Heart failure – what the general physician needs to know

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ABSTRACT

The British Society of Heart Failure (BSH) meetings highlight the latest advancements within the field of heart failure (HF) and provide education for training and revalidation for cardiologists and general physicians. This article reviews take-home messages from the 7th BSH HF revalidation and training meeting. It emphasises what every physician needs to know about the latest acute HF guidelines, diagnostics in HF, management strategies (including pharmacotherapeutics and device therapy), and when to consider referring to a transplant centre for mechanical circulatory support or transplantation. It describes the practical challenges faced and provides clinicians with a framework to assist with service development and commissioning of resources to deliver optimal, integrated services that meet the ever-advancing needs of our HF communities.

KEYWORDS: Heart failure, guidelines, diagnosis, transplant, ventricular assist device, commissioning, service development

Introduction

Heart failure (HF) is defined, clinically, as a syndrome in which patients have typical symptoms (eg breathlessness, ankle swelling and fatigue) and signs (eg elevated jugular venous pressure, fluid retention, pulmonary congestion and a displaced apex beat) resulting from an abnormality of cardiac structure or function (either left ventricular systolic dysfunction (LVSD) or diastolic dysfunction (termed HF with preserved ejection fraction (HF-PEF)) which fails to deliver oxygen to meet the requirements of metabolising tissues.[1–3] It is a common acute and chronic medical problem with an estimated prevalence of 2–4% in Europe.[1] This figure continues to grow as a result of an ageing population, an increasing prevalence of diabetes and hypertension, and better survival from cardiovascular disease.

Acute heart failure (AHF) can present as new-onset HF, or as an acute decompensation of chronic HF (CHF). Most cases of HF are acquired and caused by dysfunction of the heart due to muscle damage, coronary artery disease (CAD), hypertension, diabetes, valvular heart disease or arrhythmias (including atrial fibrillation).[1,4] Epidemiological studies suggest that genetics predispose individuals to HF through maladaptive modulation of responses to common pathophysiological stressors (such as CAD) or maladaptive responses to pharmacotherapy. Other causes of HF include inflammatory cardiomyopathies and genetic disorders ('inherited cardiomyopathies', such as dilated cardiomyopathy) which are complex, rare and clinically heterogeneous heart muscle diseases predominantly inherited as autosomal dominant disorders that require family screening, risk stratification and early institution of disease modifying or device therapy.[5]

Survival and quality of life remains poor in HF with a reported four-year survival following diagnosis of only 50%.[1] Admission to hospital is common and is often associated with significant length of stay. Acute HF accounts for over 67,000 admissions per year in England and Wales and is the leading cause of hospitalisation in those aged over 65 years.[6] Early discharges are often counter-productive and contribute significantly to re-hospitalisation rates, particularly within the post-discharge 'vulnerable period'. These place considerable pressures on resources. Radical improvements in the delivery of HF services to ensure early and effective diagnoses and management of patients, careful optimisation prior to discharge and targeted planning pre- and early discharge to tackle 'vulnerable periods' are warranted to minimise high post-discharge morbidity, mortality and re-hospitalisation rates.

Advances in research provide a clear evidence base for treatments to reduce morbidity and mortality. These underpin the development of practice guidelines and quality standards in HF. However, there remain diverse unmet needs, and delivering the burgeoning demand for comprehensive HF care within the ever-increasing socioeconomic restraints requires major service redesign beyond the use of pharmaceuticals alone. This review summarises key HF guidelines and describes strategies for the future to deliver an engaged, collaborative and 'seamless' system of HF care to enable the optimal management of each and every patient along their healthcare journey.

AHF guidelines

The AHF National Institute for Health and Care Excellence (NICE) guidelines highlight key priorities for organising the totality of acute HF care (diagnosis, assessment, monitoring, management and stabilisation) for patients aged over 18 years who are admitted with a new diagnosis of HF or decompensation

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Box 1. Key priorities for the implementation of the NICE acute HF guidance. Adapted with permission.[7]

Organisation of care

- Hospitals admitting people with suspected AHF should provide specialist HF services based on a cardiology ward and additional outreach services.
- All patients admitted to hospital with suspected AHF should receive early and continuing input from dedicated specialist HF teams.

Diagnosis, assessment and monitoring

- Use a single measurement of serum natriuretic peptides (BNP or NT-proBNP) to rule out the diagnosis of HF in people presenting with new suspected AHF.
- Where natriuretic peptide level is raised, perform transthoracic Doppler 2D echocardiography to establish the presence or absence of cardiac abnormalities within 48 h of admission to enable early specialist management.

Treatment after stabilisation

- In a person presenting with AHF who is already taking beta-blockers, continue the beta-blocker treatment unless they have a heart rate <50 bpm, second- or third-degree atrioventricular block, or shock.
- Start (or restart) beta-blocker treatment during the hospital admission for LVSD once condition has stabilised (ie once iv diuretics are no longer required).
- Offer an ACEi and MRA to patients with AHF and LVSD during their hospital admission.
- Ensure that the person's condition is stable for 48 h before discharging from hospital and/or after starting or restarting beta-blockers.
- Plan a person's discharge from hospital after the acute phase. Communicate information about a person's condition, treatment, prognosis and subsequent management plan to primary and secondary care (including HF MDT).
- Ensure that a follow-up appointment is made with the specialist HF team within two weeks of discharge from hospital.

ACEi = angiotensin-converting enzyme inhibitor; AHF = acute heart failure; BNP = B-type natriuretic peptide; HF = heart failure; MDT = multidisciplinary team; MRA = mineralocorticoid receptor antagonist; NICE = National institute for Health and Care Excellence; LVSD = left ventricular systolic dysfunction; NT-proBNP = N-terminal pro-B-type natriuretic peptide.

of previously stable HF (Box 1).[7] They promote that any hospital admitting patients with suspected AHF should provide an integrated and engaged specialist multidisciplinary HF service led by a specialist HF lead to provide early and continuing hospital-wide outreach services and liaison with the community (Fig 1) to ensure that local needs are met, that services are accessible and regulated and that education is safeguarded.

AHF diagnostic pathway and management

The diagnostic and management pathway for AHF is described in Fig 2. It utilises natriuretic peptides to 'rule out' HF and guide the use of echocardiography to determine aetiology. It signposts clinicians to presentation-guided management and to the instigation and up-titration to maximum tolerated doses of disease-modifying pharmacological therapies according to symptomatic hypotension, renal function and potassium levels (Fig 3). It also considers advanced therapies (including devices) as disease severity escalates and New York Heart Association (NYHA) class worsens.

HF discharge planning

Hospitalised patients should be discharged once stable for 48 hours on optimal medical therapy. All pertinent clinical information, including aetiology and echocardiographic evidence for HF, major comorbidities, electrocardiography parameters (rhythm, QRS duration, morphology or paced), peptide level, discharge parameters (dry weight, blood pressure, heart rate, haematology and biochemistry values) and medications (including limitations and advice for up-titration) should be communicated to the community HF team. Documentation should specify contact details and the date of follow-up by the HF team within two weeks of discharge, alongside the future care plan (detailing device, rehabilitation, palliative needs) to ensure continuity of care, early review and the delivery of ongoing holistic and meaningful care within the community.

Traditional drug therapies for CHF and recent updates

The evidence base for treating HF primarily relates to LVSD.[3,8] Neurohormonal activation underpins the pathophysiological basis of LVSD (Fig 4). Thus neurohormonal inhibition using pharmacotherapy (beta-blockers, angiotensin-converting enzyme inhibitors (ACEi) and mineralocorticoid receptor antagonists (MRAs)), provides the bedrock for all treatments (Fig 3).

Drugs that block the sympathetic nervous system (beta-blockers) reduce morbidity and mortality. MERIT-HF, COPERNICUS and CIBIS-2 have demonstrated significant reductions in mortality with the use of metoprolol CR/XL (relative risk reduction (RRR) 34%), carvediolol (RRR 35%) and bisoprolol (RRR 34%), respectively.[9–11] SENIORS demonstrated a 12% RRR in event rate with the use of nebevilol.[12]

Beyond beta-blockers, two trials, CONSENSUS and SOLVD, underpin the guidance for using renin–angiotensin system (RAS) blockers (ACEi) in HF, demonstrating that ACEi (namely enalapril) improve survival.[13,14] Where ACEi cannot be instituted due to cough or other reason (eg angioedema), angiotensin receptor blockers (ARB) offer an alternative block within the RAS for most. Cautious commencement should be adopted in cases of prior angioedema with ACEi to ensure the absence of cross-reactivity with ARB use in up to 10% of cases. The CHARM-Alternative trial used candesartan to a target dose of 32 mg per day and demonstrated a significant 20% RRR for death or HF hospitalisation when compared with placebo.[15] Importantly, there is no evidence to suggest that an ARB is any less likely to cause hypotension, renal dysfunction or hyperkalaemia than an ACEi. Renin inhibitors also offer neurohormonal

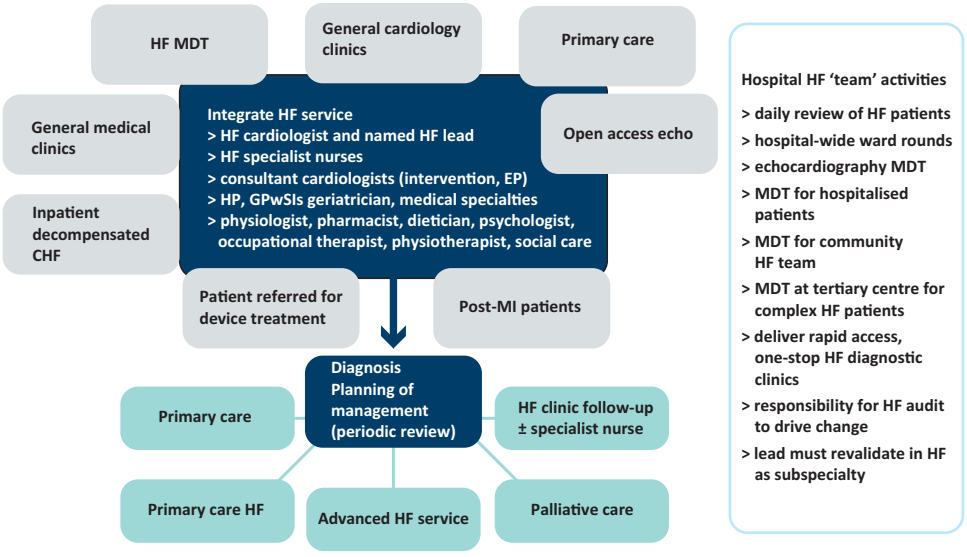

Fig 1. Elements of an integrated and multidisciplinary HF service. CHF = chronic heart failure; HF = heart failure; MDT = multidisciplinary team MI = myocardial infarction. Adapted with permission.[48]

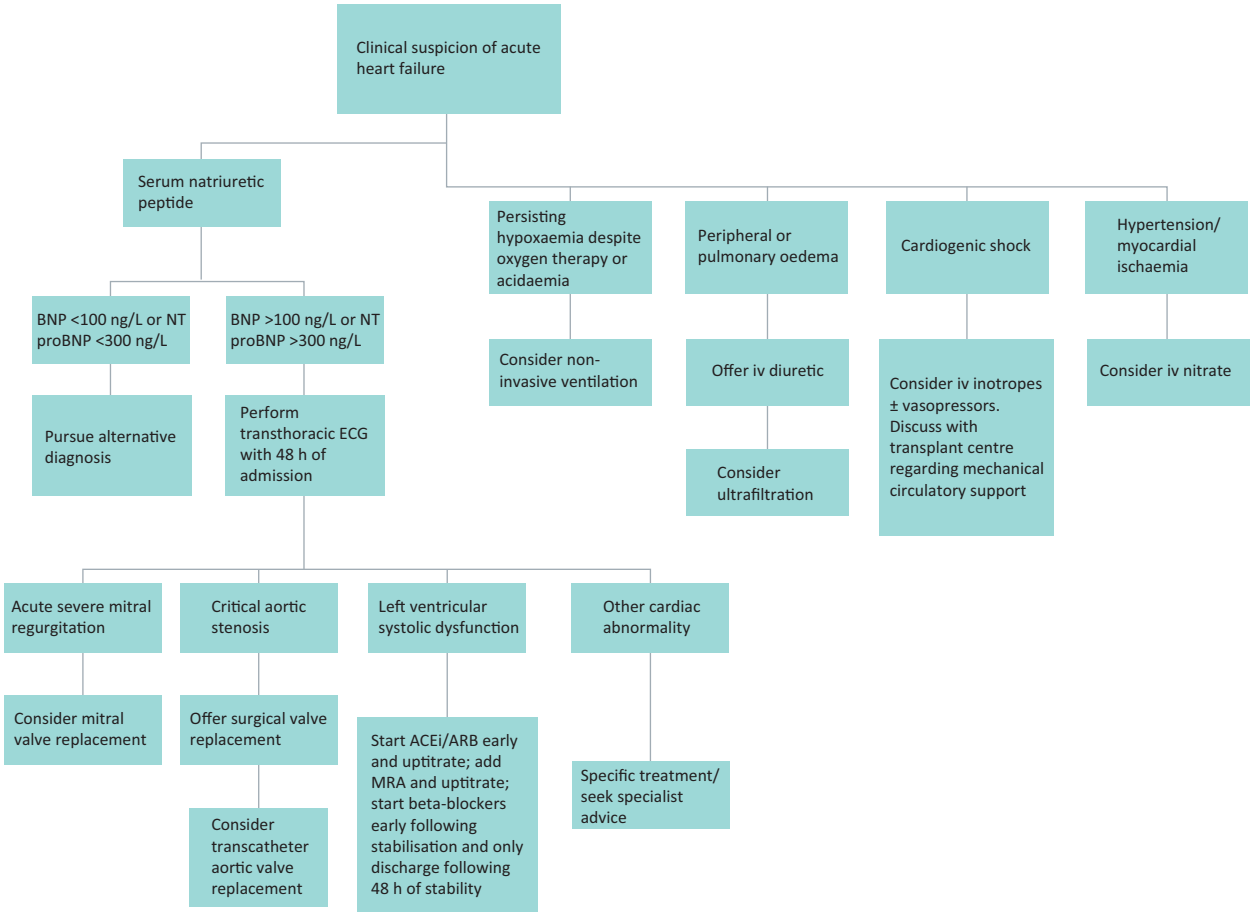

Fig 2. Diagnostic and management algorithm for acute HF.[7] ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BNP = brain natriuretic peptide; ECG = electrocardiogram; HF = heart failure; MRA = mineralocorticoid receptor antagonist; NT proBNP = N-terminal pro-B-type natriuretic peptide.

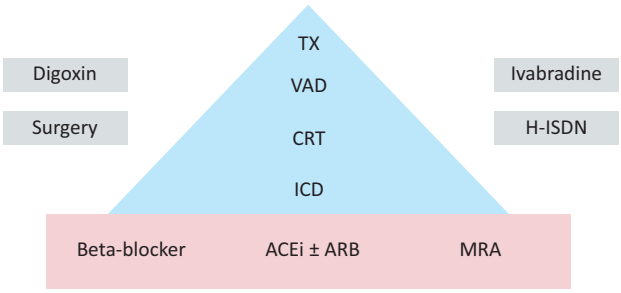

Fig 3. The building blocks of pharmacological and advanced therapies in HF with LVSD (HF-REF). ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CRT = cardiac resynchronisation therapy; HF-REF = heart failure reduced ejection fraction; H-ISDN = hydralazine-isosorbide dinitrate; ICD = implantable cardioverter defibrillator; LVSD = left ventricular systolic dysfunction; MRA = mineralocorticoid receptor antagonist; Tx = transplantation; VAD = ventricular assist device.

blockade. The ATMOSPHERE trial of 7,063 patients is underway to compare the use of enalapril (10 mg twice daily) versus the direct renin inhibitor, aliskiren (separately and in combination), in HF.[16]

Other RAS inhibitors that are used in combination with disease-modifying agents are MRAs. RALES and EMPHASIS-HF are the two pivotal trials that demonstrated reduced morbidity and mortality with spironolactone or eplerenone, respectively.[17,18] Heightened interest in combining MRAs with potassium-combining resins which bind potassium in the colon to avoid hyperkalaemia (such as sodium zirconium cyclosilicate examined in the HARMONIZE trial), may enable the wider use of MRAs in clinical practice.[19]

Additional therapies in HF are occasionally considered in addition to beta-blockers, ACEi and MRAs. The DIG study compared the use of digoxin alongside ACEi versus placebo.

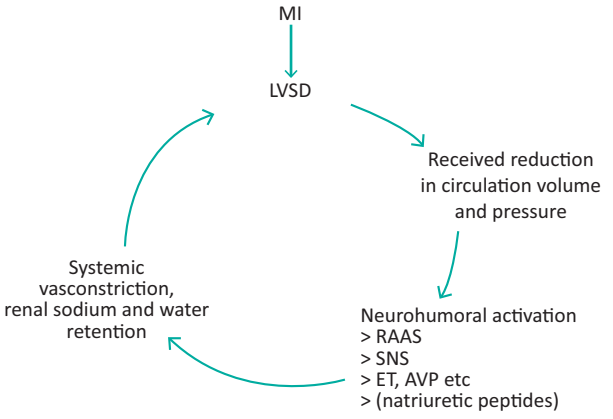

Fig 4. The pathophysiological basis of therapy in HF. Myocardial injury leads to LVSD which invokes a perceived reduction in circulating volume and pressure. This promotes neuro-hormonal activation of the RAAS, the SNS, ET, AVP, natriuretic and other systems. These invoke systemic vasoconstriction and renal sodium and water retention which worsen LVSD and promote the downward cyclical process in HF. AVP = arginine vasopressin; ET = endothelial; HF = heart failure; MI = myocardial infarction; LVSD = left ventricular systolic dysfunction; RAAS = renin angiotensin aldosterone system; SNS = sympathetic nervous system.

The lack of comparison with optimal medical therapy limited the value of this study.[20] Nevertheless, subgroup analysis within those with severe functional limitation (NYHA III/IV), a very low ejection function (<25%) or with an increased cardiothoracic ratio on chest radiograph, showed striking improvements in HF hospitalisation and mortality with digoxin therapy, suggesting that digoxin does provide some benefit in HF.

For patients in sinus rhythm, the SHIFT trial demonstrated reductions in the primary composite end point of cardiovascular death and HF hospitalisation in those with a persistently high heart rate when ivabradine was added alongside conventional HF therapy.[21] In contrast, the V-HeFT trial failed to show a significant benefit with the use of hydralazine and isosorbide dinitrate.[22] However, a possible benefit was demonstrated in sub-selected African Americans in the A-HeFT trial, although the mechanistic basis of this remains uncertain.[23]

HF management not only blocks the destructive RAS pathways as described above, but also aims to simultaneously augment beneficial neuro-humoral systems to restore imbalance through 'neuro-humoral modulation.' The angiotensin receptor neprilysin inhibitor, LCZ696, is a novel therapy examined in PARADIGM-HF that has demonstrated a marked independent reduction in hospitalisation and mortality benefit 16% over and above that achieved with the use of enalapril alone.[24] LCZ696 further lessened symptomatic deterioration, admissions to hospital and the need for intensification of oral HF therapy versus enalapril. LCZ696 therefore has the potential to slow the progression of LVSD and thus lends itself as a novel therapy in the management of HF.

Finally, PEP-CHF, I-PRESERVE, CHARM-Preserved and TOPCAT are major trials that have been conducted in patients with HF-PEF.[25–28] Each of these studies have failed to demonstrate significant reductions in event rates in those administered with perindopril, irbesartan, candesartan and spironolactone, respectively.

Intravenous therapies in AHF

The treatment goals in AHF are to achieve decongestion, preserve renal function and avoid harm. Beyond the instigation and up-titration of disease-modifying therapies, intravenous (iv) therapies are utilised. To alleviate decongestion, the DOSE trial examined the administration of bolus versus infusion iv diuretic therapy and found no significant differences with respect to weight loss or symptomatology.[29] Unsurprisingly, higher doses of diuretics promoted an enhanced fluid loss and improved symptoms. Clinicians are guided to cautiously increase the dose of diuretics in AHF for those already taking diuretics, unless there has been recent non-adherence with diuretic therapy. Renal function, weight and urine output should be monitored to avoid over-diuresis.

Beyond diuretics, NICE guidance suggests that the routine use of opiates, nitrates, sodium nitroprusside, inotropes or vasopressors in people with acute HF should be avoided unless there is evidence of concurrent myocardial ischaemia, severe hypertension, valvular, or diseases other pathology which warrant their institution within an advanced care setting. Small studies suggest that the use of low-dose dopamine (<3 µg/kg/min) and nesiritide (a recombinant brain natriuretic

peptide (BNP)) may promote renal vasodilation and enhance diuresis. However, the ROSE AHF trial conferred no benefit with their use.[30,31] Therefore, NICE corroborates American and European guidelines suggesting that such therapies are best avoided and only considered in end-stage disease or cardiogenic shock.

Finally, RELAX-AHF a placebo-controlled study using serelaxin, a novel recombinant human hormone with known renal protective properties and systemic vasodilator effects, demonstrated significant improvements in symptomatology (dyspnoea and congestion) and was associated with significantly fewer cardiovascular deaths at 180 days following acute treatment.[32]

Differentiating the aetiology of HF is important

Felker *et al* (2000) demonstrated that the underlying cause of HF has prognostic value in patients with unexplained cardiomyopathy.[33] They detailed that patients with peripartum cardiomyopathy have improved outcomes when compared with those suffering from other cardiomyopathies. They noted that the prognosis in idiopathic cardiomyopathy was better when compared with cardiac sarcoidosis, amyloid or haemochromatosis and that the prognosis within each of these conditions becomes sequentially worse. Those with cardiomyopathy related to infiltrative myocardial diseases, HIV infection or doxorubicin therapy have an especially poor prognosis.

The clinical history, electrocardiography, echocardiography (including strain imaging) and cardiac magnetic resonance imaging (CMR) tissue characterisation and nuclear imaging techniques help differentiate between underlying aetiologies. While HF is treated along the same algorithm irrespective of cause, establishment of the aetiology enables the targeted allocation of specific therapies to disease processes. In thalassaemia, CMR T2* imaging has enabled diagnoses, monitoring and instigation of chelating therapies, resulting in a dramatic reduction in mortality by 80%.[34,35] Similarly, differentiation between amyloid light chain (AL) versus transthyretin (ATTR) amyloid is key for delivering specific chemotherapy and in guiding prognosis.[36] Furthermore, establishment of aetiology can guide risk stratification and the need for device therapy or family screening, such as in the presence of a lamin (LMNA) variant in a patient with dilated cardiomyopathy.

The use of devices in HF

Unified and pragmatic device therapy guidance based on the best available evidence, have recently been updated to include primary prevention device therapy in non-ischaemic LVSD and expanding indications for cardiac resynchronisation therapy (CRT) or defibrillator (ICD) therapy (Table 1 and Fig 5).[37] These include data from the SCD-HeFT primary prevention study which demonstrated a 23% reduction in mortality following the implantation of an ICD in those with severe LVSD (ischaemic and non-ischaemic cardiomyopathy) and NYHA II-III symptoms.[38] ICD implantation can now also be considered in patients with a narrow QRS complex providing there is a high risk of sudden cardiac death. Furthermore, since the advent of MADIT-CRT and RAFT,

Table 1. NICE Guidance for device therapy according to NYHA class and QRS duration. Reproduced with permission.[37]

	NYHA Class			
QRS Interval	**I**	**II**	**III**	**IV**
<120 msec	ICD if there is a high risk of sudden cardiac death			ICD and CRT not clinically indicated
120–149 msec without LBBB	ICD	ICD	ICD	CRT-P
120–149 msec with LBBB	ICD	CRT-D	CRT-P or CRT-D	CRT-P
>150 msec with or without LBBB	CRT-D	CRT-D	CRT-P or CRT-D	CRT- P

CRT = cardiac resynchronisation therapy; CRT-D = defibrillator; CRT-P = pacemaker; ICD = implanted cardioverter defibrillator; LBBB = left bundle branch block; NYHA = New York Heart Association.

which demonstrated a superior response in those with a broad QRS irrespective of bundle branch morphology versus narrow QRS, CRT-D can now be utilised.[39,40] Since the PROSPECT trial there is no longer evidence that dyssynchrony echocardiography improves patient selection for CRT.[41] Therefore dyssynchrony echocardiography is no longer warranted in the device selection process. Finally, the use of ventricular tachycardia stimulation to guide device suitability has been excluded.

Clinicians must remain mindful of the impact of an ICD and ensure its appropriate use, particularly in those with NYHA class IV symptoms. Consideration of the cost versus benefit of device implantation, and awareness that ICD implantation does not improve symptoms, LV function or the mode of death, and can be associated with inappropriate shocks, psychological burden and implications for driving, is vital.

Finally, the advantages of devices in atrial fibrillation, bradycardia, HF-PEF, or in those with paced cardiac rhythms who are considered for 'upgrading' to CRT, remain unknown. Nevertheless, advancements in device technology and 'tele-monitoring' capabilities (including leadless technology, subcutaneous extra-cardiac devices, quadripolar

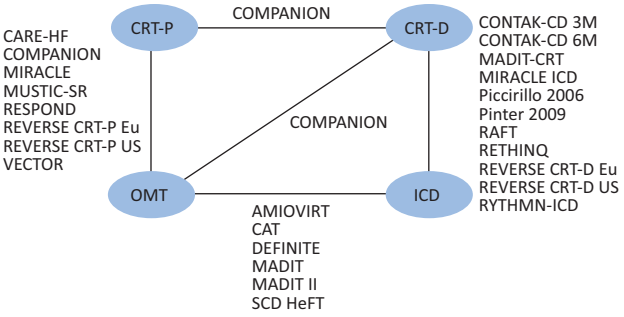

Fig 5. Diagrammatic summary of device trials in heart failure. CRT = cardiac resynchronisation therapy; CRT-D = defibrillator; CRT-P = pacemaker; ICD = implanted cardioverter defibrillator; OMT = oral medical therapy.

Box 2. Conventional criteria for HTx and clinical indicators that should prompt consideration for referral to a transplant centre. Adapted with permission.[42]

Conventional criteria for HTx:

- impaired left ventricle systolic dysfunction
- NYHA III or IV symptoms
- receiving optimal medical treatment (target or maximum tolerated doses of beta-blockers, ACEi, MRA)
- CRT, ICD or CRTD (if indicated)
- evidence of poor prognosis:
  - cardiorespiratory exercise testing (VO^2 max <12 mL/kg/min if on beta-blockade, <14 mL/kg/min if not on beta-blockade, ensuring respiratory quotient ≥1.05)
  - markedly elevated BNP (or NT-proBNP) serum levels despite full medical treatment
  - using established composite prognostic scoring system (eg HFSS or SHFM).

Clinical indicators that should prompt consideration for referral:

- two or more admissions for treatment of decompensated HF within the last 12 months
- persistent overt HF despite optimal medical treatment
- SHFM score indicating ≥20% 1-year mortality
- echocardiographic evidence of right ventricular dysfunction or increasing PA pressure on optimal medical therapy
- anaemia, involuntary weight loss, liver dysfunction or hyponatraemia attributable to HF
- deteriorating renal function attributable to HF or inability to tolerate diuretic dosages sufficient to clear congestion without change in renal function (refer before creatinine clearance falls below 50 mL/min or the eGFR drops below 40 mL/min/1.73 m^2)
- significant episodes of ventricular arrhythmia despite full pharmacological and device treatment
- increasing plasma BNP or NT-proBNP levels despite adequate HF treatment
- refractory angina where debilitating, significant and recurrent myocardial ischaemia is evident and is not amenable to revascularisation or full anti-anginal treatment
- restrictive or hypertrophic cardiomyopathy with persisting NYHA III/IV symptoms refractory to conventional treatment ± recurrent admissions with decompensated HF.

Clinical indicators that should prompt urgent inpatient referral for HTx:

- the need for continuous inotrope infusion (± IABP to prevent multiorgan failure
- persistent circulatory shock due to a primary cardiac disorder
- no scope for revascularisation in the setting of persistent coronary ischaemia.

Relative (R) and absolute (A) contraindications for transplantation:

- microvascular complications of diabetes (excluding non-proliferative retinopathy) (A)
- active malignancy other than localised non-melanoma skin cancer (A)
- extracardiac vascular disease (peripheral or cerebrovascular) (R)
- sepsis and active infection (A); chronic viral infections (R)
- recent pulmonary embolism (A) due to the risks of RV failure post-operatively
- autoimmune disorders (R)
- aggressive skeletal myopathies (A)
- substance misuse (tobacco or excessive alcohol consumption) (R)
- a history of non-adherence to treatment or follow-up (R)
- those with a BMI >32 kg/m^2 are advised to loose weight (R)
- age is not a contraindication, but age <75 years is associated with lower risk
- multiple prior sternotomies increases the risk, but is not a contraindication.

ACEi = angiotensin-converting enzyme inhibitor; BNP = B-type natriuretic peptide; CRT = cardiac resynchronisation treatment; CRTD = cardiac resynchronization therapy defibrillator; eGFR = estimated glomerular filtration rate; HF = heart failure; HFSS = heart failure survival score; HTx = heart transplantation; IABP = intra-aortic balloon pump; ICD = implantable cardioverter defibrillator; MRA = mineralocorticoid receptor antagonist; NT-proBNP = N-terminal pro-B-type natriuretic peptide; NYHA = New York Heart Association; PA = pulmonary artery; SHFM = Seattle heart failure model.

leads, multi-point pacing, vagal nerve stimulation and sensors to measure pulmonary pressures), are postulated to reduce HF admissions and are likely to become increasingly accessible.

When to refer for transplantation and mechanical circulatory support

Patients with advanced HF have a poor quality of life and dismal prognosis. Heart transplantation (HTx) provides an effective lifeline for selective patients and an HF specialist plays a vital role in the timely identification and referral of potential candidates prior to the development of irreversible complications (pulmonary arterial hypertension or cardiorenal syndrome). Banner *et al* (2011) summarise the pertinent aspects in deciding when and whom to refer for transplantation and the role of left ventricular assist devices (VADs) as a 'bridge to transplantation'.[42] The International Society for Heart and Lung Transplantation (ISHLT) guidelines and policy standards provide corroborative guidance.[43] Recent NICE guidance (2015) has extended funding for VADS as a 'bridge to transplantation' to include 'destination therapy' for those who are ineligible for HTx.[44]

Conventional criteria for referral for HTx are described in Box 2. Those with LVSD are more commonly referred, however HF-PEF is not excluded. Triggers for referral include poor prognostic markers – worsening HF symptoms, a significantly raised or increasing BNP, renal or liver dysfunction, rising diuretic requirements, the need for dose reduction of disease modifying medications, ventricular arrhythmias, or the need for inotropic or balloon pump support. Composite risk scoring systems, such as the Seattle HF Model, Heart Failure Survival Score or Meta-Analysis Global Group in HF Calculator help derive a one-year mortality risk greater than 20%.[45–47]

Contraindications for transplantation are also noteworthy. Advanced age >75 years, body mass index >30, active smoking and excessive alcohol intake infer a worsened outcome and are relative contraindications. There are few absolute contraindications but these comprise neoplasia (unless in remission for five years), recent pulmonary embolism, marked pulmonary artery hypertension (with a transpulmonary gradient >15 mmHg or pulmonary vascular resistance (>5 Woods units)), and the presence of microvasular complications of diabetes.

Recommending HTx requires specific expertise and evaluation of the risks, benefits, alternative options and scarce availability of donor hearts to optimise receiver suitability and the potential for functional recovery, survival and improved quality of life. Early referral is therefore key. Patient selection is based on clinical need, the capacity to benefit from transplantation, biological matching (blood group compatibility, donor versus recipient size and HLA status) and logistical factors that influence the cardiac ischaemia time and fairness. There are significant potential risks with HTx and mechanical circulatory support, as described within the ISHLT and the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS Registry).[43,48] Perioperative, early postoperative and long-terms risks to transplantation (including risks of antibody and cell-mediated rejection, among others) and risks with VADs (bleeding, the acquisition of von Willebrand syndrome following continuous-flow VAD placement, infection (particularly drive-line related), thromboembolic events, VAD thrombosis and the potential for worsening aortic valve insufficiency), must be discussed with patients. It is therefore imperative to seek early involvement from transplantation centres to access advanced HF expertise.

Box 3. Future training opportunities offered by the BSH.

- 8th BSH Heart Failure Day for Revalidation and Training, 3 March 2016, Golden Jubilee Hotel, Glasgow
- 6th BSH Heart Failure Nurse Study Day, 4 March 2016, Golden Jubilee Hotel, Glasgow
- 19th BSH Annual Autumn Meeting, 24–25 November 2016, QE II Centre, London.

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The future of HF and service development

HF is a young, albeit growing, cardiac subspecialty. Over the last ten years, multidisciplinary engagement from the BSH have driven the development of NICE guidelines and quality standards for delivering optimal HF care. The National HF Audit, introduced in 2007 to monitor the care and treatment of patients with AHF in England and Wales, has documented improved clinical standards in HF. The Care Quality Commission recognises the delivery of HF care within a trust as a key discerning determinant of acute hospital care. HF care should be delivered in a multi-professional manner, adhere to common guidelines and should function across all sectors of care. Multidisciplinary strategies, lifestyle recommendations and rehabilitation programs should be incorporated to improve functional capacity, reduce symptoms, improve adherence with therapy and reduce hospitalisation. Clinicians must focus on improving communication, consistency and continuity of care for HF patients throughout their patient journey.

Structurally, it is suggested that hospitals ensure that 25% of their cardiologists have a remit in HF and that larger trusts have a specific HF cardiologist.[49] Services should aim to have one HF nurse per 100,000 of population to enable home visits, telephone contact, facilitation of telemonitoring, conducting of nurse-led clinics and provision of education, patient self-care and optimisation of medical therapy, particularly in the early discharge stages. Diagnostic services must be collaboratively developed between primary and secondary care to enable efficient and effective evaluation of patients. Therapeutic pathways must enable the timely delivery of evidence-based therapy with referral pathways in place for surgical and device input alongside palliative care. Structured follow-up of stable patients, with more frequent review of those recently discharged or with deteriorating symptoms, must be available to all to enable the provision of complex care. Furthermore, careful evaluation of services using audit, quality improvement, quality indicators and monitoring through registries are key to ensuring continuous service refinement.

Commissioning drivers, forthcoming acute HF quality standards and the development of the best practice tariff are a

reality for HF services, and will stimulate future improvements in the delivery of HF care. HF is increasing in prevalence, is high in morbidity and mortality and has an increasingly complex management. We must get the basics right and ensure that the evidence-based disease-modifying treatments are initiated and up-titrated. Each and every clinician must also play a proactive role in developing services and driving clinical standards to ensure the optimal delivery of acute and chronic HF services. ■

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