Heart Failure in the United States¹

- 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 nation an estimated $32 billion each year. This total includes the cost of health care services, medications to treat heart failure, and missed days of work.

¹CDC.gov
TYPES OF HEART FAILURE

• Systolic Heart Failure
  • Normal left ventricular ejection fraction is 50-55%
  • Systolic heart failure occurs when the LVEF is < 50%
  • Causes: Ischemic, Stress (Takutsubo), Hypertension, Viral infection, Arrythmia, Diabetes, Valvular disease

• Diastolic Heart Failure
  • Increased End Diastolic Pressure due to decreased left ventricular compliance
  • As the left heart becomes less compliant and the LV wall thickens the LV does not relax as well.
  • Causes: Ischemia, Hypertension, AS, Hypertrophic Cardiomyopathy, Pericardial Disease, Amyloidosis
PATHOPHYSIOLOGY

- Cardiac output is a function of preload and afterload.
  - Preload: Volume and pressure of blood in the heart at the end of diastole.
  - Afterload: Volume and pressure of blood in the heart at the beginning of systole.

- Starling’s Law: Under normal circumstances, the more the myocardium stretches (for instance from increasing blood volume or pressure), the harder it contracts hence maintaining cardiac output. However, there is a limit to how much the myocardium will comply with this rule i.e. overstretching the heart will cause it to lose the ability to pump harder and therefore fail.

- Once the heart fails, the body will try and increase the afterload and preload through counter-regulatory neurohormonal mechanisms (renin-angiotensin-aldosterone system mainly) which under normal circumstances will support cardiac function by maintaining cardiac output. It does this in 3 ways:
  - Retain sodium and water to increase intravascular volume and preload
  - Vasoconstriction of arteries to increase blood pressure and afterload
  - Increased sympathetic activation leading to increased cardiac contractility

- In patients with impaired ventricular function and weak cardiac muscles, increases in preload and afterload are harmful and will set off a cycle which will further deteriorate cardiac function.

Adapted from fastbleep.com
SIGNS AND SYMPTOMS

• Congestive Heart Failure
  • Resulting fluid overload state with decompensated heart failure
  • Causes: dietary indiscretion with salt and water, acute or chronic renal failure, arrhythmia, worsening heart failure, accelerated or malignant hypertension, medication non-compliance
  • Symptoms: DOE, dyspnea at rest, orthopnea, PND, lower extremity edema, increased abdominal girth, weight gain, fatigue/weakness
  • Signs: rales on lung exam, or dullness to percussion of the lower lung fields suggestive of pleural effusion. Elevated JVP, lower extremity edema, use of accessory muscles of respiration, speaking in broken sentences, tachypnea, irregular heart rhythm or rapid heart rate
NYHA CLASSIFICATION

- New York Heart Association (NYHA)
- Based on a functional classification system
  - **Class I**: No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea.
  - **Class II**: Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.
  - **Class III**: Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
  - **Class IV**: Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.
• Diagnosis is based on clinical exam, laboratory and imaging assessment
  • Clinical suspicion should be elevated in a patient with symptoms/signs of CHF
  • Labs include:
    • Elevated BNP or Pro-BNP
    • Dilute urine (specific gravity may be low)
    • Acute renal failure.
  • Imaging include:
    • Chest x-ray showing cardiomegaly, Alveolar edema (Bat wings), cephalization, pleural effusions, Kerley-B or A lines.
    • Chest CT: showing ground glass alveolar infiltrates, pleural effusions, cardiomegaly
    • Echocardiogram: reduced LVEF, increased E/A, LVH (may suggest diastolic dysfunction), valvular abnormalities, thickened septum
Stage I CHF: Views of the upper lobe vessels of a patient in good condition (left) and during a period of CHF (right).

Notice the increased width of the vascular pedicle (red arrows).

Also notice cephalization (blue arrows) which is increased pulmonary vascular congestion. Hint, the pulmonary vasculature in the upper lobes should be nearly transparent in the normal chest x-ray.

Adapted from radiologyassistant.nl
Stage II of CHF is characterized by fluid leakage into the interlobular and peribronchial interstitium as a result of the increased pressure in the capillaries.

When fluid leaks into the peripheral interlobular septa it is seen as Kerley-B or septal lines.

Kerley-B lines are seen as peripheral short 1-2 cm horizontal lines near the costophrenic angles. These lines run perpendicular to the pleura.

Adapted from radiologyassistant.nl
CHEST X-RAY

- Stage III CHF
  - Alveolar infiltrates (Bat Wing's)
  - Fluid in the alveolar space
  - If this stage progresses with fluid leaking out of the capillaries at a rate greater than lymphatic reabsorption, then pleural effusions will develop

Adapted from radiologyassistant.nl
CHEST X-RAY

• Putting it all together
  • Cardiomegaly
  • Cephalization
  • Alveolar infiltrates (Bat Wing’s)
  • Kerley-B lines
  • Pleural effusions (blunting of the costophrenic margins)

Adapted from radiologyassistant.nl
TREATMENT

• Treatment CHF is based on the type of Heart Failure, acute or chronic
  • Chronic Systolic
    • Loop diuretics: promote salt and fluid excretion
      • Non-loop K sparing diuretics: spironolactone
    • Beta blocker: decrease myocardial oxygen demand, lower BP and HR
    • Acel or ARB: decrease conversion of angiotensin I to II
    • Long acting nitrates (isosorbide)
    • Antiarrythmics
    • Dietary: 2 gram salt restriction, 2L fluid restriction
    • Manage underlying etiology: CAD, DM, HTN, etc
  • LVAD
  • Heart Transplant
  • AICD for LVEF < 35% for primary prevention of sudden cardiac death
  • Chronic Diastolic
    • Loop diuretics are the mainstay of treatment
    • Dietary: 2 gram salt restriction and 2L fluid restriction
    • Manage underlying etiology: CAD, DM, HTN, etc
TREATMENT

• Acute decompensated heart failure (CHF)
  • Supplemental oxygen or NIPPV if acute hypoxemic respiratory failure
  • Rapid reduction of BP in accelerated/malignant hypertension
  • Systolic CHF
    • Loop diuretic: bolus versus drip
    • Vasodilators: nitroglycerin drip, nitropatch
    • Beta blocker (if hemodynamically stable)
    • If hemodynamically unstable
      • Inotropes (norepinephrine, dobutamine, milrinone)
      • IABP or LVAD
      • Heart transplant

• Diastolic CHF
  • Loop Diuretics primarily
MONITORING

• Close follow up with PMD
  • Office weight, BP, HR check
  • Discussion regarding symptoms, medication compliance, exercise, barriers

• HF teaching
  • Dietary restrictions: 2 gram salt restriction, 2L fluid restriction
  • Daily weights: call PMD if weight increases by 3#/day or 5#/week
  • Medication compliance
  • Exercise
    • Cardiac Rehabilitation

• Laboratory
  • Lytes including K and Mg
  • Renal function
  • No need for routine CXR or BNP/ProBNP in the compensated patient