

Objectives

- Review incidence and prevalence for cardiovascular disease and CHF.
- Identify pathophysiological changes due to CHF.

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Discuss objectives.



Objectives

- Discuss symptomatology of CHF and relate it back to the underlying pathophysiology.
- Review current treatment standards for patients suffering from CHF.

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Discuss objectives.



Introduction

- Cardiovascular disease results in multiple pathologies.
- Congestive heart failure (CHF) is one such diagnosis that occurs when the heart muscle begins to fail.
- Patient emergencies that may arise from this include chest pain, pulmonary edema, and systemic hypotension.

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Cardiovascular emergencies are the number one cause of death in the United States today.

The disease pathology underlying cardiovascular disease can actually cause multiple emergencies such as CHF, MI's, strokes, and hypertension to name a few.

It has been estimated that the prevalence rate for CHF is about 1 to 2 percent in the adult population. In more specific numbers, more than 400,000 patients are diagnosed yearly with CHF, and currently about 3 million Americans have this disease state. In fact, this pathology is a common reason for hospital admittance, as up to 40 percent of CHF patients are hospitalized every year. CHF has been found to be the cause of death in blacks more often than in whites (blacks are 1.5 times more likely to die from CHF) and has a higher prevalence in men than in women.

Pathophysiology

- Disease state results in dysfunction of left, right, or both ventricles
 - Changes can occur to heart rate or stroke volume ($CO = HR \times SV$)
- Blood pressure often deteriorates
 - $B/P = CO \times SVR$
- Failure of the heart muscle can result in either “backward” or “forward” failure

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Discuss that heart failure results in the reduction of cardiac output and may be caused by a decrease in stroke volume or a change in heart rate. By definition, cardiac output is the amount of blood pumped by the heart for 60 seconds.

A reduction in cardiac output leads to compensatory mechanisms that act to restore cardiac output. For instance, when a patient sustains an MI, the dead heart muscle prevents the heart from pumping normally, thus leading to decreased cardiac output. The body senses the decrease in cardiac output by way of baroreceptors in the aortic arch and carotid bodies and tries to compensate by increasing sympathetic tone.

Ultimately the heart is unable to:

- Pump blood effectively forward (frontwards failure)
- Keep up with incoming preload (backwards failure)

Pathophysiology

- Left ventricular failure
 - Forward failure results in low systolic B/P
 - Backward failure results in lung congestion

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Left ventricular failure occurs when the left ventricle is unable to pump adequately.

Dysfunction of the heart muscle itself, as is seen with MI, is one of the main causes of left ventricular pump failure.

Dysrhythmias also inhibit the heart's ability to pump normally.

With *backward failure* of the left ventricle:

- Pulmonary congestion (pulmonary edema) results
- Leading to signs and symptoms that are primarily respiratory in nature

With *forward failure* of the left ventricle:

- Diminished peripheral perfusion
- Systemic circulation result

Pathophysiology

- Right ventricular failure
 - Forward failure results in poor pulmonary perfusion
 - Backward failure results in venous congestion

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With right heart failure, the right side of the heart fails to function as an adequate pump to the lungs, which commonly leads to back pressure of blood into the venous and systemic circulation with *backward failure* of the right ventricle.

Backward failure of the right ventricle results in excess fluid that accumulates in the body, often in dependent extremities and may cause:

- Jugular venous distention
- Enlargement of the liver
- Possible abdominal distention in severe cases

Figure 25-1 Edema to the lower extremities is a classic sign of congestive heart failure.



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Figure 25-2 Jugular vein distention is a late sign of congestive heart failure. (© David Effron, M.D.)



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Pathophysiology

- Biventricular failure
 - Often left backward failure overlaps with right forward failure
 - The most common cause of right ventricular failure is left ventricular failure

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In severe or prolonged disease states, usually failure of one ventricle will eventually cause the failure of the other ventricle.

This creates a mixing of signs and symptoms of both syndromes.

The important point for the EMT is to:

- Recognize the clinical emergency
- Provide symptom specific management

TABLE 25-1 Pathophysiologic Changes in Right and Left Heart Failure

	Pathophysiologic Findings
Right heart failure	Right heart fails because of infarction, increased workload, valvular dysfunction, or a combination of these. It results in the congestion of blood in the vena cava, resulting in jugular venous distention, peripheral edema, enlarged liver, clear breath sounds, and probably hypotension.
Left heart failure	Left heart fails also because of infarction, increased workload (systemic hypertension), valvular dysfunction, or a combination of these. It results in the congestion of blood in the lungs, which increases pressure to a point at which fluid escapes into the alveoli, causing respiratory distress and pulmonary edema. Lung sounds often reveal crackles or "cardiac asthma," blood pressure is commonly normal to high, and peripheral congestion is absent.

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Clinical Findings

- Rapid breathing (tachypnea)
 - Hypoxia, CO₂ retention, sympathetic discharge
- Dyspnea
 - Changes in O₂ and CO₂ diffusion
- Orthopnea
 - Excessive fluid accumulation in lungs while lying
 - PND

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Changes in the respiratory pattern can be from:

- Hypoxia
- Acidosis
- Poor perfusion through the lungs (right heart failure)
- Fluid accumulation in the lungs (left heart failure)

Often, while lying flat, the fluid accumulation seen in the lungs with left heart failure is exacerbated and the patient becomes dyspneic while lying down (orthopnea).



Clinical Findings

- Anxiety, tremors, nausea/vomiting
 - Sympathetic discharge
- Low pulse oximeter readings
 - Diminished lung perfusion
 - Fluid accumulation in lungs
- Inspiratory crackles
 - Left ventricular backward failure

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With sympathetic discharge, there is an increase in nausea and with some, vomiting may occur. N/V are also common findings when a patient's blood pressure dips dangerously low.

As blood oxygenation through the lungs fail, so will the pulse oximeter reading. This may be due to:

- Poor perfusion through the lungs
- Fluid accumulation in the lungs



Clinical Findings

- Tripod positioning
 - Eases breathing due to improved diaphragm excursion
- Cool, pale, clammy skin
 - Sympathetic discharge
- Chest discomfort/pain
 - Possible angina or infarction

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Keeping the abdominal contents in a dependent position makes it easier for diaphragmatic excursion.

Again, with sympathetic discharge, peripheral vasoconstriction will make the skin become cool and sweaty.

Clinical Findings

- Wheezing (cardiac asthma)
 - Fluid accumulation in lungs stimulating “irritant” receptors
- Distended neck veins (JVD)
 - Right ventricular failure
- Failing systolic blood pressure
 - Left ventricular forward failure
 - Heightened SVR from sympathetic discharge

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Cardiac asthma occurs when fluid accumulation in the lungs from left sided failure starts to migrate up the bronchioles (due to breathing), and stimulates the irritant receptors.

This causes bronchiole constriction as the body attempts to stop the migration of the fluid.

The problem is that excessive bronchoconstriction inhibits good alveolar ventilation which in turn diminishes oxygenation and promotes dyspnea.



Clinical Findings

- Objective respiratory distress
 - Nasal flaring, retractions, tachypnea, mouth breathing, tripod position, etc.
 - Evidence of the compensatory mechanisms of the respiratory system trying to overcome insult

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These findings are consistent with general respiratory distress and are indicative of the body laboring harder to maintain normal oxygenation.



Emergency Medical Care

- Patient positioning
- Ensure airway adequacy
- Provide oxygen per protocol
- Utilize CPAP if allowed
- Nitroglycerin administration
- Arrange for ALS backup or intercept
- Ensure rapid transport to emergency department

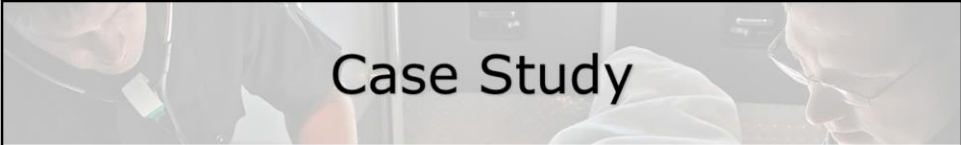
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The goal of management is to:

- Improve oxygenation
- Improve cardiac output
- Limit ischemia or infarction
- Deliver the patient to the ED rapidly with ALS intercept if possible



Case Study

You are called one night for a male patient with respiratory distress. You arrive on scene and find the patient sitting up on the edge of the bed. Patient is conscious and looks scared.

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Present case study.



Case Study

- Scene Size-Up
 - Elderly male, 290 pounds, appears to be in distress
 - No sign of struggle or trauma
 - Patient located on 2nd floor of home

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Present case study.



Case Study

- Primary Assessment Findings
 - Patient alert, responds appropriately
 - Complains of chest pain and trouble breathing
 - Airway patent with clear speech pattern
 - Breathing labored, nasal flaring and tripod positioning noted
 - Peripheral perfusion intact, radial pulse tachycardic

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Present case study.



Case Study

- Is this patient a high or low priority? Why?
- What benefit does the sitting upright position offer?
- Why is the pulse tachycardic?
- What is causing the nasal flaring and retractions?

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The patient could probably be considered a “stable but potentially unstable” patient at this time since there are no gross disturbances to vital functioning (airway, breathing, circulation). A patient with cardiovascular chest pain could rapidly deteriorate into cardiac arrest.

Sitting in an upright position helps to improve diaphragmatic motion as well as displace any fluid accumulation in the lungs to a more dependent position.

Tachycardia is secondary to sympathetic discharge, which is being stimulated due to probable baroreceptor and chemoreceptor influence on the brainstem.

Nasal flaring and retractions indicate more vigorous inspiratory muscle use to overcome resistance of airflow into the lungs. The negative intrathoracic pressure generated starts to draw in soft-tissues that overlay the bony thoracic structures – causing retractions.



Case Study

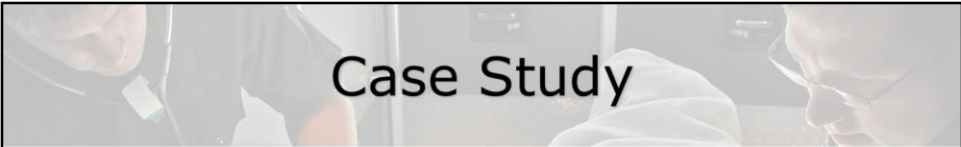
- Medical History
 - 2 previous MIs with stent placements
 - Hypertension and hypercholesteremia
- Medications
 - Nitroglycerin PRN
 - Hydrochlorothiazide
 - Prevacid
 - Lipitor

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Discuss case study.



Case Study

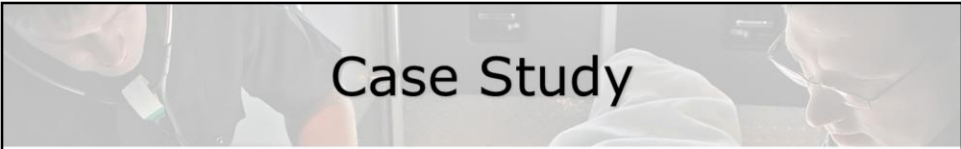
- Allergies
 - None known

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Discuss case study.



Case Study

- Pertinent Secondary Assessment Findings
 - Objective respiratory distress noted
 - Inspiratory crackles with expiratory wheezing
 - Pulse oximeter reads 91% on room air
 - JVD and peripheral edema noted
 - Dull chest pain, similar to previous MI but not as intense
 - Skin cool and clammy
 - B/P 180/104, Pulse 122, Respirations 24

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Discuss case study.



Case Study

- What pathologic change is causing the abnormal breath sounds?
- Explain why there is JVD and peripheral edema.
- Why might this patient also start to complain of nausea and/or vomiting?

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The inspiratory crackles occurs when the left ventricle cannot pump as much blood out into the aorta as it is receiving from the lungs. As such, there is a “backup” of fluid in the pulmonary vasculature that starts to leak out of the capillary beds and into the alveoli where it promotes alveolar collapse during exhalation. When the patient does breathe in, the alveoli try to reopen and it creates the characteristic “crackling” identified with crackles.

JVD and peripheral edema is likely from failure of the right ventricle to pump as much blood towards the lungs as it's receiving from the body. When the blood backs up behind the right side of the heart, it causes peripheral edema.

Nausea and vomiting occur secondary to low blood pressure and sympathetic discharge.



Case Study

- Explain the reason for the tachycardia and tachypnea.
- Why would this patient be prescribed these medications by his physician?
- If left untreated, or improperly treated, what would be the likely clinical outcome?

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Tachycardia and tachypnea are mediated by the sympathetic nervous system in response to influence from the chemoreceptors and baroreceptors monitoring blood gases and blood pressure.

These medications are to treat:

- The occasional angina (nitro)
- The excessive fluid buildup from CHF (hydrochlorothiazide)
- The high cholesterol (lipator)

The prevacid is for stomach ulcers.

If left untreated, the fluid buildup in the lungs would continue to worsen to the point that the patient will no longer oxygenate properly.

Along with the untreated angina, the patient would likely experience a severe MI and ultimately die.



Case Study

- Care provided:
 - Positioning maintained
 - High-flow oxygen administered by nonrebreather mask
 - CPAP initiated per protocol
 - EMS assists with nitro administration
 - ALS intercept initiated
 - Patient packaged and transported in ambulance

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Relate care to how it attempts to restore physiologic normalcy.

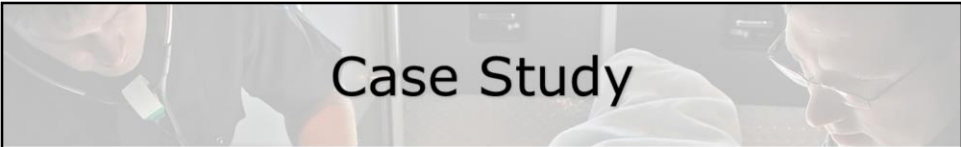
Figure 25-4 CPAP is a form of noninvasive positive pressure ventilation used in the awake and spontaneously breathing patient who needs ventilatory support. (© Ken Kerr)



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Case Study

- Explain how the following interventions may help improve the patient's condition:
 - Oxygen administration
 - CPAP
 - Nitroglycerin administration

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O₂ administration enhances diffusion of gases into the blood stream.

The use of CPAP provides a “back pressure” to help shift fluid out of the alveoli and back into the vascular system—also resulting in improved oxygenation.

Nitro will improve coronary blood flow which hopefully prevents a worsening MI.



Case Study

- If the patient improves, what would be the expected findings with:
 - Vital signs
 - Pulse oximeter
 - Breath sounds
 - Chest discomfort
 - Degree of respiratory distress

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- Vital signs will normalize.
- Pulse oximeter will improve.
- Breath sounds will become more clear.
- Chest discomfort should ease.
- Respiratory distress should start to relinquish.



Case Study

- What would be the likely assessment findings should the patient continue to deteriorate despite treatment?

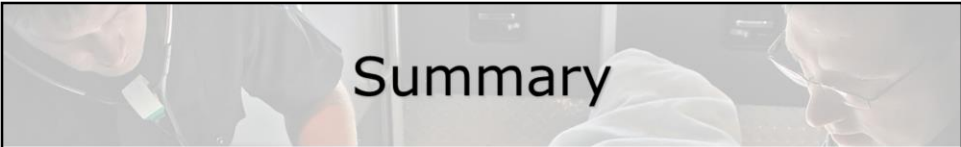
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Worsening of the following:

- Mental status
- Blood pressure
- Pulse oximeter
- Breath sounds
- Chest pain
- Skin characteristics



Summary

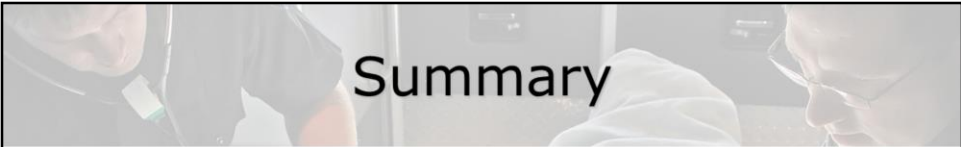
- CHF may present mildly with fatigue, or severely with hypotension with chest pain and pulmonary edema.
- Acute CHF patients can be extremely difficult to manage due to their instability.

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Review as appropriate.



Summary

- Management is geared toward improving oxygenation, alleviating dyspnea, eliminating chest pain, and maintaining normotension.

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Review as appropriate.