BRITISH SOCIETY FOR HEART FAILURE

Newsletter

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This issue reports highlights from the heart failure-related sessions at the British Cardiovascular Society (BCS) Annual Conference and Exhibition, held in Manchester on 7–9 June 2010. The British Society for Heart Failure (BSH) was involved in three conference sessions, which were organised jointly with other affiliated groups of the BCS. There was also a symposium in memory of Professor Philip Poole-Wilson, entitled "From the cell to the bedside".

Forthcoming events

13th BSH Annual Autumn Meeting
25–26 November 2010
Queen Elizabeth II Conference Centre, Westminster, London

BSH Medical Training Meeting 2011
9 February 2011
NHLI, London

BSH Nurse Training Meeting
10 February 2011
NHLI, London

Cancer and the heart

Joint session with the British Nuclear Cardiology Society

Speakers in a session on the cardiac toxicity of cancer drug therapy and radiotherapy agreed that better monitoring strategies are needed to identify long-term toxicity.

Perry Elliott (London) discussed the impact of chemotherapy on the heart. He said that the incidence of cardiac toxicity is variable but severe toxicity is probably uncommon.

Attention tends to focus on the impact on left ventricular function, but other cardiovascular toxicity can occur, including hypertension and arrhythmia. There is also a suggestion that chemotherapy might damage the endothelium, increasing the future risk of atherosclerosis.

Dr Elliott said that data on cardiac complications of chemotherapy are limited. Most trials were of short duration, whereas toxicity can occur many years after the drug has been administered. There is also confusion in the literature on whether cardiotoxicity refers to symptomatic heart failure or to sub-clinical change.

The anthracyclines, in particular, are associated with cardiac toxicity. This toxicity probably relates to oxidative stress, with the drugs forming a potent free-radical complex. Toxicity is dependent on the cumulative drug dose and is irreversible. "The more drug you give, the more myocytes you kill," Dr Elliott said.

Symptomatic heart failure and asymptomatic left ventricular dysfunction (LVD) have been reported in 1–5% and 10–30% of patients receiving anthracyclines, respectively. Following the use of these drugs in childhood malignancies, there is a slow increase in the risk of heart failure, reaching around 5% after 15 years.

Dr Elliott said that tyrosine kinase drugs could also be toxic to the heart. Trastuzumab is a monoclonal antibody against HER-2 (human epidermal growth factor receptor 2), a tyrosine kinase receptor that is overexpressed in 25% of cases of breast cancer. Trastuzumab treatment leads to asymptomatic LVD in 5–17% of patients and to symptomatic heart failure in 1–3%. There is still debate as to whether trastuzumab toxicity is truly reversible.

Risk factors for cardiac toxicity with this drug include age, coadministration with anthracyclines and low ejection fraction (EF) after chemotherapy.

Bevacizumab, another tyrosine kinase inhibitor (TKI), inhibits angiogenesis by inhibiting vascular endothelial growth factor. There is a high incidence of hypertension with this drug.

Dr Elliott said that cardiologists perceive cardiac toxicity as a problem that is going to increase as cancer cure rates improve. His view was that the rapid pace of development of TKI drugs might lead to more toxicity, but that anthracycline toxicity might actually become less of a problem because oncologists have developed strategies to reduce toxicity. These include treatment up to a 'safe dose' and cardiac monitoring throughout therapy.

Different formulations can be used, with liposomally encapsulated doxorubicin reported to be associated with a lower incidence of toxicity than the non-lipid formulation. Dose timing is also important: cardiac toxicity is much lower if trastuzumab is given after anthracycline therapy rather than at the same time.

Regarding cardioprotective drugs, dexrazoxane is an ion chelator and might prevent the production of a free-radical complex with anthracyclines. However, its use is limited, in part because oncologists believe that some therapeutic effect of anthracyclines might be mediated through oxidative stress; hence, blocking this might reduce anticancer efficacy.

Dr Elliott suggested that the key to future progress is a better understanding of individual susceptibilities, with increased study of pharmacogenomics. This could help to identify patients at highest risk of toxicity.

Asked about treatment of LVD related to chemotherapy, he said that there was a question over this because the mechanism of damage may be different from that in other forms of heart failure. "There is some suggestion that beta-blockade with carvedilol, a free-radical scavenger, may be more effective than, say, bisoprolol. It might be that fibrosis is an important driver and so early spironolactone would be useful, but we do not know."

Monitoring patients during and after cancer chemotherapy

Mark Harbinson (Belfast) described current practice in monitoring patients for left ventricular systolic dysfunction (LVSD) and heart failure.

He said that anthracycline cardiotoxicity seems to be dose- and patient-related, impairment may be permanent and monitoring is required. But there is currently no guideline on when to start monitoring, how frequently to monitor or how far EF should be allowed to fall before intervening. Noting the expense of long-term monitoring to detect disease in a relatively small number of patients, Dr Harbinson said that it seemed logical to monitor according to the individual's risk of cardiotoxicity, which related to factors such as pre-existing cardiac disease, dose and concomitant therapy.

More guidance is available on trastuzumab monitoring, and the serial assessment of EF is required. Dr Harbinson noted that the recommendations of the National Institute for Health and Clinical Excellence (NICE) often require a patient to have six imaging investigations over a course of treatment. "So, 90–95% of women will have six investigations, which will all be normal. We need more flexibility," he commented.

In Belfast they are now using a new national protocol on trastuzumab monitoring from the UK National Cancer Research Institute (NCRI),[1] which indicates that fewer scans are needed in low-risk patients. Following this guideline, a patient with an uncomplicated course may only need four echocardiography scans.

Dr Harbinson said that the guideline also gives useful advice on when to start angiotensin-converting enzyme (ACE) inhibitor treatment in these patients and when to refer to a cardiologist (**Figure 1**).

NICE does not specify a type of imaging investigation for trastuzumab monitoring. Studies have used echocardiography,

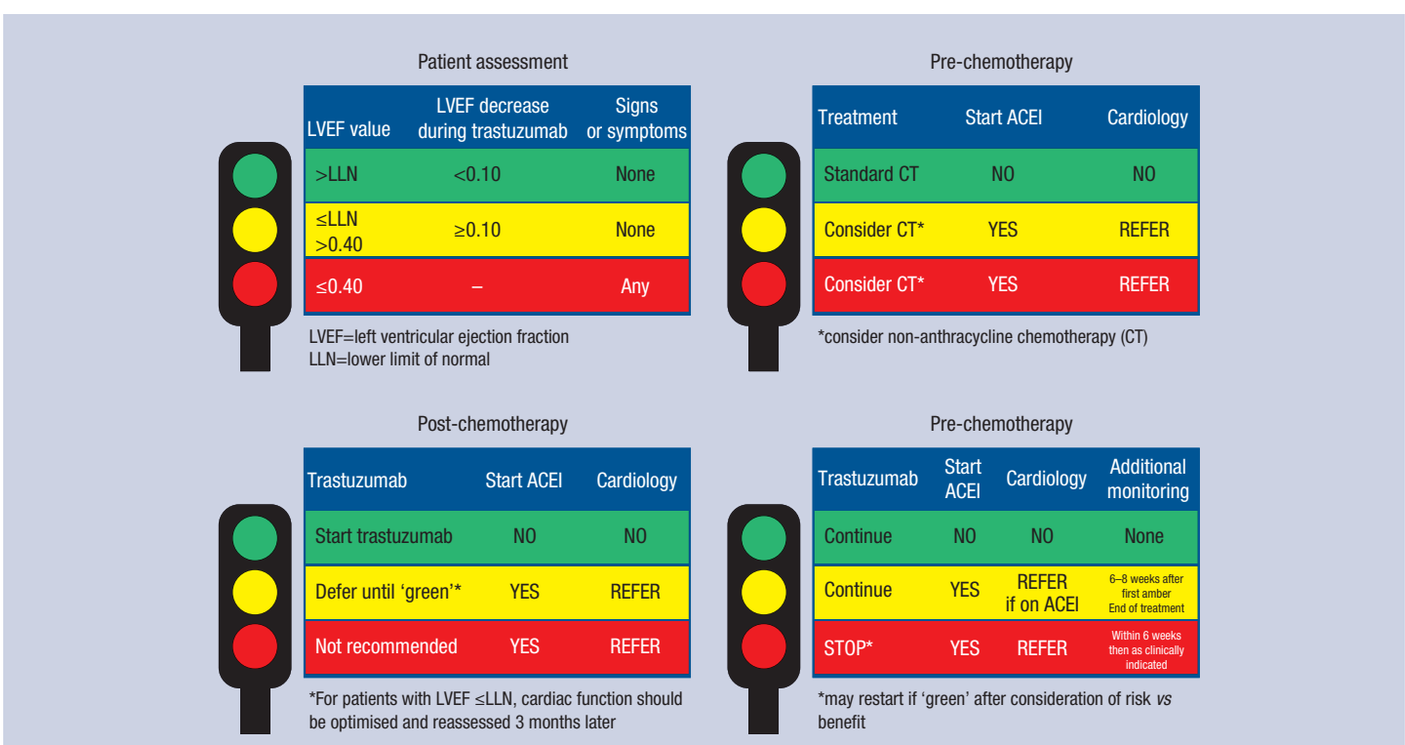

Figure 1: UK NCRI recommendations on trastuzumab monitoring.[1] ACEI = ACE inhibitor.
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Cardiac pathology and cancer

Primary tumours of the heart are extremely rare. Secondary cardiac tumours are 20 times more common, but still relatively rare, according to Mary Sheppard (London).

She said that in a series of 3314 malignant autopsies, cardiac metastases were found in 11.8%. The most common sites of the primary tumour were lung, lymphoma, breast, leukaemia, stomach, melanoma, liver and colon.

Tumours can appear as multiple nodular metastases on the heart surface, infiltrating into the myocardium, or the patient might have pericardial disease mimicking constrictive pericarditis. Intracardiac extension of the tumour can occur, with the tumour growing into the inferior vena cava and into the right atrium. Renal cell carcinoma has extended into the lumen of the inferior vena cava in 4% of patients at the time of diagnosis but it often does not infiltrate the vessel and can be removed.

Dr Sheppard said that the specialist unit at the Royal Brompton Hospital had recently published data on 116 primary cardiac tumours seen over the past 20 years.[2] The majority of these tumours (70%) were benign and were predominantly myxomas.

Tumours were more common in the left side of the heart, but right-sided tumours were more likely to be malignant. Some were found on routine echocardiography, and some patients presented with palpitation, heart failure, arrhythmia, stroke due to embolisation, chest pain or with sudden death.

Myxomas typically presented in the left atrium. Although not malignant, if not completely excised they would recur. For clinicians, it was important to differentiate myxomas from thrombi, which could have a similar size and shape.

Papillary fibroelastoma can occur in the elderly and is not really considered a tumour but is seen as degenerative proliferation of thrombi on the valve surface. It carries risk of embolisation. With increasing use of echocardiography, fibroelastomas are being diagnosed more frequently, Dr Sheppard said.

In the Royal Brompton series, malignant tumours accounted for 25% of all primary cardiac tumours. These were predominantly sarcomas (95%), which have a poor prognosis.

Dr Sheppard commented that advances in cardiac imaging have increased identification of primary cardiac tumours at an early stage, and also improved prognosis.

radionuclide ventriculography and cardiac magnetic resonance (CMR). Many centres follow patients with serial echocardiography and the British Society of Echocardiography has produced guidance on this.

Dr Harbinson said that it is not important which imaging test is used as long as the same test is used on each occasion as there is poor agreement between methods. Also, he said, imaging investigations must undergo rigorous quality control to ensure that change in EF is a real change. “This is important if we might be denying patients the benefits of trastuzumab.”

Noting that EF is quite late to change in cardiac disease, Dr Harbinson said that, in future, advanced echocardiography markers of left ventricular performance might help with the early detection of toxicity. Blood biomarkers, such as B-type natriuretic peptide (BNP) and troponin, are also being looked at as markers of risk, and might have a role in early detection.

Effects of radiotherapy on the heart

Martin Denvir (Edinburgh) explained that there is a clear relationship between dose of radiation to the heart and risk of heart disease. Exposure to radiation doses >1500 cGy is associated with excess cardiac morbidity and mortality emerging 10 years after treatment.

Adults at risk of cardiac damage include those who have received radiotherapy for breast cancer, lymphoma, lung cancer and oesophageal cancer. For childhood cancer, there is a risk after acute lymphoblastic leukaemia and lymphoma, and other malignancies in which the heart is exposed to radiation.

The longer-term effects of radiation on the heart include heart failure/LVSD, constrictive pericarditis, pericardial effusion, coronary heart disease and valvular heart disease.

A recent US questionnaire survey of survivors of childhood cancer reported a clear association between dose of radiation received and incidence of cardiac sequelae, including heart failure, pericardial disease and myocardial infarction.[3] The cumulative incidence of adverse cardiac outcomes in cancer survivors continued to increase up to 30 years after diagnosis.

In breast cancer, radiotherapy reduces local recurrence in node-positive disease and long-term mortality from breast cancer. But this comes at a cost, Dr Denvir said, with a reported 25% increase in risk of cardiovascular deaths.

US registry data show that an increase in the risk of heart disease deaths in women treated with radiotherapy for left-sided breast cancer starts to appear after 10–15 years. “So, studies with a 1–5 year follow-up are not going to find toxicity. We need studies that go beyond 10 years,” he commented.

Martin Denvir (left) and Perry Elliott

Dr Denvir noted that modern technologies and clinical strategies are reducing radiation doses to the heart and, consequently, cardiovascular risk.

He pointed out that in breast cancer patients, there is an interaction between radiotherapy and anthracyclines, leading to increased cardiac toxicity. There is little evidence of interaction between trastuzumab and radiotherapy but long-term data are not available.

Interaction between radiotherapy and conventional cardiovascular risk factors is uncertain and he thought there was insufficient evidence to consider primary prevention with treatment of cardiovascular risk factors at a lower threshold, although this had been suggested.

A cardiac sub-study of the Medical Research Council SUPREMO* breast cancer trial is looking at serial BNP up to 10 years after radiotherapy to see if this can identify patients with LVSD and predict those who might develop it in future. “This could be a way of trying to prevent the long-term sequelae,” Dr Denvir said.

Dilemmas in the management of patients with heart failure and atrial fibrillation

Joint session with BSH and Heart Rhythm UK

In this session, speakers discussed the considerable challenges in treating patients who have both atrial fibrillation (AF) and heart failure, as well as new research on the underlying disease in AF.

Maurits Allessie (Maastricht, The Netherlands) said that there is plenty of room to improve AF therapy. At present, there is no diagnosis of the patholological changes in the atrial wall that cause AF. “We don’t know what we are dealing with,” he said.

He explained that there is a growing belief that a single rapid driver somewhere in the atria is responsible for maintaining AF. In theory, therefore, if the driver is found and ablated, the patient is cured. In paroxysmal AF this has been quite successful, with the ablation of sites of rapid impulse formation in the pulmonary veins (PVs). But in permanent AF the sites are not clustered in the PV area, and attempts to ablate high dominant frequency sites have never succeeded in terminating the arrhythmia.

So there is still doubt as to what mechanism is operating in persistent AF, or indeed whether the mechanism is the same in different diseases: AF in patients with heart failure may differ from that in hypertension, or that associated with ageing.

Professor Allessie described electrophysiological studies that he has been carrying out. He has a database of thousands of fibrillation maps from 25 patients in sinus rhythm in whom AF was induced acutely and 24 patients in persistent AF. This database is being used to search for a driver of AF.

Numerous ‘focal’ fibrillation waves have been found in the right atrium, left atrium and PV area in patients with longstanding persistent AF. Professor Allessie said that these fibrillation waves – which perpetuate AF – are being recorded at the epicardial surface but appear to come from deeper layers.

His theory is that on ageing, or in conditions such as heart failure, hypertension or valvular disease, the endocardial and epicardial layers of the atrial wall become separated. Fibrillation is propagated in the endocardial layer, and may transmit into the epicardium and spread radially.

The mapping studies have shown great inter-patient variability in AF substrate in patients with persistent AF. This might eventually lead to a scenario, Professor Allessie suggested, where it would be possible to choose a treatment for patients according to the stage of their disease. Although this is a long way off (for one thing, non-invasive methods of diagnosis will be needed), in theory, different therapeutic approaches might be taken according to the degree of conduction block in the atrial wall; for example:

- stage 2 AF: drug therapy may be appropriate
- stage 3 AF: ablation therapy may be appropriate
- stage 4 AF: ablation is inadvisable because the substrate is already severely affected.

Dilemmas in medical management

By the time patients with heart failure come to medical attention, they have structural changes to the heart, so it is not surprising that this predisposes to and perpetuates AF, said Suzanna Hardman (London). This was illustrated by reflecting on some of Maurits Allessie's earlier studies showing that over time persistent AF produces structural change in the heart which maintains AF and is not reversible. Similar changes are found in heart failure (including experimental models).

AF and heart failure often co-exist. Depending on the dataset, 10–50% of patients with heart failure also have AF. The conditions are mutually causative and each can adversely affect the other. “We really don’t understand the haemodynamics in AF. But we know that AF can cause heart failure, AF can cause worsening of heart failure and heart failure can cause AF,” Dr Hardman said.

*A list of study acronyms can be found on page 8.

Data from 2008/9 from the National Heart Failure Audit showed that 37% of patients admitted to hospital with heart failure also had AF. And in the prospective US Cardiovascular Health Study, which followed over 4000 patients (mean age 73 years) for 10 years, with incident AF the hazard ratio for developing heart failure was 3.96, and with incident heart failure the hazard ratio for developing AF was 4.4.

At acute presentation, all AF patients require heparin, whether or not they have heart failure. Patients with uncontrolled AF and acute heart failure will also need intravenous diuretics and rate control, irrespective of the long-term plan. A small proportion may even need emergency direct current cardioversion. Digoxin has no role in this context – it will have no impact in the presence of the high level of adrenergic drive and there is a risk of inducing further arrhythmias. In contrast, a low-dose, short-acting beta-blocker – possibly intravenously – with frequent clinical review can be very efficacious.

For the longer-term strategy, thromboembolic risk has to be considered and a decision made with regard to rate or rhythm control. Dr Hardman said that although it was not perfect, warfarin remained the anticoagulant of choice. But use has to be balance against bleeding risks, and this decision requires involvement of the patient.

She said that, at present, the new thrombin inhibitors do not seem to have a role in AF: they do not abolish the bleeding risks, and some have high renal clearance which could be hazardous. There is little evidence for aspirin in this population and, indeed, there is evidence that aspirin may attenuate the effect of ACE inhibitors in heart failure, Dr Hardman said.

On the choice of rate or rhythm control, sinus rhythm would be expected to be better in heart failure patients, but studies to date had shown no mortality advantages of rhythm over rate control, and there was some suggestion, from sub-set analysis, that rate control may be preferential. Better clinical trials were needed, Dr Hardman said, emphasising that antiarrhythmic drugs can have risks, particularly in the heart failure population.

The role of ablation in AF and heart failure is yet to be established since studies were relatively small, often reflected highly selected patients and were rarely powered for mortality. That said, this was a useful option “but it is not for the faint hearted as repeated procedures may be necessary.”

Dr Hardman noted data emerging from animal models that activation of the renin–angiotensin system is associated with atrial cell growth, the proliferation of fibroblasts and atrial fibrosis – all components that predispose to AF — and that these effects are attenuated with ACE inhibitors. For the future, therefore interfering with that system may help to prevent AF.

Role of devices and ablation

Ablation and devices are important treatment strategies for heart failure patients with AF when optimal medical therapy fails.

Gerhard Hindricks (Leipzig, Germany) said that cardiac resynchronisation therapy (CRT) is clearly indicated in asynchronous patients with heart failure. It was surprising, he said, that in the CARE-HF study, CRT had no protective effect on the development of new AF, despite an improvement in haemodynamics.

In heart failure patients with AF, rate control (with atrioventricular [AV] node ablation) without CRT is almost obsolete, Professor Hindricks suggested. “Patients require 100% right atrial stimulation and I think this is a first-class indication for CRT.”

On choice of ablation procedure, he said that AF ablation (PV ablation) is superior to AV node ablation in patients with heart failure. The PABA-CHF study, which compared AF ablation with AV node ablation plus CRT in heart failure patients, showed superior improvements in exercise capacity, EF and quality of life with AF ablation.[4] “The clinical benefit of curative treatment – of restoring sinus rhythm – is significantly higher than palliative treatment with AV node ablation plus CRT,” Professor Hindricks said.

He suggested that AF ablation would become the leading therapy for most patients with AF.

Studies assessing the use of AF ablation in heart failure patients have had promising results, although the studies have been quite small and with relatively short follow-up, so clinical data are only preliminary. Freedom from AF after ablation ranged from 77% to 96%, but there was a high need for re-ablation. A study from a group in Bordeaux compared AF ablation in 58 patients with heart failure and 58 control patients without heart failure. After 12 months, 78% of heart failure patients and 84% of control patients remained in sinus rhythm, and the heart failure patients had a significant improvement in EF.

Professor Hindricks outlined experience in Leipzig of catheter ablation in 36 patients with AF and heart failure. Patients had a mix of ischaemic and dilated cardiomyopathy; 89% were in persistent AF. Circumferential PV ablation was used, after cardioversion. Of the 36 patients, freedom from AF was seen in 70%. Treatment also had a significant impact on EF, which increased from a median of 32% before ablation to 53% after ablation.

Current practice in his hospital is for patients with AF, heart failure and asynchrony to be first treated with CRT (with or without a defibrillator). CRT delivery and clinical status are assessed on follow up and, based on this, AF ablation is considered. AV node ablation is a possible option if AF ablation fails.

Professor Hindricks emphasised that further studies are needed to assess the impact of AF ablation (versus antiarrhythmic drug therapy and AV node ablation) on survival in heart failure patients. “As ejection fraction is one of the most important predictors of mortality, and considering catheter ablation can

Maurits Allessie (left) and Gerhard Hindricks

improve ejection fraction, it would be surprising if it did not have an impact on mortality. But the studies need to be done and are under way. It will be a couple of years before we know the true role of AF catheter ablation in heart failure patients," he said.

Patients with congenital heart disease

Discussing special considerations in adults with congenital heart disease, Lorna Swan (London) said that atrial arrhythmias are inevitable in many patients. AF itself is infrequent and tends to be a manifestation of end-stage disease; however, other atrial arrhythmias, including intra-atrial re-entrant tachycardia (IART), are more common.

Adult congenital heart disease patients with rhythm problems are difficult to look after as many patients have very complex cardiac anatomy. Care requires a multidisciplinary approach and should ideally be carried out in a specialist centre.

The typical patient at risk of IART is a patient who has undergone a Fontan surgical procedure for a single ventricle. At the moment there is no good long-term medical therapy for these patients. Rate control is often not appropriate as atrial tachycardia may be poorly tolerated haemodynamically and there is a risk of thrombosis.

Onset of atrial arrhythmias merits aggressive rhythm control. However, medical therapy for rhythm control has a high failure rate. Electrical cardioversion is often highly effective in IART, but is not without risk. It usually needs to be done urgently. Patients often have recurrent arrhythmia and it is not uncommon for some patients to undergo 15 or 20 cardioversions.
Dr Swan emphasised the need for a back-up pacing plan for implementation if the patient becomes bradycardic after cardioversion.

Because of the failure of medical therapy and the dangers of cardioversion, at the Royal Brompton Hospital they had a low threshold for ablation. "We are often forced into doing relatively high-risk ablations," she said.

Ablation planning is important to improve the success rate. Some patients have very unusual underlying anatomical and arrhythmic substrate, for example, some have two AV nodes or two sinoatrial nodes. Access to the heart can also be difficult. CMR and electrophysiological mapping studies are useful to understand the cardiac anatomy, plan access to the heart and identify targets for ablation.

"From the cell to the bedside" A symposium in memory of Philip Poole-Wilson

The first clinical trial of gene therapy in heart failure has shown some indication of efficacy, Sian Harding (London) told the BCS conference. The data are from a phase 2 US trial of SERCA2a (sarcoplasmic reticulum Ca^{2+}ATPase) therapy in patients with advanced heart failure. A similar UK trial is in the final stages of EU approval.

Professor Harding explained that SERCA2a was identified as a potential therapeutic target after discovery of the enzyme's role in calcium handling in cardiac myocytes and studies showing that enzyme activity is reduced in advanced heart failure, leading to impaired myocardial relaxation and contraction.

There is no small-molecule drug that targets SERCA2a, so it was decided to attempt enzyme replacement with gene therapy, using an adeno-associated virus vector. Six-month results from the phase 2 CUPID trial were reported at the Heart Failure 2010 conference. The gene therapy is given by intracoronary artery infusion.

Professor Harding said that the safety profile seems acceptable and, although numbers were small (25 patients received active treatment), there were positive trends in BNP level, left ventricular end-systolic volume and EF. The UK trial will involve patients with an implanted left ventricular assist device.

Professor Harding was one of the speakers in a BCS symposium held in memory of Philip Poole-Wilson. She said that Professor Poole-Wilson had been instrumental in her work on cardiac myocytes and had provided the impetus to get to the stage of the gene trial.

Sian Harding

With the title of "From the cell to the bedside", the symposium covered other areas in which Professor Poole-Wilson had specific interest. Stuart Cobbe (Glasgow), who was Professor Poole-Wilson's first research fellow, spoke on cellular cardiac electrophysiology, and Roberto Ferrari (Ferrara, Italy) described their

collaborative work on the understanding of neuroendocrine response in heart failure.

Martin Cowie (London) described how Professor Poole-Wilson had encouraged his early work on the epidemiology of heart failure in the UK in the 1990s. Professor Cowie emphasised that the prognosis for patients with heart failure is now much improved from that found in the early studies, but that it remains essential to cascade good practice across the country. He noted the challenges to come in delivering high-quality care to increasing numbers of patients with fewer resources.

The failing right heart

Joint session with the British Society of Echocardiography

In a session on the failing right heart, delegates had an update on the anatomy of the heart, followed by a presentation from Lynne Williams (Birmingham) on diastolic ventricular interaction (DVI) – the effect of the distended right ventricle on the filling of the left ventricle. Dr Williams said that a significant proportion of heart failure patients have evidence of DVI at rest. She said that DVI provides a potential target for treating patients with acute decompensated heart failure (intravenous nitrates and diuretics would be expected to have an effect) or chronic heart failure, where there is evidence that CRT (and specifically left ventricular pacing) may reduce DVI.

Richard Steeds (Birmingham) discussed methods of assessing the right ventricle in patients with heart failure. “Ignore the right ventricle at your peril,” Dr Steeds said, noting that a recent study showed a baseline right ventricular EF of <20% to be a significant independent predictor of mortality and hospitalisation in patients with systolic heart failure.[5] He said that echocardiography (particularly 3-dimensional echocardiography) and CMR were quite good at assessing the structure of the right ventricle, but there was still some way to go in assessing function: various techniques (e.g. radionuclide ventriculography, CMR and 2-dimensional echocardiography) were available but were not always reliable in the clinical situation.

Theresa McDonagh (London) outlined treatment of the failing right ventricle, which she said was essentially the management of congestion. Right heart failure is characterised by peripheral oedema, but patients can also develop tissue oedema (anasarca) and can present with ascites or pleural and pericardial effusions. Fluid retention in right ventricular failure (RVF) is very gradual, in contrast to pulmonary oedema in left ventricular failure.

Dr McDonagh said that 3% of patients admitted to hospital with heart failure present with predominantly right-sided heart failure and this condition has a poor prognosis, with an in-hospital mortality of 9%. Specific causes include acute right ventricular infarct, pulmonary hypertension and cor pulmonale (end-stage chronic obstructive pulmonary disease); congenital heart disease is another important cause. But by far the most common cause of right-sided heart failure is left-sided heart failure: in the majority of patients right ventricular failure is secondary to LVSD.

The mainstay of treatment is diuretics, with general management as for LVSD. Dr McDonagh emphasised the need to use the minimum diuretic dose necessary to remove the signs of congestion. It was important not to drop the filling pressure too much as this could exacerbate the syndrome in conditions such as restrictive cardiomyopathy or pericardial constriction.

For patients with pulmonary hypertension and RVF, there is interest in the use of phosphodiesterase type 5 inhibitors, and some data were emerging with sildenafil, but much bigger, long-term trials with these drugs were still needed.

BSH award for ‘Awareness Day’ activities

The BSH received an award from the Heart Failure Association of the European Society of Cardiology (ESC) for the publicity and initiatives that took place in the UK in connection with Heart Failure Awareness Day held on 7 May 2010.

Theresa McDonagh (BSH Chair) thanked all members, Friends of BSH and others that took part in arranging events and publications, as well as everyone that attended the events. The day had been a great success in raising the awareness of heart failure across the country. She said that next year the date for Heart Failure Awareness Day would be announced much earlier, which would give everyone more time to plan activities.

Study acronyms

CARE-HF	Cardiac Resynchronization – Heart Failure
CUPID	Calcium Up-Regulation by Percutaneous Administration of Gene therapy In Cardiac Disease
PABA-CHF	Pulmonary vein antrum isolation versus AV node ablation with bi-ventricular pacing for treatment of atrial fibrillation in patients with congestive heart failure
SUPREMO	Selective use of postoperative radiotherapy after mastectomy

References

1. Jones AL, Barlow M, Barrett-Lee PJ *et al*. Management of cardiac health in trastuzumab-treated patients with breast cancer: updated United Kingdom National Cancer Research Institute recommendations for monitoring. *Br J Cancer* 2009;100:684–92.
2. Patel J, Sheppard MN. Pathological study of primary cardiac and pericardial tumours in a specialist UK centre: surgical and autopsy series. *Cardiovasc Pathol* 2009; Sep 9. [Epub ahead of print].
3. Mulrooney DA, Yeazel MW, Kawashima T *et al*. Cardiac outcomes in a cohort of adult survivors of childhood and adolescent cancer: retrospective analysis of the Childhood Cancer Survivor Study cohort. *BMJ* 2009;339:b4606.
4. Khan MN, Jais P, Cummings J *et al*. Pulmonary-vein isolation for atrial fibrillation in patients with heart failure. *N Engl J Med* 2008;359:1778–85.
5. Meyer P, Filippatos GS, Ahmed MI *et al*. Effects of right ventricular ejection fraction on outcomes in chronic systolic heart failure. *Circulation* 2010; 121:252–8.

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