This Presentation

• Heart Failure Statistics
• Causes of Heart Failure
• Signs and Symptoms of Heart Failure
• Treatment of Heart Failure
• Special Considerations in the Elderly
• What You can Do
• Case Study

Heart Failure in the Elderly

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Heart Failure Frequency and Survival

• Overall prevalence 0.3-2%
  • 3-5% in over 65yo
  • 8-16% in over 75yo
  • 75% of all HF patients are over 75
• Heart failure is a progressive condition with a median survival of ~5years
  • ~10% annual mortality
  • Pump failure, sudden death, recurrent MI
• Heart failure is responsible for ~5% of all adult medical admissions to hospital

What is Heart failure?

A pathophysiological state in which the heart is unable to pump blood at a rate sufficient to meet the needs of the body

• A complex clinical syndrome with typical symptoms (eg, dyspnoea, fatigue) that can occur at rest or on effort that 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 exercise).
• A diagnosis of CHF may be further strengthened by a beneficial clinical response to treatment(s) directed towards amelioration of symptoms associated with this condition.
**Aetiology of Heart Failure**

- The result of chronic cardiovascular disease leading to pump failure
  - Hypertension
  - Coronary artery disease
  - Arrhythmias- esp atrial fibrillation
  - Cardiomyopathy
  - Valvular heart disease

**Risk Factors**

- Tobacco use, sedentary lifestyle, poor diet
- Diabetes, obesity
- Age, genetics

**Pathophysiology of Heart Failure**

- Healthy cardiac output ~ 5L/minute
  - Left Ventricular Ejection Fraction >50%
  - 70 BPM, 70mL stroke volume (130mL Left ventricular volume)

- Reduced cardiac output in heart failure
  - Systolic Heart Failure
  - Reduced LVEF
  - Heart Failure with Preserved Ejection Fraction (HFrEF or Diastolic Heart Failure)
  - LVEF “normal”
    - Definitions vary: >40%, >36%, >50%
    - Note that some studies of HFrEF include treated SHF patients where LVEF is increased to the HFrEF range

**Clinical Manifestations**

- Non-Specific
  - Exercise Intolerance, Fatigue, Weakness, Nocturia, CNS symptoms
  - Tachycardia, Pallor, Cyanosis of digits, Cardiomegaly

- Right Ventricular Failure
  - Abdominal pain, anorexia, nausea, bloating, constipation
  - Peripheral oedema (ankle swelling), Jugular Venous Pressure (JVP) Elevation, Hepatomegaly, Ascites

- Left Ventricular Failure
  - SOBOE, Orthopnoea, Paroxysmal nocturnal dyspnoea (PND), nocturnal cough, tachypnoea (rapid breathing)
  - Crackles, pulmonary oedema, pleural effusion, Cheyne-Stokes respiration (apnoea followed by rapid breathing)

**Classification of Heart Failure**

<table>
<thead>
<tr>
<th>Classification of Heart Failure: ACC/AHA Stage vs NYHA Class</th>
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</thead>
<tbody>
<tr>
<td><strong>ACC/AHA Heart Failure Stage</strong></td>
</tr>
<tr>
<td>A. At risk for heart failure but without structural heart disease or symptoms</td>
</tr>
<tr>
<td>B. Structural heart disease but without heart failure</td>
</tr>
<tr>
<td>C. Structural heart disease with prior or current heart failure symptoms</td>
</tr>
<tr>
<td>D. Refractory heart failure requiring specialized interventions</td>
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</table>

**Estimated 1 year mortality**

- 5-10%
- 15-30%
- 50-60%
ACC-AHA Staging of Heart failure

<table>
<thead>
<tr>
<th>ACC-AHA Stage</th>
<th>NYHA Functional Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>None</td>
</tr>
<tr>
<td>B</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>C</td>
<td>Symptomatic with moderate exertion</td>
</tr>
<tr>
<td>D</td>
<td>Symptomatic with minimal exertion</td>
</tr>
</tbody>
</table>

Natural History of CCF

Mechanism of death:
- Sudden death: 40%
- Worsening CHF: 40%
- Other: 20%

Annual mortality:
- Asymptomatic: <5%
- Mild: 10%
- Moderate: 20% to 30%
- Severe: 30% to 80%

Left ventricular dysfunction and symptoms

Responses to Reduced Cardiac Output

- Extracellular fluid volume
- Cardiac output
- Activation of ventricular and arterial receptors
- Nonosmotic vasopressin stimulation
- Stimulation of sympathetic nervous system
- Activation of the renin-angiotensin-aldosterone system
- Renal water retention
- Peripheral and renal arterial vascular resistance
- Renal sodium retention
- Maintenance of arterial circulatory integrity

Maladaptive Neurohumoral Response in Heart Failure

- ↓ Cardiac Output
- ↑ SNS
- ↓ Renal Perfusion
- ↑ Contractility
- → RAA
- → Na/H2O Retention
- Vasoconstriction
- HR
Diagnosis of Heart Failure

- Symptoms:
  - Dyspnoea, chronic fatigue, oedema, and exercise intolerance.

- Signs:
  - Third or fourth heart sounds, heart murmur, cardiomegaly, pulmonary crackles, raised jugular venous pressure, and dependent oedema.

- Causative factors:
  - Angina, previous myocardial infarction, hypertension, valvular heart disease/rheumatic fever, and cardiomyopathy.

- Patients have possible CHF if they have:
  - > 2 symptoms,
  - > 2 signs,
  - > 1 symptom and > 1 sign, or
  - > 1 symptom and > 1 causative factor.

Conditions that Mimic Heart Failure

- Obesity
- Hypoalbuminaemia
- Intrinsic renal or hepatic disease
- Intrinsc renal or hepatic disease
- Pulmonary embolic disease
- Depression and/or anxiety disorders
- Severe anaemia or thyroid disease
- Bilateral renal artery stenosis

Diagnosis of Heart Failure

- Suspected Heart Failure
- Clinical History
- Physical Examination
- Initial Investigations

- Clinical Heart Failure
- Echocardiogram

Diagnosis of Heart Failure

- Suspected Heart Failure
- Clinical History
- Physical Examination
- Initial Investigations

- Clinical Heart Failure
- Echocardiogram

Clinical diagnosis of CHF

- Echocardiogram

 Structural diagnosis
  (e.g., hypertrophic, valvular)

 Pathophysiological diagnosis
  Systolic dysfunction (LVEF < 40%)
  Diastolic dysfunction (LVEF > 40%)

 Consider specialist referral for further investigation
 Proceed to treatment guidelines
Management of Heart Failure

- Prevention
- Early detection
- Amelioration of disease progression
  - Improvement of stage of disease
- Relief of symptoms
- Minimisation of exacerbations
- Prolongation of survival

Defining the Goal of Treatment

Symptomatic status
- Asymptomatic
- Mild
- Moderate
- Refractory

% survival

- Improve survival
- Halt disease progression
- Improve remodelling
- Prevent complications
- Relieve symptoms

Management of Heart Failure

- Treat reversible causes or exacerbating factors
- Non-pharmacological therapy
- Drug therapy
  - Improve pump function
  - Treatment of major consequences
    - Fluid retention, vasoconstriction, increased sympathetic activity
- Heart transplantation, mechanical devices

Exacerbating Factors

- Inappropriate changes to CHF treatment and/or poor adherence to treatment
- Physical, environmental, and emotional excesses
- Systemic infection or development of related illness
- Pulmonary embolism
- Cardiac infection (e.g. endocarditis) and inflammation (e.g. pericarditis)
- Development of a second form of heart disease (e.g. hypertension or IHD)
- Excessive intake of water and/or sodium
- Administration of cardiac depressants or drugs that cause salt retention
- High output states (e.g. anaemia, thyrotoxicosis, infection, myxoedema, obesity, nutritional deficiencies)
Re-consider drugs that worsen HF

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Agent/drug class</th>
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<tbody>
<tr>
<td>Negative inotropic action</td>
<td>β-blockers (high doses, unstable disease)</td>
</tr>
<tr>
<td></td>
<td>CCBs (nifedipine, verapamil, diltiazem)</td>
</tr>
<tr>
<td></td>
<td>Antiarrhythmics (other than amiodarone)</td>
</tr>
<tr>
<td></td>
<td>Itraconazole, TCAs</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>Cholinergic agents (bethanechol)</td>
</tr>
<tr>
<td></td>
<td>Cholinesterase inhibitors (donepezil, rivastigmine, galantamine)</td>
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<tr>
<td>Vasodilators</td>
<td>CCBs (as above)</td>
</tr>
<tr>
<td></td>
<td>Direct-acting vasodilators (minoxidil, prazosin, terazosin)</td>
</tr>
<tr>
<td>Sodium / water retention</td>
<td>NSAIDs, salicylates (high doses), corticosteroids, glitazones, oestrogens, androgens, lithium, minoxidil, drugs with high sodium content, liquorice</td>
</tr>
<tr>
<td>Direct cardiotoxins</td>
<td>Cardiomyopathies: cytotoxics, cocaine, amphetamines, clozapine, Valvular heart disease: ergot alkaloids, pergolide</td>
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</table>

Pharmacological Treatment of Symptomatic HF

- ACE Inhibitors
- Diuretics
- β-blockers
- ATII Receptor Antagonists
- Spironolactone
- Digoxin
- Drugs for associated conditions

*Drug therapy for cardiac failure is initiated and modified according to the patient’s signs and symptoms.*

Maladaptive Responses Guide Treatment Strategies

- Digoxin
- Beta-Blockers
- ACEIs and other vasodilators
- Spironolactone
- ACEIs
- SNS
- Renal Perfusion
- Contractility
- HR
- Na/H+0 Retention
- RAA

Pharmacological Management of CCF

- Aimed at:
  - Improving cardiac pump function
  - Digoxin, inotropes
  - Reducing workload
  - Rest, weight loss, vasodilators, beta blockers
  - Control of excess Salt and water retention
  - Diuretics, sodium restriction
Treatment of CCF: Digoxin

- Positive inotrope, Negative chronotrope
- Increases LVEF and exercise tolerance
- People on an ACEI with digoxin get worse if digoxin is ceased
- No improvement in mortality in patients given digoxin as well as diuretics and ACEI (DIG Study).
- but reduced admissions to hospital for CHF
- Reduced number of deaths due to worsening heart failure

<table>
<thead>
<tr>
<th>Trial</th>
<th>Ejection fraction</th>
<th>Patients</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main</td>
<td>≤ 0.45</td>
<td>6800</td>
<td>0.69 (0.63-0.76)</td>
</tr>
<tr>
<td>Ancillary</td>
<td>&gt; 0.45</td>
<td>988</td>
<td>0.72 (0.53-0.99)</td>
</tr>
</tbody>
</table>

Note: RR = relative risk, CI = confidence interval.
Data are from GassSmithHaw's fall 2001 prescribing information for Lanoxin digoxin tablets, and Ahmed et al. 12

ACE Inhibitors

- Benefits of ACEIs:
  - delay the onset and progression of heart failure
  - improve signs and symptoms of all grades of heart failure
  - improve exercise tolerance and quality of life
  - improve survival
  - delay the development of heart failure in those with asymptomatic LV dysfunction

- All patients -- not some or most but all patients -- with heart failure due to left ventricular systolic dysfunction should receive an ACE inhibitor, unless they are intolerant of the drug or have a contraindication to its use. Treatment should not be delayed until symptoms are severe or resistant to other drugs.

ACEIs- Adverse Effects

- Adverse Effects
  - Hypotension (esp with diuretics)
  - Renal
    - Dose adjusted for renal dysfunction
    - Acute renal failure in volume depleted patients or patients with bilateral renal artery stenosis
  - <= 20% change in Cr Cl OK
  - Hyperkalaemia
  - Cough
  - Rash/Neutropaenia (Captopril only)
  - Angiooedema
Angiotensin 2 blockers (ATII blockers): the Sartans

- As an alternative to ACEI when side effects are encountered
- Block the effects of angiotensin breakthrough from ACEIs (? Combination)
- Don’t inhibit bradykinin breakdown so reduced incidence of cough

Treatment of CCF: Diuretics

- Reduce symptoms of congestion
  - SOB, Oedema
- Therapeutic Use
  - Thiazide if good renal function, otherwise loop
  - Should be accompanied by salt restriction
  - Combined thiazide/loop effective in diuretic resistance
  - Close monitoring of potassium levels required (may increase sudden death)
  - Spironolactone particularly useful in addition to Loop diuretics

Diuretics

- All patients with symptoms of heart failure who have a predisposition to fluid retention.
  - These drugs should not be used alone, even if they are effective in controlling symptoms.
  - The goal is to eliminate signs and symptoms of fluid retention e.g. Elevated JVP and/or oedema.
  - Measurement of body weight is the best way of monitoring when to initiate and/or titrate a diuretic regimen.
  - Diuretics may alter the efficacy and toxicity of other drugs used to treat heart failure, such as ACE inhibitors and b-blockers.

Diuretics - choice of agent

- Loop diuretics should be commenced at low doses e.g. frusemide 20-40mg mane. Dosage is gradually increased/modified in response to symptoms (but use lowest dose that controls symptoms)
- If large doses of frusemide are required (e.g. > 120mg/day), the addition of a thiazide may have an additive or synergistic effect.
- Severe fluid overload or pulmonary oedema - may require IV therapy or very high oral dose
Frusemide in heart failure

- Diuretics relieve breathlessness and oedema
- Evidence
  - withdrawal in advanced CCF worsens symptoms
  - in mild heart failure, may be withdrawn
  - ? Daily or intermittent use
  - “Mass action” effect may be useful
  - Normal bioavailability~60%, reduced in RHF due to “soggy gut” syndrome
  - Effects diminished by renal failure

Diuretics – monitoring therapy

- Monitoring diuretic therapy for safety, efficacy
  - Daily weights
  - Symptoms - dyspnoea, orthopnoea, oedema
  - Renal function
  - Electrolytes - esp. potassium and magnesium
- Consequences of excessive diuresis
  - Hypotension, esp. orthostatic hypotension
  - Electrolyte disturbances
  - Pre-renal renal failure

Treatment of CCF: Beta Blockers

- Traditional beta-blockers may be useful in patients with IHD worsened CCF (reduce cardiac work)
- Also, post infarction reduced the risk of dying in all patients, even those with heart failure
- Patients with heart failure are at a higher risk of dying, so the absolute benefit of beta blockers is greater in these patients.
- Newer beta-blockers have combined vasodilator effects and improve exercise tolerance, LV function and symptoms of CCF
- These effects evident in non-ischaemic CCF
- Many patients worsen initially

β-blockers

- Traditionally contraindicated in heart failure, but recent trials have shown that in small doses β-blockers may be useful in some types of heart failure, acting by affecting neurohumoral activation and ventricular remodelling.
**β-blockers**

- Large, well-conducted studies have shown that selected beta-blockers improve survival and decrease hospitalisations in stable symptomatic heart failure.
- Overall, beta-blockers reduce the annual mortality rate by an absolute 4.5% (translating to a number-needed to-treat of 22 to prevent one death in a year).
- This survival benefit is in addition to the benefits gained with ACE inhibitors.

**β-blockers**

- Start “low and go slow”

<table>
<thead>
<tr>
<th>Beta-blocker</th>
<th>Starting dose</th>
<th>Titration regimen</th>
<th>Target dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bisoprolol</td>
<td>1.25 mg once daily</td>
<td>Double dose every 2-4 weeks if patient is stable</td>
<td>10 mg once daily</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125 mg twice daily</td>
<td>Double dose every 2-4 weeks if patient is stable</td>
<td>25 mg twice daily</td>
</tr>
<tr>
<td>Metoprolol succinate</td>
<td>23.75 mg once daily</td>
<td>If moderate-severe heart failure: half tablet daily for 1 week then 1 tablet daily for 1 week</td>
<td>190 mg once daily</td>
</tr>
</tbody>
</table>

- Watch for: worsening CCF, bradycardia, hypotension, dizziness.

**Beta blockers in CCF: The evidence**

- Appears likely that patients with symptomless LVD as well as mild or moderate CHF will benefit (CIBIS II, ANZ Carvedilol)
- **Cause an initial exacerbation, so should be avoided in NYHA Class IV patients**
- “Beta blockers should be used with great caution, or not at all in patients who have had a recent exacerbation of heart failure”

**Spironolactone in CCF**

- **RALES study**
  - 1663 patients in class III or IV heart failure randomised to spironolactone 25mg or placebo in addition to other therapy.
Treatment of heart failure

60% of all cases of CHF are due to LV systolic dysfunction.
Hobbs R, Boyle A. Heart failure. Cleveland Medical Clinic 2004
http://www.clevelandclinicmeded.com/disease_management/cardiology/heartfailure/heartfai lure.htm

What You can Do

- Monitor for signs and symptoms
  - In patients with a diagnosis of CCF
  - In patients with a potential cause for CCF
- Monitor for efficacy of treatment
  - Weight/fluid gain/loss
  - Exercise tolerance
  - Peripheral oedema
  - SOB/Orthopnea/PND
- Monitor for Adverse Effects
  - Overdiuresis, electrolyte disorders, renal

Source: A step-wise approach to heart failure management.
NPS News 36, 2004

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Figure 1 - Algorithm for pharmacological management of heart failure due to LV systolic ventricular dysfunction (adapted from the NCC Chronic Heart Failure Guidelines, 2002)