

# 22. Heart Failure-Physiology

**Severity and Disability**-New York Heart Association Classification (Depends on age)

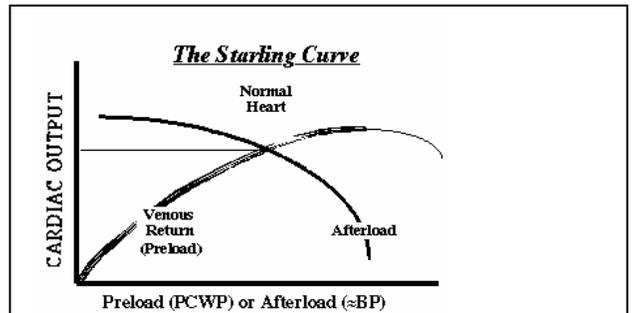
- **Class I** - No symptoms on activities of daily living (ADL) (>7 METS)
- **Class II** - Mildly limited ADL. Symptoms on normal exertion (<7 METS)
- **Class III** - Marked limitations ADL. Symptoms on minimal exertion (<4 METS)
- **Class IV** - Severely symptomatic at rest, restriction in all activities (<2 METS)

**BNP Test:** Brain natriuretic peptide >100 pg/ml = Heart or Lungs, high negative predictive value

## Etiologies:

- 1) Hypertrophic
  - a. Hypertensive
  - b. Aortic Stenosis
  - c. Hereditary
- 2) Dilative
  - a. Viral
  - b. Alcoholic
  - c. Post-MI (ie. infarction due to coronary disease)
  - d. Mitral Regurgitation > Aortic Insufficiency
  - e. Familial
  - f. Small Vessel: microvascular angina
  - g. Hemochromatosis, Cobalt toxicity
  - h. Tachycardia induced dilation - may be reversible
- 3) Restrictive
 

Pericardial, Amyloidosis, Hemochromatosis  
Scleroderma, Sarcoidosis



**Cardiogenic Shock** PCW>18 BP< 90 CI< 1.8

## Starling Curve

1. Preload (Venous Return) (PCW)
2. Afterload (Vascular Resistance) (BP)
3. Cardiac Output (Tissue Perfusion)

CO=stroke volume x heart rate  
Misleading with shunting (sepsis)

## Vascular Considerations

1. Underperfusion of vasculature leads to systemic vasoconstriction
2. Vasoconstriction exacerbates the disease because increased resistance
3. But the whole neurohormonal cascade is not dysfunctional, BNP is adaptive
4. Afterload increases lead to reduced cardiac output, and increased filling pressures
5. The venous return (preload) curve shifts up so that fluid is retained
6. Certain highly effective treatments for CHF focus on reduction in R, reshifting curves

## Neurohormonal Determinants of Vasoconstriction in CHF

1. Sympathetic nervous system activation occurs with elevated norepinephrine levels
2. Renin, Angiotensin and Aldosterone levels are highly elevated (renal sensor)
3. Reduction in baroreceptor triggering leads to increased anti-diuretic hormone (ADH)
4. ADH retains fluid (increases preload), and angiotensin II is a very potent vasoconstrictor
5. Aldosterone leads to fibrosis

## Systolic Heart Failure /Neurohormonal modification

1. Renin-angiotensin roles remodeling. Block with ACEI or ARB
2.  $\beta$ -adrenergic blockade, alpha and  $\beta$ -blockade may be more effective. Block with Carvedilol or Metoprolol-xl
3. Inhibition of aldosterone improves mortality and morbidity. Block with Spironolactone or Eplerenone
4. Diuretic = The minimum dose of Loop Diuretic
5. Chronic heart failure, Short burst of BNP with Natreacor/ Nesiritide

**Functional Classification of CHF** Clinical signs are unreliable indicators of type of CHF

1. Systolic Dysfunction
  - a. LV Ejection Fraction (EF) < 40% , Occurs in ~ 60% of CHF cases
  - b. Compensation by increased preload, tachycardia, hyperadrenergic ("cold, clammy") state
  - c. Systolic dysfunction has associated diastolic dysfunction
2. Diastolic Dysfunction
  - a. Occurs in ~30-40% of CHF cases; up to 50% of cases in elderly
  - b. Control of HTN is most important

