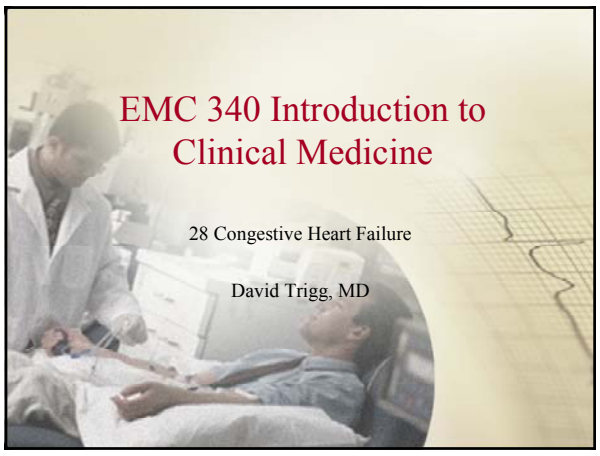
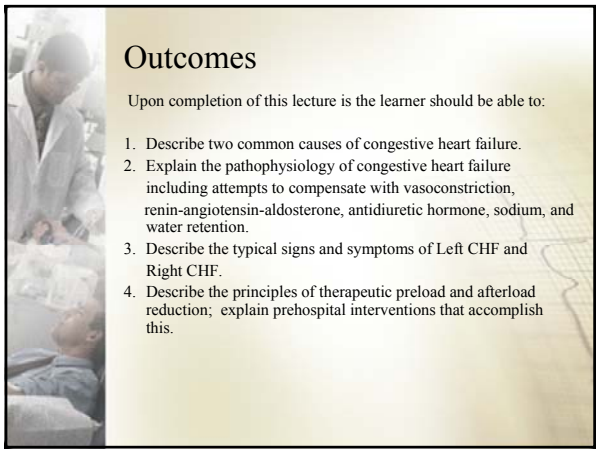


EMC 340 Introduction to Clinical Medicine

28 Congestive Heart Failure

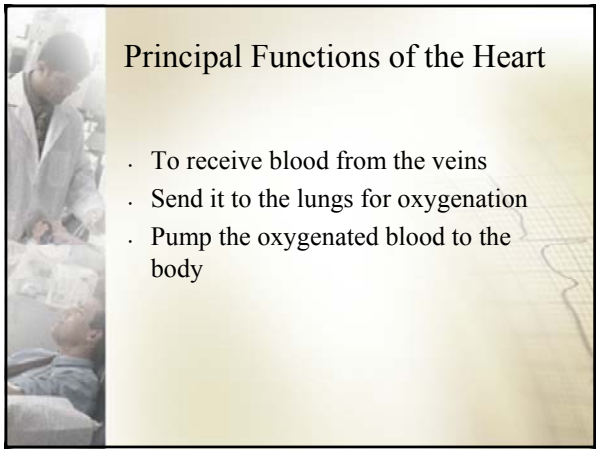
David Trigg, MD



Outcomes

Upon completion of this lecture is the learner should be able to:

1. Describe two common causes of congestive heart failure.
2. Explain the pathophysiology of congestive heart failure including attempts to compensate with vasoconstriction, renin-angiotensin-aldosterone, antidiuretic hormone, sodium, and water retention.
3. Describe the typical signs and symptoms of Left CHF and Right CHF.
4. Describe the principles of therapeutic preload and afterload reduction; explain prehospital interventions that accomplish this.



Principal Functions of the Heart

- To receive blood from the veins
- Send it to the lungs for oxygenation
- Pump the oxygenated blood to the body

Heart Failure

- Occurs when there is a disruption of the principle functions.
- Heart failure is secondary to a loss of normal contractile ability.
- When heart failure is associated with abnormal fluid retention it is referred to as *congestive heart failure* (CHF).

CHF

In the acute setting, CHF is most commonly due to:

- Loss of heart muscle because of infarction.
- Loss of nourishment due to the ischemia.
- Sudden loss of heart muscle efficiency, meaning the heart's contractile ability cannot meet the demand.

Common Causes of CHF

I. Myocardial ischemia ***NB***
   A. Acute
   B. Chronic

II. Hypertension ***NB***

III. Valvular dysfunction
   A. Aortic valve disease
   B. Mitral valve disease

IV. Acquired cardiomyopathy
   A. Toxic
      1. Alcohol
      2. Cocaine
   B. Metabolic
      1. Thyrotoxicosis
      2. Myxedema

V. Miscellaneous
   A. Anemia
   B. Cardiac arrhythmias
      1. (Atrial Fibrillation) ***NB***

Pathophysiology

- Factors determining ventricular stroke volume
  - Contractility
  - Preload
  - Afterload
- Most heart failure is associated with decreased contractility.
- Frank-Starling
  - relationship between stroke volume, preload, and contractility.
  - relationship differs in the normal and failing heart.

Heart Failure Categories

Heart failure is classified into categories related to physiology and functional anatomy.

Categories

- High output failure versus low cardiac output
- Right heart failure versus left heart failure
- Systolic heart failure versus diastolic dysfunction.

High Output Failure and Low Cardiac Output

- low-output failure is due to an inherent problem in myocardial contraction
- high-output failure is due to an inability of intact myocardium to keep up with excess functional demands
  - Anemia
  - Thyrotoxicosis
  - beriberi (vitamin B_1 deficiency)
  - Paget's disease of the bone

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Left Heart Failure

- In congestive heart failure, excess fluid accumulates behind the affected chamber.
- Left ventricular dysfunction is due to:
  - mechanical overload.
  - Infarction.
- Either results in excess fluid that develops in the lungs leading to congestion of lungs, or pulmonary edema.

Right Heart Failure

Right ventricle dysfunction is due to "blocked outflow".

- pulmonary embolus
- severe COPD
- right ventricular MI

Resulting in excess fluid on the venous side (↑CVP).

Compensatory Mechanisms

- Neurohormonal compensatory mechanisms:
  - adrenergic tone or increased tone caused by arteriolar vasoconstriction (shunting of blood flow to the brain and myocardium).
  - reducing blood flow to the skin, kidneys, gastrointestinal tract, and skeletal muscle.
- Increased adrenergic tone and arteriolar vasoconstriction result in:
  - significant increase in afterload
  - increased cardiac workload
  - decreased myocardial efficiency

Heart Failure Compensatory Mechanisms

- Reduction in blood flow to the kidneys causes shock-like compensatory mechanisms to be set in motion.
  - increased stimulation of the renin-angiotensin-aldosterone
  - secretion of antidiuretic hormone*
- The end result of these processes
  - sodium and water retention by the kidneys
  - leading to fluid overload and CHF

* Hormonal responses include increased ANP & BNP.

Clinical Features of Heart Failure

The clinical features of impaired perfusion (oxygenation).

- CNS→ confusion
- Coronary arteries → chest pain
- Lungs → shortness of breath
- Elevated venous pressures

Onset:

Either gradual, chronic progressive symptoms of heart failure, or acute pulmonary edema due to sudden left-sided decompensation.

Left-Sided Heart Failure

- Most common symptom is breathlessness (dyspnea) particularly with exertion (DOE).
- Pulmonary venous pressure
  - If greater than the critical level (20 mmHg), then the movement of fluid into the pulmonary interstitium, compresses airways and alveoli.
- Pulmonary results:
  - increased resistance to airflow, thus
  - increasing the work of breathing, causing
  - stimulation of the juxtacapillary receptors (J receptors)
  - rapid shallow breathing

Left-Sided Heart Failure

- Orthopnea (inability to breathe except in an upright position)
  - Degree of orthopnea assessed by the number of pillows (1, 2, 3…sitting upright, etc) the patient uses at night to avoid breathlessness.
- Paroxysmal nocturnal dyspnea (PND)
- Non-pulmonary symptoms due to poor blood flow
  - Fatigue
  - Altered mental status
  - Reduced urine output (during the day)
  - Nocturia

Left-Sided Heart Failure

- Skin
  - dusky or pale, diaphoresis, and cool
- Vital Signs
  - tachypnea (respiratory rate > 24/min)
  - Cheyne-Stokes
- Chest
  - bilateral inspiratory crackles
  - rhonchi and wheezing (“cardiac asthma”)
- Cardiovascular
  - S_3

Common S/S of Heart Failure

Symptoms

LEFT *SIDED FAILURE*

- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Fatigue

RIGHT *SIDED FAILURE*

- Peripheral edema
- Right upper quadrant pain

Signs

LEFT *SIDED FAILURE*

- ALOC
- BP changes
- Diaphoresis
- Tachycardia, tachypnea
- Wet rales, wheezing
- S_3 gallop

RIGHT *SIDED FAILURE*

- Jugular venous distension
- Hepatomegaly
- Hepatojugular reflux
- Peripheral edema
- Presacral region (in bedridden patients)

Treatment of Chronic CHF

Preload Reducers

- "hold" ***volume*** in the peripheral space – which causes decreased venous return
  - decreased venous return → decreased myocardial work and O_2 demand
  - process same as for the vasodilators (NTG, morphine, magnesium)
- eliminating ***volume***
  - causes decreased venous return
  - decrease in venous return ⇒ ↓ myocardial work and ↓ O_2 demand
- Pharmacological Agents
  - diuretics (lasix)
  - vasodilators (NTG, morphine, magnesium) allow a "parking" of fluid in the peripheral space, which causes decreased venous return. Also decreasing the venous return,decreases myocardial work and O_2 demand.
- Positioning (dangling feet off the gurney)

Vasodilators

- Reduce the high neurohormonal afterload, resulting from compensatory (shock-like) response to CHF.
- Cause afterload reduction (even modest), which may elevate the stroke volume
- Eliminate ***back pressure*** against which the heart has to empty by decreasing peripheral vascular resistance (PVR)
  - decreased pressure against which the heart has to empty causes decreased myocardial work and O_2 demand
- Agents
  - NTG
  - Morphine
  - magnesium

Contractility Increasers

Currently none are available that are

- simple, easy, and highly safe
- rapidly effective

Prehospital inotropic agents.

- Dobutamine: relatively safe and effective inotrope
- Digoxin: too slow
- Isoproterinol and Aminophylline: benefit/toxicity range is too risky

Summary

We have discussed:

- Common causes of congestive heart failure.
- The pathophysiology of congestive heart failure with discussion of compensation with vasoconstriction, renin-angiotensin-aldosterone, antidiuretic hormone, and sodium and water retention.
- The typical signs and symptoms of LCHF and RCHF.
- The rationale for preload and afterload therapeutic reductions.