The 16th BSH Annual Autumn Meeting was held, as ever, in the Queen Elizabeth II Conference Centre overlooking Westminster Abbey in London on 28–29 November 2013. The theme for the meeting was making sense of acute heart failure. As ever, the meeting was well-attended, with over 700 registrants for the first time. The buzz and good humour surrounding the meeting was extraordinary: many overseas visitors remarked on the friendly atmosphere and how such a meeting never happens elsewhere!

The Programme Directors were Paul Kalra (Portsmouth), Jim Moore (Cheltenham) and Iain Squire (Leicester).

The meeting was opened by the BSH Chair, Professor Andrew Clark (Hull).

**Session 1: Counting the cost of acute heart failure**

In Session 1, Professor John McMurray (Glasgow) gave the keynote lecture ‘What is acute heart failure and how do we treat it?’ He indicated that acute heart failure encompasses a spectrum of acute cardiovascular presentations, including atrial fibrillation (AF) and acute coronary syndromes. Professor McMurray described European Society of Cardiology (ESC) and North American Guidelines for the management of acute heart failure and concluded his presentation by emphasising that acute is not a useful term, and that the label of ‘hospitalised heart failure’ is gaining favour. Professor John Cleland (London) then described the causes of hospitalisation in heart failure, emphasising that most patients are not breathless at rest when admitted, but on exertion only. The dominant symptoms at admission are fluid retention and peripheral oedema. Professor Cleland highlighted the almost complete absence in the UK of recruitment to clinical trials in acute heart failure.

Professor Theresa McDonagh (London) presented data from the National Heart Failure Audit 2012–13. Up to 92% of acute trusts are now contributing data to the audit. As in previous years, the patients included are elderly and have advanced heart failure symptoms, with ongoing differences in the demographic characteristics of patients admitted to cardiology compared with those managed in other specialities. Inhospital mortality in 2012–13 was 9.4%, with a 30-day mortality of 6% in those discharged alive. Professor McDonagh concluded by saying that the audit data suggest that mortality appears to be falling year-on-year and that specialist management of the patient is associated with a better outcome.

The session concluded with a lively question and answer panel, highlighting the need for more data before the management of acute heart failure can improve.
Session 2: Optimising service delivery – how to make sense out of the chaos

Session 2 aimed to explore some of the different systems teams had developed to make sure that, as Dr Nigel Rowell (Middlesbrough), on behalf of commissioners, stressed, the right patient got to meet the right doctor at the right time. Dr Gerry Carr-White (London) described the two approaches to inpatient screening, using either natriuretic peptides or echocardiography. Whilst the natriuretic peptide approach is more sensitive, it involves a lot of extra work as it is not very specific.

Ms Jayne Masters (Southampton) and Dr Jackie Taylor (Glasgow) gave overviews of their integrated inpatient heart failure team models. Dr Angus Nightingale (Bristol) discussed the specific problem of heart failure post-myocardial infarction (MI) and the need to screen all MI patients for left ventricular systolic dysfunction to make sure that appropriate treatment is started. Dr Suzanna Hardman (London) described the workings of a dedicated heart failure unit, which must surely be the ultimate aim for all of us.

Dr Jim Moore (Cheltenham) and Ms Annie MacCallum (Gloucestershire) presented their successful community-based model for targeting heart failure patients.

Session 3: Clinical challenges in the management of acute heart failure (1)

In Session 3, common clinical complications and precipitants of acute heart failure were discussed. Dr Martin Thomas (London) talked about the problems of diuretic resistance in patients with fluid retention and discussed the possible beneficial role of ultrafiltration in selected patients. The majority of patients respond to standard pharmacological measures – intravenous loop diuretics and progressive nephron blockage (that is the combined use of loop and thiazide diuretics). He presented data on group of 79 patients with decompensated heart failure from three UK centres (60% men; mean age 71 years) who were treated with ward-based ultrafiltration. The mean total volume of fluid removed per patient was 8.1 L, with a mean duration of treatment of 58 hours. To date, clinical trials have not shown consistent benefits with ultrafiltration and further, appropriately powered, studies are required.

Dr Dominic Kelly (Hampshire) discussed the very common issue of arrhythmias in patients with acute decompensated heart failure (ADHF). Evidence for the management of such patients is lacking, however, largely due to their exclusion from randomised controlled trials. One or more precipitants, such as structural heart disease, mechanical factors, neurohormonal activation, electrolyte imbalance, ischaemia and pharmacotherapy, may be present. In the absence of clinical evidence, the primary therapeutic intervention is the management of the underlying heart failure, removal of volume overload and optimisation of heart failure medications.

The most common arrhythmia seen is AF. Framingham data suggest that the presence of heart failure increases the risk of AF by around 5–6-fold. In addition, AF becomes more prevalent with increasing heart failure severity. AF is seen in 20–60% of patients in registries of ADHF. Large trials suggest that rate and rhythm control strategies in patients with stable heart failure are equivalent, but there are few data regarding the management of AF in ADHF. We need more robust evidence before recommending pulmonary vein isolation more widely.

Bradyarrhythmia is also common in heart failure, and pacemaker implantation is often necessary. The recently published results of the BLOCK-HF* study suggest that biventricular rather than conventional pacing may be better.

Dr John Baxter (Sunderland) again showed why he is one of the Society’s favourite speakers, and why it is always unfair to have to speak after him. Using the extended metaphor of Steptoe and Son, he stressed the importance of the heart failure team offering an outreach service and not being an isolationist.

*A list of study acronyms can be found on page 7.
service inhabiting the ivory tower next to the catheter laboratory. Heart failure happens in older patients, and they stand to benefit from meeting the team interested in treating their condition.

Finally, Dr Simon Williams (Manchester) discussed the problem of myocarditis as a potential cause of acute heart failure. There is very little information to guide the treatment of myocarditis specifically. The rare patient with giant cell myocarditis might benefit from steroid therapy, and making the diagnosis is probably the only indication for myocardial biopsy in acute heart failure. Treatment should otherwise be with conventional medication: and the outlook is good compared with heart failure following MI.

Session 4: Philip Poole-Wilson Lecture

The Philip Poole-Wilson lecture was given this year by Professor Sian Harding (London). Professor Harding had worked with Philip over many years, and is a key figure in the development of isolated cardiac myocyte models. Her extraordinarily stimulating lecture focused on novel and potentially important features of β-blockers in heart failure. She highlighted the potential role of ligand bias agonism, the process by which different signal transduction paths can be followed following the stimulation of a single receptor by different ligands.

She concluded by exploring a rat model of Takotsubo cardiomyopathy. Takotsubo is characterised by cardiac failure with very high circulating levels of catecholamines. In the rat model, adrenaline was found to induce (reversible) heart failure, which could be prevented by blocking cardio-inhibitory pathways linked to the β2-receptor. Thus, low levels of adrenaline acting at the β2-receptor cause cardiac stimulation, but very high levels result in cardiac inhibition.

We were delighted to welcome Mary Poole-Wilson who presented the Poole-Wilson medal to Professor Harding.

Session 6: Hyde Park

Beyond the good humour (and occasional assault with table tennis balls), the Hyde Park session allows some unusual, but potentially important, ideas to be aired. Dr Nigel Rowell (Middlesbrough) outlined his proposals for modelling the NHS on budget airlines, rather than on Emirates; Dr Kevin Goode (Hull) exposed concerns that telemonitoring of patients might become a way of rationing care – if patients don’t comply, why should the system continue to care for them?; Dr Darrel Francis (London) explored the statistically woolly thinking underlying the concept

Session 5: Heart failure research in the UK

In Session 5, Professor Andrew Clark (Hull) highlighted the potential role of the BSH in facilitating research in heart failure in the UK. The first ever BSH Research Fellowship was awarded to Dr Jane Cannon of Glasgow, who will carry out research on the vital area of cognitive impairment in heart failure. We are delighted to acknowledge the very generous support of Servier UK in making the fellowship possible, and were pleased to welcome Mr Brad Lloyd, the Sales Director for Servier UK, who made the award in person.

The young research worker presentations followed. Dr Pierpaolo Pellicori (Hull) presented his work on the relation between cardiac morphology and surface ECG variables; Dr Ahmad Shoaib (Hull) presented data emphasising that the commonest symptom amongst patients admitted with heart failure is not breathlessness at rest; and Dr Donah Zachariah (Portsmouth) presented her work on the potential benefits of cardiac resynchronisation therapy (CRT) in patients with renal failure. The award was ultimately made to Dr Pellicori.
of ‘response’ (or otherwise) to CRT; and Dr Simon Williams (Manchester) demonstrated his amazement at the lack of evidence for the old wives’ tale that exercise during a viral infection might induce heart failure by jogging out of the auditorium in Lycra.

Dr Simon Williams

A middle-aged man in Lycra: Dr Simon Williams

Session 7: Cases

Day two began with Session 7, which comprised four case presentations that spanned the four corners of the heart failure world: from the gradual fall from grace of digoxin – the oldest treatment of all – to the organisational complexities of establishing the full evidence base for optimal therapy in a young mother with a dilated cardiomyopathy; from the ‘goddess of echo’ and the Olympus of the first ‘reverse transcatheter, aortic valve implantation (TAVI)’ across the mitral valve to the mortal pain of the patient living with heart failure. A session of progress, but also of perspective: the highlight being a recording of an interview between Dr Rob Howlett (Thaxted) and his patient, whose life was turned around by deliberate neglect of a diagnosis of diabetes.

Session 8: Heart failure with normal ejection fraction

Session 8 was given over to the problem of heart failure with normal (or preserved) ejection fraction, or possibly diastolic heart failure. Professor Walter Paulus (Amsterdam, Netherlands) delivered one of the highlights of the meeting: the keynote lecture ‘Heart failure with normal ejection fraction [HFNEF] – what is it?’ In his entertaining, yet fascinating, presentation he proposed a new paradigm for HFNEF, which shifts our thinking from it being a condition caused by increased systemic vascular resistance to that of a systemic proinflammatory state caused by common metabolic co-morbidities such as obesity, diabetes and hypertension. This promotes coronary microvascular endothelial inflammation resulting in the stiffening of cardiomyocytes and cardiac interstitial fibrosis leading to reduced diastolic compliance and consequently raised left ventricular end-diastolic pressures. His description of his case history of an archetypal sufferer, the legionnaire in a van, will live with the audience for some time.

In his presentation on echocardiography beyond ejection fraction, Professor Alan Fraser (Cardiff) discussed the central role of echocardiography in assessing left ventricular diastolic function and in confirming a diagnosis of HFNEF. The E/e’ ratio, although still commonly used, has many limitations to the assessment of diastolic function. It should not be used alone, but in combination with other variables such as left atrial volume. Where appropriate, diastolic stress testing may be useful where initial assessment is inconclusive.

In his presentation on the treatment of HFNEF, Professor Martin Cowie (London) reminded us that no treatment has been shown convincingly to reduce mortality or morbidity in this condition. Diuretic therapy remains central to managing breathlessness and fluid retention, as does addressing co-morbidities such as hypertension, AF, myocardial ischaemia and diabetes. The recently published results of the TOPCAT trial using spironolactone in patients with HFNEF failed to show benefit for the primary clinical composite outcome of cardiovascular death, heart failure hospitalisation and surviving cardiac arrest.

In the last presentation of this session Dr Luke Howard (London) addressed pulmonary hypertension in heart failure, outlining the importance of dealing with the primary causes of raised left atrial pressure and treating contributory co-existing conditions such as chronic lung disease.
**Session 9: Acute heart failure trials update**

In Session 9, Professor John McMurray (Glasgow) presented an overview of recent and ongoing trials in heart failure. Beginning with a summary of the large number of historical trials showing no evidence of benefit, Professor McMurray then moved on to describe recent trials with similar outcomes, focusing on the recent TOPCAT trial with spironolactone in patients with HFNEF. Professor McMurray described the disappointing lack of treatment benefit in this study and touched on geographical differences in event rate that may have had bearing on the final trial result. If the study had been restricted to North America only, then it may have proved positive.

Low-dose dopamine is often thought to enhance renal perfusion and maintain renal function in patients with worsening heart failure. However, neither the addition of low-dose dopamine nor of nesiritide (not licensed in the UK) made any difference to clinical outcome when added to standard treatment in the ROSE-AHF trial.

Professor McMurray then went on to describe and discuss ongoing trials, including those with the novel agents, omecamtiv and serelaxin. The findings of the RELAX-AHF study suggested that there might be a role for serelaxin in patients with acute heart failure, but the beneficial effects on outcome were not statistically robust. Further work is needed.

**Session 10: What else can we do in advanced heart failure?**

Session 10 examined the possible treatment options for people with very severe heart failure in hospital. Dr Roy Gardner (Glasgow) discussed the approach to the hypotensive patient, recognising that hypotension was often simply an indicator that the outcome was very poor. However, it’s vital to realise that the absolute level of blood pressure is not important if the brain and kidneys are well perfused: many patients are content with systolic blood pressure well under 100 mmHg, and a low blood pressure is not of itself an indication to stop treatment.

Dr Peter Cowburn (Southampton) described his experience in delivering CRT therapy to patients hospitalised with severe heart failure. In carefully selected patients, particularly those with very broad QRS complexes and with a good ‘on-table’ blood pressure response, excellent outcomes are possible. Dr Steve Shaw (Manchester) then discussed indications for ventricular assist device (VAD) implantation. His major concern was that the devices should be implanted only in patients for whom a long-term definitive strategy is available: there is little worse than realising that a patient has had a left VAD implanted and that there is no prospect of either a long-term VAD or a transplant.

**Session 11: Clinical challenges in the management of acute heart failure (2)**

Session 11 dealt with further problems associated with acute heart failure. Dr Derek Connelly (Glasgow) discussed the role of implantable cardioverter defibrillator (ICD) implantation in patients with acute heart failure. We were reminded of the indications for implantation of an ICD. Clearly, for those presenting with a life-threatening arrhythmia, implantation during the index admission is strongly indicated. However, for primary prevention, he highlighted that to be eligible for an ICD, patients with dilated cardiomyopathy should have had heart failure for at least 3 months; and those with ischaemic heart disease should wait 6 weeks after any MI. Patients presenting with severe heart failure may also have indications for biventricular pacing and in these circumstances the addition of a defibrillator may also be appropriate.

A more challenging group of patients is those with poor left ventricular function who are at high risk of sudden cardiac death within the first 3 months, but who are not yet considered ICD candidates. These patients should be carefully monitored and any ‘red-flag’ symptoms, such as
syncope, should trigger reconsideration. The role of other technologies, such as wearable defibrillators, was discussed – but the evidence for their use is weak. Although imaging biomarkers (late gadolinium enhancement on cardiac magnetic resonance or MIBG [metaiodobenzylguanidine] imaging) are used for risk stratification, they lack strong evidence in the acute setting.

Dr Carol Whelan (London) talked about amyloidosis and the heart. She discussed the various forms of cardiac amyloidosis. Whilst AL amyloidosis (arising from a blood dyscrasia) is relatively uncommon (6–10 cases per million population), it is thought that other forms of cardiac amyloid (in particular senile systemic amyloidosis) is under diagnosed. She presented the clinical features differentiating between the different types of amyloid, but there is significant overlap and so cardiac biopsy with appropriate staining and gene sequencing are required in order to make the correct diagnosis with confidence – for example, a case was presented where a patient with monoclonal gammopathy of unknown significance had wild-type transthyretin amyloid and did not require chemotherapy.

Imaging plays an important role in the initial diagnosis of amyloid and the features of CMR and DPD (diphosphono-1,2-propanodicarboxylic acid) scintigraphy were demonstrated. Although the outlook for AL amyloid remains poor and there are currently no specific therapies for transthyretin amyloid, there are new therapies on the horizon.

The final talk of the session was on peripartum cardiomyopathy (PPCM) by Dr Mark Petrie (Glasgow). The outcomes are, perhaps, not as bad as one might think. He discussed the management of PPCM – essentially conventional heart failure therapy. The evidence for the routine use of bromocriptine is not strong and a number of patients recover left ventricular function without it. During the discussion there were questions over the use of heart failure medications in breast-feeding mothers, and to the question of whether he ever stopped heart failure medication in these patients, Dr Petrie answered a rather emphatic “no”. His final plea was to encourage clinicians to participate in the ESC Heart Failure Association’s PPCM registry.

**Session 12: The debate**

In the final session, the traditional trouncing of Professor Andrew Clark (Hull) in the annual debate took place as he tried to defend the notion that hospitalisation for heart failure might be good for you. Dr Dargoi Satchi (Stoke-on-Trent) won hands down and advanced the cause of the ambulatory heart failure unit somewhat – surely a development for the future.

**Closing remarks**

The annual meeting of the Society goes from strength to strength. Over 700 registered delegates was a first for us this year, meaning that we will need to re-configure the meeting room next year! The excited buzz and great good humour surrounding the meeting come from you, the membership, and long may that continue.

A particular word of thanks is due to those who took the time to fill in the feedback sheets. The meeting is owned by the membership of the Society, and the feedback we get is invaluable in helping next year’s directors in developing the programme. We have already planned the layout of the meeting next year to accommodate the increasing number of delegates, and we look forward to seeing you again in November 2014.

Thanks to Andrew Clark, Ceri Davies, Dominic Kelly, Hugh McIntyre, Jim Moore and Iain Squire for their contributions to this article. Photos courtesy of Dr Roy Gardner.
Study acronyms

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<tr>
<th>Acronym</th>
<th>Description</th>
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<tbody>
<tr>
<td>BLOCK-HF</td>
<td>Biventricular versus Right Ventricular Pacing in Heart Failure Patients with Atrioventricular Block</td>
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<tr>
<td>RELAX-AHF</td>
<td>Efficacy and Safety of Relaxin for the Treatment of Acute Heart Failure</td>
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<tr>
<td>ROSE-AHF</td>
<td>Renal Optimization Strategies Evaluation in Acute Heart Failure</td>
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<tr>
<td>TOPCAT</td>
<td>Aldosterone Antagonist Therapy for Adults With Heart Failure and Preserved Systolic Function</td>
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BSH Secretariat
‘Nought’ The Farthings, Marcham, Oxfordshire OX13 6QD, UK
Telephone: 01865 391836
Email: info@bsh.org.uk
Website: www.bsh.org.uk
Twitter: @BSHeartFailure

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