Heart Failure

• Complex clinical syndrome resulting in insufficient blood supply/oxygen to tissues and organs
  – Diastolic or systolic dysfunction
  – Ejection fraction (EF) is amount of blood pumped by LV with each heart beat
    • Heart failure with reduced EF (HFrEF) and
    • Heart failure with preserved EF (HFpEF) to describe systolic and diastolic HF.

OXYGENATION NEEDS

• Ventilation
• Perfusion
• Diffusion

*What concept is involved?*
*How will you know when your patient is in a heart failure?*
*What nursing physical assessments are involved?*

Heart Failure

• ↑ In incidence and prevalence
  – Aging population
  – Costly, most common cause for hospital admission/readmissions in adults over age 65
  *manage at home*

• Primary risk factors: HTN, CAD, and MI
  – Why is this?
• Co-morbidities (DM, metabolic syndrome, advanced age, tobacco, and vascular disease) contribute to development of HF
Etiology of Heart Failure

- Anything that interferes with mechanisms that regulate cardiac output (CO):
  1. Preload
  2. Afterload
  3. Myocardial Contractility
  4. Heart rate
- Any changes in these factors can lead to decreased ventricular function and HF.

Primary causes - conditions that directly damage the heart
- Hypertension, including hypertensive crisis
- Coronary artery disease, including myocardial infarction
- Rheumatic heart disease
- Congenital heart defects (e.g., ventricular septal defect)
- Pulmonary hypertension
- Cardiomyopathy (e.g., viral, postpartum, substance abuse)
- Hypertrophic
- Valvular disorders (e.g., mitral stenosis)
- Myocarditis

Precipitating causes - conditions that increase workload of ventriles
- Anemia
- Infection
- Thyrotoxicosis
- Hypothyroidism
- Dysrhythmias
- Bacterial endocarditis
- Obstructive sleep apnea
- Pulmonary embolism
- Paget’s disease
- Nutritional deficiencies
- Hypervolemia

Audience Response Question
A patient with a history of chronic heart failure is hospitalized with severe dyspnea and a dry, hacking cough. Assessment findings include pitting edema in both ankles, BP 170/100 mm Hg, pulse 92 beats/minute, and respirations 28 breaths/minute. Which explanation, if made by the nurse, is most accurate?

a. "The assessment indicates that venous return to the heart is impaired, causing a decrease in cardiac output."
b. "The manifestations indicate impaired emptying of both the right and left ventricles, with decreased forward blood flow."
c. "The myocardium is not receiving enough blood supply through the coronary arteries to meet its oxygen demand."
d. "The patient’s right side of the heart is failing to pump enough blood to the lungs to provide systemic oxygenation."
### Pathophysiology

#### Left-Sided Heart Failure

- Left-sided HF (most common form of HF)
  - Results from inability of LV to
    - Empty adequately during systole
    - Fill adequately during diastole
- Blood backs up into left atrium and pulmonary veins
- Increased pulmonary pressure causes fluid leakage → pulmonary congestion and edema

- Further classified as
  - Systolic
  - Diastolic
  - Mixed systolic and diastolic

#### Systolic HF

- HFrEF – HF with reduced EF (<45%)
  - Normal EF is 55% to 60%
- Inability to pump blood forward
- Decreased LV ejection fraction (EF)
- Overtime LV dilated and hypertrophied
- Caused by
  - Impaired contractile function (MI)
  - Increased afterload (HTN)
  - Cardiomyopathy
  - Mechanical abnormalities (valve disease)

#### Diastolic HF

- HFpEF – HF with preserved EF (55-60%)
- Impaired ability of the ventricles to relax and fill during diastole, resulting in decreased stroke volume and CO
- LV stiff and noncompliant
- Caused by
  - left ventricular hypertrophy from hypertension, older age, female, diabetes, obesity
- End result of diastolic failure is same as systolic failure = pulmonary congestion

Pathophysiology
Mixed Heart Failure

• Mixed systolic and diastolic failure
  – Poor EFs (<35%)
  – High pulmonary pressures
  – Biventricular failure
    • Both ventricles may be dilated and have poor filling and emptying capacit
  – Seen in disease states such as dilated cardiomyopathy (DCM)

Pathophysiology
Right-Sided Heart Failure

• RV fails to pump effectively
• Fluid backs up in venous system
• Fluid moves into tissues and organs (peripheral edema, ascites, JVD)
• Left-sided HF is most common cause

Pathophysiology
Heart Failure in General

• Ventricular failure leads to:
  – Low blood pressure (BP)
  – Low CO
  – Poor renal perfusion
• Abrupt (as w/ acute MI) or subtle onset
• Compensatory mechanisms mobilized to maintain adequate CO
Compensatory Mechanisms

• Neurohormonal Response:
  • Renin-Angiotensin-Aldosterone-System (RAAS)
    – Homeostatic regulatory system
    – BP control and fluid and electrolyte balance
    – Fluid and sodium retained in response to stress
      • Causes vasoconstriction to ↑ BP
  • SNS – release catecholamines (epinephrine and norepinephrine)
    • ↑ HR
    • ↑ Myocardial contractility
    • Peripheral vasoconstriction

Compensatory Mechanisms

• As CO falls, blood flow to kidneys ↓ and is sensed as ↓ volume
  1. SNS is activated to ↑ BP and HR
  2. Release of aldosterone from adrenal cortex results in sodium and water retention
  3. Peripheral vasoconstriction and ↑ BP
  4. Pituitary gland releases ADH which results in water reabsorption
• Initially helpful, but outcome results in further water and sodium retention in an already overloaded state, and increased workload of the failing heart.

Compensatory Mechanisms

• Other factors contributing to development of HF
  • Endothelin is produced (stimulated by ADH, angiotensin II, and catecholamines)
    • Causes further arterial vasoconstriction and ↑ cardiac contractility and hypertrophy
  • Cytokines are released (proinflammatory)
    • Further depress heart function by causing hypertrophy, contractile dysfunction, and cell death (SIRs)
• Ventricular remodeling occurs as:
  • Continuous activation of neurohormonal responses (RAAS and SNS)
  • Hypertrophy of ventricular myocytes
  • Ventricle larger but less effective in pumping
  • Risk factor for life-threatening dysrythmias and sudden cardiac death
Compensatory Mechanisms

- **Hypertrophy**
  - Increase in muscle mass and cardiac wall thickness
  - Initially effective
  - Over time leads to poor contractility, increased O$_2$ needs, poor coronary artery circulation, and risk for ventricular dysrhythmias
  *Compare and contrast hypertrophy and dilation

Compensatory Mechanisms

- **Dilation**
  - Enlargement of chambers of heart that occurs when pressure in left ventricle is elevated
  - Initially effective
  - Eventually this mechanism becomes inadequate and CO decreases
  - Compare and contrast dilation and hypertrophy

Counterregulatory Mechanisms

- **Natriuretic peptides**
  - Atrial natriuretic peptide (ANP)
  - b-type natriuretic peptide (BNP)
  - RENAL & CARDIOVASCULAR EFFECT — diuresis, vasodilation, and lowered BP
  - HORMONAL EFFECT — inhibits SNS and RAAS
  
  - Cardiac vs respiratory dyspnea
  - BNP > 100 pg/ml
Compensatory Mechanisms

- **Compensated HF** occurs when compensatory mechanisms succeed in maintaining an adequate CO that is needed for tissue perfusion.
- **Decompensated HF** occurs when these mechanisms can no longer maintain adequate CO and inadequate tissue perfusion results.

*What will happen and what will you see?*

Acute Decompensated Heart Failure (ADHF) Clinical Manifestations

- **ADHF**
  - Sudden onset of signs and symptoms of HF
  - Requires urgent medical care
  - Pulmonary and systemic congestion due to ↑ left- and right-sided filling pressures
- Early → increased pulmonary venous pressure
  - Increase in the respiratory rate
  - Decrease in PaO₂
- Later → interstitial edema
  - Tachypnea
- Further progression → alveolar edema
  - Respiratory acidemia

Acute Decompensated Heart Failure (ADHF) Clinical Manifestations

- Can manifest as pulmonary edema
- Life-threatening situation – alveoli fill with fluid
- Most commonly associated with left-sided HF
Pulmonary Edema
Clinical Manifestations

• What will you see?
  – What about the skin?
  – What about breathing pattern?
  – What about lung sounds?
  – What about the HR? BP?

Acute Decompensated Heart Failure (ADHF)
Clinical Manifestations

• Based on hemodynamic and clinical status, patients can be categorized into one of four groups
  1. Dry-warm
  2. Dry-cold
  3. Wet-warm (most common)
  4. Wet-cold

Chronic Heart Failure
Clinical Manifestations

• Dependent on age, underlying type and extent of heart disease, and which ventricle is affected
• FACES
  – Fatigue
  – Limitation of Activities
  – Chest congestion/cough
  – Edema
  – Shortness of breath

Note: Fingertip-shaped impressions that do not rapidly refill after an examiner has exerted pressure.
Chronic Heart Failure
Clinical Manifestations

- Fatigue
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Tachycardia
- Edema
  - Dependent, liver, abdominal cavity, lungs
  - Edema may be pitting in nature
  - Sudden weight gain of >3 lb (1.4 kg) in 2 days may indicate ADHF, an exacerbation of chronic HF
- Nocturia
- Skin changes
- Behavioral changes
- Chest pain
- Weight changes

Heart Failure Complications

- Pleural effusion
- Dysrhythmias – atrial and ventricular
- Left ventricular thrombus
- Hepatomegaly
- Renal failure

Diagnostic Studies

- Determine and treat underlying cause
- Echocardiogram
  - Provides information on EF, heart valves and heart chambers
- ECG, chest x-ray, cardiopulmonary exercise stress test, heart catheterization, endomyocardial biopsy (EMB)
- BNP, serum electrolytes, BUN, creatinine, LFTs, CBC, cardiac biomarkers, coagulation studies, ABG
ADHF Interprofessional Care

- Continuous monitoring and assessment
  - VS, cardio/pulmonary, renal
- Hemodynamic monitoring if unstable
- Supplemental oxygen—what type?
- Mechanical ventilation if unstable
- High Fowler’s position
- Administer prescribed drugs
- I and O

- Daily weights
- Monitor edema
- Alternate rest with activity
- Provide diversionary activities
- Monitor response to activity
- Collaborate with OT/PT
- Reduce anxiety
- Evaluate support system
- Patient teaching

ADHF Interprofessional Care

- Ultrafiltration (aquapheresis) for patients with volume overload and resistance to diuretics
  - Intraaortic balloon pump (IABP)
  - Ventricular assist devices (VADs)
  - IABP and VADs as bridge to transplant (BTT) or as destination therapy (DT)
  - Implantable cardioverter-defibrillator (ICD)
  - Biventricular pacing/cardiac resynchronization therapy (CRT)
- Heart Transplantation—treatment of choice for patients with refractory end-stage HF, inoperable CAD, and cardiomyopathy

Audience Response Question

A patient with left-sided heart failure is prescribed oxygen at 4 L/min per nasal cannula, furosemide (Lasix), spironolactone (Aldactone), and enalapril (Vasotec). Which assessment should the nurse complete to best evaluate the patient’s response to these drugs?

a. Observe skin turgor
b. Auscultate lung sounds
c. Measure blood pressure
d. Review urine output
ADHF
Drug Therapy

• **Diuretics**
  – Decrease volume overload (preload)
  – Loop diuretics - Furosemide (Lasix)
• **Vasodilators**
  – Reduce circulating blood volume and improve coronary artery circulation
    • IV nitroglycerin
    • Sodium nitroprusside
    • Nesiritide (Natrecor)
• **Morphine**
  – Reduces preload and afterload
  – Relieves dyspnea and anxiety
• **Positive inotropes**
  – β-agonists (dopamine, dobutamine, norepinephrine (Levophed))
  – Phosphodiesterase inhibitor (milrinone)
  – Digitalis

Chronic HF
Interprofessional Care

• Main treatment goals
  – Treat the underlying cause and contributing factors
  – Maximize CO
  – Reduce symptoms
  – Improve ventricular function
  – Improve quality of life
  – Preserve target organ function
  – Improve mortality and morbidity

Chronic HF
Interprofessional Care

• Oxygen therapy
  – Relieves dyspnea and fatigue
• Physical and emotional rest
  – Conserve energy and decrease oxygen needs
  – Dependent on severity of HF
• Structured exercise program
  – CR associated with better outcomes
Chronic HF Drug Therapy

- Diuretics (Loop, Thiazide, K+ sparing)
- RAAS inhibitors
  - ACE inhibitors
  - Angiotensin II receptor blockers
- β-Blockers
- Vasodilators
- Combination therapy
- Positive inotropic agents

- What teaching will you include about these medications?

Chronic HF Nutritional Therapy

- Low sodium diet
  - Individualize recommendations and consider cultural background
  - www.nhlbi.nih.gov/health/dietary-approaches-to-stop-hypertension (DASH) diet
  - Sodium is usually restricted to 2 g/day

- Fluid restriction not generally required
  - If required, < 2L/day
    - Ice chips, gum, hard candy, ice pops to help thirst
- 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 should be reported to HCP

Chronic HF Planning

- Overall Goals
  - Decrease in symptoms
  - Decrease in peripheral edema
  - Increase in exercise tolerance
  - Compliance with the treatment regimen
  - No complications related to HF
Chronic HF Nursing Intervention

- Basic principles of care
  - HF is a progressive disease: establish treatment plans and quality-of-life goals
  - Use of self-management tools for symptom management
  - Restrict salt (and water at times)
  - Conserve energy
  - Maintain support systems

Chronic HF Patient Teaching

- Signs and symptoms of HF exacerbations – what to do/report
- Importance of early detection
- Can have positive outlook with chronic health problem if treatment plan is followed
- Drug therapy
- Dietary therapy
- Activity/rest

- Explain to patient and caregiver physiologic changes that have occurred
- Assist patient to adapt to both physiologic and psychologic changes
- Include patient and caregiver(s) in overall care plan

Acute/Chronic HF Nursing Diagnoses

- Impaired gas exchange
- Decreased cardiac output
- Excess fluid volume
- Activity intolerance