Primary Care Management of Congestive Heart Failure

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Practical Healthcare Solutions, Inc.

Objectives
- Describe the physiology and Pathophysiology of heart failure
- Understand the etiology of heart failure.
- Describe the pharmacologic management of heart failure.
- Describe the non pharmacologic / nursing management of heart failure.
- Identify patient education interventions in the management of chronic heart failure.

Part I: Overview of Heart Failure

Heart Failure (HF) Definition
A clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood

Prevalence of HF by Age and Gender
United States: 1988-94

What is the big deal
The Problem
- HF affects 5,800,000 people in the US
- Results in 6.5 million hospital days/year
- Results in 300,000 deaths (direct and indirect) each year.
- $39 billion in health care cost.
- Incidence has doubled in the last 10 years

Gottdiener, J. JACC 2000; 35:1628
Bueno et al. JAMA 2010, 303(21)
Lloyd-Jones D. et al Circulation 2009
O’Connell J. J Heart Lung Transplant 1993

Prevalence of HF by Age and Gender

Percent of Population

Source: NHANES III (1988-94); CDC/NCHS and the American Heart Association
New York Heart Association Functional Classification

Class I: No symptoms with ordinary activity.

Class II: Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, dyspnea, or angina.

Class III: Marked limitation of physical activity. Comfortable at rest, but less than ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.

Class IV: Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency may be present even at rest.

Stages of Heart Failure

Stage A: At high risk, no structural disease

Stage B: Structural heart disease, asymptomatic

Stage C: Structural heart disease with pronounced symptoms of HF

Stage D: Refractory HF requiring specialized interventions

People with:
- Obesity
- Hypertension
- Elevated Cholesterol
- Diabetes
- Drug users

People with:
- LV Hypertrophy
- Previous MI
- Rheumatic heart disease
- New AV blockage
- Aortic Disease

People with:
- Prior admission to HF with or without symptoms
- NYHA class IV or V

Etiology of Heart Failure

What causes heart failure?
- Ischemic Heart Disease
- Hypertension
- Valvular Disease
- Idiopathic Cardiomyopathy
- Infections (e.g., viral myocarditis)
- Toxins (e.g., alcohol or cytotoxic drugs)
- Prolonged Arrhythmias

Prognosis

Cardiac Mortality %

LVEF

Post MI n=196

Prognosis

Lilly, L. Pathophysiology of Heart Disease. Second Edition p 200

Left Ventricular Dysfunction

Systolic: Impaired contractility/ejection
- Approximately two-thirds of heart failure patients have systolic dysfunction

Diastolic: Impaired filling/relaxation

Part II: Hemodynamics of Heart Failure
Cardiac Output

Cardiac output is the amount of blood that the ventricle ejects per minute

\[ \text{Cardiac Output} = \text{HR} \times \text{SV} \]

Determinants of Ventricular Function

- Contractility
- Preload
- Afterload
- Stroke Volume
- Heart Rate

Cardiac Output

Determinants of Ventricular Function

- Contractility
- Preload
- Afterload
- Stroke Volume
- Heart Rate

Cardiac Output

Left Ventricular Dysfunction

- Volume Overload
- Pressure Overload
- Loss of Myocardium
- Impaired Contractility

LV Dysfunction

\[ \text{EF} < 40\% \]

- ↑ End Systolic Volume
- ↑ End Diastolic Volume
- Hypoperfusion
- Pulmonary Congestion

Compensatory Mechanisms

Ventricular Remodeling

Alterations in the heart’s size, shape, structure, and function brought about by the chronic hemodynamic stresses experienced by the failing heart.

Compensatory Mechanisms

Neurohormonal Activation

Many different hormone systems are involved in maintaining normal cardiovascular homeostasis, including:

- Sympathetic nervous system (SNS)
- Renin-angiotensin-aldosterone system (RAAS)
- Vasopressin (a.k.a. antidiuretic hormone)

Compensatory Mechanisms: Sympathetic Nervous System

- Decreased MAP
- ↑ Contractility
- Tachycardia
- Vasoconstriction

\[ \text{MAP} = (\text{↑SV} \times \text{↑HR}) \times \text{↑TPR} \]
Sympathetic Activation in Heart Failure

- ↑ CNS sympathetic outflow
- ↑ Cardiac sympathetic activity
- ↑ Sympathetic activity to kidneys + peripheral vasculature
- Myocardial toxicity
- Increased arrhythmias
- Vasoconstriction
- Sodium retention
- Disease progression

Compensatory Mechanisms: Renin-Angiotensin-Aldosterone (RAAS)
- Renin
- Angiotensin
- Converting Enzyme
- Angiotensin I
- Angiotensin II
- AT1 receptor
- Vascular remodeling
- Cell Growth
- Proteinuria

Vicious Cycle of Heart Failure

- LV Dysfunction
- Increased cardiac output
- Increased preload and afterload
- Increased blood pressure
- Frank-Starling Mechanism
- Increased cardiac workload

Neurohormonal Responses to Impaired Cardiac Performance

<table>
<thead>
<tr>
<th>Response</th>
<th>Short-Term Effects</th>
<th>Long-Term Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salt and Water Retention</td>
<td>Augments preload</td>
<td>Pulmonary Congestion, Anasarca</td>
</tr>
<tr>
<td>Vasoconstriction</td>
<td>Maintains BP for perfusion of vital organs</td>
<td>Exacerbates pump dysfunction (excessive afterload), increases cardiac energy expenditure</td>
</tr>
<tr>
<td>Sympathetic Stimulation</td>
<td>Increases HR and ejection</td>
<td>Increases energy expenditure</td>
</tr>
</tbody>
</table>

Assessing Heart Failure

- Patient History
- Physical Examination
- Laboratory and Diagnostic Tests
Diagnostic Evaluation of New Onset Heart Failure

Initial Work-up:
- ECG
- Chest x-ray
- Blood work
- Echocardiography

Diagnostic Evaluation of Chronic Heart Failure

- Routine evaluation of LV systolic function
  - Echocardiogram
  - Nuclear Medicine Imaging (MUGA scan)
  - Cardiac Cath
- Routine Laboratory evaluations
  - Renal function
  - Electrolytes
  - CBC
- Routine 12 Lead EKG’s

Part IV: Current Treatment of Heart Failure

The Vicious Cycle of Heart Failure Management

Treatment Objectives

- Survival
- Morbidity
- Exercise capacity
- Quality of life
- Neurohormonal changes
- Progression of CHF
- Symptoms

Treatment
- Prevention. Control of risk factors
- Life style
- Treat etiologic cause / aggravating factors
- Drug therapy
- Personal care. Team work

All

- Revascularization if ischemia causes HF
- ICD (Implantable Cardiac Defibrillator)
- Ventricular resynchronization
- Ventricular assist devices
- Heart transplant
- Artificial heart

Selected patients
Treatment Approach for the Patient with Heart Failure

Stage A
At high risk, no structural disease

- Treat Hypertension
- Treat lipid disorders
- Encourage regular exercise
- Discourage alcohol intake
- ACE inhibition

Stage B
Structural heart disease, asymptomatic

- Therapy
  - All measures under stage A
  - ACE inhibitors in appropriate patients
  - Beta blockers in appropriate patients
  - AICD

Stage C
Structural heart disease with persistent symptoms of HF

- Therapy
  - All measures under stage A & B
  - ACE inhibitors
  - Beta blockers
  - Digitalis
  - Angiotensin receptor blockers
  - Diuretics
  - Dietary salt restriction
  - AICD

Stage D
Refractory HF requiring specialized interventions

- Therapy
  - All measures under stages A, B, and C
  - Mechanical assist devices
  - Heart transplantation
  - Continuous (not intermittent) IV inotropic infusions for palliation
  - Hospice care

Therapy
- • Diuretics
- • ACE inhibitors
- • Beta Blockers
- • Spironolactone
- • Digitalis
- • Other

Diuretics
- Essential to control symptoms secondary to fluid retention
- Prevent progression of heart failure
- Spironolactone improves survival

Loop Diuretics Practical Use
- Start with variable dose. Titrate to achieve dry weight
- Monitor serum K+ at “frequent intervals”
- Reduce dose when fluid retention is controlled
- Teach the patient when, how to change dose
- Combine to overcome “resistance”

Loop diuretics. Dose (mg)

<table>
<thead>
<tr>
<th></th>
<th>Initial</th>
<th>Maximum</th>
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</thead>
<tbody>
<tr>
<td>Bumetanide</td>
<td>0.5 to 1.0 / 12-24h</td>
<td>10 / day</td>
</tr>
<tr>
<td>Furosemide</td>
<td>20 to 40 / 12-24h</td>
<td>400 / day</td>
</tr>
<tr>
<td>Torsemide</td>
<td>10 to 20 / 12-24h</td>
<td>200 / day</td>
</tr>
</tbody>
</table>

Diuretics. Indications
- Symptomatic HF, with fluid retention
  - Edema
  - Dyspnea
  - Lung Rales
  - Jugular distension
  - Hepatomegaly
  - Pulmonary edema (Xray)

AHA / ACC HF guidelines 2009
ESC HF guidelines 2009
Diuretic Resistance
- Neurohormonal activation
- Rebound Na+ uptake after volume loss
- Reduced tubular blood flow (renal failure, NSAIDs)
- Decreased renal perfusion (low output)
- Altered absorption of diuretic
- Noncompliance with drugs

Managing Resistance to Diuretics
- Restrict Na+/H2O intake (Monitor Natremia)
- Increase dose (individual dose, frequency, i.v.)
- Combine: furosemide / spiro / metolazone
- Dopamine (increase cardiac output)
- Reduce dose of ACE-i

ACE-i. Mechanism of Action

ACE-I. Clinical Effects
- Improve symptoms
- Reduce remodeling / progression
- Reduce hospitalization
- Improve survival

Mortality Reduction with ACE-i

ACE-i

CONSENSUS
SOLVD treatment
AIRE
Vheft-II
TRACE
SAVE
SMILE
HOPE

Study

ACE-i
Enalapril
Enalapril
Ramipril
Enalapril
Trandolapril
Captopril
Zotenopril
Ramipril

Clinical Setting
CHF
CHF
CHF
CHF
CHF / LVD
LVD
High risk
High risk

Probability of Death

CONSENSUS
ACE-i

**% Mortality**

- SOLVD (Treatment)

- SOLVD (Treatment) - CHF - NYHA II-III - EF < 35
  - n = 2589

- SAVE
  - n = 2231
  - 3 - 16 days post AMI
  - EF < 40
  - 12.5 - 150 mg / day

- ACE-i - Placebo
  - Enalapril
  - n=1285

- ACE-i - Placebo
  - Captopril
  - n=1115

ACE-i Indications

- Symptomatic heart failure
- Asymptomatic ventricular dysfunction
  - LVEF < 55%
- Selected high risk subgroups

ACE-i Contraindications

- Intolerance (angioedema, anuric renal fail.)
- Bilateral renal artery stenosis
- Pregnancy
- Renal insufficiency (creatinine > 3 mg/dl)
- Hyperkalemia (> 5,5 mmol/l)
- Severe hypotension
- Cough??

ACE-i Practical Use

- Start with very low dose
- Increase dose if well tolerated
- Renal function & serum K+ after 1-2 w
- Avoid fluid retention / hypovolemia (diuretic use)
- Dose NOT determined by symptoms
Angiotensin II Receptor Blockers (ARB)

- Candesartan, Eprosartan, Irbesartan
- Losartan, Telmisartan, Valsartan
- Efficacy not equal / superior to ACE-I
- Not indicated with beta blockers and ACE-I
- Indicated in patients intolerant to ACE-I

Angiotensin II Receptor Blockers (ARB)

\[ \text{Angiotensinogen} \rightarrow \text{Angiotensin I} \rightarrow \text{Angiotensin II} \]

\[ \text{ACE} \]

\[ \text{AT}1 \rightarrow \text{AT}2 \]

\[ \text{AT}1 \rightarrow \text{RECEPTORS} \]

\[ \text{Vasoconstriction} \rightarrow \text{Proliferative Action} \rightarrow \text{Vasodilatation} \rightarrow \text{Antiproliferative Action} \]

\[ \text{RENIN} \]

\[ \text{Other pathways} \rightarrow \text{ANGIOTENSIN II} \]

\( \beta \)-Adrenergic Blockers

Mechanism of action
- \( \downarrow \) Density of \( \beta \), receptors
- Inhibit cardiotoxicity of catecholamines
- \( \downarrow \) Neurohormonal activation
- \( \downarrow \) HR
- Anti-ischemic
- Antihypertensive
- Antiarrhythmic

\( \beta \)-Adrenergic Blockers Clinical Effects
- Improve symptoms (only long term)
- Reduce remodeling / progression
- Reduce hospitalization
- Reduce sudden death
- Improve survival

\( \beta \)-Adrenergic Blockers

\% Survival

- NYHA II-IV
  - Carvedilol (n=598)
  - Placebo (n=398)

\% Mortality

- Placebo
  - Metoprolol

US Carvedilol HF
NEJM 1996; 334: 1349-55

\( \beta \)-Adrenergic Blockers

\% Mortality

- Placebo
  - Metoprolol

\% Risk Reduction

- Risk Reduction 34%

\( \beta \)-Adrenergic Blockers

\% Survival

- Carvedilol (n=598)
- Placebo (n=398)

\% Risk Reduction 65%

\( \beta \)-Adrenergic Blockers

\% Mortality

- Placebo
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\( \beta \)-Adrenergic Blockers

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\% Mortality

- Placebo
  - Metoprolol

\% Risk Reduction

- Risk Reduction 34%
**β-Adrenergic Blockers**

**Indications**
- Symptomatic heart failure
- Asymptomatic ventricular dysfunction
  - LVEF < 55%
- After AMI

**When to start**
- Patient stable
  - No physical evidence of fluid retention
  - No need for i.v. inotropic drugs
- Start ACE-I / diuretic first
- No contraindications
- In hospital or not

**Dose (mg)**

<table>
<thead>
<tr>
<th>Initial</th>
<th>Target</th>
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<tbody>
<tr>
<td>Bisoprolol</td>
<td>1.25 / 24h</td>
</tr>
<tr>
<td></td>
<td>10 / 24h</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125 / 12h</td>
</tr>
<tr>
<td></td>
<td>25 / 12h</td>
</tr>
<tr>
<td>Metoprolol succinate</td>
<td>12.5-25 / 24h</td>
</tr>
<tr>
<td></td>
<td>200 / 24h</td>
</tr>
</tbody>
</table>

- Start Low, Increase Slowly
  - Increase the dose every 2 - 4 weeks

**Adverse Effects**
- Hypotension
- Fluid retention / worsening heart failure
- Fatigue
- Bradycardia / heart block

- Review treatment (+/- diuretics, other drugs)
- Reduce dose
- Consider cardiac pacing
- Discontinue beta blocker only in severe cases
**β-Adrenergic Blockers**

Contraindications

- Asthma (reactive airway disease)
- AV block (unless pacemaker)
- Symptomatic hypotension / Bradycardia
- Diabetes is NOT a contraindication

**Aldosterone Inhibitors**

**Spironolactone**

- Competitive antagonist of the aldosterone receptor (myocardium, arterial walls, kidney)
  - Retention $Na^+$
  - Retention $H_2O$
  - Excretion $K^+$
  - Excretion $Mg^{2+}$

- Collagen deposition
- Fibrosis
  - myocardium
  - vessels

**Spironolactone**

- Do not use if hyperkalemia, renal insuf.
- Monitor serum $K^+$ at “frequent intervals”
- Start ACE-i first
- Start with 25 mg / 24h
- If $K^+ > 5.5$ mmol/L, reduce to 25 mg / 48h
- If $K^+$ is low or stable consider 50 mg / day

**Digoxin**

**Mechanism of Action**

- Blocks $Na^+ / K^+$ ATPase => $Ca^{2+}$
- Inotropic effect
- Natriuresis
- Neurohormonal control
  - ↓ Plasma Norepinephrine
  - ↓ Peripheral nervous system activity
  - ↓ RAAS activity
  - ↑ Vagal tone

**Clinical Effects of Digoxin**

- Improve symptoms
- Modest reduction in hospitalization
- Does not improve survival

**Spironolactone Survival**

N = 1663
NYHA III-IV
Mean follow-up 2 y

RALES
NEJM 1999;341:709

**Annual Mortality**

Aldactone 18%; Placebo 23%

$\text{N} < 0.0001$

NEJM 1988;318:358

NEJM 1988;319:358
**Digoxin Contraindications**

- Digoxin toxicity
- Advanced A-V block without pacemaker
- Bradycardia or sick sinus without PM
- Marked hypokalemia
- W-P-W with atrial fibrillation

**Other Drugs**

- Inotropics: refractory HF
- Nitrates: ischemia, angina, pulmonary congestion
- Antiarrhythmics: High risk arrhythmia
- Anticoagulants: High risk of embolism
- Ca channel blockers: (only amlodipine)
- NSAIDS- Avoid

**NITRATES HEMODYNAMIC EFFECTS**

1- VENOUS VASODILATATION

- Preload

2- Coronary vasodilatation

- Myocardial perfusion

3- Arterial vasodilatation

- Afterload

4- Others

**Other Management Issues**

- Medication Adherence
- Sodium restriction
  - 2-3gms per day
- Nutrition
  - High protein diets
- Vitamins and Herbals
- Sleep Apnea
- Oxygen therapy
- Vaccinations

- Insomnia
- Depression
- Stress
- Smoking cessation
- Alcohol
  - Limit to 1-2 standard drinks per day
- Daily exercise
  - 6 minutes a day
- Sexual Dysfunction

**Other Therapies**

- Outpatient IV Infusions
  - Dobutamine
  - Natrecor
- Ventricular assist devices
- Ventricular reduction Surgery (STICH Trial)
- Transplant
- Cardiac Re-synchronization Therapy (CRT)

**Cardiac Rhythm Management in the Management of Chronic Heart Failure**
Prevalence of Ventricular Dyssynchrony in Heart Failure

Left Bundle Branch Block More Prevalent with Impaired LV Systolic Function

<table>
<thead>
<tr>
<th>Preserved LV Systolic Function (1)</th>
<th>Impaired LV Systolic Function (1)</th>
<th>Moderate/Severe HF (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8%</td>
<td>24%</td>
<td>38%</td>
</tr>
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</table>


Deleterious Effects of Ventricular Dyssynchrony on Survival

Moderate Term—1 Year

<table>
<thead>
<tr>
<th>QRS &lt; 120 ms</th>
<th>QRS &gt; 120 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>34%</td>
<td>49%</td>
</tr>
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</table>

Long Term—45 Months

<table>
<thead>
<tr>
<th>QRS &lt; 120 ms</th>
<th>QRS &gt; 120 ms</th>
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</thead>
<tbody>
<tr>
<td>34%</td>
<td>49%</td>
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</tbody>
</table>

Cardiac Resynchronization Therapy

Goal: Mitigate dyssynchrony through atrial synchronous biventricular pacing
Transvenous approach for left ventricular lead via coronary sinus
Back-up epicardial approach

Clinical Benefits of Cardiac Resynchronization Therapy

Results from Randomized Trials of over 3,000 Patients

- Enhances functional capacity
  - Quality of life
  - NYHA functional class
  - Exercise
- Limits disease progression
  - Reverse remodeling
  - Improved cardiac function
- Effects on mortality and hospitalization summarized

Inclusion Criteria & Status of Randomized Controlled Trials

<table>
<thead>
<tr>
<th>Study (n randomized)</th>
<th>NYHA</th>
<th>QRS</th>
<th>Sinus</th>
<th>ICD</th>
<th>Status</th>
<th>Results</th>
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<td>PATH CHF (41)</td>
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<td>MIRACLE ICD (169)</td>
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<td>CONTAK CD (490)</td>
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<td>PATH CHF II (89)</td>
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<td>Both</td>
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<td>CARE HF (814)</td>
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<td>≥120</td>
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</tbody>
</table>

LVEF ≤ 35% for all trials
* RV paced QRS 1 Primary endpoint not met; key secondary endpoints reached

CRT Improves Quality of Life & Functional Capacity in Moderate to Severe Heart Failure

<table>
<thead>
<tr>
<th>NYHA Class</th>
<th>Proportion Changing 1 or more Classes</th>
<th>Results</th>
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</thead>
<tbody>
<tr>
<td>III, IV</td>
<td>P=0.001</td>
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<tr>
<td>III, IV</td>
<td>P&lt;0.001</td>
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<tr>
<td>III, IV</td>
<td>P=0.017</td>
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<tr>
<td>III, IV</td>
<td>P&lt;0.001</td>
<td>+</td>
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</table>

Data sources:
MIRACLE: Circulation 2003;107:1985-90
MUSTIC (SR): NEJM 2001;344:873-80
MUSTIC AF: JACC 2003;41:217-23
PATH CHF: JACC 2003;41:217-23
MIRACLE ICD: JACC 2003;41:217-23
CONTAK CD: JACC 2003;41:217-23
Cardiac Resynchronization Therapy Improves Exercise Capacity in Moderate to Severe Heart Failure

Data sources:
- MIRACLE, Circulation 2003;107:1985-90
- MUSTIC SR, NEJM 2001;344:613-20
- MIRACLE ICD, JAMA 2003;289:2855-64
- Contak CD, J Am Coll Cardiol 2003;42:1454-59

Severity of Heart Failure

Indications for CRT & CRT-D

Cardiac Resynchronization Therapy: Creating Realistic Patient Expectations

Summary

- Heart failure is a chronic, progressive disease that is generally not curable, but treatable
- Most recent guidelines promote lifestyle modifications and maximization of medical management with ACE inhibitors, beta blockers, diuretics, digoxin, and Aldactone
- Close follow-up of the heart failure patient is essential, with necessary adjustments in medical management


Note: CRT is adjunctive and is not intended to replace medical therapy. Patients will continue to be followed by HF Specialist and Physician managing implantable devices.
Questions?