Heart Failure (I)

Ronald Zolty, MD, PhD
Case Presentation

• 42 year old African American man, who recently moved from Kansas
• PMH: HTN diag 3 years ago
• Diagnosed in May 07 with severe CHF

Echocardiogram
– LVEDD 6.6cm, LVEDS 5.6
– Severe global hypokinesis, LVEF 30%
– Moderate right ventricular hypokinesis
– Severe MR, mod-severe TR
Case Presentation

- Coronary Angiogram: Clean cors
- LV Angiogram: LVEF 27%, MR3+
- PSH: None
- FMH: unremarkable
- Allergy: NKDA
Case Presentation

- MEDS:
  - Cozaar 100 mg QD
  - Atenolol 50 mg QD
  - Spironolactone 50mg QD
  - Lasix 80 mg BID
  - Amlodipine 10 mg QD
Case Presentation

PE

- BP 95/60 HR 88, RR 20, Anicteric, Afebrile Acyanotic
- JVP ~ 6-8cm
- Chest: Clear, no crackles, no rales, no wheezing
- CV: RRR, PMI displaced
- Abd: Soft, 0 tenderness, 0 HSM
- Legs: No edema
Case Presentation

LAB

6.8  15  177
  51

134  98  21
  89
  3.6  24  1.2

LFTs: Normal
UA: WNL

EKG: Normal Sinus Regular Rhythm, LBBB QRS 150 msec

CXR: Cardiomegaly, no infiltrates
Case Presentation

• Adequate Rx ? (6 mistakes)
• What is the next step ?
• Is Pt a candidate for Mitral Valve Repair ?
• Does Pt need Transplant evaluation ?
Heart failure

• Heart Failure an Important Health Problem
• Pathophysiology
  – Diastolic LV Dysfunction
  – Systolic LV Dysfunction
• Clinical Presentation in Patients with HF
• Assessment of Heart Failure
• Hemodynamics
Heart Failure Definition

• Clinical Syndrome characterized by:
  – Impaired left ventricular performance (systolic, diastolic or both)
  – Reduced functional capacity
  – Neurohormonal dysregulation
  – Fluid retention
  – Impaired quality of life
  – Increased mortality
Heart Failure: an Important Health Problem

- Approximately 10 million Americans have HF (~1.5% of the US population)
- Incidence of HF has markedly increased over last three decades (250,000 in 1970, currently 550,000 new cases annually).
- Is associated with aging: incidence and prevalence increase in elderly population (Incidence of 10/1000>65 years of age).
- Hospital discharges 1,000,000 (2001).
- Single largest expense for Medicare.
- Five-year mortality rate as high as 50%.

AHA 2001, Heart and Stroke Statistical Update
Increasing Prevalence of HF


Heart Failure: an Important Health Problem

Prevalence of CHF, by Age, 1988-91

Congestive Heart Failure: an Important Health Problem

Deaths From Congestive Heart Failure, 1968 to 1993

Deaths (Thousands)

Year

ICD Code 428.0.
The sharp drop occurring in 1989 is attributed to revision of the death certificate.

Increase in Coronary Artery Disease as Etiology of Heart Failure Over Four Decades

<table>
<thead>
<tr>
<th>% CAD Absent</th>
<th>% CAD Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>78</td>
<td>22</td>
</tr>
<tr>
<td>64</td>
<td>36</td>
</tr>
<tr>
<td>47</td>
<td>53</td>
</tr>
<tr>
<td>33</td>
<td>67</td>
</tr>
</tbody>
</table>

n = 652

Ho KK et al Circulation. 1993; 88: 107-115
Relation Between Severity of Heart Failure and One-year Mortality

<table>
<thead>
<tr>
<th>NYHA Functional Class</th>
<th>Annual Mortality (%)</th>
<th>Sudden Death (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>II</td>
<td>5-15</td>
<td>50-80</td>
</tr>
<tr>
<td>III</td>
<td>20-50</td>
<td>30-50</td>
</tr>
<tr>
<td>IV</td>
<td>30-70</td>
<td>5-30</td>
</tr>
</tbody>
</table>

NEW YORK HEART ASSOCIATION (NYHA) FUNCTIONAL CLASS

Class I: No limitation of functional activity

Class II: Slight limitation of activity. Dyspnea and fatigue with moderate physical activity

Class III: Marked limitation of activity. Dyspnea with minimal activity

Class IV: Severe limitation of activity. Symptoms are preset even at rest
Etiology of Sudden Death

Heart rhythm recorded at onset of sudden death in 20 patients with HF awaiting heart transplant

Luu et al. Circulation, 1989; 80:1675
Heart Failure in Men vs. Women

<table>
<thead>
<tr>
<th></th>
<th>MEN</th>
<th>WOMEN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual Incidence (Age &gt;45)</td>
<td>7.2/1000</td>
<td>4.7/1000</td>
</tr>
<tr>
<td>Median Survival (excluding &lt;90 day mortality)</td>
<td>3.2 years</td>
<td>5.4 years</td>
</tr>
</tbody>
</table>
Total HF Costs (Calculated)

Health Care Financing Administration. Medicare Provider Analysis and Review (MEDPAR) Inpatient Hospital Fiscal Year 1997

Estimate calculated at 4.93% of National Health Expenditures
Comparison of Health Care Financing Administration expenditures of HF compared with cancer and MI according to Medicare Program

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Heart Failure due to Cardiomyopathy

Cardiomyopathy is a disease of the heart muscle

<table>
<thead>
<tr>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Dilated</td>
<td>• Ischemic</td>
</tr>
<tr>
<td>• Hypertrophic</td>
<td>• Valvular</td>
</tr>
<tr>
<td>• Restrictive</td>
<td>• HTN</td>
</tr>
<tr>
<td>• Arrhythmogenic</td>
<td>• Others</td>
</tr>
<tr>
<td>• Unclassified</td>
<td></td>
</tr>
</tbody>
</table>

Heart Failure = Forward and/or Backward Heart Failure

**FORWARD Heart Failure**
- Ejection of blood into aorta and pulmonary artery
- Inadequate Tissue Perfusion

**BACKWARD Heart Failure**
- Emptying of venous reservoirs
- Circulatory Congestion

*Often Coexist*
Left vs. Right Sided Heart Failure

- **Left Heart Failure**
  - Increased Pulmonary Artery Pressure
  - Decreased Cardiac Output
  - Dyspnea
  - Pulmonary Congestion
  - Pulmonary Edema

- **Right Heart Failure**
  - Increased Right Atrial Pressure
  - Decreased Cardiac Output
  - Fatigue
  - Renal Insufficiency
  - Poor Mentation
  - Edema
  - Renal Insufficiency
  - Hepatic Insufficiency
Normal and Heart Failure
Hemodynamics

Normal

- CO = 5.0
- SVR = 1000
- HR = 80
- PA = 25/10
- PCW = 6
- RA = 4
- RV = 25/6
- LV = 120/8
- EF = 65%

Decompensated Heart Failure

- CO = 3.2
- SVR = 1500
- HR = 120
- PA = 55/30
- PCW = 30
- RA = 16
- RV = 55/18
- LV = 90/35
- EF = 20%
**Useful Definitions**

**PRELOAD**
- Ventricular *end-diastolic* pressure and volume that determine resting sarcomere length just prior to contraction.

**AFTERLOAD**
- Pressure and volume *after the onset of contraction* and therefore, the resistance that the ventricle must overcome to eject its contents.
Factors Influencing Cardiac Output

Contractility

Afterload

Stroke Volume

Cardiac Output

Vascular Tone

Preload

Blood Volume

Heart Rate

(+)

(-)

(+)

(+)

(+)

(+)

(+)

Frank-Starling Curve

Heart Failure results in a downward shift of the curve resulting in hypoperfusion (b), pulmonary congestion (c), or both.
Ventricular Pressure-Volume Relationships

The right and left ventricle are phasic circulatory pumps that convert biochemical energy into mechanical pressure-volume work.

Phases of the cardiac cycle which occur with heart beat (1) Isovolemic systole, (2) Systolic ejection, (3) Isovolemic relaxation, (4) Diastolic filling.
In LV systolic dysfunction, the end-systolic pressure-volume curve moves from 1 to 2. This leads to a decrease in systolic pressure, stroke volume despite compensatory increase in the operating point on the diastolic pressure-volume curve.
Pressure-Volume Loop in Diastolic Dysfunction

In LV diastolic dysfunction, the diastolic pressure-volume curve shifts from 1 to 2. This leads to an increase in diastolic pressure, a decrease in end-diastolic volume, and a decrease in ventricular stroke volume.
Pathophysiology of LV Diastolic Dysfunction

Decreased left ventricular relaxation, increased passive chamber stiffness, or both will lead to an upward shift in the left ventricular diastolic pressure-volume curve and LV diastolic dysfunction.
Pathophysiology of LV Diastolic Dysfunction

**ABNORMAL RELAXATION**
Ventricular relaxation affects isovolemic relaxation, rapid ventricular filling, and mid-diastolic filling. Abnormal relaxation will result in an increase in LV pressure especially in the first two-thirds of diastole.

**INCREASED CHAMBER STIFFNESS**
Increased chamber stiffness implies a change in the passive stretch properties of the LV muscle. It will predominantly affect the last two thirds of diastole.
In LV diastolic dysfunction, the diastolic pressure-volume curve shifts from 1 to 2. This leads to an increase in diastolic pressure, a decrease in end-diastolic volume, and a decrease in ventricular stroke volume.
Of the patients presenting with pulmonary edema, ~ half had preserved LV function by echocardiogram and MUGA

Etiology of Heart Failure in the SOLVD Registry

- Idiopathic: 12.9%
- Other: 11.3%
- Hypertension: 7.2%
- Ischemic Heart Disease: 68.6%

What are the Causes of Diastolic Dysfunction

Diagram:
- Is left ventricular wall thickness increased (>11mm)?
  - Yes:
    - Hypertensive Heart Disease
    - Yes
    - No
  - No: Coronary Artery Disease
  - Hypertrophic or Restrictive Cardiomyopathy
Neurohormonal Activation in LV Systolic Dysfunction

Beyond hemodynamic effect alone, neurohormonal stimulation of the cardiovascular system in HF leads to progressive circulatory dysfunction and subsequent increased morbidity and mortality
Neurohormonal Activation in LV Systolic Dysfunction

INSULT

↓↓ ↓↓ LV Function

↑↑ ↑↑ RAS

↑↑ ↑↑ SNS

↓↓ ↓↓ CO

↑↑ ↑↑ PCWP

↑↑ ↑↑ Afterload

↓↓ ↓↓ Preload

↑ CO

↑ PCWP

↑ RAS

↑ SNS
Pathophysiology of LV Systolic Dysfunction

- Cardiac Injury
- Increased Load
- Activation of RAS and SNS
- Reduced Systemic Perfusion
  - Ischemia and Energy Depletion
  - Direct Toxicity
  - Apoptosis
  - Necrosis
  - Cell Death
  - Altered Gene Expression
  - Growth and Remodeling

Pathophysiology of LV Systolic Dysfunction

Two Main Protagonists

Renin-Angiotensin System (RAS)
Angiotensin II (A II)

Sympathetic Nervous System (SNS)
Norepinephrine (NE)

Hypertrophy, apoptosis, ischemia, arrhythmias, vasoconstriction, remodeling, fibrosis
Sympathetic nervous system and Heart Failure

Plasma levels of Norepinephrine in patients with stable CHF treated with digitalis, but no diuretics or vasodilators versus normal controls

Francis et al, Ann Intern Med; 1984;101:370
Sympathetic nervous system and Heart Failure

Plasma Norepinephrine and survival in CHF  Cohn JN et al NEJM 1984; 311:819
Basic pathway of the Renin-Angiotensin-Aldosterone (RAA) System in HF

- Angiotensinogen
- Renin
- Angiotensin I
- ACE
- Angiotensin II

Juxtaglomerular cells

- Vasoconstriction
- Increased Aldosterone Synthesis
- Increased Antidiuretic Hormone
- \( \uparrow \text{Na}^+ \text{ Retention} \)
- \( \uparrow \text{Free Water Retention} \)

\( \downarrow \text{BP} \)

Negative feedback

Worsening HF
Heart failure

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Cardiac Presentations in patients with Heart Failure

- Congestion
- Arrhythmia
- Myocardial Ischemia
- 02
- Palpitations
- Syncope
- Sudden Death
- Chest Pain

↓ End-organ Perfusion
# Clinical Criteria for Heart Failure

<table>
<thead>
<tr>
<th>Major Criteria</th>
<th>Minor Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute pulmonary edema</td>
<td>Dyspnea on exertion</td>
</tr>
<tr>
<td>PND or orthopnea</td>
<td>Night cough</td>
</tr>
<tr>
<td>JVD</td>
<td>Tachycardia (&gt;120 beats/min)</td>
</tr>
<tr>
<td>Rales</td>
<td>Pleural effusion</td>
</tr>
<tr>
<td>S3 gallop</td>
<td>Hepatomegaly</td>
</tr>
<tr>
<td>Abdominojugular reflux</td>
<td>Ankle edema</td>
</tr>
<tr>
<td>Cardiomegaly on CXR</td>
<td>Vital capacity decrease (1/3 from max)</td>
</tr>
<tr>
<td>Increased venous pressure (&gt;16cm H20)</td>
<td></td>
</tr>
</tbody>
</table>

*Weight loss >4.5 kg 5 days into treatment can be classified as a major or minor criterion*

Heart failure, in the Framingham HF study, requires 2 major or 1 major and 2 minor criteria to be present concommitently.
Predictive Value of Clinical Findings for Estimating Hemodynamics in Heart Failure

<table>
<thead>
<tr>
<th>Capillary Wedge Pressure</th>
<th>&gt;22mmHg</th>
<th>≤18mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthopnea +</td>
<td>39</td>
<td>0</td>
</tr>
<tr>
<td>Orthopnea -</td>
<td>4</td>
<td>7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>39</th>
<th>0</th>
<th>39</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4</td>
<td>7</td>
<td>11</td>
</tr>
</tbody>
</table>

|       | 43 | 7 | 50 |

**Sensitivity** = 39/43 = 91%  
**Specificity** = 7/7 = 100%  
**PPV** = 39/39 = 100%  
**NPV** = 7/11 = 64%

Stevenson LW et al. *JAMA* 1989; 261:884-888
Proportional Pulse Pressure and Heart Failure

Proportional Pulse Pressure = (BP systolic – BP diastolic)/BP systolic
Normal ratio is around 0.42.
Value less than 0.25 indicates a low cardiac index and is associated with a poor prognosis in a patient with heart failure.

<table>
<thead>
<tr>
<th>Cardiac Index (L/min/m²)</th>
<th>≤2.2</th>
<th>&gt;2.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPP ≤ 25%</td>
<td>29</td>
<td>3</td>
</tr>
<tr>
<td>PPP &gt; 25%</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>

|          | 32  | 18  | 50  |

Sensitivity = 29/32 = 91%
Specificity = 15/18 = 83%
PPV = 29/32 = 91%
NPV = 15/18 = 83%

Stevenson LW et al. JAMA 1989; 261:884-888
Use of Jugular Venous Distension (JVD) to Assess Fluid Status

<table>
<thead>
<tr>
<th>Capillary Wedge Pressure</th>
<th>&gt;18mmHg</th>
<th>≤18mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>+JVD</td>
<td>21</td>
<td>1</td>
</tr>
<tr>
<td>- JVD</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>37</td>
<td>15</td>
</tr>
</tbody>
</table>

Sensitivity = 21/37 = 57%
Specificity = 14/15 = 93%

PPV = 21/22 = 91%
NPV = 12/19 = 47%

Butman et al. JACC 1993; 22:968-974
Use of Inducible Abdominojugular Reflux to Increase Sensitivity of Physical Exam in Heart Failure

<table>
<thead>
<tr>
<th>Capillary Wedge Pressure</th>
<th>&gt;18mmHg</th>
<th>≤18mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>+JVD or +AJR</td>
<td>30</td>
<td>3</td>
</tr>
<tr>
<td>- JVD and - AJR</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>37</td>
<td>15</td>
</tr>
</tbody>
</table>

Sensitivity = 30/37 = 81%
Specificity = 12/15 = 80%
PPV = 30/33 = 91%
NPV = 12/19 = 63%

Butman et al. JACC 1993; 22:968-974
Heart failure

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Assessment of Heart Failure

Three-Step Assessment of Heart Failure

1. Does presentation fit the diagnosis of HF
2. Is systolic or diastolic LV function abnormal
3. Are there treatable causes of HF present
Step 1 Basic data

- History and Physical Exam
- Lab: Chem 20, CBC, UA
- ECG
- CXR
- Echocardiogram
Step1 Basic data

• History is the key to the diagnosis of the presence of heart failure
• Cardinal Symptoms in History for Possible HF
  – Shortness of breath
  – Fatigue
  – Edema
  – Chest pain
Physical exam
Is the patient “wet” or “dry”

“Dry”
- Fatigue
- Dizziness
- ↓Weight
- ↑BUN/Cr

“Wet”
- Shortness of Breath
- Edema
- ↑Weight
- JVD/HJR
Heart Failure “Lethal Triad”

- BP systolic < 100
- HR > 100
- [Na⁺] < 130
**Step1 Laboratory**

**Chemistry**

- **Na\(^+\):** ↓ *c/w* high ADH and angiotensin II levels
- **K\(^+\):** *Often* ↑ *with* ARF *due to inadequate renal perfusion*
- **BUN/Creat:** ↑ *c/w with inadequate renal perfusion*
- **Bilirubin, SGOT/SGPT, Alk Phosphatase:** *may indicate passive congestion*
- **Troponin:** *to r/o ischemia*
- **BNP**
Laboratory

**CBC**
- Anemia: may contribute to heart failure decompensation or high output heart failure is sustained

**Urinalysis**
- Proteinuria: may explain edema due to inadequate oncotic pressures
<table>
<thead>
<tr>
<th>Findings</th>
<th>Suspected Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute ST-T changes</td>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td>Atrial fibrillation, other</td>
<td>Thyroid disease</td>
</tr>
<tr>
<td>tachyarrhythmia</td>
<td></td>
</tr>
<tr>
<td>Q waves</td>
<td>HF due to reduced LV performance</td>
</tr>
<tr>
<td>Low voltage</td>
<td>Pericardial effusion, amyloidosis</td>
</tr>
<tr>
<td>LVH</td>
<td>Diastolic dysfunction</td>
</tr>
</tbody>
</table>
Step 2 Echocardiogram

Why Heart Failure

- Systolic Dysfunction (EF $\leq 40\%$)
- Diastolic Dysfunction (EF $> 40\%$)
- Other Dysfunction (e.g., valvular disease, pericardial disease)
Step 3 Nuclear vs DSE for Detecting Myocardial Ischemia

Brown KA et al. Am J Cardiol 1998; 81:1050-1053
Causes of Non-Ischemic Dilated Cardiomyopathy

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
<td>Familial, Peripartum</td>
</tr>
<tr>
<td>Genetic</td>
<td>Dystrophin</td>
</tr>
<tr>
<td>Infectuous</td>
<td>Echovirus, Coxsackie, Chagas, Q fever, Typhus</td>
</tr>
<tr>
<td>Granulomatosis disease</td>
<td>Sarcoidosis, Wegener’s</td>
</tr>
<tr>
<td>Metabolic/endocrine</td>
<td>Pheochromocytoma, Hypothyroidism, Hemochromatosis</td>
</tr>
<tr>
<td>Collagen vascular disease</td>
<td>LED, Scleroderma</td>
</tr>
<tr>
<td>Neuromuscular disease</td>
<td>Duchenne’s, Becker’s</td>
</tr>
<tr>
<td>Toxins</td>
<td>ETOH, Amphetamines, Cocaine, Adriamycine, Cyclophosphamide, 5-FU</td>
</tr>
</tbody>
</table>
## Precipitating Factors of Decompensation

<table>
<thead>
<tr>
<th>Lack of compliance</th>
<th>Inadequate/inappropriate therapy (meds, fluid)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Diet</td>
<td>• Infection</td>
</tr>
<tr>
<td>• Drugs</td>
<td>• Myocardial ischemia</td>
</tr>
<tr>
<td>Uncontrolled</td>
<td>• Endocrine disorders (thyrotoxicosis)</td>
</tr>
<tr>
<td>hypertension</td>
<td></td>
</tr>
<tr>
<td>Cardiac arrhythmias</td>
<td></td>
</tr>
<tr>
<td>Anemia</td>
<td></td>
</tr>
</tbody>
</table>
HF and anemia

• Anemia is common in CHF patients
• Treating anemia improves heart function and reduces hospitalizations.
• Silverberg et al reviewed the records of 142 heart failure patients and found that 56% were anemic (hemoglobin less than 12g/dL).
• The worse the CHF, the more likely patient was anemic: from 9% anemic in class I patients to 79% in class 4.
HF and anemia

- 26 were still anemic and had severe CHF even after 6 months of maximum HF treatment.
- Erythropoietin and IV iron raised hemoglobin and EF, even though their heart failure meds were not changed.
- This treatment also reduced hospitalizations by 92%, improved heart class, reduced Lasix doses, and slowed progression of kidney failure.
Drugs That Can Exacerbate Heart Failure

- Class I antiarrhythmics
  (e.g. procainamide, quinidine, disopyramide, flecainide)
- Calcium channel blocker (except Amlodipine)
- Beta-blockers
- TZD
- Non-steroidal anti-inflammatory drugs
  (e.g., indomethacin, naproxen)
- Alcohol or illicit drugs
  (e.g., cocaine, amphetamines, etc.)
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Swan-Ganz Catheter

- Clinical estimation of patient’s volume status can be difficult……
- Patients with pulmonary edema generally do not require SG catheter.
- SG, may be useful in patients not responding appropriately to therapy or if it is unclear whether pulmonary edema is due to cardiac or non-cardiac causes.
- SG catheter is useful in cardiogenic shock.
- RHC with Swan-Ganz catheter is the GOLD STANDARD METHOD to measure intracardiac pressures, cardiac output, and the hemodynamic response to therapy.
- Unfortunately, it is an invasive procedure that can be associated with significant complications…..
Possible Indications for Pulmonary Artery Balloon Catheter (Swan-Ganz)

**Suspected hypoperfusion**
- Life-threatening organ dysfunction
- Refractory hypotension
- Possible need for LVAD

**Hemodynamic Profile**
- Severe symptoms out of proportion to clinical examination
- Intolerance to diuresis
Swan-Ganz Catheter
Swan-Ganz Catheter

SVR = (mean Arterial Pressure – CVP) x 80
   cardiac output

PVR = mean PAP – Wedge
   cardiac output

Trans-pulmonary gradient = mean PAP – Wedge

If Pulmonary hypertension secondary to ↑ filling pressures (Trans-pulm Grad < 15mmHg)
## Swan-Ganz Catheter Examples

<table>
<thead>
<tr>
<th>PA</th>
<th>Mean PA</th>
<th>Wedge</th>
<th>TP Gradient</th>
<th>Consistent with</th>
</tr>
</thead>
<tbody>
<tr>
<td>50/35</td>
<td>40</td>
<td>31</td>
<td>9</td>
<td>Elevated left sided filling pressures</td>
</tr>
<tr>
<td>92/37</td>
<td>55</td>
<td>12</td>
<td>43</td>
<td>Intrinsic pulmonary process</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>PAP (mmHg)</td>
<td>Wedge (mmHg)</td>
<td>BP (mmHg)</td>
<td>Cardiac Output</td>
</tr>
<tr>
<td>------------</td>
<td>------------</td>
<td>--------------</td>
<td>-----------</td>
<td>----------------</td>
</tr>
<tr>
<td>3</td>
<td>20/10</td>
<td>9</td>
<td>120/80</td>
<td>5.3</td>
</tr>
<tr>
<td>1</td>
<td>15/3</td>
<td>4</td>
<td>90/60</td>
<td>3.5</td>
</tr>
<tr>
<td>15</td>
<td>50/35</td>
<td>32</td>
<td>78/60</td>
<td>3.2</td>
</tr>
<tr>
<td>16</td>
<td>75/32</td>
<td>12</td>
<td>80/62</td>
<td>3.1</td>
</tr>
<tr>
<td>0-1</td>
<td>25/0-2</td>
<td>6</td>
<td>85/56</td>
<td>7.8</td>
</tr>
<tr>
<td>18</td>
<td>30/18</td>
<td>18</td>
<td>88/60</td>
<td>3.1</td>
</tr>
</tbody>
</table>
## Swan-Ganz Catheter Hemodynamics Parameters

<table>
<thead>
<tr>
<th></th>
<th>CVP (mmHg)</th>
<th>PAP (mmHg)</th>
<th>Wedge (mmHg)</th>
<th>BP (mmHg)</th>
<th>Cardiac Output</th>
<th>SVR (Dynes/sec/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal</strong></td>
<td>3</td>
<td>20/10</td>
<td>9</td>
<td>120/80</td>
<td>5.3</td>
<td>1100</td>
</tr>
<tr>
<td><strong>Hypovolemia</strong></td>
<td>1</td>
<td>15/3</td>
<td>4</td>
<td>90/60</td>
<td>3.5</td>
<td>1600</td>
</tr>
<tr>
<td><strong>Cardiogenic shock</strong></td>
<td>15</td>
<td>50/35</td>
<td>32</td>
<td>78/60</td>
<td>3.2</td>
<td>1800</td>
</tr>
<tr>
<td><strong>Pulmonary Embolism</strong></td>
<td>16</td>
<td>75/32</td>
<td>12</td>
<td>80/62</td>
<td>3.1</td>
<td>1500</td>
</tr>
<tr>
<td><strong>Septic shock</strong></td>
<td>0-1</td>
<td>25/0-2</td>
<td>6</td>
<td>85/56</td>
<td>7.8</td>
<td>800</td>
</tr>
<tr>
<td><strong>Tamponade</strong></td>
<td>18</td>
<td>30/18</td>
<td>18</td>
<td>88/60</td>
<td>3.1</td>
<td>1850</td>
</tr>
</tbody>
</table>
### Swan-Ganz Catheter

#### Hemodynamics Parameters

<table>
<thead>
<tr>
<th></th>
<th>CVP (mmHg)</th>
<th>PAP (mmHg)</th>
<th>Wedge (mmHg)</th>
<th>BP (mmHg)</th>
<th>Cardiac index</th>
<th>SVR (Dynes/sec/cm(^5))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal</strong></td>
<td>0-5</td>
<td>25/6-12</td>
<td>6-12</td>
<td>120/80</td>
<td>≥2.5</td>
<td>900-1200</td>
</tr>
<tr>
<td><strong>Hypovolemia</strong></td>
<td>0-1</td>
<td>15/2-6</td>
<td>&lt;6</td>
<td>&lt;90/60</td>
<td>&lt;2</td>
<td>&gt;1500</td>
</tr>
<tr>
<td><strong>Cardiogenic shock</strong></td>
<td>&gt;8</td>
<td>50/30</td>
<td>30</td>
<td>&lt;90/60</td>
<td>&lt;2</td>
<td>&gt;1500</td>
</tr>
<tr>
<td><strong>Septic shock</strong></td>
<td>0-1</td>
<td>25/0-2</td>
<td>&lt;6</td>
<td>&lt;90/60</td>
<td>&gt;4</td>
<td>&lt;800</td>
</tr>
<tr>
<td><strong>Tamponade</strong></td>
<td>12-18</td>
<td>30/18</td>
<td>18</td>
<td>&lt;90/60</td>
<td>&lt;2</td>
<td>&gt;1500</td>
</tr>
</tbody>
</table>
Conclusion

“I can’t breathe”  

“I can walk 30 minutes without stopping”
Heart Failure Reduces Peak Oxygen Consumption with Exercise

\[
O_2 \text{ consumption} = HR \times SV \times \Delta AV02 \\
\Delta AV02 = O_2 \text{ content in the arterial system} - O_2 \text{ content remaining when blood flow converges back to the central venous circulation}
\]
Heart Failure Reduces Peak Oxygen Consumption with Exercise

\[ \text{Normal} \]

\[ \Delta \text{with exercise} \]

\[ \begin{align*}
HR & \times 3X \\
SV & \times 2X \\
\Delta \text{AVO}_2 & \times 3X
\end{align*} \]

\[ \text{Consumption} \]

\[ 18X \]
Heart Failure Reduces Peak Oxygen Consumption with Exercise