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HEART FAILURE

The principal functions of the heart are to receive blood from the veins, send it to the lungs for oxygenation, and pump the oxygenated blood to the body. *Heart failure* occurs when there is a substantial disruption of these functions secondary to a loss of normal contractile ability. If heart failure is associated with abnormal fluid retention, it is referred to as *congestive heart failure* (CHF). The major clinical manifestation of heart failure is shortness of breath, especially with exercise. In the acute setting, heart failure is commonly due to a loss of heart muscle because of infarction or because of a loss of nourishment due to the ischemia.

Common Causes of Heart Failure and Pulmonary Edema

I. Myocardial ischemia **NB**

- A. Acute
- B. Chronic

II. Valvular dysfunction

- A. Aortic valve disease
- B. Mitral valve disease

III. Acquired cardiomyopathy

- A. Toxic
  - 1. Alcohol
  - 2. Cocaine
- B. Metabolic
  - 1. Thyrotoxicosis
  - 2. Myxedema

IV. Systemic hypertension

V. Miscellaneous

- A. Anemia
- B. Cardiac arrhythmias (*A. Fibrillation*) **NB**

Left ventricular (LV) performance (Frank Starling) curves relate preload, measured as LV end-diastolic volume (EDV) or pressure (EDP), to cardiac performance, measured as ventricular stroke volume or cardiac output. On normal individuals, cardiac performance continuously increases as a function of the preload. Decreased LV contractility (commonly associated with heart failure) is characterized by a curve that is shifted downward (lower line). (see chart) Point *a* is an example of a normal individual at rest. Point *b* represents the same individual after developing systolic dysfunction and heart failure (e.g. after a large MI): stroke volume has fallen, and the decreased LV emptying results. Further augmentation (e.g. increased circulating volume) in the heart failure patient is represented by point *c,* which resides on the relatively flat part of the curve: stroke volume is only slightly augmented, but the markedly increased EDP results in pulmonary congestion.

Pathophysiology of Heart Failure

The three factors of contractility, preload, and afterload determine ventricular stroke volume. Coupled with heart rate, stroke volume determines cardiac output. Cardiac *contractility* is related to the amount of myocardial stretch, known as *preload*. Clinical measurements of cardiac stretch include the ventricular end-diastolic pressure and volume. *Afterload* is defined as the ventricular wall tension that develops during systole and reflects the resistance to outward blood flow. It is clinically estimated by the systolic arterial pressure. Many sorts of heart failure are associated with decreased contractility. The Frank-Starling relationships between stroke volume, preload, and contractility in both the normal and failing heart are illustrated.

Heart failure can be further classified into three categories related to physiology and functional anatomy. These categories are: high versus low cardiac output, right versus left heart failure, and systolic versus diastolic dysfunction.

Heart failure can produce either low or high cardiac output. While *low-output failure* is due to an inherent problem in myocardial contraction, *high-output failure* is due to an inability of functionally intact myocardium to keep up with excess functional demands.
[The causes of high-output failure are relatively few and include anemia, thyrotoxicosis, large arteriovenous shunts, beriberi and Paget disease of the bone].

In congestive heart failure, excess fluid accumulates behind the affected chamber of the heart. In patients with left ventricular dysfunction due to either mechanical overload or infarction, excess fluid develops in the lungs. This resulting *pulmonary edema*, or congestion, is the cardinal manifestation of *left-sided heart failure*. In patients where the right ventricle is compromised (pulmonary embolus, severe COPD, right ventricular infarction), jugular venous distension and other signs of *right-sided heart failure* occur. Long-standing heart failure, however, usually results in compromise of both ventricles.

Systolic heart failure is characterized by an impairment of myocardial contraction, and diastolic failure by an impairment in myocardial relaxation. *Systolic failure* can occur from excessive afterload (systemic hypertension) or from damaged myocytes (infarction). *Diastolic failure* can be seen in both acute and chronic heart failure.

Once heart failure has developed, several neurohormonal compensatory mechanisms occur. Alterations in adrenergic tone redistribute blood flow to the brain and myocardium, reducing blood flow tot he shin, kidneys, gastrointestinal tract, and skeletal muscle. The reduction in blood flow to the kidneys results in increased stimulation on the renin-angiotensin-aldosterone axis and secretion of antidiuretic hormone. The end result of these processes is enhanced sodium and water retention by the kidneys, which leads to fluid overload and CHF. Additionally, the increased adrenergic tone leads to arteriolar vasoconstriction, a significant raise in afterload, and, finally to increased cardiac work.

Clinical Features of Heart Failure

The clinical features of heart failure may be due to impaired perfusion or elevated venous pressures and relate to which ventricle is primarily affected. Patients may present with either the chronic progressive symptoms of heart failure or with acute pulmonary edema due to sudden left-sided decompensation.

The most common symptom of left-sided heart failure is breathlessness, or *dyspnea,* particularly with exertion. When pulmonary venous pressure reaches a critical level (20 mmHg), there is movement of fluid into the pulmonary interstitium, compressing airways and alveoli. The increased resistance to airflow intensifies the work of breathing. In addition, stimulation of the juxtacapillary receptors (J receptors) causes rapid shallow breathing. Other manifestations of

pulmonary congestion include orthopnea and paroxysmal nocturnal dyspnea (PND). Orthopnea is the sensation of breathlessness while lying flat and is relieved by sitting upright. Orthopnea results from the redistribution of intravascular blood from the gravity-dependent portions of the body to the lungs. The degree of orthopnea is usually assessed by the number of pillows the patient uses at night to avoid breathlessness. PND is severe breathlessness that awakens the patient from sleep 2 to 3 hours after lying down. PND is due tot he gradual reabsorption of interstitial lower extremity edema after lying down, with subsequent greater venous return to the heart and lungs.

Clinical manifestations of left-sided heart failure due to alterations in blood flow include fatigue, altered mental status, and reduced urine output (especially during the day). At night, urine output may increase do to increases in venous return. The increased urinary frequency at night is termed *nocturia.*

Right-sided heart failure causes increased systemic venous pressures and peripheral edema.

Physical findings of left-sided heart failure include dusky or pale skin, diaphoresis, and cool extremities due to poor perfusion and peripheral arterial vasoconstriction. Pulmonary congestion commonly results in tachypnea (respiratory rate > 24/min) and bilateral inspiratory crackles on ausculatation. Additional auscultatory findings include rhonchi and wheezing ("cardiac asthma") due to airway edema. In advanced heart failure, *Cheyne-Stokes respiration* can occur and is a respiratory pattern characterized by periods of hyperventilation separated by periods of absent breathing (apnea). Cardiac auscultation may reveal a third heart sound (S_3), and early diastolic sound resulting from abnormal filling into the dilated ventricle.

Right-sided heart failure may result in additional physical findings due to elevated systemic venous pressures. *Jugular venous distension* (JVD) is common. Engorgement of the liver results in hepatomegaly and the presence of the hepatojugular reflux (JVD with liver palpitation). Peripheral edema accumulates in the dependent areas of the body, including the ankles and legs of ambulatory patients and the presacral region of bedridden patients.

The electrocardiogram (ECG) may reveal evidence of acute myocardial infarction or ischemia as a cause of acute decompensation in CHF. Hypoxia is a frequent finding with pulmonary edema and when associated with hypercarbia and acidosis can portend impending respiratory failure.

Treatment of Chronic Congestive Heart Failure

Vasodialators help to reduce the increase in afterload that occurs with the neurohormonal compensatory response to CHF. Even a modest reduction in afterload may elevate the stroke volume of the heart and reduce ventricular end-diastolic pressures.

Common Symptoms and Physical Findings in Heart Failure

Symptoms	**Physical Findings**
LEFT *SIDED FAILURE*	
Dyspnea	Diaphoresis
Orthopnea	Tachycardia, tachypnea
Paroxysmal nocturnal dyspnea	Pulmonary rales, wheezes
Fatigue	S_3 gallop
RIGHT *SIDED FAILURE*	
Peripheral edema	Jugular venous distension
Right upper quadrant pain	Hepatomegaly
	Hepatojugular reflux
	Peripheral edema

Common Drugs Used in Acute* Heart Failure

Afterload Reducers

- eliminates ***back pressure*** against which the heart has to empty
  - this is caused by decreased peripheral vascular resistance (PVR)
    - decreased pressure against which the heart has to empty causes decreased myocardial work and O_2 demand
- vasodilators allow a "holding" of fluid in the peripheral space
  - which causes decreased venous return
    - decreased venous return causes decreased myocardial work and O_2 demand
- agents :
  - NTG
  - morphine
  - magnesium

Preload Reducers

- "holds" ***volume*** in the peripheral space – which causes decreased venous return
- decreased venous return causes decreased myocardial work and O_2 demand
  - same vasodilators as above : NTG, morphine, magnesium
- eliminates ***volume*** – which causes decreased venous return
- decreased venous return causes decreased myocardial work and O_2 demand
  - lasix
  - positioning (dangling of feet off the gurney)

* currently there are no simple, highly safe, rapidly effective, and routinely available prehospital inotropic agents. (Amrinone and Dobutamine: relatively safe and effective inotropes)
(Dig.: too slow)
(isoproterinol and aminophylline : too risky benefit/toxicity range)

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