Heart Failure: Trends and Treatment

Cardiovascular Boot Camp

Presented By:
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www.cardionursing.com

Definition

- Heart Failure is a complex clinical syndrome resulting from any structural or functional cardiac disorder impairing the ability of the ventricle to either fill or eject.
Clinical Syndrome Resulting from Clinical Manifestations

- Dyspnea and fatigue
  - May limit exercise tolerance
  AND / OR
- Fluid overload
  - May lead to pulmonary congestion and peripheral edema

Impaired functional capacity and quality of life

Causes

- Majority of heart failure patients have *impaired left ventricular function*

- Clinical syndrome of heart failure can also result from disease of the pericardium, myocardium, endocardium, valves and great vessels
Etiology of Impaired Left Ventricular Function

- Coronary Artery Disease
  - Ischemic Heart Disease
- Hypertension
- Valve Disease
- Thyroid Disease
- Viral
- Alcohol Abuse
- Idiopathic (no known cause)
- Advancing Age

Systolic Dysfunction

- Impaired wall motion and ejection
- Dilated chamber
- 2/3 of HF Population
- Hallmark: Decreased LV Ejection Fraction < 40%
- Coronary artery disease is cause in 2/3 of patients
- Remainder – other causes of LV dysfunction

Cardiomyopathy not synonymous with HF
Diastolic Dysfunction

- Filling impairment
- Normal chamber size
- 20 to 40% of patients with HF have preserved LV function
- Normal EF or elevated
- Caused by
  - Hypertension
  - Restrictive myopathy
  - Ischemic heart disease
  - Ventricular hypertrophy
  - Valve disease
  - Idiopathic

Primarily disease of elderly women with HTN

Diastolic Dysfunction

- Diagnosis is made when rate of ventricular filling is slow
- Elevated left ventricular filling pressures when *volume and contractility are normal*

In practice:
The diagnosis is made when a patient has typical signs and symptoms of heart failure and has a normal or elevated ejection fraction with no valve disease.
Left and Right Sided Heart Failure

- Reflects signs/symptoms that develop as each respective ventricle fails
- Two sides of the heart form a circuit, neither side can pump significantly more blood than the other for long

Left-Sided Heart Failure

- Decrease in CO/stroke volume
- Pressure rises in the LV, LA, and pulmonary vasculature
- Hydrostatic forces can cause intracellular fluid to accumulate in the pulmonary capillary bed (pulmonary congestion)
- Signs/symptoms result from elevated pulmonary pressures (SOB/paroxysmal nocturnal dyspnea)
Right-Sided Heart Failure

- Typically follows LS failure
- Isolated RV failure (COPD, pulmonary hypertension)
- Pressure increases in the RS of heart
- Hydrostatic pressures force more blood into the systemic venous circulation
- Causing neck vein distention, peripheral edema, weight gain, engorgement of hepatic and gastric vessels

Pathophysiology

- Complex process involving continually emerging symptoms and deterioration
- Myocardial dysfunction initially results from any number of triggers
- Compensatory mechanisms help, then harm
Pathophysiology: The Real Culprit = Neurohormonal Response

- Three significant events occur
  1. Sympathetic Nervous System (SNS) stimulation
  2. Activation of the Renin-Angiotensin-Aldosterone System (RAAS)
  3. Ventricular Remodeling

Activation of SNS

- First Responder
  - Decreased CO $\rightarrow$ ↓ BP $\rightarrow$ activates baroreceptors and vasomotor regulatory centers in medulla

- Increase circulating catecholamines
  - Stimulates alpha and beta receptors
    - Increase HR
    - Peripheral vasoconstriction
    - Contractility

Positive effect: ↑ CO and BP
Negative effect: ↑ O2 demand $\rightarrow$ ischemia, arrhythmias, sudden death
Chronic Stimulation of SNS

- Norepinephrine (circulating catecholamine) is cardiotoxic
  - Decreases heart’s ability to respond to sympathetic stimulation
  - Down regulation of B1 receptor sites (less sensitive)
  - Contributes to decreased exercise tolerance
  - Can also lead to ventricular remodeling

Activation of RAAS

- Kidney’s response to decreased perfusion due to decreasing CO
- Concentrations of angiotensin II, and aldosterone rise as end result
  - Potent vasoconstriction
  - Sodium/water absorption increases
- Result = enhanced preload and afterload
Harmful Result of RAAS Activation

- Enhanced preload increases end-diastolic volume dilating the LV
- LV becomes overstretched
- LV changes size and shape (ventricular remodeling)
- Contractility decreases
- Congestive symptoms develop

Vasopressin Release

- Release of Vasopressin
  - Antidiuretic Hormone
  - Response to angiotensin II
  - Reabsorption of water and additional vasoconstriction
Endothelin

- Endogenous hormone
  - Released in response to angiotensin II
  - Vasoconstriction
  - Fluid Retention
  - Increase Contractility
  - Hypertrophy

Cytokines

- Release causes inflammatory response
- Promotes cell growth (hypertrophy)
- Promotes cell death (apoptosis)
- Contributes to ventricular remodeling
HF as Progressive Disorder

- Initial injury or stress on myocardium
- Change in geometry of left ventricle
  - Dilates
  - Hypertrophies
  - Becomes more spherical
- Decreases mechanical performance of LV and increases regurgitation thru mitral valve
- These effects sustain and enhance the remodeling process

Ventricular Remodeling

- Process of pathological growth
- Can occur from prolonged activation of SNS/RAAS
- Involves
  - Hypertrophy of myocytes
  - Pressure – thicken (concentric)
  - Volume – elongate (eccentric)
  - Genetically abnormal – inefficient contraction
  - Increased ventricular muscle mass, change in ventricular shape
  - Collagen matrix becomes fibrotic
Apoptosis (a component of remodeling)

- Preprogrammed cell death without inflammation/scarring (necrosis)
- Process is accelerated in HF but in a random pattern
- Cell slippage
  - Bricks – myocytes
  - Morter – collagen
  - Degredation (slippage) or Fibrotic

Diagnosing Heart Failure – The Good Guys: Natriuretic Peptides

- Cardiac hormones secreted by myocytes
  - **Atrial natriuretic peptide (ANP)**
    - Produced in atria
  - **Brain natriuretic peptide (BNP)**
    - Produced in ventricles in response to increased ventricular pressure/stretching
    - Stronger release than ANP
- Promote vasodilatation (preload/afterload reduction)
- Reduce sodium/water retention (diuretic response)
- Reduce production/action of vasoconstrictor peptides
- Plasma concentrations elevated in patients in fluid overload
- Nesiritide (Natrecor) is the synthetic form of BNP
## Stages of Heart Failure
### ACC / AHA

<table>
<thead>
<tr>
<th>Stage A</th>
<th>Stage B</th>
<th>Stage C</th>
<th>Stage D</th>
</tr>
</thead>
<tbody>
<tr>
<td>At high risk for HF but without structural heart disease or symptoms of HF. HPTN CAD DM Obesity Metabolic syndrome Family HX CM</td>
<td>Structural heart disease but without signs or symptoms of Heart Failure</td>
<td>Structural heart disease with prior or current symptoms of HF.</td>
<td>Refractory HF requiring specialized interventions.</td>
</tr>
<tr>
<td></td>
<td>Previous MI LV Remodeling including LVH and low EF Asymptomatic valvular disease</td>
<td>Know structural disease and SOB, fatigue, reduced exercise tolerance.</td>
<td>Marked symptoms of HF at rest despite maximal medical therapy.</td>
</tr>
</tbody>
</table>

## Classification of Heart Failure
### New York Heart Association

<table>
<thead>
<tr>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac disease no resulting limitation in physical activity. Ordinary activity free of fatigue, palpitation, dyspnea or anginal pain.</td>
<td>Cardiac disease with slight limitation of physical activity.</td>
<td>Cardiac disease with marked limitation on physical activity.</td>
<td>Cardiac disease resulting in inability to carry out any physical activity without discomfort. May have symptoms of cardiac insufficiency at rest.</td>
</tr>
<tr>
<td></td>
<td>Comfortable at rest but ordinary activity results in fatigue, palpitations, dyspnea, or anginal pain.</td>
<td>Comfortable at rest but less than ordinary activity results in fatigue, palpitations, dyspnea, or anginal pain.</td>
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</tbody>
</table>
Diagnosing Heart Failure

- **History**
  - Causative diseases
  - Related diseases
  - Family history
  - Symptoms
- **Physical Examination**
- **Diagnostic Testing**
  - Echocardiogram
  - CXR
  - EKG
  - Heart catheterization
  - Stress testing
  - Lab Work

Diagnosing Heart Failure - Symptoms

- Exercise intolerance (hallmark)
  - Fatigue
  - Dyspnea
- Paroxysmal nocturnal dyspnea
- Frequent night urination with less during the day
- Peripheral edema/weight change
- Chest Pain
- GI problems
- Confusion/altered mental status
Diagnosing Heart Failure -
Symptoms

- Symptoms in the elderly
  - Many don't experience exertional dyspnea because they are sedentary
  - More common:
    - Daytime oliguria/nocturia
    - Mental disturbances
    - Anorexia
    - GI disturbances

Diagnosing Heart Failure –
Physical Exam

- General Appearance (resting dyspnea, cyanosis, cachexia)
- Weight gain
- BP/HR
- JVD
- Displaced apical impulse
- S3/S4
- Lung sounds
- Hepatojugular reflux
- Edema
Diagnostic Testing

- Lab work
  - Serum electrolytes
  - Blood urea nitrogen/creatinine
  - Liver function tests
  - CBC
  - Thyroid function tests
  - BNP Level

BNP Lab Test

- Useful to differentiate “SOB”
- High negative predictive value (<100 pg/ml we know it is not HF)
- Must be used with assessment
- May be elevated for other reasons
### Disease Management Strategies

- Continuum of Care
- Multidisciplinary System
- Evidence Based Guidelines
- Case Management with Physician Direction
- Focus on Patient Education

### Stages to Guide Therapy

<table>
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<tr>
<th>Stage A</th>
<th>Stage B</th>
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</table>
| • Treat HTN  
  • Treat DM  
  • Smoking Cessation  
  • Treat Lipids  
  • Regular Exercise  
  • DC Alcohol / Drug Use  
  • ACE-I in select patients | • All measures as stage A  
  • ACE-I in select patients  
  • Beta Blockers in select patients  
  • Implantable defibrillators | • All measures under stage A  
  • Dietary salt restriction  
  • Daily Weight  
  • Medications for routine use: Diuretics  
  • ACE – I  
  • Beta-Blockers  
  • Medications in select patients: Digitalis  
  • Aldosterone Antagonists  
  • Devices in select patients  
  • Resynchronization Therapy  
  • Implantable defibrillators | • All measures under A, B and C  
  • Mechanical assist  
  • Transplantation  
  • Palliative Care  
  • Hospice |

33

34
Medication – Ace-Inhibitors

- Definitive evidence of mortality/morbidity reduction
- Inhibits conversion of angiotensin I to II
- Interferes with ventricular remodeling
- Slows disease progression
- Used for mortality benefit not symptom relief
- May need to adjust diuretic dose and monitor K+
- Renal function and ACE-I
- Cough / angioedema
- Use of ARB or hydralazine/isordil
- ACE Inhibitor suffix: “pril”
  - Captopril, enalapril, lisinopril, perindopril, ramipril, trandolapril

Medications - ACE Inhibitors

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<th>Stage A</th>
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<tbody>
<tr>
<td>In patients at high risk for developing or</td>
<td>All pts. with recent or remote history of</td>
<td>Class I recommendations</td>
<td>Same as Stage C</td>
</tr>
<tr>
<td>history of atherosclerotic vascular disease,</td>
<td>MI regardless of EF or presence of HF.</td>
<td>Stage A/B (IA,B,C)</td>
<td></td>
</tr>
<tr>
<td>DM or HPTN. (IIA)</td>
<td>(IA)</td>
<td></td>
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<tr>
<td></td>
<td>All pts. reduced EF and no symptoms of HF.</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>(IA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Beneficial in pts with HPTN &amp; LVH with no HF</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>symptoms. (IIB)</td>
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Medications

Angiotensin Receptor Blockers

- ACE Inhibitors remain the first choice for inhibition of RAAS
- ARB’s are a reasonable alternative to ACE Inhibitor if intolerant to ACE Inhibitor due to cough or angioedema
- Directly block angiotensin II
- Combination of ACE I and ARB
- Reasonable alternative to ACE I as 1st line therapy for patients with mild / moderate HF & reduced LVEF, especially if already take ARB for other reason (HTN)
- ARB Suffix: “sartan”

Medication – Beta Blockers

- Decrease mortality/hospitalization
- Even better in combination with ACE Inhibitor
- Enhances overall well being
- Slows disease progression
- Inhibits ventricular remodeling and apoptosis
- Inhibits adverse effects of SNS
- Decrease myocardial oxygen consumption
- When to initiate?
- Not all beta blockers are equal
  – Metoprolol, bisoprolol, carvedilol
- Beta blocker suffix: “olol”
**Medications**

**Beta-Blockers**

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<tbody>
<tr>
<td>All pts. with recent or remote history of MI regardless of EF or presence of HF. (IA)</td>
<td>All pts. reduced EF and no symptoms of HF, (IA)</td>
<td>Class I recommendations Stage A/B (IA,B,C)</td>
<td>Same as Stage C</td>
</tr>
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**Medication - Diuretics**

- **Goal** = decrease congestive symptoms
- **Added for fluid overload** – Stage C
- **Loop diuretics preferred**
  - Thiazide ok with overloaded HTN patients
- **Keep K >4 and Mg >2**
- **Consider adding NA restriction as well**
Medication - Digoxin

- Stage C
  - Added in patients with persistent symptoms already on ACE Inhibitor, Beta-blocker and diuretic
- Positive inotropic effect – weak effect
- Enzyme inhibition in noncardiac tissues – reduces sympathetic flow
- Improved symptoms, exercise tolerance and quality of life
- No reduction in mortality
- Beta-blocker better for rate control
- Low dose: 0.125mg daily
- No need for loading dose

Medications
Aldosterone Antagonists

- Stage C and D
- New York Heart Association Class IV, systolic dysfunction
  - EF <35%
- Already on ACE I, Beta Blocker, Diuretics, Digoxin
- Reduce death and rehospitalizations
- Spironolactone (RALES Trial)
  - Survival benefit in NYHA Functional Class 3 or 4 HF
- Epleranone
- High risk for HYPERkalemia
  - Do not initiate in patients with elevated creatinine (M>2.5 mg/dL; W>2.0 mg/dL) or elevated potassium (>5.0 mEq/L)
**Medication - Other**

- **Warfarin** recommended if HF and a fib
- **Antiarrhythmics**
  - ICD preferred if had VF or unstable VT
  - Amiodarone for symptomatic ventricular arrhythmias and/or not ICD candidate
- **Avoid:**
  - NSAIDS
  - Avandia

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**Newer / Future Agents**

- **Direct Renin Inhibitors**
  - Aliskiren (Tekturna)
  - Currently used as antihypertensive
- **Vasopressin 2 Antagonists**
  - Vasopressin 1 (vascular)
  - Vasopressin 2 (renal collecting ducts)
  - Vasopressin 3 (pituitary)
  - Tolvaptan – in addition to standard IV therapy in acute decompensated HF
- **Adenosine A1 Receptor Antagonists**
  - Investigational
  - Enhances response to diuretics
Medications for Diastolic Dysfunction

- Probably the same for systolic dysfunction
- Calcium Channel Blockers
  - Control of BP
  - Decrease HR (better fill)
  - Control atrial fib
  - Calcium Channel Blockers Suffix: “pine"
  - **Worsens systolic dysfunction**
  - Amlodipine ok with systolic dysfunction

Acute Decompensated Heart Failure

<table>
<thead>
<tr>
<th>Warm and Dry</th>
<th>Warm and Wet</th>
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<tbody>
<tr>
<td>Normal Perfusion</td>
<td>Normal Perfusion</td>
</tr>
<tr>
<td>No Congestion</td>
<td>Congestion</td>
</tr>
<tr>
<td><strong>Cold and Dry</strong></td>
<td><strong>Cold and Wet</strong></td>
</tr>
<tr>
<td>Low Perfusion</td>
<td>Low Perfusion</td>
</tr>
<tr>
<td>No Congestion</td>
<td>Congestion</td>
</tr>
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Assessment of Skin and Lungs
Fluid Overload vs. Hypoperfusion

- **Fluid Overload**
  - Weight gain
  - Peripheral edema
  - Jugular venous distention
  - SOB
  - Crackles in lungs

- **Hypoperfusion**
  - Narrow pulse pressure
  - Resting tachycardia
  - Cool Skin
  - Altered mentation
  - Decreased urine output
  - Increased BUN/Creatinine
  - Cheyne Stokes Respirations

Treatment for Acute Decompensated Heart Failure

- **Congestion with adequate perfusion**
  - Reduce Preload

- **Hypoperfusion with no congestion**
  - Inotropic support
    - Assure adequate preload
    - Assure decreased afterload
    - Increase contractility
Treatment for Acute Decompensated Heart Failure

#1 Reduce Preload

#2 Reduce Afterload

#3 Increase Contractility

## Reduce Preload
- Diuretics
- Venous vasodilators
  - Low dose Nitroglycerin
  - Neseritide
- Ultrafiltration

## Reduce Afterload
- Arterial vasodilators
  - High dose Nitroglycerin
  - Nitroprusside
  - Neseritide
- Intra aortic balloon pump

## Increase Contractility
- Positive Inotropes
  - Dobutamine
  - Milronone
Cardiac Resynchronization Therapy

- Treatment modality for heart failure not just pacing
- Treatment modality in conjunction with drug therapy
- Goals:
  - Improve hemodynamics by restoring synchrony of ventricular contraction
  - Improve quality of life
  - Decrease mortality and morbidity

Normal Ventricular Depolarization

Mitral valve closed to prevent regurgitation
Septum moves leftward and functions as part of LV to eject blood
Papillary muscles contract with LV
Ventricular Depolarization with LBBB

A
Abnormal electrical activation

B
Reduced ejection of blood into aorta
Septum bulges into RV

Mitral regurgitation
Abnormal mechanical function

Cardiac Resynchronization Therapy
Cardiac Resynchronization Therapy

- Significant reduction in all-cause mortality - 25%
- Number needed to treat = 27
- Survival benefits seen after 3 months
- Significant reduction in hospitalizations - 32%
- Improved 6 minute walk tests
- Improvement by at least one NYHA class
- Clinical improvement in quality of life
- Improved ejection fraction
- Improved peak oxygen consumption

Indications for CRT

- Stage C
- NYHA Class III
- EF< 35%
- NSR
- QRS duration > 120 ms
- Optimal medical therapy
  - ACE Inhibitor
  - Beta Blocker
  - Diuretic
Implantable Cardiovertor Defibrillator - Indications

- Asymptomatic (NYHA Class I) LV dysfunction AND
  - EF < 30% and prior MI ≥ 40 days
  - EF 31-35% and NSVT, CAD, prior MI, and inducible / sustained VT by EPS

- NYHA II-III LV Dysfunction
  - EF ≤ 35%

Not recommended in Stage D

ICD Functions

- ATP-Antitachycardia Pacing
- Cardioversion Shock
- Defibrillating Shock
Intra Aortic Balloon Pump

- Intra Aortic Balloon (IAB) is inflated during diastole and deflated during systole
- The IAB is a volume displacement device
- Placement
  - Descending thoracic aorta
  - 1 to 2 cm below the subclavian artery origin
  - Above renal and mesenteric arteries

Hemodynamic Impact of IAB Counterpulsation

- Decreased systolic aortic pressure
- Decreased afterload
  - Decreased MVO2
- Decreased LV wall tension
- Decreased preload
  - Decreased pulmonary congestion
- Decreased HR
Implantable Hemodynamic Monitoring

- Record hemodynamic pressures
- Resembles a pacemaker
- Continuously stores date and records real-time beat to beat signals
- External pressure reference calibrates to barometric pressure
- Data downloads via telephone

Left Ventricular Assist Devices

- Profound failure
  - Mean blood pressure < 60 mmHg,
    Systolic blood pressure < 90 mmHg,
    Cardiac index < 2.0 L/min/m²
- Temporary replacement of pumping function of the left ventricle
- Blood diverted from LA and LV to the LVAD
- Blood returned to the aorta
- Continuous flow vs. pulsatile flow
- Portability

Figure 1: The implantable hemodynamic monitor. The Chronic implantable hemodynamic monitor is an investigational device, limited by United States law to investigational use.

Illustration courtesy of Medtronic, Inc.

Figure 2: Courtesy of World Heart, Inc. Oakland, CA
Other Attempts at Treatment

- Continuous Renal Replacement Therapy
- Enhanced External Counterpulsation
- Gene Therapy
- Partial Left Ventriculectomy
- Endoventricular Patch
- Dynamic Myoplasty
- Myosplint
- Acorn Cardiac Support Device

Exercise Training

*Exercise training should be considered for all stable outpatients with chronic HF who are able to participate in protocols needed to produce physical conditioning. Exercise training should be used in conjunction with drug therapy.*
Refractory End-Stage HF

- **Stage D**
  - Prognosis: Survival and Function
  - Transplant
  - Chronic Intermittent Inotropic Support
  - Compassionate End-of Life Care
    - Advanced Directives
    - Hospice Referrals
    - Palliative Care

The Best Treatment
Patient Education & Self-Care Management

- What does the patient need to know to care for themselves at home?
- Patient education is a clinical practice recommendation!
- SURVIVAL skills
Barriers to Self-Care Management

- Lack of knowledge
- Literacy
- Multiple medications
- Fear of medication side effects
- Living alone (lack of social support)
- Memory problems
- Higher acuity
- Multiple needs (comorbidities)
- Shorter LOS
- Noncompliance
- Transportation
- Financial

Who should be involved?

- Patient
- Family/support system
- Physicians
- Dietician
- Case Manager/Social Service
- Heart Failure Nurses
Self-Care Concepts

- Daily management
- Education should focus less on disease process and etiologies
- Key areas to teach
  (CORE MEASURES)
  - Prognosis of heart failure
  - Signs/symptoms: Daily weights
  - Low sodium diet
  - Medications
  - Activity
  - Follow up

Signs & Symptoms

- Focus on changes!
  - Is there a change in their activity tolerance?
  - Impact on their ADL's

Criteria for physician notification
Daily Weights

- Use same scale
- Same amount of clothing
- Empty bladder and before breakfast
- 2 pounds in one day or 4 pounds in a week
- Mark on their calendar
- Many patients don’t call because they feel “OK”, but fluid increase can be a first sign of impending decompensation
- Decrease fluid intake
- Decrease sodium intake

Low Sodium Diet

- Discuss how sodium impacts fluid retention
- Salt = sodium
- Focus on what they can eat
- What about the salt shaker?
- High sodium foods (majority of sodium intake is already in foods)
Medications

- Need to know trade/generic names
- Don’t wait until discharge!
- Are the discharge instructions clear/legible/lay terms used?
- Create a schedule
- Be careful of “meds as at home”
- Understanding of what medications are for – not everything is for blood pressure
- Alternatives for routine schedule
  - Lasix at 4pm
  - ACE Inhibitor at night

Medications

- Include the family
- What system is used at home?
- Compliance history
  - Financial concerns
  - Wallet card
  - Need to understand they feel better because of taking their meds
  - Need to understand the progress made with HF management
  - Need to understand the importance of not running out of their medication
  - Regular follow-up with physician
Core Measures

- Discharge Instructions
  - Prognosis of heart failure
  - Pharmacotherapy
  - Dietary Restrictions
  - Activity Restrictions
  - S&S of Deterioration
  - Follow Up Plan

- LV Assessment for EF
- ACE-I or ARB for Left Ventricular Dysfunction
- Smoking Cessation Counseling

JCAHO Standards for Certification

- Patient Education
  - Diet
  - Weight Monitoring
  - Activity Level
  - Medications
  - Symptom Management

- Blood pressure control < 140/90
- 90 day readmit rate after ED visit for HF
QUESTIONS??

Thanks for Attending
Cardiovascular Boot Camp

You may contact us at www.cardionursing.com