Special Report

Concise Guide to the Management of Heart Failure

World Health Organization/Council on Geriatric Cardiology of ISFC: Task Force on Heart Failure Education.

Local Sponsor: Council on Heart Failure for Pakistan.

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OBJECTIVE OF GUIDELINE

Heart failure is a major and growing public health problem worldwide. It is common, costly, disabling and deadly. Early diagnosis and effective treatment reduce morbidity, mortality, and cost. The aim of these guidelines is to provide a concise summary, for primary care physicians (general practitioners), of the modern diagnostic and therapeutic approach to a patient with suspected heart failure.

DEFINITIONS OF HEART FAILURE

Pathophysiological Definition

Cardiac failure is an inability of the heart to deliver blood (and therefore oxygen) at a rate commensurate with the requirements of the metabolising tissues at rest or during light exercise. This leads to characteristic systemic pathophysiological responses (neural, hormonal, renal and others), symptoms and signs.

Clinical Definition

Clinically the term "heart failure" is applied to the syndrome of breathlessness and fatigue associated with cardiac disease. It is often accompanied by fluid retention ("congestion"), as indicated by an elevated jugular venous pressure and oedema. Conditions leading to a mismatch between tissue oxygen delivery and demand (eg. anemia) may mimic the clinical signs of heart failure as many conditions causing fluid retention (eg. renal and hepatic failure). *The clinical diagnosis of heart failure, therefore, necessitates both the presence of significant cardiac disease and typical symptoms and signs.*

CLINICAL DIAGNOSIS OF HEART FAILURE

The clinical assessment of the patient with suspected heart failure seeks to answer two questions: (Figure 1; Diagnostic Algorithm).

* Are the patient's symptoms cardiac or non-cardiac in origin, i.e., is heart disease present?
* Where there is cardiac disease, what is the precise nature of the cardiac problem?

1. *Clinical History*

Although dyspnoea and fatigue are the hallmarks of heart failure, they are also common in other conditions (eg. respiratory disease, obesity). Knowledge of a pre-existing myocardial (eg. past myocardial infarction) or valvular problem increases the probability that the patient's symptoms are due to heart failure. A history of angina, hypertension, rheumatic fever or previous cardiac surgery is also helpful. Palpitations may indicate a cardiac rhythm or conduction disorder that may be the cause (or result) of heart failure. Conversely, a history of other relevant medical problems (eg. anaemia, pulmonary, renal or hepatic disease) may reduce the probability of heart failure.

2. *Clinical Examination*

Many patients with heart failure have few and/or subtle clinical signs. Conversely, some physical signs, such as ankle oedema, are very non-specific and may

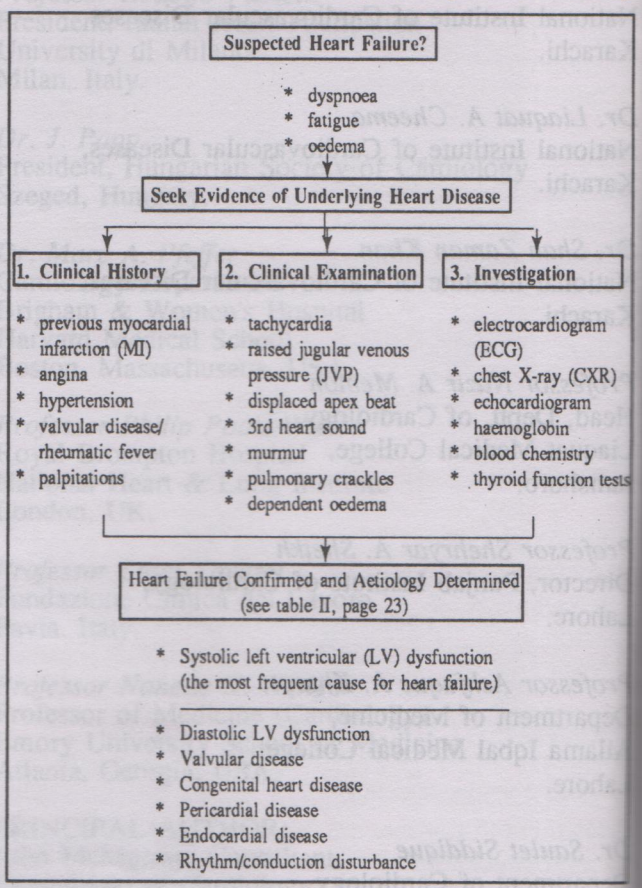


Fig. 1
Diagnostic Algorithm

be seen in patients without cardiac disease. A raised jugular venous pressure (in the absence of anaemia, pulmonary, renal or hepatic disease), a rapid low amplitude pulse, a third heart sound and displacement of the apex beat are specific signs of cardiac disease.

Blood pressure is usually normal or low for the patient's age provided that hypertensive heart disease is not the cause of the heart failure. Pulmonary crackles are non-specific in the absence of other signs of cardiac disease.

Conversely, dyspnoea in the absence of cardiac signs as mentioned above is more suggestive of pulmonary or other disease.

3. ***Investigations***

The purpose of investigation is to:

1) Confirm the diagnosis of heart failure by demonstrating underlying cardiac disease.

2) Define the cause of heart failure by characterizing the underlying cardiac problem.

3) Assist in the choice of optimal therapy (and avoid inappropriate treatment) by defining the precise cause of heart failure.

4) Obtain prognostic information.

5) Provide a reference point from which to measure the effects of treatment.

Blood tests help exclude anaemia, thyroid, hepatic and renal disease. The most commonly used cardiac investigations are the 12 lead electrocardiogram (ECG), the chest radiograph ("X-ray") and, where available, the echocardiogram.

CLINICAL DIAGNOSIS OF HEART FAILURE

The Resting ECG

A completely normal 12 lead ECG is uncommon in a patient with heart failure. Even in pericardial disease the QRS complexes are usually of low voltage and the patient often has atrial fibrillation. However, a breathless patient with an abnormal ECG may not necessarily have heart failure. Table I shows how the ECG can help in defining the cause of heart failure.

Chest Radiograph

In acute, or decompensated chronic, heart failure the chest x-ray may show florid alveolar pulmonary oedema, interstitial pulmonary oedema, basal pleural effusions or pulmonary venous engorgement. In older patients the most common finding is cardiac enlargement. Cardiomegaly is indicative of significant cardiac disease though it does not define the precise cardiac problem. For example, conditions as diverse as pericardial effusion, or pulmonale, left ventricular aneurysm and mitral stenosis all cause radiographic cardiomegaly.

It is important to realise, however, that significant left ventricular dysfunction can occur in the absence of radiographic cardiomegaly.

Echocardiogram

An important investigation in a patient with suspected heart failure is the echocardiogram. This ultrasonic examination visualises the cardiac chambers and valves. Systolic and diastolic ventricular contractile function can be measured, as can chamber size and wall thickness. Similarly, Doppler ultrasound enables valvular stenosis and regurgitation to be detected and quantified. Congenital heart defects, valvular vegetations, intracardiac tumours and intracavitary thrombus can also be detected.

Ideally all patients with suspected heart failure should have an echocardiogram though this investigation is often not available. If a definite diagnosis of heart failure can be made clinically, e.g., in a patient who has had a large myocardial infarction in the past or who has the characteristic auscultatory findings of mitral stenosis, echocardiography is not essential if access is limited. On the other hand echocardiography is strongly recommended where there is diagnostic uncertainty on clinical grounds (see pages).

TABLE I

ECG FINDINGS IN PATIENTS WITH SUSPECTED HEART FAILURE

Finding	Significance
Q-waves; poor R wave progression	Indicates previous MI; LV systolic dysfunction probable.
Left ventricular hypertrophy (LVH)	May be due to hypertension, aortic stenosis, hypertrophic or dilated cardiomyopathy. May have either systolic or diastolic LV dysfunction.
Left bundle-branch block (LBBB)	Usually indicates underlying heart disease.
Right bundle-branch block (RBBB)	May not denote underlying heart disease. Incomplete RBBB may indicate an atrial septal defect. RBBB and left anterior hemiblock (LAHB) are common in Chagas disease.
Atrial fibrillation	Common in old age. May be caused by any cardiac disease. Should specifically consider mitral stenosis, pulmonary embolism, and thyrotoxicosis. If ventricular rate is very rapid may have rate related heart failure despite normal myocardial and valve function.
Bradyarrhythmias	Heart failure may be rate related.
Atrioventricular block	Heart failure may be rate related. May be present in Chagas disease.
Low voltage QRS complexes	May indicate pericardial constriction or effusion, infiltrative disease.

DIAGNOSIS AS THE BASIS OF TREATMENT

Optimal treatment is based on accurate diagnosis. Enough diagnostic information to enable appropriate treatment may be available from the history, examination and simple investigations. For example, a patient presenting with breathlessness for a number of months or years after myocardial infarction, and who has Q waves on the ECG, will almost certainly have heart failure due to left ventricular systolic dysfunction. The probability is even greater if the chest x-ray shows cardiomegaly and/or pulmonary congestion. Other patients may need more thorough investigation, possibly necessitating referral to a cardiologist. Ultimately the precise cause of each patient's heart failure should be ascertained as different causes of heart failure require different treatments (Table II - page 23 and Figures 2 and 3, pages 24 and 25).

TREATMENT OF HEART FAILURE DUE TO LEFT VENTRICULAR SYSTOLIC DYSFUNCTION

Non-Pharmacological Treatment/Life-Style Modification

Diet

All patients need support and dietary advice regarding maintenance of optimal weight. Obesity increases the workload on the heart, especially during physical activity. Weight reduction, through restriction of dietary fat and calories is imperative for those who are obese, and is advised for those who are overweight. In patients with coronary heart disease and raised lipids, a low fat diet may delay recurrence of significant cardiovascular events. Conversely, maintenance or improvement of the nutritional status in wasted, undernourished or alcoholic patients is also important.

Salt intake should be restricted as this may aggravate a patient's condition. Salt should not be added during cooking or at the table.

Fluid intake

Patients with heart failure often have an intense thirst, which can lead to excessive fluid intake and hyponatraemia. Fluid intake should be limited where possible, to about 2 liters a day for most patients. During periods of hot weather, diarrhoea, vomiting or fever, fluid intake may be increased or the dose of diuretic reduced.

Alcohol intake

Alcohol can damage the myocardium and precipitate arrhythmias. It should be avoided or used only in moderation.

Smoking

Smoking increases the risk of many cardiovascular, pulmonary and other problems, including cancers, and must be avoided at all costs.

TABLE II

SUMMARY OF THE TYPES OF HEART FAILURE AND THEIR TREATMENTS

Aetiology and Pathophysiology	Treatments in Common Practice
Myocardial Systolic Failure Heart failure is most commonly due to systolic dysfunction where the myocardium fails to contract normally; the left ventricle is usually dilated. Previous myocardial infarction, chronic hypertension, dilated cardiomyopthy, viral myocarditis, Chagas' disease and alcoholic heart disease are common causes of this type of heart failure.	It is important to identify these patinets, because prognosis in systolic heart failure is improved if an angiotensin converting enzyme (ACE) inhibitor is given in addition to diuretic and/or digoxin. Heart failure in patients with alcoholic cardiomyopathy often improves or resolves if they stop drinking.
Myocardial Diastolic Failure Heart failure is sometimes due to diastolic ventricular dysfunction, where the myocardium is stiff, often because of hypertrophy, and fails to relax normally. This problem seems to be more common in the elderly and may have a better prognosis than systolic heart failure. Hypertension can cause this type of heart failure.	The optimal treatment for diastolic heart failure has yet to be determined. Underlying problems should be treated appropriately, eg., antihypertensive therapy should be given to lower elevated blood pressure, aiming to induce regression of left ventricular hypertrophy. "Congestion" should be relieved with diuretics.
Valvular Disease Valvular disease remains a cmmon cause of heart failure in areas of the world where rheumatic fever is prevalent. Calcific aortic stenosis is also a frequent problem in elderly patients.	Surgery and other interventional procedures such as balloon valvuloplasty are of potentially great benefit. Inoperable regurgitant valvular disease may be helped by vasodilators.
Pericardial Disease Pericardial constriction and effusion, for example, due to tuberculosis or viral disease may compromise pump function. Cardiac tamponade should be considered.	If conservative treatment fails, pericardiocentesis, balloon pericardiotomy and surgical pericardiectomy may be of benefit.
Endocardial Disease Endocardial or endomyocardial fibrosis and its variant, Loffler's disease (endocarditis prietalis fibroplastica), cause a form of diastolic, or restrictive, heart failure.	The pathophysiology of heart failure due to these conditions is poorly understood and its treatment is not well studied. Diuretics and digoxin are commonly given for symptomatic relief.
Congenital Heart Disease Many forms of congenital heart disease may cause heart failure in infancy or childhood. Some types (eg. atrial septal defect and aortic stenosis) may not present until late in life as heart failure.	Surgical as well as medical treatment is often required.
Metabolic Heart Disease Thyroid disease causes heart failure. Thiamine deficiency (beri beri) may cause heart failure in specific populations. Iron overload (haemochromatosis and haemosiderosis) may also cause myocardial injury.	Replacement or removal of the relevant nutritional, hormonal or metabolic factor is usually curative.

Exercise

Bed rest is an important part of the treatment of acute heart failure or decompensated chronic heart failure, though early mobilization is important. Otherwise regular, and moderate physical activity for the condition of the patient, should be encouraged. This has significant symptomatic and other benefits in patients with heart failure. Dynamic exercise activities such as walking, cycling, swimming, bowling, gardening, etc. should be continued at a pace that is comfortable for the patient.

Vaccination

Heart failure may predispose to and be exacerbated by pulmonary infection, which is a common cause of hospitalization. Therefore, influenza and pneumococcal vaccinations are recommended.

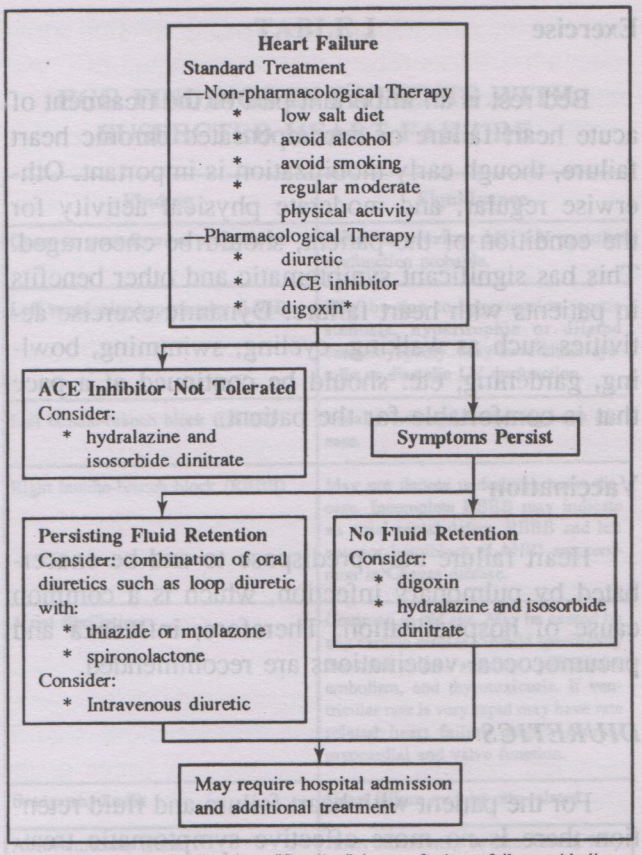
DIURETICS

For the patient with heart failure and fluid retention there is no more effective symptomatic treatment than a diuretic. Normally diuretics are used in combination with an ACE inhibitor and/or digoxin (see footnote, page 24). Four main principles underlie the use of diuretics in heart failure (see page 24). Loop diuretics are commonly used though in mild heart failure thiazide diuretics may suffice. The effective daily dose of frusemide is 40 mg (equivalent to 1 mg of bumetanide), but a reduced response may lead to a need for 80 to 120 mg daily.

Overtreatment can cause hypovolaemia, hypotension and renal impairment.

Patient Guidelines for Use

All dieuretics cause inconvenience for patients who usually have to organize their daily activities around the period of most intense diuresis. Thiazide diuretics cause a prolonged mild diuresis whereas loop diuretics cause a shorter, more vigorous diuresis. The effect of loop diuretics usually diminishes four hours after dosage. Patients should be informed that there is generally no fixed time of day that diuretics must be taken and, according to individual circumstances, the dose may be taken in the morn-



* Some physicians use digoxin as "first line" therapy for heart failure, with diuretics and ACE inhibitors, whereas others reserve its use to those patients with atrial fibrillation or those patients whose symptoms persist.

Fig. 2:
Treatment Algorithm

ing, afternoon or evening (but not too late as the diuresis may interrupt sleep). The patient may also be flexible with the diuretic dose according to need. Patients can be instructed to record their daily weight (on rising, after voiding, before breakfast) and, if there is a consistent (more than three consecutive days) increase in weight of more than 0.5 kg., they are advised to increase the diuretic dose until 'dry weight' is regained. If the weight gain or symptoms worsen, the patient should be instructed to seek medical help.

ANGIOTENSIN CONVERTING ENZYME (ACE) INHIBITORS
(See figure 3 and Table III)

ACE inhibitors have been shown to be of benefit in all symptomatic classes of heart failure due to left ventricular systolic dysfunction. ACE inhibitors reduce vasoconstriction, improve pump function, and increase renal and skeletal muscle blood flow in heart failure. When given with diuretics, ACE inhibitors improve the symptoms and signs of all classes of heart failure and improve exercise tolerance. Increased severity of heart failure and need for hospital admission are reduced. Survival is improved in all classes of heart failure with ACE inhibitor treatment. The risk of myocardial infarction may also be reduced.

Guidelines for use

Certain precautions should be taken before treatment is started. Potassium supplements and potassium sparing diuretics should be withdrawn. It is desirable that a baseline measurement of blood chemistry is made. The patient should be observed for two to four hours after the first dose. A low dose should be given initially - for example, enalapril 2.5 mg or captopril 6.25 mg - and regular treatment can then usually be started at an intermediate dose - enalapril 2.5 mg twice daily or captopril 12.5 mg three times daily. It is desirable that the patient should be reviewed after one or two weeks to check blood chemistry and for symptoms of hypotension; the drug dose should be modified accordingly. Provided the patient has not experienced significant hypotensive symptoms or a significant rise in serum creatinine or potassium concentration (>200 mmol/l or 5.5 mmol/l respectively), the dose of ACE inhibitor should be increased as tolerated. Larger doses such as enalapril 10 mg given twice daily or captopril 50 mg three times daily are recommended as these dosages have been shown to be beneficial in clinical trials. The correct dose of ACE inhibitors in heart failure should be guided by clinical trials and individual patient

Principles of using diuretics for heart failure

* Use in moderation; avoid excessive doses of any single drug
* Make use of synergy between different classes of drugs, especially in cases of diuretic resistance (the principle of sequential nephron blockade)
* Monitoring of blood chemistry may help to avoid uraemia, hypokalaemia, and hyponatraemia
* Use in combination with an angiotensin converting enzyme (ACE) inhibitor and/or digoxin (see footnote, page 12), unless this is not tolerated

response. For certain patients at high risk (Table III), special assessment is desirable and hospital admission may be advisable.

Adverse effects of angiotensin converting enzyme inhibitors

Hypotension

Some reduction in blood pressure is expected but it usually induces no symptoms. However symptomatic hypotension may occur in a small percentage of patients. If it does occur, the patient may be hypovolemic, in which case treatment can often be restarted after correction of dehydration. Ensure that obstructive valve disease is not present, and that the patient does not have diastolic rather than systolic left ventricular dysfunction.

Renal dysfunction

In patients with mild and moderate heart failure in clinical trials only small changes in serum creati-

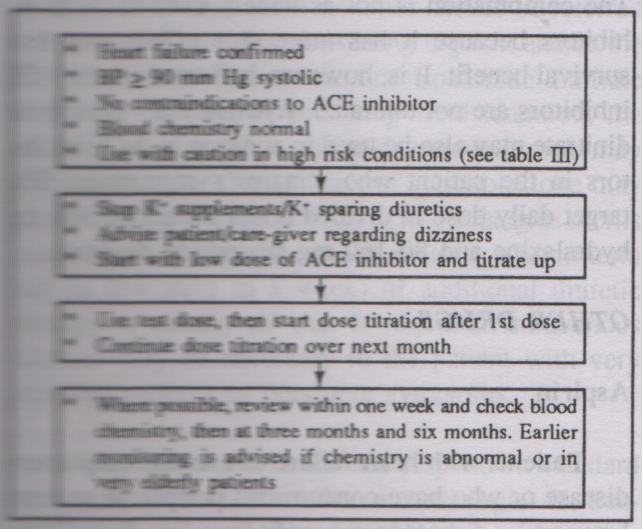


Fig. 3:
Initiation of an ACE Inhibitor in Heart Failure

nine and urea concentration 8.8 µmol/l [0.10 mg%] and 1.2 mmol/l [3.2 mg%] respectively) occurred after treatment. Among patients with severe heart failure, many of whom had abnormal baseline blood chemistry, serum creatinine concentration was as likely to fall as it was to increase after starting ACE inhibitor treatment. As with symptomatic hypotension, renal dysfunction is often exacerbated by dehydration. Non-steroidal anti-inflammatory drugs (NSAIDs) may also cause renal dysfunction and should be avoided if possible in patients receiving ACE inhibitors. Patients should be advised not to purchase and use "over the counter" NSAIDs. See co-prescribing, page 27.

Hyperkalaemia

TABLE III

HIGH-RISK CONDITIONS FOR WHICH SPECIALIST ASSESSMENT IS DESIRABLE

* Severe heart failure (New York Heart Association class III or IV - shortness of breath on slight effort or at rest); patients with a dose of 80 mg of frusemide or equivalent
* Low systolic blood pressure (<90 mmHg)
* Low serum concentration of sodium (<90 mmHg)
* Low serum concentration of sodium (<130 mmol/l) or high potassium concentration >5.5 mmol/L
* Existing renal dysfunction (serum creatinine concentration [2.26 mg%] >200µmol/L)
* Severe generalized atherosclerosis (especially if intermittent claudication and arterial bruits are present) ie., risk of renal artery stenosis
* Severe chronic obstructive airways disease and pulmonary heart disease (cor pulmonale) ie., risk of fixed pulmonary hypertension

Few patients with mild and moderate heart failure develop worrisome hyperkalaemia, i.e., K^+>5.5 mmol/l. This has rarely been a cause of withdrawal in the large trials using an ACE inhibitor. Dehydration, NSAIDs, and potassium-sparing diuretics increase the risk of hyperkalaemia. If these are not responsible the dose of ACE inhibitor should be reduced and K^+ monitored carefully.

Cough

Cough is common in patients with heart failure and pulmonary oedema/congestion should always be excluded. ACE inhibitors can also cause cough, though this was only troublesome enough to cause treatment withdrawal in a very small percentage of patients in the large ACE inhibitor clinical trials.

Advice to the patient and care-givers

Symptomatic patients should be informed that

they are likely to notice a gradual improvement in their symptoms, and that this may take some weeks to develop fully. Patients who become asymptomatic should be informed that their treatment is being continued to keep them well. Patients can also be advised that future hospitalization is less likely and that life expectancy is improved by treatment with an ACE inhibitor.

Patients should be warned that dizziness may occur initially, after starting treatment, though this is usually transient and resolves with lying down. If it is persistent and troublesome, a doctor should be notified before further doses are taken. If patients become dehydrated (e.g., due to diarrhoea and vomiting or a hot climate), dizziness may occur. A temporary increase in fluid intake and/or reduction in diuretic dose usually results in resolution.

Cough and, less commonly, taste disturbance may occur within the first few weeks of starting treatment. If the adverse effect is not particularly troublesome, patients may be willing to accept it in light of the substantial benefits of ACE inhibitor therapy.

DIGOXIN

Digoxin should be used to control the ventricular rate, where necessary, in patients with heart failure and atrial fibrillation. Digoxin is also of benefit in patients with heart failure who are in sinus rhythm.

Although usually considered a positive inotropic agent, digoxin is known to have other important effects in heart failure. These include neuroendocrine suppression, especially sympathetic nervous system inhibition, and arterial vasodilation. Digoxin also has complex direct and indirect electrophysiological effects.

Digoxin is of symptomatic benefit in patients with heart failure in sinus rhythm though survival data will not be available until the DIG (Digitalis Investigation Group) Trial reports in 1996. Digoxin is also of symptomatic benefit when given with an ACE inhibitor and diuretic and this is its main indication in patients with heart failure who are in sinus rhythm.

Most trials showing clinical benefit have used daily doses between 0.125 and 0.375 mg, though a lower dose may be required in elderly patients and those with impaired renal function.

The incidence of digoxin toxicity in outpatients appears to be low, about one episode for every 20 years of treatment. This is supported by the recent large digoxin trials where the incidence of adverse effects with digoxin has been no different from that with placebo. However, the presence of renal insufficiency augments the risk of toxicity. Some potential drug interactions are relevant to heart failure. Hypokalaemia (caused by diuretics) increases the risk of digoxin toxicity. Amiodarone and quinidine increase serum digoxin concentrations due to a pharmacokinetic interaction.

OTHER VASODILATORS

Hydralazine and isosorbide dinitrate

This combination improves symptoms, exercise tolerance, and survival in patients with heart failure. The combination is not as widely used as ACE inhibitors because it has more side effects and less survival benefit. It is, however, indicated where ACE inhibitors are not tolerated. Hydralazine - isosorbide dinitrate may also be used in addition to ACE inhibitors in the patient who remains symptomatic. The target daily dose in divided doses should be 300 mg hydralazine and 80-160 mg isosorbide dinitrate.

OTHER DRUGS

Aspirin

Patients with heart failure due to coronary artery disease or who have concomitant peripheral or cerebrovascular disease may benefit from low dose (75mg -325 mg) aspirin, because of its platelet antiaggregant property.

Warfarin

Patients with atrial fibrillation and heart failure should always be considered for warfarin treatment. Any other patient who has had a thromboembolic episode or who has been shown to have intracardiac thrombus should be considered for treatment with

warfarin. The place of anticoagulation in patients with heart failure who are in sinus rhythm and who do not have intracardiac thrombus or a history of thromboembolism is uncertain.

Beta Adrenoreceptor Antagonists

Initiation of conventional doses of beta blockers in patients with heart failure can cause profound haemodynamic and clinical deterioration. There is, however, some evidence that cautious introduction of a very low dose of beta blocker, under careful observation, followed by gradual dose titration may lead to symptomatic and, possibly, survival benefit. At present the precise role of beta blockers in the treatment of heart failure is uncertain and the results of ongoing trials are awaited.

APPARENTLY INTRACTABLE HEART FAILURE

(also see Figure 2 - page 24)

Patients who do not respond to standard therapy with a diuretic and ACE inhibitor should be referred for specialist assessment. Adding digoxin and/or the combination of hydralazine and isosorbide dinitrate may help. If there is persisting fluid retention, the addition of a thiazide diuretic or metolazone may induce diuresis. The addition of spironolactone may also initiate a diuresis. Each of these strategies merits careful monitoring of the blood chemistry. Short term use (a few days to a week) of additional diuretic therapy is usually all that is required. Intravenous diuretic may be necessary in the patient with very resistant oedema or persistent symptoms.

Many patients who do not respond to standard treatment are best referred to a cardiologist for hospital admission and some combination of bed rest, fluid restriction, intravenous diuretic, dobutamine, sodium nitroprusside or dopamine. Invasive haemodynamic monitoring is often helpful in establishing treatment goals and efficacy. Cardiac transplantation may be considered in selected cases.

In patients with severe diuretic resistant heart failure, careful nursing attention is important. Sleeping with the head elevated in bed or in a comfortable chair may prevent distressing nocturnal breathlessness.

OTHER ASPECTS OF MANAGEMENT

CO-PRESCRIBING

Care in co-prescribing cannot be over-emphasized. The following drugs should be used with caution and usually avoided:

* NSAIDs
* Calcium channel blockers (except possibly amlodipine)
* Antiarrhythmics (except amiodarone)
* Beta blockers
* Corticosteroids
* Tricyclic antidepressants
* Lithium
* Carbenoloxone

NON-ADHERENCE

Careful advice about the rationale behind treatment, especially diuretic treatment, and an explanation about flexible timing of doses may help to prevent non-compliance. When ACE inhibitors are prescribed for patients rendered asymptomatic by diuretics the patient should be told of their prophylactic benefit in maintaining stability, preventing hospitalisation and improving survival.

MANAGEMENT OF CONCOMITANT PROBLEMS

Many patients with heart failure have concomitant problems which either reflect the underlying cause of their heart failure (e.g., angina) or are a consequence of it (e.g., ventricular arrhythmias). The management of these concomitant problems in patients with heart failure is often different from that in patients who do not have heart failure. This section briefly highlights the concomitant problems typically

sion to heart failure can also be reduced. Infarct size can be limited by thrombolytic therapy and reinfarction reduced by aspirin, beta blockers, and, possibly, ACE inhibitors. In asymptomatic patients with substantial impairment of left ventricular function prophylactic ACE inhibitor therapy has been shown to reduce the risk of developing heart failure and to improve survival.

CONCLUSION

In a patient with suspected heart failure, precise diagnosis of the underlying cardiac problem is the basis of optimal treatment. Non-pharmacological, pharmacological, and surgical treatments are available. With modern treatment, both morbidity and mortality from heart failure can be substantially reduced.

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present in patients with heart failure.

ATRIAL FIBRILLATION

Up to 30 per cent of patients with heart failure have concomitant atrial fibrillation. Five questions should be asked before management is started (see below). Control of ventricular rate is usually achieved with digoxin; if there is difficulty, consider amiodarone but remember that digitalis toxicity may develop. Thromboembolism should be prevented, and recent trials have shown substantial benefit from treatment with warfarin. Cardioversion, possibly preceded and followed by treatment with amiodarone, should be considered as, ideally, sinus rhythm should be restored.

ANGINA

As the commonest cause of heart failure is coronary artery disease, may patients also have angina. Coronary artery bypass grafting should be considered if the patient is otherwise suitable for surgery. Coronary angioplasty or other transcatheter revascularization procedure may also be appropriate. Prognosis may be improved by surgery in patients with extensive coronary artery disease and left ventricular dysfunction. Advanced age (>75 years) and severe left ventricular dysfunction (left ventricular ejection fraction <20%) are relative contraindications. The only antiischaemic drugs available that do not exacerbate pump dysfunction are the nitrates and possibly amlodipine (see beta blockers, page 27).

Issues affecting management of atrial fibrillation in patients with heart failure

* Is atrial fibrillation the cause or consequence of heart failure?
* Could the patient have mitral valve disease?
* Could the patient have thyrotoxicosis?
* Is atrial fibrillation part of sick sinus syndrome? (Bradycardia may aggravate heart failure, and digoxin may aggravate bradycardia.)
* Are there any contraindications to the use of warfarin?

VENTRICULAR ARRHYTHMIA

Patients with symptoms of palpitations, dizziness, and blackouts should be investigated for arrhythmias, since symptomatic ventricular arrhythmia requires treatment. Before an antiarrhythmic drug is given, possible precipitating or aggravating factors must be excluded. (see below). The drug to consider is amiodarone; others are likely to worsen a patient's overall condition and even the arrhythmia. Beta blockers may be useful, if tolerated. The role of implantable pacemaker and defibrillator devices in patients with heart failure is not yet clear.

PREVENTION OF HEART FAILURE

Prevention of heart failure is a major aim of modern cardiological practice because the burden of symptoms is so high and prognosis is so poor once overt cardiac failure is established. This aim can be achieved by a) preventing the development of causal heart disease, e.g., myocardial infarction, hypertensive heart disease and b) preventing the progression of established cardiac disease to heart failure.

The role of smoking, cholesterol, sedentary lifestyle and other factors in promoting the development of coronary heart disease (CHD) are well recognised and can be altered. Hypertension also increases the risk of developing CHD and the risk of developing heart failure through direct, chronic, myocardial damage. Antihypertensive therapy has been clearly shown to reduce the risk of developing heart failure. Improved socioeconomic conditions and antimicrobial therapy have reduced the incidence of rheumatic heart disease in many societies.

Possible precipitating or aggravating factors for ventricular arrhythmia

* Electrolyte disturbance, e.g., hypokalaemia, hypomagnesaemia, hyperkalaemia
* Digoxin toxicity
* Drugs exacerbating pump dysfunction, e.g., most antiarrhythmic drugs and some calcium channel blockers
* Drugs causing electrical instability, e.g., most antiarrhythmic drugs (except amiodarone) and antidepressants
* Recurrent myocardial ischaemia
* Respiratory disease, infection, hypoxaemia, hyperthyroidism

Once cardiac disease is established its progres-

Cardiology For The Trainee:

(Physicians in training in cardiology can have their questions addressed to in this section-Ed.)

Following guide is reproduced from: Heart beat No. 3, September, 1995.

Guide to comprehensive risk reduction for patients with coronary and other vascular disease

<table>
<tr><th>Risk Intervention</th><th colspan="5">Recommendations</th></tr>
<tr><td>Smoking:
<u>Goal</u>
complete cessation</td><td colspan="5">Strongly encourage patient and family to stop smoking.
Provide counselling, nicotine replacement, and formal cessation programmes as appropriate.

Start AHA Step II Diet in all patients: ≤30% fat, <7% saturated fat, <200 mg/d cholesterol.</td></tr>
<tr><td>Lipid management:
<u>Primary goal</u>
LDL<100 mg/dL
<u>Secondary goals</u>
HDL>35 mg/dL;
TG<200 mg/dL</td><td colspan="5">Assess fasting lipid profile. In post-MI patients, lipid profile may take 4 to 6 weeks to stabilize. Add drug therapy according to the following guide:</td></tr>
<tr><td rowspan="5">LDL<100 mg/dL

No drug therapy</td><td colspan="3">LDL 100 to 130 mg/dL

Consider adding drug therapy to diet, as follows:</td><td colspan="2">LDL>130 mg/dL

Add drug therapy to diet, as follows:</td><td rowspan="5">HDL<35 mg/dL

Emphasize weight management and physical activity. Advise smoking cessation.
If needed to achieve LDL goals, consider niacin, statin, fibrate.</td></tr>
<tr><td colspan="4">Suggested drug therapy</td></tr>
<tr><td>TG<200 mg/dL</td><td>TG 200 to 400 mg/dL</td><td colspan="2">TG>400 mg/dL</td></tr>
<tr><td>Statin
Resin
Niacin</td><td>Statin
Niacin</td><td colspan="2">Consider combined drug therapy (niacin, fibrate, statin)</td></tr>
<tr><td colspan="4">If LDL goal not achieved, consider combination therapy.</td></tr>
<tr><td>Physical activity:
<u>Minimum goal</u>
30 minutes 3 to 4 times per week</td><td colspan="5">Assess risk, preferably with exercise test, to guide prescription.
Encourage minimum of 30 to 60 minutes of moderate-intensity activity 3 or 4 times weekly (walking, jogging, cycling, or other aerobic activity) supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, using stairs, gardening, household work). Maximum benefit 5 to 6 hours a week.
Advise medically supervised programmes for moderate - to high - risk patients.</td></tr>
<tr><td>Weight management:</td><td colspan="5">Start intensive diet and appropriate physical activity intervention, as outlined above, in patients>120% of ideal weight for height.
Particularly emphasize need for weight loss in patients with hypertension, elevated triglycerides, or elevated glucose levels.</td></tr>
<tr><td>Antiplatelet agents/ anticoagulants:</td><td colspan="5">Start aspirin 80 to 325 mg/d if not contraindicated.
Manage warfarin to international normalized ratio=2 to 3.5 for post-MI patients not able to take aspirin.</td></tr>
<tr><td>ACE inhibitors post-MI:</td><td colspan="5">Start early post-MI in stable high-risk patients (anterior MI, previous MI, Killip class II (S3 gallop, rales, radiographic CHF)). Continue indefinitely for all with LV dysfunction (ejection fraction ≤ 40%) or symptoms of failure.
Use as needed to manage angina, rhythm or blood pressure in all other patients.</td></tr>
<tr><td>Beta-blockers:</td><td colspan="5">Start in high-risk post-MI patients (arrhythmia, LV dysfunction, inducible ischemia) at 5 to 28 days. Continue 6 months minimum. Observe usual contraindications.
Use as needed to manage angina, rhythm or blood pressure in all other patients.</td></tr>
<tr><td>Oestrogens:</td><td colspan="5">Consider oestrogen replacement in all postmenopausal women.
Individualize recommendation consistent with other health risks.</td></tr>
<tr><td>Blood pressure control:
<u>Goal</u>
≤140/90 mm Hg</td><td colspan="5">Initiate lifestyle modification - weight control, physical activity, alcohol moderation, and moderate sodium restriction - in all patients with blood pressure > 140 mm Hg systolic or 90 mm Hg diastolic.
Add blood pressure medication, individualized to other patient requirements and characteristics (i.e., age, race, need for drugs with specific benefits) if blood pressure is not less than 140mm Hg systolic or 90 mm Hg diastolic in 3 months or if *initial* blood pressure is > 160 mm Hg systolic or 100 mm Hg diastolic.</td></tr>
<tr><td colspan="6">*ACE indicates angiotensin-converting enzyme; MI, myocardial infarction; TG, triglycerides; and LV, left ventricular.*</td></tr>
</table>