

# Newsletter

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This issue reports highlights from the British Society for Heart Failure (BSH) sessions at the British Cardiovascular Society (BCS) Annual Scientific Conference, held in Manchester 2–4 June 2008.

The BSH was involved in seven conference sessions, several of which were organised jointly with other affiliated groups of the BCS.



## Forthcoming BSH event in 2008

### 11<sup>th</sup> BSH Annual Autumn Meeting

#### Heart failure in transition

20–21 November 2008  
Queen Elizabeth II  
Conference Centre,  
Westminster, London

## Cardiovascular aspects of pregnancy

### Joint session with the British Congenital Cardiac Association and Heart Care Partnership UK

In a session on cardiovascular diseases that can complicate pregnancy, Dawn Adamson (London) pointed out that cardiac disease is the most common cause of maternal mortality in the UK. The latest data from the Confidential Enquiry into Maternal and Child Health (CEMACH)<sup>1</sup> show that maternal deaths from cardiac disease have increased over the past 15 years and this is due to an increase in acquired disease.

Preconception advice is essential for women with a pre-existing serious medical condition that may be aggravated by pregnancy. CEMACH also now recommends preconception counselling for women at higher risk of developing cardiac disease in pregnancy: those who are obese, who smoke, have hypertension and/or diabetes, or a family history of heart disease, or are aged >35 years. "Since up to 50% of pregnancies are unplanned, you should not wait for people to come to you for pre-pregnancy counselling," Dr Adamson commented.

There are five conditions in which pregnancy is "contraindicated" (World Health Organisation 4 classification):

- pulmonary arterial hypertension (PAH)
- severe systemic ventricular dysfunction (New York Heart Association [NYHA] functional class III–IV or left ventricular ejection fraction <30%)
- previous peripartum cardiomyopathy with any residual left ventricular impairment
- severe left heart obstruction
- Marfan syndrome with aorta >40 mm diameter.

If pregnancy is not contraindicated, the woman needs advice on the risks of the pregnancy on her condition and her condition on the pregnancy, and whether any drugs she takes, such as warfarin, statins and antiarrhythmics, should be stopped prior to conception. For women with a metal valve, warfarin is best for the mother but heparin is best for the baby. Embryopathy risk is lower at a low warfarin dose, so continuation of warfarin might be considered if the woman is stable on a low dose. If swapping to heparin, it is best to do this by 6 weeks to minimise foetal risk.

### Pulmonary arterial hypertension

Sara Thorne (Birmingham) emphasised that women with PAH must be advised to avoid pregnancy and termination should be discussed if pregnancy occurs.

PAH is associated with very high maternal mortality because the heart and pulmonary circulation cannot meet the physiological demands of pregnancy. The mortality rate is unclear but is generally quoted as 40%. Problems can occur at any stage in pregnancy.

If a woman with PAH becomes pregnant and cannot or will not terminate the pregnancy, specialist multidisciplinary management is required. Pulmonary arterial vasodilator therapy may increase chances of survival. The best experience to date comes from Sheffield where in 14 consecutive pregnancies in women with PAH there were five terminations/miscarriages and nine completed pregnancies, with no deaths.

The Sheffield approach is to stop bosentan if possible, because of its embryopathic effect, and change to inhaled iloprost. If necessary, sildenafil may be used and, as third-line, intravenous prostacyclin or bosentan. Low-molecular-weight heparin is also given.

### Contraception in high-risk women

For patients with heart disease in whom pregnancy must be avoided, barrier methods of contraception are unreliable and the combined pill is contraindicated because of thrombogenic risk. Cerazette® (desogestrel 75 µg) has better efficacy than other progestogen-only pills. Even better are long-acting progestogens, such as the intra-uterine progestogen-only system (Mirena®) and implant (Implanon®) – these are more effective than sterilisation. However, Mirena is not a good option for women with PAH, because of the risk of a vasovagal reaction on insertion.

Special advice is needed for women taking bosentan as this is an enzyme inducer and can affect the efficacy of some progestogen preparations, including Implanon. Depo-Provera® is not affected by bosentan and is a good option.

### Postpartum cardiomyopathy

Peri- or postpartum cardiomyopathy (PPCM) is a condition of unknown aetiology in which symptoms of heart failure occur between the last month of pregnancy and 5 months postpartum. In Western Europe and the USA, the condition occurs in up to 1:1350 pregnancies, but in Africa it is between 1:100 and 1:1000, and in Haiti 1:300.

The condition can progress rapidly and mortality is 10–25% despite standard therapy for heart failure. Recovery of normal cardiac function occurs in less than 30% of patients.

There is currently no specific treatment for PPCM. But Denise Hilfiker-Kleiner (Hanover, Germany) said there is now evidence that bromocriptine might be useful, either for preventing PPCM in high-risk pregnancies or as early treatment for acute PPCM. There have been promising results in individual cases and a randomised controlled trial is underway.

Interest in bromocriptine, a prolactin inhibitor, stems from her team's studies in transgenic mice suggesting that PCCM may be related to increased production of a harmful form of prolactin. Their hypothesis is that increased oxidative stress in late pregnancy leads to enhanced activity of cardiac cathepsin D enzyme. This, in turn, is associated with increased cleavage of



Denise Hilfiker-Kleiner: her studies have led to investigation of bromocriptine use in postpartum cardiomyopathy

the dominant 23 kDa form of prolactin, an angiogenic protein, to 16 kDa prolactin, which (in the mouse model) has antiangiogenic properties, impairs the cardiac microvasculature and reduces cardiac function.

Dr Hilfiker-Kleiner said that the major risk with PPCM is delayed diagnosis, with gynaecologists and general physicians not suspecting cardiac disease in young women. This point was reinforced by Susan Sneddon, from Edinburgh, who presented a patient's viewpoint. Her initial symptoms of PCCM – swelling in her hands and feet, and severe breathlessness – were seen as normal symptoms related to her twin pregnancy, and the condition was not immediately diagnosed even when her symptoms worsened after delivery.

### Late cardiovascular complications of pre-eclampsia

The association between pre-eclampsia and later cardiovascular disease was discussed by Jill Pell (Glasgow). A 2007 meta-analysis of around 200,000 pregnancies involving pre-eclampsia<sup>2</sup> showed a relative risk (RR) of 2.16 for later ischaemic heart disease events. There was also an association with hypertension and stroke.

Risk of ischaemic heart disease is particularly high with early onset pre-eclampsia (before week 37) (RR=8) and is also increased with more severe pre-eclampsia, pre-eclampsia in serial pregnancies and when the pregnancy is also associated with intrauterine growth restriction or preterm delivery.

Absolute risk increases with time, as the baseline cardiovascular risk increases. Professor Pell said: "Having pre-eclampsia does not have a great impact early on, but in population terms, in much later age groups, it is associated with a huge increase in absolute number of events."

The two main hypotheses of why pre-eclampsia is associated with future cardiovascular disease are shared predisposition (different manifestations of the same disease process) or causal link (with pre-eclampsia causing persistent metabolic, inflammatory and vascular changes that predispose to later coronary heart disease). Whichever theory holds, women who have had pre-eclampsia are an obvious target group for primary prevention and could be monitored for early signs of cardiovascular disease.

## Update on left ventricular non-compaction

Left ventricular non-compaction (LVNC) is a “new” condition, first described in 1984. There is still considerable uncertainty over its diagnosis and management.

Perry Elliott (London) said that non-compaction is currently being overdiagnosed. This is largely because diagnostic criteria are not robust and there is failure to distinguish normal and abnormal trabecular patterns. “There clearly is an entity of hypertrabeculation which is pathological and also a range of normal trabeculation. The diagnostic challenge is in the ‘grey area’ in between. We haven’t yet defined the upper limit of normal trabeculation,” he said.

LVNC, an “unclassified” cardiomyopathy, is a developmental abnormality. Dr Elliott explained that in embryonic development the heart goes through a period of hypertrabeculation. As development progresses these trabeculae disappear, leaving a residual thin trabeculated surface and a thick compact layer. LVNC is a failure of that process, with characteristic features of

deep trabeculae and marked hypertrabeculation, particularly in the posterolateral segments.

It is now recognised that LVNC, like other cardiomyopathies, is probably a familial disorder caused by many different genetic abnormalities. Importantly, there appears to be overlap with mutations in genes associated with dilated cardiomyopathy and hypertrophic cardiomyopathy, in particular the genes coding for sarcomeric proteins that are expressed at an early stage of cardiac development.

Dr Elliott emphasised the need to keep an open mind when assessing family members because of the range of different phenotypes associated with LVNC. There is no single clinical scenario and different patterns of LVNC can occur in thick hearts, dilated hearts and otherwise normal hearts.

Alison Duncan (London) said that echocardiography can be used to diagnose LVNC, but this is complicated. There are at least three different echo definitions for non-compaction, each of which is based on only small patient numbers.

The most commonly used echo definition (the Jenni criteria) depends on identifying two distinct layers of myocardial wall (compacted [C] and non-compacted [NC]) on short axis view. These are expressed as a ratio with NC:C >2 being diagnostic for non-compaction. The Jenni criteria also require demonstration of blood flow into the intertrabecular recesses using colour Doppler.

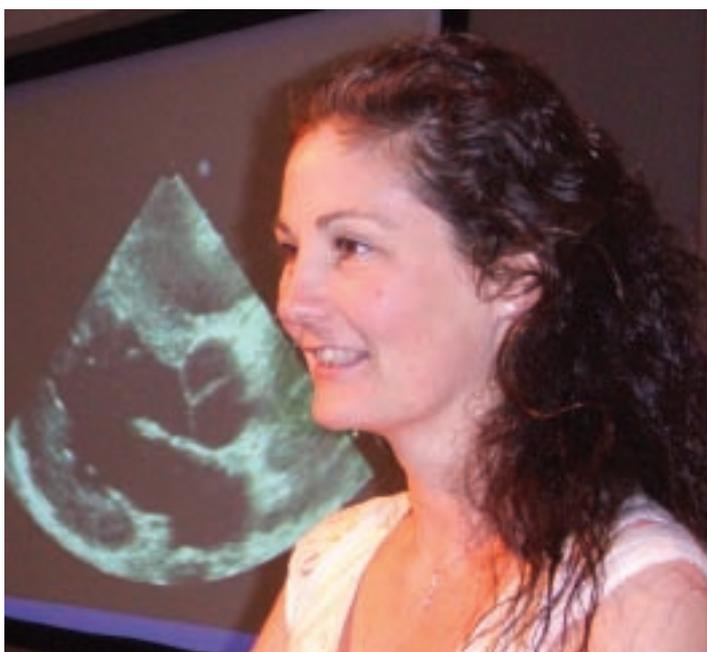
Many patients diagnosed with LVNC go on to develop “other” cardiomyopathies, particularly dilated cardiomyopathy. “There’s a school of thought that in diagnosing non-compaction on echo what we are doing is seeing the first stage of something that is going to progress to another more recognisable type of cardiomyopathy,” Dr Duncan commented.

Echo can help with differential diagnoses. There are many of these, but the most important are:

- normal variant (up to 70% of people have trabeculated hearts)
- apical hypertrophic cardiomyopathy
- arrhythmogenic right ventricular dysplasia.

Once a diagnosis of non-compaction is made, a real strength of echo is the ability to assess risk by looking for the presence of dyssynchrony, and for specific complications, such as ventricular thrombi, for which there is increased risk because of stasis of blood in the trabecular recesses.

Sanjay Prasad (London) explained how cardiac magnetic resonance (CMR) imaging can complement echo assessment. It can help distinguish whether there is true non-compaction or whether the condition is being confused with, or is concurrent with, other cardiomyopathy, thrombus or localised hypertrophy.



Alison Duncan discussed the use of echocardiography to diagnose left ventricular non-compaction

The CMR technique of late gadolinium enhancement can be used to look for thrombi and also for scar tissue (fibrosis).

Little is known yet about the prognostic value of CMR imaging in LVNC. It would be useful to collect registry data on these patients, and it is also important to establish the significance of non-compaction in asymptomatic patients, Dr Prasad said.

## Treatment implications

It may ultimately be possible to identify high-risk individuals with non-compaction and to intervene to prevent development of heart failure.

For now, said Theresa McDonagh (London), there is no specific evidence-based treatment for LVNC, and management involves standard heart failure treatment (both for left ventricular systolic dysfunction [LVSD] and asymptomatic systolic dysfunction), assessing arrhythmia risk and prophylaxis of thromboembolism.

In the largest and most modern series, involving 67 cases of non-compaction,<sup>3</sup> 66% of patients had LVSD at presentation, 12% of patients were asymptomatic, 55% had breathlessness and 6% presented with stroke. At 30-month follow up, 15% had died (one-third from progressive heart failure and two-thirds from sudden cardiac death). Outcome was better than in previous

series, probably because heart failure treatment is improving, and a broader spectrum of severity of cases was included.

In addition to standard heart failure drugs, cardiac resynchronisation therapy (CRT) might be considered. The CARE-HF (Cardiac Resynchronisation in Heart Failure) study population presumably included some patients with non-compaction. Also, a recent retrospective series of eight patients with LVNC indicated that CRT response is similar to that in patients with heart failure due to ischaemic or dilated cardiomyopathy. Indications for the use of an implantable cardioverter defibrillator (ICD) are likely to be the same as for other non-ischaemic cardiomyopathies. However, 2008 guidelines from the American College of Cardiology/American Heart Association/Heart Rhythm Society say that an ICD “can be considered” in non-compaction, indicating that there is insufficient evidence to recommend use.

For thrombotic risk, while some authors have suggested use of warfarin for all patients with non-compaction, more recent studies have used aspirin routinely, reserving warfarin for patients at greatest risk. However, one study using this schedule<sup>3</sup> reported thromboembolism in 9% of patients, a rate which Dr McDonagh suggested was difficult to accept. Her minimum treatment for a patient with non-compaction and LVSD would probably be an angiotensin-converting enzyme (ACE) inhibitor, beta-blocker and warfarin.

# Tracking changing prognosis in heart failure

## Joint session with the British Nuclear Cardiology Society and British Society of Echocardiography

Biomarkers, echocardiography, nuclear techniques and CMR were all covered in a session on ways of tracking changing prognosis and tailoring therapy in heart failure.

Roy Gardner (Glasgow) said there is good evidence for use of B-type natriuretic peptide (BNP) as a prognostic marker. Using this biomarker should help with patient management since changes in BNP concentration are a marker of risk: if the concentration is high and fails to fall, evidence suggests that the patient is at particularly high risk.

Treatment monitoring using BNP also looks promising. In STARS-BNP (Systolic Heart Failure Treatment Supported by BNP), patients with NYHA II–III heart failure received treatment according to current guidelines or BNP-guided treatment (to target BNP level). Dosage of ACE inhibitors and beta-blockers was higher in the BNP-guided group. At 15-month follow-up, significantly fewer patients (24% vs 52%,  $p < 0.001$ ) had reached endpoint (chronic heart failure-related death or hospitalisation) in the BNP group.<sup>4</sup>

The larger ongoing BATTLE-SCARRED (BNP-Assisted Treatment to Lessen Serial Cardiac Readmission and Death) study is also assessing BNP-guided treatment. The majority of patients

recruited so far have NYHA class II heart failure, and are aged >75 years – they therefore represent a “real world” population and the results are awaited with interest.

Discussing how echocardiography can be used for prognosis and to tailor treatment, Simon Ray (Manchester) said that his practice is to measure left ventricular volumes and ejection fraction (using the Simpson rule where possible), left arterial volume, mitral E wave and deceleration time; and E/Ea (E/E') on tissue Doppler.

These measurements, taken at baseline and follow-up, provide sound grounds for making decisions about treatment. But echo parameters should not be used in isolation and the likely way forward is to use a combination of E/Ea and measurement of BNP.

The potential value of this combination was shown in a community-based study published earlier this year<sup>5</sup> involving 228 elderly patients with newly diagnosed heart failure in which cardiovascular events were assessed over 18 months. Patients with low BNP at baseline had good prognosis, a group with high BNP but relatively low E/Ea had intermediate prognosis, whereas those with elevated BNP and elevated E/Ea did very badly.

Alexander Jacobsen (Cambridge) said that nuclear techniques offer a targeted investigation in heart failure. He outlined two new research techniques that have excited considerable clinical interest. One is cardiac neurotransmission imaging using <sup>123</sup>I-MIBG, a partial agonist for the adrenergic receptor. This can be used to quantify the extent and severity of regional neuronal injury. Its prognostic value in heart failure is promising but further trials are needed to support clinical use. The technique may also be used to also monitor and predict response to therapy, including CRT.

Another new tracer is <sup>123</sup>I-BMIPP, a sensitive potential marker for aberrations in cardiac metabolism. This can be used to identify viable but damaged myocardium.

Where does CMR imaging fit in? Sanjay Prasad (London) said that one of the areas where this technique has had the biggest

impact in gauging prognosis is in the evaluation of scar (fibrous) tissue using the technique of late-gadolinium enhancement. Gadolinium is a paramagnetic contrast agent: it accumulates in areas of scar tissue and can detect 1 mm of scar.

This test can identify patients who need more intensive therapy; for example, those with little scar but substantial hibernating myocardium. It can also investigate non-response to CRT, since placing leads in an area of scar tissue will not generate response and may even have a deleterious effect.

Presence of scar tissue is a driver for re-entry arrhythmias and sudden cardiac death. Identifying scar, for example, in a patient who has had a silent myocardial infarction (MI), might therefore help select patients who need an ICD. Gadolinium enhancement can also detect microvascular ischaemia and capillary damage in acute heart failure.

## Hypertension and heart failure

Joint teach-in session with the Primary Care Cardiovascular Society

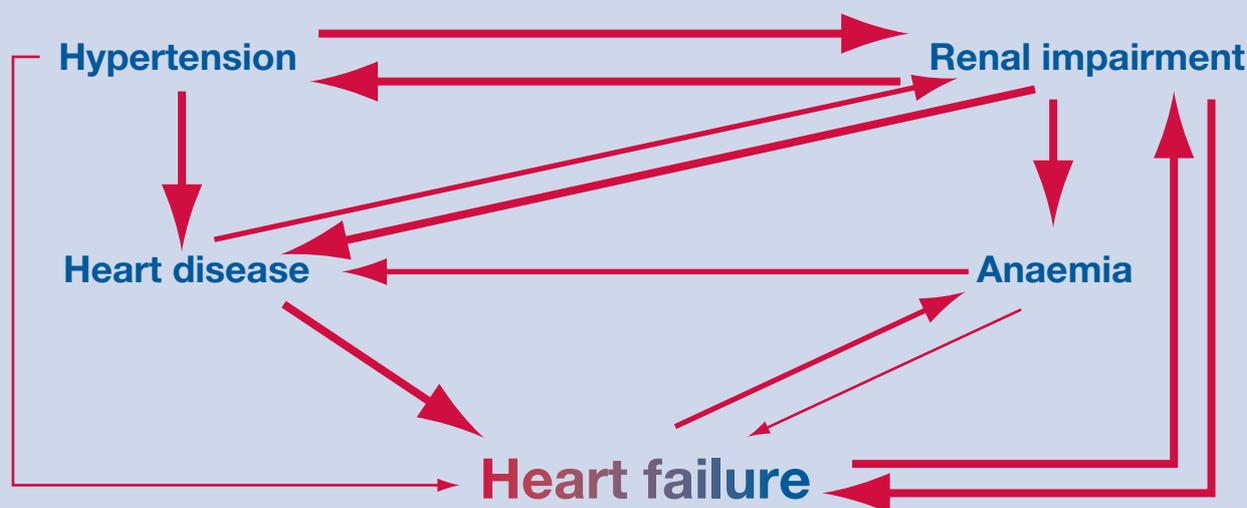
Speakers: Richard Hobbs (Birmingham), John Sanderson (Birmingham), Rosaire Gray (London), Henry Dargie (Glasgow)

Hypertension is a major risk factor for heart failure, largely because it is a driver of coronary heart disease. Treating hypertension is associated with a significant reduction in heart failure and the challenge for primary care is better screening of the population to detect disease and better management strategies to achieve blood pressure targets.

There is a view, particularly in USA, that heart failure with normal ejection fraction (HFNEF) and systolic heart failure are

two separate conditions, and that left ventricular hypertrophy (LVH) is not a precursor of systolic heart failure. This is not the case, said John Sanderson: there is a single syndrome of heart failure – a continuum – with some patients progressing from HFNEF to typical LVSD. This process is accelerated by MI and diabetes, and also by alcohol.

Treatment should be aimed at reversing LVH and preventing ventricular remodelling to try to stop this progression.



Asked about measuring diastolic function, Professor Sanderson said that BNP measurement might come in useful here. There is certainly a trap in assuming that a patient with “normal ejection fraction” does not have heart failure. GPs should ask the echo lab if they have looked at:

- evidence of LVH – which would raise suspicion of heart failure
- left atrial size – a good measure of raised left atrial pressure over time
- long axis function – a sensitive measure of left ventricular function.

Laboratory experiments are providing a clue to the intracellular mechanism by which hypertension is associated with heart failure. Altered intracellular ion regulation, specifically raised intracellular sodium, is seen in a guinea pig model of LVH and could explain the increased arrhythmia risk associated with LVH.

There are complex associations between hypertension, kidney disease and heart failure. Renal dysfunction is common in heart failure, and *vice versa*.

All heart failure treatments (diuretics, ACE inhibitors, beta-blockers, spironolactone and angiotensin receptor blockers [ARBs]) can cause or aggravate renal dysfunction either directly through specific mechanisms or indirectly by reducing renal blood flow. However, it is important to try to keep the patient on these drugs where possible.<sup>6</sup>

The use of diuretic plus ACE inhibitor plus ARB plus spironolactone plus beta-blocker is challenging and requires close monitoring. Not uncommonly, treatment may have to be temporarily reduced (e.g. intercurrent illness) and occasionally it may be necessary to completely stop all “lifesaving” heart failure medicines to let the kidney recover, after which the drugs can be cautiously reintroduced.

## Cognitive dysfunction in heart failure

### Joint session with the British Geriatric Society-Cardiovascular Section

*Speakers: Duncan Forsyth (Cambridge), Jackie Taylor (Glasgow), John Baxter (Sunderland), John Starr (Edinburgh)*

Cognitive dysfunction in elderly patients with heart failure is common, under-recognised and has an important impact on management. Epidemiological data suggest that patients with heart failure have twice the likelihood of developing cognitive dysfunction as age-matched controls.

Any patient who appears confused should have cognitive assessment. An easy and quick way of screening is to use the MMSE (Mini-Mental State Examination) and CLOX-1 (a clock drawing task). CLOX-1 is useful because, unlike the MMSE, it assesses frontal executive function. The patient is asked to “draw a clock as a child would recognise it with the numbers inside and the hands showing [specified time].”

Differential diagnoses in patients with cognitive dysfunction include depression, delirium and dementia. In inpatients, delirium should be assumed until proven otherwise, with possible causes including infection, drugs, electrolyte disturbance and heart failure *per se*.

The best way to find out if a patient is depressed is simply to ask: “Do you feel depressed?” Citalopram or mirtazapine are favoured antidepressants; the latter has an appetite-stimulating effect and so is useful if the patient has problems with appetite.

Cognitive impairment impacts on the identification of heart failure because patients present with less-specific symptoms, in part due to a difficulty in explaining symptoms. It also limits patients’ capacity to understand complex treatment regimens, and leads to difficulty with self-management. Cognitive impairment is associated with increased disability and increased mortality in elderly heart failure patients: these patients are a frail and high-risk group who need a specific type of heart failure management.

### What is the link?

The traditional view is that cerebral hypoperfusion is the major factor linking heart failure and cognitive dysfunction. But John Starr suggested that while this may be the case in patients with advanced heart failure, especially if surgery or a procedure such as cardiac catheterisation is being carried out, there is little convincing evidence of a link in most patients with heart failure. Microemboli are also an unlikely cause of cognitive dysfunction unless a cardiac procedure is being carried out.

More likely, he suggested, are hormonal/cytokine effects. For example, BNP has widespread receptor distribution in the brain and there is evidence of a possible link with cognition. Pro-inflammatory cytokines may also be relevant.

# Advanced heart failure: optimum clinical care includes embedded supportive care

Joint session with National Council for Palliative Care and Health Care Partnership UK

Speakers: James Beattie (Birmingham), Yvonne Millerick (Glasgow), Miriam Johnson (Scarborough), Suzanna Hardman (London)

Provision of palliative care for patients with advanced heart failure is improving, although many barriers remain. Current challenges include problems at point-of-care interfaces, deciding when to turn off devices, and how to ensure patient preferences are met and goals are reappraised as the disease progresses. A paper published last year identified 17 different models of heart failure end-of-life care in the UK – hospital-, community- and hospice-based – with good examples of collaboration between palliative-care physicians and cardiologists.

A heart failure palliative care project has been set up in Glasgow, funded by the British Heart Foundation, in which heart failure nurses work with health- and social-care professionals to produce a written management plan for patients with advanced heart failure who are progressively symptomatic despite optimal treatment. There is a fast-track process for patients who require hospitalisation for intravenous diuretic.

Patients in Glasgow are electronically “flagged” in the out-of-hours system to alert staff that the patient requires only palliative care. In Scarborough, the importance of a problem-based approach rather than a prognosis-based approach is emphasised.

Suzanna Hardman suggested there is perhaps too much emphasis on the need to tell patients they will die from their heart failure, rather than optimising their heart failure care, which will both alleviate symptoms and improve mortality. Many patients are very elderly and a future cause of death, always uncertain, may not be their prime concern. For some, self-management is an important strategy. These patients fare better



Martin Cowie (left), chair of the BSH, and Michael Connolly (Manchester), who jointly chaired the advanced heart failure session

as they learn to control the condition, yet this may be undermined by contradictory advice and overemphasis on the inevitability of a heart failure death.

Quantity and quality of life improvements are not mutually exclusive for people with heart failure. Yet too often patients are not getting an early accurate diagnosis, or seeing a cardiologist, or receiving optimised pharmacological treatment, all of which can improve mortality and ameliorate symptoms.

## Echo or ECG to select for CRT?

Joint session with British Society of Echocardiography

The question of whether echocardiography should be used to identify patients most likely to respond to CRT is controversial. National Institute for Health and Clinical Excellence (NICE) guidelines say echo should be used to assess dyssynchrony if the QRS duration is between 120 msec and 149 msec. But European Society of Cardiology guidelines say patients should be selected on the basis of the ECG, and several recent papers have argued against echo measurements.

A debate on this led by John Sanderson (Birmingham) (pro) and Andrew Clark (Hull) (against) showed the majority of the audience to be in favour of using echo. Professor Sanderson

suggested that a combination of clinical factors, new echo techniques and magnetic resonance imaging criteria may help identify those most likely to benefit, though there is no conclusive evidence yet for this approach. Dr Clark pointed out that before CARE-HF none of the trials demonstrating improved prognosis with CRT used echo as entry criteria, and in CARE-HF echo was used in only 92 patients.

### Improving response to CRT

There are complex reasons for failure to respond to CRT, said John Cleland (Hull). These include failure to deliver effective

therapy, inability of the patient to respond and choice of criteria for declaring success or failure (a major problem in interpreting studies).

It is important not to confuse outcome and response, he said. A patient who has a poor outcome with CRT might have had a much worse outcome without CRT. He would advise patients who are considering CRT that most people do get some benefit (though this may not be immediate), and that while some patients feel they have not benefited from the device there is a chance they would have deteriorated without it.

He suggested that clinicians should concentrate on implanting CRT devices in patients who fit NICE criteria, and then maximise response by good post-implantation management. "This is where I think most of our effort should go, rather than in trying to select patients beyond what the clinical trials have told us to do."

Rakesh Sharma (London) said that post-implantation monitoring involves attention to fluid status, titration of medicines, a pacemaker check and echocardiography to optimise pacing parameters.

The haemodynamic response to CRT requires adjustment of a patient's drug therapy. Diuretic requirements may reduce within a few days of implantation. Failure to recognise this means the patient develops pre-renal failure, masking the benefit from CRT. There is also an opportunity to improve patients' heart failure medicines: it may be possible to increase ACE inhibitor dose, and beta-blockers can sometimes be restarted in patients who were previously intolerant to them.

Co-morbidity is a major reason for non-response and is often forgotten when patients are being assessed for CRT. For example, in a patient with brittle chronic obstructive pulmonary disease it is unlikely that CRT will make the patient less breathless. Renal failure is associated with a higher risk of non-response, though there are limited data on this because trials have excluded patients with severe renal disease. Also, pacing can potentially make symptoms worse in a patient with active cardiac ischaemia. At the Brompton Hospital, CMR imaging is done before CRT implantation to check perfusion and to look for scar tissue.

## National Heart Failure Audit

The BSH is encouraging clinicians to participate in the National Heart Failure Audit of patients coming into hospital with heart failure. This is being carried out in conjunction with the Healthcare Commission. Results will inform the Trust's "star" rating, and will be a useful lever for clinicians wishing to highlight areas for improvement in heart failure care.

More information is available at: [www.ic.nhs.uk/heartdiseaseaudits](http://www.ic.nhs.uk/heartdiseaseaudits)

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## Becoming a Member or a Friend of the BSH

Membership is open to anyone involved in the diagnosis, management or science of HF. If you are interested in becoming a Member or Friend of the BSH, please contact:

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