Northwest Community EMS System

Heart failure/Pulmonary Edema and CV Conditions

Connie J. Mattera, M.S., R.N., EMT-P

KNOWLEDGE OBJECTIVES:

Upon completion of the class, each participant will independently do the following with at least an 80% degree of accuracy and no critical errors:

1. Describe the evolution of HF and explain the various classification of HF.
2. Compare & contrast the most common causes of HF.
3. Differentiate by class and general action the most common prescription drugs used to treat HF.
4. State the components of history to obtain and the physical exam to be performed on a pt with HF.
5. Interpret presenting S&S to distinguish HF from other cardiac or respiratory pathology.
6. Sequence the appropriate treatment for a patient with HF and cardiogenic shock.
7. Explain the physiologic benefits and value the importance of using CPAP for pts with HF.
8. State the drug profile for NTG and defend appropriate dosing in HF.
9. Identify the patient with HF that may benefit from the use of midazolam for anxiety.
10. Describe the purpose and function of alternate interventions for HF such as left ventricular assist devices (LVAD).

Abbreviations used in this presentation	
ADHF: Acute decompensated heart failure	ETOH: Alcohol
ACEI: Angiotensin-converting enzyme inhibitors	HF: Heart failure
ACS: Acute coronary syndromes	HTN: Hypertension
AF: Atrial fibrillation	Hx: History
AMS: Altered mental status	LA: Left atrium
ARB: Angiotensin receptor blocker	LV: Left ventricle
BS: Breath sounds	MI: Myocardial infarction
CAD: Coronary artery disease	NTG: Nitroglycerin
c/o: Complaining of	Pts: Patients
CO: Cardiac output	RAAS: Renin angiotensin aldosterone system
COPD: Chronic obstructive pulmonary disease	SOB: Shortness of breath
CPAP: Continuous positive airway pressure	S&S: Signs & symptoms
CVD: Cardiovascular disease	SNS: Sympathetic nervous system
DM: Diabetes Mellitus	SV: Stroke volume
DOE: Dyspnea on exertion	WOB: Work of breathing
EF: Ejection fraction	Y/O: Years old

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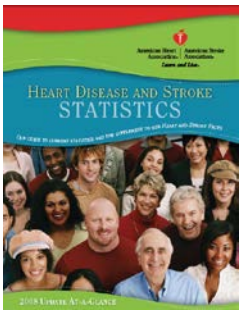
I. **Introduction; epidemiology**



A. Heart failure affects over 5.7 million people in the U.S. and represents the only cardiovascular disease with an increasing prevalence. This may be explained by improved survival rates in those sustaining an acute MI and by the demographic shift to an aging society. Over 1 million are hospitalized/yr

B. Mortality rates within one year of diagnosis are 20% and five year mortality rates are 50% for all New York Heart Association classes combined.

C. Cost: $37.2 billion (2009)

D. Data from the Framingham Heart Study *(Circulation. 2002; 106:3068–3072)*

1. Heart failure (HF) incidence approaches 10 per 1,000 population after age 65.
2. Seventy-five percent of HF cases have antecedent hypertension. About 22% of males and 46% of females
3. Heart attack (MI) victims will be disabled with HF within six years.
4. At age 40, the lifetime risk of developing HF for both men and women is one in five.
5. At age 40, the lifetime risk of HF occurring without antecedent MI is one in nine for men and one in six for women.
6. The lifetime risk doubles for people with BP greater than 160/90 mmHg compared to those with BP less than 140/90 mm Hg.

E. A study conducted in Olmsted County, Minnesota, showed that the incidence of HF (ICD9/428) has not declined during two decades, but survival after onset has increased overall, with less improvement among women and elderly persons. *(JAMA. 2004; 292:344–350.)*

II. **Heart failure** is a state of impaired cardiac function in which the heart is unable to pump sufficient blood to meet the body's metabolic demands.

A. It occurs when the workload demand exceeds the pump's ability to supply blood; when the heart's ability to pump effectively decreases; or when blood flow through the heart is impeded (sclerosed heart valves) and compensatory mechanisms are activated.

B. Several factors may predispose a patient to heart failure. See table 1.

Clinical hemodynamic subsets of patient with AMI

Subset I:	Patients who are normotensive and have adequate peripheral perfusion
Subset II:	Patients with pulmonary congestion
Subset III:	Patients with systolic hypertension
Subset IV:	Patients with peripheral hypoperfusion but no pulmonary congestion
Subset V:	Patients with *both* pulmonary congestion and peripheral hypoperfusion (pump failure)

Classifications of heart failure: Heart failure can be clinically classified in a variety of overlapping ways depending on the severity of symptoms or the predominant underlying pathology. It may be categorized by the scale first introduced by the New York Heart Association, as systolic or diastolic, high-output or low-output, right-sided or left-sided, backward or forward failure, and acute or chronic.

Table 1 - Clinical classifications of heart failure

New York Heart Association Functional classification Scale	Based on the degree of effort required to cause symptoms. Class I: No limitation of physical activity. Asymptomatic during ordinary physical activity. Class II: Slight limitation of physical activity; comfortable at rest; mild symptoms (dyspnea, fatigue or chest pain) during ordinary activity Class III: Moderate limitation; comfortable at rest, but minimal activity causes symptoms Class IV: Severe limitation; Symptoms at rest; 50% 1 year mortality rate.
Am College of Cardiology Scale	A. High risk for HF but no structural heart disease & no S&S B. Have developed structural heart disease but no S&S (yet) C. Past or current HF symptoms with structural heart damage. D. End stage or terminal HF requiring specialized treatment strategies or hospice care
Systolic vs. diastolic	Systolic failure is caused by an impaired ability of the heart to contract. The weakened heart is not able to pump effectively, resulting in an ever increasing systolic remainder of blood which ultimately increases the heart's size. The degree of failure may be quantified by measuring left ventricular ejection fraction. Ejection fractions < 40% signify systolic failure. Diastolic failure is caused by an impaired ability of the heart to relax or fill as the myocardial wall becomes stiff or thickened.
Low output vs. high output	Low-output failure is most common and is associated with a decreased cardiac output (< 4 L/min). The heart may function adequately at rest and only fail on exertion. This type is common with ACS, hypertension, and valvular disease. High-output failure is rare, where CO may be normal or even elevated, but cannot meet increased metabolic needs such as with anemia, sepsis and hyperthyroidism, beri-beri.
Left-sided versus right-sided	The left and right sides may fail independently resulting in different S&S. However, most patients have some degree of failure in both ventricles owing to excessive stress on an impaired heart. In left HF, pulmonary congestion induces symptoms such as dyspnea and orthopnea. Failure of the RV as an effective forward pump to the lungs results in ↑ pressure and congestion in the systemic venous circulation due to lack of forward blood flow into the Rt heart. Evidenced clinically as peripheral edema, hepatomegaly, and systemic venous distention. Left sided failure causes pulmonary HTN that may secondarily precipitate Rt sided failure. Other causes: Chronic hypertension, obstructive pulmonary diseases (emphysema, chronic bronchitis, asthma), pulmonary embolus (acute onset), Infarction of R atria or ventricle. Primary right sided failure secondary to lung disease is called cor pulmonale.
Backwards vs. forwards failure	In backwards failure, the ventricle cannot empty adequately, leading to elevated filling pressures and venous congestion in the beds which empty into that side of the heart. Forward failure is manifested by decreased cardiac output and perfusion deficits in the tissues that receive blood from that side of the heart. Extreme example is cardiogenic shock.
Acute vs. chronic	Failure may present as an acute complication of an AMI/pulmonary embolus or as a chronic and progressive condition secondary to slow structural changes of the heart. Over 5 million Americans suffer from chronic heart failure of which about 50% die within five years. Those who develop chronic failure will experience the same compensatory mechanisms as those with acute failure which help the patient to complete activities of daily living without continuous respiratory distress. They are usually on a host of medications to help keep them asymptomatic. Patient's previously diagnosed with chronic HF may decompensate due to increased exertion, too much salt intake, fluid imbalances, noncompliance with medication orders, sudden increases in metabolic demand related to fever or anemia, or an acute MI.

S&S Chronic HF (Many are S&S of right sided HF (RHF))	**S&S Acute HF**
▪ Fatigue & weakness ▪ Rapid or irregular heartbeat ▪ Dyspnea on exertion; orthopnea ▪ Exercise intolerance ▪ Cough or wheezing w/ white or pink-tinged sputum ▪ + JVD, dependent (pitting) edema (RHF) ▪ S3 gallop ▪ Ascites, hepatomegaly (RHF) ▪ Sudden weight gain from fluid retention ▪ Anorexia or nausea ▪ Difficulty concentrating or deceased alertness	▪ Sudden fluid accumulation in lungs; bilateral crackles or wheezes ▪ Rapid or irregular heartbeat with palpitations or dysrhythmias, weak pulse, HTN or hypotension ▪ Sudden severe dyspnea ▪ Orthopnea, PND ▪ Falling SpO_2 ▪ Ventilatory failure possible (capnography) ▪ Pink, foamy sputum possible ▪ Signs of ischemia if accompanied by AMI ▪ Skin: pale, gray, or cyanotic

III. **Compensatory mechanisms**

A. Regardless of etiology, inadequate cardiac function triggers a common set of compensatory mechanisms

B. When patient suffers an MI, has uncontrolled HTN, valve disease, or dysrhythmia, the LV becomes damaged and does not pump effectively

C. A reduction in LV stroke volume results in decreased perfusion to target organs that is perceived by baroreceptors (pressure) and stretch receptors in the aortic arch, carotid sinus, and kidneys and osmoreceptors in the brain as a reduction in circulating volume and pressure. This triggers a series of compensatory mechanisms. Neurohormonal activation has only recently been recognized as an important contributor to clinical symptoms and progression of heart failure.

D. **Primary neurohormonal compensatory mechanisms**

1. **Activation of the sympathetic nervous system**

a. With the onset of LV dysfunction, the normal autonomic NS balance is disrupted resulting in greater input from the sympathetic side. Norepinephrine is released from nerve endings and epinephrine is released from the adrenal gland to improve heart performance and to maintain mean arterial pressures. Epinephrine speeds HR and increases stroke volume by interacting with ß-1 receptors in the SA node and the myocardial cell membranes.

b. Norepinephrine stimulates alpha-1 receptors on smooth muscles in the arterioles resulting in vasoconstriction and increased afterload. The pump must now work harder to overcome resistance to emptying the LV. The prognosis in HF varies inversely with the concentration of norepinephrine in the blood. ↑ workload = ↑ O_2 demand

2. As pressure overload continues, the LV ventricle becomes thicker (hypertrophic) and simultaneously becomes stiffer & more rigid, decreasing in elasticity.

3. **Activation of the renin-angiotensin-aldosterone system** (RAA)

a. Juxtaglomerular cells (baroreceptors) around the renal nephrons sense reduced perfusion. They release **renin** which converts **to angiotensin I** in the liver. **Angiotensin I** is converted to **Angiotensin II** via angiotensin converting enzyme **(ACE),** found on the interior lining of blood vessels.

b. **Angiotensin II** "tenses the angios", i.e., it constricts arterioles and increases the BP. Angiotensin II also stimulates release of **aldosterone** from the adrenal cortex causing the kidneys to retain sodium and water, thus expanding vascular volume, ↑ preload, and decreasing urinary output.

c. Under normal circumstances, aldosterone is detoxified by a healthy liver. In HF, liver function is compromised due to fluid congestion and inadequate circulation. Therefore, aldosterone continues to generate decreased urinary output and fluid retention, augmenting the development of edema.

d. The RIGHT heart continues to receive blood from the body & pumps it into lungs. Increased preload activates Starling's law (↑ stretch = ↑ contractility) to create greater contraction.

E. **Secondary compensatory mechanisms**

1. Release of endothelin from vascular linings results in vasoconstriction of most vascular beds. It also causes + inotropic and chronotropic effects on heart muscle. It is now thought to play a major role in the perpetuation of heart failure.

2. Antidiuretic hormone (ADH) acts on renal collecting ducts to make them more permeable to water, thus decreasing urinary output. Also a potent vasoconstrictor.

3. Brain natriuretic hormone (BNP) is released from the atria and ventricles in response to fluid volume overload. These levels can help differentiate HF from medical conditions in symptomatic patients (Frazier & Arthur, 2009). These peptides inhibit renal Na reabsorption, augment Na and water excretion, suppress the rennin, angiotensin, aldosterone system and sympathetic NS, and produce arterial and venous dilation. The FDA approved a rapid point-of-care assay in 2000.

F. Over time, these compensatory mechanisms are detrimental and add to the burden on the failing heart, shortening survival times. Excessive myocardial dilation **overstretches the muscle fibers** resulting in diminished contractility and stroke volume. Increased chamber hydrostatic (fluid) pressures cause **congestion in the venous beds** leading to the affected ventricle(s). Tachycardia decreases coronary artery filling as these vessels are perfused during ventricular diastole. **Hypertrophy** increases oxygen demand at a time when coronary blood supply is already limited.

G. Ventricular cells begin to change their structure, function, and gene expression (**remodeling**) resulting in myocytes (heart cells) that are large, genetically abnormal, and cannot contract as efficiently as normal cells.

H. When the heart is damaged by infarction, the necrotic area expands while surrounding tissues dilate (compensatory mechanism to ↑ radial pressure from Starling's mechanism and ↑ contractility). The heart becomes **stiff** with less compliant muscle fibers and may transition to an **enlarged spherical shape** that cannot pump as effectively.

I. Remodeling causes death of cardiac cells at an accelerated rate. Given that cardiac cells do not divide and reproduce, those that die are not replaced. The time course for remodeling after AMI or in the presence of heart failure is not completely known, but early intervention in the cycle of increased wall tension and increased cavity size is key to preventing LV dysfunction.

J. Neurohormonal stimulation has a direct effect in stimulating remodeling and cell death. Control of this activity has become a management objective in treating HF with ACEI.

IV. Fluid dynamics in the lungs

A. When ventricular filling &/or emptying is impaired (muscle damaged and stiff), additional blood remains in the LV after systole. The LV & LA overfill until they cannot hold more.

B. Blood cannot move forward from lungs to the left heart so pulmonary vessels engorge with blood. Increased hydrostatic pressure leads to venous bed congestion in the lungs. **Blood does NOT flow backwards into the lungs from the left side of the heart**.

C. The lungs are then perfused in excess of ventilation causing a ventilation/perfusion mismatch. When pressure in pulmonary vessels exceeds pressure in the tissues → fluid leaks into interstitial spaces. Bronchovascular cuffs of the interstitial spaces can hold about 500 mL of edema. The lymph system can remove 10-20 mL of fluid/hr in healthy lung. Under stress, it can remove more through ↑ flow.

D. In HF, fluid accumulates faster than it can be removed = interstitial pulmonary edema (pt experiences dyspnea and wheezes may develop)

E. In normal lungs, the pressure of the blood (hydrostatic pressure) generated by the RV is balanced by the air pressure in the alveoli (among other things). The presence of surfactant in the alveoli keep O_2 and CO_2 exchanging rapidly. Surfactant also keeps the alveoli open and contributes to keeping the alveoli dry.

F. When hydrostatic pressures in the interstitium increase above alveolar pressures, tight junctions of alveolar walls are damaged and fluid leaks into alveolar sacs resulting in a decreased functional reserve capacity (FRC), surfactant washout, microatelectasis & **alveolar pulmonary edema** with an increase in the work of breathing (WOB), and decreased oxygen diffusion. Gas exchange is impaired, crackles & hypoxia may develop.

G. **Basic problems**

1. Too much fluid (hydrostatic) pressure in pulmonary vessels and tissues
2. Loss of surfactant, atelectasis & alveolar flooding
3. Impaired ventilations & gas exchange
4. ↑ myocardial workload causing ↑O_2 demand

V. **Assessment**

A. Assess for hypoperfusion & cardiorespiratory compromise. Differentiate HF from other life threatening conditions by: history, meds, S&S (exam).

B. **Consider cause: rate, rhythm, volume, or pump problem; treat per appropriate SOP based on etiology.**

1. It is often impossible for EMS personnel to determine the exact etiology of pump failure. Attempt to determine the cause based on PMH, history of chief complaint, and physical exam. Even in the best case scenario, EMS personnel must often treat presumptively based on clinical presentations that suggest a pump origin.

2. **Not all pulmonary edema has a cardiac etiology**. The **non-cardiac** (low pulmonary pressure**) form** can be caused by an altered alveolar-capillary membrane permeability which allows rapid fluid extravasation such as with pneumonia, head trauma, bacterial sepsis, inhaled toxins, drug overdose (narcotics), lymphatic insufficiency or obstruction, high altitudes, pulmonary emboli, eclampsia, recovery from anesthesia and idiosyncratic reactions. They are not treated the same way.

3. It may be difficult to differentiate asthma or bronchitis from acute pulmonary edema, especially in the elderly who may have multiple diseases and/or a complicated PHM.

C. **Differential of acutely decompensated heart failure to consider**

1. **Cardiovascular**

a. Heart failure
b. AMI; unstable angina
c. Acute valvular/septal rupture: Rapid transport and surgery can reverse shock due to rupture of chordae tendinae, rupture of the interventricular septum or acute valvular dysfunction.
d. Aortic dissection, arrhythmia
e. Endocarditis, myocarditis
f. Hypertensive crisis

2. **Pulmonary**

a. COPD
b. Pneumonia (multilobar); pulmonary emboli
c. Pneumothorax; anaphylaxis; aspiration; ARDS

3. Other

a. Pure volume overload: Renal failure; post-transfusion
b. Sepsis

D. **Past medical history to obtain if time allows**

1. SOB, cough, dyspnea? Asthma/COPD?
2. Other pulmonary conditions: pneumonia, pulmonary embolus, pneumothorax, pulmonary effusion, etc.
3. CVD: History of peripheral arterial disease; uncontrolled HTN (> 140/90)
4. Valve disease
5. Diabetes; drug abuse
6. OPQRST of S&S

E. **Most common causes of HF**

1. LV systolic dysfunction (60% of pts)
2. LV diastolic dysfunction: Decreased compliance: The chambers can't fill with enough blood if the heart muscle is stiff.

Table 2	
Risk factors/causes of HF	**Description**
Cardiovascular disease; Coronary artery disease & myocardial infarction	CAD is one of the most common causes of HF. CVD include history of coronary artery disease (e.g., heart attack, angina); stroke or transient ischemic attack (TIA); arrhythmias; or HF. Over time, coronary arteries narrow from atherosclerosis. Heart muscle downstream from narrowed arteries may be hypoperfused. It may be enough to allow the muscle to remain alive but not function well. Once an MI occurs, necrotic muscle will not pump as well.
Hypertension	(75% of pts): Resistance from stiff or constricted blood vessels forces heart to work harder. Over time, the LV enlarges to compensate for the extra work. Eventually, it may become too stiff or too weak to pump blood effectively. Data from the Framingham Heart Study indicate that HTN is a very common risk factor contributing to a large proportion of HF cases *(JAMA. 1996; 275:1557–1562).*
Diabetes (DM)	Diabetes increases risk of CAD & HTN. A study of the predictors of HF among women with CAD found that DM was the strongest risk factor. *(Circulation. 2004;110:1424–1430.)* The prevalence of DM is ↑ among older adults with HF and is a significant independent risk factor for death in these pts. Mayo Clinic researchers found the odds of having DM for those first diagnosed with HF in 1999 was nearly 4 X higher than for those diagnosed 20 yrs earlier.*(N Engl J Med. 2006;355:251–259.)*
Select diabetes meds	Avandia (rosiglitazone) and Actos (pioglitazone) have been found to increase the risk of HF.
Dysrhythmias	Tachycardias cause increase in cardiac workload which can cause the heart to weaken and fail. Bradycardias can cause hypotension and prevent coronary artery filling (MAP must be 60 or above to fill CA) leading to HF.
Faulty valves	A damaged valve forces the heart to work harder to keep blood flowing in the right direction. Valves that leak or don't open wide enough interfere with blood flow through the heart so it can't pump properly. Over time, this can weaken the heart muscle.
Cardiomyopathies	Form of myocardial disease related to viral infections, injury from toxic effects of alcohol, drugs (cocaine), some drugs used for chemotherapy, and malnutrition. Lupus and thyroid disease can also damage heart muscle. Types: Idiopathic, alcoholic, toxin-related (adriamycin), postpartum, hypertrophic (heart is abnormally enlarged. Less blood can be pumped by the LV that cannot fully relax during diastole), dilated (less blood can be pumped by the LV which is enlarged and weakened), tachyarrhythmia-induced.
Myocarditis	Inflammation of heart muscle often associated with a bacterial, viral, or syphilitic infection. Myocarditis, cocaine or alcohol abuse, and cardiotoxic chemotherapies are important causes of HF in younger patients (Kosowsky & Kobayashi, 2002).
Congenital heart defects	Increase risk of heart disease which can lead to failure.
Kidney conditions	Any renal disease the leads to HTN can also lead to fluid retention and HF
Hyperkinetic states	Anemia, arteriovenous fistula, thyroid disease, Beri-beri
Others	Acute papillary muscle dysfunction, ruptured chordae tendinae, acute aortic insufficiency, prosthetic valve dysfunction, ruptured septum
Secondary causes	Drugs that alter cardiac function (the toxic effects of certain drug overdoses [beta or calcium blockers] can be reversed if identified early), cardiac tamponade, pulmonary embolism (causes acute RV failure), atrial myxomata (benign primary tumor covered by thrombus formation arising from the lining of the atria; may simulate mitral or tricuspid stenosis), superior vena cava syndrome.

F. **Common precipitants of existing HF decompensation**

1. Medication noncompliance; non-adherence to dietary restrictions
2. Uncontrolled hypertension; myocardial ischemia/infarction
3. Acute valvular dysfunction
4. Cardiac arrhythmias
5. Pulmonary and other infections
6. Administration of inappropriate meds (neg inotropes)
7. Fluid overload; missed dialysis
8. Anemia; alcohol withdrawal

G. **Medications – see Table 3**

1. **Important questions to ask**

   a. **"What medications do you take?"** Avoid asking only about medications taken every day – some are taken every other day, some are injected every other week, etc. Also avoid asking if they take any prescription medications. These questions suggest that you don't want to hear about the aspirin they take every day or the numerous over-the-counter antihistamines they have been taking.

   b. **"What other medications, drugs, vitamins, etc. do you take?"** No matter how many medications a patient initially says they take, ask "What else?" until the patient says, "That's all." Patients often forget some or get interrupted and lose their train of thought.

   c. **"What medications are you supposed to be taking?"** and **"What medications have you been on before?"** These are open-ended questions that should be asked if a patient denies taking any medications. They often reveal something about the past medical history or the fact that a patient may not be taking or have stopped taking a medication without consulting with their physician.

   d. **"Have you been taking your medicines the way you were instructed to?"** Patients often decide to alter their medication routine for various and strange reasons. They may or may not admit to it.

   e. **"When was your last dose?" and "How much did you take?"**

   f. **"Has there been any recent change in your medications?"** Increases or decreases in types of meds or doses may shed light on their current condition.

2. **Ace inhibitors**: All pts. With LV systolic dysfunction should be treated with an ACEI unless contraindicated or not tolerated. Considered first-choice drug therapy for HF. ACEI have proven to slow the progression of HF. They prevent the conversion of angiotensin I to angiotensin II which is a potent vasoconstrictor. Thus they **vasodilate the patient, lower the BP, and decrease the heart's workload and restore the balance between myocardial oxygen supply and demand.** They reduce LV mass, reduce sympathetic stimulation and have a direct antiatherogenic effect. Blunts aldosterone release so prevents salt and water retention.

   SE: Some people develop a persistent cough. Common to feel weak or dizzy when first taking these drugs due to the reduction in BP and inability to compensate rapidly for changes in position. Other SE: Skin rashes, altered sense of taste, hyperkalemia. Pts may need their K levels checked regularly, especially if they also take diuretics or potassium supplements. Nausea and HA possible.

3. Angiotensin Receptor Blockers: "Sartans"
4. Beta Blockers: "LOLs"
5. Calcium Channel Blockers
6. Diuretics
7. Vasodilators
8. Anticoagulants (AF)
9. Antiarrhythmics

H. **Signs and symptoms – how will they present?**

1. **Dyspnea** (DOE): suggests that the patient is unable to meet metabolic or oxygen demands as a result of impaired cardiac output or decreased alveolar diffusion.
2. **Tachypnea w/ ↑ WOB;** decreased compliance, airflow obstruction, development of dynamic hyperinflation, and ↑ WOB (↑ pressure/↓ volume) occurs as lungs become "stiffer" overwhelming the capacity of the respiratory muscles.
3. Accessory muscle use; ✓ for inspiratory retraction of the intercostal spaces and supraclavicular fossae

4. ✓position (tripod) will help expand chest and shows that they have muscle tone
5. **Orthopnea** (inability to lie flat): Indicates a progression of dyspnea
6. **Paroxysmal nocturnal dyspnea** (PND): Progression of orthopnea – Patient wakes soon after going to bed or after lying down with sudden onset of dyspnea caused by progression of heart failure when dependent body water enters back into the central circulation and floods the lungs. Requires them to sit up or stand in order to breathe more easily.
7. Frequent nocturia: fluid returns to kidneys when supine
8. Inspiratory/expiratory ratio – prolonged expiratory phase
9. Breathing w/ pursed lips – own PEEP
10. Ask pt to take deep breath & ask about pain: Pleuritic chest pain may suggest cardiac infection, pleurisy, pleural effusion, pneumonia, or costochondritis. Complaint of chest, neck, jaw or arm pain if ischemia or AMI present may be obscured or ignored due to the sudden severe dyspnea.
11. Agitation, severe apprehension, confusion, & weakness may be due to decreased perfusion to brain from decreased cardiac output – especially in the elderly.
12. Cool, pale, ashen or cyanotic skin with diaphoresis – compromised perfusion. Ask about chills or fever (pneumonia).
13. **Cough**: frothy (occasionally pinked-tinged) sputum (surfactant washing out) vs. yellow or green (COPD/pneumonia)
14. **SpO_2 less than 94%** on room air.
15. **Capnography**
    a. Provides valuable information about the adequacy of ventilations, changes in dead space, and pulmonary perfusion so care is purposeful and tailored to patient's needs.
    b. In HF, should have a normal, squared off waveform vs. asthma/COPD that usually presents with a shark-fin waveform due to delayed exhalation.
    c. As the CO improves, capnography should show an ↑ in end tidal CO_2 from 28 up to 40 mmHg.
    d. If compensatory mechanisms fail, bradypnea and hypoventilation will cause CO_2 retention resulting in confusion, head bobbing, then progress to lethargy and unresponsiveness as patient goes into respiratory failure. If patient decompensates, will need to be intubated and ventilated.
16. **Breath sound assessment**
    a. **Crackles**
        (1) **Definition** - Short explosive discontinuous sounds caused by sudden equalization of gas pressures between two compartments of the lung. Lower airways close first on exhalation. If high surface tension within alveolus, reopening on inspiration results in popping sounds or crackles. Described according to pitch, timing and location. Timing indicative of underlying pathology.
        (2) **Late inspiratory bi-basilar crackles**: Produced by the sudden opening of collapsed airways and adjoining alveoli; usually described as high-pitched, explosive sounds that vary in intensity. Although usually associated with left-sided ventricular failure, they may be heard in poorly ventilated atelectatic lung areas.

        Note: The pattern of crackles typically changes over different areas of the lung bases. Localized crackles are heard over areas of early or non-consolidating pneumonia.

(3) **Characteristics:**

(a) Variable intensity: If the patient has a very hairy chest, hold the stethoscope firmly against the skin to minimize the crackling noises produced by the hair.

(b) Basilar and symmetrical

(c) Gravity-related traction on small peripheral airways

(d) No change from coughing; secondary to LV heart failure, early pneumonia or pulmonary fibrosis. Crackles secondary to atelectasis usually clear with coughing.

(4) **Early inspiratory and expiratory crackles**

Produced by widespread airflow obstruction in the central airways; loud, low-pitched, scanty and well conducted, especially to the lower lobes. Can be heard throughout both lung fields in patients with chronic bronchitis.

b. **Wheezes**

(1) Definition: Continuous, harmonic, musical sounds

(2) Described according to pitch, duration, timing and complexity

(3) Pathology: Produced when air passes rapidly through a bronchus so narrowed that it is almost closed. As the bronchial walls fluctuate between closed and barely open, they generate audible sounds.

(4) **All that wheezes is NOT asthma or COPD**! The bronchioles are sensitive to any type of irritation. When body water collects around the terminal bronchioles and irritation occurs, the bronchioles constrict in response to that irritation. This also causes wheezing (cardiac asthma). Do NOT treat these patients with albuterol/ipratropium or epinephrine!

c. **Diminished or absent sounds**

(1) Usually associated with a pneumothorax, COPD, severe airway obstruction, severe atelectasis, or pleural effusion. Breath sounds will be diminished bilaterally in patients who have COPD or are obese, but will differ from the right to left lung with unilateral pneumothorax or pleural effusion. Reflection of sounds away from the chest wall is due to a mismatch of acoustical properties of diseased areas and the chest wall. Even a shallow pneumothorax will reflect sounds. Sounds will not return until all air is absorbed and pleurae are in contact again.

(2) Wheezes may diminish if the bronchoconstriction becomes so severe that airflow velocity drops below the level necessary to produce audible wheezes. The disappearance of wheezes needs to be correlated with the patient's ability to move a large enough volume of air. **Access their ability to speak in complete sentences without gasping for breath.**

d. **Accurate documentation**: Lung sounds must be documented in all patients with a chief complaint or physical exam evidence of ventilatory, respiratory, or cardiovascular disease or distress. Note the underlying sounds as normal, diminished, or absent for the both the left and right sides and any adventitious sounds (wheezes, crackles, stridor).

17. **Cardiovascular assessment/monitoring/support**

a. ✓general rate and quality of pulse: would expect strong with HTN; if weak, suspect HF; check for pulse deficit

b. **Apply ECG monitor and interpret rhythm.**

Sinus tach (HR 101 up to 150; can range as high as 180) - Physiological response due to ↑ sympathetic tone with the release of catecholamines caused by pain, anxiety stress, exertion, or hypoxia. Catecholamines have a negative impact on myocardial ischemia, automaticity, cardiac work and the threshold for VF (Circulation 45:703). HF (due to myocardial damage) and hypovolemia should be considered as causes of ST.

Palpitations or irregular pulse: ask if hx of AF; PVCs common: A-tach, A-flutter and A-fib may be due to atrial ischemia but are often associated with HF as they are caused by the same underlying conditions: HTN, CAD, overstretched atria, or valve disease. In pts with abnormal systolic or diastolic function, loss of "atrial kick" can have profound consequences.

In AF, blood in the atria tends to collect rather than be ejected which causes **microthrombi** to form on the interior atrial walls. If one of these clots breaks loose, it can cause a pulmonary embolism (right heart) or stroke (left heart). If patient has had AF, they will usually be anticoagulated.

If AF is accompanied by a rapid ventricular response, the tachycardia is problematic because it means reduced filling time and ↑ myocardial oxygen demand. When hemodynamic compromise (hypoperfusion/↓ BP) occurs, restoration of a normal rate (at hospital) is a goal of therapy.

Since tachycardia further increases myocardial oxygen demand, the ideal HR is 60-90. However, treatment should be aimed at removing the cause of the tachycardia. **Do NOT directly slow the HR** by giving negative inotropes (NO Verapamil) to a patient in LV failure. If new-onset AF has precipitated acute pulmonary edema, cardioversion may be indicated at the hospital. EMS should cardiovert if hemodynamically unstable.

c. BP is usually high in HF; if low suspect cardiogenic shock. If hemodynamic compromise (hypoperfusion/↓ BP) occurs due to bradycardia (unusual), restoration of a normal rate must be an immediate goal of therapy.

d. Obtain review, and transmit **12-L ECG ASAP** to r/o AMI as cause. Obtain while stationary; may transmit while moving. If unable to transmit, read interpretation to hospital. If 12 lead ECG indicates **AMI** (STEMI)**:** Notify receiving hospital ASAP.

(1) **The ECG is likely to be abnormal in patients with HF**. LV hypertrophy is seen in almost ½ and evidence of old MI may be seen in up to 70% of patients. A completely normal 12L is actually evidence against LV dysfunction and should prompt consideration of other alternative diagnoses.

(2) If **acute ischemia**; give NTG and treat per ACS SOP.

(3) If **age undetermined**, may use NTG dosing for pulmonary edema.

(4) Typically, if an AMI is severe enough to cause acute pulmonary edema, there will be associated hypotension.

(5) Ventricular hypertrophy will be evident in the leads that overlie the affected ventricle. R wave may be very tall in V5 or may be seen in V6, exceeding 25 mm in height. Also look for a deep S wave (>25 mm) in V1 or V2. Look for ST depression or T wave inversion in those leads suggesting strain on the ventricle.

(6) Leave a copy of the 12 L tracing with the PCR in the ED.

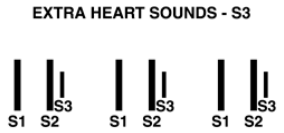
18. **Heart sounds**

a. **S1 (lub)**: Occurs at the beginning of ventricular systole with the closure of the mitral and tricuspid valves. Most audible at the apex and lower sternal border. Fairly high-pitched; harder to hear during tachycardia. If may be

"split" or heard as two sounds clumped together in the presence of right bundle branch block. Varies in intensity with atrial fibrillation.

b. **S2 (dub)**: Occurs at the beginning of diastole when the aortic and pulmonic valves close. Most audible at the base of the heart (aortic area). Higher in pitch than S1.

c. **S3** (3rd sound; **ventricular gallop)**: Dull, low pitched sounds, occur early in diastole with a cadence like "Ken-tuck-y". Thought to be caused by the early, rapid filling of a dilated LV. Most audible at the apex with the pt on his left side. Normal in children and young adults. Considered abnormal in patients over 30. Seventy percent of pts > 40 yrs with an **ejection fraction < 30% will have an S3** due to ventricular overdistention.



d. **S4 (atrial gallop)**: Also normal in children and young adults. Associated with the forceful ejection of blood into a stiff ventricle. Most audible at the apex with the pt on his L side. Also dull and low pitched. It is especially hard to hear as it is very soft. Abnormal in adults. Occurs late in diastole and may be caused by pulmonic stenosis or any condition that affects LV compliance, e.g., aortic stenosis, hypertension, MI, and cardiomyopathy.

19. A laterally displaced, often diffuse, apical impulse is due to ventricular enlargement.

20. **Assess neck veins for JVD**

a. Best measured with patient supine and head elevated 45° (20°- 60° range). Adjust angle so top of jugular vein is in the middle of the neck. Measure height of venous column from angle of Louis; add 5 cm to determine measurement from RA

b. Results from elevated RA pressure so may not be present with acute LV failure. May be present if chronic right HF and fluid retention.

21. **Hepatojugular reflux**

a. Inspect for ascites, apply steady pressure to upper abdomen

b. If hepatomegaly with portal hypertension, blood column in jugular vein will immediately rise.

c. If veins remain distended after patient resumes normal breathing (within 10-15 seconds), the finding suggests right HF.

22. **Peripheral edema**

a. When systemic venous pressure becomes too high (RHF), serum is forced into interstitial tissues, resulting in peripheral edema. Ask the patient about their normal baseline. Over what time did the edema develop?

(1) Lower extremities on upright patient
(2) Sacral region in bedridden patient
(3) **Pitting**: Assess how high is it pitting?
(a) 0 - ¼": +1 pitting edema
(b) ¼ - ½: +2 pitting edema
(c) ½ - 1": +3 pitting edema
(d) 1" +4 pitting edema
(4) Entire body - *anasarca* (moonface, distended abdomen)

b. **Fluid accumulation in serous cavities**

(1) Abdominal cavity – *ascites*
(2) Pleural space - pleural effusions
(3) Pericardium - pericardial effusion/rub

c. Check warmth of legs to r/o DVT

VI. **Guidelines for treating cardiogenic pulmonary edema**

If NONE to MILD cardiorespiratory compromise: Alert, normotensive or hypertensive **(SBP ≥ 90 and DBP ≥ 60):**

A. **Main treatment goals:** EMS care is **aimed at restoring a balance to oxygen supply and demand to tissues**.

1. ↓ cardiac workload ;↓ fluid pressure in pulmonary vessels; ↓ lung water
2. ↓ work of breathing; ↓ O_2 demands; Keep alveoli open
3. ↑ O_2 diffusion; ↑ pulse ox; ↑ cardiac output and improve systemic tissue perfusion by improving the pumping action of the heart

B. **Position**: Place patient at rest. If not in shock or intubated, **sit them up** with the legs in a dependent position. High Fowler's position will increase lung volumes and vital capacity, decrease the work of breathing, and decrease venous return to the heart (AHA, 2010).

C. **Airway assessment and control**: If severe respiratory distress: Assess need for DAI.

1. DAI is only indicated for patients in or near the late stages of respiratory failure or arrest.

2. **Avoiding intubation** is preferable to prevent complications:

a. Having to sedate or paralyze patients more than desired
b. Unexpected difficulties while intubating or improperly performed procedure/placement
c. Oral trauma, misplacement
d. Barotrauma (see more with intubation and BVM ventilation than CPAP)
e. Nosocomial infections: sinusitis, Otitis, MRSA, Klebsiella
f. Aspiration risk during the procedure
g. Anxiety and discomfort (patients still awake); communication challenges
h. Ventilator-associated pneumonia

3. Up to 90% of pts with pulmonary edema can avoid intubation if placed on CPAP

D. **Ventilations/gas exchange**

1. **Oxygen**: Most patients are hypoxic due to ventilation/perfusion (V_A/Q) abnormalities. Supplemental O_2 is given to reverse hypoxia and decrease pulmonary vascular resistance. Give O_2 via **CPAP** mask if not contraindicated. If CPAP is contraindicated, apply O_2 15 L/NRM or assist with BVM.

2. **Physiological benefits** of C-PAP

a. Ventilatory O_2 consumption in healthy patients is < 4%. When ill, this can escalate to 20%, remarkably increasing the work of breathing (WOB).

b. Most alveoli collapse during exhalation except at the apices. If the lungs are dry, surfactant allows them to reopen without resistance. In pulmonary edema, the surfactant is washed out, producing stiff air sacs that only open with considerable effort.

c. If one exhales against resistance, alveoli and smaller airways don't collapse, energy is not consumed on the next inhalation, **WOB is reduced**, and gas exchange time can be doubled; thereby ↑ **pO_2** (pulse ox) and **improving exhalation of pCO_2** (capnography).

d. Keeping alveolar pressures high throughout the respiratory cycle increases the driving pressure of oxygen across the pulmonary capillary membranes, reducing hypoxia. CPAP will not immediately drive water out of alveoli, but will prevent more from flooding in.

e. ↑ **cardiac output (CO):** With a healthy heart, the pulmonary capillary wedge pressure (PCWP) is < 23 and CO is determined by preload. Thus ↑ fluid = ↑ output. In pulmonary edema, PCWP is high. CO now becomes dependent on afterload. CPAP ↑ pressure throughout the thoracic cavity,

including that surrounding the LV making it easier to move blood out of the LV. Similarly, pressure surrounds the thoracic but not the abdominal aorta, giving the impression of reduced afterload outside of the thoracic cavity.

3. **Indications for CPAP if HF suspected** (NWC EMSS)

   a. 18 years or older; alert, intact airway, good ventilatory drive
   b. Normotensive or hypertensive BP (SBP ≥ 90 and DBP ≥ 60): CPAP will ↑ intrathoracic pressure which can ↓ preload and drop BP. Need MAP of >60 to fill coronary arteries.
   c. Bilateral crackles or wheezes w/ paramedic impression of left HF

E. While prepping for 12 Lead: **ASPIRIN 324 mg** (4 tabs 81 mg) chewed and swallowed. AMI is one cause of acute HF. Patients with HF at ↑ risk for arterial or venous thromboembolic events. In addition to AF and poor ventricular function (promote stasis and ↑ risk of thrombus formation), pts with HF have other manifestations of hypercoagulability.

F. IV access; NS TKO

G. **First line drugs if BP normal or elevated.** Patients with pump failure may require treatment for a co-existing rate or volume problem and/or correction of other underlying problems (hypoxia, hypoglycemia, drug overdose, or poisoning) in addition to supporting the failing heart. No single drug or drug class is sufficient to provide complete relief. Pump support may ultimately require venodilators to reduce preload; vasodilator therapy to reduce arterial resistance (afterload); inotropes to improve contractility, mechanical assistance (intra-aortic balloon pump) or surgery. Use caution in dosing drugs if RV failure is also present as liver function may be compromised and drug metabolism and detoxification may be impaired.

Profile: NITROGLYCERIN for HF	
Classification	Nitrate; antianginal, antihypertensive, vasodilator
Indications	Cardiogenic pulmonary edema if **SBP ≥ 90 and DBP ≥ 60** **No other class of drugs improves the symptoms of congestion as rapidly as nitrates.**
Action for PE	• NTG metabolizes into nitric oxide, which forces calcium out of vascular smooth muscle cells, causing them to relax and dilate. Low doses initially dilate the veins (tabs 1-3) • Pools blood in the periphery, **reduces RV preload** and pulmonary blood flow (↓congestion) • Dilates coronary arteries -Increases myocardial O2 supply. • Decreases LV filling pressure. Reduces systolic wall tension and ventricular work; thus **↓ O_2 demand** • At higher doses (tabs 4+): Dilates systemic arteries; reduces the pressure against which the ventricle pumps (**↓ afterload**), also reducing cardiac work and facilitating cardiac emptying.
Indication	LV failure w/ cardiogenic pulmonary edema or HF if SBP > 90 & DBP > 60
Dose and route	0.4 mg SL q.3 - 5 minutes as long as systolic BP > 90 systolic - **NO DOSE LIMIT** SL route has approx. a 75% absorption rate of the NTG. Pt. will get about 300 of the 400 mcg dose, or 60 mcg/min.
Onset of action	1-3 minutes SL half life 5 min
Contraindications	Hypotension (SBP < 90 or more than 30 mmHg below baseline), hypovolemia, known RV MI (relative) Patients with shock caused by RV failure have a mortality rate similar to that for pts with shock due to LV failure. Patients with RV dysfunction and acute infarction are dependent on RV "filling" pressure (preload) to maintain cardiac output Thus, nitrates, diuretics, and other vasodilators (ACE inhibitors) should be avoided because severe hypotension may result. This hypotension is often easily treated with an IV fluid bolus. Recent use of Viagra or Levitra (vardenafil) w/in 24 hrs or Cialis (tadalafil) w/in 48 hrs
Side effects	- CV: Decrease in BP of 10% in the normotensive patient; 20% in the hypertensive pt. This reduction may cause a reflex tachycardia with an increase in myocardial oxygen demand and worsening ischemia. Limit BP drop to 10% if patient is normotensive; 30% if hypertensive.. An IV fluid bolus of 200 mL should be administered if lungs are clear. - CNS: Headache, dizziness, light-headedness, syncope, ringing in ears - Burning sensation under tongue, flushed skin, N/V - HEME: Methemoglobinemia

Anxiolytic (anti-anxiety): Used to ↓ anxiety and ↓ myocardial oxygen demands. Midazolam in **2 mg increments** every 30-60 sec IVP (0.2 mg/kg IN) up to 10 mg IVP/IN/IM. May repeat to 20 mg if BP > 90.

VII. **Cardiogenic shock**

A. The most severe form of pump failure occurs when LV function is so compromised that the heart cannot meet the metabolic needs of the body and compensatory mechanisms are exhausted. The hypotension that accompanies this form of shock aggravates the situation by decreasing coronary perfusion, further suppressing cardiac performance, and ultimately resulting in total pump failure (Bledsoe, 2006).

B. Definition: Hypoperfusion persists after correction of existing dysrhythmia, hypovolemia, or altered vascular tone.

C. Cardiogenic shock is usually due to extensive MI of the LV, diffuse ischemia, or decompensated HF. There is a poor prognosis when > 35-40% of the LV is destroyed. Historically, about 7.5% of patients with AMI develop cardiogenic shock and mortality rates range as high as 80% even with appropriate therapy.

D. Hypoxia, resulting from ventilation/perfusion abnormalities in the lung, occurs in early shock and decreases contractility. In late shock it worsens, and becomes "malignant" or irreversible because of the low perfusion state.

E. Acidosis results from anaerobic metabolism with release of lactate and pyruvic acids accompanied by decreased renal perfusion and accumulation of organic acids. Myocardial ischemia develops when arterial pressure falls and further compromises contractility.

F. **Triad of cardiogenic shock**

1. Pump failure
2. Pulmonary edema
3. Hypotension: Systolic below 80 to 90 mm Hg or a reduction of 70 mm Hg or more.

G. **Differential diagnosis of cardiogenic shock**: When presented with a hypotensive patient who appears to be in cardiogenic shock, also consider the possibility of the following:

1. Aortic dissection
2. Massive PE
3. Septic shock

H. **Clinical presentation**

1. Signs and symptoms reflect evidence of forward and backward failure
2. **Hypotension**: Systolic BP < 90 mmHg or a 30-60 mmHg drop from previous baseline levels. Patients in early cardiogenic shock may not be hypotensive.
3. **Evidence of decreased blood flow to major organ systems**
   a. Peripheral vasoconstriction associated with cold, diaphoretic skin that has a dusky or ashen color.
   b. Symptoms of cerebral hypoxia such as restlessness, apprehension, and confusion which may progress to apathy, lethargy and coma.
4. Evidence of left-sided heart failure: tachypnea; impaired gas exchange; profound respiratory failure; **pulmonary congestion/edema**
5. When dysrhythmia exist, it may be difficult to know if it is the cause of the hypotension or the result of cardiogenic shock. Correct any major dysrhythmias.
6. Compensatory tachycardia with weak, thready pulses (carotid)
7. Elevated filling pressures (requires invasive monitoring)

I. **Emergency interventions for cardiogenic shock**

1. **Time sensitive patient**: Prolonged stabilization in the field is contraindicated - expedite transport
2. Place patient in supine position

3. Secure an open airway; suction prn; monitor SpO_2; administer 15 L O_2/NRM or BVM – NO CPAP due to hypotension. Consider drug assisted intubation (DAI) if necessary to ↓ work of breathing (WOB), protect airway or ventilate patient
4. Assess carefully for S&S of hypovolemia/dehydration
5. Monitor ECG; obtain 12-lead ASAP
6. Treat underlying problems or dysrhythmias per appropriate SOP
7. **IV access** with NS. Some patients appear to be in cardiogenic shock when they are actually volume depleted or experiencing a RV infarct. Auscultate lungs. If **clear and respirations are not labored**, may try 200 mL NS fluid challenges to increase preload. Frequently re-evaluate lung sounds.
8. **Drug therapy: If alert with gag reflex:** Chewable **ASA** 4 tabs PO.

   Inotropes: Aortic root pressures must be maintained at a minimum MAP of 60 mmHg to create a pressure head for the flow of blood into the coronary arteries. If MAP < 60 mmHg, most patients will die. Getting the pressure up with vasopressors may be done at the temporary expense of other target organs.

Profile: DOPAMINE	
Generic name	Dopamine (Intropin)
Indications	Hemodynamically significant hypotension (SBP ≤ 70-100) not caused by hypovolemia. Cardiogenic shock (low doses); anaphylaxis, neurogenic, septic shock (higher doses)
Action	Dopamine is chemically related to epinephrine and norepinephrine and increases BP by stimulating alpha and beta-1 receptors. **Action is dose dependent.** • Dilates renal and mesenteric blood vessels at very low doses (1-2 mcg/kg/min) • At low doses, it acts as a beta agonist (by stimulating adenocyclase in the cell which converts ATP to cyclic AMP). C-AMP allows or increases calcium entry into the cell allowing the cross bridging of myosin and actin in the sarcomere. This causes a **positive inotropic effect** (↑ myocardial contractility). It does not increase myocardial O_2 demand as much as Isuprel and epinephrine and does not have the same powerful chronotropic effects. • In higher doses, it acts predominately on **alpha** receptors causing peripheral vasoconstriction. When used in therapeutic doses, dopamine maintains renal and mesenteric blood flow because of its effects on the dopaminergic receptors. For these reasons, dopamine is currently the most commonly used vasopressor. It will increase the BP and pulse pressure, but there is generally less effect on the DBP.
Onset of action	5 minutes
Duration of action	10 minutes IV
Contraindications	Hypovolemia unless fluid volume resuscitation is underway. Pheochromocytoma (tumor of the adrenal gland) Presence of tachydysrhythmias
Precautions	• Unfortunately, the patient is often maximally SNS stimulated resulting in a feedback mechanism which creates more phosphodiesterase (the enzyme that breaks down C-AMP) and dopamine may not work. • Treat severe brady/tachycardias (HR > 140) first if present • Dopamine will ↑ HR and can induce or worsen SVT and VT
Side effects (common)	Tachycardia, palpitations, dysrhythmias, chest pain Nervousness, headache, dyspnea, N/V. Many of these are dose related.
Dose and route	Beta dose: 2-10 mcg/kg/min Alpha predominates > 10-20 mcg/kg/min Start at 5 mcg/kg/min; titrate up to 20 mcg/kg/min or until BP maintained > 90
How supplied	Drips are typically mixed at 800 mg/500 mL or 400 mg/250 mL IVPB. Concentration is 1600 mcg/mL
Interactions	Like all catecholamines, can be deactivated by an alkaline solution (sodium bicarb)

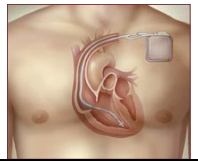
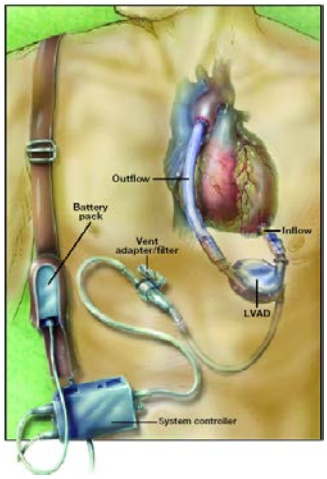
Quick notes on dosing: Obtain patient's weight in pounds. Take first 2 numbers: Ex: 200 pound patient.

Subtract 2 from that number: 20 - 2 = 18
Drip is run at 18 microdrops per minute to administer 5 mcg/kg/min.

Definitive care at hospital: Bedside echocardiogram; cath lab; intra-aortic balloon pump; VAD

Table 3 Prescription drugs used in the treatment of Heart Failure – See SOPs		
Angiotensin converting enzyme (ACE) Inhibitors (ACEI)	Benza***pril***/Lotensin Captopril/Capoten Enalapril/Vasotec Fosinopril/monopril Lisinopril/Prinivil/Zestril Moexipril/Univasc Perindopril/Aceon Quinapril/Accupril Ramipril/Altace Trandolapril/Mavik	Generic names end in ***PRIL*** Considered first-choice drug therapy for HF. ACEI have proven to slow the progression of HF. They prevent the conversion of angiotensin I to angiotensin II which is a potent vasoconstrictor. Thus they vasodilate the patient, lower the BP, and decrease the heart's workload. Blunts aldosterone release so prevents salt and water retention. SE: Some people develop a persistent cough. It is common for them to feel weak or dizzy when they first start taking these drugs due to the reduction in BP and inability to compensate rapidly for changes in position. Other SE: Skin rashes, altered sense of taste, hyperkalemia. Pts may need their K levels checked regularly, especially if they also take diuretics or potassium supplements. Nausea and HA possible.
Angiotensin II Receptor Blockers (ARBs)	Cande***sartan***/Atacand Eprosartan/Teveten Irbesartan/Avapro Losartan/Cozaar Olmesartan/Benicar Telmisartan/Micardis Valsartan/Diovan	Generic name ends in ***sartan*** These drugs prevent angiotensin II from having any effect on the heart or blood vessels. Lowers the BP without the SE cough of ACEIs. SE: very few. May cause nausea, dizziness, headaches and low BP.
Anticoagulants	Heparin Clopidogrel/Plavix Warfarin/Coumadin Rivaroxaban/Xarelto ASA-containing products	Used as prophylaxis against clots in CA, in those with AF (blood pooling in atria can cause mural thrombi) or in those with Hx of stroke. SE: nosebleeds, bleeding gums, easy bruising. Minor inj can cause major bleeding.
Beta Blockers (BB)	Acebuto***lol***/Sectral Atenolol/Tenormin Betaxolol/Kerlone Bisoprolol/Zebeta Carvedilol/Coreg Labetalol Metoprolol/Lopressor/Toprol Nadolol/Corgard Pembutolol Pindolol Propranolol/Inderal Timolol/Blocadren Sotalol/Betapace	Generic name ends in ***"LOL"*** The failing heart tries to compensate for its weakened pumping action by beating faster, which adds an additional strain. Beta blockers slow the HR by blocking beta-1 receptors. Used for mild to moderate HF and often with other classes of drugs (diuretics, ACEI and digoxin) SE: May be able to tolerate less strenuous physical activity. Nausea & weakness after lifting or exertion. May also cause hypotension, dizziness, headache, and worsening asthma symptoms (beta-2 receptors may be blocked in lungs producing bronchoconstriction).
Calcium channel blockers (CCB)	Amlodipine/Norvasc	Muscle cells need calcium to contract. Calcium blockers can be used to treat HTN associated with HF. SE: Because Ca blockers relax smooth muscle, they all can cause hypotension, HA, facial flushing, dizziness, and nausea
Digoxon	Digoxin/Lanoxin Digitoxin	Digoxin slows HR and conduction through AV node. This optimizes the Starling reflex (increased stretch = increased contractility). Increased SV relieves HF symptoms, especially if pt is not responding to ACEI and diuretics. Slows the rate of AF. SE: Can be severe with dig toxicity. Anticipate a slow HR and prolonged PR intervals.
Diuretics	Amilor***ide***/Midamor Bumetanide/Bumex Chlorothiazide/Diuril Diazide Furosemide/Lasix Hydrochlorothiazide/Hydrodiuril Indapamide/Lozol Metolazone/Zaroxolyn Polythiazide Torsemide	Diuretics cause the kidneys to excrete more sodium, magnesium, and water in the urine. Less total blood volume reduces the heart's workload. They also reduce the build up of fluid in the lungs and dependent areas of the body (feet and ankles). Variety of mechanisms of actions. Some act on the renal tubules, others block aldosterone (Spironolactone/Aldactone and Inspra/eplerenon) or produce changes in the osmolar balance. Some may prevent heart scarring. SE: A therapeutic dose level may be difficult find to avoid over or dehydration. Some cause potassium losses in the urine and others are potassium sparing,

	Trianterene/Dyrenium	
Vasodilators	Hydralazine/Apresoline Isosorbide/Isordil Minoxidil/Loniten Nesiride/Natrecor Nitrates/NTG	Those that don't tolerate an ACEI may be prescribed a different vasodilator to prevent hypertension and improve their tolerance for exercise. SE: See NTG profile

Table 4 Definitive long-term interventions	
Implantable cardioverter defibrillators 	Device implanted under the skin and attached to the heart with small wires. An ICD monitors the heart rhythm. If the HR transitions to a shockable rhythm, the unit will automatically discharge at preset J settings. Is sometimes combined with a biventricular pacemaker for those with severe HF.
Cardiac resynchronization therapy (CRT) or biventricular pacing	Unit sends timed electrical impulses to both ventricles so they pump in synchrony in a more coordinated manner. Up to half of pt in HF have dysrhythmias that cause already-weak heart muscle to beat asynchronously. This inefficient muscle contraction worsens the failure.
Left ventricular assist device (LVAD) 	Battery operated, mechanical blood pump that is surgically implanted next to the native heart. It takes over pumping function of a sick or weakened heart to maintain perfusion to other organs. It is considered a life sustaining device and is a "bridge to transplant" as people await a heart transplant or may be considered an alternative to transplant as they are portable and can be used for weeks to months. A tube pulls blood from the L ventricle into a pump that sends blood directly into the aorta. The goal is to help the weakened ventricle. The pump is placed in the upper part of the abdomen. Another tube attached to the pump is brought out of the abdominal wall and is attached to the battery and control system. In the event of a power failure of system malfunction, if the pt is unconscious and not breathing: ■ **Initiate CPR** & **use standard BLS and ALS SOPs**. Chest compressions are allowed. ■ **Defibrillate if indicated**. Do NOT place pads over the pump. You may **defibrillate at the nipple line or above**. ■ Due to the nature of this rotary pump, that patient **will not have a palpable pulse and you will need a Doppler to obtain a BP**. These patients are anticoagulated with warfarin (Coumadin) (goal INR 2.0-2.5). The patient will have a caregiver who has been trained in the use of the device and MUST remain with the patient at all times. If the patient presents in a medical emergency, contact the Acute Care Nurse Practitioner/VAD Coordinator at the site where the VAD was implanted. Their phone numbers should be readily available with the patient.
Cardiac wrap therapy	Wraps the failing heart in a mesh bag helping to prevent further failure. A surgeon pulls he mesh wrap over the base of the heart (top) and attaches it with sutures. The goal is to prevent a weakened heart from enlarging and failing further.
Ventricular restoration surgery 	Used experimentally to treat patients with HF caused by AMI. Surgeons remove scar tissue in ventricular muscle and reform the remaining healthy muscle to restore a more normal elliptical LV shape. Reducing the size and shape of the LV helps restore normal pumping function.
Enhanced external counterpulsation (EECP)	Noninvasive technique has been used as a treatment for heart-related chest pain and researchers are studying it to see if it would be beneficial for patients with HF. Inflatable pressure cuffs are placed on the calves, thighs, and buttocks. They are inflated and deflated in sync with the heartbeat. In theory, the EECP should promote venous return to the heart.

VIII. **Peripheral vascular & other CV emergencies**

A. **Aneurysm**

1. **Definition:** Non-specific term meaning dilation of a vessel. Types include atherosclerotic, dissecting, and traumatic. Known as the “great imposter”, a century ago, Sir William Osler stated, "There is no disease more conducive to clinical humility than aneurysm of the aorta".

2. **Abdominal aneurysm (AAA)**

a. Caused by atherosclerosis weakening the wall of the aorta causing it to balloon out. Most common site is below renal artery bifurcation (branching); may involve iliac and renal arteries. Ten times more common in men, most prevalent in ages 60-70.

b. **Signs and symptoms**: Abdominal, back or flank pain (can present similar to a kidney stone), hypotension may be present, pulsatile mass - can be palpated when 5 cm, palpate gently, decreased or absent femoral pulse.

c. **Management**

(1) High index of suspicion important.
(2) Treat shock with 02, IV fluids, MAST/PASG if dissecting/ruptured.

3. **Dissecting aortic aneurysm**

a. Small tear in inner wall of aorta allows blood to go under and create false passage between the layers of the vessel wall and hematoma forms. Most (60-70%) involve ascending aorta. Once process begins it may extend to involve all of thoracic and abdominal aorta as well as tributaries, coronary arteries, aortic valve, carotids, and subclavian. It can rupture at any time usually into pericardial or pleural cavity. The etiology is degenerative disease of connective tissue of the aorta.

b. Predisposing factors: HTN in 75-85% of cases, there is some familial tendency, usually involves pts greater than 40-50 years, pregnancy.

c. **Signs and symptoms**

(1) **Pain** - characteristic ripping, tearing, substernal - may radiate to back between scapulae. BP often elevated, yet pt looks "shocky" due to impaired perfusion.

(2) Dissection into other arteries and structures may cause: syncope, stroke, absent or reduced pulses, differences in arm blood pressure, heart failure, pericardial tamponade, acute MI, may also produce neuro deficit secondary to mass effect on spinal roots of nerves - this deficit is often asymmetrical.

d. **Management:** High index of suspicion important

(1) Keep pt quiet.
(2) 100% Oxygen
(3) IV enroute, if possible - no fluid challenges.
(4) PASG if leaking or ruptured
(5) Transport as soon as possible.

4. Thoracic aneurysm (non-traumatic)

a. This is particularly problematic for patients with Marfan's Syndrome.

b. Prior to the advent of aortic surgery, the prognosis for these patients was very grim.

B. **Traumatic thoracic aorta rupture will be reviewed in trauma module**

C. **Acute arterial occlusion**

1. Sudden occlusion of arterial flow by one of the following mechanisms: trauma, thrombosis, embolus (thrombus which forms in L ventricle, arterial thrombi secondary to atrial fib, thrombus from abdominal aortic atherosclerosis), idiopathic. May involve vessels in abdomen or extremities (most common).

2. **Signs and symptoms**

   a. Pain present in 75-80% of cases, sudden, excruciating, peaks within several hours, if absent may be due to paresthesias
   b. Pallor, may be mottled or cyanotic in affected part
   c. May have decreased temp in limb
   d. Shock, if larger vessels, particularity mesenteric (gut) are occluded
   e. Pulselessness distal to occlusion, check symmetry

3. **Management**: Treat shock with oxygen, IV fluids. Protect any affected limb - don't allow pt to walk.

D. **Right ventricular failure**

1. Failure of R ventricle as an effective forward pump results in increased blood pressure in the systemic venous circulation, resulting in venous congestion due to lack of forward blood flow into the heart.

2. Most common cause is L ventricular failure

3. Other causes

   a. Chronic hypertension
   b. Obstructive pulmonary diseases (emphysema, chronic bronchitis, asthma)
   c. Pulmonary embolus (acute onset)
   d. Infarction of R atria or ventricle

   Primary right sided failure secondary to lung disease is called **cor pulmonale**

4. **Signs and symptoms**

   a. Tachycardia – compensatory

   b. **Venous congestion**: When the right ventricle fails, it is unable to keep up with venous return. As stroke volume lessens (lack of forward flow), R atrial pressure rises and pressure is transmitted to vena cava and rest of the venous system (JVD).

   c. **Organ engorgement:** Tender RUQ from liver engorgement

   d. **Peripheral edema in dependent body parts**: When systemic venous pressure becomes too high, serum portion of blood forced into interstitial tissue of body, resulting in peripheral edema. What is baseline? Over what time did condition develop?

      (1) Lower extremities
      (2) Sacral region in bedridden patient
      (3) Pitting: Assess how high is it pitting?
      (4) Entire body - *anasarca* (moonface, distended abdomen)

   e. **Fluid accumulation in serous cavities**

      (1) Abdominal cavity – *ascites*
      (2) Pleural space - pleural effusions
      (3) Pericardium - pericardial effusion/rub

   f. **History**

      (1) Often previous MI's in pts with chronic pump failure

      (2) Frequently give history of taking digitalis and diuretic, (dyazide, Lasix, Bumex, Tenoretic, Moduretic- contain diuretic) or ACE inhibitor (captopril, lisinopril, enalapril).

      (3) Patient may use lay terms to describe their heart failure

g. **Management**

(1) Usually not an emergency in itself- more important to realize relationship to LV failure and potential for acute decompensation
(2) Place pt at rest with head and chest elevated
(3) Room air pulse ox
(4) Oxygen per SpO2; capnography
(5) Monitor ECG
(6) Take baseline VS
(7) Start IV NS TKO
(8) Treat symptoms of L ventricular failure if present

IX. **Non-critical peripheral vascular conditions**

A. **Deep vein thrombosis (DVT)**

1. **Definition**: Thrombosis and inflammation of a vein - commonly occurs in calf or thigh

2. **Predisposing factors (Virchow's triad)**

a. Venous stasis
b. Damage to lining of vessels
c. Predisposition to clotting

3. **Signs & symptoms**

a. Gradually increasing pain – tenderness
b. Swelling of leg & foot distal to DVT
c. Signs & symptoms improve with leg elevation

4. **Management**

a. Prone to develop pulmonary emboli
b. Palpate calf gently
c. Do not allow patient to walk
d. Elevate leg

B. **Varicose veins**

1. **Definition**: Non-critical condition caused by dilation of superficial veins, usually in lower extremities

2. Predisposing factors include genetic predisposition, pregnancy and obesity

3. **Signs & symptoms**

a. Visible distention of leg veins
b. Lower leg discomfort and swelling, especially at end of day
c. Skin color & texture changes in legs & ankles
d. May develop stasis ulcers, over or distal to varicosity
e. Severe bleeding from rupture is the only associated emergency - control hemorrhage with direct pressure

C. **Peripheral arterial atherosclerotic disease**

1. Chronic disease caused by atherosclerosis of abdominal aorta & its tributaries to lower extremities.

2. A gradual, progressive disease - not an emergency. Commonly seen in diabetics.

3. **Signs & symptoms**

a. Characterized by *intermittent claudication* "angina of the calves"; tropic changes in feet; skin rubor on dependency; or ulcers and gangrene.

b. Only associated emergency is development of acute arterial occlusion

X. **Malignant hypertension** (HTN)

A. Occurs in < 1% of pts. with HTN, usually poorly controlled or untreated

B. **"Hypertensive Crisis"** is the name given to the syndrome of blurred vision, focal neuro deficit, seizure, stupor or coma

C. **Signs & symptoms**

1. SBP ≥ 220 and DBP ≥ 130 with S&S of **end organ failure**, including brain, heart, lungs, and kidneys resulting in neurovascular symptoms (i.e. visual disturbances, seizure activity, altered mental status, paralysis,) chest pain and/or pulmonary edema.
2. Mental status change: Restlessness, confusion, somnolence
3. Neurological deficit: Blurred vision, headache
4. Nausea, vomiting, chest pain, dyspnea may also develop L ventricular failure and pulmonary edema

D. **Management**

1. Do not flex neck or knees; maintain head and neck in neutral alignment
2. Assess and record GCS and neuro signs as a baseline.
3. History of or presenting with: HTN, CAD, pulmonary edema from L ventricular failure, dissecting aortic aneurysm, renal disease, DM, hypertension of pregnancy, stroke, or adrenal tumor. HTN in these conditions is usually the result of the primary problem. Treatment is aimed at the primary organ dysfunctions rather than the HTN.
    a. The goal of treatment is to maintain cerebral perfusion and achieve a slow, controlled reduction in BP, rather than an abrupt fall which may cause further neurological complications and ischemia.
    b. Assess for chest pain and/or pulmonary edema. If present: treat per appropriate SOP.
    c. If patient is hypertensive but **without** CV or neurologic signs:
        (1) Reassess and document patient condition/VS q. 15 minutes
        (2) It is important not to use drug therapy to rapidly lower the BP in chronically hypertensive patients.
    d. **IMC** special considerations:
        (1) Keep patient as quiet as possible; reduce environmental stimuli
        (2) Intubate if GCS ≤ 8: consider using DAI and Lidocaine 1.5 mg/kg IVP
        (3) Elevate head of stretcher 10°-15° to ↓ risk of aspiration
        (4) Seizure/vomiting precautions; suction only as needed
        (5) If chest pain or pulmonary edema present: **NITROGLYCERIN 0.4 mg SL**. Treat per appropriate Cardiac SOP.

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DOPAMINE Dose by Weight			
BODY WEIGHT		**DOSE RANGES**	
		Start at 5 mcg/kg/min ß predominates	Do not exceed 20 mcg/kg/min α predominates
100 lbs	45 kg	8 mcgtts/min	32 mcgtts/min
121 lbs	55 kg	10 mcgtts/min	40 mcgtts/min
143 lbs	65 kg	12 mcgtts/min	48 mcgtts/min
165 lbs	75 kg	14 mcgtts/min	56 mcgtts/min
187 lbs	85 kg	16 mcgtts/min	64 mcgtts/min
210 lbs	95 kg	19 mcgtts/min	76 mcgtts/min
240 lbs	109 kg	22 mcggts/min	88 mcgtts/min
260 lbs	118 kg	24 mcgtts/min	96 mcgtts/min

CARDIOLOGY HOMEWORK QUESTIONS

ACS/PATIENT ASSESSMENT - HISTORY (Hx)

1. Compare and contrast the pathophysiology that results in atherosclerosis vs arteriosclerosis:

   Atherosclerosis:

   Arteriosclerosis:

2. List two non-modifiable risk factors of atherosclerosis

   _____

   _____

3. List three modifiable risk factors of atherosclerosis

   _____

   _____

   _____

4. List three pathophysiological results of atherosclerosis:

   _____

   _____

   _____

5. What conditions may result from atherosclerosis?

   _____

   _____

   _____

6. Myocardial oxygen "demand" is determined by:

   _____

   _____

   _____

7. What can compromise myocardial oxygen "supply"?

   _____

   _____

   _____

8. What is coronary artery disease?

9. What happens when a plaque ruptures?

10. When do the majority of sudden deaths from heart disease occur?

    Why has the mortality for cardiovascular disease declined?

11. Differentiate between ischemia and necrosis of heart muscle

    Ischemia:

    Necrosis:

12. List the two main causes of myocardial ischemia:

    _____

    _____

13. Stable angina usually lasts < _____ minutes.

    Pain from an acute MI usually lasts > _____ minutes.

14. List symptoms associated with acute cardiac ischemia besides pain:

    _____

    _____

    _____

    _____

    _____

15. A 63 year old female is complaining of chest pain. If she tells you that the pain was brought on by physical exertion and was relieved by rest, what should a paramedic suspect?

16. If she tells you that she has a history of angina, but this attack lasted longer than usual, and rather than the usual 1 NTG that she takes, it required 3 NTG to relieve the pain you should consider an impression of:

17. If she tells you that the pain came on 20-30 minutes ago while she was watching TV and still isn't gone despite 3 NTG you should consider an impression of:

18. Can discomfort in the jaw, neck, shoulders or arms be due to ischemia even without chest pain?

19. Can a patient be experiencing an MI without any chest pain?

    If so, in what types of patients is this more likely to occur?

20. List two major changes in myocardial tissue produced by ischemia.

    _____

    _____

21. Therefore, list two major complications of an acute MI.

    _____

    _____

22. Why is ECG monitoring important in the patient with myocardial ischemia?

23. Why is it important to check the equality of peripheral pulses in the patient with chest discomfort?

24. List 3 causes of tachycardia in the patient with myocardial ischemia:

    _____

    _____

    _____

    _____

25. Why is tachycardia harmful to a patient with myocardial ischemia?

PRE-HOSPITAL TREATMENT

26. List 5 goals of prehospital management of ACS:

27. How can oxygen administration impact myocardial ischemia?

28. Why should multiple IV attempts be avoided in the patient suspected of having a MI?

    What is the maximum number of attempts allowed by SOP?

29. Why is ASA given to a patient with acute ischemia?

30. What is the prehospital dose of ASA per SOP?

31. List one contraindication for giving ASA to a patient with suspected ACS.

32. Why is pain relief important for a myocardial ischemia patient in the field?

33. Per SOP, what is the minimum systolic blood pressure required to give Nitroglycerin to a patient with ischemic chest pain prior to starting an IV?

34. What are two of the benefits of giving NTG to a patient with ischemia?

35. List four contraindications for giving a patient NTG

36. How long should you wait to assess for a response/pain relief following NTG administration?

37. How should you determine if the NTG tab has retained its potency?

38. If the pain is not gone after the first NTG, should you repeat it? If so, how soon and how many times?

39. Is the pain of MI usually completely relieved by NTG? Why?

40. What are the two most common side effects from NTG?

_____

_____

41. If you have given 3 NTGs and the patient still has pain, what should you give?

42. What is the classification of fentanyl?

43. List two contraindications for giving a patient fentanyl.

_____

_____

44. What is the dose of fentanyl for adults?

45. By what routes can fentanyl be given to a patient with ACS?

46. Under what circumstances can this drug be repeated? Up to how much?

47. What are two side effects of fentanyl?

_____

_____

48. Why is it important to minimize pre-hospital time with a patient suspected of having an AMI?

HF/Pulmonary Edema/Cardiogenic Shock

48. An 86 y/o female complaining of an acute onset of shortness of breath is found sitting upright in bed coughing up frothy sputum. BP 180/90; P 130 and irregular; RR 36 and labored. Lung sounds: bilateral crackles halfway up both lung fields. There is no JVD noted. ECG: atrial fibrillation.

    What should a paramedic suspect?

    A. Right ventricular failure
    B. Acute pulmonary edema
    C. Cardiogenic shock
    D. Myocardial infarction

49. How should oxygen be applied to this patient?

50. What is the benefit of this type of oxygen delivery system?

51. List three contraindications for using the above oxygen delivery system.

    _____

    _____

    _____

52. What drug should be given first to reduce preload?

    How does this drug accomplish that action?

53. What is the initial dose?

54. How often can the drug be repeated?

55. What drug should be given instead if the patient presents with signs/symptoms of hypoperfusion (decreased LOC, pale, cool, diaphoretic, hypotension, decreased capillary refill)?

    _____

56. How many mcg/kg/min should the patient receive initially?

    _____

57. At this dose, do alpha or beta receptors predominate?

    _____

58. If the patient weighs 180 lbs, how many microdrips per minute of the above drug should he receive?

    _____

59. If you infused 10-20 mcg/kg/min, would alpha or beta receptors predominate?

60. What drug (dose & route) is indicated if a patient in pulmonary edema has severe anxiety?

61. List three major S&S of right sided heart failure

62. List two causes of sudden death other than ACS

Vascular diseases

63. What is an aneurysm?

64. Where does an abdominal aortic aneurysm typically develop?

65. List 4 S&S of a dissecting AAA

66. What is the EMS treatment for a AAA?

67. List two mechanisms that would cause acute arterial occlusion

68. List three S&S of acute arterial occlusion

69. What S&S should cause a paramedic to suspect a deep vein thrombosis?

70. What is intermittent claudication?

What disease state would it suggest?

71. How high must the BP get before a paramedic should suspect hypertensive crisis?

What conditions may cause the BP to climb that high?

What neuro deficits are often present in hypertensive crisis?

True or false: Drugs should be given to rapidly reduce the BP in HTN crisis.

72. What drug should a paramedic give first to a patient with hypertensive crisis with pulmonary edema?