Cardiomyopathies

- Primary disorder of cardiac muscle causing abnormal myocardial performance
- Not the result of disease or dysfunction of nonmuscular cardiac structures
- Excludes:
  - Myocardial infarction
  - Hypertension
  - Valvular Disease

Cardiomyopathies

- Hypertrophic
- Dilated (ischemic and nonischemic)
- Stressed Induced (Takotsubo)
- Restrictive *

Hypertrophic Cardiomyopathy (HCM)

- Myocardial hypertrophy without the presence of associated hemodynamic stress (no ↑ in afterload)
- Hypertrophy of the heart muscle including the septum and ventricular free wall
- Previously called IHSS – idiopathic hypertrophic subaortic stenosis
- Leading cause of death in athletics < 35 y/o

Hypertrophic Obstructive Cardiomyopathy (HOCM)

- Subgroup of patients with HCM develop obstruction
- Once obstruction occurs it is called: Hypertrophic obstructive cardiomyopathy (HOCM)
Dilated Cardiomyopathy

- Enlarged, dilated cardiac chamber
- Can affect one or all four chambers
- As chamber enlarges, its ability to contract becomes impaired, resulting in systolic dysfunction
- Most common cause of HF

Dilated Cardiomyopathy

<table>
<thead>
<tr>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
</tr>
<tr>
<td>Valvular</td>
</tr>
<tr>
<td>Genetic</td>
</tr>
<tr>
<td>Ischemic</td>
</tr>
<tr>
<td>Hypertensive</td>
</tr>
<tr>
<td>Myocarditis (Infection/inflammatory)</td>
</tr>
<tr>
<td>Chemotherapy</td>
</tr>
<tr>
<td>Periparum syndrome related to toxemia</td>
</tr>
<tr>
<td>Cardiotoxic effects of drugs or alcohol</td>
</tr>
</tbody>
</table>

Idiopathic Dilated Cardiomyopathy (IDC)

<table>
<thead>
<tr>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>50% of IDC is familial</td>
</tr>
<tr>
<td>Suspected when other causes are excluded</td>
</tr>
<tr>
<td>CAD</td>
</tr>
<tr>
<td>Thyroid disease</td>
</tr>
<tr>
<td>Valvular abnormalities</td>
</tr>
<tr>
<td>Infiligrative causes</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Alcohol</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitors</td>
</tr>
<tr>
<td>Beta blockers</td>
</tr>
<tr>
<td>Anticoagulation is required due to the risk of thromboembolism</td>
</tr>
<tr>
<td>Improvement of LV function is often better in IDC than in patients with IDM (ischemic dilated cardiomyopathy)</td>
</tr>
</tbody>
</table>

Ischemic Dilated Cardiomyopathy (IDM)

<table>
<thead>
<tr>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most common type of dilated cardiomyopathy</td>
</tr>
<tr>
<td>Occurs when CAD or ischemic heart disease causes remodeling of the LV with an associated reduction of EF</td>
</tr>
<tr>
<td>Remodeling is the compensatory response of the ventricles to improve its function.</td>
</tr>
<tr>
<td>- Harms ventricular muscle</td>
</tr>
<tr>
<td>- Worsens stroke volume</td>
</tr>
<tr>
<td>- Develops ventricular dilation</td>
</tr>
<tr>
<td>- Decreases EF</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitors</td>
</tr>
<tr>
<td>Beta blockers</td>
</tr>
<tr>
<td>Diuretics/spironolactone</td>
</tr>
<tr>
<td>Anticoagulation is required due to the risk of thromboembolism</td>
</tr>
<tr>
<td>ICD</td>
</tr>
<tr>
<td>Amiodarone to prevent dysrhythmias</td>
</tr>
<tr>
<td>Monitor electrolytes</td>
</tr>
<tr>
<td>Prognosis is worse for IDM than nonischemic cardiomyopathy.</td>
</tr>
</tbody>
</table>

Hypertensive Dilated Cardiomyopathy

<table>
<thead>
<tr>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosed when systolic function remains depressed despite adequate treatment of hypertension</td>
</tr>
<tr>
<td>Myocardial systolic function is depressed out of proportion to the increase in wall stress</td>
</tr>
<tr>
<td>Prognosis is influenced by other comorbidities</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Same as IDM (ischemic)</td>
</tr>
<tr>
<td>Afterload reduction is the most important goal</td>
</tr>
<tr>
<td>Antihypertensive vasodilators</td>
</tr>
<tr>
<td>Amiodipine (Norvasc, bisoprol, metoprol or maleata)</td>
</tr>
<tr>
<td>Alpha–blocking agents</td>
</tr>
<tr>
<td>Alfuzosin (Uroxatral)</td>
</tr>
<tr>
<td>Doxazosin (Cardura)</td>
</tr>
<tr>
<td>Prazosin (Minipress)</td>
</tr>
<tr>
<td>Terazosin</td>
</tr>
<tr>
<td>Tamulosin (Flomax)</td>
</tr>
</tbody>
</table>

Afterload reduction - Alpha–blocking agents

Treat ischemic disease - prevent remodeling with ACEI & BB
### Valvular Dilated Cardiomyopathy

**Causes**
- Myocardial systolic function is depressed out of proportion to the increase in wall stress secondary to valvular abnormalities
- Most caused by left sided valves
  - MR & AR
  - AS less common cause
- Nitrates with peripartum than during the last trimester of pregnancy
- With AR – calcium channel blockers

**Treatment**
- Valve replacement or repair – improves wall stress but not depressed LVF
- ACEI & BB
- Aggressive afterload reduction
  - Hydralazine
  - Nitrates
  - nitroglycerin, isosorbide dinitrate, isosorbide mononitrate.

Valve repair – Afterload reduction: hydralazine & nitrates

### Peripartum Dilated Cardiomyopathy

**Causes**
- Occurs when myocardial systolic dysfunction occurs during the last trimester of pregnancy or within 6 months of childbearing.
- Outcomes are better with peripartum than with other dilated cardiomyopathies
- Treatment is aggressive & consistent with IDC (idiopathic)
- 50% will recover completely
- Small minority will need transplant

### Alcohol–Related Dilated Cardiomyopathy

**Causes**
- Diagnosed when there is a history of sustained and heavy alcohol consumption and other causes of dilated cardiomyopathy are excluded.
- Toxic effects of alcohol are thought to cause the nonspecific changes in the myocardium
- Thiamine deficiencies can compromise cardiac function

**Treatment**
- Alcohol abstinence
- Same as for IDC (idiopathic)
- Prognosis is somewhat better than for IDC depending on the degree of myocardial impairment and alcohol abstinence

### Restrictive Cardiomyopathy (RC)

**Causes**
- Restrictive filling and reduced diastolic volume of either or both ventricles
- Least common cardiomyopathy (5%)

### Cardiomyopathy vs Heart Failure

<table>
<thead>
<tr>
<th>Cardiomyopathy</th>
<th>Heart Failure</th>
</tr>
</thead>
</table>
| Primary disorder of cardiac muscle causing abnormal myocardial performance | Complex clinical syndrome
| Heterogeneous group of diseases of the myocardium. | Develops from any cardiac disorder that impairs the ability of the ventricle to fill or eject adequately
| Disease that affects primarily the myocardial layer | Pathologic state in which the heart is unable to pump enough oxygenate blood to meet the metabolic needs of the body
| Associated with mechanical and/or electrical dysfunction | Usually exhibits ventricular hypertrophy or dilation
| Usually leads to progressive heart failure | Often leads to heart failure

**Cardiomyopathy**
- Primary disorder of cardiac muscle causing abnormal myocardial performance
- Heterogeneous group of diseases of the myocardium.
- Disease that affects primarily the myocardial layer
- Associated with mechanical and/or electrical dysfunction
- Usually exhibits ventricular hypertrophy or dilation
- Often leads to progressive heart failure

**Heart Failure**
- Complex clinical syndrome
- Develops from any cardiac disorder that impairs the ability of the ventricle to fill or eject adequately
- Pathologic state in which the heart is unable to pump enough oxygenate blood to meet the metabolic needs of the body
Heart Failure

- Syndrome preceded by an initiating cardiovascular event (MI, hypertension, etc)
- On the cardiac continuum HF is an end event – represents the most severe manifestation of cardiovascular disease

Heart Failure

<table>
<thead>
<tr>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Arises from alterations in systolic and diastolic dysfunction</td>
</tr>
<tr>
<td>- Systolic Dysfunction</td>
</tr>
<tr>
<td>- Diastolic Dysfunction</td>
</tr>
<tr>
<td>2. Systolic and Diastolic Dysfunction are progressive syndromes that develop over the course of many years</td>
</tr>
<tr>
<td>3. Heart Failure preferred term</td>
</tr>
<tr>
<td>- Not all HF patients (especially those with diastolic dysfunction) exhibit symptoms of congestion</td>
</tr>
</tbody>
</table>

HF is a progressive syndrome ---- develops over many years

Acute Exacerbation vs Chronic HF

<table>
<thead>
<tr>
<th>Acute HF</th>
<th>Chronic HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Also called: Decompensated HF</td>
<td></td>
</tr>
<tr>
<td>New or worsening signs and symptoms of the HF syndrome</td>
<td></td>
</tr>
<tr>
<td>Frequently leads to ED visits or hospitalization</td>
<td></td>
</tr>
<tr>
<td>May also be: Sudden onset of HF signs and symptoms that occur in patients with no previous HF history</td>
<td></td>
</tr>
<tr>
<td>Denotes the slow progression and continuance of the HF syndrome</td>
<td></td>
</tr>
<tr>
<td>Chronic HF patients frequently experience “exacerbations of HF” also known as acute HF or decompensated HF</td>
<td></td>
</tr>
</tbody>
</table>

Etiology of Heart Failure

What causes heart failure?

- The loss of a critical quantity of functioning myocardial cells after injury to the heart due to:
  - Ischemic Heart Disease
  - Hypertension
  - Idiopathic Cardiomyopathy
  - Infections (e.g., viral myocarditis, Chagas’ disease)
  - Toxins (e.g., alcohol or cytotoxic drugs)
  - Valvular Disease
  - Prolonged Arrhythmias

Pathophysiology of HF

- Myocardial Injury
  - Fail in LV Performance
- Myocardial Toxicity
  - Change in Gene Expression
  - ANP
  - BNP
- Peripheral Vasodilation
  - Sodium/Water Retention
- Remodeling and Progressive Worsening of LV Function
- HF Symptoms


4/9/2013
Compensatory Mechanisms

- End result of body's attempt to compensate...
  Ventricular Remodeling

Standard Testing for HF

- Echocardiogram
- Lab: BNP, CMP, CBC, TSH, Cardiac enzymes
- Chest X-ray
- Coronary angiogram (left heart catheterization)

An echocardiogram is a procedure used to visualize the pumping action of the heart. The EF is calculated by measuring the ventricle in systole and diastole as noted with the blue lines.

Ejection Fraction

- The amount of blood leaving the heart with each contraction.
- The amount ejected is measured as a fraction of the total amount of blood in the heart at the beginning of contraction.
- Normal is 55–75%.
  - EF < 40% = needs ACE I/ARB
  - EF < 30% = poor outcomes
  - EF < 18% = transplant

BNP Test (Brain Natriuretic Peptide)

- Measures concentration of BNP in blood
- BNP increases in response to LV dysfunction
- Normal = 38 ± 4
- Screen for HF
  - If BNP > 80 – 100 have HF
  - If BNP < 80 = respiratory problem
- Monitor effects of medication
  - BNP Greater than 400 admit to hospital

BNP

- BNP is manufactured in the ventricles
- BNP is a natural hormone that is released in response to a distended, overloaded ventricle to maintain normostasis.
- BNP counters the RAAS system
- The more compensated the heart is the more the RAAS is activated.
- The more the RAAS is activated the more BNP released to counteract
- The higher the BNP, the harder the ventricles are working to counteract the RAAS
Pharmacological Actions of hBNP

- Hemodynamic (balanced vasodilation)
- • veins
- • arteries
- • coronary arteries
- Neurohormonal
- • aldosterone
- • norepinephrine
- Renal
- • diuresis & natriuresis

BNP Concentration and Degree of HF Severity

<table>
<thead>
<tr>
<th>BNP Concentration (pg/mL)</th>
<th>Mild n = 27</th>
<th>Moderate n = 34</th>
<th>Severe n = 36</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>186</td>
<td>791</td>
<td>2013</td>
</tr>
<tr>
<td>500</td>
<td>22</td>
<td>165</td>
<td>266</td>
</tr>
<tr>
<td>1000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2500</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Types of heart failure

Diastolic Dysfunction
- Heart failure symptoms with EF ≥ 40%
- Hypertrophic CMP, HTN, Ischemia, Age
- Imbalance in volume/pressure relationship

Systolic Dysfunction
- Depressed contractility EF ≤ 40%
- CAD, Valve disease, Ischemic and idiopathic CMP

Systolic vs Diastolic HF

- Inability of LV to contract against a load and eject blood into the aorta
- Hallmark signs:
  - Reduced ejection fraction (EF)
  - Reduced stroke volume
  - Abnormal diastolic function

Systolic Dysfunction

Pathophysiology

1. LV wall thins and the cavity dilates (eccentric hypertrophy)
2. Thin, dilated ventricle unable to contract effectively
3. EF decreases
4. Leads to ↓ CO, ↑ LVEDV, ↑ preload → pulmonary congestion
5. Dilated cardiomyopathy common cause of systolic HF
6. Cardiomyopathy and systolic dysfunction should NOT be used interchangeably

1. EF < 40% systolic dysfunction
Diastolic Dysfunction

Pathophysiology

1. Ventricular muscle thickens (concentric hypertrophy)
2. Ventricular cavity size may remain normal or become smaller
3. Noncompliant ventricle unable to relax, impairing filling
4. To ↑ filling, left atrial pressure ↑; leading to pulmonary congestion

1. EF normal in diastolic dysfunction

Clinical Presentation

- Symptomatic with exertion and ↑ HR
  - Faster HR ↓ filling time & ↓ CO
  - Exercise → ↑ catecholamines → ↑ HR → worsens diastolic function
  - Flash pulmonary edema can develop during periods of ischemia

Elevated heart rate worsens diastolic function

Diagnosis

Three conditions required:
1. Signs and symptoms of HF
2. Normal or only slightly decreased EF
3. Increased diastolic filling pressures and abnormal relaxation of LF
4. Diagnosis made in patients presenting with the clinical syndrome of HF with no evidence of systolic dysfunction

Commonly associated with chronic hypertension or ischemic heart disease

New York Heart Association (NYHA) Classification of Heart Failure

<table>
<thead>
<tr>
<th>Class</th>
<th>Patient Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I (Mild)</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea).</td>
</tr>
<tr>
<td>Class II (Mild)</td>
<td>Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea).</td>
</tr>
<tr>
<td>Class III (Moderate)</td>
<td>Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea).</td>
</tr>
<tr>
<td>Class IV (Severe)</td>
<td>Unable to carry out any physical activity without discomfort. Symptoms of fatigue, rapid/irregular heartbeat (palpitation) or shortness of breath (dyspnea) are present at rest. If any physical activity is undertaken, discomfort increases.</td>
</tr>
</tbody>
</table>

Heart Failure

Right-Sided Heart Failure
- Dependent edema
- Jugular Venous distention
- Abdominal distension
- Hepatomegaly
- Splenomegaly
- Anorexia/Nausea
- Weight gain
- Nocturnal diuresis
- (Systemic circulation)
- (Pulmonary system)

Left-Sided Heart Failure
- Dyspnea
- Tachypnea
- Crackles in the lungs
- Dysrhythmias
- Orthostatic Hypotension
- Weight gain
- Nocturnal diuresis
- (Systemic circulation)
- (Pulmonary system)
**Right sided symptoms – think Circulation**
- Swelling of feet & legs
- Swollen or tender abdomen w/loss of appetite
- Increased urination at night

**Left sided symptoms – think Lungs**
- Shortness of breath
- Cough
- Difficulty sleeping at night due to breathing problems

**Right Ventricular Failure**

**Causes**
- Usually results from prolonged LV failure
- Right ventricular MI or inferior wall MI
- Primary pulmonary hypertension
- Acute or chronic lung disease
- Chronic severe tricuspid regurgitation

**Neurohormonal Responses in HF**

1. **Activation of Sympathetic Nervous System (SNS)**
2. **Renin–Angiotensin–Aldosterone System (RAAS) Kicks in…**

**SNS Activation**
- ↓ Cardiac Output
- Activates SNS
- Compensates by releasing neurohormones (Norepinephrine, Aldosterone, Vasopression)
- ↓ Sodium & Water Retention
- ↓ Congestion
- ↓ Vasoconstriction
- ↑ Afterload

**Renin–Angiotensin–Aldosterone System (RAAS)**
- Low Cardiac Output / Hypotension / Hypovolemia
- Decreased Renal perfusion
- Afferent Arteriole (baroreceptors)
- Release Renin (α-messenger)
- Go to Liver to stimulate Angiotensin I → production
- Angiotensin I goes to the Lung
- Angiotensin Converting Enzyme (ACE) located in the pulmonary vascular membrane
- Converts Angiotensin I to Angiotensin II
- ↓ Angiotensin II
- ↓ Growth Factor
- ↓ Potassium
- ↑ Aldosterone
- ↑ Adrenal Cortex
- ↑ Increases S/F
- ↑ Distal Renal Tubule
- ↑ Increases H2O & Na+ + Resorption
- Excretes K+ for Na+
Heart Failure

**Pharmacological Treatment**

- Three primary goals of medical treatment
  1. Reduce preload
  2. Reduce afterload
  3. Increase contractility

---

**Paradigm for Outpatient Management of Heart Failure**

<table>
<thead>
<tr>
<th>Stabilize patient</th>
<th>Stabilize the disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretic</td>
<td>ACE inhibition</td>
</tr>
<tr>
<td>β-blockade</td>
<td>Aldosterone inhibitors</td>
</tr>
<tr>
<td>Treat residual</td>
<td>Digoxin</td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
</tr>
</tbody>
</table>


---

**Reduce Preload**

- **Diuretics**
  - Venous Vasodilators
    - ACE (Angiotensin–converting enzyme) Inhibitor
      - Blocks the RAAS effect of reabsorption of sodium and water and thus decreases volume overload
    - Aldosterone antagonists
      - Spironolactone & eplerenone
      - Added to increase diuretic effect if symptoms at rest
    - Nitrates
      - Dilates veins, allowing more blood to remain in the vascular system and sending less to the heart

---

**ACEI: Therapeutics**

- Contraindications:
  - history of angioedema, hyperkalemia, creatinine > 3.0
- Complications:
  - hyperkalemia, worsening renal failure, cough, dysgeusia, angioedema
- Patient education:
  - check BP & call in, report dizziness, orthostasis, syncope, or lip swelling, take separately from beta blocker dose
ACE Inhibitors “Prils”
- Benazepril Lotensin
- Captopril Capoten
- Lisinopril Zestril Prinivil
- Quinapril Accupril
- Ramipril Altace

ARBs: Benefits in HF
- Prevent remodeling after MI; reduce LV dilation & scar thinning
- Reduce cytokine levels
- May additively attenuate neurohormonal levels when combined with ACEI therapy

ARBs “Sartans”
- Candesartan Atacand
- Irbesartan Avapro
- Losartan Cozaar
- Valsartan Diovan
- Telmisartan Micardis
- Eprosartan Teveten

ARBs: Therapeutics
- Contraindications
  - hyperkalemia, history of angioedema, creatinine >3.0
- Complications
  - angioedema, headache, dizziness, hyperkalemia
- Patient education
  - similar to ACEI

Aldosterone Antagonists
- Spironolactone and Eplerenone
  - Data:
    - Spironolactone studied in Class III & IV patients
    - Eplerenone studied in post MI pts with HF
  - Contraindications
    - hyperkalemia
    - renal failure, creatinine >2.5
  - Use cautiously:
    - patients on potassium supplements
    - hyponatremia
    - renal insufficiency
    - hepatic disease

Aldosterone Antagonists
- Patient education:
  - May need to teach patient to eat foods low in potassium
  - discontinue potassium–based salt substitutes
  - Check K+ levels one & three weeks after initiation, after dose changes, and regularly thereafter
Diuretics

- Used to relieve fluid retention in symptomatic patients; use as little dose as possible
  - Improves exercise tolerance
- Facilitate the use of other drugs indicated for heart failure
- Patients can be taught to adjust their diuretic dose based on changes in body weight

Reduced Afterload

- Arterial Vasodilators
  - ACE (Angiotension-converting enzyme) Inhibitor
    - Blocks the RAAS effect of vasoconstriction
    - Enhances the action of kinins, which promotes a positive vasodilatory effect
    - Slows disease progression
  - Angiotension Receptor Blockers
    - Directly blocks angiotension II – results vasodilatory effect
    - Used if patient cannot tolerate ACEI due to cough or angioedema
  - Hydralazine and Nitrate Combination
    - Hydralazine – arterial vasodilator
    - Recommended in African Americans with systolic dysfunction
      - Oral nitrates are venous vasodilators (preload reduction)
  - Milrinone (Primacor) Phosphodiesterase inhibitor
    - Causes vasodilation to ↓ preload and afterload

Beta Blockers

- Blocks the neurohormonal response of chronic SNS stimulation
- Slows heart rate for better diastolic ventricular filling
- Not initiated when fluid overload or in a decompensated state
- Initiate after fluid status optimized (no longer needing IV diuretics or IV vasodilators)
- Reduces arrhythmias
- Slows disease progression
  - Carvedilol (Coreg), Metoprolol (Lopressor), and Bisoprolol (Zebeta)

How to Initiate Beta Blockers in Heart Failure

- Ensure patient is not fluid overloaded or dehydrated
- Start at the lowest dosage
- Increase to next dosage level every 2 weeks – as tolerated
- Encourage patient to continue, even if somewhat more fatigued
Managing Side Effects During β-Blocker Uptitration

- Vasodilator side effects
  - Reassure patient that side effect is usually temporary
  - Separate the dosing of β-blocker and ACE inhibitor
  - If persistent, reduce vasodilators/diuretics
  - If persistent, reduce β-blocker dosage

Managing Side Effects During β-Blocker Uptitration

- Fluid retention
  - Increase diuretic to restore weight to baseline level
  - If persistent, reduce β-blocker dosage
  - Delay uptitration until weight is at baseline

- Bradycardia/AV block
  - Reduce dosage (or discontinue) drugs with effects on sinus and AV nodes
  - Measure digoxin levels
  - May reduce digoxin dose or discontinue
  - Reduce β-blocker dose

Beta Blockers “Olols”

- Acebutolol  Sectral
- Atenolol  Tenormin
- Betaxolol  Kerlone
- Bisoprolol  Zbeta
- Metoprolol  Lopressor
- Nadolol  Corgard
- Pindolol  Visken
- Propanolol  Inderal
- Timolol  Blocadren

Increase Contractility

- Increase contractility by first ↓ afterload
  - Digoxin
    - Oral medicine of choice to assist with contractility
    - Enhances inotropy of cardiac muscle
    - Reduces activation of SNS and RAAS
    - Most effective in patients with low EF
  - Dobutamine
    - ↑ contractility by stimulating beta receptors
  - Milrinone (Primacor) Phosphodiesterase inhibitor
    - Increases calcium ion uptake.
    - Has positive inotropic effect

Medications to Avoid in HF

- NSAIDS (non-steroidal anti-inflammatory drugs)
  - ↑ risk of fluid retention and renal failure
  - May diminish the efficacy of diuretics and ACE inhibitors
- Most antiarrhythmics
  - Poorly tolerated due to proarrhythmic & cardiodepressant effects
  - Amiodarone – does not adversely affect HF survival
- Calcium channel blockers
  - Amlodipine is okay
- Avandia (Rosiglitazone Maleate)
  - Can cause fluid retention and exacerbate HF

CRT Systems

- Atrial lead in right atrial appendage (same as conventional pacing)
- Right-ventricular lead at the RV apex or RV outflow tract
- Left-ventricular lead is passed through the coronary sinus and into one of the vessels on the outside of the heart
Cardiac Resynchronization Therapy

**Patient Indications**

**CRT device:**
- Moderate to severe HF (NYHA Class III/IV) patients
- Symptomatic despite optimal, medical therapy
- QRS ≥ 130 msec
- LVEF ≤ 35%

**CRT plus ICD:**
- Same as above with ICD indication

Nonpharmacological Treatments of HF

- Diet
- Daily weights
- Exercise
- Teach back

Health care reform

What does that mean to me as a Fairbanks Memorial Hospital Employee?

How do readmissions come into play?

- Beginning in 2013 Medicare and Medicaid will not pay for patients who are discharged from the hospital with those four diagnosis and readmitted back to the hospital within 31 days.
- As you know, many of these types of patients have complex needs both medically and socially which can often put them back in the acute care hospital within 31 days of discharge.
- If a person is readmitted within 31 days then we get zero pay for the second stay
Readmissions

- Heart Failure
- COPD
- Pneumonia
- AMI

Methodist Approach is an Enterprise Approach

- We need everyone who interacts with the patient as part of the healthcare team to all function from a common set of standardized tools and interventions – with the goal to assist in managing these patients more effectively to keep them out of the hospital with improved management on the outpatient side.

- Hence an enterprise wide approach

Heart Failure is the nation’s most rapidly growing heart problem

The Vicious Cycle of Heart Failure Management

Causes of Hospital Readmission for Heart Failure
Removing table salt does not constitute a low sodium diet.
Daily weight documentation following weight standards: Weigh on admission to unit, including transfers, then:

- Weigh between 4 - 6 am
- Same scale
- Empty bladder
- Do not weigh monitor
- Stand to weigh if able
- Document in computer which type of scale used
- Document the weight difference from previous day’s wt (±) in the computer

Wt Gain/Loss – should be charted daily

Weights
- 126 lb
- 127
- 124.9
- 112.7
- 120.9
- 120.3

Quiz Time
How many ml = 2.2 pounds or 1 kg?
A. 220 ml  
B. 500 ml  
C. 1000 ml  
D. 2200 ml
You are the nurse taking care of this patient on 5/9. What are your concerns?

Example of good diuresis

How to check 24 hour & LOS Net

Educating Patients About Daily Weights

- The vascular bed can hold 10 pounds of fluid before it starts to seep out into the tissues
  - 2 pounds = 1 quart of water extra in the circulation
- Keep track – standard is to notify MD of a 2–3 pound weight gain overnight or 5 pound gain in one week

Assessment of Fluid Volume Status

- Teaching points for patients/families:
  - Ankle swelling (shoes too tight, socks make marks on leg)
  - Abdominal swelling ("bloated", belt too tight)
  - Orthopnea
  - Paroxysmal nocturnal dyspnea
  - Weight gain
  - Shortness of Breath with walking
Exercise Recommendations
- Start slow, increase slowly
- Avoid the extremes of intertemperate climates
  - mall walking
  - indoor treadmills or tracks
- exercise cycle indoors
- May not initially tolerate exercise
  - may see increased symptoms (2–6 weeks)
  - increased blood volume
  - fatigue
- Don’t be discouraged by inevitable interruptions in activity/training schedule

Patient Teaching – Exercise
Exercise such as walking is important but don’t overdo it. Set realistic goals and don’t “push.” Stop exercising immediately if you feel tired, have chest pain, or are very short of breath. Using your activity in the hospital as a guide start with that amount of exercise and gradually increase.
- Remember these tips as well:
  - Do things at a slow to moderate pace – don’t rush.
  - Space out activities throughout the day taking 30-30 minute rest periods. Do “easy” activities alternating with harder ones.
  - Don’t exercise for at least one hour after meals.
  - Avoid extremely hot or cold temperatures.
  - Avoid heavy exercise and weight lifting. Ask the doctor about returning to work and doing active recreational activities.
  - Keep the general guidelines above and use positions easy for you when you feel able to resume your sexual activity.

Get with the Guidelines HF Silver and Gold Plus
- In addition to Achievement Award
- Select 4 or more of the HF Quality Measures
- Demonstrate at least and average of 75% or above compliance

Get with the Guidelines HF Silver and Gold Plus
- Anticoagulation for atrial fibrillation: Percent of patients with atrial fibrillation or recent ischemic stroke on appropriate anticoagulation therapy at discharge.
- Fibrinolysis at discharge: Percent of eligible patients with left ventricular systolic dysfunction (LVEF ≤ 35%) who received appropriate fibrinolytic therapy within 1 hour of hospital arrival.
- Hemoglobin A1c at discharge: Percent of patients with left ventricular systolic dysfunction (LVEF ≤ 35%) who received appropriate insulin or diabetes medication therapy at discharge.
- HF inpatient hospitalization: Percent of eligible patients with left ventricular systolic dysfunction (LVEF ≤ 35%) who received appropriate discharge education.
- HF Readmission: Percent of eligible patients with left ventricular systolic dysfunction (LVEF ≤ 35%) who were readmitted within 30 days of discharge.

HF ACHIEVEMENT MEASURES
- ACE/ARB at discharge: Percent of heart failure patients with left ventricular systolic dysfunction (LVEF ≤ 35%) and without both angiotensin converting enzyme inhibitor (ACE) and angiotensin receptor blocker (ARB) contraindications who are prescribed an ACE or ARB at hospital discharge.
- BNP at hospital discharge: For purposes of this measure, we excluded patients with a point-of-care troponin test and/or elevated cardiac troponin elevation (CTE) test levels ≤ 40000 or a review description of an elevated peak troponin (PT) patient with moderate or severe systolic dysfunction.
- Evidence-based specific beta blockers: Percent of heart failure patients who were prescribed with evidence-based specific beta blockers (bisoprolol, carvedilol, metoprolol succinate) at discharge.
- Measure 4: Function: Percent of heart failure patients with documentation in the hospital record that left ventricular function (LVEF) was assessed before admittance, during hospitalization, or is planned for after discharge.
- Post-discharge appointment for heart failure patients: Percent of eligible heart failure patients for whom a follow-up appointment was scheduled and documented including which practitioner and when (i.e., care provider, follow-up visit, or location and date for home health visit).
Patient Education

- Studies have shown that 40–80 percent of the medical information patients receive is forgotten immediately.¹
- Nearly half of the medical information retained is incorrect.²


Patient Education – Teach Back

- One of the easiest ways to close the gap of communication between clinician and patient is to employ the “teach-back” method, also known as the “show-me” method or “closing the loop.”³
- Teach-back is a way to confirm that you have explained to the patient what they need to know in a manner that the patient understands. Patient understanding is confirmed when they explain it back to you. It can also help the clinic staff members identify explanations and communication strategies that are most commonly understood by patients.

Teach-back: A Health Literacy Tool to Ensure Patient Understanding

Presentation created by The Iowa Health System Health Literacy Collaborative

The Challenge

- Research shows that patients remember and understand less than half of what clinicians explain to them.

Ley, Communicating with patients: improving communication satisfaction, and compliance 1988
Rost, Predictors of recall of medication regimens and recommendations for lifestyle change in elderly patients 1987

Everyone benefits from clear information.

Many patients are at risk of misunderstanding, but it is hard to identify them.

Testing general reading levels does not ensure patient understanding in the clinical setting.

Adapted from: Reducing the Risk by Designing a Safer, Shame-Free Health Care Environment. AMA, 2007

Universal Communication Principles

- **Everyone** benefits from clear information.
- Many patients are at risk of misunderstanding, but it is hard to identify them.
- Testing general reading levels does not ensure patient understanding in the clinical setting.

Talking with Patients & Families

**Always:**
- Use Plain Language.
- Slow down.
- Break it down into short statements.
- Focus on the 2 or 3 most important concepts.
- Check for understanding using teach-back.

Teach-back

- Why do I use it?
- What is it?
- How do I use it?
- When do I use it?

Teach-back is...

- Asking patients to repeat in their own **words** what they need to know or do, in a non-shaming way.
- **NOT** a test of the patient, but of how well you explained a concept.
- A chance to check for understanding and, if necessary, re-teach the information.
“Asking that patients recall and restate what they have been told” is one of 11 top patient safety practices based on the strength of scientific evidence.”
AHRQ, 2001 Report, Making Health Care Safer

“Physicians’ application of interactive communication to assess recall or comprehension was associated with better glycemic control for diabetic patients.”
Schillinger, Arch Intern Med Vol 163, Jan 13, 2003, “Closing the Loop”

 Asking for a Teach-back - Examples

Ask patients to demonstrate understanding, using their own words:
- “I want to be sure I explained everything clearly. Can you please explain it back to me so I can be sure I did?”
- “What will you tell your husband about the changes we made to your blood pressure medicines today?”
- “We’ve gone over a lot of information, a lot of things you can do to get more exercise in your day. In your own words, please review what we talked about. How will you make it work at home?”

Teach-back... 

Teach-back – Additional Points

Do not ask yes/no questions like:
- “Do you understand?”
- “Do you have any questions?”

For more than one concept:
- “Chunk and Check”
  - Teach the 2-3 main points for the first concept & check for understanding using teach-back...
  - Then go to the next concept

Teach-back – Using it Well: Elements of Competence

Responsibility is on the provider.
Use a caring tone of voice & attitude.
Use Plain Language.
Ask patient to explain using their own words (not yes/no).
Use for all important patient education, specific to the condition.
Document use of & response to teach-back.

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10 Elements of Competence for Using Teach-back Effectively

1. Use a caring tone of voice and attitude.
2. Display comfortable body language and make eye contact.
3. Use plain language.
4. Ask the patient to explain back, using their own words.
5. Use re-echoing, open-ended questions.
6. Avoid asking questions that can be answered with a simple yes or no.
7. Emphasize that the responsibility to explain clearly is on you, the provider.
8. If the patient is not able to teach back correctly, explain again and re-check.
9. Use read-aloud prints materials to support learning.

Heart Failure Teach Back Questions Day 1

Patient Interview:
1. What did you think triggered your admission to the hospital?
2. What are the symptoms of your heart failure?
3. What is the name of your heart pill(s)?
4. What weight gain should you tell your doctor about? Do you have a scale at home?
5. What foods should you avoid when you have heart failure?

Interventions:
- Give scale & weight documented on card.
- Give and document heart failure packet.
- Teach follow-up visit and treatment education.
- Document triggers in intake section of Day 1 education.
- Document all education in computer.

Repeat Day 1 questions until patient understands.

For newly diagnosed, change question to “This is your heart pill. Call about weight gain…” etc.

Heart Failure Teach Back Questions Day 2

Continue with Day 2 after patient understands Day 1 questions.

Patient Interview:
1. Why is it important to take your medication for heart failure?
2. Why is it important to avoid foods with sodium (salt)?
3. Why is it important to watch for the symptoms of heart failure?
4. Why is it important to watch for weight gain?

Interventions:
- Dietitian follow-up.
- Change order.
- Reading labels.
- Doing out and ordering from a menu.
- Medication video.
- Nutrition video.
- Document education in computer.

Teach Back Questions Day 3

Continue with Day 3 after patient understands Day 1 & 2 questions.

Patient Interview:
1. How do you feel your medication and when you will take them.
2. How will you keep yourself from being rehospitalized?

Interventions:
- Discharge video.
- Give HR support group handout and encourage attendance.
- Review meds.
- Nurse/F weight goal/ target weight.
- Call MD.
- Follow-up appointment made within one week or home health referral.
- Verify all care measures documented including HF packet given.

Teach Back Questions Discharge

Heart Failure Call 911 for:
- Uncontrolled chest pain
- Uncontrolled shortness of breath
- Weight gain up to 5 pounds in 1 day or 5 pounds in 1 week
- New swelling in feet, ankles, hands, abdomen
- Cough that does not go away
- Increased shortness of breath especially with rest or when lying down
- Loss of energy, weakness
- Unexplained weight loss
- Unexplained diarrhea, confusion or anxiety

You are doing well when:
- Weight is stable
- Able to do normal activities
- No change in symptoms

Call Home Health or Doctor for:
- Home health or doctor visit