Outpatient Management of Heart Failure

What is CHF?

“CHF is a complex clinical syndrome with typical symptoms (e.g. dyspnoea, fatigue) that can occur at rest or on effort, and is characterised by objective evidence of an underlying structural abnormality or cardiac dysfunction that impairs the ability of the ventricle to fill with or eject blood (particularly during physical activity).”

A diagnosis of CHF may be further strengthened by improvement in symptoms in response to treatment.

National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand Guidelines for the prevention, detection and management of chronic heart failure in Australia, 2006

Common causes of CHF are

- ischaemic heart disease (present in over 50% of new cases),
- hypertension (about two-thirds of cases)
- idiopathic dilated cardiomyopathy (around 5-10% of cases).

NHF 2006 guidelines

Diagnosis is based on

- clinical features
- chest x-ray
- objective measurement of ventricular function (e.g. echo)
- plasma levels of B-type natriuretic peptide may have a role in diagnosis.

Diagnosis may be strengthened by improvement in symptoms in response to treatment.

LV function assessments with treadmill performance

Naughton J Exercise Testing Futura 1988

Clinical Trap

Oedema is not always a sign of CHF

Mrs DD Albumin 40

Practice points from 2006 NHF Guidelines

“Clinical diagnosis of CHF is often unreliable, especially in obese patients, those with pulmonary disease and the elderly. Therefore, it is important to perform investigations to confirm the diagnosis.”

“All patients with suspected CHF should undergo an ECG, chest x-ray and echocardiogram, even if the physical signs are normal.”
Risk factors for heart failure:
- CHD or history of MI
- Hypertension
- Valvular heart disease
- Alcoholism
- Sleep apnoea
- Cardiotoxic drugs e.g. chemotherapeutic agents
- Thyroid dysfunction
- Atrial fibrillation and other arrhythmias
- Diabetes
- Congenital heart defects
- Obesity
- Age
- Smoking
- LVH on echo

Obstructive Sleep Apnoea
A randomised controlled study in obstructive sleep apnoea patients with systolic LV dysfunction and heart failure has shown CPAP treatment to lead to a significant improvement in LVEF, a fall in systolic blood pressure and a reduction in LV chamber size.


Practice point NHF 2006 guidelines
“If sleep apnoea is suspected, referral to a sleep physician is indicated.”

The modified ‘Schroeder approach’ to CHF

Step 1. Look for treatable causes
- Ischaemia - including painless (silent).
- ‘Surgical’ lesions eg valvular lesions, HCM
- Tachycardia-induced cardiomyopathy.
- Alcohol and toxins
- Myocarditis
- Infiltrations: Sarcoidosis, Haemochromatosis
- Thyroid disease
- Diabetes
- Sleep apnoea.

Step 2. Use the basic tools
- Stress Testing
- Serial OP Labs and Echo
- Cardiac Cath, Coronary angio, Myocardial Biopsy, (Nuclear medicine tests and MRI)

Step 3. The hierarchy of medical Rx
- Initiate ACE Inhibitor and monitor creatinine
- Avoid Xs diuresis (“short term loop diuretics OK”)
- Switch to ARB if cough truly troublesome

Once stable:
- Initiate Carvedilol/B blockade - HR >/= 55 and SBP 90 if OK
- Diuretics ( monitor EUC - * beware aggravating DHF - echo )
- *Aldosterone antagonists, Thiazide etc
- Frusemide - am weight based.
- Digoxin (conservative dose)
- Consider adding ARB to ACE I if Creat OK and SBP > 110.
- Treat endothelial dysfunction to promote vasodilation (Statin)
- “Mandate regular walking program”
- Correct anaemia, infections, Obesity, DM etc
- Consider RFA Rxs, anticoagulation with Warfarin,
- ICD with pacemaker CRT
**Echocardiography**

**Heart failure with preserved systolic function (HFPSF)**

Causes of heart failure with preserved systolic function (impaired relaxation)

**Common causes**
- Hypertension (especially systolic hypertension): Patients tend to be female and elderly. This cause now represents 40-50% of all hospital admissions for CHF.
- CHD, which may lead to impaired myocardial relaxation.
- Diabetes — men with diabetes are twice as likely to develop heart failure than men without diabetes, and women with diabetes are at fivefold greater risk than women without diabetes. These differences persist after taking into account age, blood pressure, weight, cholesterol and known coronary artery disease. Myocardial ischaemia is very common in diabetes and is aggravated by hyperglycaemia, as well as concomitant hypertension and hyperlipidaemia. However, diabetes is additionally associated (independent of ischaemia) with interstitial fibrosis, myocyte hypertrophy and apoptosis, as well as both autonomic and endothelial dysfunction, all of which may contribute to the diabetic cardiomyopathic state.

**Less common causes**
- Valvular disease, particularly aortic stenosis.
- Hypertrophic cardiomyopathy — most cases are hereditary.
- Restrictive cardiomyopathy, either idiopathic or secondary to infiltrative disease, such as amyloidosis.

**Diastolic Heart Failure**

**Exercise Intolerance in Patients With Heart Failure and Preserved Left Ventricular Systolic Function: Failure of the Frank-Starling Mechanism.**


Seven patients with congestive heart failure with normal ejection fraction and no significant coronary or valvular disease underwent invasive cardiopulmonary exercise testing using upright bicycle ergometry. The results were compared with that of 10 age-matched normal subjects. The patients had hypertension or cardiomyopathy. They had a 48% reduction in peak oxygen consumption, with a 41% reduction in peak cardiac index. During exercise, there was a failure of left ventricular stroke volume index to increase normally, accompanied by a marked increase in pulmonary wedge pressure.

The left ventricular end-diastolic pressure-volume relationship was abnormal, indicating limited left ventricular diastolic filling.

The authors conclude that diastolic dysfunction limited these patients’ ability to increase stroke volume by means of the Frank-Starling mechanism.

(Photograph D. Evans, M.D.)

**Exercise Intolerance in Patients With Heart Failure and Preserved Left Ventricular Systolic Function**

- **Delay sx – clinical and echo FU at 12 mths 0-1**
- **Borderline – review 6 mths – clinical and echo 2**
- **Operate 3 or >**

**Points Decision regarding surgical intervention**

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<th>Clinical EF</th>
<th>LV end systole</th>
<th>Exercise Capacity</th>
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**Aortic Regurgitation Algorithm for Timing of Surgery**

**Heart Failure with Preserved Systolic Function — Just as Common, Just as Deadly**


The presence of LVH in a patient with hypertension increases the risk of congestive heart failure 2 to 3 times

**Heart Failure with Preserved Systolic Function**

- New research confirms that the clinical syndrome of heart failure in conjunction with preserved ejection fraction is common and associated with high risk outcomes.
- This year, three studies looked at the relative incidence of reduced and preserved ejection fraction (EF) in heart failure patients and at outcomes in each group.
- In the first Canadian researchers studied more than 2000 patients admitted with heart failure whose EF was assessed. The majority (87%) had reduced (EF ≤40%), but a substantial minority (13%) had preserved EF (40%–50%). (One-year mortality was only slightly higher among those with reduced EF than among those whose EF was preserved [39% vs. 37%].)
- In the second study, Mayo Clinic researchers reviewed the records of more than 6000 patients presenting with heart failure from 1987 to 2001. Of the 6000 patients with echocardiographic assessment, 65% had reduced (EF ≤40%) and 35% had a preserved EF (40%–50%). Patients with preserved EF were older and more likely to be female, and more commonly had hypertension or atrial fibrillation. Patients with reduced EF were more likely to have underlying coronary artery disease or significant left atrial disease. Median 1-year mortality was slightly higher among those whose EF was preserved [37% vs. 36%].
- Finally, in a recent prospective population-based study, Mayo researchers recruited 558 patients who underwent echocardiography around the time of their diagnosis. Preserved EF (32%) was present in 558 patients, while isolated diastolic dysfunction was present in 45%. Preserved EF was associated with older age, diabetes, and no history of previous myocardial infarction. Patients with preserved EF were more likely to be women and to have diabetes, and less likely to have prior myocardial infarction. The average brain natriuretic peptide (BNP) level was not significantly different between groups, but within each EF category, BNP levels correlated with severity of diastolic dysfunction. No association was seen in both groups after adjustment for age, sex, and mortality risk was lower among patients with preserved EF (hazard ratio, 0.85), but not significantly.

These 3 studies confirm that the clinical syndrome of heart failure in conjunction with preserved ejection fraction is common, representing roughly half of all heart failure. It is also associated with high-risk outcomes, underscoring the need for more effective therapy for heart failure in association with preserved systolic function.

Fleischmann, Journal Watch General Medicine December 20, 2006

**NHF guidelines 2006**

Heart failure with preserved systolic function (HFPSF), or diastolic heart failure, is common and may account for up to 40% of patients with CHF.

The presence of LVH in a patient with hypertension increases the risk of congestive heart failure 2 to 3 times.
Diastolic heart failure is an LV filling problem

BNP and NT-pro BNP
- When diagnosing systolic left ventricular (LV) heart failure, these markers are good.
- But when we are talking about diastolic LV failure or RV failure, they are not quite so good.
- Clinical judgment is still very important. Complements but does not replace the role of clinical evaluation.

In the PRIDE study, among patients with combined pulmonary and heart disease, clinical assessment missed 75% of the diagnosis of CHF whereas NT-proBNP testing gave the correct diagnosis in 88% of these patients.

LV Remodelling
Change in geometry of the left ventricle whereby it dilates, hypertrophies, and becomes more spherical.
The change in chamber size increases the haemodynamic stresses on the walls of the failing heart, and depresses mechanical performance. Mitral regurgitation is also increased. These changes refeed the remodelling.

Consensus Recommendations for the Management of Chronic Heart Failure - Amer J cardiol, Jan 21, 1999 83(2A)

Interplay between cardiac function and neurohumoral and cytokine systems. Myocardial injury, of many etiologies, can depress cardiac function, which in turn causes activation of the sympathoadrenal system (SAS) and the renin-angiotensin-aldosterone system (RAAS) and the elaboration of endothelin, arginine vasopressin (AVP), and cytokines such as tumor necrosis factor (TNF) a. In acute heart failure (left), these are adaptative and tend to maintain arterial pressure and cardiac function. In chronic heart failure (right), they cause maladaptive hypertrophic remodelling and apoptosis, which cause further myocardial injury and impairment of cardiac function.

Harrison’s Textbook of Medicine 15th edit 2001 - Braunwald
Sympathetic activation

\[
\beta_1 \quad \beta_2 \quad \alpha_1
\]

ββββ receptors

Metoprolol

Propranolol

Carvedilol

Cardiotoxicity

Checklist before starting beta-blockade in CHF

- Class II, III or IV symptoms ie 6 or less mets
- On diuretics + ACE inhibitor
- No contraindications
- No acute medical illness
- No physical evidence of fluid retention

CHARM study (Candesarten)
15% reduction in CV death or CHF hospitalisation when ARB added to ACEI-based regimen

Heart Foundation and Cardiac Society of Australia and New Zealand – Guidelines for the prevention, detection and management of chronic heart failure in Australia, 2006”

ARBs “should also be considered for reducing morbidity and mortality in patients with systolic CHF who remain symptomatic despite receiving ACEIs”

Practice point - Drugs to avoid in CHF:

- anti-arrhythmic agents (apart from beta-blockers and amiodarone)
- non-dihydropyridine calcium-channel blockers (verapamil, diltiazem)
- tricyclic antidepressants
- non-steroidal anti-inflammatory drugs and COX-2 inhibitors
- clozapine
- metformin and thiazolidinediones (pioglitazone, rosiglitazone)
- corticosteroids (glucocorticoids and mineralocorticoids)
- tumour necrosis factor antagonist biologicals.

NHF guidelines 2006

Pacemakers - the ugly

Mode Selection Trial (MOST) Effect of Ventricular Pacing on Heart Failure

CRT is delivered by a small device implanted under the skin in the shoulder area (much like a pacemaker). Three leads are inserted through the venous system and advanced to the heart. CRT restores synchronous ventricular contraction, thereby improving pump efficiency.
CARE - HF study
Primary Endpoint
(All-cause Mortality or Unplanned Hosp. for Major CVS Event)

<table>
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<th>OPT</th>
<th>CRT</th>
<th>CRT-D</th>
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HR 0.63 (95% CI 0.51 to 0.77)

No statistical significant heterogeneity in subgroups

Event-free Survival

Days

P < .0001

CRT

Medical Therapy

COMPANION:
% Reductions in Primary and Secondary Endpoints
Reduction Compared With OPT
12-Month Outcomes

OPT versus CRT, OPT versus CRT-D

Primary endpoint (combined all-cause mortality and all-cause hospitalization) 18.6% 19.3%

Secondary endpoint (all-cause mortality) 23.9% 43.4%

Combined all-cause mortality and heart failure hospitalization
35% 39%

CRT = cardiac resynchronization therapy; CRT-D = CRT plus implantable cardioverter defibrillator; OPT = optimal pharmacologic therapy

Echo in CHF Advantages

- Detect preclinical disease
- Determine aetiology of CHF and exclude mimics
- Quantification: systolic and diastolic function, chamber size, LV mass, haemodynamics, valve regurgitation.
- Stress echo: functional status, presence and severity of CAD, perfusion and viability.
- Evaluation of therapy and prognosis

Consensus Recommendations for the Management of Chronic Heart Failure - Amer J cardiol, Jan 21, 1999 83(2A)

Recommendations for diagnostic investigation of CHF
2006 NHF Guidelines

“...All patients with suspected CHF should undergo an echocardiogram to improve diagnostic accuracy and determine the mechanism of heart failure.”

TRAP
Ejection fraction inaccuracies

“Ejection Fraction Measurement Should be Banned”
Feigenbaum ASE meeting 2005
Take Home Messages:
1. The importance of an ordered approach to management
2. Beware diastolic heart failure and get serious with BP, weight control and diabetic control.
3. Consider the concept of remodelling and heart failure prevention when approaching pharmacotherapy and have an ordered (menu) approach to pharmacotherapy
4. Pacemakers - the good and the ugly
5. Beware the clinical traps – oedema, creps, ejection fractions
6. The essential role of echocardiography in management