The partial update of the National Institute for Health and Clinical Excellence (NICE) guideline on chronic heart failure, published in August, was one of the key topics of the conference. There was lively debate regarding the interpretation and implementation of NICE’s recommendations.

Outlining the NICE diagnostic pathway, Abdallah Al-Mohammad (Sheffield), who was Clinical Adviser for the Guideline Development Group, said that the guideline had simplified but not compromised diagnosis (see Box). “The diagnostic algorithm is based on clinical and cost-effectiveness evidence, as well as pragmatic clinical opinion,” he said.

A key change is the recommendation for natriuretic peptide (NP) testing rather than electrocardiography (ECG) in primary care, with referral for echocardiography only for patients with raised NP levels. Also, to increase the speed of diagnosis there are timescales for echocardiography depending on the level of NP. For patients with a previous myocardial infarction (MI) – who are at particularly high risk of heart failure – direct referral for echocardiography is recommended, without NP testing.

Getting the diagnosis right

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NICE recommendations on diagnosis

- Patients with a previous MI should have echocardiography and specialist assessment within 2 weeks
- Patients with B-type natriuretic peptide (BNP) >400 pg/ml or N-terminal-pro-BNP (NT-proBNP) >2000 pg/ml should have echocardiography and specialist assessment within 2 weeks
- Patients with BNP 100–400 pg/ml or NT-proBNP 400–2000 pg/ml should have echocardiography and specialist assessment within 6 weeks

On the specifics of NP testing, Allan Struthers (Dundee) said the test is sensitive but not specific, making it a good rule-out test but a poor rule-in test. Specificity is poor because pathologies other than left ventricular systolic dysfunction (LVSD) can increase NP level. These include left ventricular (LV) hypertrophy, myocardial ischaemia, pulmonary embolism, renal failure, sepsis, chronic obstructive pulmonary disease (COPD), pulmonary hypertension and diastolic dysfunction.

Some caution was needed, he said, in interpreting NP results because levels are reduced by obesity and by drug therapy (diuretics, angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs] and aldosterone antagonists). He thought it useful that NICE emphasised different levels of urgency of echocardiography, but said that, certainly in
Scotland, getting an echocardiogram within 2 weeks would be difficult. He also thought that the availability of NP testing is higher in England than in Scotland.

In discussion, Ahmet Fuat (Darlington) expressed concern that the rule-out levels recommended by NICE are too high and might miss patients with heart failure. He noted that many patients in whom NP tests are carried out in primary care will be on an ACE inhibitor, and perhaps a diuretic for another indication. His local laboratory has age-related cut-offs. Professor Struthers said that age-related cut-offs are a good idea but they detract from the simplicity and ease of use of the test.

Another controversial area relates to NICE’s recommendation of the need for “specialist” assessment in heart failure diagnosis. NICE says that a specialist should make the final diagnosis. It defines a specialist as “a physician with sub-specialty interest in heart failure (often a consultant cardiologist) who leads a specialist heart failure multidisciplinary team.”

There were many general practitioners with a special interest (GPSIs) in heart failure in the audience who asked if they would be seen by NICE as a specialist for making a diagnosis and instituting a management plan. Dr Al-Mohammad said that the specialist as defined in the guideline is essentially a consultant physician. The intention is that all patients have a face-to-face consultation with a specialist, not just that the specialist interprets the echocardiogram. “It is important that the specialist sees the patient at least once and determines the diagnosis,” he said.

Theresa McDonagh (London) agreed that consultants must see new patients and patients admitted to hospital. These are the patients at highest risk. But chronic disease management is different, and it is not necessary for consultants to see patients for routine follow up or for up titration of drugs. “We have to start dividing up tasks more sensibly,” she said. In a discussion on whether a diagnosis might be missed if general practitioners (GPs) no longer carry out an ECG in suspected heart failure, Dr McDonagh said that NPs have greater sensitivity than an ECG. An ECG will certainly be part of routine secondary-care assessment, but primary care should plan its service around NP tests.

**Echocardiography beyond ejection fraction**

Discussing echocardiography in patients with suspected heart failure, Antoinette Kenny (Newcastle) said that it is important not just to measure ejection fraction. The echo assessment should include tissue Doppler imaging (TDI) to measure long axis function. This new, more sensitive, measure of LV function can identify early systolic impairment that is not seen when looking just at ejection fraction.

Dr Kenny said that some 50% of patients with heart failure have “normal” systolic function as assessed by ejection fraction, and so are thought to have impaired diastolic function. But measurement of long axis function suggests that these patients do not have pure diastolic dysfunction. “Their ejection fraction is normal but they have stiff hearts and their long axis function is abnormal in systole. They have early LV systolic dysfunction.”

She suggested that long axis measurement should be incorporated into routine clinical practice. The test would only take an extra 30 seconds. Dr Kenny accepted there were as yet no longitudinal data to show progression of LVSD in patients with predominantly diastolic dysfunction. The main thing at present was not to dismiss symptoms as non-cardiac as these patients would benefit from diuretics.

**Rational management of heart failure**

Drug treatment of heart failure is intended to make patients feel better and live longer, and this may involve accepting changes in renal function, said Iain Squire (Leicester).

He emphasised the importance of clinical examination for assessing the adequacy of drug therapy, in particular of diuretics. Salt and water retention, associated with activation of the renin–angiotensin system, was central to the pathophysiology of heart failure, and the rational use of diuretics could be challenging.

Clinicians must ensure that patients are given enough diuretic. When estimated glomerular filtration rate was low – and it often would be – more diuretic would be needed. “But this is often the
situation when they are tempted to reduce diuretic because they think they are poisoning the kidneys,” Professor Squire said. “It is obviously important to watch urea and creatinine but don’t forget the place of appropriate clinical examination in deciding whether a patient needs more diuretic.”

He said that asking patients about nocturnal dyspnoea and their ability to lie flat is a sensitive marker for fluid status/intravascular volume. He uses this to drive the diuretic dose up a little more than he otherwise might. Professor Squire also commented that clinicians are sometimes too conservative about uptitration of ACE inhibitors.

Discussing clinical trials in heart failure, Henry Dargie (Glasgow) said that large randomised trials had provided a robust evidence base for the management of patients with heart failure and a reduced ejection fraction (HeFREF). Mortality had been the most important endpoint in the earlier trials but hospitalisation not only for heart failure but for all causes is increasingly being seen as a valid trial endpoint. Patients do not want to be in hospital and since hospital admissions are very expensive their reduction is assuming greater fiscal importance. Improving symptoms remains a very important endpoint for patients and is essential for regulatory approval.

Although many drugs have been proven to work in terms of symptoms and outcomes, there was a very long list of failures, including, most recently, vasopressin antagonists, endothelin antagonists and adenosine A1 antagonists and, previously, some beta blockers. Results from trials of medicines for acute heart failure and in patients with a preserved ejection fraction (HeFPEF) have been, relative to those in HeFREF, disappointing.

He suggested that some drugs have failed because of faulty trial design. “In clinical trial design it is extremely important to have the right population of patients and the right dose. It is particularly important not to introduce the drug in too high a dose.” Professor Dargie thought that the neuroendocrine approach had been very successful and could be explored further. He stressed that, although it was essential to define novel targets and to develop more, individualised, treatments, it was equally important to use what we know works much more effectively. The national audits of heart failure in the UK have revealed a continually high and unacceptable mortality associated with inadequate prescription rates of proven medicines and devices.

Andrew Clark (Hull) emphasised the need for scepticism in interpreting clinical trial data. He was wary of subgroup analyses: “We must always treat a subgroup analysis, whether prespecified or not, with a huge pinch of salt.” Subgroup analyses usefully point the way to the next trial, but should not be used as a guide to current practice, he said.

Combined endpoints could also be problematic. In such cases, the individual components should be of comparable clinical importance, occur with similar frequency and be similarly sensitive to treatment intervention. Results could otherwise be misleading; there may, for example, be an overall positive result on the basis of one component that is less important while being negative on the more important endpoints.

Professor Clark reminded delegates to be alert to the presentation of misleading trial data: for example, a graph where the y-axis does not start at zero. He was also not happy with the notion of “class effects” and suggested that clinicians should use the specific drugs that have been proven to work in clinical trials.

Management of heart failure: what’s new?

The state of play with telemonitoring (remote monitoring) in heart failure was discussed by Martin Cowie (London). “Make no mistake, this will have an impact on your practice within the next 5 or 10 years,” he said, noting the political drive to increased home care and also the projected doubling in number of heart failure patients, which would require changes to models of care.

Professor Cowie explained that monitoring technology varies from simple to complex but data transmission to the healthcare team is straightforward. Many physiological features can be remotely monitored, either with stand-alone systems or implanted systems. With the latter, data can be collected from a therapeutic device (implantable cardioverter defibrillator [ICD] or cardiac resynchronisation therapy-defibrillator [CRT-D]) or from diagnostic systems, such as devices that measure pulmonary artery pressure.

Patient acceptability of remote monitoring is high; it is more likely to be health professionals’ attitudes that are a barrier to adoption of the technology, Professor Cowie said.

Iain Squire: “Ensure that patients are given enough diuretic”
The rationale is that telemonitoring will give early warning of decompensation so that care can be adjusted as necessary. It will not be for all patients, or necessarily for use long term for any one patient. Challenges still remain, including the best combination of variables to monitor and how best to present the information so that health professionals are not swamped by data but can use them to make better decisions for their patients.

Professor Cowie said that a recent Cochrane review1 of randomised trials of telemonitoring with stand-alone systems found a 34% reduction in mortality and a 21% reduction in heart failure hospitalisation compared with usual care. He pointed out that a new UK trial to assess remote monitoring using implanted devices has just got the go-ahead, with nearly £4 million funding from the British Heart Foundation and industry. This trial would help to secure the evidence base.

**New clinical trial results**

John Cleland (Hull) gave an overview of important heart failure clinical trials reported in 2010. These included EMPHASIS-HF,2 which assessed eplerenone given in addition to standard therapy in mild heart failure (New York Heart Association [NYHA] functional class II) and showed a 37% reduction in the primary endpoint of cardiovascular death or hospitalisation for heart failure, and a 24% reduction in both all-cause mortality and cardiovascular death (secondary endpoints). There were no safety issues.

SHIFT investigated ivabradine, a heart rate-lowering drug, in class II–IV heart failure and reported an 18% reduction in risk of cardiovascular death or hospitalisation for heart failure and a trend to reduction in all-cause mortality and cardiovascular death (secondary endpoints). There were no safety issues.

The RAFT trial4 compared ICD and CRT-D in NYHA class II or III heart failure. There was 25% reduction in death/hospitalisation for heart failure and also a 25% reduction in death in the CRT-D group after a mean follow up of 40 months. The result encourages use of CRT-D rather than ICD in patients with few symptoms and a broad QRS on the surface ECG, Professor Cleland commented.

In acute heart failure, the DOSE trial compared low- and high-dose intravenous diuretic, and continuous versus twice-daily bolus therapy. The higher dose was more effective but there was no clear difference between continuous or bolus therapy.

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**Assessing prognosis/identifying high-risk patients**

In a session on assessing prognosis in patients with heart failure, Simon Woldman (London) said that sympathetic imaging is a new technique with potential value. The ADMIRE-HF study showed that metaiodobenzylguanidine (mIBG) imaging could predict risk of arrhythmia. More recently, data have shown that use of the test before ICD implantation can predict defibrillator discharge.5 This raises the possibility that mIBG might be used to identify patients who are at particularly high risk of sudden cardiac death and would most benefit from an ICD. An intervention study to assess this is now planned.

Kevin Damman (Groningen, The Netherlands) described new urinary markers that might identify patients who are at risk of worsening renal function. He said that one of the pathological features of the cardio-renal syndrome is tubular damage and this can be measured with the new markers which include N-acetyl-beta-D-glucosaminidase and KIM-1 (kidney injury molecule 1).

There are no data yet to show that these markers can be used in clinical practice to guide therapy. However, Dr Damman said that urinary levels of these markers add to prognostic information in patients with heart failure and may predict worsening renal function long before there is any observed change in serum creatinine.

The new NICE guideline on unstable angina and non-ST-elevation MI has an emphasis on coronary intervention and ischaemia testing. It mentions the need for screening for LVSD but as this is not specifically flagged up as a key priority it may be overlooked and lifesaving therapies may not be started early enough, said Martin Cowie (London). He emphasised the importance of early detection of post-MI ventricular damage. These patients are very high risk and their risk can be reduced with early initiation of therapy (ACE inhibitor, beta-blocker and eplerenone). Waiting 6 weeks to start drug therapy probably misses most of the opportunity to modify the condition. Professor Cowie said that hospitals should ensure their protocols include assessment of LV function: “Don’t use the excuse that patients are not in hospital long enough. If you have a heart attack, it should not be too much to expect that someone assesses your LV function and gets you on to the right drugs before you go home.”

Cardiopulmonary exercise testing should be routine in the management of patients with chronic heart failure said Klaus Witte (Leeds). An exercise test – on a treadmill or bike – is not complex, the equipment is relatively inexpensive, and the test gives an objective measure of severity, helps with diagnosis and

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*A list of study acronyms can be found on page 7.
Heart failure audit

The BSH is hoping to get a grant to carry out a trial to investigate the feasibility of heart failure units (similar to stroke units). The need for such units is highlighted by findings from the National Heart Failure Audit that survival is higher in patients who have access to specialist cardiology care, said Theresa McDonagh (London).

The audit shows that patients who receive specialist care on admission are more likely than those who do not to be discharged on evidence-based drug therapy and to have specialist heart failure follow-up, and that both these factors are associated with better survival.

Reporting the latest data from the National Heart Failure Audit (April 2009–March 2010), Dr McDonagh said that overall 1-year mortality was 30% and inpatient mortality was 9.6% (6.4% in patients treated on cardiology wards and 12.4% in patients treated elsewhere in the hospital). Outcome was therefore still very poor and a long way from the results achieved in clinical trials. Lack of specialist care during or after admission was associated with worse 1-year outcomes.

Overall, 46% of patients were treated in cardiology wards and 42% in general medical (including care of the elderly) wards. A higher proportion of men were treated on cardiology wards. For patients with LVSD, around 80% were discharged on an ACE inhibitor or ARB, 65% on a beta-blocker and 40% on an aldosterone antagonist. Prescribing of all these therapies was higher in younger patients. Dr McDonagh commented that the audit data are robust as the audit is now large. Participation has increased and in the past year data were recorded on over 21,000 admissions.

can help identify deterioration. This will help with assessment of prognosis.

Dr Witte said it is important to carry out a functional test that reproduces the patient’s symptoms rather than just performing an ECG and echocardiography. Three basic variables are measured in an exercise test: oxygen uptake, carbon dioxide output and ventilation. From these, peak oxygen uptake, anaerobic threshold and an impression of ventilatory response to exercise can be derived. In a group of 1100 patients, he had found peak oxygen consumption to be much better than LV function as a prognostic marker. For patients who cannot cope with stress testing, the 6-minute walk test, performed correctly, could also be a good marker of symptom severity and outcome, he said.

Getting drug treatment right

In brief presentations highlighting the evidence for major drug groups, Henry Dargie (Glasgow) suggested that aldosterone antagonists should now join ACE inhibitors and beta-blockers as routine first-line therapy for patients with chronic and post-MI heart failure. The drugs have shown consistent clinical benefit in randomised controlled trials and in clinical practice. Hyperkalaemia has not been a significant problem when the usual safeguards are observed.

With regard to ARBs, Hugh McIntyre (Hastings), who was a member of the Development Group for the NICE guideline update, explained how NICE had used all-cause mortality as the basis for comparing ACE inhibitors and ARBs. It had concluded that ACE inhibitors should have primacy but that ARBs could be considered for patients who have intolerable side effects with ACE inhibitors and, on specialist advice, as an option for second-line treatment in patients who remain symptomatic despite optimal first-line treatment (especially in mild–to-moderate heart failure).

Abdallah Al-Mohammad (Sheffield) explained that NICE sees the isosorbide dinitrate/hydralazine combination as an option, on specialist advice, where there is intolerance to both ACE inhibitors and ARBs, and as a second-line treatment (particularly in patients of African-Caribbean origin).

Discussing NICE’s treatment algorithm for LVSD, Suzanna Hardman, who was also a member of the Development Group for the NICE guideline update, said that it was clear that all patients need an ACE inhibitor and a beta-blocker. It does not seem to matter which drug is started first. There are many “historical” reasons for not prescribing beta-blockers, but these drugs could, and should, be used in the elderly, and in patients with peripheral vascular disease, diabetes, erectile dysfunction, interstitial lung disease and COPD without reversibility.

She said that ARBs are useful in patients who have a true intolerance to ACE inhibitors. But patients are often swapped from an ACE inhibitor because of cough when this may just be a sign that the patient’s heart failure is inadequately treated.

Discussing the NICE recommendations for second-line treatment, Dr Hardman said that the guideline had gone to press before publication of the EMPHASIS-HF data. It was
now apparent that aldosterone antagonists produced benefit on top of ACE inhibitors and beta-blockers, and could be used in patients with all types of chronic heart failure.

Moving on to the recommendation that advice is sought from a heart failure specialist when a patient is admitted to hospital with heart failure, Dr Hardman said that inpatient mortality is lower when a cardiologist with an interest in heart failure leads the service and ensures optimum treatment. “We have got to get it right for the inpatient population and then pick it up in the community. We want an integrated multidisciplinary team that works across all sectors of care. The audit data show that at present we are not getting it right. The guideline is about increasing the quality of care across the board so all patients have the best care.”

Giving a primary-care perspective on the guideline, Jim Moore (Cheltenham) said that a high proportion of healthcare communities do not currently have access to NP testing. Testing is now central to the initial assessment of patients with suspected heart failure and this, together with the requirement for early specialist assessment, will have significant implications for the commissioning and provision of services. He suggested that the price of NPs might be reduced by procurement on a wider basis and also pointed out that NHS Improvement has cost modelling data that might support communities wanting to introduce this test.

The Gloucestershire Heart Failure Service is not typical: the primary care-based service has nine full-time equivalent heart failure nurses and four GPSIs. Dr Moore said that, at present, NP testing is not used but it is being seriously considered. A recent study in Gloucestershire found that one-third of patients who would normally have been referred for echocardiography would not be referred if NP testing were used. The decision was still to be made on whether they would have open- or restricted-access NP testing.

Dr Moore said that the NICE guideline had been well received in primary care as simple, comprehensible and practical. And he felt that GPs were getting more comfortable with use of beta-blockers in heart failure.

A secondary-care view on the guideline was given by Simon Woldman (London) who thought that the major problem with implementation would be getting sufficient consultant appointments to see all new heart failure patients. However, many more patients could be seen if services were reorganised. With the amount of money currently spent in North Central London on heart failure services (£12 m) there was good opportunity to restructure services. “We have [a] great resource, and just need to use it more efficiently.”

Dr Woldman suggested that all heart failure staff should work together, in one place. This would avoid the current artificial barrier between primary and secondary care. Patients could be “worked up” by a specialist nurse or registrar, receive their ECG and echocardiography, and then be seen by a consultant who could decide the management plan. This sort of change would be difficult but was essential, he said.

Patients’ unmet needs

In a session on patients’ unmet needs, John Buckley (British Association for Cardiovascular Prevention and Rehabilitation) said that cardiac rehabilitation (CR) for stable heart failure patients is recommended by NICE but at present less than 2% of eligible patients take it up. CR in heart failure poses challenges that differ from the usual CR triggered by an acute “behaviour changing” event or surgery where there is an obvious “fix” and patients can more easily see a pathway of benefits.

He emphasised that CR for patients with heart failure involves more than exercise. Psycho-behavioural aspects are also essential. The goals are to prevent hospital readmissions and unnecessary primary-care appointments, and to help patients to self-manage.

Dr Buckley said that CR does not always need to be hospital based and the exercise component of a CR programme for heart failure does not always need to involve an exercise class. For some low functioning patients, simply managing and better pacing in activities of daily life, which leads to more activity, can
achieve the same as a structured programme. He also suggested that for some patients muscle strength training might be the place to start, rather than aerobic training, as it is short-term muscle activity that often limits patients’ daily function and affects quality of life.

Simon Conroy (Leicester) discussed advance-care planning in heart failure. He said that patients would make different decisions about priorities (e.g. some would wish to focus on survival, others on comfort), so it is important to ask what they would like. Not everyone wants to be treated at home at the end of life.

A majority of patients want to talk about the future but find it difficult to initiate the conversation. Others do not want to talk about it and advance-care planning should not be forced on them. Advance-care planning must involve family and carers, and be carried out at the right time for the individual.

Dr Conroy said that heart failure specialist nurses are a fantastic resource in advance-care planning. The heart failure nurse can be the key worker who facilitates discussion with the patient. A recent study has shown that there is already considerable joint working between heart failure nurses and palliative care teams.

Jim Beattie (Birmingham) said that end-of-life care is now part of mainstream heart failure care, appropriate to all care settings. Challenges remain in diagnosing the dying phase and ensuring that treatment options are still appropriate with the changing goals of care. It is difficult to know when to withdraw drugs but he suggested pulling back sequentially on medicines that are no longer contributing. First to stop might be drugs such as statins and digoxin for patients in sinus rhythm. The next step would be to consider drugs with medium-term benefit, such as ACE inhibitors/ARBs, beta-blockers, and drugs for co-morbidities such as hypertension, oral hypoglycaemics or thyroxine.

Dr Beattie noted that there is a trend for patients who are actively dying to receive subcutaneous diuretic. That is fine but the evidence base for this is still limited.

He emphasised that there should be discussion about deactivating the ICD whenever the goals of care are discussed. Doctors should not forget about a patient's ICD; they must accept the patient's choice, and having made a decision to deactivate “make sure you have access to someone who can deactivate without delay.” He pointed out that the Liverpool Care Pathway has a protocol on the deactivation of a device. There are also guidelines on this from the British Heart Foundation and, recently published, from the European Heart Rhythm Association.

Stephen Oxberry (Huddersfield) urged heart failure professionals to get to know their palliative care physician. He emphasised that hospice staff do not just want to get involved in the last 48 hours of a patient’s life. “End-of-life care is difficult for us if we only have a few hours left to get to know the patient and carers,” he said.

Patients’ expectations may not meet reality, in part because they may have recovered from many previous acute episodes. It is difficult to shift the emphasis to involve a palliative care approach and honest communication is key to helping patients through this process.

As a local example of cardiology/palliative care interface, Dr Oxberry said that Bradford Hospice runs a weekly group with referrals from heart failure specialist nurses. This is a multidisciplinary session during which patients can see a range of healthcare staff, and receive complementary therapies and social support. A similar drop-in scheme is planned for Huddersfield. The aim is to demystify hospices so that rather than being seen as places where you die they are seen as places offering symptom support.

A patient’s perspective of the BSH conference was given by Richard Mindham, who participated in a discussion panel after the “Unmet needs” session. He said he had been impressed by speakers’ concern for patients’ wellbeing. On specific points, Mr Mindham referred to the discussion on clinical trial endpoints and said that his focus would be symptom relief rather than mortality/hospitalisation. He also noted the major impact of a heart failure diagnosis on patients’ sense of self worth. Patients would have many questions they wanted to ask about the implications of their diagnosis. “Every time we meet a healthcare professional we go in with 10 questions and come out with 10 more questions,” he said.

Study acronyms

**ADMIRE-HF**  
AdreView myocardial imaging for risk evaluation in heart failure

**DOSE**  
Diuretic optimization strategies evaluation in acute heart failure

**EMPHASIS-HF**  
Eplerenone in mild patients hospitalisation and survival study in heart failure

**RAFT**  
Resynchronisation-defibrillation for ambulatory heart failure trial

**SHIFT**  
Systolic heart failure treatment with the I f inhibitor ivabradine trial
References


BSH AGM

At the Annual General Meeting of the BSH on 26 November 2010, the chair (Dr Theresa McDonagh) reported that membership was stable, currently standing at 733. The majority of members were cardiologists (199) and nurses (410).

Dr McDonagh noted that the BSH had been involved in three high-profile sessions at the British Cardiovascular Society Annual Conference and Exhibition in June 2010. The second BSH training day for cardiology registrars was held in April 2010. During 2010, the BSH collaborated with a number of other professional organisations, including NICE (on the partial update to the heart failure guideline) and the European Society of Cardiology (ESC). The BSH won an award for its activities in connection with the ESC’s 2010 Heart Failure Awareness Day.

In 2011, the BSH training day for cardiology registrars would take place on 9 February and the first BSH study day for heart failure nurses on 10 February. Heart failure Awareness Day would take place on 6 May 2011.

Election of a new board would take place in 2011, with nomination papers being sent out in February. Dr Suzanna Hardman would take over as chair.

The BSH accounts for the year to 31 May 2010 were presented by the Treasurer (Professor Iain Squire) who reported that the finances were in a healthy position.

13th BSH Annual Autumn Meeting: acknowledgements

Gold exhibitors
Alere, GE Healthcare

Silver exhibitors
Pfizer, Servier

Bronze exhibitors

Other contributors
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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 heart failure.
If you are interested in becoming a Member or Friend of the BSH, please contact:

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