Session 1: Advances in pharmacological therapy for heart failure

Angiotensin-receptor blockers

Professor Stephen Ball (Leeds) described recent studies of angiotensin II antagonists (AIIAs) and identified key learning points for the audience. Although there are numerous studies of AIIAs (comprehensively reviewed and analysed in papers by McMurray and Pfeffer and Jong et al.) the two principal randomised controlled trials of AIIAs in heart failure are ELITE II and Val-HeFT.

ELITE II followed on from ELITE (Evaluation of Losartan in the Elderly), a trial that claimed additional survival benefits for the AIIA losartan versus captopril – an angiotensin converting enzyme inhibitor (ACEI) – in HF. ELITE II was designed specifically to investigate the potential mortality benefits of losartan. However, although it was better tolerated than captopril, a survival advantage was not confirmed (Figure 1).

Val-HeFT investigated the morbidity and mortality benefits of adding the AIIA valsartan to current best therapy (ACEIs). Similar to ELITE II, a neutral mortality result was found, but there was a slight benefit for hospitalisations. In fact, the important finding was that mortality was increased in those taking the combination of beta-blockers, ACEIs and valsartan. It was suggested that the trial could be interpreted as showing that in clinical practice, valsartan might benefit patients who could not tolerate ACEI therapy. However, the numbers were small and an ACEI-intolerant population was not studied.

Summarising other key trials, Professor Ball assessed how his interpretation of the studies differed slightly from reported claims. Of particular relevance to HF patients are LIFE (in hypertensive patients) and OPTIMAAL (enrolment post-MI patients with clinical evidence of HF) as these conditions often underlie HF. Ongoing AIIA trials include CHARM (a trial of candesartan in HF) and VALIANT (enrolling post-MI patients).

Although there is “no strong evidence for the use of AIIAs as a first line therapy for HF” they should be considered for ACEI-intolerant patients, Professor Ball said.

New information on beta-blockers

A wealth of data from clinical trials now exists to substantiate the use of beta-blockers in HF. Professor Andrew Coats (London) overviewed the major trials.
The US Carvedilol Programme was the first large trial to look at beta-blocker use in HF\(^{10}\). Although the mortality result was encouraging, it was not a primary endpoint and therefore unable to convince sceptics to prescribe beta-blockers for this indication. However, results from the CIBIS II\(^{11}\) and MERIT HF\(^{12}\) trials soon substantiated the mortality claims.

Although these studies indicated that people with moderate HF could benefit from beta-blockers, evidence was sparse in patients with mild or severe HF. Professor Coats explained how the recent CAPRICORN\(^{13}\), COPERNICUS\(^{14}\) and BEST\(^{15}\) trials have helped fill in the evidence gaps.

CAPRICORN was a post-MI HF study. Mortality was a co-primary end point and was reduced by 23%, similar to results of previous beta-blocker studies. The other co-primary end point of all-cause mortality or cardiovascular hospitalisations was not reduced.

The BEST and COPERNICUS trials enrolled patients with severe HF. In BEST, overall no additional survival benefit was found in the bucindilol group. Although NYHA class was not used to assess severity in COPERNICUS and greatly uncompensated patients were not enrolled, results were positive for mortality, hospitalisations, quality of life and cost-effectiveness – regardless of ejection fraction.

Professor Coats concluded that all patients with stable chronic HF due to left ventricular systolic dysfunction should receive a beta-blocker, unless contraindicated or not tolerated.

**Vasopeptidase inhibitors, endothelin and cytokine antagonists**

Professor John McMurray (Glasgow) summarised the lessons learned from an unfortunate tale about three classes of drugs that had once seemed to have a promising future in HF: anti-cytokine (anti-tumour necrosis factor (TNF)) therapy, endothelin antagonists and vasopeptidase inhibitors.

**Cytokine antagonists**

Experimental studies had indicated that cytokines, and in particular, TNF, were involved in myocardial damage. When experimental anti-cytokine studies validated this theory, the scene was set for the concept to be tested in a major outcome trial (RENEWAL) involving HF patients. The hypothesis was that a fusion protein ‘etanercept’ designed to mimic the TNF receptor would bind excess circulating TNF in extracellular fluid, reducing myocardial damage. However, the trial was stopped prematurely because of ‘futility’. Professor McMurray said that it was possible that the complexity of the two-part, multiple-dose trial design had adversely affected the results.

**Endothelin receptor antagonists**

The endothelins are a family of peptides with similar activities to those of the hormone angiotensin II and it was hoped that endothelin receptor antagonists would have similar benefits to renin-angiotensin-aldosterone system inhibitors. The ENABLE studies\(^{16}\) looked at the effects of bosentan (an endothelin receptor antagonist) in high risk HF patients. The results suggested that taking bosentan was associated with early worsening of chronic HF, leading researchers to question whether the endothelin hypothesis was wrong, or worse, whether the neurohormonal theory is misleading.

It is possible, however, that the doses used in the study were wrong.

**Dual angiotensin converting enzyme/neutral endopeptidase inhibitors**

It was hoped that inhibiting the actions of both ACE and neutral endopeptidase (NEP) would:

- reduce the production of angiotensin II from angiotensin I (mediated by ACE), a hormone implicated in the progression of HF
- increase circulating bradykinin and natriuretic peptides (both of which are vasodilators degraded by NEP)

The OVERTURE trial\(^{17}\) studied whether omapatrilat (a NEP inhibitor) had additional mortality and morbidity benefits over ACE-inhibition in severe HF patients. Disappointingly, the results were neutral for this endpoint, although those for the secondary endpoint were more promising. It is unlikely now that the drug will be studied further because of the association between bradykinin (which the drug increases) and angioedema.

Professor McMurray concluded that these trials suggest that “perhaps we don’t fully understand the pathophysiological basis of treatment in HF or how to select the right drug dose”. In addition, the rush to get the molecule to market is perhaps compromising the design of clinical trials. Clinicians might also have to contemplate the possibility that a therapeutic ceiling has been reached for neurohormonal antagonism.

**Statins for patients with heart failure: The Debate**

Dr Stefan Anker (London and Berlin) and Dr Richard Wray (Hastings) presented a lively debate about the use of statins (cholesterol-lowering agents) in HF.

Presenting the ‘for’ argument, Dr Wray pointed out that about 65% of UK HF patients have coronary artery disease (CAD)\(^{12}\). In addition, large randomised controlled trials have clearly shown the importance of coronary events in increasing the risk of the development of HF, or worsening the prognosis of HF patients.

Evidence from large randomised controlled trials shows that statins are extremely valuable in reducing cardiac events in those with CAD\(^{18,19}\). The Heart Protection Study has also shown that statins have cardioprotective effects *even in people with low or normal cholesterol levels*\(^{20}\). This leads Dr Wray to believe that they may play an important role in reducing morbidity and mortality in HF patients. To help address the lack of specific evidence in HF patients, the GISSI group in Italy plans to study the effects of statins in people with HF of different aetiologies.

Presenting the ‘against’ argument, Dr Stefan Anker explained that although statins are clearly beneficial in reducing coronary events in CAD patients, CAD and HF are two quite different syndromes “the evidence base for using statins in HF is non-existent”. Since the mortality rate is high for HF patients and they are seriously ill it would be unwise to expose patients to a treatment that can cause severe side effects, even if in other settings they are very rare, he said. In addition, statins damage a patient’s antioxidant defence, which is compromised in HF\(^{21,22}\).
However, his principal argument against using statins in the HF population, was that cholesterol-lowering agents are harmful to these patients because cholesterol has a cardioprotective effect against damaging inflammatory mediators as lipoproteins can bind to bacterial endotoxin and prevent its deleterious effects.

Finally, he did suggest that if a statin-like drug was created which did not lower cholesterol but had ancillary properties such as “immune modulatory and anti-inflammatory actions”, it might be a useful addition to HF therapy – an approach endorsed by Dr Wray.

However, despite Dr Anker’s spirited argument against statins, the audience vote endorsed Dr Wray’s approach.

Note: Although Dr Anker had been asked to present against the use of statins, in real life (knowing the available data, including his own) he agrees with Dr Wray’s opinions.

Session 2: A clinical research network in the UK – providing definitive answers to key clinical questions

Dr Davies (Birmingham) said that the BSH Board would like the BSH members to consider whether the development of a clinical network in HF would be an effective project for the Society to pursue. And should it be a network in research, teaching or training, or a combination of all three? The goal would be to improve and modernise HF research and management through collaboration.

UK-HEART

Dr Jim Nolan (Stoke-on-Trent) provided an example of how the development of a clinical research network can be beneficial in improving HF management.

The UK-HEART study was established in response to the fact that although it is recognised that severe HF patients have a high mortality risk, it is extremely difficult for clinicians to risk-stratify those with mild to moderate HF who comprise the majority of the population.

It was designed by a group of collaborating cardiologists as a large, prospective, multicentre trial with a long follow-up, to provide statistically sound data that would help to develop a clinically useful prognostic model.

The study revealed that this population of HF patients has an overall 5-year mortality risk of 36.5% and the factors associated with this poor prognosis were identified. From this information, the investigators derived an index with good sensitivity and specificity for pinpointing patients at increased risk of premature death. Further refinement of this approach allows patients at increased risk of sudden or progressive HF death to be identified. These results provide useful insights into the pathophysiology of HF, and will help clinicians target therapies accordingly. Most importantly this approach to risk stratification is practical as it relies on clinicians carrying out simple, widely available tests.

The study shows how, on a modest budget (£15,000), collaboration between different research centres can help advance our knowledge of HF and improve its management.

Dr Nolan said that the group has now grown to include other centres both in the UK and abroad, who will be collaborating in a planned UK-HEART II study.

After Dr Nolan’s presentation, representatives from the UK’s principal research-funding organisations explained how they are involved in HF research and education.

Summary from the Wellcome Trust

The Wellcome Trust is one of the most well-known independent research funding charities in the UK and was established to “foster and promote research with the aim of improving human and animal health”. Dr Huehns, who trained as a cardiologist and now works as a Science Programme Officer at the Wellcome Trust, explained the charity’s role in cardiovascular research.

In 1996 it launched a cardiovascular research initiative to promote multidisciplinary centres of excellence for cardiovascular research. After a national competition the centres of Oxford and Edinburgh were selected to receive funding of approximately £7 million over 7 years, with Oxford concentrating on the molecular genetics of heart disease and Edinburgh on cardiovascular development and vascular injury. In addition the Trust supports five other academic research facilities in the UK.

It also provides project-specific grants and funds fellowships relating to cardiovascular disease. Dr Huehns said that competition is fierce for the different fellowships, which are available for basic scientists and clinicians, from junior through to the senior research fellowship level. Anyone interested in applying for a grant or fellowship should either contact one of the Wellcome Trust advisors or visit the website for more information (http://www.wellcome.ac.uk).

Developing a strategy for cardiovascular research in the UK (Summary from the Medical Research Council)

The Medical Research Council (MRC) currently spends £24 million per annum on cardiovascular research, split between heart, vascular and metabolic research. This portfolio is further underpinned by significant investment in basic research. The MRC is probably the principal funder of non-commercial cardiovascular clinical trials in the UK, corresponding to 19% of its cardiovascular portfolio.

Dr Rob Buckle explained how the Council is currently creating an overall research strategy for the next 5–10 years, the two priority areas being post-genomic research and the health of the public.

Part of this strategy involves collaboration with other major funding charities and health departments in key areas, and such an approach lead to the formation of the ‘Cardiovascular Research Funders’ Forum’ in 2001. By working together, the member organisations hope to better co-ordinate cardiovascular research within the UK and provide strategic guidance, for example by identifying research needs as well as providing input to aid development of the National Service Framework for Coronary Heart Disease.

The clinical trials that the MRC funds are mainly large, multicentre phase III randomised controlled trials and approximately half are for non-drug interventions. The largest...
trial the Council helped fund was the Heart Protection Study on statin use.

Drawing on past experience, Dr Buckle said that a successful clinical research network requires clear aims and should integrate existing networks. It is also important to consider potential funding sources and how they can be convinced that a network would be well used. Once designed, centralised co-ordination is crucial.

For more information on the MRC, please visit their website: http://www.mrc.ac.uk.

Summary from the British Heart Foundation
The British Heart Foundation (http://www.bhf.org.uk) plays a leading role in the fight against heart disease through a variety of approaches, the most prevalent of which is funding research into the causes, diagnosis, prevention and treatment of heart disease. In addition, it has a vital education role – by providing support and information to patients and their relatives and raising public awareness. Training is also important.

Professor Charles George, the Medical Director of the BHF, said that the decrease in the mortality rate from CHD over recent years is validation for the Foundation’s work (Figure 2). However, the reality is that people with CHD are now living longer, and this is associated with a rise in the incidence of HF. For this reason it is focusing more attention on the syndrome and this began with the launch of the campaign ‘It’s time to tackle heart failure’ in British Heart Week in 2002. This was also accompanied by a special HF-focused supplement to the annual CHD statistics publication27.

Specific projects in HF include:
• Development of a HF nurse training course at the Glasgow Caledonian University, alongside the BSH
• Funding of HF rehabilitation programmes
• A rapid training programme for echocardiography technicians

Professor George also outlined to the audience how cardiovascular research is being funded by the BHF, and referred to a joint initiative with the Department of Health for research into heart failure, for which a sum of £1.5 million is available.

Discussion
The key messages from discussion about a potential clinical network for HF were:
• It should not become an exclusive network of people otherwise they could wield too much influence over policies and the direction of research. It should be multidisciplinary and smaller centres should be involved.
• A research network should support basic research projects as well as clinical trials.
• A HF database would be pivotal in the development of the network.
• It should not just be devoted to research (although it is fundamental to good clinical practice) but to knowledge sharing between healthcare professionals: the logical first step would be to develop a ‘clinical network’. Training and education should also be addressed.

Professor John Cleland then presented the membership with an idea for a study that could be supported by the BSH as part of the network, looking at the benefits of allopurinol in treating HF.

Dr Davies said that further suggestions about the structure and purpose of the network would be welcomed by the BSH Board.

Session 3: Management problems in heart failure 1 – how to manage
Atrial fibrillation; cardioversion never, anticoagulation always?
Atrial fibrillation is associated with a large increased risk of stroke and thromboembolism. It is common for HF patients to have atrial fibrillation and the combination of both increases the stroke risk even more28. Professor Gregory YH Lip (Birmingham) investigated the merits of cardioversion and anticoagulation therapy for HF patients who also have atrial fibrillation.

Large randomised controlled trials have established the benefits of antithrombotic therapy, particularly warfarin, in atrial fibrillation – the evidence for aspirin is less strong29. Therefore, in high-risk groups such as HF patients, Professor Lip advocates the use of warfarin, despite the fact that it is less convenient than aspirin, requiring regular monitoring of anticoagulation intensity.

Having outlined the case for antithrombotic therapy, Professor Lip then discussed cardioversion. This approach has often been used in the past to return patients in atrial fibrillation back to normal sinus rhythm. However, recent large randomised control trials (RCTs) such as AFFIRM30, RACE31 and PIAF32, which have compared a strategy of rhythm control (cardioversion) with ‘rate control’ (using heart rate slowing medication and anticoagulants) have not shown that cardioversion is the superior strategy. Indeed, the choice of
whether to adopt a strategy of rate control or rhythm control should be considered on an individual basis, with reference to risk stratification schemes33.

Hypotension

Professor Allan Struthers (Dundee) said that clinical experience generally lessens concerns about hypotension. He explained its causes in chronic HF and how and when to treat it, with reference to the principal clinical trials.

Overdiuresis is a common cause of hypotension in HF and a simple clinical examination can provide confirmation. It is important for the dose of diuretic to be individualised to avoid this, although recommending a correct dose is difficult based on current evidence.

Hypotension is also common in a HF patient because they have a low cardiac output. Many HF drugs exacerbate this by lowering blood pressure even further.

However, there are established methods for reducing the incidence of hypotension and dizziness. If the patient is taking an ACEI they can be switched to another brand, for example, perindopril reduces blood pressure less34. However, there is no firm evidence to support this strategy. Another option is to reduce the ACEI dose, but although this would reduce dizziness, it might also increase the risk of hospitalisation slightly35.

Reducing the beta-blocker dose appears to be better, Dr Struthers said, because studies show that dizziness can be reduced without worsening outcome. However, it would be better for a patient to receive a lower-dose of a beta-blocker plus an ACEI, rather than a higher dose of one, if blood pressure is the limiting factor. If for any reason a patient could only receive one drug, trials show that low dose beta-blockers are more effective in treating HF36.

Preventing early readmission

Kirstin Russell from the Glasgow Heart Failure Liaison Service, explained how and why the service was developed and how it has benefited patients so far.

It is acknowledged that readmission rates for heart failure patients are high and this is considered in part due to poor patient management post follow-up. In response to this issue, the Glasgow Heart Failure Liaison Service began in July 2000 with the aim of identifying hospitalised patients with deteriorating HF and providing them with nursing support post-discharge. The specialist nurses liaise with primary and secondary care physicians to optimise the management of heart failure patients as well as provide HF education and psychological support.

Ms Russell summarised the RCT on which the service was based, comparing nurse-led intervention with usual care. After twelve months follow-up the investigators concluded that although there was no mortality difference between the groups, the nurse intervention significantly reduced readmission rates and the number of patient bed days37. Ms Russell said that following on from the trial, the results have been reinforced by data obtained from 84 patients receiving the intervention at two centres in Glasgow.

The scheme has now been extended to five acute hospitals in Glasgow and over 800 patients have been recruited. Ms Russell said that the labour intensity of the intervention has raised resourcing and funding issues, which will be investigated, and further research will be carried out into areas such as quality of life, palliative care and carers.

Natriuretic peptides as an aid to diagnosing symptoms suspicious of heart failure

It has been acknowledged for over a decade that circulating natriuretic peptide concentrations are elevated in chronic HF patients38,39,40. Dr Theresa McDonagh (Glasgow), who has been at the forefront of UK research in this area, provided an insight into how a simple test for brain natriuretic peptide (BNP) can be used in the clinic to diagnose heart failure. Of greatest importance in the use of this test is its sensitivity and specificity.

Dr McDonagh outlined the evidence for using BNP as a HF test from studies in the acute41,42 post-MI43 and outpatient hospital setting44, as well as in the community44.

The issue is how to translate the findings from the studies to real-life clinical practice. The real value of the test is in its negative, rather than positive, predictive value39. Therefore, it would be most likely to be used to ‘rule out’ suspected cases of HF. Interpretation becomes more difficult when patients (who would have had a positive test in primary care) are referred to secondary or tertiary care. There are no ‘abnormal values’ to which a clinician or nurse can refer easily. Values need to take into account the age of the patient, as levels rise with age (exponentially over the age of 70) and their gender. In addition false negatives can occur with certain drugs. Therefore these issues, as well as the cost implications, need to be fully researched before this screening procedure becomes widely used in clinical practice.

Initiation of beta-blockers in primary care

According to Dr Ahmet Fuat (Darlington) primary care can no longer ignore the impressive evidence base in favour of prescribing beta-blockers in all patients with stable HF.

Mentioning the landmark studies, all showed that beta-blockers confer significant morbidity and mortality benefits when added to conventional therapy and have a reasonably low adverse event rate compared with placebo.

However, compared with other European practitioners, our GPs and selected hospital specialists seem reluctant to prescribe them46. Although some observational studies have suggested they can cause adverse effects such as dizziness and hypotension46, others indicate that with simple precautions, most patients with heart failure, including the elderly and those with severe LVSD, can safely be established on a beta-blocker in a community setting47.

He summarised the procedure for initiation with reference to a useful review by Mead48. Before starting a beta-blocker regime, the GP must make a series of checks about the patient and educate them about the treatment they will receive, its long term benefits and explain that they may expect a temporary worsening of symptoms. Compliance should be encouraged, and the target dose should be mentioned.

Initiation should begin with a slow uptitration and a useful mnemonic to follow is STAPLE (Figure 3)46. Perseverance is the key to successful initiation, Dr Fuat said, since many
Renal dysfunction is common and can be expected routinely in chronic HF patients, said Dr Robert MacFadyen (Birmingham). Although it is often a consequence of unsuspected renovascular disease (associated with ischaemic or post-hypertensive systolic left ventricular dysfunction), tubular glomerular disease or obstructive nephropathy, iatrogenic causes are also common.

Clinicians need to understand how to prevent renal problems from developing and worsening. In the case of a patient presenting with suspected renovascular disease they should be examined thoroughly, investigated appropriately and treated promptly by revascularisation if feasible. Preserving renal function is crucial in these patients because cardiac surgery cannot take place in those without adequate renal reserve.

Loop diuretic therapy is particularly associated with iatrogenic injury to the kidneys because they lower blood volume and consequently, renal perfusion. Unfortunately, there is little evidence to support current methods of blood volume assessment.

In addition, many HF drugs lower blood pressure, and when combining drugs it is important to consider that there is a threshold where renal function declines precipitously if arterial perfusion pressure is decreased. The goal is to balance preservation of arterial pressure and functioning renal parenchyma, whilst treating HF.

Concluding, Dr MacFadyen said that prescribers should carefully consider whether every drug therapy for HF is necessary for every HF patient. The impact of drug therapy on renal function in each case should be a matter of concern. This is particularly pertinent for combinations involving spironolactone.

Hyponatraemia

Dr Iain Squire (Leicester) explained that hyponatraemia often co-exists with renal dysfunction and it is more likely to occur in severe HF patients. Although part of the cause is neurohormonal activation, there is also an iatrogenic component. Hyponatraemia often indicates too much or too little treatment, he said.

A basic clinical examination of the patient is key to assessing the extent of the problem – patients are more likely to be fluid overloaded than dehydrated, with hyponatraemia indicating a relative excess of water in proportion to the retention of salt. In this case optimising both diuretic and other treatment is vital. Fluid and salt restriction can also help to rectify electrolyte imbalance, although it can be difficult and should always be considered in the context of the patient’s current pharmacological therapy. Dr Squire briefly described the guidelines for carrying out this process.

Iatrogenic causes should always be suspected, Dr Squire said, particularly excessive diuretic use as they increase intravascular salt and water depletion. He recommended that if a hyponatraemic patient is receiving diuretics but not ACEIs, addition of an ACEI should be considered, but initiated with care. However, in stable chronic HF patients who have become hyponatraemic, ACEIs might instead contribute to the problem by reducing renal perfusion. In this case their temporary or permanent withdrawal should be considered. Aspirin and NSAIDs have also been implicated as contributors and if an episode occurs after their recent initiation, their use should be reviewed.

The key message is that even mild hyponatraemia should be taken seriously as this predicts a worse outcome in HF.

Hibernation and ischaemia

Coronary artery disease is one of the commonest causes of heart failure and over 30% of all HF patients have angina. In the CHRISTMAS study, 79% of the HF patients with left ventricular dysfunction secondary to CAD had reversible ischaemia or hibernating myocardium.

Investigation

Professor John Cleland (Hull) explained that symptoms of angina do not necessarily indicate that the patient has myocardial ischaemia. Therefore investigation for myocardial ischaemia and hibernation is not justified on the basis of angina symptoms alone.

Ischaemia is notoriously difficult to diagnose and it can only be confirmed by carrying out stress imaging. A diagnosis of stunning and/or hibernation can be achieved by assessing wall thinning, the contractile response to low dose dobutamine, uptake of radioisotopes or the lack of uptake of gadolinium on cardiovascular magnetic resonance imaging.

Treatment

An anginal HF patient should be treated with symptomatic therapies first – beta-blockers are the first choice. If
pharmacological therapy fails then revascularisation should be considered for symptomatic relief. There is no evidence that revascularisation improves the prognosis of HF patients with coronary artery disease, even if a large proportion of the myocardium is affected by hibernation or ischaemia. Evidence for or against revascularisation in this context will be sought in the HEART-UK trial52.

The same pharmacological therapies as for angina are used in patients with a diagnosis of ischaemic myocardium, but the beta-blocker carvedilol has been proven to be useful for patients with hibernation53. ACEIs should be considered as they reduce the incidence of myocardial infarction and warfarin may also be preferred over aspirin, but this will be confirmed in the ongoing WATCH trial54.

Recurrent syncope

Syncope is quite common, said Dr Phil Batin (Wakefield). It is defined as ‘a sudden and transient loss of consciousness associated with the inability to maintain postural tone, followed by prompt, spontaneous and complete recovery’. There is a difference between syncope and the many conditions that resemble it and all aetiologies share one common pathway – transient global cerebral hypoperfusion.

It is difficult to investigate the epidemiology of syncope because so few cases are reported. However, data from the Framingham study show that it is much more likely to occur in the elderly. It accounts for 3–5% of Accident and Emergency attendances and 1–3% of hospital admissions55.

Although in many cases of syncope the aetiology remains unknown, patients with cardiac disease are more likely to have cardiac-related syncope56. Cardiac syncope confers a worse prognosis than other aetiologies55.

A history of syncope in HF is a serious concern – a study of severe HF patients showed that those who had suffered from syncopal episodes had a 45% risk of sudden death, compared with 12% in those who did not56. This was independent of aetiology. Recurrence rates are also higher in patients with cardiological disease55. There is therefore a need for clinicians to take syncope seriously and investigate, risk-stratify and treat patients appropriately.

Selecting patients with heart failure for an ICD

Selecting HF patients for an ICD is quite difficult in practice, said Dr Mark Kearney (London). There are two ways in which a HF patient dies – either through sudden death or from gradual decompensation. Studies have shown that implanting a cardioverter defibrillator can prevent sudden death57,58. However, as the cost is high and the operative risk also, it is important that a strategy is employed to target those who would benefit most.

As Dr Nolan discussed, the UK-HEART study derived an index based on four independent predictors of sudden death that can identify patients at greatest risk. The index was found to have a negative predictive value of 100% if none of the risk factors were present, and could also discriminate between those at low, medium and high risk.

When validated, this simple test could be used by clinicians to risk-stratify HF patients and identify those who are at greatest risk of sudden death and would benefit from an ICD.

Anemia: the red revolution

Dr Ivor Cavill, a haematologist from Cardiff, explained why clinicians must not underestimate the importance of anaemia in HF. On the whole it seems that “doctors have a high tolerance for anaemia, but patients don’t”, he said. From his experience patients often consider the chronic fatigue associated with anaemia to be worse than pain59.

Another attitude to anaemia is that a patient being slightly anaemic isn’t a real concern. However, Dr Cavill explained that even small decreases in haemoglobin concentrations can have a negative effect on exercise tolerance.

Not only is the haemoglobin concentration an important factor in anaemia, but so is the balance between red blood cell production and destruction. When it is in balance this is reflected in haemoglobin concentrations. Giving a blood transfusion to anaemic patients only acts as a quick fix, temporarily increasing haemoglobin levels60. Instead the replacement of red blood cells is needed alongside erythropoetin. In this way patients can be freed from the fatigue of anaemia, allowing them to benefit from an increased quality of life.

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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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