Heart Failure
A New Look at an Old Problem
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Background

- Critical Care Nursing
- Cardiology Nursing
- Family Practice Nurse Practitioner
- North Central Heart Institute
- Cardiology Nurse Practitioner
- American College of Cardiology (ACC) CVT Member
- ACC Nurse Core Competency Certification
Disclosures
Objectives

1) Understand basic pathophysiology related to Heart Failure
2) Understand the different terminology used to describe various Heart Failure conditions
3) Understand abnormal exam findings in Heart Failure
4) Understand basic guidelines to heart failure treatment
5) Identify new treatment modalities
Definitions

HEART FAILURE IS NOT A DISEASE

IT'S NOT
A TUMOR!!!
IT’S A CLINICAL SYNDROME

- End stage of many cardiovascular disorders

- Structural / Functional disorder that does 1 of 2 things
  - Impairs ability of ventricle to fill with blood
  - Impairs ability of ventricle to eject blood
Technical

- Abnormality of the cardiac function causes the heart to **FAIL TO PUMP BLOOD AT A RATE REQUIRED** by metabolizing tissue or when the heart can only do so with **ELEVATED FILLING PRESSURE**

Operational

- A clinical syndrome resulting from cardiac decompensation and characterized by signs and symptoms of **INTERSTITIAL VOLUME OVERLOAD** and / or **INADEQUATE TISSUE PERFUSION**
Decreased Cardiac Output

- **STROKE VOLUME** = volume of blood pumped from ventricle of the heart with each beat (70 ML in healthy young man)
Pathophysiology

- Contractility reduced by diseases that disrupt myocyte (muscle cell) activity

- Ventricular remodeling results in
  - Hypertrophy and dilation of myocardium
  - Causes progressive myocyte contractile dysfunction over time
  - Deposition of collagen between myocytes which disrupts integrity of the muscle

Contractility decreased → Stroke volume falls → LVEDP increases → Dilation of heart → Increased preload
Increased Afterload

- Most commonly from increased peripheral vascular resistance (PVR) ie. Hypertension, Aortic valvular disease

- Causes resistance to ventricular emptying

- Heart responds with hypertrophy of myocardium
  - Mediated by Angiotensin II and Catecholamines
  - Results in increased O2 and energy demand & thick myocardium

- ATP production impaired by myocytes with impaired mitochondrial (energy production) function
**Preload and Afterload**

**Preload**
Volume of blood in ventricles at end of diastole (end diastolic pressure)

Increased in:
- Hypervolemia
- Regurgitation of cardiac valves
- Heart Failure

**Afterload**
Resistance left ventricle must overcome to circulate blood

Increased in:
- Hypertension
- Vasoconstriction

↑ Afterload = ↑ Cardiac workload
Ventricular Remodeling

Myocardial dysfunction
- Myocardial infarction
- Ischemic heart disease
- Hypertension
- Other

↓ Cardiac output
↓ Systemic blood pressure

↓ Perfusion to kidneys

Baroreceptors activated
- Left ventricle
- Aortic arch
- Carotid sinus

Vasomotor regulatory centers in medulla stimulated

Sympathetic nervous system activated

↑ Catecholamines (epinephrine and norepinephrine)

Renin-angiotensin-aldosterone system activated

Angiotensin II
- Aldosterone released into kidneys
- Renin

Angiotensin I

Angiotensinogen

↑ Angiotensin II
↑ Aldosterone

- Retain sodium and water
- Arginine vasopressin
- Endothelin
- Cytokines (tumor necrosis factor-α)

Vasoconstriction
- ↑ Afterload
- ↑ Blood pressure
- ↑ Heart rate

Ventricular remodeling
- Hypertrophy and dilation of ventricle
- Genetically large cells
- Impaired contractility

Remodeled
Normal

Lungs

↑ Angiotensin II
↑ Aldosterone
Neuro-humoral/ Inflammatory/ Metabolic

RAAS (Renin Angiotensin Activating System)
Activation angiotensin II
- ↑ preload/ afterload
- Direct myocyte toxicity, induction of myocyte death, remodeling, down-regulation of adrenergic receptors, arrhythmias, potentiation of auto- immune effects on heart muscle.

Aldosterone
- Salt and water retention by kidneys
- Contributes to myocardial fibrosis
- Autonomic dysfunction
- Dysrhythmias
- Endothelium dysfunction
- Prothrombotic effects
Neuro-humoral/ Inflammatory/ Metabolic

Arginine vasopressin (Antidiuretic hormone ADH)
- Peripheral vasoconstriction
- Renal fluid retention
- Exacerbate hyponatremia and edema

Natriuretic Peptides (Atrial ANP, Brain BNP)
- Increased in heart failure
- Inadequate compensation in heart failure

Inflammatory Cytokines
- Endothelium hormones
- Endothelin potent vasoconstrictor
- Associated with poor prognosis in heart failure
- TNF-α –contributes to myocardium remodeling, down-regulates synthesis of Nitric Oxide (potent vasodilator)
Neuro-humoral/ Inflammatory/ Metabolic

Myocyte Calcium Transport
- Changes in intracellular transport implicated in decreased myocardial contractility

Insulin Resistance
- Cause abnormal myocyte fatty acid metabolism
- Abnormal generation of ATP
- Contributes to decreased myocardial contractility/ remodeling

Diabetes
- Disturbs calcium metabolism
- Causes oxidative stress
- Changes fatty acid and glucose metabolism
- Causes mitochondrial dysfunction
Old Nomenclature

Ischemic
- Left ventricle enlarged, dilated and weak
- Caused by ischemia - a lack of blood supply to the heart muscle caused by coronary artery disease and heart attacks
- Now falls under category of dilated (most common cause of dilated)

Non-ischemic
- These forms of cardiomyopathy are not related to coronary artery disease
New Nomenclature

Heart Failure with Reduced Ejection Fraction (HFrEF) = EF ≤ 40% (Systolic)

Heart Failure with Preserved Ejection Fraction (HFpEF) = EF > 40, 45, 50, 55% depending on the study (Diastolic)

(2013 ACCF/AHA Heart Failure Guidelines, Yancy, CW et. Al)
What is Cardiomyopathy?
Is Cardiomyopathy Heart Failure?
Is Heart Failure Cardiomyopathy?
Cardiomyopathy

- **Definition** = “Diseases of Heart Muscle”

- Term used for predominantly genetically determined diseases that have recognizable phenotypes

- **NOT THE SAME AS HEART FAILURE**

- May or may not manifest in clinical heart failure
Various forms of Dilated Cardiomyopathy

- Ischemic (Most Common)
- Idiopathic
- Endocrine / Metabolic (Obesity, Diabetic, Thyroid disease [hyper and hypo], Acromegaly / Growth Hormone Deficiency)
- Toxic (Alcoholic, Cocaine, Cancer therapy, Ephedra, Cobalt, Anabolic Steroids, Chloroquine, Clozapine, Amphetamine, Methylphenidate, Catechloamine)
- Tachycardia Induced
- Myocarditis (10% of unexplained)-most often post-viral
- AIDS
- Chagas’ Disease (Central & South America) occurs after Trypanosoma cruzi infection
- Inflammation-induced (Non-infectious) Hypersensitivity
- Rheumatological / Connective Tissue (Pericarditis, Pericardial effusion)
- Peripartum/ Postpartum
- Iron overload (Hemochromocytosis)
- Amyloidosis
- Sarcoidosis
- Stress (Takotsubo)
Types of Cardiomyopathy

- Dilated Cardiomyopathy (ischemic most common)

- Hypertrophic Cardiomyopathy (HOCM, IHSS)

- Restrictive Cardiomyopathy (Amyloid, Sarcoid, Carcinoid, Idiopathic)

- Arrhythmogenic Right Ventricular Dysplasia (ARVD) 
  [Genetic mutations of heart muscle proteins, frequently causes sudden cardiac death]

- Left Ventricular Non-Compaction Cardiomyopathy 
  (Congenital gene mutation, failure of embryonic heart muscle undergo compacting transformation)
Other Terminology

- Chronic
- Acute
- Acute on Chronic
- Compensated
- Decompensated
- Left Ventricular
- Right Ventricular
- Systolic
- Diastolic
- Cardiomyopathy
- Low-output
- High-output
Internal Anatomy of the Human Heart

Superior vena cava

Aorta

Pulmonary trunk

Right atrium

Left atrium

Pulmonary semilunar valve

Aortic semilunar valve

Tricuspid valve

Mitral / bicuspid valve

Right ventricle

Left ventricle

Inferior vena cava

Cardiac muscle
Normal Heart

Chambers relax and fill, then contract and pump.

Heart with Dilated Cardiomyopathy

Left Ventricles

Right Ventricles

Muscle fibers have stretched. Heart chambers enlarge.
Systolic Heart Failure
Heart failure with Reduced Ejection Fraction (HFrEF)

- Impaired contractility
- LV Cannot pump effectively during systole
- Blood backs up to pulmonary system
- Primary mechanism in dilated cardiomyopathy
Most common Causes of Systolic Heart Failure

- Coronary ischemia = 50-75%
- Valvular disease = 10-12%
- Idiopathic dilated
- Hypertension
Diastolic Heart failure
Heart Failure with Preserved Ejection Fraction (HFpEF)

- Normal or near normal LV function
- Abnormal LV filling
- Elevated LV filling pressures
- “Problem of relaxation”
- Elevated pressure transmitted to pulmonary system
- Don’t tolerate hemodynamic stress
  - Atrial fib / tachycardia
  - Hypertension
Left sided Heart Failure
- Predominately affect LV
- Usual causes are MI, HTN, valvular disease, global dysfunction
- **THINK PULMONARY CONGESTION**
  - Dyspnea
  - Orthopnea
  - Paroxysmal Nocturnal Dyspnea (PND)

Right sided Heart Failure
- Commonly the result of left sided failure
- Can occur independently
- RV infarct
- Acute Pulmonary Embolus
- **THINK SYSTEMIC CONGESTION**
  - Edema
  - Ascites
  - Hepatic congestion
  - Jugular venous distention
Low-output Heart Failure

- Not enough blood pumped to meet body metabolic needs
- Various systolic and diastolic HF

High-output Heart Failure

- Heart functions normally
- Unable to keep up with increased metabolic needs
  - Thyrotoxicosis
  - Anemia
  - A-V fistulas
  - Sepsis (↓ PVR)
Acute Heart Failure
- Sudden development of HF symptoms
- Most commonly with:
  - Myocardial infarct or ischemia
  - Severe hypertension
  - Sudden valve dysfunction (ischemic MR, ruptured AV or MV)

Chronic Heart Failure
- Has existed for a long time
- Chronic symptoms
- Well controlled symptoms on meds
Impact

- About 5.1 million people in the United States have heart failure.
- One in 9 deaths in 2009 included heart failure as contributing cause.
- About half of people who develop heart failure die within 5 years of diagnosis.
- Heart failure costs the U.S. ~ $32 billion each year. Total includes cost of health care services, medications, and missed days of work.
Deaths from Heart Failure Vary by Geography

Heart Failure Death Rates, 2007-2009
Adults Ages 35+, by County

Rates are spatially smoothed to enhance the stability of rates in counties with small populations.

ICD-10 codes for heart failure: I50; deaths with heart failure mentioned in any of the 20 listed causes of death on the death certificate.

Data Source: National Vital Statistics System and the U.S. Census Bureau
Risk Factors

- Coronary Artery Disease
- Smoking
- Hypertension
- Obesity
- Diabetes
- Valvular Heart Disease
Classification

Why Classify?

- Provides a way of defining stage of disease
- Useful information about the presence and severity of disease
  - New York Heart Association (NYHA)
  - American College of Cardiology (ACC) / American Heart Association (AHA)
New York Heart Association

- Most common
- Functional classification
- Exercise capacity
- Symptomatic status
**Class I**
- Symptoms only @ activity levels that would limit normal people (>7 mets ~ Carrying 24# up 8 steps)

**Class II**
- Symptoms with ordinary exertion, slight limitation

**Class III**
- Symptoms with less than ordinary exertion (walking 20-100 meters, comfort only @ rest)

**Class IV**
- Symptoms @ rest
- Emphasizes development and progression
- Can be used to describe individuals and populations
- Recognizes that risk factors and abnormalities of cardiac structure are associated with HF
- Stages are progressive
- Once patient moves to higher stage, regression to earlier stage not observed
- Progression in stages associated with reduced 5 year mortality
Class A
- Risk of Heart Failure - No symptoms

Class B
- Structural disease - No symptoms

Class C
- Structural disease with prior or current symptoms

Class D
- Refractory Heart Failure - Requires special intervention
Diagnosis

- No diagnostic test for Heart Failure
- Largely clinical diagnosis
- Based on history and exam
Clinical Assessment

- Establish diagnosis
- Assess acuity / severity
- Determine etiology
History

- Dyspnea
- Orthopnea / Paroxysmal Nocturnal Dyspnea (PND)
- Edema
- Hepatic congestion / ascites
- Fatigue / weakness (most pronounced with exertion)
- Angina
- Recent Flu-like symptoms (Viral myocarditis)
- Longstanding hypertension
- Alcohol Abuse
- Family history of unexplained cardiomyopathy, amyloid
- Low voltage EKG, heavy proteinuria
- LVH criteria on EKG or echo
- Medications-anti-arrythmic Norpace (disopyramide) Flecainide, Ca Channel blockers (esp. Verapamil), NSAID
- Recent transfusion (transfusional volume overload)
- History of chemotherapy (Adriamycin)
Physical Exam

Looking for signs of ↑ filling pressures
- Volume overload
- Ventricular enlargement
- Pulmonary Hypertension
- ↓ Cardiac output ~ ↓ Tissue perfusion
Vitals and appearance

- Resting sinus tachycardia (irregularly irregular pulse is suggestive of atrial fibrillation which frequently accompanies HF)
- Narrow pulse pressure (<25)
- Diaphoresis
- Peripheral vasoconstriction cool, pale sometimes cyanotic extremities
Volume assessment

Pulmonary Congestion
- Rales more prominent in acute or sub-acute
- Chronic HF is associated with increases in venous capacity and lymphatic drainage of the lungs
  - Rales are often absent even though the pulmonary capillary pressure is elevated.
  - Chronic elevation in pulmonary venous pressure can lead to pleural effusions.

Peripheral edema
- Swelling of the legs (which is more prominent when the patient is upright), ascites, scrotal edema, hepatomegaly, and splenomegaly
- + Hepatojugular reflux (>3cm)

Elevated jugular venous pressure
Heart sounds - An S3 gallop

- Extra heart sound that occurs soon after the normal two "lub-dub"
- S3 is thought to be caused by the oscillation of blood back and forth between the walls of the ventricles initiated by the inflow of blood from the atria
- Tensing of the chordae tendinae during rapid filling and expansion of the ventricle
- "KENTUCKY"
- Heart best at apex in left lateral decubitus position with bell
Pulsus Alternans

- Present in severe left ventricular failure
- Evenly spaced alternating strong and weak peripheral pulses
- Pathophysiology not well understood
- Severe ventricular dysfunction may be associated with variations in contractility secondary to shifts in afterload, preload, and electrical excitability
Precordial Palpation

- An apical impulse that is laterally displaced past the mid-clavicular line
- Parasternal lift
Diagnostic Testing
EKG

- Look for Cause
- Always Compare to Previous
- Ischemia- ST changes
Heart Block

- Common cause
- $1^\circ$ Atrial-Ventricular Block
- Left Bundle Branch Block
- Left Anterior Fascicular Block
- Complete Heart Block ($3^\circ$)
- Consider Sarcoidosis
Other EKG Findings in Heart Failure

- **Left Ventricular Hypertrophy (LVH)**
  - Secondary to HTN
  - Increased Ventricular Mass

- **Low Limb Lead Voltage**
  - Suggestive of infiltrative disorder such as amyloid

- **Low limb lead voltage + Precordial LVH =**
  - Idiopathic cardiomyopathy-Wide QRS, LBB

- **Persistent Tachycardia**
  - Rate related CM
Chest X-ray

- Cardiomegaly
- Evaluate for pulmonary edema
- Look for structural abnormality
Initial Blood Testing

- **Complete Blood Count (CBC)**
  - Looking for concurrent or alternate condition (Anemia/Infection)

- **Electrolytes / Renal Panel**
  - Low Sodium = Severe Heart Failure
  - Renal failure- may be caused by HF or contribute to HF
  - Needed for baseline prior to treatment

- **Liver function test (LFT)**
  - Elevated with hepatic congestion

- **Fasting Blood Sugar**
  - Diabetes underlying HF
Natriuretic Peptides

- Both assays have **very high negative predictive values** and are excellent for ruling out heart failure in patients with shortness of breath.

- Generated by myocytes in response to myocardial stretch.

- Levels of both BNP and NT-proBNP tend to increase with age.

- Levels of NT-proBNP and BNP may be increased in persons with kidney disease.

- Both BNP and NT-proBNP will rise with left ventricle dysfunction and either can be measured, they are not interchangeable and the results cannot be directly compared.
<table>
<thead>
<tr>
<th>NYHA Class</th>
<th>BNP</th>
<th>Severity</th>
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<tr>
<td></td>
<td>&lt; 100</td>
<td>No HF</td>
</tr>
<tr>
<td>I</td>
<td>100-300</td>
<td>Failure present</td>
</tr>
<tr>
<td>II</td>
<td>&gt;300</td>
<td>Mild</td>
</tr>
<tr>
<td>III</td>
<td>&gt;600</td>
<td>Moderate</td>
</tr>
<tr>
<td>IV</td>
<td>&gt;900</td>
<td>Severe</td>
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<tr>
<th>Interpretation</th>
<th>Age</th>
<th>NT-Pro BNP Range</th>
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<tr>
<td>Congestive Heart Failure likely</td>
<td>≤ 75</td>
<td>≥ 125 pg/mL</td>
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<tr>
<td></td>
<td>&gt;75</td>
<td>&gt; 450 pg/mL</td>
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Other labs

- **Thyroid studies** (especially with atrial fib)
- **Iron studies**- Screen for hemochromocytosis
- **ANA / RF**- Lupus, Autoimmune disorder
- **Viral serology**- Antimyosin antibody, Coxackie, Mycoplasma
- **24 hour urine** – R/O pheochromocytoma
- **Thiamine, Carnitine, Selenium**
Echocardiogram

- Especially in new onset
- Ventricular size and function (Ejection Fraction)
- Pericardial thickening
- Intra-arterial / Intra-ventricular shunts
- Abnormal myocardial texture- “sparkling” pattern= amyloid
- Right ventricular size and function
- Estimate Pulmonary pressures
- Diastology
Other testing

- Exercise testing
- Cardiac catheterization
- Cardiac MRI (CMR)
- CT Angiogram
- Endo-myocardial biopsy
- Genetic
Treatment
Goals of Therapy

- Clinical Improvement
- Reduction in Mortality Risk
- Reduce rate of hospitalizations for Heart Failure
Components of therapy

- Correction of systemic factors (Thyroid dysfunction, Infection, uncontrolled DM)

- Control of comorbidities (COPD, Sleep apnea)

- Lifestyle modification
  - Smoking cessation
  - ETOH restriction
  - Salt restriction (2-3 GM or less / Day)
  - Weight reduction (goal to be within 10% IBW)
  - Daily weight monitoring to detect fluid accumulation

- Review of meds that may contribute (NSAID, antiarrhythmic, Ca Channel block, thiazolidinedione)
Components of Therapy (cont.)

- Pneumococcal and yearly Influenza immunization
- Treatment of underlying Heart Disease
- Pharmacologic therapy
- Device therapy (Automatic implantable cardioverter-defibrilator AICD, Cardiac resynchronization therapy CRT/biventricular pacing)
- Specialized management (transplantation, LVAD)
- Comprehensive discharge planning for hospitalized patients
Acute Decompensated Heart Failure

REMEMBER…..

- Many conditions can cause cardiogenic pulmonary edema in the absence of heart disease
  - Primary volume overload-blood / fluid
  - Severe hypertension
  - Severe renal disease
Initial Stabilization

- Airway, Oxygen, Ventilation, Continuous Pulse Oximetry
- Vitals - Hypertension / Hypotension
- Continuous Cardiac Monitor
- IV access
- Seated Posture
- Diuretic Therapy
- Vasodilator
- Urine output

Aggressiveness depends on hemodynamics / volume status
Flash Pulmonary Edema r/t HTN

Aggressive Vasodilating therapy

Normotensive & Volume Overload

Diuretic + Vasodilation

Hypotensive/Volume Overload

DO NOT TOLERATE VASODILITATION

Diuretic +/- Inotropes
Oxygen / Ventilation

- Supportive Oxygen in absence of hypoxia **NOT RECOMMENDED**

- High Flow Non-Rebreather
- Noninvasive Positive Pressure Ventilation (NPPV, BIPAP)-Preferred if not contraindicated
  - Decreases need for intubation
  - Improves respiratory parameters
  - Especially beneficial with hypercapnia
    - Respiratory Distress
    - Respiratory Acidosis
    - Hypoxia

- Failed BIPAP
  - Intubation with PEEP
  - Titrate FiO₂ >90%
Diuretics

- ADHF = usually volume overload
- Improve symptoms and oxygenation
- Exceptions???
  - Severe hypotensive cardiogenic shock
  - Aortic stenosis - use with caution

ALWAYS USE IV AS INITIAL THERAPY

- Greater and more consistent drug availability
- GI edema may inhibit absorption
- Peak response 30min after dosing

- Titrate according to response
- If on chronic loop diuretic may need higher doses (2.5 X oral)
Hemodynamic Effects

Positive
↓ Intravascular volume, CVP, PCPW
Initial Morphine like effect (vasodilatation) mediated by prostaglandins

Negative
- Symptomatic hypotension-r/t lag in re-equilibrium of vascular volume
- HFpEF- more sensitive r/t ↓ preload
- Rising BUN / CREAT

- If substantial congestion persists- diuresis not working- May need Ultrafiltration / Dialysis
Inadequate Diuretic Response = Re-evaluate

- Sodium Restriction (<2 GM/ day)
- Double loop diuretics until euvolemia or max dose
- Add second diuretic
  - Chlorothalidone (50-100mg daily)
  - Metoloazone (2.5-5mg qod-qd)
  - Spironolactone (up to 100 daily)
  - Oral HCTZ (25-50 BID)
Sodium and Fluid Restriction
2GM/ Day (American Heart Failure Society of America)

Hospitalized patient a CAPTIVE AUDIENCE

- Indiscretions can be overridden by ↑ diuresis
- Reinforce salt restriction at home
- Predict discharge diuretic dose based on home diet
- Routine fluid restriction NOT recommended except with severe hyponatremia

Hyponatremia in Heart failure = Volume Overload

- Fluid restriction <2 Liters / day if Serum Sodium < 130
- Stricter if Sodium <125
Vasodilators

- Use with systemic fluid overload + No hypotension

- Nitroglycerine or Nipride + Diuretics = Rapid symptom relief with severe pulmonary edema

### Nitro
- Starting dose 5-10mcg/kg/min- can ↑ 5-10 every 3-5 min
- Contraindicated with phosphodiesterase inhibitor (Viagra, Cialis, Levitra)
- IV – fast, reliable, easily titrated

### Nipride
- 5-10mcg/kg/min-titrate every 5 min (5-400mcg/kg/min)
- Venous and arterial effects
- Potent, works very rapidly
- Keep MAP >65
- Metabolizes to cyanide→ toxic, only use 24-48 hr.
- Reflex tachycardia
- Rebound vasoconstriction when stopped
Ace Inhibitor

**MAINSTAY OF CHRONIC THERAPY FOR HF R/T SYSTOLIC DYSFUNCTION**

- Should be used in ALL patients with HFrEF unless contraindicated
  - Angioedema, pregnancy, bilateral RAS, hyperkalemia, severe hypotension
- Reduces risk of death and hospitalizations
- Whether its used in ADHF depends on if already on it
- For patients on chronically can usually continue if hemodynamically stable
- Stop or decrease if:
  - Hypotension
  - Acute Renal Failure
  - Hyperkalemia
  - Use cautiously with low BP
  - Can add once stable
- Angiotensin Receptor Blockers are alt. for pt. who don’t tolerate ACE
Beta Blockers

- Proven to reduce mortality
- Recommended for all patients with current or prior symptoms unless contraindicated
- Improves symptoms
- Enhances sense of well-being
- Benefits seen regardless of whether patient had CAD or DM
- Three beta blockers have been shown to have benefit
  - Bisprolol (Zebeta)
  - Metoprolol succinate (Extended release)- short acting less effective in HF
  - Carvedilol (blocks alpha 1, beta 1, and beta 2)
Beta Blockers

- Use with caution in ADHF
- Can worsen Acute HF
- May exacerbate bronchospasm
- Patients on chronic BB
  - May continue during decompensation if mild
  - Decrease or hold if mod-sever decompensation
  - HOLD IF RECEIVING INOTROPES
- Do not initiate in early management if BB naive
- Plan to initiate prior to discharge
- START LOW, GO SLOW!
Severe LV Systolic Failure

**Inotropes (Dobutamine/ Milrinone)**
- Low output failure (↓ peripheral perfusion, end organ dysfunction)
- Symptomatic hypotension despite adequate filling pressures
- Intolerant or unresponsive to vasodilator
- Not recommended unless normal filling pressures
- Monitor vitals frequently (stop if hypotension or tachycardia)
- Increase myocardial O2 demand- may provoke ischemia
- Exacerbates A-fib and Ventricular arrhythmias

**Vasopressors**
- Only used in persistent symptomatic hypotension and evidence of end organ damage
- Increases afterload
- Decreases cardiac output
Other Considerations

Aldosterone Antagonists (Spironolactone, Eplerone)
- Decrease mortality
- Used in long term management

DVT prophylaxis
- Heparin or Low Molecular Weight Heparin (Enoxaparin)
- Indicated if not already anti-coagulated and not contraindicated

Morphine
- Limited data
- May reduce anxiety and work of breathing
- Retrospective studies show ↑ frequency of need for mechanical ventilation & ↑ mortality

Ivabradine (Corlandor)
- Hyperpolarization-activated beta blocker
- Stable symptomatic HFREF <35%
- Resting HR > 70 on max tolerated BB or contraindication to BB
Mechanical Support
- Intra-Aortic Balloon Pump (IABP)
- LV assist device (bridge or destination)
- CI < 2, SPB < 90, PCPW > 18

Ultrafiltration
- Inferior to stepped pharm treatment
- Increased adverse events
- Used only if can’t achieve adequate response to aggressive diuresis
- Lifevest wearable defibrillator
- Implantable AICD
- Bi-Ventricular Pacemaker
- Bi-Ventricular AICD
Transplant

Contraindications

- Age >70
- Organ failure
- Severe Pulmonary Hypertension
- Malignancy within 2 years
Treatment of Diastolic Heart Failure

- TREATMENT OF UNDERLYING PROCESS
  - Hypertension (ACE, ARB, CA Channel, BB)
  - Diuretics to control volume overload
    - Careful use to prevent excessive preload reduction = hypotension
  - CAD / Ischemia
  - CPAP for sleep apnea
  - Weight loss for obesity
- Slowing heart rate to allow for longer filling times
  - Beta Blockers
  - Ca Channel Blockers
- Studies do not demonstrate any morbidity / mortality benefit with use of Beta blockers, ACE, ARB as they do in Systolic HF

- MORE STUDY NEEDED
Pitfalls

- Under-diuresis
- All peripheral edema is not heart failure
- Don’t forget to teach dietary management and early symptom recognition
- Recognizing diastolic dysfunction
What’s New?

- Remote electronic monitoring for outpatients
- Cardiomems-Implantable PA pressure monitor
- Increase in placement of destination LVADs
- Lifevest wearable defibrillator
- Implantable Defibrillator AICD
- Bi-Ventricular Pacemakers
- Bi-ventricular AICD
Summary

- Heart Failure is a significant problem within the United States
- Heart Failure pathophysiology involves a constellation of factors
- Having a better understanding will improve your ability to care for your patients
- Honing history and exam skills can help to identify early onset of Heart Failure
- Lab and imaging can be useful in diagnosing and monitoring Heart Failure
- Understanding the classification system helps objectify and risk stratify your patients
Correction of systemic factors, control of comorbidities, lifestyle modification, pharmacologic and device therapy are all mainstays of therapy.

Systolic and Diastolic Heart failure differ in pathophysiology and treatment.

Ace Inhibitors, Diuretics, and Beta Blockers are the foundation of Systolic Heart Failure treatment.

Prevention and treatment of the underlying causes of Diastolic Heart failure are the current best practices, however more study is needed.
Questions?
References


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http://www.cdc.gov/heartdisease/facts.htm


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Uptodate.com

Thank you