Heart Failure
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Demographics—CHF in the United States

- Nearly 1,100,000 hospital admissions for primary diagnosis of heart failure
  - Accounts for ~ 6.5 million hospital days
- More than 12M - 15M office visits
- 6-month readmission 33-50%
- One year mortality ~ 5-15%
  - Systolic dysfunction 10-15%
  - Diastolic dysfunction 5-8%
- Five-year mortality rate approaches 50%
  - 6-year mortality (80% for men; 65% for women)
- In-hospital mortality
  - 2.8% in diastolic vs. 3.9% in systolic dysfunction, p=0.005

Advanced Heart Failure
- Demographics & Etiology
- What puts the “Congestion” in Heart Failure??
- Diagnostic Tools
- Systolic or Diastolic?
- Management & Treatment Options

Etiology
- CAD / Ischemic Myocardial Disease
  - 32% of all HF patients have suffered at least one heart attack
- Hypertension
  - 72% of patients reported hx. of chronic HTN (ADHERE)
- Cardiomyopathy
  - Hypertrophic, dilated, restrictive, idiopathic, post-partum, viral, bacterial
- Valvular disease
- Temporary Causes
  - Cardiac tamponade
  - Cardiac dysrhythmias
- Comorbidities complicating HF management
  - Diabetes: 44%
  - Renal insufficiency 31% (Mean Cr (male) 3.8mg/dL; (female) 2.5mg/dL)
What puts the “Congestion” in Heart Failure?

CHF...A Syndrome of Congestive Symptoms

• Because the ventricle is inadequately emptied, ventricular end-diastolic pressure and volumes increase. This is transmitted to the atrium.
• On the left side of the heart, the increased pressure is transmitted to the pulmonary vasculature, and the resultant hydrostatic pressure leads to extravasation of fluid into the lung parenchyma, causing pulmonary edema.
• On the right side of the heart, the increased pressure is transmitted to the systemic venous circulation and systemic capillary beds, leading to extravasation of fluid into the tissues of target organs and extremities, resulting in peripheral edema.

Clinical Manifestations “Left-sided Heart Failure” Pulmonary Congestion

• Acute pulm. edema
• Dyspnea
• Orthopnea
• Paroxysmal Nocturnal Dyspnea
• Pulmonary-cough, crackles, tachypnea, pulmonary congestion, pulmonary edema
• Bloody sputum
• Cardiomegaly
• Presence of S3
• Tachycardia (w/PVCs)
• Weakness, fatigue
• Decreased cardiac output
• Hypotension
• Peripheral hypoperfusion
• RV failure

Clinical manifestations “Right-sided Heart Failure” “Body” Congestion

• Peripheral edema
• Jugular venous distension (JVD) > 3cm
• Fatigue, lethargy
• Hepatic congestion-palpable liver, ascites
• Anorexia, GI Distress
• Wt gain (usually 2-3 pounds “overnight”)
• RV failure usually occurs as a result of LV failure and pulmonary congestion
• Without these factors, is a result of:
  – RV infarction
  – Pulmonary hypertension

Examination

Diagnosis of ADHF

• Rales / pulmonary edema / SOB
• Tachycardia (HR x SV = CO)
• Peripheral edema
• S3 gallop and/or murmur suggesting obstructive or regurgitant valvular disease
• JVD (> 3cm) or other evidence of increased LV filling pressure (hepatojugular reflex, ascites, hepatomegaly)
• Increased LV filling pressure [PAOP] confirmed by pulmonary artery catheter

Advanced Heart Failure

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Diagnosis

- There is no single diagnostic test for heart failure because it is largely a clinical diagnosis based on careful history and physical examination
  - Symptom assessment
  - Exercise tolerance [VO2 stress test; 6 min. walk]
  - CXR
  - Cardiac ECHO
  - BNP (natriuretic peptide)
  - Invasive hemodynamic monitoring [PA catheter]
  - Cardiac catheterization [right heart cath for CVP/PAOP]
  - MUGA—pharmacologic stress
  - Cardiac MRI

Heart Failure Classification: NY Heart Association

- **Class I:** No limitation in physical activity
- **Class II:** Comfortable at rest, but symptoms with ordinary activity—slight limitation in physical activity
- **Class III:** Comfortable at rest, but symptoms with less than ordinary activity—marked limitations in physical activity
- **Class IV:** Symptoms at rest—unable to carry out physical activity without discomfort or symptoms

Cardiac Natriuretic Peptides in HF

- BNP (B-type Natriuretic Peptide)
  - Primarily secreted by the ventricles in response to increased volume
  - Body’s own “defense” against pressure and volume increases
  - Reduce preload by:
    - Increase venous capacitance (venous vasodilitation)
    - Promotion of diuresis
  - By virtue of their counter-regulatory nature, BNP levels should be able to act as an index of the severity of left ventricular dysfunction
    - BNP levels correlate to the degree of heart failure

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Systolic v. Diastolic Heart Failure

Is the problem filling the ventricle

or

is the problem emptying the ventricle?
Systolic Dysfunction

- Defect in the expulsion of blood from the ventricles into the aorta /pulmonary artery
- “Dilated” ventricle
- Inadequate emptying of the ventricles decreased inotropy [forward failure]

Causes:
- CAD/Infarction (decreased myocardial contractility)
- Viral, bacterial, post-partum, ETOH/drug
- Idiopathic dilated cardiomyopathy

Diastolic Dysfunction

- Preserved ejection fraction (EF > 40%)
- “Non-dilated” ventricle
- Occurs when the filling of the ventricles is impaired
- ACC/AHA guidelines suggest a diagnosis based on typical symptoms of HF in the setting of a normal LVEF & no valvular abnormalities

Causes:
- Chronic hypertension
- Hypertrophic cardiomyopathy
- Aortic stenosis
- Scarred heart muscle (s/p MI)

Advanced Hemodynamics

- CVP: 2-6 RV preload [volume]
- PAOP: 5-12 LV preload [volume]
- PVR: <250 RV afterload [resistance]
- SVR: 800-1400 LV afterload [resistance]
- PAS: 15-30 PAH if PAM > 25mmHg & PAOP < 15
- PAD: 8-15 [equal to PAOP in absence of lung dx]
- CO: 4-8 L/min stroke volume x HR
- CI: 2.5-4.0 L/min/m2 CO/BSA
- LVEDV: 120-130 ml end diastolic volume
- SV: 60-70ml stroke volume
- EF: >55% Ejection fraction (SV/EDV)

Systolic vs. Diastolic?

CO = HR x Stroke Volume

CD: 3.6L/min = Heart Failure symptoms
HR: 90 beats/min
SV: 40 mL/beat

Systolic Heart Failure

Dilated ventricle
LVEDV: 200mL
EF: 40mL/200mL = 20%

Diastolic Heart Failure

Restrictive/Hypertrophic
LVEDV: 80mL
EF: 40mL/80mL = 50%
Comparison of Systolic & Diastolic HF

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Systolic HF</th>
<th>Diastolic HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>HF Symptoms</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Neurohormonal Activation (BNP)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Volume Overload</td>
<td>Reduced</td>
<td>Increased</td>
</tr>
<tr>
<td>LV Mass</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>LA Size</td>
<td>Reduced</td>
<td>Increased</td>
</tr>
<tr>
<td>LV End-Diastolic Volume</td>
<td>Increased</td>
<td>Reduced</td>
</tr>
<tr>
<td>LV End-Diastolic Pressure</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Exercise Capacity</td>
<td>Reduced</td>
<td>Reduced</td>
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<tr>
<td>Cardiac Output Augmentation</td>
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Angiotensin Converting Enzyme Inhibitors (ACE-I)
- Prevents worsening of heart failure by blocking the RAAS compensatory mechanism
  - Prevents conversion of angiotensin I to angiotensin II
- Vasodilator—decreases SVR & PVR
- Increases CO, ex. tolerance, coronary & cerebral blood flow
- Increased renal blood flow—diuresis & natriuresis
- “PRIL” suffix
  - Captopril (Capoten)
  - Enalapril (Vasotec)
  - Ramipril (Altace)
  - Lisinopril (Prinivil/Zestril)

Angiotensin II Receptor Blockers (ARBs)
- Used typically when ACE-I are contraindicated
- Blocks Angiotensin-II receptor
- Valsartan
- Losartan
- Irbesartan
- Candesartan/HCTZ (Atacand)

Medications in the Heart Failure Arsenal

- Angiotensin II Inhibition (ACE-I or ARB)
- Beta-Blockers
- Diuretics
- Digoxin (Class IIa: benefit > risk)
- Intravenous Positive Inotropic Agents
  - Dobutamine
  - Milrinone
- Aldosterone antagonists
- Calcium Channel Blockers
- Vasodilators: IV-NTG, Oral-hydralazine/nitrates
- Natriuretic Peptides (Acute Decompensated HF)—Nesiritide (Natrecor)
Diuretics

- Thiazide diuretics - work in ascending loop of Henle. First line diuretic in heart failure.  
  - HCTZ
- Loop diuretics - work in thick segment of ascending loop. Very potent diuretic, used in most patients with heart failure. **WATCH for hypokalemia**  
  - Lasix, Bumex
- K-sparing - work in distal convoluted and collecting tubules. Not strong enough on its own for diuresis, but commonly used as an adjunct therapy. **WATCH for hyperkalemia.**  
  - Spironolactone

**Effects of Diuretics on Neurohormones**

- **Direct effects of diuretics:**  
  - Natriuresis, diuresis  
  - Electrolyte excretion: K⁺, Ca²⁺, Mg²⁺
- **Indirect effects of diuretics:**  
  - Volume depletion, decreased circulating volume  
  - Decreased renal perfusion and increased release of antidiuretic hormone  
  - Increased renin production, RAAS/sympathetic activation  
  - Metabolic alkalosis

**Diuretics and Heart Failure**

There have been few long-term studies of diuretic therapy for the treatment of heart failure and, thus, its effects on morbidity and mortality are not known.

- Patients may become unresponsive to high doses of diuretics if they:
  - Consume large amounts of dietary sodium  
  - Are taking agents that can block the effects of diuretics (NSAIDs)  
  - Have significant impairment of renal function or perfusion
- Diuretic resistance can generally be overcome:
  - By the IV administration of diuretics  
  - The use of two or more diuretics in combination

**Beta Blockers**

- Selective inhibition of B1 stimulation in the heart
- Negative chronotropic and inotropic effects  
  - May be especially helpful in patients with tachycardia or atrial fibrillation
- Reduction in renin secretion from the kidneys
- Directly reduces sympathetic activity
  - Propranolol  
  - Atenolol  
  - Esmolol  
  - Metoprolol  
  - Carvedilol (has additional α-blocking activity)  
  - Labetalol (has additional α-blocking activity)

**Intravenous Positive Inotropes**

- **Dobutamine** – stimulates beta-1 receptors  
  - Positive inotrope and chronotrope  
  - 2.5-10.0 mcg/kg/min
- **Milrinone**  
  - Positive inotrope and vasodilator  
  - 0.375-0.75 mcg/kg/min
- Patients should be acute decompensated or refractory to other therapy before adding these agents

**Conventional Treatments of Acute Heart Failure**

- **Diuretics**  
  - Reduce volume preload
- **Vasodilators**  
  - Decrease preload and/or afterload
- **Inotropes**  
  - Augment contractility

Stroke Volume

Stroke volume (SV) depends on several key factors including preload (end-diastolic volume), afterload and contractility. Valvular dysfunction and ventricular geometry can also affect stroke volume.

Preload
- The degree to which the ventricles are stretched prior to contracting (end-diastolic volume/poressure).
- An increase in circulating volume will increase preload. Decreased venous return has the opposite effect, causing a reduction in stroke volume.

Afterload
- Resistance to forward flow from the ventricles.
- An increased afterload will hinder the ventricles in ejecting blood, causing reduced stroke volume.

Contractility
- Measures the speed and force of contraction (inotropy)

Advanced Hemodynamics

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<td>stroke volume</td>
</tr>
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<td>end diastolic volume</td>
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Case Study

- 53 y.o. Caucasian male
- Present to the OSUMC ED with increased DOE (2-3 wks) with walking just a few steps
- Increased edema 3-4+ up to scrotum/abdomen/BLE & BUE
- D/C’d six days ago from FL hospital for CHF exacerbation while on vacation

Patient Education!!!

- Counseling: Life-long disease
- Symptoms and symptom management
- Dietary Restrictions (2-3gm Na; Fluid Restriction)
- Control Blood Pressure
- ETOH abstinence
- Smoking Cessation
- Exercise--Cardiac Rehab
  - Improve exercise capacity
- Depression
- Compliance with medications, diet, activity, etc.

Case Study

- Vitals
  - 98.0 – 95 – 28 – 110/57
  - SaO2 94% on JI
  - Weight: 156.5 kg
  - Sinus rhythm (0-2PVCs/min); sinus tachy with activity
- CXR
  - Cardiomegaly
  - Positive pulmonary congestion
- ECHO
  - EF 20%
  - 3+ mitral regurgitation
  - Dilated LV
Q & A

- Is this Systolic or Diastolic heart failure?
- What else would you like to know?
  - Labs (Chem 7; CBC; BNP)
    - K 3.5
    - Na 133
    - Hgb 12.1
    - BNP 1220

- Troponins—negative
- Any signs of infarction?

Case Study

- Medications on admission
  - Lasix 80mg PO BID
  - NPH 30u AM / 35u PM
  - I:CHO w/ correction factor
  - Lisinopril 10mg QD
  - Lopressor 25mg BID
  - KCL 20 QID

Interventions--day 1

- IV nesiritide 0.01 mcg/kg/min.
- IVP Lasix 80 mg q 12 h
- Diet:
  - ADA 2gm Na+
  - 2000mL fluid restriction
- Daily weights / I&Os

Left ventricular failure can be caused by which of the following?

1. Aortic regurgitation and hypertension
2. Aortic stenosis and COPD
3. Mitral regurgitation and pulmonary embolus
4. Mitral stenosis and AMI

A Direct Effect of Sodium Nitroprusside (Nipride) is

1. Decrease stoke volume
2. Increase venous return
3. Decrease afterload
4. Increase PVR
Which of the following parameters indicates successful management of right ventricular failure?

1. Decreased CVP
2. Decreased PAOP
3. Increased PAD
4. Increased RV pressure

Answer Now

A patient admitted with CHF develops hypotension, tachycardia, decreased urine output, cool clammy skin, decreased LOC, and tachypnea. Which of the following should be included in the patient’s plan of care?

1. Positive inotropes, diuretics, vasodilators
2. ACE-inhibitors, adenosine, B-blockers
3. B-blockers, diuretics, calcium-channel blockers
4. Negative inotropes, digoxin, antiarrhythmics

Answer Now

Which of the following should the nurse assess for complications related to an arteriogram?

1. Renal function
2. Acid-base balance
3. Liver enzymes
4. Mean arterial pressure

Answer Now

Which of the following dysrhythmias is most frequently associated with CHF?

1. Ventricular bigeminy
2. Second-degree Type-II AV block
3. Junctional tachycardia
4. Atrial fibrillation

Answer Now

When pulmonary hypertension and heart pressure measurement are desired, which of the following is the best diagnostic test?

1. Left-sided heart cath
2. Transthoracic ECHO
3. Right-sided heart cath
4. Transesophageal ECHO

Answer Now