BSH Board Election

The result of the BSH Board Election was announced at the EGM during the British Cardiac Society (BCS) Annual Scientific Conference in April. We are delighted to welcome some new faces to the BSH Board. Lynda Blue (Glasgow), a previous Observer to the Board, who has been working hard developing the HF nurses’ training programme, was elected as a Councillor, together with Professor Martin Cowie (Imperial College Heart and Lung Institute, London) and Dr Iain Squire (University of Leicester).

The new Chairman, Professor Henry Dargie, thanked Dr Mick Davies for his hard work over the past two years during which he has been Chairman. In this time, alongside the Committee, he has developed an exciting programme of meetings and new activities for the BSH.

The Society is also extremely grateful to two further founding members who are now retiring having served on the BSH Board since the initiation of the Society in 1998:
- Professor Richard Hobbs, Treasurer throughout this time, who has helped develop sound accounting systems and a secure financial base for the Society.
- Professor Philip Poole-Wilson, the original Chairman of the Society whose vision and hard work in the field has, without doubt, helped to raise awareness of HF.

New Board

We are pleased to confirm that the BSH Board for the period June 2003–May 2005 comprises:

Chairman: Professor Henry Dargie (Glasgow)
Past Chairman: Dr Mick Davies (Birmingham)
Chairman-Elect: Professor John Cleland (Kingston upon Hull)
Deputy Chairman: Dr Suzanna Hardman (London)
Treasurer: Dr Theresa McDonagh (Glasgow)
Councillor: Mrs Lynda Blue (Glasgow)
Councillor: Professor Martin Cowie (London)
Councillor: Dr Iain Squire (Leicester)

Meetings and activities

BSH meetings in 2003

This newsletter reports on the BSH symposia held at the BCS Annual Scientific Conference in April this year. The practical theme of the sessions attracted record numbers of delegates and it was deemed an outstanding success.

The principal focus of this year’s Annual Autumn Meeting is on how to manage advanced HF in patients for whom medical treatment alone is no longer sufficient. The Programme Director is Mr Stephen Westaby, a distinguished cardiac surgeon from Oxford, well known for his pioneering research into the use of left ventricular assist devices. It will take place in Oxford on Friday 28 November. For more details please contact the BSH Secretariat or see our website (www.bcs.com/affiliates/bsh.html).

Heart failure nurse training programme

The BSH, in partnership with the British Heart Foundation and the Glasgow Caledonian University, is offering the first formal academically accredited educational programme to prepare registered nurses and other allied health care professionals to manage chronic HF. The course, entitled ‘Principles of Care Management’ was established in 2002 and runs three times a year. The starting dates for 2004 are February, June and September. In addition, an e-learning format is being developed to commence in 2004.

For further information on the course content please contact m.wright@gcal.ac.uk or y.millerick@btopenworld.com.

Application forms can be obtained from:
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From atrial fibrillation to heart failure and from heart failure to atrial fibrillation

A BSH session at the BCS Annual Scientific Conference on 28th April 2003, chaired by Suzanna Hardman (London) and Mick Davies (Birmingham)

Nearly a third of HF patients have atrial fibrillation (AF) and both AF and HF are associated with embolic phenomena. There is considerable interest amongst clinicians about how to manage these patients as they can be difficult to treat. One of the topics debated passionately in the medical world is whether AF patients should be restored back to sinus rhythm or not. The speakers reviewed the evidence for and against this approach, touching upon electrophysiology of the heart, the treatments available and how to translate the results from clinical trials into real-life practice.

From atrial fibrillation to heart failure and from heart failure to atrial fibrillation. The epidemiology and prognosis of these interactions

Dr Russell Davis (Birmingham) discussed the relationship between left ventricular systolic dysfunction (LVSD) – the most common cause of HF – and AF.

Both LVSD and AF share similar risk factors: ischaemic heart disease, hypertension, diabetes and advancing age. Atrial fibrillation can precipitate the development of overt HF in patients with LVSD through reduction of cardiac output, and the presence of both conditions confers a high risk of embolic stroke. Dr Davis discussed evidence from small trials about the negative effects of AF on haemodynamics and cardiac output, before moving on to look at much larger epidemiological studies that confirm the AF-HF link:

- The Framingham study\(^1\) revealed that men and women with AF have a 4.5-fold or 5.9-fold greater risk of developing HF, respectively, than those in sinus rhythm.

- In the Hillingdon study\(^2\), just over 30% of those admitted to hospital with suspected HF also suffered from AF episodes, with AF being considered to be the sole aetiology in 5% of the patients.

- The ECHOES screening study\(^3\) investigated over 3000 randomly selected patients aged over 45. Similar proportions of patients were found to have either a low left ventricular ejection fraction (EF) (1.8%) or AF (2.0%) and the survival rates for both conditions were also similar (84.7% and 87.2% respectively) and worse than for the population overall (96.5%). Nearly 40% of patients with AF were symptomatic and 0.6% had HF due to AF.

It would therefore perhaps be expected that it would be beneficial for a patient with left ventricular dysfunction (LVD) to be in sinus rhythm. Data from the Italian Network on Congestive Heart Failure study\(^4\) and the ECHOES study\(^3\) do support the theory that HF patients in sinus rhythm have a better prognosis than those in AF. However, results from the Hillingdon HF study do not: at a median of 16 months follow-up, the hazard ratio was significantly worse for HF patients who were in sinus rhythm, rather than AF\(^5\).

Dr Davis said that the major HF treatment trials give conflicting evidence about the benefits of ACE inhibitor or beta blocker therapy in patients with AF and HF. For example, in the CIBIS II trial\(^6\), bisoprolol only reduced mortality for those in sinus rhythm, whereas the US carvedilol studies showed a significant benefit for AF patients taking carvedilol compared with the placebo group\(^7\).

It is clear that there is a relationship between HF and AF: many people have both conditions and as LVD progresses the AF risk increases. What are currently unclear are the prognostic implications of this combination and how it should be managed effectively.

Recent developments in our understanding of normal and abnormal pacemaker activity in the atria

Dr Mark Boyett (Leeds) took a detailed look at the normal and abnormal pacemaker activity of the heart from a basic scientist’s point of view and updated the audience on recent advances in scientific knowledge.

Beginning with the sino-atrial (SA) node, he explained that until recently sodium (Na\(^+\)) channels were not thought to have a role in pacemaking. Scientists only thought that the cardiac Na\(^+\) channel, which is absent in the SA node, was expressed in the heart. However, the neuronal Na\(^+\) channels Nav1.1 and Nav1.3 have since been found in the SA node. In experiments with mice, TTX (an agent to which only the neuronal sodium channels are sensitive) both slowed the heart rate of the mice and increased heart rate variability, producing an effect that is similar to sick sinus syndrome in humans\(^8\). This confirms that Nav1.1 does play a role in pacemaking.

Dr Boyett then turned his attention to the role of intracellular calcium in pacemaking in the SA node. It is suggested that before an action potential arises a ‘calcium transient’ occurs: calcium is released from the sarcoplasmic reticulum (SR) of the cell, which activates an inward Na\(^+\)-Ca\(^{2+}\) exchange current. This leads to depolarisation of the cell and generation of an action potential. It is generally agreed that activation of the ryadonine receptor (RYR2) leads to release of Ca\(^{2+}\) from the SR. Researchers have carried out experiments to block this receptor with ryadonine, therefore inhibiting normal Ca\(^{2+}\) release and the calcium transient. Some have reported that blocking this receptor and reducing the calcium transient abolishes pacemaking\(^9\), whereas others say it only slows it down\(^10\). Curiously, whereas ryadonine has been shown to slow or block the activity of the SA node, it has conversely been shown to promote pacemaker activity to the pulmonary veins instead and this may be important for AF\(^11\).

Dr Boyett described briefly other potential pacemaking sites within the atria. One site is around the tricuspid
valve, where the ion channel HCN4, only previously identified in the tissue of the SA and atrio-ventricular (AV) nodes, has been found. The role of this ion channel in the tricuspid valve tissue is unclear, however it does play an important role in the pacemaking of the SA and AV nodes.

The atria have a multitude of potential pacemaker tissues, which could be involved in AF as well as the heart’s normal rhythm. Fortunately, scientists are now beginning to identify the molecular basis of pacemaking in these tissues.

From heart failure to atrial fibrillation and back again: understanding the underlying electrophysiology

Professor Maurits Allessie (Maastricht, The Netherlands) gave the audience a detailed introduction to the underlying electrophysiology of AF. He explained that over time, paroxysmal AF (where AF episodes stop on their own) often becomes persistent and it grows increasingly difficult to return patients to sinus rhythm with antiarrhythmic drugs or cardioversion. This progression occurs because during the first days of AF, a process termed ‘electrical remodelling’ occurs, whereby the atrial refractory period gradually shortens, increasing the frequency of fibrillation and consequently promoting the stability of the arrhythmia – hence the phrase ‘AF begets AF’.

There are other processes, however, that are involved in the change from sinus rhythm to AF. Professor Allessie’s experiments have led him to believe that chronic atrial stretch is one of the mechanisms that plays a key role in the pathophysiological relationship between AF and HF and that it is both a cause and a consequence of AF.

Looking at how atrial stretch may be a cause of AF, Professor Allessie described experiments in chronically instrumented goats that showed a significant correlation between the degree of atrial dilatation and the duration of AF episodes \(p<0.01\). Studies in humans and animals have suggested that chronic atrial stretch induces activation of signalling pathways that lead to cellular hypertrophy, fibroblast proliferation and tissue fibrosis, all of which cause atrial dilatation and promote conduction disturbances.

Other experiments in the goat have shown that induced episodes of AF can significantly reduce the work index of the atria so that they are almost paralysed. Of interest is the fact that the loss of atrial contractility (contractile remodelling) followed exactly the same time course as the electrical remodelling, suggesting that they are due to the same mechanism, possibly the downregulation of L-type calcium channels.

Concluding, Professor Allessie said that the structural cascade of remodelling is the most important drive behind AF in HF and to prevent AF in future it is crucial that research focuses on the cellular mechanisms involved in atrial stretch.

Overview of recent studies: to restore sinus rhythm or not?

There are many obvious advantages for people’s hearts to remain in sinus rhythm, said Professor Stuart Cobbe (Glasgow), these are:

- Superior rate control
- Improved ventricular filling
- Maintenance of the haemodynamic contribution of atrial systole (provided that the atria are capable of contracting)
- A reduced thromboembolic risk.

However, there are disadvantages associated with antiarrhythmic drugs used to preserve sinus rhythm, as ironically they can have the reverse effect and be pro-arrhythmic. Also there are concerns about the futility of maintaining sinus rhythm in patients with substantial changes to the structure and function of their atria. In the long term it may be wiser to accept that many patients will remain in AF and seek other ways of improving symptoms and survival instead, said Professor Cobbe.

In a trial that studied people with persistent AF, even an aggressive protocol of electrical cardioversion and antiarrhythmic therapy failed to maintain sinus rhythm in the majority long term – at 4 years only 27% remained in sinus rhythm. The group identified 3 major predictors of failure: prolonged AF (>36 months), poor effort tolerance and age >56 years. Most HF patients are above this age.

Professor Cobbe looked at the issue of ‘rate control’ (using heart rate slowing medication) versus ‘rhythm control’ (cardioversion and antiarrhythmic drugs), with reference to three major randomised controlled trials. The largest of these was the AFFIRM study, which enrolled over 4000 AF patients. Over 20% had a history of HF or echocardiographic evidence of LV dysfunction. Patients were randomised to either rate or rhythm control strategies and all additionally received warfarin, which could be discontinued if prolonged sinus rhythm was achieved. There was a slight mortality difference between the two groups in favour of the rate control strategy \(p=0.08\) (Figure 1). Of note is that the majority of strokes that occurred in the trial were in patients in whom warfarin had been discontinued – a powerful reinforcement of its benefits.

No clinical trials have solely investigated the effects of maintenance of sinus rhythm in HF patients with AF. However, retrospective analyses of the CHF-STAT and DIAMOND trials have been carried out focusing on AF patients. In CHF-STAT the antiarrhythmic drug amiodarone was effective in controlling ventricular rate \(p=0.001\) and achieving rhythm control \(p=0.002\) in those in AF at baseline. Although treatment did not decrease the mortality risk in all AF patients, those who converted to sinus rhythm did benefit \(p=0.04\). In the overall study population, fewer patients in the amiodarone group developed AF \(p=0.005\).
A similar story is true for the DIAMOND studies, which investigated the effects of dofetilide, alone or in conjunction with electrical cardioversion, in post-MI or HF patients. A pooled analysis of 506 patients in AF or flutter at randomisation revealed that, again, although treatment did not reduce the overall mortality risk, those in the treatment group who converted to sinus rhythm achieved better survival rates. Consequently, it can be concluded from these two studies that people who remain in or relapse into AF have a poorer prognosis than those who are restored to sinus rhythm. However, Professor Cobbe remarked that this might be more due to the extent of the underlying disease found in these patients than the actions of the treatments.

Although Professor Cobbe said that he was in no doubt that restoration of sinus rhythm is associated with haemodynamic benefits, he explained that the balance between the positive and negative aspects of therapies to restore sinus rhythm has not been fully established. There is a clear need for new antiarrhythmic drugs and therapies to be developed that are safer and more effective for this condition.

Management of atrial fibrillation in heart failure: from clinical trials to patient care

Professor Martin Cowie (London) looked at the more practical aspects of treating HF patients with AF.

Between 5 and 10% of the elderly population have AF24 and the prevalence of this arrhythmia in the HF population is much greater – in the Euroheart Heart Failure Survey25 it was 43% and in the Hillingdon Study2 it was present in 30% of all new HF patients. Although some people are asymptomatic and therefore unaware of being affected by AF, it does greatly increase the risk of thromboembolism and, consequently, stroke.

Historically, cardiologists have been taught to opt for rhythm, rather than rate control, using escalating doses of antiarrhythmic drugs, from the least to the most toxic, alongside electrical cardioversion and anticoagulation therapies. The cycle of restoring a patient to sinus rhythm, followed by relapse into AF, and returning them to sinus rhythm again often continues until the clinician agrees that it is futile and then a rate control approach is taken. However, this rigid strategy that has been taught to generations of medical students is now being questioned, chiefly because there is little evidence to support it. For patients with HF, there will be no specific trial data available until the results from the ‘rate versus rhythm’ control study in HF patients with AF (AF-CHF) are reported in 2005. In the meantime, the ESC, AHA and ACC have published joint guidelines on how to manage these patients in different clinical situations (see: http://www.escardio.org/scininfo/guidelines/atrialfibrillation.pdf). These guidelines question the perceived superiority of the rhythm control strategy (as mentioned by various speakers), discuss the safety and efficacy of various pharmacological options and advise on the newer non-pharmacological approaches available. NICE have also published some guidelines on HF in July this year, which discuss the care of patients with both AF and HF. They can be downloaded from the NICE website at this address: http://www.nice.org.uk/pdf/C5NICEguideline.pdf.

Professor Cowie advised that before any treatment is initiated a careful history and assessment of the patient should be taken. When treating HF patients with AF, stabilising the HF will often be the most sensible first step (some HF drugs, such as ACE-inhibitors and angiotensin receptor antagonists even have antiarrhythmic effects). Once this has been achieved, aggressive treatment is likely to be given; firstly with amiodarone alone and then with electrical cardioversion, if necessary. However, once this process has been tried once or twice without success, adopting a ‘rate control’ strategy is appropriate. The most important focus of therapy, however, is to prevent patients from suffering an embolic stroke, and unless there is a strong reason not to do so, anticoagulation with warfarin is necessary. Unless, it is also crucial to carry out regular clinical reviews of patients, particularly addressing their symptoms, treatment and adequacy of anticoagulation.

Screening for asymptomatic left ventricular dysfunction – has the time come?

A BSH session at the BCS Annual Scientific Conference on 29th April 2003, chaired by Henry Dargie (Glasgow) and Philip Poole-Wilson (London)

Brain natriuretic peptide (BNP) testing is a simple procedure to screen for HF in symptomatic patients and it is now almost routinely used in clinical practice. Several studies have suggested that it is also possible to screen for asymptomatic left ventricular dysfunction (ASLVD) using BNP. This session investigated the evidence supporting this rationale, whether it is cost-effective and how it could be targeted to identify those with ASLVD at greatest risk.
Asymptomatic left ventricular dysfunction – is it common and does it reside in recognisable groups?

Professor David Mant (Oxford) explained that data from five big community studies suggest that the prevalence of definite (moderate or severe LVSD) is between 2 and 4%, and that the prevalence of mild or borderline systolic dysfunction is between 7 and 12%. Approximately half of these cases are asymptomatic.

Similar to hypertension, the distribution of LVSD is not bimodal and therefore there is no clear echocardiography 'cut off point' to define it (Figure 2).

However, not all ventricular dysfunction is systolic, and including patients with preserved systolic function might double the estimated prevalence of LVD. The overall LVD prevalence can be assessed by echocardiography (i.e. by measuring the E/A ratio in diastole as well as the systolic ejection fraction), but it is more easily and reliably estimated by testing the person’s blood BNP level. The BNP level not only shows a more consistent relationship with functional status and prognosis than systolic ejection fraction, but it is also raised in HF syndromes with preserved left ventricular systolic function. BNP testing was used in the Poole community study, and with a BNP cut-off point of >30pmol/l as a marker of LVD (an appropriate level for characterising systolic dysfunction in that study) the overall prevalence estimate of LVD was 20% in patients aged between 70 and 85.

Does ASLVD reside in recognised groups? Data from the principal community studies show that, unsurprisingly, the prevalence is higher in those with recognised vascular disease: diabetes, MI, stroke, angina and hypertension. Systolic dysfunction is extremely common in people after MI (over 40% have at least borderline dysfunction, approximately a third of which is asymptomatic); diastolic dysfunction may be commoner in the other conditions which are more associated with decreased cardiac compliance. Systolic dysfunction increases with age and is more common in men than women (pooled data from Rotterdam and Birmingham reveal that there is a 3-fold difference between men and women aged 85+). Therefore targeting screening by age and gender and risk factors is sensible, said Professor Mant.

He concluded that ASLVD is common and can be found in patient populations with vascular disease. Including diastolic dysfunction, approximately 10% of all those aged over 70 may have some degree of LVD that could be treated in the context of a screening trial.

Asymptomatic left ventricular dysfunction – is it serious and treatable?

Professor Martin Cowie (London) discussed whether clinicians need to identify and treat people with ASLVD – does it have a serious implication on life expectancy, morbidity or quality of life? As Professor Mant discussed, the prevalence of ASLVD is quite high. In the Echocardiographic Glasgow Study, the prevalence of asymptomatic and symptomatic LVD was the same in the adult population studied (1.5%) and, surprisingly, the mortality rate after 4 years was also the same (21%)3. This finding is fairly consistent with data from the SOLVD and SAVE studies.

Further evidence for the prognostic implications of ASLVD is gained from the Framingham study, which revealed that people with no symptoms but a dilated left ventricle are at much greater risk of developing HF than the general population. Although it may seem sensible to screen for ASLVD for this reason, often it is an acute cardiac event that shifts the person from a ‘normal’ left ventricular ejection fraction to asymptomatic (or even symptomatic) LVD. Targeting efforts at coronary heart disease prevention should pay dividends in preventing subsequent HF.

Having established that ASLVD is serious, Professor Cowie assessed whether it is treatable. The only evidence from treatment trials comes from those that...
principally enrolled patients with pre-existing coronary artery disease (often with an acute event in the recent past), such as SOLVD prevention\textsuperscript{33}, SAVE\textsuperscript{34} and TRACE\textsuperscript{36}. These trials provided convincing evidence that ACE inhibitors are beneficial for such patients in reducing the risk of the development of HF and hospitalisations for HF. The results also suggested a reduction in mortality. Similarly, in the CAPRICORN study, the beta blocker carvedilol improved survival in recent MI patients with evidence of LV systolic dysfunction\textsuperscript{37}. Yet it is important to remember that the groups studied in these trials are selected and are not wholly representative of those who would be identified as having ASLVD in the general population.

In addition, it is likely that if a screening programme were to be carried out, many of those identified as having ASLVD would already be on medication that prevents or slows the onset of HF, in particular ACE inhibitors, for other indications. In the Heart of England study\textsuperscript{38} for example, nearly 30% of patients with previously undetected ASLVD were taking ACE inhibitors or beta blockers at the time and the proportion would probably be even greater now.

Professor Cowie said that although evidence suggests that the presence of ASLVD increases the risk of morbidity and mortality, he remains undecided as to whether screening for ASLVD is cost effective. If it were to be carried out it would have to be carefully targeted to high risk groups. Instead, it may be more efficient to invest resources into high risk primary and secondary prevention measures, which are likely to have an impact on the development and progression of ASLVD.

**Asymptomatic left ventricular dysfunction – can we detect it using natriuretic peptides?**

Dr Theresa McDonagh (Glasgow) examined the theory and evidence supporting the potential use of BNP testing in screening for ASLVD. Also she touched on how the screening could be carried out and what clinicians could learn from the tests.

It has long been known that circulating plasma concentrations of A and B type natriuretic peptides are higher in those with LVD, even if it is asymptomatic, and there is good evidence that BNP and N-terminal atrial natriuretic peptide (ANP) are useful markers for this condition\textsuperscript{39,40,41}. However, Dr McDonagh pointed out that although these peptides are markers it doesn’t necessarily mean that they are effective as screening tools for ASLVD.

Some evidence supporting the use of natriuretic peptide testing in this context comes from the MONICA study\textsuperscript{42}. This investigated 1252 randomly selected people aged 25–74 and 1.4% had ASLVD on echocardiography. BNP was more effective than N-terminal ANP in screening, with a reasonable area under the curve. More recently Smith et al.\textsuperscript{27} carried out a study on elderly patients and found that BNP had a high negative predictive value – i.e. if you have a low concentration you are unlikely to have LVD.

However, data from the Framingham study was not as complimentary to this screening technique\textsuperscript{43}. BNP testing was not effective at detecting ejection fractions below 50%, but at below 40%, the results were better. Dr McDonagh pointed out that the measurement of LVD in this study was not as rigorously performed as in others.

Targeting screening to a high risk population is much more sensible strategy, Dr McDonagh said. In the North Glasgow Study\textsuperscript{44}, subjects aged over 55 with ischaemic heart disease were screened for asymptomatic LVD. BNP performed well, with a similar effectiveness to mammography for breast cancer screening and cytology for cervical carcinoma. Following on from this, the BEACON study\textsuperscript{45} tested patients from 7 general practices in South Glasgow, who were receiving medication for cardiovascular disease or diabetes. The investigators found that the best method of testing was using BNP to ‘rule out’ those without LVD, rather than referring to ‘abnormal’ BNP values to confirm whether a patient has LVD or not. This is because there is no real ‘normal’ BNP value as concentrations rise with age (exponentially over the age of 70).

In this fairly high risk population, BNP testing identified 7.7% and 18% of the population as having LVD using ejection fraction cut off points of <35% and <40%, respectively. It also identified 2.7% and 8.3% of patients as having ASLVD, using the same criteria. BNP testing did not pick up as many cases of definite LVD as the ECG, but when the EF cut-off point was extended to <40% to include mild cases, BNP picked up more. Both tests identified similar numbers of people with untreated LVD who would benefit from ACE inhibitors or angiotensin receptor antagonists.

Dr McDonagh agreed that BNP could be used to screen for ASLVD as a ‘rule out’ test and it would be preferable to performing an ECG, but it is currently difficult to know what a positive test would mean for the patient. For this reason, she believes that further research is required to confirm whether it should be used for this purpose.

**Asymptomatic left ventricular dysfunction – would screening using BNP testing be cost effective?**

Dr Olav Nielsen (Copenhagen, Denmark) explained that in healthcare there are three main types of economic analysis: cost minimisation, cost-effectiveness and cost utility:

- **Cost minimisation analysis**: Compares strategies of equal effectiveness to determine which is least expensive.
- **Cost effective analysis**: Compares ratios of the incremental cost over the incremental effectiveness of alternative strategies.
- **Cost utility analysis**: Compares ratios of the incremental cost over the incremental utility. For example, cost per life year or cost per quality adjusted life years.

Adapted from Earle et al. Annals of Oncology, 1998; 9: 475–82
However, problems can be encountered when making these calculations. Firstly, it is very difficult to state what is an acceptable cost per life-year gained. Secondly, when assessing the impact of a screening programme, two analyses need to be carried out. In the case of ASLVD, firstly the screening procedure needs to be sensitive to identify an adequate number of people from the population with the condition. Then to realise the benefit of screening, the treatment needs to be cost effective.

Dr Nielsen began by examining the second phase of this process first – whether it is cost effective to treat LVSD, referring to analyses from the SAVE47,48 SOLVD prevention49 and HOPE ACE inhibitor studies50 (medium and high risk patients). Although they did not study the ASLVD population specifically, they enrolled patients who were either at a presumably similar risk of major cardiovascular events or had evidence of LVD and in these patients ACE inhibitor treatment is generally considered to be cost effective (Figure 3). Based on these studies Dr Nielsen expected that the numbers needed to treat (NNT) to prevent one event would be between 20 and 40 people over a 5-year treatment period. However, he stressed again that this was an estimate as none looked exclusively at patients with ASLVD, and, in addition, none of the patients were detected incidentally in the community.

Having established that treatment is worthwhile, Dr Nielsen returned to the first question: how many people need to be screened for a given time period in order to prevent one death or adverse event if screening is followed by treatment? The number of subjects needed to screen (NNS) to prevent one event can be obtained from clinical trials directly answering that question. As these data are not available it can be estimated from knowing the prevalence of ASLVD and the number needed to treat (NNT). This is the number of subjects with ASLVD that would need to be treated for a given duration in order to prevent one cardiovascular event. Dividing the NNT (e.g. 20) by the prevalence of the disease (e.g. 3%) yields the NNS = 667 for five years’ treatment.

Calculations based on these estimates reveal that the numbers needed to screen for ASLVD with echocardiography are comparable with other strategies, such as cancer screening51.

Yet, although echocardiography is the most effective screening tool for ASLVD, screening large numbers of people with echocardiography would be extremely demanding on resources. Studies have shown that using a BNP test as pre-screening measure before an echocardiogram is significantly cost minimising52 even with a high rate of false positive BNP values. Choosing a higher BNP concentration cut off point would reduce the false positive rate and focus on high risk subjects, thereby possibly improving the overall cost effectiveness of a screening programme.

Dr Nielsen concluded that screening for ASLVD is likely to be cost effective, with a cost per life saved of between £4000 and £8000 over 5 years52. In addition, the number needed to screen is lower or comparable with accepted strategies for other diseases. BNP testing is useful in pre-screening before echocardiography, bringing down the cost of detecting LVSD. However, there are many questions that still need to be answered, such as what the effect would be of identifying other treatable conditions during screening would be and whether asymptomatic patients would be compliant with treatment.

References

12. M Yamamoto and MR Boyett, unpublished findings

Figure 3. Comparison of cost effectiveness of ramipril treatment with other cardiovascular and non-cardiovascular interventions.

14. Neuberger et al. unpublished, presented at the 23rd Annual Scientific Meeting of the Heart Rhythm Society, San Diego, USA


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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. Members receive a regular newsletter as well as the opportunity to become involved in a stimulating programme of meetings.

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