



GUIDELINES ON THE MANAGEMENT OF CHRONIC HEART FAILURE IN NORTHERN IRELAND

February 2005

These guidelines have been published by the Clinical Resource Efficiency Support Team (CREST), which is a small team of health care professionals established under the auspices of the Central Medical Advisory Committee in 1988. The aims of CREST are to promote clinical efficiency in the Health Service in Northern Ireland, while ensuring the highest possible standard of clinical practice is maintained.

The guidelines have been produced by a sub-group of health care professionals from varied backgrounds including Medical (Primary and Secondary care), Nursing, Management and Public Health, Chaired by Dr Gary McVeigh. CREST wishes to thank them and all those who contributed in any way to the development of these guidelines.

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ISBN 1-903982-10-3

Executive Summary ISBN 1-903982-11-1



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ACKNOWLEDGEMENTS:

NHS Modernisation Agency: Coronary Heart Disease Collaborative Service
Improvement Guide: Heart Failure
www.modern.nhs.uk/chd

NHS National Institute for Clinical Excellence: Management of Heart Failure:
Understanding NICE guidance - information for people with heart failure, their
carers, and the public
www.nice.org.uk

Department of Health: Coronary Heart Disease
www.doh.gov.uk/heart/heartfailure/index.html

NHS: National Service Framework for Coronary Heart Disease
www.nelh.nhs.uk/nsf/chd/nsf/main

NSW: Clinical Service Framework for Heart Failure
www.health.nsw.gov.au

British Heart Foundation
www.bhf.org.uk

British Society for Heart Failure
www.bcs.com/affiliates/bsh.html

SIGN Guideline 35 Section 2: Diagnosis of heart failure due to LVSD
www.show.scot.nhs.uk/sign/guidelines/fulltext/35/section2.5html

NSW Clinical Service Framework for Heart Failure
www.health.nsw.gov.au

Wolverhampton Heart Failure Service
www.doh.gov.uk/heart/heartfailure/resources



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ABBREVIATIONS

ACE	Angiotensin converting enzyme
BNP	B-type natriuretic peptide
CHD	Coronary heart disease
CHF	Chronic heart failure
CREST	Clinical Resource Efficiency Support Team
DIG	Digitalis Intervention Group
ECG	Electrocardiogram
ECHO	Echocardiogram
IHD	Ischaemic heart disease
NHS	National Health Service
NICE	National Institute for Clinical Excellence
NSF	National Service Framework
NYHA	New York Heart Association
SIGN	Scottish Intercollegiate Guidelines Network
CHARM	Candesartan in Heart Failure - Assessment of Reduction in Mortality and Morbidity Programme
COMET	Carvedilol or Metoprolol European Trial



CHRONIC HEART FAILURE

(i) PREFACE

Expert committees in the United States and Europe and national bodies including the Scottish Intercollegiate Guidelines Network (SIGN) and the National Institute for Clinical Excellence (NICE) have developed guidelines on the management of chronic heart failure (CHF) in adults. Under the auspices of the Clinical Resource Efficiency Support Team (CREST), a group was established to produce guidelines for the management of CHF for Northern Ireland. In formulating these guidelines the group took account of the evidence reviewed by the expert committees and national bodies and where recent evidence permitted, updated best practice recommendations.

These guidelines focus on aspects of care most strongly supported by evidence. For the interested reader, recent references provide further details of current and promising diagnostic, therapeutic and monitoring approaches or interventions that may become standard practice in the future. The appendices outline practical guidelines for diagnosing CHF, instituting drug therapy and a sample service model to improve organisation and standards of care.

The management of CHF can be improved and the challenge will be translating the knowledge set out in this document into organised systems that effectively deliver care for the benefit of all patients across Northern Ireland.

(ii) BACKGROUND

The National Service Framework for Coronary Heart Disease

The National Service Framework (NSF) for Coronary Heart Disease was issued by the Department of Health in March 2000 as the Government's blueprint for tackling heart disease in England and Wales¹ (www.nelh.nhs.uk). It set out national standards for preventing and treating coronary heart disease by defining models of service provision within primary and secondary care. Standard 11 outlined best practice standards for the prevention, diagnosis and care of people with chronic heart failure in England and Wales. This approach aims to provide co-ordinated and integrated care for people with CHF, detailed standards for the prevention, diagnosis and management of CHF and includes models of care which demonstrate the way in which care can be delivered in accordance with the standards. To achieve these standards, the NSF detailed explicit milestones, goals



and performance indicators against which progress is measured. The provision of an equitable, consistent and systematic approach to the diagnosis, investigation and management of patients with CHF will translate into improved outcomes for patients.

**(iii) MANAGEMENT OF CHRONIC HEART FAILURE IN ADULTS:
NICE GUIDELINES**

In July 2003, the National Institute for Clinical Excellence (NICE) issued a guideline to the National Health Service (NHS) in England and Wales on the management of CHF². The recommendations cover all aspects of the care pathway from first suspicion of diagnosis, through chronic disease management, to end-of-life issues. The guideline offers best practice advice on the care of adult patients who have symptoms or a diagnosis of CHF. It also provides evidence-based guidance on prevention, treatment, monitoring and support of patients with CHF.

The NSF initiative and the NICE guidelines for the management of CHF do not extend to Northern Ireland. In recognition of this, CREST established a working group to provide guidance for the management of adult heart failure for health care professionals in Northern Ireland.



1. INTRODUCTION

With an ageing population and improved care for coronary heart disease (CHD), CHF is an increasingly common condition^{3,4}. There is no cure for this progressive condition and it has a dramatic effect on the quality of life of patients. A great deal can be done to manage CHF more effectively, with better outcomes for patients and their families and with more cost-effective use of staff, resources, beds and medicines in the local health economy^{2,5}.

2. DEFINITION OF CHRONIC HEART FAILURE

CHF is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to function as a pump to support a physiological circulation⁶. CHF is not a diagnosis in itself. It is a clinical syndrome - a collection of symptoms (breathlessness, fatigue) and signs (fluid retention) - but the term is commonly used as a diagnostic label in clinical practice. CHF has a number of causes with most cases in the United Kingdom due to CHD and about a third from hypertensive heart disease^{6,7}.

3. INCIDENCE AND PREVALENCE OF CHRONIC HEART FAILURE

The prevalence and population burden of CHF due to CHD is increasing despite the declining overall mortality from CHD⁸. This is due to both an ageing population and to more people surviving acute heart attacks but with a damaged myocardium and residual left ventricular dysfunction.

It is estimated that the incidence of CHF is about one new case per 1,000 population per year and is rising at about 10% per year. This increases with age to more than 10 cases per 1,000 population in those aged 85 years and over. The median age of clinical presentation is 76 years⁵. The male : female ratio is 2:1. Population prevalence rates have been estimated between 3 and 20 people per 1,000 increasing to at least 80 cases per 1,000 among people aged 75 and over¹.

CHF accounts for a total of 1 million inpatient bed days (2% of all NHS inpatient bed days) and 5% of emergency admissions. These figures are projected to rise by 50% over the next 25 years. CHF is responsible for 1-2% of healthcare expenditure with approximately 70% of the total due to the cost of hospitalisation². Total healthcare costs increase markedly with disease severity⁹. On average, a general practitioner will look after 30 patients with heart failure and suspect a new

diagnosis in approximately 10 patients annually. Most individuals with CHF will be seen in primary care between 10 to 15 times per year. The total general practitioner cost has been estimated at £104 million per annum³.

These figures represent best estimates. Accurate quantification of the prevalence is complicated by the fact that many patients with left ventricular systolic dysfunction or with early CHF have few symptoms and some patients with typical symptoms are shown on investigation not to have CHF¹⁰⁻¹². We do not have an accurate picture of the extent of the problem locally. However, sufficient information on incidence and prevalence is available to make decisions about future service provision and the resources required.

4. DEMOGRAPHICS AND CONCOMITANT DISORDERS IN CHRONIC HEART FAILURE

Community-based epidemiological studies have provided important information on the demography of CHF, providing insight into the impact of CHF on public health¹³. CHF is often accompanied by a range of concomitant disorders that both contribute to the cause of the disease and play a key role in its progression and response to treatment. Information from large-scale multicentre trials on the most common co-morbidities, ischaemic heart disease (IHD), hypertension and diabetes mellitus, provide guidance in therapeutic decision-making process. Furthermore, as CHF is often an endpoint in intervention trials of both hypertension and diabetes these studies afford important information on the prevention of CHF in these common diseases¹⁴.

There are sex-based differences in co-morbidities in patients with CHF with females tending to be older, to have associated diabetes mellitus and hypertension, and to have more preserved ventricular function¹⁴. Sex-based differences in mortality have generally not been observed in females treated with angiotensin converting enzyme (ACE) inhibitors and beta blockers. In the digitalis intervention group (DIG) trial females did have a higher morbidity than their male counterparts¹⁵. This observation may have resulted from the higher plasma concentration of digoxin found in female patients.

Age does not appear to influence the beneficial effects of ACE inhibitors or beta blockers in chronic systolic heart failure although tolerability of treatment may decrease with advance in age. Ongoing trials are currently addressing therapeutic interventions in elderly patients more representative of those treated in the community with CHF^{16,17}.

Hypertension contributes pathogenetically to the development of systolic and diastolic heart failure and is also a major risk factor for IHD, stroke and renal failure. The effect of antihypertensive therapies in limiting the development of chronic heart failure in patients with systo-diastolic and isolated systolic hypertension supports a major contribution of this co-morbidity to onset and progression of CHF¹⁸⁻²⁰. African-American patients tend to respond less well to ACE inhibition for treatment of hypertension²¹. In established CHF, the benefits of ACE inhibitor therapy has been demonstrated in some, but not all, CHF patients of African-American origin²².

Coronary artery disease features prominently as a cause of CHF²³. The optimum management of IHD, especially the early provision of thrombolysis and secondary prevention is therefore of great importance. Many patients with myocardial ischaemia and CHF may have hibernating, but potentially viable, myocardium²⁴. Undoubtedly, part of the beneficial effect of neurohumoral antagonists in CHF may result from improvements in underlying ischaemia²⁵.

Diabetes mellitus is a common co-morbidity in CHF²⁶. Patients with diabetes are not only at higher risk of developing CHF but also tend to be more symptomatic for a given degree of systolic dysfunction and have a higher mortality than non-diabetic individuals^{27,28}. Several intervention trials have shown the therapeutic benefits of blocking the renin-angiotensin system and highlight the importance of this therapeutic approach in prevention and treatment of CHF in diabetes mellitus¹⁴. The major trials of beta blockers in CHF indicate similar benefits in the diabetic sub-groups to the overall benefit^{29,30}. Care is required with the prescription of spironolactone in CHF patients with diabetes in whom hyporeninaemic hypoaldosteronism is common and diligent monitoring of serum potassium concentration is recommended.

Atrial fibrillation is a common co-morbidity with CHF, present in up to 1/3 of all patients enrolled in the major intervention trials. However, there is no evidence that restoration of sinus rhythm is better than control of ventricular response in patients with CHF and atrial fibrillation³¹. The benefits of pharmacological management of ventricular arrhythmias in patients with established CHF are not well established. Recent evidence indicates that selected patients may benefit from implantation of cardioverter defibrillators.

A recent community based epidemiological study examined the prevalence of left ventricular systolic dysfunction in patients at high risk for heart failure³². This targeted echocardiographic study showed definite systolic dysfunction (ejection fraction <40%) in 22% of patients with a previous myocardial infarction and 6% of patients with diabetes mellitus. Based on these results the authors suggested a targeted screening programme may be warranted in this group of patients.

5. DIAGNOSIS

The full evaluation of a patient with CHF requires more than confirmation of a diagnosis of the syndrome. A complete evaluation requires consideration of the underlying abnormality of the heart, an assessment of severity, aetiology, precipitating and exacerbating factors, identification of concomitant disease relevant to management and an estimation of prognosis.

Diagnosing CHF on clinical grounds is neither straightforward nor reliable³³⁻³⁷. Importantly, the historical basis for a diagnosis of CHF should be reviewed and only patients whose diagnosis is confirmed should be managed in accordance with guideline advice. A comprehensive clinical history and examination is of great value in eliciting symptoms and signs that lend support for the diagnosis of CHF and can provide clues to the underlying aetiology. The most likely underlying cause for CHF is CHD and evidence for CHD should always be sought. Hypertension, diabetes mellitus, anaemia and thyroid disease can also lead to CHF. The identification of valvular disease on clinical examination may be aetiologically important. Drugs (especially alcohol) and multisystem diseases such as haemochromatosis, amyloidosis and collagen vascular disorders should not be overlooked.

The symptoms of CHF are non-specific with dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea and fatigue having relatively low sensitivities and specificities. Similarly, signs compatible with the diagnosis such as a raised jugular venous pressure, tachycardia, third heart sound and displaced apex beat are non-specific, especially if found in isolation. Therefore, clinical features may suggest the diagnosis but are not sufficient to establish the diagnosis of CHF. A table of symptoms and signs and conditions that may mimic CHF are shown below.

SYMPTOMS AND SIGNS THAT MAY MIMIC CHF	
SYMPTOMS	SIGNS
<ul style="list-style-type: none"> • Shortness of breath on exertion • Short of breath on lying flat • Wake up short of breath • Fatigue • Weight loss (muscle loss) • Weight gain (oedema) • Poor appetite • Irregular heart beat • Wheezy chest • Irritating cough 	<ul style="list-style-type: none"> • Tachycardia • Tachypnoea • Raised jugular venous pressure pulse • Gallop rhythm (3rd or 4th heart sound) • Fine end-inspiratory crepitations • Rhonchi • Dependent oedema • Cachexia

OTHER CONDITIONS THAT MAY PRESENT WITH SIMILAR SYMPTOMS

- | | |
|--|--|
| <ul style="list-style-type: none"> • Obesity • Chest disease - including lung, diaphragm or chest wall • Venous insufficiency in lower limbs • Drug-induced ankle swelling (eg dihydropyridine calcium channel blockers) • Drug-induced fluid retention (eg NSAIDs) • Hypoalbuminaemia | <ul style="list-style-type: none"> • Intrinsic renal or hepatic disease • Pulmonary embolic disease • Depression and/or anxiety disorders • Severe anaemia or thyroid disease • Bilateral renal artery stenosis |
|--|--|

Although individual symptoms and signs have poor predictive value in the diagnosis of heart failure, establishing a clinical impression or suspicion of the syndrome is essential in determining the need for further evaluation and investigation. Typically blood (biochemical profile, full blood count, thyroid function tests, fasting glucose, liver function tests and lipid profile), respiratory (peak flow, spirometry), radiology (chest x-ray) and urine analysis tests or investigations may be requested in patients with suspected heart failure. The investigations help confirm or refute the diagnosis, identify the aetiology of heart failure, exclude conditions that exacerbate myocardial ischaemia and provide an estimate of cardiovascular risk and prognosis. Appendix 1 shows an algorithm employed by NICE summarising the recommendations for the diagnosis of heart failure.

Echocardiography (ECHO) is the single most important investigation in the evaluation of a patient with suspected CHF^{38,39}. It often confirms the diagnosis and at the same time identifies the underlying cause of the problem. The test is most informative after a careful history, physical examination, electrocardiogram (ECG) and chest radiograph have been obtained so that appropriate questions can be addressed. Most doctors will require the technical findings of echocardiography to be interpreted. Sometimes it is difficult to get an optimal scan and reporting is not always in a standardised format. Alternative methods of cardiac imaging such as transoesophageal echocardiography, radionuclide ventriculography and magnetic resonance imaging should be considered when poor image quality is reported on echocardiography.

Recognising that ECHO is a limited resource and must be used well, other screening tests have been suggested to rule out CHF. A 12-lead ECG, in the

absence of any history of CHD, is a valuable screening tool to identify those who do not require ECHO⁴⁰. The measurement of B-type natriuretic peptide (BNP) offers another screening tool. It is a simple blood test with a high negative specificity which means that it is very useful as a test to exclude CHF without the need for more complicated or expensive tests. BNP improves accuracy in diagnosing CHF in patients presenting with acute dyspnoea^{41,42}. It also holds promise in predicting outcome and response to therapy⁴³⁻⁴⁵. However, as with all biomarkers, it must be used in the context of good clinical judgement and physicians should be aware of important biological variability. BNP levels increase with age, hypertension, renal insufficiency, atrial fibrillation and sleep apnoea⁴⁶. Appropriate cut-off values should be agreed with the local biochemistry laboratory.

Economic analyses and recent clinical trial data suggest that knowledge of the BNP value can result in more efficient use of ECHO facilities, more rapid initiation of treatment, shorter hospital stays and lower costs^{2,42}. Use of this assay is recommended by NICE in the diagnostic work-up of a patient with suspected CHF. A normal ECG and BNP value effectively excludes a diagnosis of CHF. If one or both tests are abnormal, transthoracic ECHO with doppler ultrasonography should be performed. A patient with breathlessness found to have a clinically significant murmur should be referred specifically for ECHO.

6. DIASTOLIC HEART FAILURE

Clinicians are willing to accept a diagnosis of systolic CHF and if the left ventricular ejection fraction is low the diagnosis is seldom questioned. Diastolic heart failure refers to the clinical syndrome of CHF with a preserved left ventricular ejection fraction (>50%) in the absence of major valve disease and accompanied by evidence of abnormal diastolic relaxation⁴⁷. Heterogeneity in the reported populations and diagnostic criteria has led to uncertainty regarding the true prevalence of the condition. Some studies suggest that around 30% of older patients with apparent CHF have diastolic CHF⁴⁸. Typically these patients are elderly, more likely to be women and often have raised blood pressure in association with ventricular hypertrophy⁴⁹. Only recently has firm evidence become available that primary abnormalities in diastolic function are present in diastolic heart failure and can occur without hypertrophy of the left ventricle⁵⁰.

Clinical characteristics alone cannot distinguish reliably between systolic and diastolic heart failure. While doppler ECHO is employed to aid the diagnosis of diastolic dysfunction, altered transmitral filling patterns are ubiquitous in elderly patients. Recent evidence indicates the use of traditional doppler ECHO indices in

the assessment of diastolic dysfunction are unreliable⁵¹. Newer non-invasive indices of diastolic function may provide more precise information⁵². The advent of the biomarker BNP may help confirm the presence of CHF in patients with suspected diastolic heart failure. As is the case with systolic heart failure, prevention of diastolic heart failure can be achieved through better control of hypertension and other cardiovascular risk factors in the community.

7. SEVERITY OF SYMPTOMS

Whilst investigations can give an indication of the physiological impact and severity of CHF, it is also useful in clinical practice to have an estimate of the functional impact on an individual patient. The New York Heart Association (NYHA) classification of severity of CHF symptoms is widely accepted and valid^{2,5}. This scale, which has 4 levels, should be used as a measure of patient incapacity and severity of symptoms. The 4 levels are:

- Class 1** - no restriction of activity at all - asymptomatic left ventricular function is included in this category
- Class 2** - slight limitation by symptoms - comfortable at rest but ordinary activity results in breathlessness, fatigue, palpitation, or angina
- Class 3** - marked limitation by symptoms - comfortable at rest but less than ordinary activity will produce symptoms
- Class 4** - unable to undertake any activity without symptoms - symptoms at rest that increase with any activity

The functional class can deteriorate unpredictably over time and symptom severity does not always reflect the underlying severity of the heart problem ie mild symptoms may be found in patients with significant cardiac damage and vice versa⁵. Changes in medication or diet can influence functional capacity in the absence of any measurable change in heart function.

In summary, establishing the diagnosis of CHF should be standardised across Northern Ireland and optimised. Clinicians should ensure that all patients suspected of having CHF undergo a comprehensive clinical assessment, with history, physical examination and appropriate diagnostic investigations. The use of good clinical acumen in primary care should continue to provide the basis to recognise possible CHF and ensure early investigation to establish a diagnosis and

underlying cause. The ideal service should provide ECHO as part of a “one stop service”, which also provides expert opinion in addition to other supporting investigations. Regional implementation of a service model would help ensure equitable access and standards of care, based on evidence and best practice, are provided for patients with CHF.

8. TREATMENT OF CHRONIC HEART FAILURE

8.1 Introduction

The aims of therapy in CHF are to improve quality of life and life expectancy. The relative importance of these aims will vary between individual patients, should take into account patients’ preferences and may change with time.

9. NON-PHARMACOLOGICAL/LIFESTYLE MANAGEMENT

9.1 Education

Education of patients and carers is an important element of non-pharmacological management. Patients adhere better with treatment when they understand CHF and the rationale for treatment.

9.2 Exercise Training

Inactivity can lead to deconditioning which leads to worsening of symptoms and exercise performance^{53,54}. Exercise training is known to reduce the debilitating symptoms of CHF, such as breathlessness and fatigue, through effects on the cardiovascular and musculoskeletal systems. A recent meta-analysis suggests that mortality and admission to hospital are significantly reduced by exercise training in patients with CHF due to left ventricular systolic dysfunction⁵⁵.

Rehabilitation programmes that combine exercise, psychological support and education may be of greater benefit than programmes that provide only one of these components⁵⁶. The optimal programme in terms of design, frequency or

duration has not been established. Little is known about the health economics of rehabilitation programmes for CHF although evidence from other disease areas suggests that if programmes can reduce the risk of hospitalisation they may represent a cost effective use of resources².

9.3 Sexual Activity

Patients with CHF and their partners are often anxious about how sexual activity may affect the heart. Healthcare professionals should be prepared to approach this sensitive issue as it is unlikely to be raised by the patient. While no evidence base is available patients with CHF who are not severely compromised may be advised that it is fine to maintain a sexual relationship within the limitations of their symptoms. If appropriate, advise about the use of nitrolingual spray prior to sexual activity. Patients and their partners should also be advised that the symptoms of CHF and certain medications may reduce sexual interest.

9.4 Smoking Cessation

Whilst there are no published studies on the effect of stopping smoking in patients with CHF, given the detrimental effects on haemodynamics and oxygen delivery, it would seem prudent and logical to advise all patients to stop smoking. Patients should be provided with counselling and education programmes to assist with quitting.

9.5 Alcohol Intake

Chronic excessive alcohol consumption can be cardiotoxic and patients with alcohol-related CHF should abstain from drinking alcohol. Alcohol may have other detrimental effects on the heart by precipitating arrhythmias (e.g. atrial fibrillation) or causing an acute deterioration in cardiac function. For patients with established CHF the volume load of alcoholic beverages may make oedema more difficult to control.

9.6 Fluid Intake

Sodium and water retention, leading to an expansion of extracellular fluid volume, are important components of the heart failure syndrome. Although excessive

amounts of fluid should be avoided and fluid restriction is commonly advocated, advice on daily fluid intake will vary depending on the clinical status of the patient. In practice a restriction of 1.5-2 litres daily is often advised. Patients and carers should be advised that fluid intake includes soups, sauces, ice cream and alcohol.

9.7 Daily Weighing

Patients with CHF should be advised to weigh themselves on a daily basis to detect sudden or unexpected weight gain or weight loss (more than 2kg in 3 days) and to alert their healthcare professional. When patients are able to self-manage they may titrate diuretic therapy according to a pre-specified management plan.

9.8 Salt Intake

The evidence base for diet and nutrition in patients with CHF is limited and conflicting advice exists about salt intake. However severe salt restriction (<2gms daily) is rarely necessary. All patients should be discouraged from adding salt to their food and should reduce the amount of salt they add during cooking. Ready-to-cook meals and convenience foods contain large amounts of salt and may also promote fluid retention and make oedema, in the setting of CHF, more difficult to control. Salt substitutes should be avoided as these are rich in potassium and when added in large quantities may lead to hyperkalaemia especially if the patient is taking an ACE inhibitor and/or spironolactone.

9.9 Obesity

Management of CHF should include weight reduction in the overweight patient. Reducing obesity will reduce the work of the heart, lower blood pressure and improve the patient's lipid profile, which are important factors in secondary prevention. Referral for dietetic advice would be appropriate.

9.10 Cachexia

This is a frequent complication in advanced CHF and involves loss of muscle mass (including cardiac muscle) as well as adipose tissue. The aim is to increase non-oedematous body weight with small frequent meals indicated to lessen nausea and bloating. Referral to a dietitian would be indicated.

9.11 Immunisations

Evidence of intercurrent infection should be sought and treated appropriately. There is a little documented evidence on the benefits of immunisation in patients with CHF. However, in practice, patients with CHF should be offered an annual vaccination against influenza and vaccination for pneumococcal infection (only required once).

9.12 Travel

Air travel should be possible for the majority of patients with CHF depending on their clinical condition at the time of travel. On long flights patients should be advised to walk around regularly and not become dehydrated. If visiting hot or humid countries, diuretic dose may need to be altered to avoid dehydration.

9.13 Psychological Management

There is little literature about psychological interventions for patients with CHF. Depression tends to be more common in patients with CHF than the general population. Drug therapy for depression can exacerbate fluid retention and appropriate arrhythmias.

NICE guidelines recommend that if depression is co-existing with CHF, it should be treated. Good communication between healthcare professionals, patients and carers is essential for best management.

9.14 Medication Advice

Advice should be given regarding the name, dose, timing and route of all medication. Desired effects and potential side effects should be discussed.

Patients should be made aware of titration schedule of some medication and that improvement in symptoms, if any, may be gradual. The importance of concordance should be emphasised. Patients should contact their healthcare professional if they have side effects of medication. The CHF team and the patient should be aware of the potential for corticosteroids, non-steroidal anti-inflammatory medications and negative inotropic medications to precipitate or exacerbate CHF. Physicians should prescribe these medications with due caution in patients who have or are at increased risk for CHF and seek specialist advice where necessary.

10. PHARMACOLOGICAL TREATMENT OF CHRONIC HEART FAILURE

The objectives of treatment for systolic (and diastolic) heart failure are to relieve symptoms, enhance exercise tolerance and quality of life, reduce hospital admission and readmission rates and improve survival. Table 1 indicates the benefits of particular drug therapies in attaining these objectives and Appendix 2 shows a treatment algorithm for the pharmacological management of CHF.

The objectives of treatment for diastolic heart failure are similar to those for systolic heart failure. The treatment of diastolic heart failure is largely empiric due to the lack of an evidence base. Chronic treatment includes restriction of dietary sodium and control of hypertension. A single randomised trial with the angiotensin receptor blocker candesartan, in patients with preserved left ventricular function, demonstrated a modest impact on reducing future hospital admissions for CHF compared with placebo⁵⁷.

The information relating to specific drug treatment represents a guide to therapy rather than a didactic protocol that must be followed in all patients. Treatment should always be tailored to the individual patient with referral for specialist advice considered where appropriate.

10.1 Angiotensin Converting Enzyme (ACE) Inhibitors

ACE inhibitors improve symptoms, reduce event rates and reduce mortality in CHF^{58,59}. The benefits tend to be more marked with more severe left ventricular systolic dysfunction although there is benefit for all NYHA classes⁶⁰. High doses of ACE inhibitors do not necessarily improve symptoms or prolong life more than low doses but do appear to be more effective in reducing the risk of hospitalisation⁶¹. All patients with left ventricular systolic dysfunction should be considered for treatment and it is recommended that an ACE inhibitor be introduced before beta-blockade. Some clinicians may wish to up-titrate both classes of agents together but this requires intensive monitoring and careful supervision. Therapy is initiated at the appropriate dose and up-titrated at two weekly intervals until the optimal tolerated or target dose is achieved. Treatment of CHF with ACE inhibitors is cost effective largely due to reduced risk of hospitalisation.

In general, the side-effect profile is low, but about 10% of patients experience a dry cough, perhaps related to bradykinin. Absolute contraindications include a history of angio-oedema with past exposure to the class of agents, bilateral renal artery

stenosis, pregnancy and cardiogenic shock. A fall in blood pressure may occur after the first dose, especially in hypotensive patients who are hyponatraemic and on large doses of diuretics. Therapy should be used with extreme caution in patients with aortic stenosis. Renal function should be closely monitored - an increase in creatinine of about 10-15% is normal after ACE inhibitor treatment. The drugs should be used with added caution in patients with a serum creatinine $>200\mu\text{mol/L}$ or serum potassium $>5.0\text{mmol/L}$. In all patients, the dose should be increased until that recommended by the trials evidence is reached, or side-effects occur. Practical recommendations and an algorithm for the use of ACE inhibitors are shown in Appendix 3.

For those patients unable to tolerate ACE inhibitors, angiotensin II receptor antagonists offer a suitable alternative⁶². There are seven currently available, but none yet has a license for first-line treatment in CHF. In patients with CHF due to left ventricular systolic dysfunction, treatment with an angiotensin II antagonist has been shown to reduce the risk of hospitalisation⁶³ and reduce mortality compared with placebo⁶⁴⁻⁶⁶. In the candesartan in CHF (CHARM - added) study additional benefits were apparent in terms of a reduction in cardiovascular mortality and all cause mortality and hospitalisations in patients receiving ACE inhibitors, beta blockers and spironolactone⁶⁶. Careful monitoring of renal function and serum potassium is required if combination therapy with an ACE inhibitor and aldosterone antagonist is contemplated; specialist supervision is recommended. In patients with preserved left ventricular function and CHF, angiotensin receptor blockade with candesartan reduced the number of investigator-reported admissions for CHF. As a class angiotensin II receptor blockers are better tolerated than ACE inhibitors.

10.2 Beta-Blockers

For many years, CHF was a contra-indication to the use of beta-blockers, but now they are known to have an important role in event prevention⁶⁷⁻⁶⁹. Only carvedilol and bisoprolol and long-acting metoprolol are licensed in the United Kingdom for use in CHF. Beta-blockers increase life expectancy in patients with CHF due to left ventricular systolic dysfunction compared with placebo; an effect seen in all functional classes of CHF⁷⁰. Results from the Carvedilol or Metoprolol European Trial (COMET)⁷¹ indicated that carvedilol reduced mortality to a greater extent than metoprolol although the mechanisms responsible for this finding remain the subject of debate⁷². When initiating beta-blocker therapy in CHF, patients should be commenced on an agent licensed for treatment of the condition and where possible, the dose of beta-blocker should be up-titrated to that shown to be of

benefit in outcome trials. In general, these doses were well tolerated by the patient populations included in the randomised outcome studies. The key to their successful use is to start treatment with a very low dose (e.g. 3.125 mg of carvedilol or 1.25 mg of bisoprolol) and build the dose up gradually over a period of weeks. This requires intensive supervision and monitoring. Relative contra-indications remain bradycardia, wheezing and cold extremities. Practical recommendations and an algorithm for the use of beta-blockers is shown in Appendix 4.

10.3 Aldosterone Antagonists

In patients with moderate to severe CHF (NYHA class III and IV) due to left ventricular systolic dysfunction the addition of spironolactone to baseline therapy reduced mortality compared with placebo⁷³. There was also a reduction in hospitalisation for cardiac causes. Approximately 10% of patients receiving spironolactone experienced gynaecomastia or breast pain due to the drug's affinity for androgen receptors. Careful monitoring of renal function and especially serum potassium is required if combination therapy with inhibitors of the renin-angiotensin system and aldosterone antagonists are contemplated. Institution under specialist supervision is recommended. Practical recommendations and an algorithm for the use of spironolactone is shown in Appendix 5.

A more recent study using the aldosterone antagonist eplerenone, in patients with CHF after myocardial infarction, demonstrated a significant reduction in mortality and in risk of hospitalisation for CHF compared to placebo⁷⁴. The incidence of gynaecomastia did not differ between the eplerenone and placebo groups due to the drug's low affinity for androgen receptors.

10.4 Diuretics

There is little hard evidence that diuretics reduce mortality compared with placebo but their role in symptom relief in CHF is undisputed⁷⁵. Diuretics should be routinely used to treat fluid overload, either pulmonary or peripheral oedema and titrated (up or down) according to need following initiation of subsequent CHF therapies. Loop diuretics are typically employed and more effective than thiazides in promoting diuresis and natriuresis. When prescribed with ACE inhibitors or spironolactone, potassium supplementation or a potassium sparing diuretic may not be required. Careful monitoring of potassium levels and renal function is essential.

10.5 Digoxin

In the DIG study there is no evidence to suggest a difference in mortality between the control group and the digoxin-treated cohort in patients with CHF in sinus rhythm⁷⁶. Digoxin therapy was associated with lower rates of hospitalisation and clinical deterioration. Digoxin would be indicated for patients with worsening or severe CHF due to systolic dysfunction in patients already receiving ACE inhibitor, beta-blocker and diuretic therapy. Digoxin retains an important role in the treatment of patients with any degree of CHF and atrial fibrillation.

Digoxin has a narrow therapeutic window, with arrhythmias and gastrointestinal side effects being the most common clinical problems. The usual daily dose being of oral digoxin is 125-250 µg; lower if the patient is elderly or has impaired renal function. A number of drugs can alter the pharmacokinetics of digoxin including antiarrhythmic drugs (amiodarone, verapamil, propafenone) and antibiotics (erythromycin and tetracycline). These interactions can significantly alter the serum digoxin concentration. Post-hoc analysis of the DIG trial suggested that the serum digoxin concentration was an independent predictor of mortality and suggested an optimal serum concentration range of 0.5 - 0.8 ng/mL⁷⁷. Two important pharmacodynamic interactions for digoxin include increased toxicity in the presence of hypokalaemia and bradycardia when combined with beta-blockers and non-dihydropyridine calcium channel blockers.

10.6 Nitrates and Hydralazine

The combination of nitrates and hydralazine reduce symptoms and events, but only when prescribed at high dose⁷⁸. The combination is less effective than ACE inhibitors in the treatment of CHF and is less well tolerated. Combination is now rarely used due to side-effects and nitrates by themselves confer no added benefit. The combination therapy may have a role in patients intolerant to ACE inhibitors or angiotensin II receptor antagonists.

Statins and aspirin should be continued in patients with underlying IHD, with the same therapeutic targets. Concern has been expressed that aspirin may reduce the beneficial effect of ACE inhibitors in heart failure and atherosclerotic arterial disease although this finding is not confirmed in all reports². Aspirin continues to be recommended in patients with the combination of heart failure and atherosclerotic vascular disease.

10.7 Warfarin

Although oral anticoagulation is indicated in certain groups of patients with heart failure (e.g. atrial fibrillation) the available data does not support the routine use of warfarin in CHF patients in sinus rhythm⁷⁹. Some experts would advocate the use of warfarin in patients with very poor left ventricular function. A large randomised trial of warfarin in CHF patients in sinus rhythm is currently in progress.

10.8 Amiodarone

The role of amiodarone in the treatment of CHF is controversial. The drug has numerous side-effects (including thyroid dysfunction, liver and lung toxicity and neuropathy) and requires careful monitoring. A recent 5-year study in patients with grade II and III CHF, not yet published in full, showed no benefit of amiodarone in improving life expectancy compared with placebo⁸⁰.

10.9 Calcium Channel Blockers

There is no evidence to suggest that calcium channel blockers improve life expectancy compared with placebo in patients with CHF. There is evidence to suggest some members of the class (e.g. verapamil, diltiazem and nifedipine) can be associated with clinical deterioration².

11. INTERVENTIONAL METHODS

Contemporary evidence-based medical regimes improve left ventricular function and reduce the risk of death from pump failure and arrhythmia in patients with CHF. Nevertheless, there remains significant morbidity and mortality in the setting of CHF. Further benefit from drugs that antagonise neurohumoral pathways may not be achieved. Trials of new agents, such as endothelin antagonists, vasopeptidase inhibitors and tumour necrosis factor receptor blockade have not shown an incremental survival benefit⁸¹⁻⁸³.

Interventional methods have been introduced with the aim of improving morbidity and mortality in CHF. In addition to cardiac transplantation, devices are now employed to reduce sudden death and to improve the mechanical disadvantage during systole that results from conduction disturbances that affect approximately 30% of patients with CHF.

11.1 Heart Transplantation

Heart transplantation currently offers unparalleled symptomatic relief and restoration of quality of life but alone cannot solve the increasing public health problem of end-stage CHF. Heart transplantation has a high early mortality with 15%-20% of recipients dying within a year of operation^{84,85}. Thereafter the death rate is constant at about 4% per year. Approximately 50% of patients can be expected to be alive after 10 years and 15% after 20 years. Recipients never regain full health as the immunological effects of the donor heart and requirement for immunosuppression introduce new sources of illness and patients require regular hospital surveillance and often repeat admission to hospital⁸⁶.

11.2 Biventricular Pacing and Cardioverter Defibrillators for Chronic Heart Failure

Cardiac resynchronisation therapy is currently accepted as an adjunct to medical treatment of symptomatic CHF in patients with severe left ventricular systolic dysfunction and ECG manifestations of ventricular conduction delay⁸⁷. The aim of biventricular pacing is to produce simultaneous stimulation of the right and left ventricles and to reduce intraventricular contraction differences. Studies indicate that biventricular pacing improves symptoms in patients with heart failure and left bundle branch block and may reduce the risk of clinical deterioration^{88,89}. However, the clinical response to biventricular pacing is heterogeneous and difficulties surround the issue of patient selection⁹⁰. Up to 30% of patients who receive an implant do not respond to the intervention⁹¹. Growing evidence suggests that the timing and extent of mechanical dyssynchrony are poorly related to QRS duration, highlighting the poor predictive value of QRS duration for the identification of mechanical dyssynchrony^{91,92}. The optimal echocardiographic technique to identify mechanical dyssynchrony has not yet been established.

In considering devices for CHF a growing body of evidence suggest some patients benefit from combined biventricular pacing and insertion of an implantable cardioverter-defibrillator device⁹³. A recent study demonstrated that cardiac resynchronisation therapy decreased the combined risk of death and first hospitalisation and when combined with an implantable defibrillator, significantly reduced mortality in patients with advanced (class III or IV) CHF⁹³. Studies using an implantable cardioverter defibrillator alone in patients with non-ischaemic cardiomyopathy demonstrate a reduction in sudden death but not death from any cause⁹⁴. However, results made available so far from the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) indicate that in patients with class II or III CHF

and ejection fraction of <35%, on excellent background medical therapy, ICD therapy is associated with significant improvements in mortality compared with amiodarone or placebo therapy⁹⁵. Current guidance from the NICE Technology Appraisal Guidance recommends the use of implantable cardioverter-defibrillators in patients with sustained ventricular tachycardia without syncope/cardiac arrest who have an associated reduction in ejection fraction of <35% but are no worse than class III NYHA functional classification of CHF⁹⁶.

Recommendations for implantation of these devices will change with the publication of future trial data⁹⁷. Patients considered as potential candidates for the implantation of these devices should be referred to a specialist with a specific interest in this area. Device therapy for CHF will likely remain an adjunct to optimal medical treatment or revascularisation. Cost and availability of appropriate expertise will probably be the limiting factors in determining how widespread the use of such devices will become.

11.3 Coronary Revascularisation

Although IHD is the most common cause of CHF, the benefits of revascularisation in patients with CHF remain uncertain. In a recent study, a significant majority of patients with CHF due to CHD had evidence of “hibernating” myocardium (viable but non-contractile cardiac muscle) or demonstrated reversible ischaemia in two or more segments of myocardium⁹⁸. A variety of imaging techniques are employed to detect non-contractile but viable myocardium including perfusion scintigraphy, stress ECHO, magnetic resonance imaging and positron emission tomography⁹⁹. Currently there is evidence from non-randomised studies supporting revascularisation for the treatment of “myocardial hibernation” and an indication that medical therapy with carvedilol may have modest benefits^{98,100}. There are no published randomised comparisons between revascularisation and medical therapy in patients with myocardial hibernation although one trial is currently ongoing⁷². Revascularisation should still be considered if there is significant angina.

In summary, modern medical management has made a considerable impact on the survival in CHF although the prognosis remains unacceptably poor. Indeed, despite advances in medical therapy, the prognosis remains worse than for many cancers. The prescription of and compliance with, evidence-based pharmacological therapy remains suboptimal. A recent survey indicated that 60% of patients with CHF were prescribed ACE inhibitors, 34% beta-blockers but only 20% received these drugs in combination¹⁰¹. Furthermore, the prescribed doses were only 50% of



those employed in the randomised outcome studies and recommended by expert panels¹⁰¹. These data highlight a deficit in prescription of therapies that improve morbidity and mortality in CHF and suggest that improved organisation of care for CHF is required. Non-adherence to therapy is an important issue and simplifying dosing regimes and educating patients and their carers about their medicines is particularly important.

It is unlikely that interventional therapies will significantly impact outcome on a population basis given the projected increase in cases of CHF and limited resource and expertise available.

12. MULTIDISCIPLINARY APPROACH TO CHF MANAGEMENT

The management of CHF is likely to be shared between health care professionals in primary and secondary care. Patients and their carers are now increasingly involved in management decisions. Currently, evidence suggests that the major failings of management relate to poor communication between health care professionals and between patients and the professionals caring for them.

Heart failure nurses seem ideally placed to address several issues in the management of patients with CHF that need to be addressed in order to improve outcomes¹⁰²⁻¹⁰⁴. Nurses can provide much needed continuity between the primary and secondary settings. They can provide education to patients with CHF in order to teach self-care. They can monitor compliance with medication, diet or symptom monitoring to improve quality of life and prevent hospital readmission¹⁰⁵. Specialist nurses can titrate medications within specified guidelines in order to optimise therapy and provide the psychological and social support to both patients and their carers. Recent evidence suggests that nurse-led heart failure clinics improve both survival and self-care behaviour in patients with CHF^{106,107}.

All CHF patients admitted to hospital should be referred to a physician with an interest in the condition for review and advice on management of their CHF. These patients should have access to allied health professionals including dietitians, pharmacy, physiotherapy, social work and cardiac rehabilitation services. The patients should be assessed by a CHF nurse specialist to ensure that CHF education is initiated in the hospital and co-ordinated management is continued on discharge from hospital in liaison with general practice and community health services¹⁰⁸.

As the clinical condition of a person with CHF may fluctuate and repeated admission to hospital is common, all patients with CHF require monitoring and

continuing care. At review an assessment of functional capacity (by history or 6-minute walk test), fluid status (physical examination), cardiac rhythm (clinical examination, ECHO) and laboratory assessment (especially electrolytes and creatinine) are recommended. Advice should be provided on non-pharmacological interventions in the control of CHF and patients should have access to programmes that equip and enable them and their carers to actively participate in self-management of CHF following discharge from hospital. An action plan which covers self-management of important aspects of the disease including recognition of symptoms and signs that signal the need for professional attention and information on how to obtain this attention should be provided for all patients. The provision of access and advice from appropriate health professionals requires effective communication and linkage between hospital and community-based services for all patients with CHF.

To achieve these goals a number of CHF service models have been developed in England and Wales under the auspices of the NSF. An example of the Wolverhampton Heart Failure Service model is shown in Appendix 6. This service model is patient-centred, works across the primary/secondary care interface and is based on evidence as well as best practice.

13. PALLIATIVE CARE FOR PATIENTS WITH END-STAGE HEART FAILURE

The aim of palliative care is to improve the quality of life for patients with an incurable disease. There is substantial evidence for considerable unmet palliative needs of patients with CHF and their carers^{110,111}. The main areas of need include symptom control, psychological and social support, planning for the future and end of life care. Issues of sudden death and living with uncertainty are pertinent to all patients with CHF¹¹². The opportunity to discuss these issues should be available at all stages of care. It is recommended that the palliative care needs of patients and carers should be identified and assessed and managed at the earliest opportunity. Patients with CHF and their carers should have access to professionals with palliative care skills within the heart failure team¹¹³. Although it is not known when, how and by whom supportive and palliative care is best provided, heart failure nurses are ideally placed to co-ordinate these services and should be accessible to patients and their carers.

14. MONITORING OF QUALITY AND OUTCOME INDICATORS

To continuously improve the quality of CHF care in Northern Ireland, local multidisciplinary teams that provide CHF care should monitor a small, core set of quality indicators. Each indicator should be clearly defined to ensure comparability over time. The indicators should include process measures that reflect these guidelines. For example, in the hospital setting, the percentage of patients who undergo assessment of left ventricular ejection fraction, percentage on ACE inhibitors at discharge, percentage of patients given lifestyle instructions and management plans prior to discharge. The CHF teams should set targets for improvement in quality indicator rates and should develop and implement ideas to improve performance against the quality indicators.

The impact of investment in additional staff to manage CHF in primary or secondary care should be evaluated against the core quality indicators. Additional investment should be seen to lead to improved quality.

In secondary care a sizeable burden on health services arise through the cost associated with hospitalisation of patients with CHF. Analysis should extend beyond the practice of focussing on the first admission as it discards aspects of hospitalisation that inform about disease burden, multiple admissions and length of stay¹⁰⁹.

In primary care the new GMS contract for general practitioners includes a left ventricular dysfunction subset of CHD as one of the ten disease areas in the clinical domain. Practices that have a register of patients with CHD and left ventricular dysfunction and can demonstrate that the diagnosis is confirmed by ECG (target 90%) and that an ACE inhibitor or angiotensin receptor antagonist is prescribed (target 70%) will qualify for additional funding. However, providing optimal care for patients with CHF goes far beyond the basic quality indicators assessed in the GMS contract.

15 CONCLUSION

Guidelines achieve nothing if they sit on the shelf. There is considerable good will for improving standards of care for patients with a diagnosis of CHF. There is a broad consensus nationally for what needs to be done and implementation of these guidelines should improve uptake of appropriate diagnostic procedures and treatments for CHF. Importantly any strategy must include a service model of delivery that will improve co-ordination and sharing of information between health care professionals to ensure equity and optimise care for all patients with CHF in Northern Ireland.

FREQUENTLY ASKED QUESTIONS

1. How should a trial fibrillation be managed in patients with CHF?

All such patients should be anticoagulated with warfarin unless contraindicated. Rate control should be instituted with digoxin and beta blockers. DC cardioversion to sinus rhythm should be attempted in selected patients and in some, may lead to an improvement in systolic function.

2. How should angina be treated in patients with CHF?

Antianginal drug treatment should be instituted with beta-blockers and if necessary nitrates. Rate-limiting calcium antagonists should be avoided. Revascularisation should be considered. Special investigations to assess myocardial ischaemia, viability and hibernation should be considered if revascularisation is an option. Many authorities recommend that hibernating myocardium should be sought in all patients with CHF and coronary artery disease to see if they would be suitable candidates for revascularisation.

3. Should I perform further risk stratification for arrhythmias?

In patients with impaired systolic function after myocardial infarction, arrhythmic risk stratification should be considered. Further investigation and specialist referral should be considered in patients without severe symptoms (probably NYHA Class I and II). The situation in patients with CHF due to causes other than coronary artery disease is uncertain.

4. How should I approach the management of patients with progressive CHF who are not responding to standard treatment?

Drug therapy should be optimised and causes for any underlying reversible cause or aggravating factor sought and treated. In patients who are suitable for further intervention, specialist advice should be sought about biventricular pacing, cardiac transplantation or other strategies. In patients unsuitable for further intervention, involvement of a palliative care team may be appropriate.

5. The new GP contract states I should refer patients with suspected heart failure for an ECHO. Why should I request an ECG and BNP instead?

Cost to the HPSS of an ECG and BNP blood test are about 10 x less than ECHO and the results can be just as accurate. It is likely that future versions of the GP contract will be amended to reflect this.

6. Is there any difference between the ACE inhibitors?

They all act in the same way and there are no major differences between the eight currently licensed for use in heart failure. However, captopril has a higher side-effect profile than the newer drugs, especially at higher doses and so tends to be less often used; it also needs to be taken three times a day.

7. Is there any difference between ACE inhibitors and angiotensin II antagonists?

Both groups of drugs act on the same pathway to produce a similar end result, but ACEIs also reduce the breakdown of bradykinin. Whilst this might contribute to cough, it also could enhance the vasodilator effect of ACEIs. A head-to-head comparison of captopril and losartan did not show any difference in terms of efficacy, although losartan caused fewer side-effects.

8. Is there any difference between the beta-blockers?

There are pharmacological differences - carvedilol has a significant component of α -blockade in its action. The significance of this is uncertain, but a recent comparison of carvedilol and long-acting metoprolol indicated that the former was superior in terms of event reduction.

9. What about impaired renal function?

Impaired renal function is common, particularly in severe heart failure, due to a combination of poor renal perfusion and treatment with ACEIs and diuretics. In addition, patients may have atherosclerotic renal artery disease, or concomitant diabetes. In general, a raised creatinine is an indication for ACE inhibition, not a contra-indication. A moderate rise in creatinine on treatment is normal, but if a marked rise occurs, the advice of a renal physician should be sought.

10. Who should I refer for a specialist cardiology opinion?

- Angina, for consideration of revascularisation.
- Atrial dysrhythmias, particularly of recent onset, for consideration of cardioversion.
- Ventricular dysrhythmias, for which pharmacological treatment will usually be amiodarone. Those at greatest risk (poorly tolerated dysrhythmias and/or severe LVSD) should be considered for an AICD.
- Significant valvular disease.

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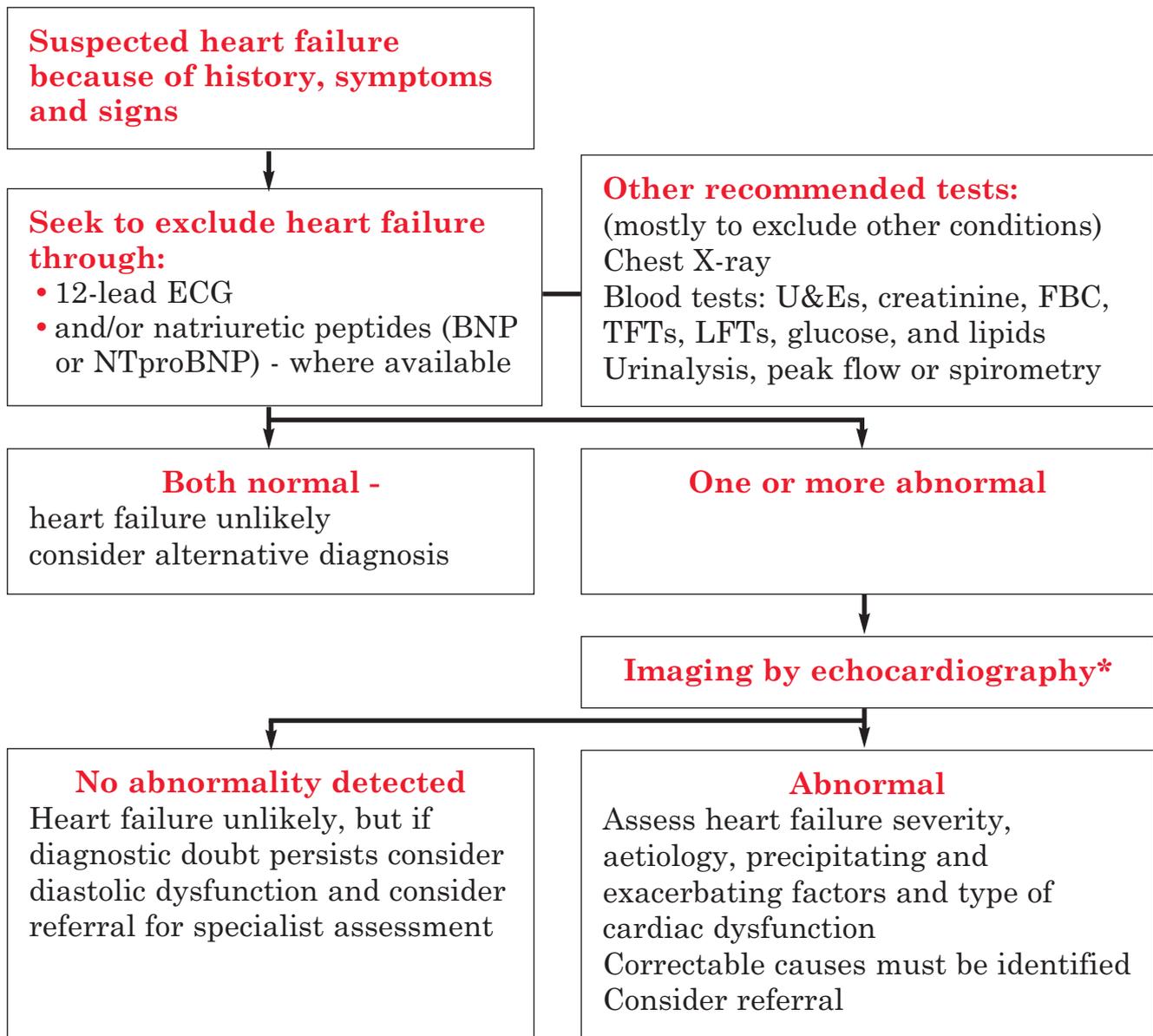
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APPENDIX 1

DIAGNOSTIC ALGORITHM FOR THE DIAGNOSIS OF CHRONIC HEART FAILURE



***Alternative methods of imaging the heart should be considered when a poor image is produced by transthoracic Doppler 2D echocardiography - alternatives include transoesophageal Doppler 2D echocardiography, radionuclide imaging or cardiac magnetic resonance imaging**

Key: ECG: electrocardiogram, BNP: B-type natriuretic peptide, NTproBNP: N-terminal proB-type natriuretic peptide, U&Es: urea and electrolytes, FBC: full blood count, TFTs: thyroid function tests, LFTs: liver function tests

TABLE 1

Drugs used to attain objectives of therapy in chronic heart failure due to LV systolic dysfunction

(NB. Drugs used for improving symptoms and exercise capacity can be adjusted according to patient responses, but drugs used with the aim of improving longevity or reducing hospitalisation rates should not usually be adjusted on this basis).

Condition	NYHA Class	Drugs for improving:			
		Longevity	Quality of life		
			Symptoms	Exercise capacity	Hospitalisation rates
Mild HF	II	ACEI, β B, ISDN+Hyd	ACEI, AIIA, Digx, Diur, Vasodil	Diur, Digx, ISDN+Hyd	ACEI, β B, Digx, Diur (AIIA if not on ACEI)
Moderate HF	III	ACEI, β B, AI α A, ISDN+Hyd	ACEI, AIIA, Digx, Diur, Vasodil	Diur, Digx, ISDN+Hyd	ACEI, β B, Digx, Diur (AIIA if not on ACEI)
Severe HF	III+IV	ACEI, β B, AI α A, ISDN+Hyd	ACEI, AI α A, AIIA, Digx, Diur, Vasodil	Diur, Digx, ISDN+Hyd	ACEI, AI α A, Diur (β B, Digx, (AIIA if not on ACEI)
End stage HF	IV		Digx, Diur, Pos-Inotr, Vasodil	Digx, Diur, Vasodil	Diur

Legend:

ACEI: Angiotensin Converting Enzyme Inhibitors

AIIA: Angiotensin II Receptor Antagonists

AI α A: Aldosterone Antagonists (only spironolactone hitherto)

β B: beta blockers (bisoprolol, carvedilol, metoprolol)

Digx: Digoxin

Diur: Diuretics

ISDN+Hyd: Combination of oral isosorbide dinitrate and hydralazine

LVSD: Left ventricular systolic dysfunction

NYHA: New York Heart Association Functional Class for Heart Failure

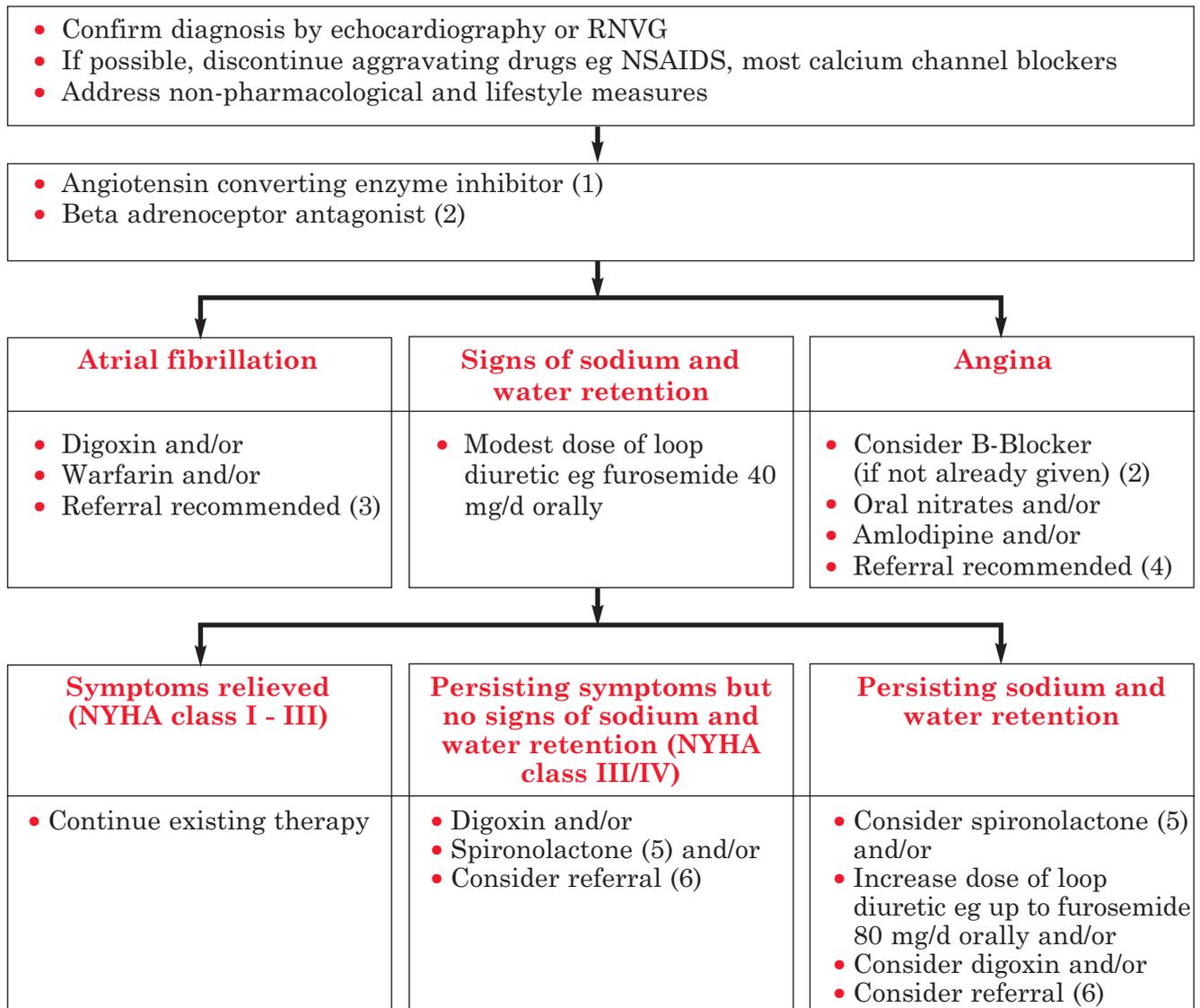
Pos-Inotr: Positive inotropes (e.g. iv dobutamine, enoximone, milrinone)

Vasodil: Vasodilators (e.g. nitrates, calcium antagonists, K Channel openers)

APPENDIX 2

TREATMENT ALGORITHM

Treatment of left ventricular systolic dysfunction



1. See use of ACE inhibitor algorithm. If not tolerated, consider angiotensin II receptor antagonist/II-ISDN combination therapy/digoxin.
2. Indicated in NYHA Class I-III heart failure. Extreme caution required in initiating β -blockers; must be done under specialist care.
3. Electrical cardioversion may be indicated; other specialist drugs such as amiodarone may be indicated.
4. Referral to a specialist with an interest in heart failure; coronary angiography and bypass surgery may be indicated.
5. Indicated in NYHA Class III-IV heart failure. Dose 25mg once daily; extreme care must be taken to avoid hyperkalaemia and renal failure, ie monitor electrolytes carefully.
6. Other specialist therapy may be indicated as an outpatient or inpatient.

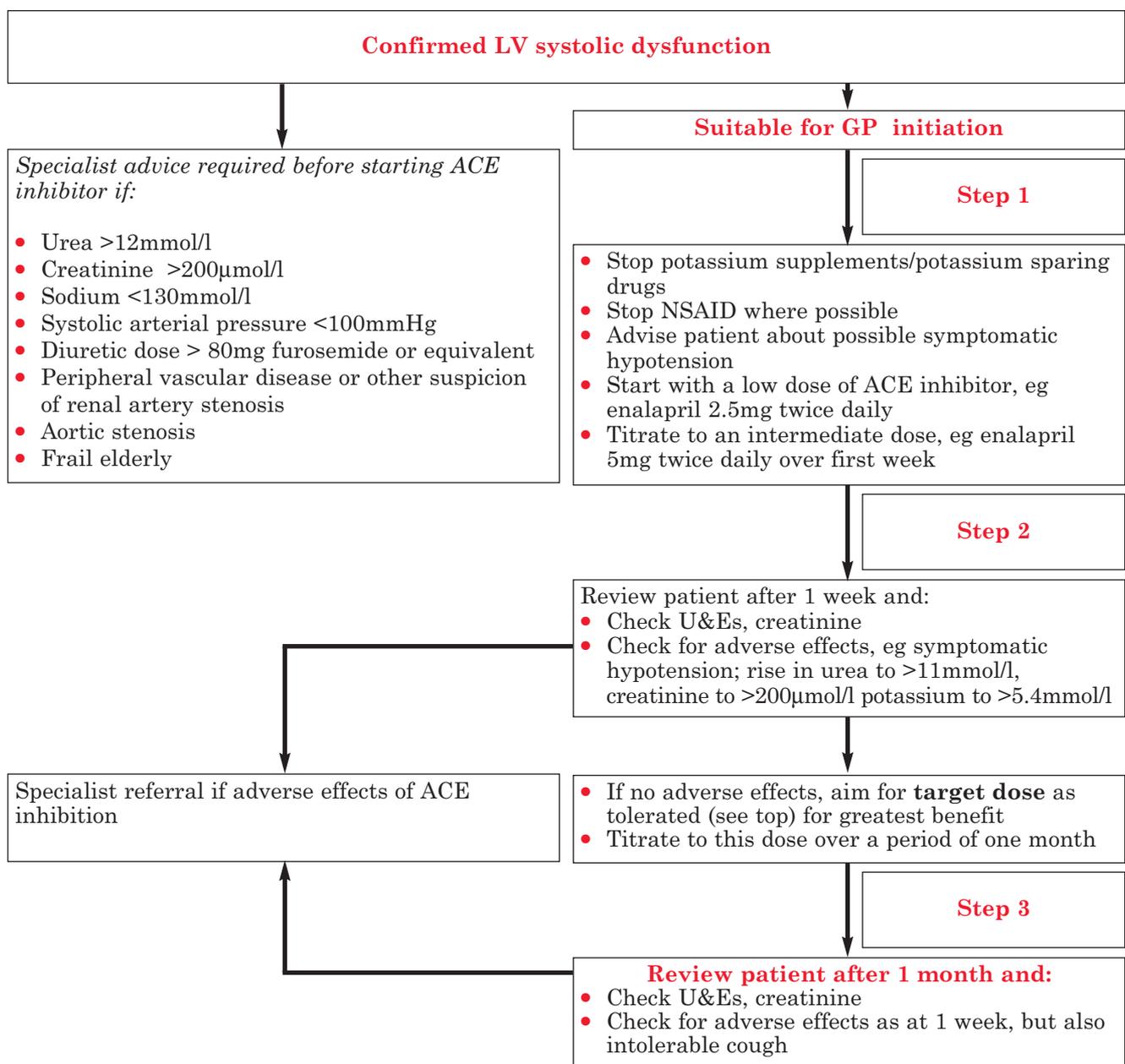
APPENDIX 3

ALGORITHM FOR THE USE OF AN ACE INHIBITOR

NB Target dose. It is important to optimise the dose of ACE inhibitor when seeking prognostic benefit, though caution should be exercised in the very elderly. Target doses are as follows:

- Captopril 50mg tds.
- Enalapril 10-20mg bd.
- Ramipril 5mg bd.
- Lisinopril 30-35mg od.

For those intolerant of ACE inhibitors, a similar approach should be used when using **angiotensin II receptor antagonists**, particularly with regard to monitoring renal function; the same adverse events, with the exception of cough, should be anticipated.



PRACTICAL RECOMMENDATIONS ON THE USE OF ACE INHIBITORS

Which ACE inhibitor and what dose?

<u>Licensed ACEI</u>	<u>Starting dose (mg)</u>	<u>Target dose (mg)</u>
Captopril	6.25 three times daily	50-100 three times daily
Cilazapril*	0.5 once daily	1-2.5 once daily
Enalapril	2.5 twice daily	10-20 twice daily
Fosinopril*	10 once daily	40 once daily
Lisinopril	2.5-5.0 once daily	30-35 once daily
Perindopril*	2.0 once daily	4 once daily
Quinapril*	2.5-5.0 once daily	10-20 once daily
Ramipril	2.5 once daily	5 twice daily or 10 once daily

*Target dose based on manufacturer's recommendation rather than large outcome study

How to use?

- Start with a low dose (see above)
- Double dose at not less than 2 weekly intervals
- Aim for target dose (see above) or, failing that, the highest tolerated dose
- Remember some ACE inhibitor is better than no ACE inhibitor
- Monitor blood chemistry urea, creatinine, K⁺ and blood pressure
- When to stop up-titration/down-titration; see PROBLEM SOLVING

Advice to patient?

- Explain expected benefits
- Treatment is given to improve symptoms, to prevent worsening of heart failure and to increase survival
- Symptoms improve within a few weeks to a few months
- Advise patients to report principal adverse effects ie dizziness/symptomatic hypotension, cough

Problem solving

- Asymptomatic low blood pressure does not usually require any change in therapy

Symptomatic hypotension

- If dizziness, light-headedness and/or confusion and a low blood pressure, reconsider need for nitrates, calcium channel blockers* and other vasodilators

- If no signs/symptoms of congestion consider reducing diuretic dose
- If these measures do not solve problem, seek specialist advice
- * Calcium channel blockers should be discontinued unless absolutely essential e.g. for angina or hypertension.

Cough

- Cough is common in patients with chronic heart failure, many of whom have smoking-related lung disease
- Cough is also a symptom of pulmonary oedema which should be excluded when a new or worsening cough develops
- ACE inhibitor induced cough rarely requires treatment discontinuation
- When a very troublesome cough does develop, one stopping the patient sleeping and can be proven to be due to ACE inhibition, (i.e. recurs after ACE inhibition withdrawal and rechallenge) substitution of an angiotensin II receptor antagonist can be considered

Worsening renal function

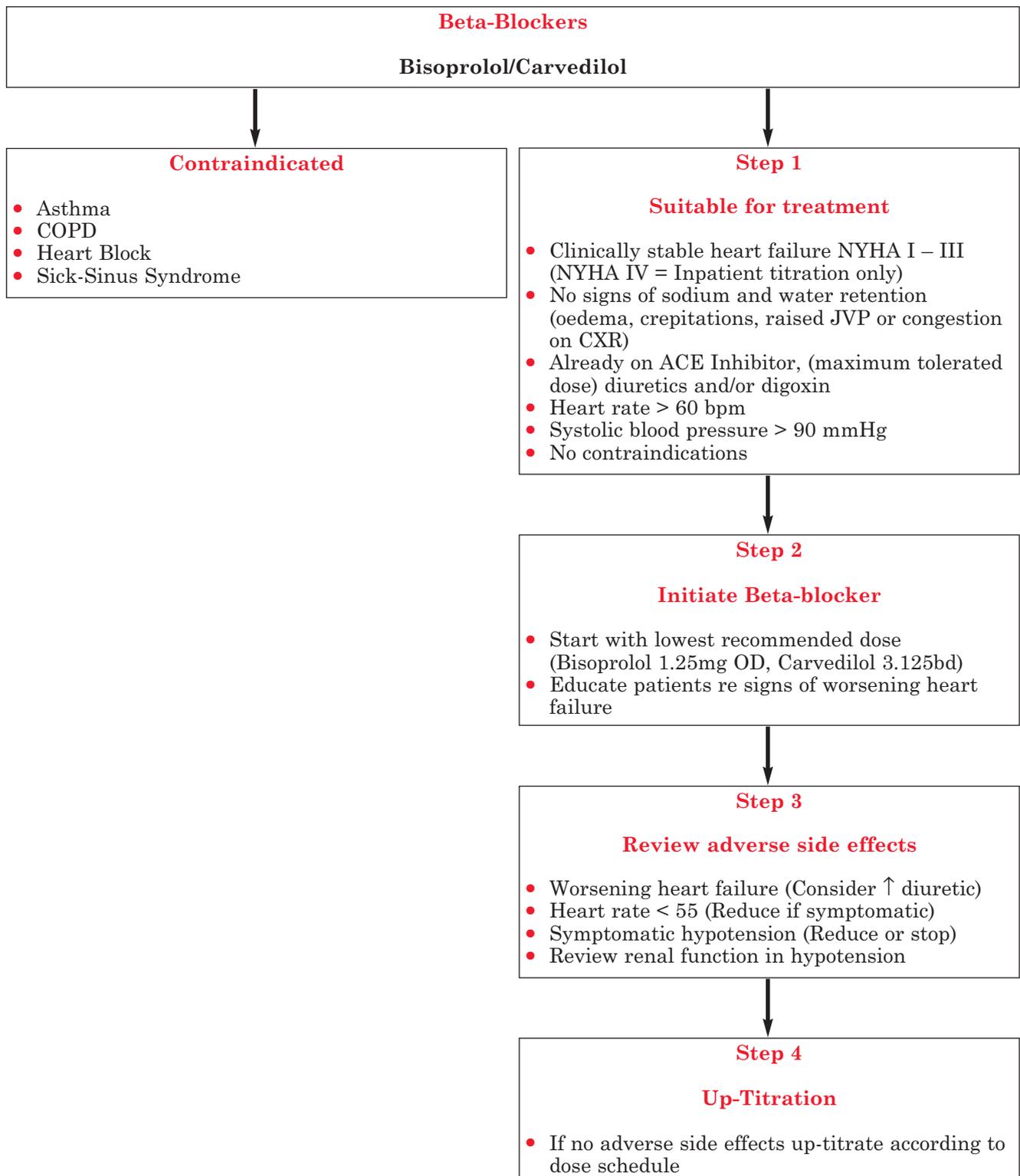
- Some rise in urea, creatinine and K^+ is to be expected after initiation of an ACE inhibitor; if the increase is small and asymptomatic no action is necessary
- An increase in creatinine of up to 50% above baseline, or to 250 $\mu\text{mol/l}$, which ever is the smaller, is acceptable
- An increase in K^+ to <6.0 mmol/l is acceptable
- If urea, creatinine or K^+ do rise excessively consider stopping concomitant nephrotoxic drugs (e.g. NSAIDs), non-essential vasodilators (e.g. calcium antagonists, nitrates), K^+ supplements/retaining agents (triamterene, amiloride) and if no signs of congestion, reducing the dose of diuretic
- If greater rises in creatinine or K^+ than those outlined above persist despite adjustment of concomitant medications the dose of the ACE inhibitor should be halved and blood chemistry rechecked, if there is still an unsatisfactory response specialist advice should be sought
- If K^+ rises to > 6.0 mmol/l or creatinine increases by $>100\%$ or to above 350 $\mu\text{mol/l}$ the dose of ACE inhibitor should be stopped and specialist advice sought
- Blood chemistry should be monitored serially until K^+ and creatinine have plateaued

Note: It is very rarely necessary to stop an ACE inhibitor and clinical deterioration is likely if treatment is withdrawn; ideally, specialist advice should be sought before treatment discontinuation.

Adapted from McMurray et al. (2001) Practical recommendations for the use of ACE inhibitors, beta-blockers and spironolactone in heart failure: putting guidelines into practice. European Journal of Heart Failure 3: 495-502.

APPENDIX 4

ALGORITHM FOR THE USE OF BETA-BLOCKERS IN CHRONIC HEART FAILURE



PRACTICAL RECOMMENDATIONS ON THE USE OF BETA-BLOCKERS

Which beta-blocker and what dose?

Only two beta-blockers are licensed for the treatment of heart failure in the UK.

	<u>Starting dose (mg)</u>	<u>Target dose (mg)</u>
Bisoprolol	1.25 once daily	10 once daily
Carvedilol	3.125 twice daily	25-50 twice daily

How to use?

- Start with a low dose (see above)
- Double dose at not less than 2 weekly intervals
- Aim for target dose (see above) or, failing that, the highest tolerated dose
- Remember some beta-blocker is better than no beta-blocker
- Monitor heart rate, blood pressure, clinical status (symptoms, signs, especially signs of congestion, body weight)
- Check blood chemistry 1-2 weeks after initiation and 1-2 weeks after final dose titration
- When to down-titrate/stop up-titration; see PROBLEM SOLVING

Advice to patient?

- Explain expected benefits
- Emphasise that treatment is given as much to prevent worsening of heart failure as to improve symptoms, beta-blockers also increase survival
- If symptomatic improvement occurs, this may develop slowly over 3-6 months or longer
- Temporary symptomatic deterioration may occur (estimated 20-30% of cases) during initiation/up-titration phase
- Advise patient to report deterioration (see PROBLEM SOLVING) and that deterioration (tiredness, fatigue, breathlessness) can usually be easily managed by adjustment of other medication; patients should be advised not to stop beta-blocker therapy without consulting their physician
- Patients should be encouraged to weigh themselves daily (after waking, before dressing, after voiding, before eating) and to increase their diuretic dose should their weight increase, persistently (>2 days), by >1.5-2.0 kg

Problem solving

Worsening symptoms/signs (e.g. increasing dyspnoea, fatigue, oedema, weight gain)

- If increasing congestion, double dose of diuretic and/or halve dose of beta-blocker (if increasing diuretic does not work)
- If marked fatigue, (and/or bradycardia, see below) halve dose of betablocker (rarely necessary)
- Review patient in 1-2 weeks; if not improved seek specialist advice
- If serious deterioration, halve dose of beta-blocker or stop this treatment (rarely necessary); seek specialist advice

Low heart rate

- If <50 beats/min and worsening symptoms, halve dose beta-blocker or, if severe deterioration, stop beta-blocker (rarely necessary)
- Review need for other heart rate slowing drugs (e.g. digoxin, amiodarone, diltiazem)
- Arrange ECG to exclude heart block
- Seek specialist advice

Asymptomatic low blood pressure

- Does not usually require any change in therapy

Symptomatic hypotension

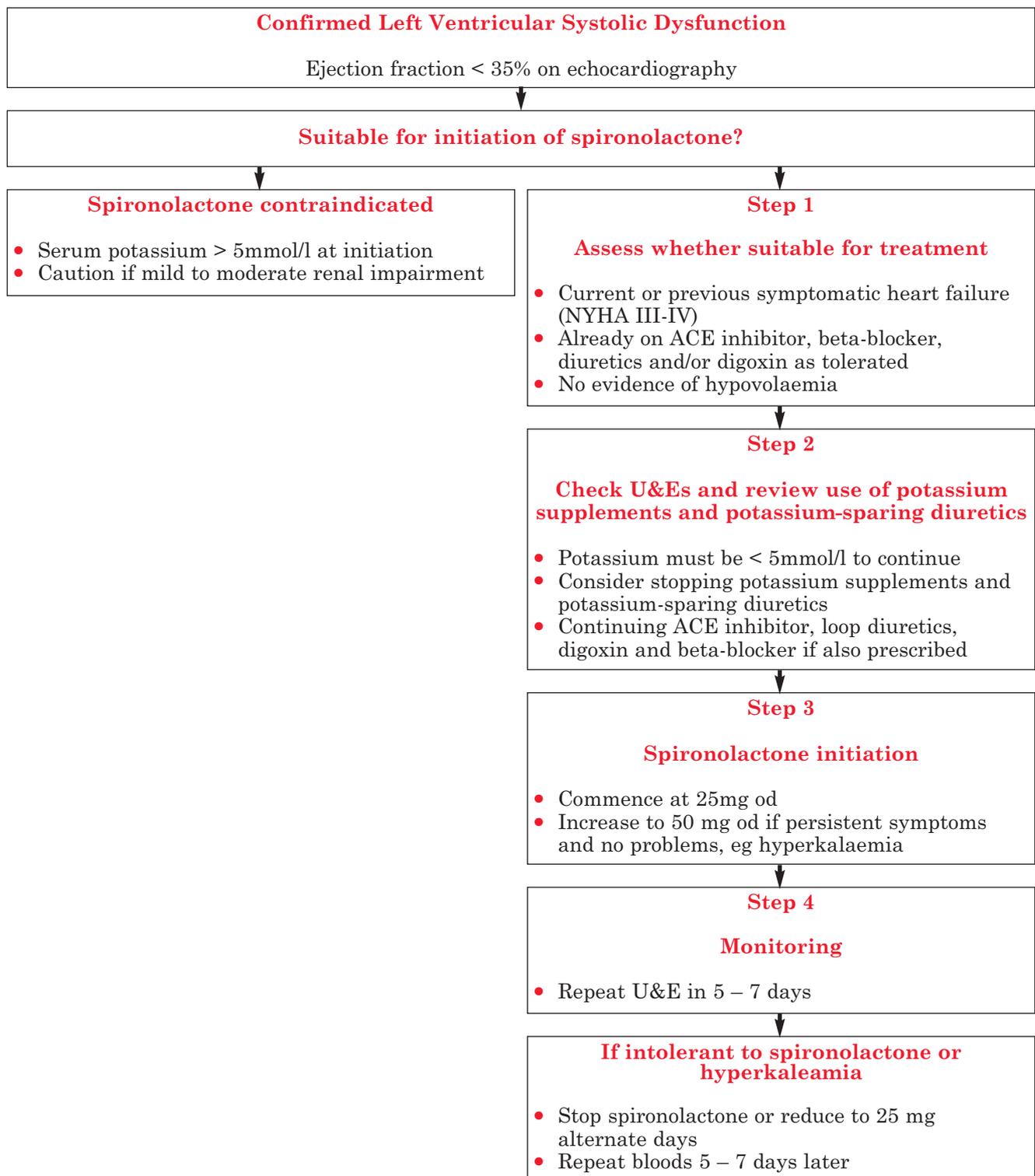
- If dizziness, light-headedness and/or confusion and a low blood pressure, reconsider need for nitrates, calcium channel blockers and other vasodilators
- If no signs/symptoms of congestion consider reducing diuretic dose
- If these measures do not solve problem seek specialist advice

Note: Beta-blockers should not be stopped suddenly unless absolutely necessary (there is a risk of a 'rebound' increase in myocardial ischaemia/infarction and arrhythmias); ideally specialist advice should be sought before treatment discontinuation

Adapted from McMurray et al: (2001) Practical recommendations for the use of ACE inhibitors, beta-blockers and spironolactone in heart failure: putting guidelines into practice. European Journal of Heart Failure 3: 495-502.

APPENDIX 5 (comment: 25mg od is quite a high starting dose for the elderly, consider 12.5mg od. MEAN dosage in the RALES study was 27mg od)

ALGORITHM FOR THE USE OF SPIRONOLACTONE IN HEART FAILURE



PRACTICAL RECOMMENDATIONS ON THE USE OF SPIRONOLACTONE

Which dose of spironolactone?	
Starting dose (mg)	Target dose (mg)
25 once daily or on alternate days	25-50 once daily
<p>How to use?</p> <ul style="list-style-type: none"> • Start at 25 mg once daily • Check blood chemistry at 1, 4, 8 and 12 weeks; 6, 9 and 12 months, 6 monthly thereafter • If K⁺ rises to between 5.5 and 6.0 mmol/l or creatinine rises to 220 µmol/l reduce dose to 25 mg on alternate days and monitor blood chemistry closely • If K⁺ rises to >6.0 mmol/l or creatinine to >350 µmol/l stop spironolactone and seek specialist advice 	
<p>Advice to patient?</p> <ul style="list-style-type: none"> • Explain expected benefits • Treatment is given to improve symptoms, prevent worsening of heart failure and to increase survival • Symptom improvement occurs within a few weeks to a few months of starting treatment • Avoid NSAIDs not prescribed by a physician (self-purchased 'over the counter' treatment e.g. ibuprofen) • Temporarily stop spironolactone if diarrhoea and/or vomiting and contact physician 	
<p>Problem solving</p> <p>Worsening renal function/hyperkalaemia:</p> <ul style="list-style-type: none"> • See HOW TO USE? Section • Major concern is hyperkalaemia (>6.0 mmol/l though this was uncommon in the RALES clinical trial; a high normal potassium may be desirable in patients with heart failure, particularly if taking digoxin) • It is important to avoid other K⁺ retaining drugs -for example, K⁺ sparing diuretics and nephrotoxic agents (e.g. NSAIDs) • Some 'low salt' substitutes have a high K⁺ content • Male patients may develop breast discomfort and/or gynaecomastia 	

Adapted from McMurray et al: (2001) Practical recommendations for the use of ACE inhibitors, beta-blockers and spironolactone in heart failure: putting guidelines into practice. European Journal of Heart Failure 3: 495-502.

APPENDIX 6



***WOLVERHAMPTON
HEART FAILURE
SERVICE***

WORKING TOGETHER TO



MAKE A DIFFERENCE



Wolverhampton Heart Failure Service

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 The full Wolverhampton Heart Failure Service Model can be accessed at: www.doh.gov.uk/heart/heartfailure/resource	



Wolverhampton Heart Failure Service

Heart failure is a common chronic debilitating disease with an extremely poor prognosis (worse than that of most cancers). It has an enormous impact on health services accounting for 2 per cent of total healthcare expenditure, two thirds of which are hospital costs. The total annual cost of heart failure in the UK is around £600m.

The incidence of heart failure is estimated at 1-5 per 1,000 population. Prevalence is estimated at 3-20 per 1,000 population (increasing prevalence is due to the ageing population and survival post Myocardial Infarction)

Heart Failure has posed the biggest challenge yet by the National Service Framework (NSF) for Coronary Heart Disease. It has been recognised that the management of heart failure is often sub-optimal, poorly diagnosed and under treated.

Wolverhampton's Local Implementation Team identified heart failure as a priority to be addressed. There was overwhelming evidence to support specialist nurse intervention and also research to suggest that half of all hospital admissions were preventable. The multidisciplinary Heart Failure Service was subsequently commenced in January 2002. The service gained national recognition in July 2002 after winning a Health and Social Care award.

Heart Failure Team

Consultant Cardiologist
Heart Failure Specialist Nurses (3 WTE)
Heart Failure Secretary

Contact Details

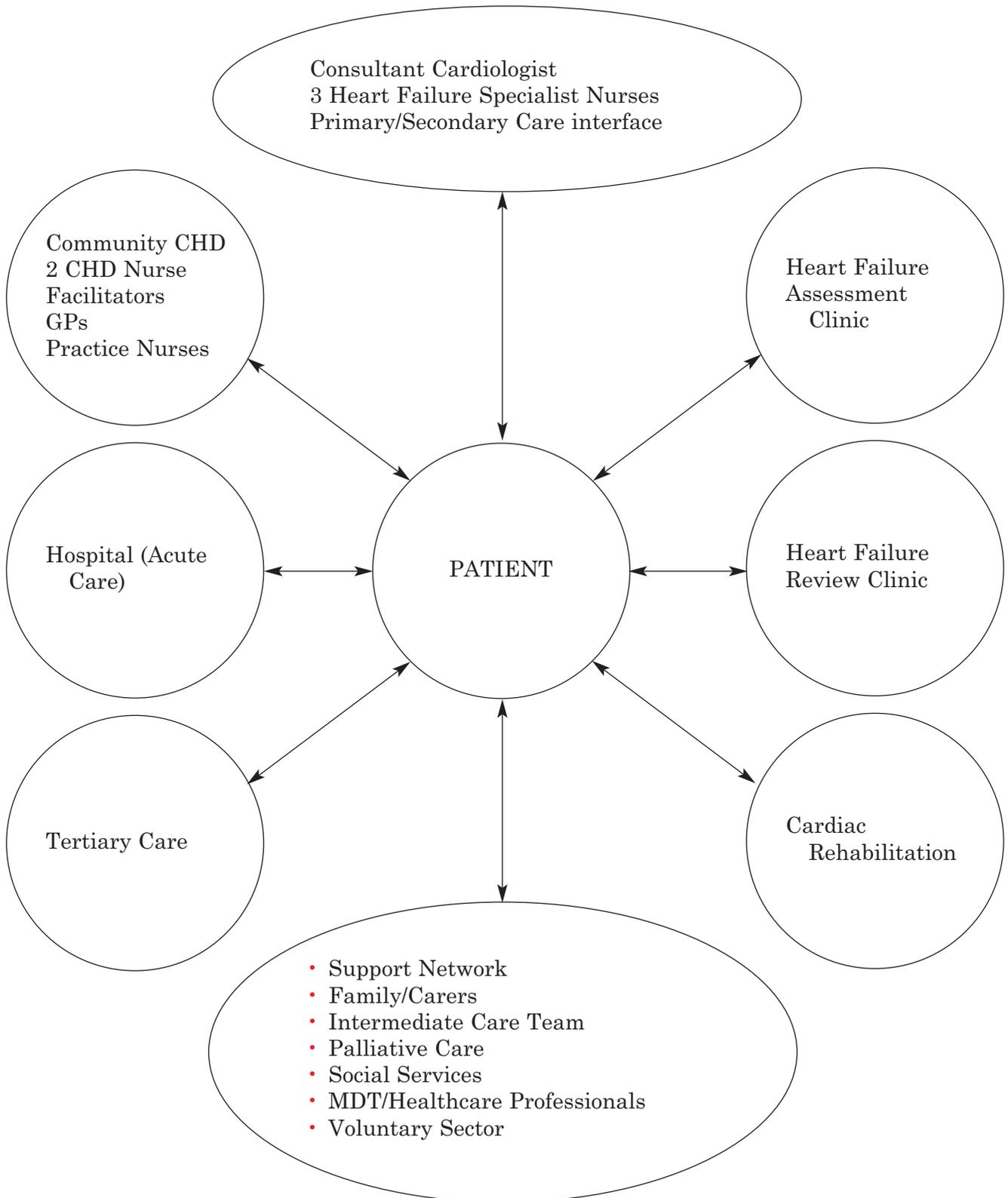
Heart Failure Team

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Tel/Fax 01902 643069

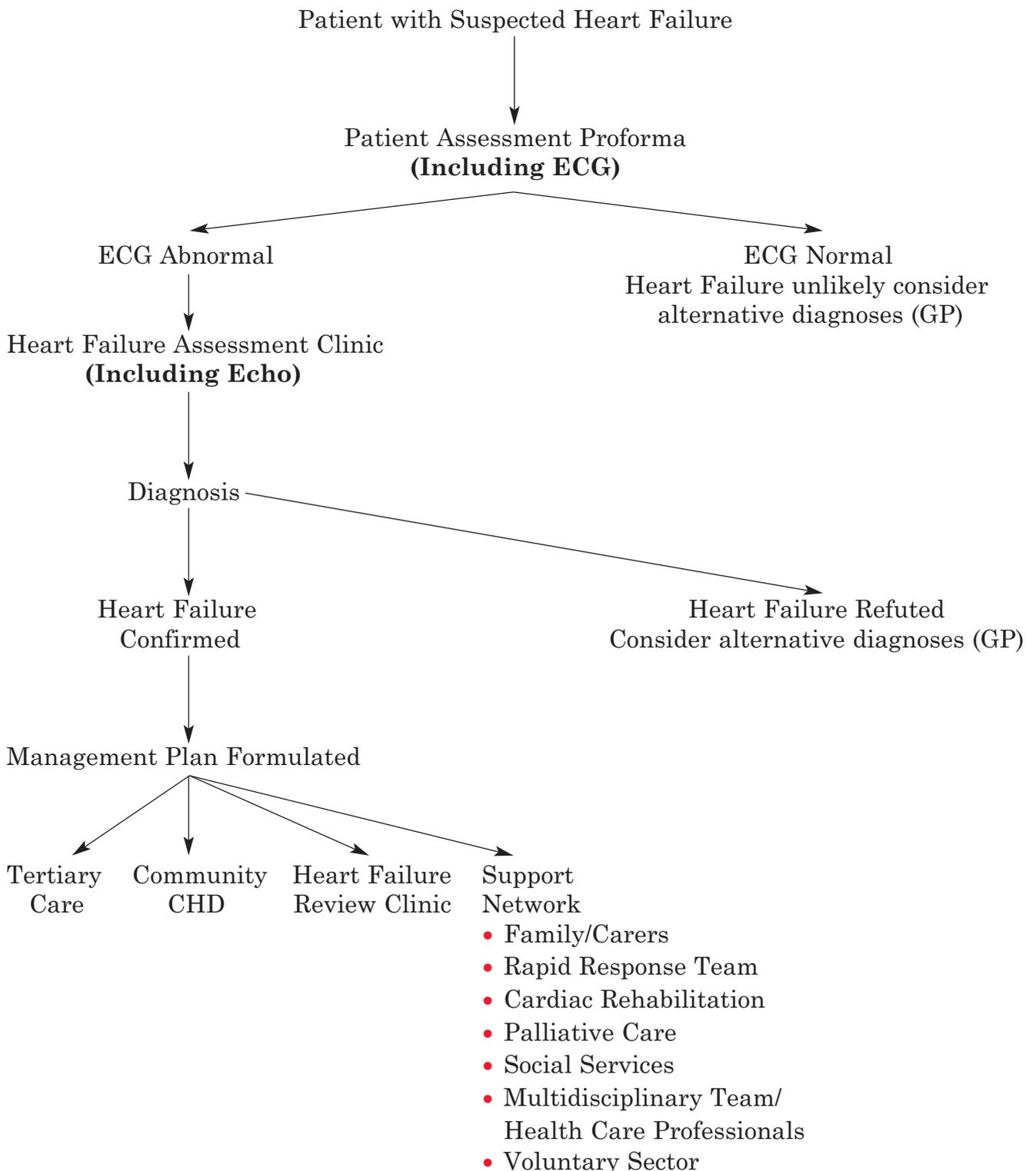
WOLVERHAMPTON HEART FAILURE SERVICE

SERVICE MODEL



WOLVERHAMPTON HEART FAILURE SERVICE

PATIENT FLOW



Wolverhampton Heart Failure Service

SERVICE PROVISION

Service Model

- Patient centred
- Works across the primary/secondary care interface (multi/interdisciplinary)
- Based on evidence as well as regional and national best practice
- Formulated to assist in achieving NSF standards

Service Aims

- Improve access to diagnostic testing to confirm or refute the diagnosis of heart failure
- Identify aetiology
- Provide evidence based treatment
- Improve quality of life by improving symptoms or slowing their deterioration
- Reduce length of hospital stay
- Reduce frequent re-admissions to hospital
- Reduce mortality
- Improve the end of life experience for patients and carers

Heart Failure Assessment Clinic

- GP referrals of new patients with suspected LVSD
- Access via faxed proforma
- Diagnosis of heart failure confirmed or refuted
- Management plan formulated which includes:
Home visit for education with ongoing contact and support
Further investigations to establish aetiology
Evidence based treatment (pharmacological and non-pharmacological)
Referral to multidisciplinary agencies as appropriate

Heart Failure Review Clinic

- Optimisation of evidence based medication
- Initiation and titration of Beta-Blockers
- Urgent symptom review and monitoring

Inpatient Service

- Multidisciplinary referral via telephone or fax accepted
- Patients must have a confirmed diagnosis of heart failure by their consultant documented in the hospital notes
- Education provided prior to discharge home to increase patient involvement and understanding, leading to empowerment and improvement in compliance
- Co-ordinating safe discharge home with appropriate multidisciplinary input
- Provision of ongoing support and follow up from the heart failure team

Telephone Contact/Advice Line

- Patients telephoned at 1, 3, 6 and 12 months
- Advice line operates from 9am to 5pm Monday to Friday with an answering machine out of hours. For urgent queries patients/carers are encouraged to contact their GP or emergency services as appropriate

Community Review

To maximise equity and service provision the aim since setting up the service has been for GPs and practice nurses to be equipped with the appropriate knowledge and skills to optimise patient's treatment and management within the practice setting, with support from the heart failure service.

To facilitate this goal the heart failure specialist nurses provide a rolling education programme for practice nurses.

Workshop 1

Recording an Accurate ECG
The Normal ECG
(2 hrs duration)

Workshop 2

Systematic Analysis of Rhythm Recognition and ECG Interpretation
(3 hrs duration)

Workshop 3

Pathophysiology of Heart Failure
Non Pharmacological Management of Heart Failure
Pharmacological Management of Heart Failure
(3 hrs duration)

Workshop 4

Systematic Review of Heart Failure in the Practice Setting
Case Histories
(2 hrs duration)

Practice Nurses also have the opportunity to shadow Specialist Nurses' clinical practice and are encouraged to do the British Heart Foundation 'Heart Save Course'.

Effectiveness

- The effectiveness of the service is audited regularly to meet the requirements of the NSF and evaluate service provision
- Comprehensive patient database is kept
- Patient satisfaction surveys are used to ensure consumer view

Clinical Guidelines

MANAGEMENT OF PATIENTS WITH CHRONIC HEART FAILURE DUE TO LEFT VENTRICULAR SYSTOLIC DYSFUNCTION

This document sets out guidelines to address the aims of the National Service Framework for Coronary Heart Disease with respect to heart failure, with a service model agreed across primary and secondary care.

Users should be aware that the commonest cause of chronic heart failure is left ventricular systolic dysfunction; this document does not cover the treatment of diastolic dysfunction, for which the evidence base is less robust.

Parts of this document have been adapted for local use from the SIGN guideline: “Diagnosis and Treatment of Heart Failure due to Left Ventricular Systolic Dysfunction” February 1999 (www.sign.ac.uk).

National Service Framework – Coronary Heart Disease Standard Eleven

“Doctors should arrange for people with suspected heart failure to be offered appropriate investigations (e.g. electrocardiography, echocardiography) that will confirm or refute the diagnosis. For those in whom heart failure is confirmed, its cause should be identified and the treatments most likely to both relieve symptoms and reduce their risk of death should be offered.”

Aims:

- To help people with heart failure live longer and achieve a better quality of life
- To help people with unresponsive heart failure (and other malignant presentations of coronary heart disease) receive appropriate palliative care

Abbreviations

ACE	Angiotensin converting enzyme
AICD	Automatic implantable cardioverter defibrillator
CHD	Coronary heart disease
CHF	Chronic heart failure
COPD	Chronic obstructive pulmonary disease
ECG	Electrocardiogram
LVSD	Left ventricular systolic dysfunction
NSAID	Non-steroidal anti-inflammatory drug
NYHA	New York Heart Association



WOLVERHAMPTON HEART FAILURE SERVICE

Heart Failure Team
New Cross Hospital Old Ward 11
Wolverhampton
WV10 0QP
Tel/Fax No 01902 643069

HEART FAILURE ASSESSMENT CLINIC

The purpose of the clinic is to:-

- Provide access to diagnostic testing to confirm or refute the diagnosis of heart failure
- Where heart failure is confirmed, to provide a management plan for the patient's continuing care, including:
 - Planning further investigations
 - Education for patients and carers
 - Optimising medications
 - Multidisciplinary support and follow up

Suitable referrals to this clinic are:-

New patients with suspected heart failure

The emphasis is on left ventricular systolic dysfunction (LVSD) where key drugs are of proven prognostic benefit – (see Wolverhampton Heart Failure Guidelines).

- **Please note, if the patient has a normal ECG, LVSD is extremely unlikely and referral to this clinic is inappropriate.**

To access this clinic:

Please complete the assessment proforma and send or fax to:-

Wolverhampton Heart Failure Service
Old Ward 11
New Cross Hospital
Wolverhampton
WV10 0QP
Tel/Fax: 01902 643069

(A copy of the 12-lead ECG must be attached)

You will be kept informed regarding your patients at all times.



WOLVERHAMPTON HEART FAILURE SERVICE

ASSESSMENT PROFORMA

(Please tick relevant boxes)

PATIENT DETAILS

Hospital No:

Name:

Sex: M F

Address:

DOB:

Ethnic Group

Interpreter req'd: Yes/No
If Yes – which language?

Tel No:

GP DETAILS

Name:

Address:

Tel No:

PATIENT'S PAST MEDICAL HISTORY

IHD:

Hypertension:

Rheumatic Fever:

Diabetes:

Other: _____

MEDICATIONS (Please list)

SYMPTOMS

Dyspnoea: Orthopnoea:
 PND: Cough:
 Fatigue/Lethargy: Peripheral Oedema:

Other: _____

EXAMINATION

Pulse: bpm BP: Elevated JVP: Y N
 Heart sounds: Normal Gallop Murmur
 Chest X-Ray: Normal Cardiomegaly Congestion

INVESTIGATIONS

*Abnormal 12-lead ECG: (Please attach copy)

Evidence of previous MI: Atrial Fibrillation:
 Bundle Branch Block: Left Ventricular Hypertrophy:
 Left Axis Deviation:

* Other minor abnormalities are not suggestive of LVSD

BLOODS

U & E

Sodium:
 Potassium:
 Urea:
 Creatinine:

FBC

Hb:
 WCC:
 Platelets:

LFT

Total Bilirubin:
 Alkaline Phosphate:
 ALT:
 Albumin:
 Globulin:

TFT

TSH:

(Referrals will not be accepted without results of blood tests, CXR and ECG)

WOLVERHAMPTON HEART FAILURE SERVICE

PATIENT REGISTRATION

Hospital No:	Address 1:
Title:	Address 2:
First Name:	Area:
Surname:	Town/City:
Registration Date: / /	County:
Consultant:	Post Code:
GP Practice:	Telephone Number:
GP Name:	PCG/PCT:
Hospital Name:	Deceased: Y / N
Gender: M / F	Date Deceased: / /
Date of Birth: / /	

**** Please note: All items in Bold must be completed****

BASIC DETAILS

Consultation Details

Consultation Date: / /	Location of Visit: Outpatient Dept / Home / Ward:-
Type of Visit: Assess Clinic / Review Clinic / In-patient/ Home / Beta-blocker Clinic / Urgent	Reason for Visit: Assessment / Education / Titrate Medication / Urgent Review
Primary Care Visit: Heart Failure Nurse / Rapid Response Team	
Secondary Visit: GP/Consultant	Reason for Urgent Review:

SYMPTOMS

Type of Symptom	Date Appeared	Date Gone	Comments
Shortness Of Breath	/ /	/ /	At Rest / On Mild Exertion / On Moderate Exertion / None
Orthopnoea/PND:	/ /	/ /	
Fatigue:	/ /	/ /	
Dizziness:	/ /	/ /	
Chest Pain:	/ /	/ /	
Palpitations:	/ /	/ /	
Cough:	/ /	/ /	
Other:	/ /	/ /	

LIFESTYLE

Smoking: <20 / <40 / 40+ (Per day)/ Ex / Recently Stopped	Smoking Cessation: Y / N
Alcohol: Y / N / Unknown	Advice on Alcohol: Y / N
Alcohol Consumption: Units per Week	Comments:-
Living Situation: Lives With:-	Advice on Fluid Intake: Y / N
Fluid Restriction: Y / N / Not Req / Unk	Advice on Salt Intake: Y / N
Salt Restriction: Y / N / Not Req / Unk	Advice on Exercise: Y / N
Exercise: Y / N / Not Req / Unk	Advice on Avoiding Infection: Y / N
Flu Vaccination: Y / N / Not Req / Unk	Advice on Weight: Y / N
	Advice on Taking Medication: Y / N

PHYSICAL CONDITION

Blood Pressure Systolic: mmhg	Oedema: Mild / Moderate / Severe
Blood Pressure Diastolic: mmhg	<u>Lower Limb Pulses:</u>
Pulse Rate: beats per min	Not Assessed/ Not Present / Present
Rhythm:	JVP: Elevated / Normal
1st /2nd/3rd Degree/ AF/ VT/ SR/ SVT/Paced	JVP Elevation: cms
Weight:	Wheezes: Y / N / Unknown
Height:	Crackles: Y / N / Unknown
BMI:	Heart Sounds: Normal/ Diastolic/ Systolic/ Pan / S3 / Not assessed
Respiratory Rate:	

Medical History Comments

<u>Please Tick(4)</u>	<u>Other:</u>
↑ BP π	
DM π	
↑ Cholesterol π	
IHD π	
PVD π	
CVA/TIA π	
Family History π	Asthma π Rh. Fever π

Symptoms	None	Slight	Moderate	Severe
NYHA Classification	i	ii	iii	iv

INVESTIGATIONS

Haematology

Haemoglobin:
Platelets:
White Cell Count:
Date: / /

Biochemistry

Sodium:
Potassium:
Urea:
Creatinine:
Date: / /

Glucose

Glucose:	Glucose Type: Fasting/ Random/ Unk	Date: / /
HbA1c:	Date: / /	

Chest X-Ray

CXR:	Y / N / Unknown
Pleural Effusion:	Y / N / Unknown
Cardiothoracic Ration:	%
<u>Pulmonary Congestion:</u>	
Generalised / Lower Lobe Diversion /	
Upper Lobe Diversion / None	

Liver

Serium Bilirubin:
AST:
ALT:
Alkaline Phosphate:
Gamma GT:
Date: / /

INVESTIGATIONS

Lipids

Cholesterol (Fasting):
Cholesterol (Random):
TG:
HDL:
LDL:
Date: / /

Serum Levels

Digoxin Level:
Digoxin Date: / /
NT – Pro BNP Level: pmol/L
NT – Pro BNP Date: / /

ECG

ECG Rate:	Axis
ECG Rhythm: 1st/2nd/3rd degree block/ AF/Atrial Flutter/ SB / SR / ST / SVT / VT/ Paced	
Heart Block:	*Y / N / Unknown
*Bundle Branch Block:	LBBB / RBBB
<u>Atrio-Ventricular Block:</u>	
1° 2° type I / 2° type II / 3°	

Pre-Excitation:	None / WPW Syndrome
LVH:	Y/N/Unknown
Pathological Q Waves:	Y/N/Unknown
LVH:	Y/N/Unknown
ST/T Wave Abnormalities:	Y/N/Unknown
Date: / /	

INVESTIGATIONS

Thyroid

Free T4:	TSH:	Date: / /
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Angiogram

Normal Coronaries:
One / Two / Three Vessel Disease:
Mild / Moderate / Severe LV Dysfunction:

TOE Findings

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ECHOCARDIOGRAM

Mitral Valve:- Abnormal / Normal / Unk	Pulmonary Valve:- Abnormal / Normal / Unk
Mitral Stenosis:- Trivial / Mild / Moderate / Severe	Pulmonary Stenosis:- Trivial / Mild / Moderate / Severe
Mitral Regurgitation:- Trivial / Mild / Moderate / Severe	Pulmonary Regurgitation:- Trivial / Mild / Moderate / Severe
Aortic Valve:- Normal / Abnormal / Calcified / Thickened/ Unknown	LV Systolic Function:- Normal / Impaired
Aortic Stenosis:- Mild / Moderate / Severe	Ejection Fraction: %
Aortic Regurgitation:- Mild / Moderate / Severe	Fractional Shortening: %
Tricuspid Valve:- Abnormal / Normal / Unknown	Qualitative Assessment of LVSD:- Mild / Moderate / Normal / Severe / Unknown
Tricuspid Stenosis:- Trivial / Mild / Moderate / Severe	<u>LV Diastolic Function:</u> Abnormal / Mild / Moderate / Normal / Severe / Unknown
Tricuspid Regurgitation:- Trivial / Mild / Moderate / Severe	

ECHOCARDIOGRAM

Chamber Dimensions

Left Ventricle Diastole: mm	Right Ventricle: Abnormal / Normal / Unk
Left Ventricle Systole: mm	Exercise Test: Negative / Positive / Requested
Left Atrium: mm	Nuclear Imaging: Negative / Positive / Requested / Unknown
Qualitative Assessment of LVH: Y / N / Unknown	Type of HF Syndrome:
Posterior Wall Diastole: mm	Principle Aetiology:
Intraventricular Septum Diastole: mm	Arrhythmias /CHD/Congenital/Hypertension Valvular / Cardiomyopathy / Unknown

Echocardiogram Result:	Normal/Abnormal/Unknown
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MEDICATION
DIURETICS/ACE INHIBITORS

Diuretics

Comments:				Importance Indicator:			
Drug Name	Dosage	Unit	Frequency	Route	Start Date	Stop Date	Reason

Ace Inhibitors

Comments:				Importance Indicator:			
Drug Name	Dosage	Unit	Frequency	Route	Start Date	Stop Date	Reason

MEDICATION
BETA BLOCKERS/SPIRONOLACTONE

Beta Blockers

Comments:				Importance Indicator:			
Drug Name	Dosage	Unit	Frequency	Route	Start Date	Stop Date	Reason

Spiro lactone

Comments:				Importance Indicator:			
Drug Name	Dosage	Unit	Frequency	Route	Start Date	Stop Date	Reason



OTHER CARDIAC DRUGS

Comments:				Importance Indicator:			
Drug Name	Dosage	Unit	Frequency	Route	Start Date	Stop Date	Reason

ADVERSE EFFECTS

Dizziness	Y / N / Unknown	Time Started:	Duration: Mins
Blurred Vision	Y / N / Unknown	Time Started:	Duration: Mins
Fainting	Y / N / Unknown	Time Started:	Duration: Mins
Cough	Y / N / Unknown	Time Started:	Duration: Mins
Allergic Reaction	Y / N / Unknown	Time Started:	Duration: Mins

COMPLICATIONS - (MEDICATION)

Ace Inhibitor Complications:	
Beta Blocker Complications:	
Other Drug Complications:	
Outcome of Clinical Appointment:	
Need Referral:	
Other Discipline:	
Has dual ACE/Diuretic Therapy been optimised:	Y / N



WOLVERHAMPTON HEART FAILURE SERVICE

Patient Label

Heart Failure Team
New Cross Hospital
Old Ward 11
Wolverhampton
WV10 0QP
Tel/Fax No 01902 643069

Date:

Dear Dr

Thank you for referring the above patient to the Heart Failure Assessment Clinic. The results of clinical assessment including echocardiogram do not support the diagnosis of heart failure.

Echocardiogram result:

Comments/suggestions:

If you require any further information please do not hesitate to contact us.

Regards

Yours sincerely

WOLVERHAMPTON HEART FAILURE SERVICE



WOLVERHAMPTON HEART FAILURE SERVICE

Patient Label

Heart Failure Team
 New Cross Hospital
 Old Ward 11
 Wolverhampton
 WV10 0QP
 Tel/Fax No 01902 643069

Date:

Dear Dr
 Thank you for referring the above patient to the Heart Failure Assessment Clinic.
 The diagnosis of heart failure has been confirmed.

History:

SOB	
Orthopnoea/PND	
Fatigue	
Dizziness	

Chest Pain	
Palpitations	
Other	

Examination:

Heart Rate	
BP	/
JVP	

Chest	
Oedema	
Weight	

Echocardiogram Result: Normal / Mild / Moderate / Severe / LV Dysfunction / Other

NYHA Class: **i** **ii** **iii** **iv**

Management Plan:

Heart Failure Review Clinic Y/N Follow up appointment.....
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Your patient will receive education and ongoing support from the Heart Failure Team. You will be kept informed regarding your patient at all times.

If you require any further information please do not hesitate to contact us.

Yours sincerely



WOLVERHAMPTON HEART FAILURE SERVICE

Patient Label

Heart Failure Team
 New Cross Hospital
 Old Ward 11
 Wolverhampton
 WV10 0QP
 Tel/Fax No 01902 643069

Date:

Dear Dr

The above patient attended the Heart Failure Review Clinic on.....

History:

SOB	
Orthopnoea/PND	
Fatigue	
Dizziness	

Chest Pain	
Palpitations	
Other	

Examination:

Heart Rate	
BP	/
JVP	

Chest	
Oedema	
Weight	

ECG:

--

Blood Results:

Sodium	
Potassium	
Urea	
Creatinine	
Other	

NYHA Class: i ii iii iv

Management Plan:

Follow up appointment for Heart Failure Review Clinic: Y/N inweeks
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If you require any further information please do not hesitate to contact us.

Yours sincerely

ISBN 1-903982-10-3

Executive Summary ISBN 1-903982-11-1