
John Cleland (Hull)

ETNA
Henry Dargie (Glasgow)

16.45 Closing remarks
John Cleland (Hull)

16.50 Reception with exhibitors and poster presentations

17.30–18.45 Educational Symposium sponsored by Takeda
‘Chronic Heart Failure Management: Sharing the Care – Regional Experiences’
A programme for this symposium can be found on the back cover
How far are we from an ideal integrated heart failure service?

Suzanna Hardman
Clinical & Academic Department of Cardiovascular Medicine, Whittington Hospital, London

This opening presentation is designed to set the scene for the morning sessions. What should ideal integrated heart failure services look like, and where are the gaps? As part of this presentation, new data will be presented from a busy DGH in an inner city setting that is trying to establish comprehensive heart failure services, and will include early data from a randomised controlled trial of a novel nurse-led intervention for patients admitted to hospital with heart failure. Are we doing enough for inpatients? Can we modify the horrendous mortality of this cohort? Are we doing enough to support British Heart Foundation nurses, and could we do better?

Many of the issues thrown up in this presentation will be discussed in greater detail in subsequent presentations and will then be revisited by Dr McDonagh in the closing presentation of the morning.
The epidemiology of acute decompensated heart failure

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

By definition, acute heart failure presents with a rapid onset of symptoms. This can be de novo, or on a background of chronic heart failure. Within the UK healthcare setting, such patients usually present to secondary care, often via the accident and emergency department. Co-morbidity is common, with acute coronary syndrome often the precipitant of the acute heart failure. Population-based studies in Europe and North America have reported a high early mortality, but many patients respond to therapy rapidly.

The professional responsibility for the care of such patients will vary between hospitals, but is likely to involve emergency room physicians, acute medicine receiving units, general and care of the elderly medical wards, and cardiology/intensive care. The need for hospital-wide protocols and a local champion for heart failure is widely recognised. Data from other healthcare systems suggest that protocol-based audits with publication of pre-specified quality indicators may help to drive up quality. The Health Care Commission has selected the emergency management of patients admitted with heart failure as an appropriate condition for a national (England) audit of the activity of acute hospitals. The results are due to be published early in 2006.
Many epidemiological, morbidity and economic indices underline the need for a change in approach to the in-hospital management of heart failure. These indices include the fact that heart failure represents one of the most common causes for admission to hospital, especially in the elderly. Moreover, 20% of the entire heart failure population will be admitted to hospital every year, with many requiring re-admission soon after discharge. The economic impact of the above figures is underlined by the fact that hospitalisation accounts for approximately 70% of the total heart failure budget, which reflects both the frequency of admission and the length of stay (approximately 9–11 days).

Many of the above statistics and characteristics of heart failure can be modified, but this will require a significant change in the management strategies for this patient group. Such changes should include the placement of all patients admitted with heart failure under the management of the cardiology service. In many hospitals this does not happen. Published data have underlined that cardiology care results in more complete investigation of patients and better use of standard proven therapy, with evidence suggesting better short- and long-term outcomes. Ideally, patients admitted with left ventricular failure should also be admitted to a designated unit in a manner similar to the management of acute coronary syndromes and myocardial infarction. Following admission, and in tandem with medical care, relevant allied healthcare professionals involved in heart failure management should become involved. Of particular importance at this time is patient and family education regarding issues of self care in the heart failure syndrome.

Finally, discharge readiness is a critical component of the inpatient heart failure care process. Criteria should be adopted to help define clinical stability and these should be adhered to in order to guard against premature discharge. The early course of a patient’s stay in hospital can be used to define the patient as either being at low or high risk of re-admission. Features underlying a low risk of re-admission would be rapid clinical response, early decline in brain natriuretic peptide levels and early discontinuation of intravenous therapy. Such low-risk patients may be suitable for early discharge once there is an appropriate chronic ambulatory care heart failure facility available for close monitoring following discharge. High-risk individuals may not be appropriate for such early discharge. To guard against early re-admission it is critically important that all patients discharged from hospital following left ventricular failure are followed-up closely in a designated unit by experienced medical personnel. As we gain more experience in this type of follow-up it may be possible to delegate low-risk individuals as defined above to nurse outreach follow-up.

By adopting the above strategies it is likely that the morbidity associated with heart failure can be lessened, the reliance of this patient population on hospitalisation reduced and the overall outlook of the sicker end of the heart failure population significantly improved.
There is an increasing need to provide accurate and timely analyses of healthcare activity in order to:

- monitor and improve standards of care
- focus healthcare resources where they are most needed and most effective.

Data collection is the key resource to make this a useful exercise.

Interventional procedures lend themselves well to this approach: single, well-defined events taking place in a small number of areas with easily defined dataset requirements.

Acute medical conditions, such as acute myocardial infarction, superficially appear to be similarly susceptible to data collection, but we already know from MINAP (Myocardial Infarction National Audit Project) that in some areas MINAP returns are filled in for fewer than one-third of the appropriate patients.

Specialist outpatient services, such as heart failure clinics, can be designed to allow full data capture if the clinic and database systems are integrated. However, this neglects entirely all those patients with chronic heart failure who are being managed elsewhere within the system (other cardiologists, physicians, geriatricians and those not even reaching secondary care).

I present an audit of patients with a discharge-or-death diagnosis of heart failure in a large district hospital with tertiary referral services for cardiology. Important points are: the difficulties of data collection; the inadequacies of classification and coding; the lack of linkage between different data collection systems; the need for dedicated personnel; and the importance of designing data collection around the pathway that an individual patient follows through a hospital stay.

Reference

The Bradford Heart Failure Nurse Specialist (HFNS) Service provides city-wide services to patients with heart failure and their carers from the three Bradford Primary Care Trusts (PCTs): North, South and West, and City.

The HFNS Service aims to support patients and their carers in the community following hospital admission with a primary diagnosis of heart failure. It also aims to help manage patients, referred by the primary care team, whose heart failure has become unstable (in order to try and prevent admission to hospital), and those with complex or palliative needs.

Our referral criteria are:

- All patients who have had at least one emergency hospital admission with a primary diagnosis of heart failure within the past month.
- Heart failure must be caused by left ventricular systolic dysfunction, as determined by echocardiography or coronary angiography/ventriculography.

Heart failure nurse specialist contact following discharge includes:

- contact by telephone within 2 working days of discharge
- home visit within 1 week of discharge
- second home visit within 1–2 weeks of first visit
- third home visit 1–4 weeks after second visit, depending on need
- subsequent contact frequency determined by individual patient needs and may be by phone or home visit, or at a heart failure clinic (primary or secondary care).

This session will present up-to-date audit figures from the Service and attempt to reflect on some of the issues and possible reasons for the data produced. I hope to demonstrate drug prescribing patterns for the service and highlight the difficulties of polypharmacy and uptitration within real patient populations.
Heart failure audit data from a GP with special interest

Nigel Rowell
James Cook University Hospital, Middlesborough

Three audits across primary and secondary care will attempt to demonstrate:

1. What makes GPs think of heart failure and how do they seek to diagnose it?
2. How well they record and act on information from secondary care?
3. What can be done to ‘track’ patients admitted acutely and bring them into the heart failure systems to optimize their therapy and management?

As a GP/hospital practitioner, I hope to show how seamless care could be engineered to put the pieces together.
Audit data of discharges from secondary care: view of the COOP physician

John Baxter
Care of the Elderly, Sunderland Royal Hospital, Sunderland

Sunderland Royal Hospital serves a population of approximately 300,000 people, of whom 7.3% are aged over 75 years. There is a high degree of social deprivation in Sunderland and there are high mortality rates from ischaemic heart disease: 865 per 100,000 population aged between 65 and 74 years. By contrast, the national average is 661 per 100,000 population.

The Department of Care of the Elderly runs an acute geriatric service admitting over 10,000 acute medical admissions a year aged over 70 years. There are over 500 acute admissions with new or decompensated heart failure every year to the unit. Most of the older persons admitted with heart failure have left ventricular systolic dysfunction with ejection fractions less than 40%.

Prior to the development of a heart failure service for older persons in Sunderland, 30% of older persons discharged from hospital following an admission with new or decompensated congestive cardiac failure were re-admitted within 3 months. The development of inpatient evidence-based management guidelines, rapid-access transthoracic echocardiography, early supported discharge with home visits from heart failure specialist nurses and the setting up of a heart failure clinic dedicated to older persons has significantly improved outcomes.

Re-admissions have decreased by 50%, and there is now a much higher rate of prescribing of angiotensin-converting enzyme (ACE) inhibitors and beta-blockers. Our data show that beta-blockers are less well tolerated in older persons than the results of initial beta-blocker trials suggested, but that if an older person does tolerate a beta-blocker they will do so at the target doses suggested by these trials. More recent evidence from the Seniors Trial is comparable with our data.

Our experience in Sunderland shows that the application of current evidence-based management guidelines in older persons with heart failure does improve outcome.
The Glasgow Heart Failure Liaison Service: reflecting audit in practice within a Specialist Nurse Service

Kirstin Russell
Heart Failure Liaison Service, NHS Greater Glasgow, Primary Care Division, Glasgow

Within the Glasgow Heart Failure Liaison Service data are collected and collated on a quarterly basis to assist with service capacity and workload issues. Items reported on each quarter include:

- new patient recruitment
- deaths and discharges
- active caseload
- patient contact
- drug regimens.

All of these data form the basis of an Annual Report, which is fed back to both clinicians and management. By looking at the Service as a whole and examining how it has developed over the past 5 years, we aim to compare results from our Baseline Report against those in the most recently published Annual Report. The Baseline Report was completed after all of the sites had been fully operational for a full calendar year, and was published in 2002.
Two large European surveys have been conducted on admissions to hospital with heart failure in Europe. UK centres participated only in the first of these studies. In 18 UK hospitals, over a 6-week period in 2000–2001, a review of 8581 consecutive deaths and discharges from medical wards revealed that 1991 (23%) patients had evidence of heart failure, although the diagnosis was not recorded in their notes in about one-third of cases. Nearly 90% (88%) of these patients had been treated with loop diuretics at the time of death or discharge. Similar data were obtained from 23 other countries belonging to the European Society of Cardiology (ESC).

Patients from the UK were older than the ESC average (75 years versus 71 years) due to the younger age of patients in Eastern Europe. Heart failure was the primary reason for admission in only 20% of cases and cardiologists managed only 21% of cases (compared with an ESC average of 43%). In only about half of patients was an echocardiogram carried out, and very few patients had undergone an exercise test or a coronary angiogram at any time prior to or during hospitalisation in the UK. Only about 40% had evidence of left ventricular systolic dysfunction and two-thirds had evidence of coronary disease. UK patients were less likely to receive angiotensin-converting enzyme (ACE) inhibitors and beta-blockers than patients in most other countries, and were more likely to receive calcium channel blockers (Figure 1). Over 30% (31%) of patients were re-admitted to hospital within 12 weeks of discharge (ESC average 24%). Mortality during admission was 9% (ESC average 7%) and 12-week mortality 16% (ESC average 14%). However, UK outcomes were similar to those of other Western European countries.
How do we move forward?

Theresa McDonagh
Department of Cardiology, Royal Brompton Hospital, London

Abstract not available at the time of going to press
The natural history of left ventricular dysfunction in the population

Theresa McDonagh
Department of Cardiology, Royal Brompton Hospital, London

Abstract not available at the time of going to press
Beta-blockers in heart failure: not just catecholamine antagonists

Sian Harding
National Heart and Lung Institute, Imperial College, London

Long-term stimulation or overexpression of the β₁-adrenoceptor (AR) is clearly associated with damaging effects on the myocardium. The benefit of β₁-AR blockade in heart failure is consistent with the prevention of further deterioration due to chronic catecholamine exposure. However, the role of the β₂-AR in heart failure is more intriguing. The range of signalling pathways modulated by the β₂-AR is greater than that for the β₁-AR, and much animal or cell-based experimentation has indicated a protective role for β₂-AR activation. Since most of the clinically useful β-blockers will bind to the β₂-AR to some extent, the role of this subtype in the therapeutic action of these agents must be considered.

Using human ventricular isolated myocytes, we have found that β-blockers can have rapid, direct negative inotropic effects on contraction, and that these effects are mediated by the β₂-AR. The effects are not due to catecholamine blockade, since they are observed in superfused myocytes with negligible noradrenaline or adrenaline content. Negative inotropic effects of β-blockers are observed only in myocytes from failing, not non-failing, human heart, and become more pronounced as failure worsens. Inhibition of Giα/βγ dissociation by the treatment of myocytes with pertussis toxin prevents this negative effect, suggesting that it is the ability of the β₂-AR to couple to Gi as well as Gs that underlies the response. When a G-protein coupled receptor couples to different G-proteins, the order of affinity of agonists/antagonists can be altered. β₂-AR-blockers that are antagonists at β₂-AR-Gs may also be agonists at β₂-AR-Gi.

Additional evidence for this is provided by animal models, in which overexpression of either the β₂-AR itself or of Giα2 (mouse, rat, rabbit; transgenic or adenoviral overexpression) produces myocytes which respond to β-blockers in the same way as the failing human heart. In these models, acute exposure will produce a negative inotropic effect but, interestingly, chronic exposure to antagonists (including carvedilol) improves myocyte contraction.

β-blockers, particularly those binding to the β₂-AR, may therefore have multiple actions in addition to their effect as catecholamine antagonists. These findings have implications for the choice and design of β-blockers in heart failure, both with respect to their initial tolerability and to their final ability to remodel the signalling pathways in the failing human heart.
The concept that the failing heart is energy starved is several decades old, but has recently found major renewed interest. The heart consumes about 6 kg of ATP per day, and it is therefore obvious that “power house failure” would lead to “mechanical failure”. Large clinical heart failure trials published over the past 15 years have consistently shown that any treatment that is energy costly to the heart worsens prognosis, whereas any treatment that is energy sparing improves prognosis. There are, in principle, three aspects to energy metabolism:

- substrate utilisation
- mitochondrial function
- downstream mechanisms of ATP transport and utilisation.

All of these can become limiting steps in heart failure. Furthermore, the thermodynamic concept of free energy change of ATP hydrolysis suggests that the free energy change has to be kept above a threshold of –52 KJ/mol, and thus the free ADP concentration has to be kept low, which is a major function of the creatine kinase system. Many experimental studies have described the characteristic energetic phenotype of the failing heart, which exhibits up to 70% reductions in phosphocreatine (PCr) and free creatine, no or mild reductions in ATP, up to a 50% reduction in mitochondrial creatine kinase activity and up to a 90% reduction in ATP transfer via creatine kinase. The free energy change of ATP hydrolysis may also be substantially lower in heart failure.

Genetically manipulated mouse models allow us to dissect the role of various pathways involved in cardiac energetics. Creatine kinase and guanodinoacetate methyl transferase knockout mice show a functional and energetic cardiac phenotype consistent with a crucial role for energy metabolism. $^{31}$P-Magnetic resonance spectroscopy allows the analysis of cardiac energetics in humans. The PCr/ATP ratio is an index of the energetic state of the heart. This ratio is substantially reduced in heart failure, correlates with ejection fraction, and is also a strong predictor of patient prognosis. Disease states that predispose to heart failure, such as diabetes and obesity, also show impaired cardiac energetics. Energetics may be a specific therapeutic target in heart failure and, again, substrate utilisation, mitochondrial function and high-energy phosphate stores and availability may all be targeted. A number of experimental and clinical studies suggest that this may be feasible, and large-scale clinical trials of this concept are now warranted.
Scar extent as a determinant of adverse left ventricular remodelling in chronic heart failure

Nikolay Nikitin

Department of Cardiology, Academic Unit, University of Hull, Kingston-upon-Hull

In chronic heart failure (CHF), beta-blockers and angiotensin-converting enzyme (ACE) inhibitors improve left ventricular systolic function, but not all patients respond. We sought to establish whether scar extent, assessed by cardiac magnetic resonance (CMR) with delayed enhancement (DE), can determine the risk of persistent adverse left ventricular remodelling despite standard pharmacological treatment. Thirty-eight patients with CHF due to left ventricular systolic dysfunction who were already receiving treatment with ACE inhibitors/angiotensin II receptor antagonists and beta-blockers underwent CMR with DE, which was repeated after 12 months. DE on CMR (interpreted as scar tissue) was present in 24 patients. The scar mass was 24±9 g (range 9–43 g) and scar extent (percentage of left ventricular mass) was 17±7% (range 6–31%). At baseline, there were no differences in clinical or CMR-derived indices between patients showing DE (DE-positive group) and no DE (DE-negative group). After 12 months, left ventricular volumes and ejection fractions were unchanged in the DE-positive group. In the DE-negative group, however, left ventricular end-diastolic and end-systolic volumes had fallen (from 231±83 to 196±71 ml, p=0.018, and from 161±74 to 120±68 ml, p=0.007, respectively) and ejection fraction had increased (from 32±11 to 41±11%, p=0.003). No DE-negative patient showed a deterioration in ejection fraction of 5% or more. Eight DE-positive patients who demonstrated a deterioration in ejection fraction had higher scar extent than 17 DE-positive patients with no deterioration (22±8% versus 14±3%, p=0.03). There was a strong negative correlation between scar extent and change in ejection fraction (p<0.001, r=−0.77). In conclusion, in patients with left ventricular systolic dysfunction, scar extent predicts the risk of persistent adverse left ventricular remodelling despite conventional pharmacological treatment.
The MAGIC trial is a randomised, double-blind study of autologous, cultured skeletal myoblasts that are injected at the time of coronary artery bypass grafting into previously infarcted tissue. The trial aims to establish proof of concept and provide preliminary dose justification and safety data. The primary endpoint is the echocardiographic assessment (in a core laboratory) of global and regional wall motion of the injected territory. The study is operational in Europe (soon in Canada) and 83 patients have been treated.
BEAUTIFUL

Henry Dargie
Clinical Research Initiative, Cardiac Department, Western Infirmary, University of Glasgow, Glasgow

Abstract not available at the time of going to press
Background: The Cardiac Resynchronization – Heart Failure (CARE-HF) study demonstrated that the benefits of cardiac resynchronisation therapy (CRT) on morbidity and mortality are additional to those of angiotensin-converting enzyme (ACE) inhibitors, beta-blockers and aldosterone antagonists in patients with advanced heart failure due to left ventricular systolic dysfunction and dyssynchrony. In March 2004, the Data Safety and Monitoring Committee recommended extension of the trial until May 2005 without providing further information. However, the Steering Committee decided to close the study as planned with follow-up to September 2004, and to create an extension phase with a primary endpoint of all-cause mortality.

Aim: To investigate the long-term effects of CRT on mortality in patients with advanced heart failure and cardiac dyssynchrony despite optimised pharmacological therapy.

Results: A total of 409 patients were assigned to CRT and 404 to the control group. By the end of the extension phase, all patients will have been followed for 2 years and the mean follow-up will be approximately 3 years. The main study reported a reduction in mortality with CRT (hazard ratio 0.64; 95% confidence interval 0.48–0.85; p<0.002). On 7 March 2005, investigators were informed of these results and requested to ensure that all appropriate patients were offered CRT. Investigators were asked to complete the planned extension phase in May 2005.

Approximately 250 deaths will have occurred by the end of the extension phase. Deaths will be classified according to mode and cause, as in the main study. Although many patients may have received a CRT device by May 2005 this will have had little impact on differences in survival between groups. This analysis will demonstrate whether the large benefit in mortality observed in the main study increases with longer follow-up. Data on the risk of sudden death and death from worsening heart failure will also be presented, particularly focusing on potential differences between modes of death between treatment groups.
ESSENTIAL

Henry Dargie
Clinical Research Initiative, Cardiac Department, Western Infirmary, University of Glasgow, Glasgow

Abstract not available at the time of going to press
Previous studies have suggested that levosimendan may reduce the mortality associated with severe and/or acute heart failure compared with placebo (RUSSLAN and CASINO) or dobutamine (LIDO and CASINO). SURVIVE was designed to compare the effects of a (single) 24-hour infusion of levosimendan or dobutamine in patients with acute heart failure (either new-onset or acute on chronic) and left ventricular systolic dysfunction on mortality over the subsequent 6 months. REVIVE was designed to compare the effects of a levosimendan with placebo on a composite clinical endpoint in patients with a hospital admission for worsening chronic heart failure. The results of both studies, which will include over 2000 patients, will be presented at the AHA.
Abstract not available at the time of going to press
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British Society for Heart Failure
8th Annual Autumn Meeting 2005

Friday 25 November 2005
Queen Elizabeth II Conference Centre, London
09.45 – 18.45

An Educational Symposium Supported by Takeda UK Ltd: 17:30 – 18:45

Chronic Heart Failure Management: Sharing the Care – Regional Experiences

Co-Chairs:
Nigel Rowell (Middlesborough) & Kirstin Russell (Glasgow)

Speakers:

• Judith Walker & Fiona Lough (London): British Association for Cardiac Rehabilitation (BACR) - Heart Failure Special Interest Group

• Jackie Austen (Abergavenny): Cardiac rehabilitation in older patients with heart failure - The Gwent Experience

• Janet Reid (Edinburgh): Working in partnership with voluntary organisations to meet the needs of patients & their carers - The Lothian Experience

• Jenny Welstand & Kate Evans (Wrexham): Multidisciplinary approach to heart failure management - The Wrexham Experience

Attendees of this final session of the day will be entered into a draw to receive one of two educational grants from the sponsor to attend next year ESC Heart Failure 2006 Congress in Helsinki.

Endorsed by: BRITISH SOCIETY FOR HEART FAILURE