Heart Failure

- The most common reason for hospitalization in adults >65 years old.
Heart Failure-
(progression)

Mild

CDHF(Pulmonary Edema)

Severe End Stage

Cardiogenic shock
Cardiomyopathy

Irreversible

Needs new ventricle

VAD
IABP
Heart Transplant

Control With

Drugs
Diet
Fluid
Restriction

Emergency-Upright, O2, morphine, etc
Heart Failure - Clinical syndrome ... can result from any structural or functional cardiac disorder that impairs ability of ventricle to fill with or eject blood

Impact!

5 million Americans- have heart failure
- 500,000 new cases every year
- 25-50 billion dollars a year to care for people with HF
- 6,500,000 hospital days / year and 300,000 deaths/year
Definition - Heart Failure (HF)

Key Concepts

- CO = SV x HR - becomes insufficient to meet metabolic needs of body
- SV - determined by preload, afterload and myocardial contractility
- EF < 40% (need to understand)
- *Classifications HF
  - Systolic failure - dec. contractility
  - Diastolic failure - dec. filling
  - Mixed
90/140 = 64% EF - 55-65 (75) normal

Click for animated EF
• Keys to understanding HF

• All organs (liver, lungs, legs, etc.) return blood to heart
• When heart begins to fail/ weaken> unable to pump blood forward-fluid backs up > Inc. pressure within all organs.
  • Organ response
  • LUNGS: congested > “stiffer”, inc effort to breathe; fluid starts to escape into alveoli; fluid interferes with O2 exchange, aggravates shortness of breath.

• Shortness of breath during exertion, may be early symptoms > progresses > later require extra pillows at night to breathe > experience "P.N.D." or paroxysmal nocturnal dyspnea .
• Pulmonary edema
• Legs, ankles, feet- blood from feet and legs > back-up of fluid and pressure in these areas, heart unable to pump blood as promptly as received > inc. fluid within feet and legs causes fluid to "seep" out of blood vessels ; inc. weight
Heart Failure

Decreased blood supply to the brain may cause dizziness.

Decreased blood supply to the kidneys causes the body to retain salt and water.

Decreased blood supply to the legs causes fatigue, particularly during exertion.
Heart Failure (ADHF) Pneumonic
(emergency mgt > recall for later!)

U Upright Position
N Nitrates
L Lasix
O Oxygen
A ACE, ARBs, Amiodorone
D Dig, Dobutamine
M Morphine Sulfate
E Extremities Down
Heart Failure

Click here for Online Lecture (Interactive)

or

Click here for Online Lecture (Read)
Heart Failure
Etiology and Pathophysiology

- Systolic failure - most common cause
  - Hallmark finding: Dec. in *left ventricular ejection fraction* (EF)
  - Due to
    - Impaired contractile function (e.g., MI)
    - Increased afterload (e.g., hypertension)
    - Cardiomyopathy
    - Mechanical abnormalities (e.g., valve disease)
Heart Failure

Etiology and Pathophysiology

- **Diastolic failure**
  - Impaired ability of ventricles to relax and fill during diastole > dec. stroke volume and CO
  - Diagnosis based on presence of pulmonary congestion, pulmonary hypertension, ventricular hypertrophy
  - *normal ejection fraction (EF)* - **Know why!**
Heart Failure

Etiology and Pathophysiology

• **Mixed systolic and diastolic failure**
  – Seen in disease states such as dilated cardiomyopathy (DCM)
  – Poor EFs (<35%)
  – High pulmonary pressures

• **Biventricular failure** (both ventricles may be dilated and have poor filling and emptying capacity)
Factors effecting heart pump effectiveness

**Preload**

- Volume of blood in ventricles at end diastole
- Depends on venous return
- Depends on compliance

**Afterload**

- Force needed to eject blood into circulation
- Arterial B/P, pulmonary artery pressure
- Valvular disease increases afterload
Cardiomegaly/ventricular remodeling occurs as heart overworked—changes in size, shape, and function of heart after injury to left ventricle. Injury due to acute myocardial infarction or due to causes that inc. pressure or volume overload as in Heart failure.
Normal

Enlarged Heart
A type of cardiomyopathy. An enlarged heart is a sign that the heart may be overworked.

Left ventricle

Right ventricle

Thin, weakened left ventricle

American Heart Assn-Media files Animations
Heart Failure
(AKA-congestive heart failure)

- **Pathophysiology**
- **A.** Cardiac compensatory mechanisms
  - 1. tachycardia
  - 2. ventricular dilation - Starling’s law
  - 3. myocardial hypertrophy
    - Hypoxia leads to dec. contractility
Pathophysiology-Summary

• B. Homeostatic Compensatory mechanisms
• Sympathetic Nervous System- (beta blockers block this)
  – 1. Vascular system- norepinephrine- vasoconstriction (What effect on afterload?)
  – 2. Kidneys
    • A. Dec. CO and B/P > renin angiotensin release. (ACE)
    • B. Aldosterone release > Na and H2O retention
  – 3. Liver- stores venous volume (ascites, +HJR, Hepatomegaly- can store 10 L. check enzymes

Counter-regulatory-
• Inc. Na > release of ADH (diuretics)
• *Release of atrial natriuretic factor > Na and H2O excretion, prevents severe cardiac decompensation
• What is BNP? What drug is synthetic form BNP?
Heart Failure

Etiology and Pathophysiology

• Compensatory mechanisms- activated to maintain adequate CO
  – Neurohormonal responses: *Endothelin*-stimulated by ADH, catecholamines, and angiotensin II >
    • Arterial vasoconstriction
    • Inc. in cardiac contractility
    • Hypertrophy
Heart Failure

Etiology and Pathophysiology

• Compensatory mechanisms - activated to maintain adequate CO
  – Neurohormonal responses: Proinflammatory cytokines (e.g., tumor necrosis factor)
    • Released by cardiac myocytes in response to cardiac injury
    • Depress cardiac function > cardiac hypertrophy, contractile dysfunction, and myocyte cell death
Heart Failure
Etiology and Pathophysiology

• **Compensatory mechanisms**- activated to maintain adequate CO
  – Neurohormonal responses: **Over time** > systemic inflammatory response > results
    • Cardiac wasting
    • Muscle myopathy
    • Fatigue
Heart Failure
Etiology and Pathophysiology

• **Counter regulatory processes**
  – Natriuretic peptides: atrial natriuretic peptide (ANP) and b-type natriuretic peptide (BNP)- *also dx test for HF

• Released in response to inc. in atrial volume and ventricular pressure
• Promote venous and arterial vasodilation, **reduce preload and afterload**
• Prolonged HF > **depletion of these factors**
Heart Failure
Etiology and Pathophysiology

• Counter regulatory processes
  – Natriuretic peptides- *endothelin and aldosterone antagonists*
    • Enhance diuresis
    • Block effects of the *RAAS*
  – Natriuretic peptides- *inhibit development of cardiac hypertrophy*; may have antiinflammatory effects
Result of Compensatory Mechanisms

Heart Failure Explained

**Figure 1. Heart Failure Cycle.**
Pathophysiology -

**Structural Changes with HF**

- Dec. contractility
- Inc. preload (volume)
- Inc. afterload (resistance)
- **Ventricular remodeling (ACE inhibitors can prevent this)**
  - Ventricular hypertrophy
  - Ventricular dilation
Ventricular remodeling

Left ventricular hypertrophy

Left ventricular dilatation
LV Remodeling Post-infarction

Normal
Infarct expansion
Dilatation & thinning
Further dilatation

Septum
Free wall
A
B
C
D
FLUID OVERLOAD > Acute Decompensated Heart Failure (ADHF)/Pulmonary Edema

> Medical Emergency!
Heart Failure

Classification Systems

- New York Heart Association Functional Classification of HF
  - Classes I to IV
- ACC/AHA Stages of HF (newer)
  - Stages A to D
### NYHA Functional Classification of Heart Disease

**Class I**  
No limitation of physical activity. Ordinary physical activity does not cause fatigue, dyspnea, palpitations, or anginal pain.

**Class II**  
Slight limitation of physical activity. No symptoms at rest. Ordinary physical activity results in fatigue, dyspnea, palpitations, or anginal pain.

**Class III**  
Marked limitation of physical activity. Usually comfortable at rest. Ordinary physical activity causes fatigue, dyspnea, palpitations, or anginal pain.

**Class IV**  
Inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of angina may be present even at rest. If any physical activity is undertaken, discomfort is increased.

### ACC/AHA Stages of Heart Failure

**Stage A**  
Patients at high risk of developing left ventricular dysfunction because of the presence of conditions that are strongly associated with the development of HF.

**Stage B**  
Patients who developed structural heart disease that is strongly associated with the development of HF but who have never shown signs of HF.

**Stage C**  
Patients who have current or prior symptoms of HF associated with underlying structural heart disease.

**Stage D**  
Patients with advanced structural heart disease and marked symptoms of HF at rest despite maximal medical therapy and who require specialized interventions.

ACC/AHA, American College of Cardiology/American Heart Association; HF, heart failure; NYHA, New York Heart Association.
ACC/AHA Stages

**Stage A**
- High risk for developing CHF
- No structural disorder of heart

**Stage B**
- Structural disorder of heart
- Never developed symptoms of CHF

**Stage C**
- Past or current symptoms of CHF
- Symptoms associated with underlying heart disease

**Stage D**
- End-stage disease
- Requires specialized treatment strategies

NY ASSN Funct Class

**Class I**
- No limitation of physical activity
- Ordinary activity does not cause fatigue, palpitations, dyspnea, or angina

**Class II**
- Slight limitation of physical activity
- Comfortable at rest
- Ordinary activity results in fatigue, palpitations, dyspnea, or angina

**Class III**
- Marked limitation of physical activity
- Comfortable at rest
- Less than ordinary activity results in fatigue, palpitations, dyspnea, or angina

**Class IV**
- Inability to carry on any physical activity without discomfort
- Symptoms present even at rest
- Symptoms exacerbated by any activity

Treatment Options

**Class IIIa**
- No dyspnea at rest

**Class IIIb**
- Recent dyspnea at rest
<table>
<thead>
<tr>
<th>Stage A</th>
<th>At high risk for developing heart failure. Includes people with:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Diabetes mellitus</td>
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<tr>
<td></td>
<td>CAD (including heart attack)</td>
</tr>
<tr>
<td></td>
<td>History of cardiotoxic drug therapy</td>
</tr>
<tr>
<td></td>
<td>History of alcohol abuse</td>
</tr>
<tr>
<td></td>
<td>History of rheumatic fever</td>
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<td></td>
<td>Family history of CMP</td>
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<tr>
<td></td>
<td>Exercise regularly</td>
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<td></td>
<td>Quit smoking</td>
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<tr>
<td></td>
<td>Treat hypertension</td>
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<td></td>
<td>Treat lipid disorders</td>
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<tr>
<td></td>
<td>Discourage alcohol or illicit drug use</td>
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<td></td>
<td>If previous heart attack/ current diabetes mellitus or HTN, use ACE-I</td>
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</tbody>
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<thead>
<tr>
<th>Stage B</th>
<th>Those diagnosed with “systolic” heart failure- have never had symptoms of heart failure (usually by finding an ejection fraction of less than 40% on echocardiogram)</th>
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<tbody>
<tr>
<td></td>
<td>Care measures in Stage A +</td>
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<tr>
<td></td>
<td>Should be on ACE-I</td>
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<tr>
<td></td>
<td>Add beta -blockers</td>
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<tr>
<td></td>
<td>Surgical consultation for coronary artery revascularization and valve repair/replacement (as appropriate)</td>
</tr>
</tbody>
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<thead>
<tr>
<th>Stage C</th>
<th>Patients with known heart failure with current or prior symptoms.</th>
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<tbody>
<tr>
<td></td>
<td>Symptoms include: SOB, fatigue</td>
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<tr>
<td></td>
<td>Reduced exercise intolerance</td>
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<tr>
<td></td>
<td>All care measures from Stage A apply, ACE-I and beta-blockers should be used +</td>
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<tr>
<td></td>
<td>Diuretics, Digoxin,</td>
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<tr>
<td></td>
<td>Dietary sodium restriction</td>
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<td></td>
<td>Weight monitoring, Fluid restriction</td>
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<tr>
<td></td>
<td>Withdrawal drugs that worsen condition</td>
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<tr>
<td></td>
<td>Maybe Spironolactone therapy</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Stage D</th>
<th>Presence of advanced symptoms, after assuring optimized medical care</th>
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<tbody>
<tr>
<td></td>
<td>All therapies -Stages A, B and C + evaluation for:Cardiac transplantation, VADs, surgical options, research therapies, Continuous intravenous inotropic infusions/ End-of-life care</td>
</tr>
</tbody>
</table>
Heart Failure

Etiology and Pathophysiology

- Primary risk factors
  - Coronary artery disease (CAD)
  - Advancing age
- Contributing risk factors
  - Hypertension
  - Diabetes
  - Tobacco use
  - Obesity
  - High serum cholesterol
  - African American descent
  - Valvular heart disease
  - Hypervolemia
CHF-due to

– 1. Impaired cardiac function
  • Coronary heart disease
  • Cardiomyopathies
  • Rheumatic fever
  • Endocarditis

– 2. Increased cardiac workload
  • Hypertension
  • Valvular disorders
  • Anemias
  • Congenital heart defects

– 3. Acute non-cardiac conditions
  • Volume overload
  • Hyperthyroid, Fever, infection
Classifications- (how to describe)

- Systolic versus diastolic
  - Systolic- loss of contractility get dec. CO
  - Diastolic- decreased filling or preload
- Left-sided versus right –sided
  - Left- lungs
  - Right-peripheral
- High output- hypermetabolic state
- Acute versus chronic
  - Acute- MI
  - Chronic-cardiomyopathy
Symptoms

- Shortness of breath
- Swelling of feet & legs
- Chronic lack of energy
- Difficulty sleeping at night due to breathing problems
- Swollen or tender abdomen with loss of appetite
- Cough with frothy sputum
- Increased urination at night
- Confusion and/or impaired memory
Left Ventricular Failure

- Signs and symptoms
  - dyspnea
  - orthopnea PND
  - Cheyne Stokes
  - fatigue
  - Anxiety
  - rales

- NOTE L FOR LEFT AND L FOR LUNGS
- Why does this occur??
Heart Failure

Clinical Manifestations

• Acute decompensated heart failure (ADHF)
  – > Pulmonary edema, often life-threatening
• Early
  – Increase in the respiratory rate
  – Decrease in PaO₂
• Later
  – Tachypnea
  – Respiratory acidemia
Heart Failure

Clinical Manifestations

- Acute decompensated heart failure (ADHF)

- Physical findings
  - Orthopnea
  - Dyspnea, tachypnea
  - Use of accessory muscles
  - Cyanosis
  - Cool and clammy skin

- Physical findings
  - *Cough with frothy, blood-tinged sputum- why???  > (see next slide)
  - Breath sounds: Crackles, wheezes, rhonchi
  - Tachycardia
  - Hypotension or hypertension
Acute Heart Failure

Acute heart failure is sudden onset of left ventricular failure as the result of an acute primary event. The most common precipitating factor is myocardial infarction. Cardiac output falls as there is less contractile mass to eject blood from the left ventricle. In response to this the sympathetic nervous system is activated increasing heart rate and inducing vasoconstriction in an attempt to maintain normal blood pressure and perfusion of vital organs.

Pressure in the left ventricle increases as it is unable to empty efficiently.

In turn this pressure is conducted back to the lungs where it pushes fluid into the alveoli causing acute shortness of breath. This breathlessness is worse on reclining and may be associated with frothy sputum, frothy sputum, pink or white, pulmonary oedema.

Complete Case study of Heart Failure in Lewis online resources
Pulmonary edema begins with an increased filtration through the loose junctions of the pulmonary capillaries.

As the intracapillary pressure increases, normally impermeable (tight) junctions between the alveolar cells open, permitting alveolar flooding to occur.

Acute Decompensated Heart Failure (ADHF) Pulmonary Edema
ADHF/Pulmonary Edema (advanced L side HF)

- When PA WEDGE pressure is approx 30mmHg
  - Signs and symptoms
    - 1. wheezing
    - 2. pallor, cyanosis
    - 3. Inc. HR and BP
    - 4. s3 gallop
    - 5. rales, copious pink, frothy sputum
Person literally drowning in secretions

Immediate Action Needed
Goals of Treatment - ADHF/Pulmonary Edema

- MAD DOG
- Improve gas exchange
  - Start O2/elevate HOB/intubate
  - Morphine – dec anxiety/afterload
  - A- (airway/head up/legs down)
  - D- (Drugs) Dig not first now- but drug
    - IV nitroglycerin; IV Nipride, Natrecor
  - D- Diuretics
  - O- oxygen /measure sats;
    - Hemodynamics, careful observation
  - G- blood gases

- Think physiology
Right Heart Failure

• Signs and Symptoms
  – fatigue, weakness, lethargy
  – wt. gain, inc. abd. girth, anorexia, RUQ pain
  – elevated neck veins
  – Hepatomegaly +HJR
  – may not see signs of LVF
What does this show?
What is present in this extremity, common to right sided HF?
Can You Have RVF Without LVF?

- What is this called?

**COR PULMONALE**

Cor pulmonale, or right-sided heart failure, is an enlargement of the right ventricle due to high blood pressure in the lungs usually caused by chronic lung disease.
Heart Failure

Complications

• Pleural effusion

• Atrial fibrillation (most common dysrhythmia)
  – Loss of atrial contraction (kick) - reduce CO by 10% to 20%
  – Promotes thrombus/embolus formation inc. risk for stroke
  – Treatment may include cardioversion, antidysrhythmics, and/or anticoagulants
Heart Failure Complications

- **High risk of fatal dysrhythmias** (e.g., sudden cardiac death, ventricular tachycardia) with HF and an EF <35%

  - HF lead to severe hepatomegaly, especially with RV failure
    - Fibrosis and cirrhosis - develop over time
  - Renal insufficiency or failure
Heart Failure

Diagnostic Studies

• Primary goal- determine underlying cause
  – History and physical examination( dyspnea)
  – Chest x-ray
  – ECG
  – Lab studies (e.g., cardiac enzymes, BNP- (beta natriuretic peptide- normal value less than 100) electrolytes
  – EF
Heart Failure

Diagnostic Studies

• Primary goal- determine underlying cause
  – Hemodynamic assessment-Hemodynamic Monitoring-CVP- (right side) and Swan Ganz (left and right side)
  – Echocardiogram-TEE best
  – Stress testing- exercise or medicine
  – Cardiac catheterization- determine heart pressures ( inc.PAW )
  – Ejection fraction (EF)
An echocardiogram uses sound waves to produce an image of the heart.
Echocardiography

- EF is the "single most important measurement in HF"
- It helps define etiology and type of HF
- There is no correlation between symptoms and EF
Nursing Assessment

• Vital signs
• PA readings
• Urine output
• -What else!!
Chronic HF

Nursing Management

• Nursing diagnoses
  – Activity intolerance
  – *Decreased cardiac output*
  – Fluid volume excess
  – Impaired gas exchange
  – Anxiety
  – Deficient knowledge
Decreased cardiac output

- Plan frequent rest periods
- Monitor VS and O2 sat at rest and during activity
- Take apical pulse
- Review lab results and hemodynamic monitoring results
- Fluid restriction - keep accurate I and O
- Elevate legs when sitting
- Teach relaxation and ROM exercises
• **Activity Intolerance**
  – Provide O2 as needed
  – practice deep breathing exercises
  – teach energy saving techniques
  – prevent interruptions at night
  – monitor progression of activity
  – offer 4-6 meals a day

• **Fluid Volume Excess**
  – Give diuretics and provide BSC
  – Teach side effects of meds
  – Teach fluid restriction
  – Teach low sodium diet
  – Monitor I and O and daily weights
  – Position in semi or high fowlers
  – Listen to BS frequently
Knowledge deficit

- Low Na diet
- Fluid restriction
- Daily weight
- When to call Dr.
- Medications
Chronic HF

Nursing Management

• Planning: Overall Goals
  – Decrease in symptoms (e.g., shortness of breath, fatigue)
  – Decrease in peripheral edema
  – Increase in exercise tolerance
  – Compliance with the medical regimen
  – No complications related to HF
How to Achieve Goals

• Decrease preload
  – Dec. intravascular volume
  – Dec venous return i.e.
    • Fowlers
    • MSO4 and Ntg

• Decrease afterload

• Inc. cardiac performance(contractility)
  – CRT (cardiac resynchronization therapy)

• Balance supply and demand of oxygen
  – Inc. O2- O2, intubate, HOB up, legs down, mech vent with PEEP (if ADHF/PE)
  – Dec. demand- use beta blockers, rest, dec B/P

Manage symptoms
Chronic HF

Nursing Management

• Health Promotion

  – Treatment or control of underlying heart disease key to preventing HF and episodes of ADHF (e.g., valve replacement, control of hypertension)

  – Antidysrhythmic agents or pacemakers for patients with serious dysrhythmias or conduction disturbances

  – Flu and pneumonia vaccinations
Chronic HF

Nursing Management

• Health Promotion
  – Treatment or control of underlying heart disease key to preventing HF and episodes of ADHF (e.g., valve replacement, control of hypertension)
  – Antidysrhythmic agents or pacemakers for patients with serious dysrhythmias or conduction disturbances
  – Flu and pneumonia vaccinations
Chronic HF

*Nursing Management*

• Health Promotion
  – Patient teaching: medications, diet, and exercise regimens
    • Exercise training (e.g., cardiac rehabilitation) improves symptoms but often underprescribed
  – Home nursing care for follow-up and to monitor patient’s response to treatment may be required
Heart Failure
Nursing and Collaborative Management

- **Overall goals** - to therapy for ADHF & chronic HF
  - Decrease patient symptoms
  - Improve LV function
  - Reverse ventricular remodeling
  - Improve quality of life
  - Decrease mortality and morbidity
ADHF
Nursing and Collaborative Management

• Improve cardiac function
  – For patients who do not respond to conventional pharmacotherapy - (e.g.- O2, even intubate, high Fowler’s, diuretics, vasodilators, morphine sulfate)

• Inotropic therapy
  – Digitalis
  – β-Adrenergic agonists (e.g., dopamine)
  – Phosphodiesterase inhibitors (e.g., milrinone)
  – Caution – re- calcium channel blockers- dec. contractility- only amiodopine (Norvasc) approved even in mild heart failure)

• Hemodynamic monitoring
Chronic HF
Collaborative Management

• Main treatment goals
  – Treat underlying cause & contributing factors
  – Maximize CO
  – Provide treatment to alleviate symptoms
  – Improve ventricular function
  – Improve quality of life
  – Preserve target organ function
  – Improve mortality and morbidity
Chronic HF
Collaborative Management

• O2 (non-rebreather if emergency); morphine, diuretics, etc-dec preload, afterload

• Physical and emotional rest

• Nonpharmacologic therapies
  – Cardiac resynchronization therapy (CRT) or biventricular pacing
  – Cardiac transplantation
CRT—Cardiac Resynchronization Therapy

**HOW IT WORKS:**

Standard implanted pacemakers—equipped with two wires (or "leads")—conduct pacing signals to specific regions of the heart (usually at positions A and C). Biventricular pacing devices have added a third lead (to position B) that is designed to conduct signals directly into the left ventricle. Combination of all three lead > synchronized pumping of ventricles, inc. efficiency of each beat and pumping more blood on the whole.
Chronic HF
Collaborative Management

• **Therapeutic objectives for drug therapy**
  – Identification of type of HF & underlying causes
  – Correction of Na & H2O retention and volume overload
  – Reduction of cardiac workload
  – Improvement of myocardial contractility
  – Control of precipitating and complicating factors
Chronic HF - Collaborative Management

Drug therapy

- **Diuretics**
  - Thiazide
  - Loop
  - Spironolactone

- **Vasodilators**
  - ACE inhibitors - pril or ril *first line heart failure*
  - Angiotensin II receptor blockers
  - Nitrates
  - β-Adrenergic blockers - al or ol
  - **Nesiritide** - Natrecor (BNP)
Chronic HF
Collaborative Management

• Drug therapy (cont’d)
  – Positive inotropic agents
    • Digitalis
    • Calcium sensitizers- (Levosimendan) new under research; cardioprotective, inc. cardiac contractility
  – BiDil (combination drug containing isosorbide dinitrate and hydralazine) approved only for the treatment of HF in African Americans
Chronic HF

Collaborative Management

• Nutritional therapy
  – Diet/weight reduction recommendations-individualized and culturally sensitive
  – Dietary Approaches to Stop Hypertension (DASH) diet recommended
  – Sodium- usually restricted to 2.5 g per day
  – Potassium encouraged unless on K sparing diuretics (Aldactone)
Chronic HF
Collaborative Management

• Nutritional therapy
  – Fluid restriction may or may not be required
  – **Daily weights** important
    • Same time, same clothing each day
  – **Weight gain** of 3 lb (1.4 kg) over 2 days or a 3-to 5-lb (2.3 kg) gain over a week-report to health care provider
Chronic HF-End Stage >ADHF
Collaborative Management

• Nonpharmacologic therapies (cont’d)
  – Intraaortic balloon pump (IABP) therapy
    • Used for cardiogenic shock
    • Allows heart to rest
  – Ventricular assist devices (VADs)
    • Takes over pumping for the ventricles
    • Used as a bridge to transplant
  – Destination therapy-permanent, implantable VAD
  – Cardiomyoplasty- wrap latissimus dorsi around heart
  – Ventricular reduction -ventricular wall resected
  – Transplant/Artificial Heart
Intraaortic Balloon Pump (IABP)

- Provides temporary circulatory assistance
  - ↓ Afterload
  - Augments aortic diastolic pressure
- Outcomes
  - Improved coronary blood flow
  - Improved perfusion of vital organs
Balloon increases blood flow to the heart and relieves some of the workload by inflating when the heart relaxes and deflating just before the heart contracts.

This perspective shows the aorta as it extends down behind the heart.
Enhanced External Counterpulsation - EECP

Pumps during diastole - increasing O2 supply to coronary arteries. Like IABP but not invasive.
Ventricular Assist Devices (VADs)

- Indications for VAD therapy
  - Extension of cardiopulmonary bypass
    - Failure to wean
    - Postcardiotomy cardiogenic shock
  - Bridge to recovery or cardiac transplantation

- Patients with New York Heart Association Classification IV who have failed medical therapy
Patient Teaching-Cleveland Clinic for Heart Failure LVAD devices
Left ventricular assist device

Left Ventricular Assist Device (LVAD)

Controller battery and reserve battery

Pump

Aorta

Vent adapter and vent filter

External battery pack

Outflow-valve housing

Inflow-valve housing

Drive line

Prosthetic left ventricle

System controller

Skin line
The HeartMate II - one of several new LVAD devices - designed to last longer with simplicity of only one moving part; also much lighter and quieter than its predecessors; major differences is rotary action which creates a constant flow of blood, not “pumping action”.
Cardiomyoplasty technique: left latissimus dorsi muscle (LDM) transposed into chest through a window created by resecting the anterior segment of 2nd rib (5 cm). LDM is then wrapped around both ventricles. Sensing and pacing electrodes are connected to an implantable cardiomyostimulator.
A. Enlarged heart before LVR surgery. Dotted lines indicate slice to be removed by surgeon.

B. Heart after LVR surgery.
Click here for UTube
Artificial Heart animation!
Cardiac Transplantation

Nursing Management

• Treatment of choice for patients with refractory end-stage HF, inoperable CAD and cardiomyopathy

  – Goal of transplant evaluation process - identify patients who would most benefit from a new heart
Cardiac Transplantation

Nursing Management

• Transplant candidates - placed on a list
  – Stable patients wait at home and receive ongoing medical care
  – Unstable patients - may require hospitalization for more intensive therapy
  – **Overall waiting period** for a transplant is long; many patients die while waiting for a transplant
Cardiac Transplantation

Nursing Management

- **Surgery** involves removing recipient’s heart, except for posterior right and left atrial walls and their venous connections.
- Recipient’s heart replaced with donor heart.
- Donor sinoatrial (SA) node is preserved so that a *sinus rhythm* may be achieved postoperatively.
- **Immunosuppressive therapy** usually begins in operating room.
Click here to Perform a Heart Transplant...(your patient with end stage heart failure may require this!)
Cardiac Transplantation

Nursing Management

- **Infection**- primary complication followed by **acute rejection** in first year post transplantation
- After first year, malignancy (especially lymphoma) and coronary artery vasculopathy = major causes of death
Cardiac Transplantation

Nursing Management

• *Endomyocardial biopsies* - obtained from right ventricle weekly for first month, monthly for following 6 months, and then yearly to detect rejection
  – *Heartsbreath test* is used along with endomyocardial biopsy to assess organ rejection

• *Peripheral blood T lymphocyte* monitoring - assess recipient’s immune status

• Care focuses:
  – Promoting patient adaptation to transplant process
  – Monitoring cardiac function & lifestyle changes
  – Providing relevant teaching
PATIENT TEACHING
Chronic HF

Nursing Management

• Implementation: Patient education
  – Medications (lifelong)
  – Taking pulse rate
    • Know when drugs (e.g., digitalis, β-adrenergic blockers) should be withheld and reported to health care provider
Chronic HF

**Nursing Management**

- **Acute Intervention**
  - HF - progressive disease—treatment plans established with quality-of-life goals
  - Symptom management controlled with self-management tools (e.g., daily weights)
  - Salt - restricted
  - Energy- conserved
  - Support systems - essential to success of entire treatment plan
Chronic HF - Nursing Management

• Ambulatory and Home Care
  – Explain physiologic changes that have occurred
  – Assist patient to adapt to physiologic and psychologic changes
  – Integrate patient and patient’s family or support system in overall care plan

• Implementation: Patient Education
  – Home BP monitoring
  – Signs of hypo- and hyperkalemia if taking diuretics that deplete or spare potassium
  – Instruct in energy-conserving and energy-efficient behaviors
What’s New in Heart Failure?

Go here for updates on Heart Failure!

Go here for UTube videos- great visuals

HeartNet/Ventricular Support System

End Stage Heart Failure- newest Therapies

Muscle cell transplant (stem cell); Angiogenesis
10 Commandments of Heart Failure Treatment

1. Maintain patient on 2- to 3-g sodium diet. Follow daily weight. Monitor standing blood pressures in the office, as these patients are prone to orthostasis. Determine target/ideal weight, which is not the dry weight. In order to prevent worsening azotemia, some patients will need to have some edema. Achieving target weight should mean no orthopnea or paroxysmal nocturnal dyspnea. Consider home health teaching.

2. Avoid all nonsteroidal anti-inflammatory drugs because they block the effect of ACE inhibitors and diuretics. The only proven safe calcium channel blocker in heart failure is amlodipine (Lotrel /Norvasc).

3. Use ACE inhibitors in all heart failure patients unless they have an absolute contraindication or intolerance. Use doses proven to improve survival and back off if they are orthostatic. In those patients who cannot take an ACE inhibitor, use an angiotensin receptor blocker like irbesartan (Avapro).

4. Use loop diuretics (like furosemide [Lasix]) in most NYHA class II through IV patients in dosages adequate to relieve pulmonary congestive symptoms. Double the dosage (instead of giving twice daily) if there is no response or if the serum creatinine level is > 2.0 mg per dL (180 µmol per L).

5. For patients who respond poorly to large dosages of loop diuretics, consider adding 5 to 10 mg of metolazone (Zaroxolyn) one hour before the dose of furosemide once or twice a week as tolerated.
The 10 Commandments of Heart Failure Treatment

6. Consider adding 25 mg spironolactone in most class III or IV patients. Do not start if the serum creatinine level is > 2.5 mg per dL (220 µmol per L).

7. Use metoprolol (Lopressor), carvedilol (Coreg) or bisoprolol (Zebeta) (beta blockers) in all class II and III heart failure patients unless there is a contraindication. Start with low doses and work up. Do not start if the patient is decompensated.

8. Use digoxin in most symptomatic heart failure patients.

9. Encourage a graded exercise program.

10. Consider a cardiology consultation in patients who fail to improve.

ACE = angiotensin-converting enzyme.
WebMD- Patient Medications for Heart Failure!
Medical Treatment-Drug Therapy (typical)

- Cardiac Glycoside-Digoxin
- Positive inotropes-dobutamine, Primacor. Natrecor
- Antihypertensives- WHY
- ACE inhibitors- stops remodeling (pril or ril)
  - Catopril, enalapril, cozar, lisinopril
- Preload reduction *MSO4- important,
  - Vasodilators-nitrates
  - Diuretics-lasix, HCTZ, (Aldactone and Inspra)
  - Beta blockers- dec. effects of SNS (Coreg)
  - *Caution with CALCIUM CHANNEL BLOCKERS- dec cardiac contractility
**Meds!**

**Angiotensin-converting enzyme inhibitors**, such as captopril and enalapril, block conversion of angiotensin I to angiotensin II, a vasoconstrictor that can raise BP. These drugs alleviate heart failure symptoms by causing vasodilation and decreasing myocardial workload.

**Beta-adrenergic blockers**, such as bisoprolol, metoprolol, and carvedilol, reduce heart rate, peripheral vasoconstriction, and myocardial ischemia.

**Diuretics** prompt kidneys to excrete sodium, chloride, and water, reducing fluid volume. Loop diuretics such as furosemide, bumetanide, and torsemide are preferred first-line diuretics because of efficacy in patients with and without renal impairment. Low-dose spironolactone may be added to a patient's regimen if he has recent or recurrent symptoms at rest despite therapy with ACE inhibitors, beta-blockers, digoxin, and diuretics.

**Digoxin** increases the heart's ability to contract and improves heart failure symptoms and exercise tolerance in patients with mild to moderate heart failure.
Other drug options include nesiritide (Natrecor), a preparation of human BNP that mimics the action of endogenous BNP, causing diuresis and vasodilation, reducing BP, and improving cardiac output.

Intravenous (I.V.) positive inotropes such as dobutamine, dopamine, and milrinone, as well as vasodilators such as nitroglycerin or nitroprusside, are used for patients who continue to have heart failure symptoms despite oral medications. Although these drugs act in different ways, all are given to try to improve cardiac function and promote diuresis and clinical stability.
ER Decision-Making

Go here for physician discussion/decision-making re- The patient with heart failure in ER

<table>
<thead>
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<th>Variable</th>
<th>Sensitivity</th>
<th>Specificity</th>
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<td>Hx of HF</td>
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Sunday in the ER

Management Strategies for Acute Decompensated Heart Failure: From ER to Discharge

U.S. News

CRISIS IN THE ER
Heart Failure Case Study! (#1)
Complete and check your answers!

Patient with Shortness of Breath (#2)

Congestive Heart Failure (#3)

Heart failure case study (#4)

Heart Failure Challenge Game
Prioritization and Delegation (22)

- Two weeks ago, a 63 year old client with heart failure received a new prescription for carvedilol (Coreg) 3.125 mg orally. Upon evaluation in the outpatient clinic these symptoms are found. Which is of most concern?
  - A. Complaints of increased fatigue and dyspnea.
  - B. Weight increase of 0.5 kg in 2 weeks.
  - C. Bibasilar crackles audible in the posterior chest.
  - D. Sinus bradycardia, rate 50 as evidenced by the EKG.
The nurse is caring for a hospitalized client with heart failure who is receiving captopril (Capoten) and spironolactone (aldactone). Which lab value will be most important to monitor?

A. Sodium  
B. Blood urea nitrogen (BUN)  
C. Potassium  
D. Alkaline phosphatase (ALP)

C. Potassium
#24

As charge nurse in a long-term facility that has RN, LPN and nursing assistant staff members, a plan for ongoing assessment of all residents with a diagnosis of heart failure has been developed. Which activity is most appropriate to delegate to an LVN team leader?

A. Weigh all residents with heart failure each morning
B. Listen to lung sounds and check for edema weekly.
C. Review all heart failure medications with residents every month.
D. Update activity plans for residents with heart failure every quarter.

B. Listen to lung sounds and check for edema weekly
A cardiac surgery client is being ambulated when another staff member tells them that the client has developed a supraventricular tachycardia with a rate of 146 beats per minute. In what order will the nurse take these actions?

A. Call the client’s physician.
B. Have the client sit down.
C. Check the client’s blood pressure.
D. Administer oxygen by nasal cannula

B, D, C, A
The echocardiogram indicates a large thrombus in the left atrium of a client admitted with heart failure. During the night, the client complains of severe, sudden onset left foot pain. It is noted that no pulse is palpable in the left foot and that it is cold and pale. Which action should be taken next?

- A. Lower his left foot below heart level.
- B. Administer oxygen at 4L per nasal cannula.
- C. Notify the physician about the assessment data.
- D. Check the vital signs and pulse oximeter.

Notify the physician about the assessment data