Congestive Heart Failure:
Comprehensive Practice Guidelines

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HPI

- 20 y/o gentleman, no significant PMEDHx
- 05/14 presentation to Millard Fillmore Suburban Hospital
- CC: Six week history of abdominal pain with nausea, vomiting, and lower extremity edema.
  - had been seen in community hospital in Pennsylvania
  - complained of dizziness with position change
  - DOE
  - PND
  - orthopnea

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Course

- Admitted to ECMC
- Rx: IV furosemide, captopril, digoxin, spironolactone, IV dobutamine
- LVEF 7%, extensive LV thrombus (mobile), mod-severe TR
- Enoxaparin and warfarin initiated
- THC positive
- Discharged 05/20

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Course

- 08/15: VO2 max 15.7 ml/kg/min (37% predicted)
- 08/15: LVEF 8%
- Medical therapy maximized as tolerated
- 02/13: VO2 max 14.5 ml/kg/min (34% predicted)
- 02/13: LVEF 6%
- 03/08: ICD (St. Jude single chamber)
- 04/07: Hospitalized for pulmonary edema; intubated
- 06/22: RHC: RA 21, W 25, CI 1.6
- 08/11: RHC: RA 18, W 23, CI 1.8

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HFSA 2010 Comprehensive Heart Failure Practice Guideline

Key Recommendations

http://www.heartfailureguideline.org/

2013 ACCF/AHA Guideline for the Management of Heart Failure: Executive Summary:

•*J Am Coll Cardiol*. 2013;62(16):1495-1539.

•A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

Prevalence of Heart Failure

- 6 million people affected in the U.S.
- 400,000-700,000 new cases of congestive heart failure (CHF) each year
- HF afflicts 10 out of every 1,000 over age 65 in the U.S.
- By year 2030, estimated 10 million Americans will be affected
- Cost $39.2 billion in 2010
- 2nd only to hypertension as outpatient diagnosis

Definition:

•Abnormality in cardiac function that leads to an inability of the heart to pump blood at a rate commensurate with the metabolic requirements.

•Results in a clinical syndrome or condition characterized by :

a) volume overload

b) manifestations of inadequate tissue perfusion

Does the heart muscle have to be weak?

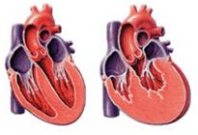
- Systolic: most common; contractile failure
- Diastolic: increased filling pressures required to maintain cardiac output despite normal contractile function

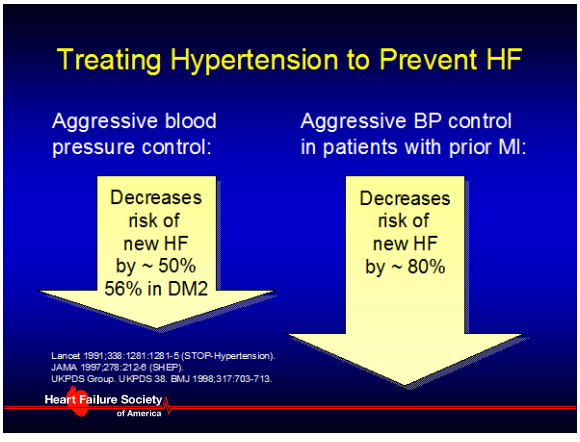
Definition of Heart Failure

Classification	Ejection Fraction	Description
I. Heart Failure with Reduced Ejection Fraction (HF*r*EF)	≤40%	Also referred to as systolic HF. Randomized clinical trials have mainly enrolled patients with HF*r*EF and it is only in these patients that efficacious therapies have been demonstrated to date.
II. Heart Failure with Preserved Ejection Fraction (HF*p*EF)	≥50%	Also referred to as diastolic HF. Several different criteria have been used to further define HF*p*EF. The diagnosis of HF*p*EF is challenging because it is largely one of excluding other potential noncardiac causes of symptoms suggestive of HF. To date, efficacious therapies have not been identified.
a. HF*p*EF, Borderline	41% to 49%	These patients fall into a borderline or intermediate group. Their characteristics, treatment patterns, and outcomes appear similar to those of patient with HF*p*EF.
b. HF*p*EF, Improved	>40%	It has been recognized that a subset of patients with HF*p*EF previously had HF*r*EF. These patients with improvement or recovery in EF may be clinically distinct from those with persistently preserved or reduced EF. Further research is needed to better characterize these patients.

Appropriate Treatment is based on cause of Heart Failure—ASK WHY!

- Coronary Artery Disease
- Idiopathic Dilated Cardiomyopathy
- Hypertension
- Valvular Heart Disease
- Toxic/Drug
- Congenital
- Metabolic
- Other: infiltrative (amyloid, sarcoid) and re





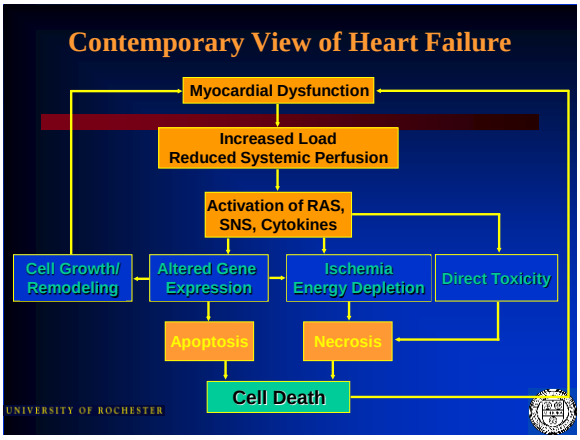
Pathophysiology

•CURRENT CONCEPTS

a) Ventricular Remodeling

b) Neurohumoral and Endocrine Activation

•What the body means to be adaptive initially, becomes maladaptive long term.



Ventricular Remodeling

- ❖ Change in ventricular shape and dimension
- ❖ Regional or global
- ❖ Increased ventricular volume
- ***Changes are occurring at cellular level:***
  - Myocyte hypertrophy, increase intracellular sarcomere
  - Myocyte slippage
  - Myocardial interstitial fibrosis, increased collagen deposition

Neurohormonal Model

- Major components
  - Naturetic Peptide System
    - BNP
  - Sympathetic Nervous System
  - Renin-Angiotensin System
  - Aldosterone

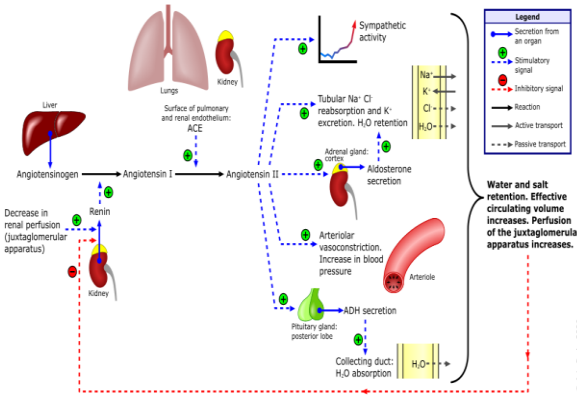
Causes for Elevated Natriuretic Peptide Levels

Cardiac	Noncardiac
• Heart failure, including RV syndromes • Acute coronary syndrome • Heart muscle disease, including LVH • Valvular heart disease • Pericardial disease • Atrial fibrillation • Myocarditis • Cardiac surgery • Cardioversion	• Advancing age • Anemia • Renal failure • Pulmonary causes: obstructive sleep apnea, severe pneumonia, pulmonary hypertension • Critical illness • Bacterial sepsis • Severe burns • Toxic-metabolic insults, including cancer chemotherapy and envenomation

Sympathetic Nervous System

1. Direct Stimulation of RAAS
2. Stimulate Beta 1 to increase contractility
3. Norepinephrine stimulates arteriolar and venous constriction
4. Increase in afterload leads to decreased cardiac output and ventricular performance
5. Increased myocardial oxygen consumption
6. Tachycardia leads to increased consumption and decreased diastolic filling time

Renin-angiotensin-aldosterone system



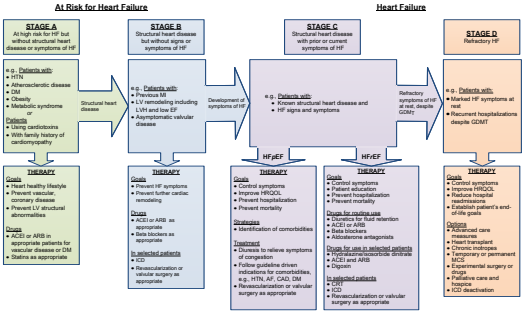
Other bad actors

- Cytokines-depress cardiac function
  - Tumor necrosis alpha: proinflammatory; cardiac cachexia
- Interleukin 6
- Peripheral Changes: Endothelial Derived factors

Key Treatment Paradigm

- Expert HF disease management program
- Excellent Self Care: sodium, weight, compliance
- Pharmacology: ACE/BBlockers
- Mechanical Therapies: AICD/BiV-CRT

Stages, Phenotypes and Treatment of HF



Classification of Heart Failure

ACCF/AHA Stages of HF		NYHA Functional Classification	
A	At high risk for HF but without structural heart disease or symptoms of HF.	None	
B	Structural heart disease but without signs or symptoms of HF.	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.
C	Structural heart disease with prior or current symptoms of HF.	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.
		II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in symptoms of HF.
		III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF.
D	Refractory HF requiring specialized interventions.	IV	Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.

HFSA 2010 Practice Guideline

Evaluation—Patients Suspected of Having HF

Table 4.3. Symptoms Suggesting the Diagnosis of HF	
Symptoms	• Dyspnea at rest or on exertion • Reduction in exercise capacity • Orthopnea • PND or nocturnal cough • Edema • Ascites or scrotal edema
Less specific presentations	• Wheezing or cough • Unexplained fatigue • Early satiety, nausea/vomiting, abdominal discomfort • Confusion/delirium • Depression/weakness (esp. in elderly)

HFSA 2010 Practice Guideline

Evaluation—Patients Suspected of Having HF

Table 4.4. Signs to Evaluate in Patients Suspected of Having HF	
Cardiac Abnormality	**Sign**
↑ cardiac filling pressures and fluid overload	• Elevated jugular venous pressure (JVP) • S3 gallop • Rales • Hepatojugular reflux • Ascites, edema
Cardiac enlargement	• Laterally displaced or prominent apical impulse • Murmurs suggesting valvular dysfunction

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Patient Evaluation

•Recommendation 4.8

•It is recommended that patients with a diagnosis of HF undergo evaluation as follows (Table 4.6):

- **Assess clinical severity of HF by history and physical examination**
- **Assess cardiac structure and function**
- **Determine the etiology of HF**
- **Evaluate for coronary disease and myocardial ischemia**
- **Evaluate the risk of life-threatening arrhythmia**
- **Identify any exacerbating factors for HF**
- **Identify co-morbidities which influence therapy**
- **Identify barriers to adherence and compliance**

Strength of Evidence = C

HFSA 2010 Practice Guideline

Initial Evaluation—ECG

•Recommendation 4.13 Electrocardiogram

•It is recommended that all patients with HF have an ECG performed to:

- **Assess cardiac rhythm and conduction (in some cases, using Holter monitoring or event monitors)**
- **Assess electrical dyssynchrony (wide QRS or bundle branch block) especially when LVEF < 35%**
- **Detect LV hypertrophy or other chamber enlargement**
- **Detect evidence of myocardial infarction or ischemia**
- **Assess QTc interval, especially with drugs that prolong QT int.**

Strength of Evidence = B

Cardiopulmonary Exercise Testing

"VO2 Max"
- normal
- athletes

mL O2/kg/min

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1 of 2

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Patient Education

Recommendation 8.1

It is recommended that patients with HF and their family members or caregivers receive individualized education and counseling that emphasizes self-care.

This education and counseling should be delivered by providers using a team approach in which nurses with expertise in HF management provide the majority of education and counseling, supplemented by physician input and, when available and needed, input from dietitians, pharmacists and other health care providers.

Strength of Evidence = B

Heart Failure Society of America

Lindenfeld J, et al. HFSA 2010 Comprehensive Heart Failure Guideline. J Card Fail 2010;16:e1-e194.

HFSA 2010 Practice Guideline

Patient Education

Recommendation 8.2

It is recommended that patients' literacy, cognitive status, psychological state, culture, and access to social and financial resources be taken into account for optimal education and counseling.

Because cognitive impairment and depression are common in HF and can seriously interfere with learning, patients should be screened for these.

Patients found to be cognitively impaired need additional support to manage their HF.

Strength of Evidence = B

Heart Failure Society of America

Lindenfeld J, et al. HFSA 2010 Comprehensive Heart Failure Guideline. J Card Fail 2010;16:e1-e194.

HFSA 2010 Practice Guideline

Nonpharmacologic—Dietary Sodium

•Recommendation 6.2

•Dietary sodium restriction (2-3 g daily) is recommended for patients with the clinical syndrome of HF and preserved or depressed LVEF.

- **Further restriction (< 2 g daily) may be considered in moderate to severe HF.**

Strength of Evidence = C

HFSA 2010 Practice Guideline

Nonpharmacologic—Fluid Intake

•Recommendation 6.3

•Restriction of daily fluid intake to < 2 liters:

- **Is recommended in patients with severe hyponatremia (serum sodium < 130 mEq/L)**
- **Should be considered for all patients demonstrating fluid retention that is difficult to control despite high doses of diuretic and sodium restriction.**

Strength of Evidence = C

HFSA 2010 Practice Guideline

Nonpharmacologic—Nutrition in Advanced HF

- *Recommendation 6.4*
- **It is recommended that specific attention be paid to nutritional management of patients with advanced HF and unintentional weight loss or muscle wasting (cardiac cachexia).**
  - **Measurement of nitrogen balance, caloric intake, and prealbumin may be useful in determining appropriate nutritional supplementation.**
- **Caloric supplementation is recommended.**
- **Anabolic steroids are not recommended for cachexic patients.**

Strength of Evidence = C

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Nonpharmacologic—CPAP

•Recommendation 6.7

•Continuous positive airway pressure to improve daily functional capacity and quality of life is recommended in patients with HF and obstructive sleep apnea documented by approved methods of polysomnography.

Strength of Evidence = B

HFSA 2010 Practice Guideline

Nonpharmacologic—Oxygen

•Recommendation 6.8

•Supplemental oxygen, either at night or during exertion, is not recommended for patients with HF in the absence of an indication due to underlying pulmonary disease.

•Patients with resting hypoxemia or oxygen desaturation during exercise should be evaluated for residual fluid overload or concomitant pulmonary disease.

Strength of Evidence = B

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Nonpharmacologic—Sexual Dysfunction

•Recommendation 6.12

•It is recommended that treatment options for sexual dysfunction be discussed openly with both male and female patients with HF.

•The use of phosphodiasterase-5 (PDE5) inhibitors such as sildenafil may be considered for use for sexual dysfunction in patients with chronic stable HF.

- **These agents are not recommended in patients taking nitrate preparations.**

Strength of Evidence = C

HFSA 2010 Practice Guideline

Nonpharmacologic—Depression

•Recommendation 6.10

•It is recommended that screening for endogenous or prolonged reactive depression in patients with HF be conducted following diagnosis and at periodic intervals as clinically indicated.

•For pharmacologic treatment, selective serotonin receptor uptake inhibitors (SSRIs) are preferred over tricyclic antidepressants, because the latter have the potential to cause ventricular arrhythmias, but the potential for drug interactions should be considered.

Strength of Evidence = B

HFSA 2010 Practice Guideline

Nonpharmacologic—Smoking & Alcohol

- *Recommendation 6.13*
- **It is recommended that patients with HF be advised to stop smoking and to limit alcohol consumption to ≤ 2 standard drinks per day in men or ≤ 1 standard drink per day in women.**
- **Patients suspected of having an alcohol-induced cardiomyopathy should be advised to abstain from alcohol consumption.**
- **Patients suspected of using illicit drugs should be counseled to discontinue such use.**

Strength of Evidence = B

HFSA 2010 Practice Guideline

Nonpharmacologic—Vaccinations

•Recommendation 6.14

•Pneumococcal vaccine and annual influenza vaccination are recommended in all patients with HF in the absence of known contraindications.

Strength of Evidence = B

HFSA 2010 Practice Guideline

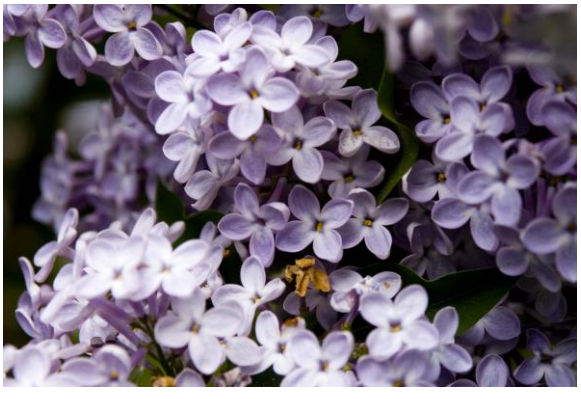
Nonpharmacologic—NSAIDs

•Recommendation 6.16

•NSAIDs, including COX-2 inhibitors, are not recommended in patients with chronic HF.

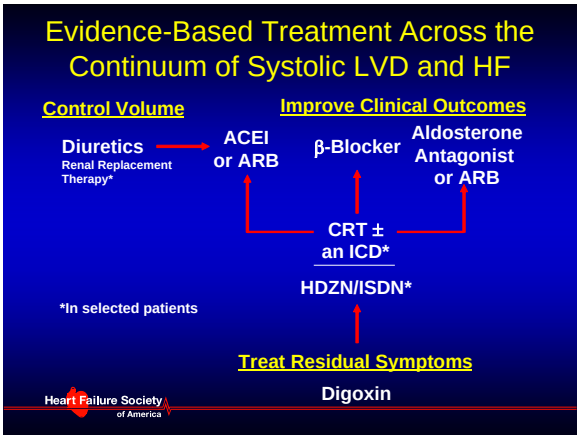
- **The risk of renal failure and fluid retention is markedly increased in the setting of reduced renal function or ACE inhibitor therapy.**

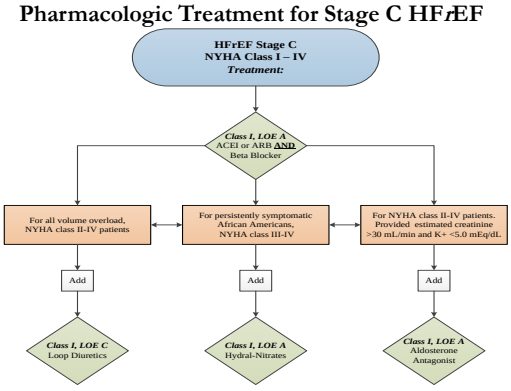
Strength of Evidence = B



Pharmacology

1. ACE Inhibitors/ Angiotensin Receptor Blockers
2. Beta Blockers
3. Aldosterone Inhibition
4. Digoxin
5. Others: Hydralazine/Nitrates, amiodarone
6. Diuretics





ACE Inhibitors

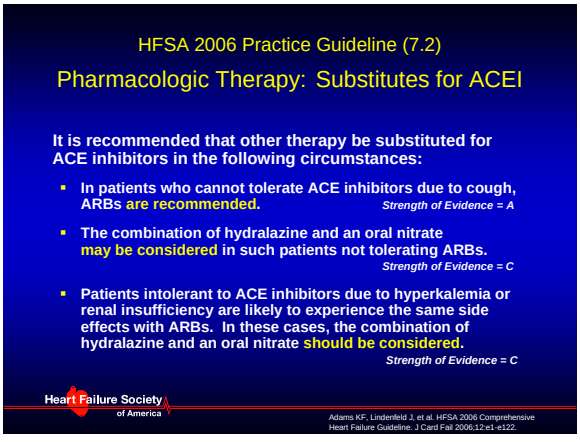
•ACE inhibitors interfere with the RAAS by inhibiting the enzyme responsible for the conversion of angiotensin I to angiotensin II.

• lisinopril, altace, enalapril, captopril

•Monitor for hypotension, renal failure, hyperkalemia, cough and angioedema

ACE Inhibitors Used in Clinical Trials

Generic Name	Trade Name	Initial Daily Dose	Target Dose	Mean Dose in Clinical Trials
Captopril	Capoten	6.25 mg tid	50 mg tid	122.7 mg/day
Enalapril	Vasotec	2.5 mg bid	10 mg bid	16.6 mg/day
Fosinopril	Monopril	5-10 mg qd	80 mg qd	N/A
Lisinopril	Zestril, Prinivil	2.5-5 mg qd	20 mg qd	4.5 mg/day, 33.2 mg/day*
Quinapril	Accupril	5 mg bid	80 mg qd	N/A
Ramipril	Altace	1.25-2.5 mg qd	10 mg qd	N/A
Trandolapril	Mavik	1 mg qd	4 mg qd	N/A



Beta-Adrenergic Receptor Blockers

- Interfere with the actions of the endogenous neurohormonal system, inhibiting the effects of the SNS
- Beta1, Beta1 and 2, and Beta1, 2 and alpha1
- Only 3 agents approved for use in HF: Toprol XL, Coreg, Zebeta
- Monitor for hypotension, fluid retention, fatigue, heart rhythm and sexual function
- **Patient education extremely important with this therapy

Effect of Beta Blockade on Outcome in Patients With HF and Post-MI LVD

Study	Drug	HF Severity	Target Dose (mg)	Outcome
US Carvedilol[1]	carvedilol	mild/ moderate	6.25-25 BID	↓48% disease progression (p= .007)
CIBIS-II[2]	bisoprolol	moderate/ severe	10 QD	↓34% mortality (p <.0001)
MERIT-HF[3]	metoprolol succinate	mild/ moderate	200 QD	↓34% mortality (p = .0062)
COPERNICUS[4]	carvedilol	severe	25 BID	↓35% mortality (p = .0014)
CAPRICORN[5]	carvedilol	post-MI LVD	25 BID	↓23% mortality (p =.031)

1. Colucci WS et al. Circulation 1196;94:2800-6.
2. CIBIS II Investigators. Lancet 1999;353:9-13.
3. MERIT-HF Study Group. Lancet 1999;353:2001-7.
4. Packer M et al. N Engl J Med 2001;3441651-8.
5. The CAPRICORN Investigators. Lancet 2001;357:1385-90.

Heart Failure Society of America

Beta Blockers Used in Clinical Trials

Generic Name	Trade Name	Initial Daily Dose	Target Dose	Mean Dose in Clinical Trials
Bisoprolol	Zebeta	1.25 mg qd	10 mg qd	8.6 mg/day
Carvedilol	Coreg	3.125 mg bid	25 mg bid	37 mg/day
Carvedilol	Coreg CR	10 mg qd	80 mg qd	
Metoprolol succinate CR/XL	Toprol XL	12.5-25 mg qd	200 mg qd	159 mg/day

HFSA 2010 Practice Guideline (7.11)

Pharmacologic Therapy: Beta Blockers

- SYMPTOMATIC EXACERBATION
- Continuation of beta blocker therapy is recommended in most patients experiencing a symptomatic exacerbation of HF during chronic maintenance treatment, unless they develop cardiogenic shock, refractory volume overload, or symptomatic bradycardia.
  Strength of Evidence = C
  - Temporary dose reduction may be considered
  - Avoid abrupt discontinuation
  - Reinstate or gradually increase prior to discharge
  - Titrate dose to previously tolerated dose as soon as possible

HFSA 2006 Practice Guideline (7.6)

Pharmacologic Therapy: Beta Blockers

CONCOMITANT DISEASE

Beta blocker therapy is recommended in the great majority of patients with LV systolic dysfunction—even if there is concomitant diabetes, chronic obstructive lung disease or peripheral vascular disease.

- Use with caution in patients with:
  - Diabetes with recurrent hypoglycemia
  - Asthma or resting limb ischemia.
- Use with considerable caution in patients with marked bradycardia (<55 bpm) or marked hypotension (SBP < 80 mmHg).
- Not recommended in patients with asthma with active bronchospasm.

Strength of Evidence = C

Heart Failure Society of America

Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

HFSA 2006 Practice Guideline

Pharmacologic Therapy: Beta Blocker Overview*

General considerations	Initiate at low doses
	Up-titrate gradually, generally no sooner than at 2 week intervals
	Use target doses shown to be effective in clinical trials
	Aim to achieve target dose in 8-12 weeks
	Maintain at maximum tolerated dose
If symptoms worsen or other side effects appear	Adjust dose of diuretic or concomitant vasoactive med.
	Continue titration to target after symptoms return to baseline
If up-titration continues to be difficult	Prolong titration interval
	Reduce target dose
	Consider referral to a HF specialist

* Consult language of specific recommendations

Heart Failure Society of America

Adapted from: Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

HFSA 2006 Practice Guideline (7.10)

Pharmacologic Therapy: Angiotensin Receptor Blockers

ARBs are recommended for routine administration to symptomatic and asymptomatic patients with an LVEF ≤ 40% who are intolerant to ACE inhibitors for reasons other than hyperkalemia or renal insufficiency.

Strength of Evidence = A

Heart Failure Society of America

Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

Angiotensin Receptor Blockers Used in Clinical Trials

Generic Name	Trade Name	Initial Daily Dose	Target Dose	Mean Dose in Clinical Trials
Candesartan	Atacand	4-8 mg qd	32 mg qd	24 mg/day
Losartan	Cozaar	12.5-25 mg qd	150 mg qd	129 mg/day
Valsartan	Diovan	40 mg bid	160 mg bid	254 mg/day

HFSA 2010 Practice Guideline (7.14-7.15)

Pharmacologic Therapy: Aldosterone Antagonists

- **An aldosterone antagonist is recommended for patients on standard therapy, including diuretics, who have:**
  - **NYHA class IV HF (or class III, previously class IV) HF from reduced LVEF (≤ 35%)**
- **One should be considered in patients post-MI with clinical HF or diabetes and an LVEF < 40% who are on standard therapy, including an ACE inhibitor (or ARB) and a beta blocker.**

Strength of Evidence = A

HFSA 2006 Practice Guideline (7.16-7.18)

Aldosterone Antagonists and Renal Function

Aldosterone antagonists are not recommended when:

- **Creatinine > 2.5mg/dL (or clearance < 30 mL/min)**
- **Serum potassium> 5.0 mmol/L**
- **Therapy includes other potassium-sparing diuretics**

Strength of Evidence = A

It is recommended that potassium be measured at baseline, then 1 week, 1 month, and every 3 months

Strength of Evidence = A

Supplemental potassium is not recommended unless potassium is < 4.0 mmol/L

Strength of Evidence = A

Heart Failure Society of America

Adapted from: Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

HFSA 2006 Practice Guideline (7.19)

Pharmacologic Therapy: Hydralazine and Oral Nitrates

A combination of hydralazine and isosorbide dinitrate is recommended as part of standard therapy, in addition to beta-blockers and ACE-inhibitors, for African Americans with LV systolic dysfunction:

- **NYHA III or IV HF** ***Strength of Evidence = A***
- **NYHA II HF** ***Strength of Evidence = B***

Heart Failure Society of America

Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

HFSA 2006 Practice Guideline (7.23)

Loop Diuretics

Agent	Initial Daily Dose	Max Total Daily Dose	Elimination: Renal – Met.	Duration of Action
Furosemide	20-40mg qd or bid	600 mg	65%R-35%M	4-6 hrs
Bumetanide	0.5-1.0 mg qd or bid	10 mg	62%R/38%M	6-8 hrs
Torsemide	10-20 mg qd	200 mg	20%R-80%M	12-16 hrs
Ethacrynic acid	25-50 mg qd or bid	200 mg	67%R-33%M	6 hrs

Heart Failure Society of America

Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive

HFSA 2006 Practice Guideline (7.23)

Potassium-Sparing Diuretics

Agent	Initial Daily Dose	Max Total Daily Dose	Elimination	Duration of Action
Spironolactone	12.5-25 mg qd	50 mg	Metabolic	48-72 hrs
Eplerenone	25-50 mg qd	100 mg	Renal, Metabolic	Unknown
Amiloride	5 mg qd	20 mg	Renal	24 hrs
Triamterene	50-75 mg bid	200 mg	Metabolic	7-9 hrs

Heart Failure Society of America

Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

HFSA 2010 Practice Guideline

Diuretics

Recommendation 7.24

Oral torsemide may be considered in patients in whom poor absorption of oral medication or erratic diuretic effect may be present, particularly those with right-sided HF and refractory fluid retention despite high doses of other loop diuretics.

Strength of Evidence = C

Intravenous administration of diuretics may be necessary to relieve congestion. *Strength of Evidence = A*

Diuretic refractoriness may represent patient nonadherence, a direct effect of diuretic use on the kidney, or progression of underlying cardiac dysfunction.

Heart Failure Society of America

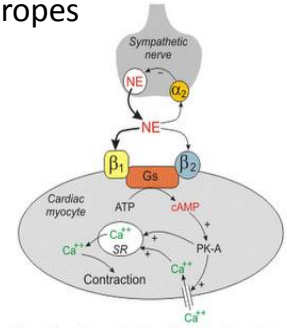
Lindenfeld J, et al. HFSA 2010 Comprehensive Heart Failure Guideline. J Card Fail 2010;16:e1-e194.

Digoxin

- not a first line drug
- DIG Trial: little effect on survival
- assists with heart rate control in atrial fibrillation

Positive Inotropes

- Mechanism
  - ↑ cAMP → ↑ Ca influx → ↑ contractility
- Available agents
  - Dobutamine
  - Milrinone
  - Dopamine



Abbreviations: NE, norepinephrine; Gs, G-stimulatory protein; PK-A, cAMP-dependent protein kinase; SR, sarcoplasmic reticulum

Dobutamine

- β_1 & β_2 agonist
  - Inotropy & vasodilation
- Effects
  - ↑ CI, ↑ SV, ↓ SVR, ↓ PCWP
  - Intravascular depletion: ↓ BP & ↑ HR
- Dosing
  - Initial infusion dose: 2-5 mcg/kg/min
  - Maintenance: 2-20 mcg/kg/min
  - May need higher dose if on outpatient β-blocker
- Monitoring
  - Heart rate/ECG
  - MAP
  - Electrolytes

Milrinone

- Phosphodiesterase inhibitor Type III
  - Increases cAMP by inhibiting the conversion of cAMP to AMP
  - Inotropy & vasodilation
- Results: ↑ CI, ↓ SVR, ↓ PCWP
  - May lead to hypotension & tachycardia
- Dosing
  - Maintenance: 0.25-0.75 mcg/kg/min
  - Consider lower initial dose in renal dysfunction

Milrinone

- Caution
  - Arrhythmias
  - Thrombocytopenia
  - **$T_{1/2}$ ~2-2.5 hrs→ Risk of ADEs persists after infusion stopped**
- Monitoring
  - Blood pressure
  - Heart rate
  - ECG/Arrhythmias
  - Electrolytes
  - Platelet count

Guidelines for Outpatient Milrinone Infusion at URMC

- Only adjust rate with large weight changes after discharge
- Labwork
- If single lumen cath: peripheral labs. Do not interrupt infusion
- Normal saline flushes ONLY, no heparin in any patient that is bridging to either transplant or LVAD: Heparin Induced Thrombocytopenia can be life threatening and will eliminate patient's candidacy for transplant!!
- No rotation of lines if double lumen cath; avoid routine interruption
- VS Parameters/Troubleshooting of line patency
- Weekly dressing change
- If PICC pulls back, okay to run as deep peripheral temporarily
- Heplock if line DC'd

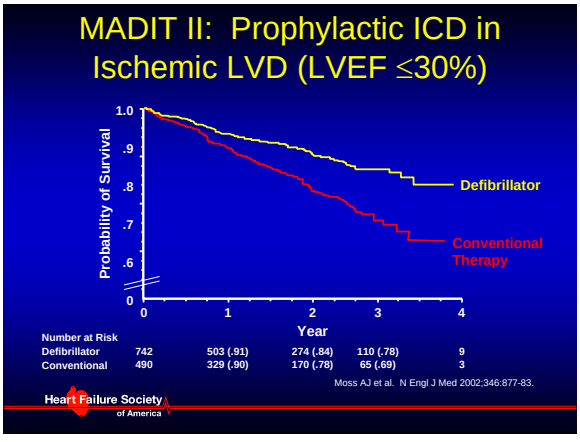
PARADIGM-HF LCZ696

- Oral, BID
- ARNI: Angiotensin Receptor Neprilysin Inhibitor (Valsartan/Neprilysin combo)
- Inc. Natriuetic Peptide and Suppress RAAS
- 09/11/2014 NEJM

HFSA 2010 Practice Guideline (9.1, 9.4)

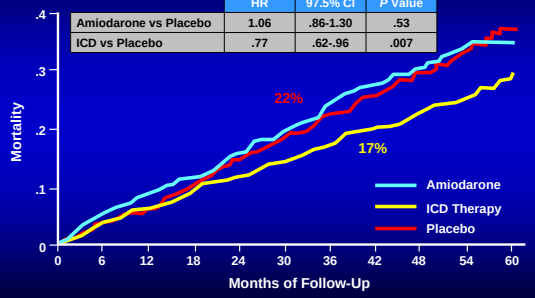
Device Therapy: Prophylactic ICD Placement

- **Prophylactic ICD placement should be considered in patients with an LVEF ≤35% and mild to moderate HF symptoms:**
  - **Ischemic etiology** *Strength of Evidence = A*
  - **Non-ischemic etiology** *Strength of Evidence = B*
- **In patients who are undergoing implantation of a biventricular pacing device, use of a device that provides defibrillation should be considered.** *Strength of Evidence = B*
- **Decisions should be made in light of functional status and prognosis based on severity of underlying HF and comorbid conditions, ideally after 3-6 mos. of optimal medical therapy.** *Strength of Evidence = C*



ICD Therapy in the SCD-HeFT Trial: Mortality by Intention-to-Treat

	HR	97.5% CI	*P* Value
Amiodarone vs Placebo	1.06	.86-1.30	.53
ICD vs Placebo	.77	.62-.96	.007



Bardy GH et al. N Engl J Med 2005;352:225-37.

HFSA 2010 Practice Guideline

Biventricular Pacing

•Recommendation 9.7

•Biventricular pacing therapy is recommended for patients with all of the following:

- **Sinus rhythm**
- **A widened QRS interval (≥120 ms)**
- **Severe LV systolic dysfunction (LVEF ≤ 35%)**
- **Persistent, moderate to severe HF (NYHA III) despite optimal medical therapy.**

Strength of Evidence = A

HFSA 2006 Practice Guideline (8.7)

Heart Failure Disease Management

Patients recently hospitalized for HF and other patients at high risk should be considered for referral to a comprehensive HF disease management program that delivers individualized care.

Strength of Evidence = A

Heart Failure Society of America

Adapted from: Adams KF, Lindenfeld J, et al. HFSA 2006 Comprehensive Heart Failure Guideline. J Card Fail 2006;12:e1-e122.

Clinical Events and Findings Useful for Identifying Patients With Advanced HF

Repeated (≥2) hospitalizations or ED visits for HF in the past year
Progressive deterioration in renal function (e.g., rise in BUN and creatinine)
Weight loss without other cause (e.g., cardiac cachexia)
Intolerance to ACE inhibitors due to hypotension and/or worsening renal function
Intolerance to beta blockers due to worsening HF or hypotension
Frequent systolic blood pressure <90 mm Hg
Persistent dyspnea with dressing or bathing requiring rest
Inability to walk 1 block on the level ground due to dyspnea or fatigue
Recent need to escalate diuretics to maintain volume status, often reaching daily furosemide equivalent dose >160 mg/d and/or use of supplemental metolazone therapy
Progressive decline in serum sodium, usually to <133 mEq/L
Frequent ICD shocks

HFSA 2010 Practice Guideline (8.13)

End-of-Life Care in Heart Failure

- **End-of-life care should be considered in patients who have advanced, persistent HF with symptoms at rest despite repeated attempts to optimize pharmacologic, device, and other therapies, as evidenced by one or more of the following:**
  - **HF hospitalization** *Strength of Evidence = C*
  - **Chronic poor quality of life with inability to accomplish activities of daily living** *Strength of Evidence = C*
  - **Need for continuous IV inotropic therapy support** *Strength of Evidence = C*

HFSA 2010 Practice Guideline

End-of-Life Care

- *Recommendation 8.16 (NEW in 2010)*
- **It is recommended that, as part of end-of-life care, patients and their families/caregivers have a plan to manage a sudden decompensation, death, or progressive decline.**
- **Inactivation of an implantable defibrillation device should be discussed in the context of allowing natural death at end of life. A process for deactivating defibrillators should be clarified in all settings in which patients with HF receive care.**

Strength of Evidence = C

Thank you for your attention!

- Please do not hesitate to contact the Program in Advanced Heart Failure and Transplantation for additional questions or if you wish to discuss a specific patient scenario.
  - 585-273-3760