

Pathophysiology Of Congestive Heart Failure

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Definition

HF is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.

Congestive Heart Failure describes a condition where the heart muscle is weakened and cannot pump as strongly as before.

Heart Failure

- This means less oxygen is reaching the organs and muscles which can make feel tired and short of breath.
- **CONGESTIVE HEART FAILURE** – refers to the state in which abnormal circulatory congestion exists a result of heart failure

Types of Heart Failure

- **Low-Output Heart Failure**

- Systolic Heart Failure:

- decreased cardiac output
- Decreased Left ventricular ejection fraction

- Diastolic Heart Failure:

- Elevated Left and Right ventricular end-diastolic pressures
- May have normal Left ventricular ejection fraction

- **High-Output Heart Failure**

- Seen with peripheral shunting, low-systemic vascular resistance, hyperthyroidism, beriberi, carcinoid, anemia
- Often have normal cardiac output

- **Right-Ventricular Failure**

- Seen with pulmonary hypertension.

Types of Heart Failure

- **Systolic Dysfunction**
 - Coronary Artery Disease
 - Hypertension
 - Valvular Heart Disease
- **Diastolic Dysfunction**
 - Hypertension
 - Coronary artery disease
 - Hypertrophic obstructive cardiomyopathy (HCM)
 - Restrictive cardiomyopathy

Etiology

- Heart failure is caused by systemic hypertension in 75% of cases.
- Structural heart changes, such as valvular dysfunction, cause pressure or volume overload on the heart.
- Heart is unable to pump enough blood to meet tissues O₂ requirements
 - Congenital heart defects
 - Severe lung disease
 - Diabetes
 - Severe anemia
 - Overactive thyroid gland (hyperthyroidism)
 - Abnormal heart rhythms

Etiology

- Increase in Pulmonary pressure results fluid in alveoli
(PULMONARY EDEMA)
- Increase in Systemic pressure results in fluid in tissues
(PERIPHERAL EDEMA)

Health conditions that either damage the heart or make it work too hard

Coronary artery disease

Heart attack

Heart muscle diseases (cardiomyopathy)

Heart inflammation (myocarditis)

Epidemiology

- Five millions Americans have CHF
- 550,000 New cases every year
- 800,000 Patients with CHF hospitalized every year
- 250,000 die every year
- 50% Patients die with in five years
- 150% increase in the last 20 year
- 2.6% total population has this disease
- Incidence and associated morbidity and mortality is expected to increase in future

Risk Factors

- Hypotension
- Fluid retention & worsening CHF
- Bradycardia & heart block
- Contraindication in pts with CHF exacerbation

Pathophysiology

- In order to maintain normal cardiac output, several compensatory mechanisms play a role as under:
Compensatory enlargement in the form of cardiac hypertrophy, cardiac dilatation, or both.
- Tachycardia [*i.e. increased heart rate*] due to activation of neurohumoral system e.g. release of norepinephrine and atrial natriuretic peptide, activation of renin-angiotensin aldosterone mechanism.

Pathophysiology

- **STARLING'S LAW**

Within limits, the force of ventricular contraction is a function of the end-diastolic length of the cardiac muscle, which in turn is closely related to the ventricular end-diastolic volume.

- This is achieved by increasing the length of sarcomeres in dilated heart
- Increases the myocardial contractility and thereby attempts to maintain stroke volume.

Pathophysiology

- Heart failure results in **DEPRESSION** of the ventricular function curve
- **COMPENSATION** in the form of stretching of myocardial fibers results
- Stretching leads to cardiac dilatation which occurs when the left ventricle fails to eject its normal end diastolic volume

Compensatory Mechanisms

- Sympathetic nervous system stimulation
- Renin-angiotensin system activation
- Myocardial hypertrophy
- Altered cardiac Rhythm

Pathophysiology

LEFT VENTRICULAR FAILURE

- Ischaemic heart disease
- Myocarditis
- Valvular heart disease
- Restrictive pericarditis

RIGHT VENTRICULAR FAILURE

- Cor pulmonale
- Right-sided valvular disease
- Right-sided myocardial disease
- Pulmonary hypertension

COMPENSATORY MECHANISMS

Activation of norepinephrine
atrial natriuretic peptide

Tachycardia

Further stress on myocardium

CONGESTIVE HEART FAILURE

Activation of renin-angiotensin-
aldosterone mechanism

Na⁺ and water retention

↑ Myocardial contractility

↑ Cardiac workload

Cell stretching

COMPENSATORY HYPERTROPHY
AND DILATATION

Pathophysiology

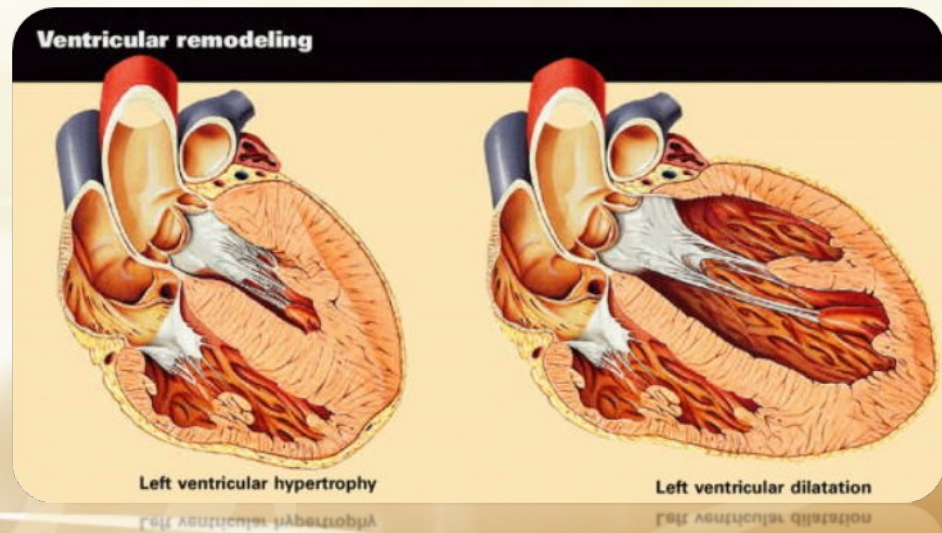
Renin-angiotensin system



Pathophysiology

Ventricular remodeling

Altered cardiac rhythm



Signs and symptoms of CHF

- Shortness of breath often with activities or while lying flat
- Weakness and fatigue
- Awakening short of breath at night
- Need for increased pillows at night – helps lungs drain of excess fluid
- Coughing or wheezing
- Swelling of feet and legs or other “dependent” areas
- Anorexia/loss of appetite
- Weight gain

Symptoms of HF

- **Fatigue**
- **Activity decrease**
- **Cough (especially supine)**
- **Edema**
- **Shortness of breath**

Complications

- Cardiac arrhythmia
- Hypotension
- N/V
- Amrinone..... Thrombocytopenia, liver enzyme
- Milirinone..... Bone marrow suppression, liver toxicity

Complications

GI

- N/V, vomiting, diarrhea, abdominal pain, constipation

Neurologic

- Headache, fatigue, insomnia, vertigo

Visual

- Color vision (green or yellow), colored halos around the subject

Miscellaneous

- Allergic, thrombocytopenia, necrosis

Complications

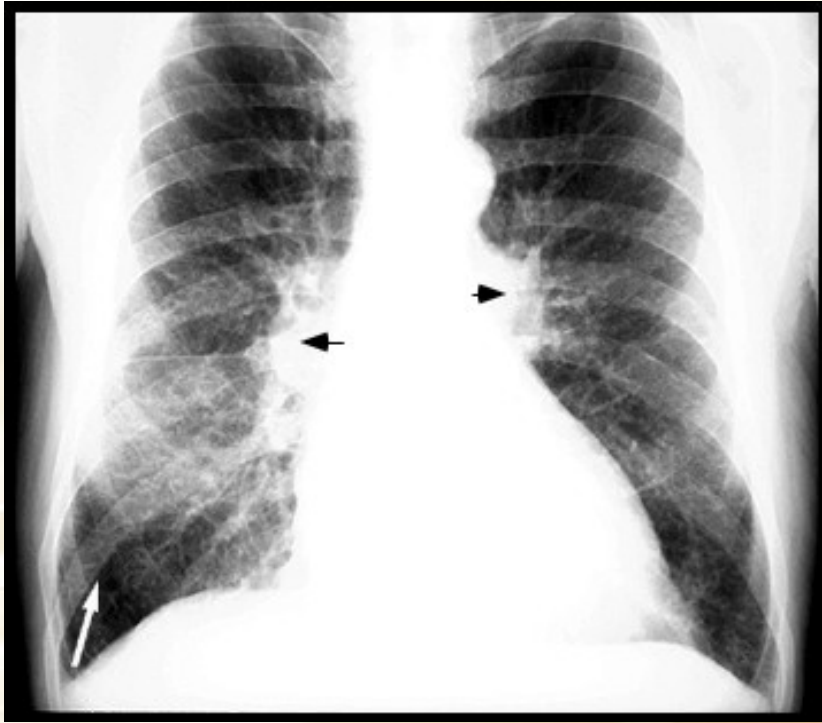
Heart

- SA and AV node suppression
- AV block
- Atrial arrhythmia
- Ventricular arrhythmia

Diagnosis

- Electrocardiogram (ECG, “EKG”)
- Chest x-ray
- Echocardiography (“Echo”)
- Heart catheterization
- Stress test
- Blood tests

Chest x-ray



DEB Approach With Heart Failure

- **Dagnose**
 - Etiology
 - Severity (LV dysfunction)
- **Initiate**
 - Diuretic/ACE inhibitor
 - β -blocker
 - Spirololactone
 - Digoxin
- **Educate**
 - Diet
 - Exercise
 - Lifestyle
 - CV Risk
- **Titrate**
 - Optimize ACE inhibitor
 - Optimize β -blocker

Treatment (Medication)

- ACE Inhibitors
- Diuretics
- Inotropic Agents
- Beta Blockers
- Calcium Channel Blockers

DRUGS USED TO TREAT CONGESTIVE HEART FAILURE

VASODILATORS

- CAPTOPRIL
- ENALAPRIL
- FOSINOPRIL
- LISINOPRIL
- QUINAPRIL
- HYDRALAZINE
- ISOSORBIDE
- MINOXIDIL
- SODIUM

DIURETICS

- BUMETANIDE
- FUROSEMIDE
- HYDROCHLOROTHIAZIDE
- METALAZONE

INOTROPIC AGENTS

- DIGOXIN
- DIGITOXIN
- DOBUTAMINE
- AMRINONE
- MILRINONE

DRUGS USED TO TREAT CONGESTIVE HEART FAILURE

Beta blocker

- Metoprolol
- Carvidilol
- Bisoprolol

Calcium channel blockers

- Nifedipine
- Diltiazem
- Verapamil
- Amlodipine
- Felodipine

BASIC PHARMACOLOGY OF DRUG USED IN

CONGESTIVE HEART FAILURE:

DIGITALIS

	<i>DIGOXIN</i>	<i>DIGITOXIN</i>
• LIPID SOLUBILITY	MEDIUM	HIGH
• ORAL AVAILABILITY	75%	>90%
• HALF-LIFE	40 HRS	168 HRS
• PLASMA PROTEIN BINDING	20-40 HRS	>90 HRS
• PERCENTAGE METABOLIZED	<20	>80
• VOLUME OF DISTRIBUTION	6.3 L/KG	0.6 L/KG

Treating Congestive Heart failure

- **U**pright position
- **N**itrates
- **L**asix
- **O**xygen
- **A**CE inhibitors
- **D**igoxin

- **F**luids(decrease)
- **A**fter load (decrease)
- **S**odium retention
- **T**est (Dig level, ABG's, Potassium level)

Patient counseling

- Lifestyle changes
- Monitoring for changes
- Medications
- Surgery

Patient counseling

Lifestyle changes

- Stop smoking
- Loose weight
- Avoid or limit alcohol
- Avoid or limit caffeine
- Eat a low-fat, low-sodium diet
- Exercise

Patient counseling

- Reduce stress
- Keep track of symptoms and weight and report any changes or concern to the doctor
- Limit fluid intake
- See the doctor more frequently

Conclusion

“PREVENTION IS BETTER THAN CURE”.

- Newer device therapies are showing promise for symptom relief and improved survival
 - Biventricular pacing.
- Transplants remain rare, but technology for mechanical assist devices continues to improve—stay tuned.

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**Thank
you.....**