HEART FAILURE

Definition:

**Physiological:** Inability of the heart to pump necessary amounts of blood to meet the metabolic demands of the body, OR
to do so with increased filling pressures

**Clinical:** Syndrome presenting with symptoms and/or signs of:

1. Low cardiac output
   - Acute transient decrease causes presyncope and/or syncope
   - Subacute decrease causes shock
   - Chronic decrease is associated with fatigue

2. Pulmonary Venous Hypertension: (Dyspnea-Pulmonary edema)

   Pulmonary venous pressure is the pressure with which the left ventricle is filled by the right ventricle. If the left ventricle is stiff (diastolic dysfunction) or unable to eject normal amount of blood due to systolic dysfunction, then the right ventricle tries to fill the left ventricle with higher pulmonary venous pressure. **Pulmonary Venous Hypertension causes pulmonary congestion, which in turn causes shortness of breath of varying severity (NYHA class I to IV) including orthopnea and paroxysmal nocturnal dyspnea.** Severe elevation (particularly acute elevation) of pulmonary venous pressure (above the oncotic pressure, which is about 28 mmhg) causes Pulmonary Edema. On physical exam crepitations or rales are present. Radiologically, chest x-ray findings may reveal cephalization of blood flow, interstitial edema, perihilar edema and then alveolar edema. Whitening of lungs is noted with increasing severity of pulmonary venous hypertension.

   Mild elevation of pulmonary venous pressure at rest may not cause any symptoms unless pt exercises which further increases pulmonary venous pressure and precipitates symptoms due to pulmonary congestion (Dyspnea on exertion).

   Explanation for Cephalization: Normally vascular markings are more marked at bases due to increased flow. In pulmonary venous hypertension equal or greater flow is directed to upper zones of the lungs with equal or greater vascularity in cephalic portions of the lungs. The possible mechanism for this pattern is as follows: since pulmonary venous pressure is maximal at the bases, alveoli tend to collapse resulting in alveolar hypoxia, which causes pulmonary arteriolar constriction and redirects the blood to the cephalic portions of the lungs.

   Symptoms and signs of pulmonary venous hypertension are called Left ventricular failure or left-sided failure or left heart failure.

3. Systemic Venous Hypertension: (Elevation of Jugular venous pressure, Enlarged liver and Edema of the dependent parts of the body)

   The right ventricle fills the left ventricle with pulmonary venous pressure. The right ventricle generates pulmonary arterial pressure, which is partly expended in overcoming the resistance in the pulmonary arterioles, and the remaining is transmitted to the pulmonary capillaries and pulmonary veins to fill the left ventricle. If the right ventricle is called upon to fill the left ventricle with higher pulmonary venous pressure, it has to generate higher pulmonary arterial pressure. The signal to generate higher pulmonary arterial pressure comes from higher systemic venous pressure, the pressure with which right ventricle is filled. Initially the right ventricle is able to generate higher pulmonary arterial pressure with minimal elevation of systemic venous pressure. However, if the right ventricle is constantly required to generate high pulmonary arterial pressure to achieve an adequate pulmonary venous pressure, the right ventricle will hypertrophy and subsequently dilate resulting in a diminished ejection fraction. This leads to increasing systemic venous pressure. Unlike pulmonary venous pressure, systemic venous pressure can be easily measured in cm of water and converted to mmhg by examination of neck veins. Systemic Venous Hypertension is characterized by three cardinal signs (three E's) Elevation of Jugular Venous pressure, Enlarged Liver and Edema of the feet (dependent parts of the body).
Systemic venous hypertension may also occur without pulmonary venous hypertension (or Left heart failure) such as in primary pulmonary hypertension, pulmonary stenosis, right ventricular infarction, myocarditis and tricuspid valvular diseases.

The signs and symptoms associated with systemic venous hypertension are also referred to as right heart failure or right ventricular failure or right-sided failure.

Note: Pulmonary venous hypertension causes pulmonary congestion and pulmonary edema, whereas systemic venous hypertension causes systemic congestion (hepatic congestion and other organs) and systemic edema (edema of the dependent parts of the body). Heart failure associated with either congestion can be called congestive heart failure. What about heart failure with low cardiac output but without pulmonary or systemic venous congestion? In such cases the word “congestive” may be inappropriate and may be simply referred to as heart failure.

PATHO-PHYSIOLOGY OF HEART FAILURE
(INCREASE IN PRELOAD, AFTERLOAD AND CONTRACTILITY)

The right ventricle normally generates a pulmonary arterial pressure of 20 mmHg (maximal mean) to overcome the resistance (10 mmHg maximal) in the pulmonary circuit and to distend the left ventricle to a volume of approximately 100 ml with a pulmonary venous pressure of 10 mmHg. The left ventricle usually ejects 70cc OR 70%. Thus the ejection fraction of normal left ventricle is about 70% ±10%. The normal heart beats approximately 70 times per minute. Therefore the cardiac output is 70x 70 or 4900 cc (say 5 liters per minute). If left ventricular ejection fraction decreases due to left ventricular systolic dysfunction, the cardiac output and blood pressure (which is a product of systemic vascular resistance and cardiac output) tend to decrease leading to activation of a cascade of events (Fig 1.)

Essentially the body attempts to increase cardiac output and thereby rectify the primary defect- low cardiac output. If the cardiac output is not restored to normal, the body defends the blood pressure by increasing the systemic vascular resistance. The low blood pressure resulting from the decrease in cardiac output activates two neuro-humoral mechanisms.

1. **Sympatho-Adrenergic system** increases the contractility and ejection fraction. Adrenaline is a powerful inotropic. It acts as an internal whip, which lashes the horse (heart) with increasing intensity proportional to its sickness. It is shown that the sicker the heart the greater the levels of adrenaline leading to sooner demise of the patient. One of the modalities of treatment of heart failure is to administer inotropic agents like digitalis, as if the inotropic whip of adrenaline is not adequate. Recently, many therapists have questioned the wisdom of the body to whip a sick heart. (Whipping a sick horse may make the horse run faster but may hasten the death). An alternate strategy is also tested resting the heart by blocking the adrenalin (the internal whip) with beta-blocker drugs. The alternate strategy proved to be beneficial.

2. **Renin-Angiotensin-Aldosterone-Vasopressin System** increases sodium and water retention. Increased blood volume raises the systemic venous pressure increasing the preload of the right ventricle (initial tension in the words of Starling). This translates into higher right ventricular systolic pressure (the final tension in Starling’s language), pulmonary arterial pressure, pulmonary capillary pressure and pulmonary venous pressure culminating in greater distension of left ventricular volume. Thus the body attempts to compensate for the decreased stroke volume due to low ejection fraction by increasing end-diastolic volume of the left ventricle (theoretically, stroke volume could be restored to normal if left ventricular end diastolic volume is doubled to 200cc given an ejection fraction of 35%, which is half of the normal). Pulmonary venous hypertension generated to fill the left ventricle to a greater volume will give rise to symptoms of “left heart failure” or “left ventricular failure”.

Pulmonary arterial hypertension, required to generate pulmonary venous hypertension to fill the left ventricle to a greater volume, in itself may not cause any symptoms immediately except minimal elevation of systemic venous pressure. But over a long period it will lead to right ventricular diastolic and systolic dysfunction. This manifests as Elevation of jugular venous pressure, Enlarged liver and Edema of the feet, right heart failure or right ventricular failure or right sided failure.

It is important to remember that the elevation of venous pressures (both pulmonary and systemic) is a compensatory mechanism to maintain the stroke volume and cardiac output.

Although systemic and pulmonary venous hypertension is beneficial to restore the cardiac output, it causes significant disease to the patient and compromises his/her overall function. Therefore the treatment is directed towards the decrease of venous pressures (preload) with diuretics to relieve the symptoms and improve the exercise capacity. How much diuretic to give? The therapeutic goal is to give diuretics to improve the exercise capacity to maximal. If compensatory mechanisms fail to raise venous pressures adequately, such as in right ventricular infarction, venous pressure should be raised therapeutically by administration of fluids.

If both the mechanisms fail to restore the cardiac output, then each one of them increases the systemic vascular resistance (afterload) to maintain the blood pressure. In addition to the sympatho-adrenergic system, angiotensin II is also a powerful vasoconstrictor. However, an increase in systemic vascular resistance can be deleterious. The sick left ventricle has to eject blood into an arterial system, which offers
higher resistance. This results in decreased stroke volume and cardiac output. Therefore, systemic vascular resistance should be kept at the minimum required to achieve minimal blood pressure to maintain perfusion to vital organs. For example in an ambulatory patient head should be perfused and in a bed-ridden patient, the kidneys should be perfused (Note the minimum pressure required to perfuse the vital organs varies from patient to patient and no particular number should be used to treat all patients). Various vasodilators have been shown to effectively relieve symptoms and improve survival. Occasionally the systemic vascular resistance in the face of low cardiac output is too low to maintain the blood pressure required to perfuse vital organs. Vasoconstrictors have to be judiciously used in those cases.

Thus compensatory mechanisms include increase in preload, afterload and contractility. Management of heart failure constitutes manipulation of these three compensatory mechanisms to improve quality and quantity of life.