This month we are going to take a look at Congestive Heart Failure. Let's take a look at a typical EMS call and see how we can determine what is wrong with your patient.

You are dispatched to the scene of a 74 year old man for respiratory distress. You and your crew know the address well. You discuss with him on your way to the scene and recall that he has a history of hypertension, coronary artery disease, CHF and emphysema. You are frequently called to his home for respiratory distress. What clues will you look for during your assessment to form the correct field impression and plan of care? He has a history of both CHF and COPD, which one is it? Or is it something like pneumonia, pulmonary emboli, pneumothorax, anaphylaxis, aspiration or a MI?

Upon arrival you begin your assessment as your partner talks to his wife to get a history. You find him sitting forward in his chair, his arms resting on his walker. His color is dusky. He does slowly respond to you when you talk to him, however his answers are brief one word responses. You note that he has pursed lips when he breathes with a prolonged expiratory phase. You do not see any retractions or hear any audible breath sounds. His pulse is rapid and irregular. Your EMT-B tells you his vitals are BP 180/110, P 118, RR 32 and labored, pulse ox of 87%. You have the patient placed on a NRB mask at 15 liters. OK... so what do we know so far?

Just looking at him you know he is not good. We need to determine if he is in respiratory distress, failure and arrest. We do this by determining how well he is ventilating and oxygenating. He is sitting upright in his chair to facilitate his breathing, this shows that he still has good muscle tone. Although his response was slow, responding to you shows he is still perfusing his brain, so he is still compensating. Therefore, at this point he is determined to be in respiratory distress. What else do we know?

Airway: It is open and he is able to maintain it

Breathing: We noted his breathing was labored, fast and he was hypoxic. He had a prolonged expiratory phase and pursed lips breathing. When you lifted his shirt to look for retractions you found none. Doing a quick listen of his lungs you hear little to no movement in the bases, but hear wheezes and fine rales as you listen higher.

Circulation: His pulse is rapid and irregular. He is warm and dry, but he does have some circumoral cyanosis.

History: Your partner tells you after interviewing his wife that the patient has no allergies, takes Nitroglycerin, Metoprolol, Warfarin, and albuterol with ipratropium. He has a history of an MI 2 years ago, CHF, COPD and pneumonia one year ago. The wife states the patient is compliant in taking his meds, but does not follow his low salt diet. The patient tells you the shortness of breath came on suddenly while watching TV. He does admit to getting short of breath with activity. He denies having a fever. When asked if he has a cough, he said he did have a cough, but it was dry with no sputum. He denies any chest pain or discomfort. He rates the severity of his distress at 8 out of 10.

After you move him to the rig, you perform a more detailed physical exam. Starting at the head you note that his mucus membranes are moist. Although sitting upright you note that there is JVD present. You listen more carefully to his lungs and in addition to the wheezes and fine crackles described earlier you hear some fine crackles in the bases bilaterally. His abdomen is soft and non-tender with no ascites noticed. You check for sacral edema, but find none. Moving down to his legs, when you remove his slippers and socks you notice the indentation they leave. You check his feet and legs for edema and note he has pitting edema to about 5 inches above his ankles. There is no redness or drainage noted. When he is placed on the monitor you note atrial fib with occasional unifocal PVCs. Repeat vitals are 188/108 HR 118 RR 32 and pulse ox is 92% on the O2. Your partner has started a line for you and is running NS at TKO. You do a 12 lead that shows no ischemic changes.
So now that you have a good history and physical completed it is time to put it all together and come up with a field impression and game plan. I have attached a table that should help. Let's look at possible etiologies:

**Aspiration:** He has no history of a choking spell, altered mentation, swallowing difficulties or a PEG tube, so it's safe to rule that out.

**Anaphylaxis:** Lungs sounds would be wheezing or silent, you would notice some angioedema or urticaria. You do not get JVD with anaphylaxis and we would be hemodynamically unstable. So we can rule this out.

**Pneumothorax:** His BP would not be elevated and there would be decreased lung sounds on one side. Ruled out

**Pulmonary emboli:** Lung sounds would be clear. There would be no JVD and we would expect his BP to be down. Ruled out

**Pneumonia:** Afibrile, no chills or sweats. No productive cough with green or yellowish sputum. So probably not.

So now we are left with COPD, heart failure or MI.

You strongly consider COPD by his pursed lips breathing, tripod position and wheezes heard. You also remember that he had crackles in addition to the wheezes that are not usually present in COPD. Plus he has JVD plus pitting edema. You rule out COPD.

Taking all this into account you decide that heart failure is the correct field impression. It is important to rule out that an acute cardiac event may have triggered pulmonary edema. Although he was tachycardic, the rate was not significant enough to be worrisome. He also showed no ischemic changes on his 12 Lead and denied chest pain or discomfort. If he had an MI severe enough to put him in pulmonary edema he would be hemodynamically unstable.

So now that we have our field impression We know that our goal is to improve ventilation and oxygenation. We also need to look at decreasing his preload and after load thereby decreasing his cardiac oxygen demand. So let's take a look at what heart failure is.

Heart failure is a complex clinical syndrome that can result from any structural as well as functional cardiac disorders that impair the ability of the ventricles to fill with and eject blood. This month's topic will look at some of the causes, symptoms and our treatment for patients with HF otherwise known as Congestive Heart Failure.

**Definition**

Heart failure, also known as congestive heart failure (CHF), means the heart can't pump enough blood to meet the body's needs. Over time, conditions such as narrowed arteries in the heart (coronary artery disease) or high blood pressure gradually leave the heart too weak or stiff to fill and pump efficiently.

It is very difficult to reverse many conditions that lead to heart failure, but heart failure can often be treated with good results. Medications can improve the signs and symptoms of heart failure. Lifestyle changes, such as exercising, reducing the salt in diets, managing stress, treating depression, and especially losing excess weight, can improve the quality of life.

The best way to prevent heart failure is to control risk factors and conditions that cause heart failure, such as coronary artery disease, high blood pressure, high cholesterol, diabetes or obesity.
Symptoms
Heart failure can be chronic — meaning the condition is ongoing — or acute, meaning the condition has started suddenly.

**Chronic heart failure symptoms**

- Shortness of breath (dyspnea) when the patients exert themselves or lie down
- Fatigue and weakness
- Rapid or irregular heart beat
- Swelling (edema) in their legs, ankles and feet
- Reduced ability to exercise
- Swelling of the abdomen (ascites)
- Sudden weight gain from fluid retention
- Persistent cough with blood tinged phlegm present
- Difficulty cooincentrating or decreased alertness

**Acute heart failure symptoms**

- Symptoms similar to those of chronic heart failure, but more severe and start or worsen suddenly
- Sudden fluid buildup
- Rapid or irregular heartbeat (palpitations)
- Sudden, severe shortness of breath and coughing up pink, foamy mucus
- Chest pain, if the heart failure is caused by a heart attack

![Normal Chest Film](image1)

![Chest Film showing Pulmonary Edema](image2)
If the patient has a diagnosis of heart failure, and if any of the symptoms suddenly become worse or they develop a new sign or symptom, it may mean that existing heart failure is getting worse or not responding to treatment.

**Causes**

Heart failure often develops after other conditions have damaged or weakened your heart. Over time, the heart can no longer keep up with the normal demands placed on it to pump blood to the rest of your body. The main pumping chambers of your heart (the ventricles) may become stiff and not fill properly between beats. Also, the heart muscle may weaken, and the ventricles stretch (dilate) to the point that the heart can't pump blood efficiently throughout your body. The term "congestive heart failure" comes from blood backing up into — or congesting — the liver, abdomen, lower extremities and lungs.

Heart failure can involve the left side, right side or both sides of your heart. Typically, heart failure begins with the left side — specifically the left ventricle, the heart's main pumping chamber.

**Type of heart failure**

<table>
<thead>
<tr>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left-sided heart failure</strong></td>
</tr>
<tr>
<td>Most common form of heart failure.</td>
</tr>
<tr>
<td>Fluid may back up in your lungs, causing shortness of breath.</td>
</tr>
<tr>
<td><strong>Right-sided heart failure</strong></td>
</tr>
<tr>
<td>Often occurs with left-sided heart failure.</td>
</tr>
<tr>
<td>Fluid may back up into your abdomen, legs and feet, causing swelling.</td>
</tr>
<tr>
<td><strong>Systolic heart failure</strong></td>
</tr>
<tr>
<td>The left ventricle can't contract vigorously, indicating a pumping problem.</td>
</tr>
<tr>
<td><strong>Diastolic heart failure</strong></td>
</tr>
<tr>
<td>The left ventricle can't relax or fill fully, indicating a filling problem.</td>
</tr>
</tbody>
</table>

Any of the following conditions can damage or weaken your heart and can cause heart failure. Some of these can be present without your knowing it:
Coronary artery disease and heart attack.
Coronary artery disease is the most common form of heart disease and the most common cause of heart failure. Over time, arteries that supply blood to the heart muscle narrow from a buildup of fatty deposits, a process called atherosclerosis. Blood moves slowly through narrowed arteries, leaving some areas of your heart muscle weak and chronically deprived of oxygen-rich blood. In some cases, the blood flow to the muscle is just enough to keep the muscle alive but not functioning well. A heart attack occurs if plaques formed by the fatty deposits in your arteries rupture. This causes a blood clot to block blood flow to an area of the heart muscle, weakening the heart’s pumping ability.

High blood pressure (hypertension). Blood pressure is the force of blood pumped by your heart through the arteries. If the blood pressure is high, the heart has to work harder than it should to circulate blood throughout the body. Over time, the heart muscle may become thicker to compensate for the extra work it must perform, enlarging the heart. Eventually, the heart muscle may become either too stiff or too weak to effectively pump blood.

Faulty heart valves. The valves of the heart keep blood flowing in the proper direction through the heart. A damaged valve, due to a heart defect, coronary artery disease or heart infection, forces the heart to work harder to keep blood flowing as it should. Over time, this extra work can weaken the heart. Faulty heart valves, however, can be fixed if found in time.

Damage to the heart muscle (cardiomyopathy). Some of the many causes of heart muscle damage, also called cardiomyopathy, include infections, alcohol abuse, and the toxic effect of drugs such as cocaine or some drugs used for chemotherapy. In addition, whole-body diseases, such as lupus, or thyroid problems can damage heart muscle.

Myocarditis. Myocarditis is an inflammation of the heart muscle. It's most commonly caused by a virus and can lead to left-sided heart failure.

Heart defects that you may be born with (congenital heart defects). If the heart and its chambers or valves haven’t formed correctly, the healthy parts of the heart have to work harder to pump blood through the heart, which in turn may lead to heart failure.

Abnormal heart rhythms (heart arrhythmias). Abnormal heart rhythms may cause the heart to beat too fast. This creates extra work for the heart. Over time, the heart may weaken, leading to heart failure. A slow heartbeat may prevent the heart from getting enough blood out to the body and may also lead to heart failure.

Other diseases. Chronic diseases such as diabetes, severe anemia, hyperthyroidism, hypothyroidism, emphysema, lupus, hemochromatosis and buildup of proteins in your muscles (amyloidosis) also may contribute to heart failure. Causes of acute heart failure include viruses that attack the heart muscle, severe infections, allergic reactions, blood clots in the lungs, the use of certain medications or any illness that affects the whole body.
Risk factors
A single risk factor may be enough to cause heart failure, but a combination of factors increases the risk. Risk factors include:

**High blood pressure.** The heart works harder than it has to if the blood pressure is high.

**Coronary artery disease.** Narrowed arteries may limit the heart’s supply of oxygen-rich blood, resulting in weakened heart muscle.

**Heart attack.** Damage to the heart muscle from a heart attack may mean the heart can no longer pump as well as it should.

**Irregular heartbeats.** These abnormal rhythms can create extra work for the heart, weakening the heart muscle.

**Diabetes.** Having diabetes increases your risk of high blood pressure and coronary artery disease.

**Some diabetes medications.** The diabetes drugs rosiglitazone (Avandia) and pioglitazone (Actos) have been found to increase the risk of heart failure.

**Sleep apnea.** The inability to breathe properly at night results in low blood oxygen levels and increased risk of abnormal heart rhythms. Both of these problems can weaken the heart.

**Congenital heart defects.** Some people who develop heart failure were born with structural heart defects.

**Viruses.** A viral infection may have damaged the heart muscle.

**Alcohol use.** Drinking too much alcohol can weaken heart muscle and lead to heart failure.

**Kidney conditions.** These can contribute to heart failure because many can lead to high blood pressure and fluid retention.

System Protocol
Our protocol for treating patients with Heart Failure/Pulmonary Edema is based on good assessment skills. Does the patient have any signs of hypoperfusion and cardiorespiratory compromise. We need to differentiate heart failure from COPD/asthma by looking at past medical history, meds, signs and symptoms, and capnography.

Consider the cause and look at the heart rate, rhythm, volume or pump problem. Treat per appropriate SOP based on the etiology. Listen to the patient’s breath sounds in all lobes, front and back and report timing and location of wheezes/crackles.
If the patient is in mild to moderate cardio respiratory compromise, we need to position the patient in an upright 90 degree sitting position and dangle their legs over the sides of the stretcher. Assess the airway and apply CPAP immediately. Start with 5-10cm PEEP. If SBP falls <90 (MAP <65) titrate PEEP down to 5cm; remove if hypotension persistsacreate the FiO2 to 95% and PEEP at 10cm to achieve SpO2 >95%. If the patient’s SBP falls < 90 you should remove the CPAP. If resp distress and CPAP contraindicated, not tolerated, or unavailable. Assess need for advanced airway and DAI.

The patient should receive Aspirin 324mg by mouth per the Acute Coronary Syndrome protocol and then Nitroglycerine 0.4mg sub lingual as long as the SBP remains >90. You can repeat the NTG 0.4mg every 3-5 minutes with no dose limit. For severe anxiety you may give Midazolam in 2mg increments every 30-60 seconds IVP up to 10mg. You may repeat to 10mg if SBP >90.

If pt has signs of cardiogenic shock due to pump failure, dysrhythmias (tachy/brady), this is a time sensitive patient and needs to be aggressively treated. Assess the need for DAI to decrease the work of breathing and to protect the airway or ventilate the patient. Assess for signs of hypovolemia/dehydration.

If the patient is hypovolemic and or dehydrated and certain that lungs are clear and respirations are not labored you may give IV NS fluid challenges in 200 ml increments until SBP is maintained above 90. Frequently assess breath sounds while treating the patient. Dopamine 400mg/250 of NS or D5W IVPB may be started at 5mcg/kg/min. Titrate up to 20mcg/kg/min until a SBP is maintained above >90 (MAP >65). If possible ACS patient you may give ASA 324mg by mouth per the ACS protocol unless contraindicated.

Ref:
http://www.mayoclinic.com/health/heart-failure/DS00061
McHenry Western Lake County EMS System Protocol
www.UpToDate.com
HEART FAILURE / PULMONARY EDEMA

- Assess for hypoperfusion and cardiorespiratory compromise. **12 Lead ECG obtained and transmitted**
- Differentiate HF from COPD/asthma by PMH, meds, S & S, capnography if available (See appendix p. 106).
- **Consider cause:** rate, rhythm, volume, or pump problem; treat per appropriate SOP based on etiology.
- Auscultate lung sounds all lobes, front & back; report timing/location of wheezes/crackles

Low Acuity to EMERGENT: Mild to Moderate cardiorespiratory/perfusion compromise
Alert, normotensive or hypertensive (SBP ≥ 90 and DBP ≥ 60) (MAP ≥ 65)

1. **IMC special considerations:**
   - Position patient sitting upright at 90˚ (if tolerated); dangle legs over sides of stretcher
   - **C-PAP:** 5-10 cm PEEP; If SBP falls < 90 (MAP < 65): Titrate PEEP down to 5 cm; remove if hypotension persists
   - If respiratory distress and CPAP contraindicated, not tolerated, or unavailable:
     - Assess need for **advanced airway** O₂ 15 L/NRM

2. **ASPIRIN 324 mg** (4 tabs 81 mg) PO per ACS SOP unless contraindicated

3. **NITROGLYCERIN 0.4 mg SL** If SBP remains ≥ 90 (MAP ≥ 65): Repeat NTG 0.4 mg every 3-5 min – no dose limit
   - NTG may be given if HR > 100 in pulmonary edema

4. Severe anxiety and SBP ≥ 90 (MAP ≥ 65): **MIDAZOLAM** standard dosing per ACS SOP

CARDIOGENIC SHOCK (CRITICAL): Pump failure due to AMI, dysrhythmia; HF; obstructive shock (tension pneumothorax, cardiac tamponade, pulmonary embolus); or drugs with SBP < 90; MAP < 65; & S&S hypoperfusion

1. **IMC special considerations:**
   - Assess need for advanced airway to ↓ work of breathing, protect airway, or ventilate patient
   - Assess for hypovolemia/dehydration

2. If hypovolemic and/or dehydrated - **lungs clear and ventilations unlabored:**
   - **NS IVF in 200 mL increments up to 1 L**; attempt to achieve SBP ≥ 90 (MAP ≥ 65). Frequently reassess lung sounds.

3. **DOPAMINE IVPB:** 5 mcg/kg/min; may titrate up to 20 mcg/kg/min to maintain SBP ≥ 90 (MAP ≥65)

4. If possible ACS: (alert with gag reflex): **ASPIRIN 324 mg** (4 tabs 81 mg) PO per ACS SOP

**Sampling of drugs prescribed for patients with CV disease/Heart Failure**

**ACE Inhibitors** (ACEI): Benza*april* (Lotensin), captopril (Capoten), enalapril (Vasotec), fosinopril, monopril, lisinopril (Prinivil/Zestril), moesipril (Univasc), perindopril (Aceon), quinapril, accupril, Ramipril (Altace),trandolapril (Mavik)

**Angiotensin Receptor Blockers** (ARB): candesartan (Atacand), eprosartan (Teveten), irbesartan (Avapro), losartan (Cozaar), olmesartan (Benicar), telmisartan (Mircardis), valsartan ( Diovan)

**Anticoagulants:** apixaban (Eliquis), aspirin, argatroban, bivalirudin (Angiomax), clopidogrel (Plavix), dabigatran (Pradaxa), endoxaban (Savaysa), epifibatide (Integrisin), lepirudin (Refudian), presugrel (Effient), rivaroxaban (Xarelto), ticagrelor (Brilinta), ticlodipine (Ticlid), warfarin (Coumadin, Jantoven); Sub-q route: dalteparin (Fragmin), enoxaparin (Lovenox), fondaparinux (Arixtra), tinzaparin (Innohep); Heparin (IV & sub-q)

**Beta Blockers:** acebutolol ( Sectral), atenolol (Tenormin), betaxolol (Betopic,Kerlone), bisoprolol (Zebeta), carvedilol (Coreg), esmolol (Brevibloc), labetalol (Normodyne, Trandate), levobunolol (Betagan), metoprolol (Lopressor/Toprol), nadolol (Corgard), pembutolol, pindolol (Visken), propranolol (Inderal), timolol (Blocadren, Timoptic), sotalol (Betapace)

**Calcium channel blockers:** amlodipine (Norvasc), felodipine, diltiazem (Cardizem), nicardipene (Cardene), nifedipine (Procardia, Adalat), verapamili (Calan, Isotin)

**Diuretics:** amiloride (Midamor),bumetanide (Bumex),chlorothiazide (Diuril), Diazide, furosemide (Lasix), hydrochlorothiazide (Hydrodiuril), indapamide (Lozol), metolazone (Zaroxolyn), Polythiazide, spironolactone (Aldactone), torsemide, triamterene (Dyrenium)

**Digoxin** (Lanoxin)

**Vasodilators:** hydralazine (Apresoline), isosorbide (Isordil), minoxidil (Loniten), nesiride (Natrecor), Nitrates/NTG
<table>
<thead>
<tr>
<th>Disease</th>
<th>Onset</th>
<th>Cough</th>
<th>Lungs</th>
<th>Exam</th>
<th>History</th>
<th>JVD</th>
<th>BP</th>
<th>HR</th>
<th>RR</th>
<th>SaO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspiration</td>
<td>Sudden</td>
<td>Possibly</td>
<td>Unilateral or bilateral crackles or wheezing, most common R.L.L.</td>
<td>Hypoxia</td>
<td>Choking, altered mentation, swallowing diff, Feeding tube</td>
<td>No</td>
<td>Unchanged</td>
<td>Up</td>
<td>Up</td>
<td>Down</td>
</tr>
<tr>
<td>Anaphylaxis</td>
<td>Sudden</td>
<td>No</td>
<td>Bilateral wheezing or silent</td>
<td>Angioedema Urticaria</td>
<td>Exposure to an allergen</td>
<td>No</td>
<td>Down !</td>
<td>Up !</td>
<td>Up</td>
<td>Down</td>
</tr>
<tr>
<td>Asthma</td>
<td>Sudden or gradual</td>
<td>Possibly non-productive</td>
<td>Bilateral wheezing or silent</td>
<td>Retractions, Pursed lips, Tripod position</td>
<td>Exposure to trigger</td>
<td>Late</td>
<td>Unchanged or up</td>
<td>Up</td>
<td>UP</td>
<td>Down</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Sudden</td>
<td>Yes</td>
<td>Unilateral absence or diminished</td>
<td>Possible injury</td>
<td>Trauma Coughing</td>
<td>Late</td>
<td>Unchanged or up</td>
<td>Up</td>
<td>Up</td>
<td>Down</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Sudden</td>
<td>No</td>
<td>Clear</td>
<td>Shock/hypoxia</td>
<td>Surgery, sedentary, long distance travel, long bone fracture</td>
<td>No</td>
<td>Down !</td>
<td>Up !</td>
<td>Up</td>
<td>Down !</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Gradual</td>
<td>Productive green/yellow</td>
<td>Unilateral or bilateral course crackles or wheezing</td>
<td>Fever, chills</td>
<td>Illness, prolonged shallow breathing, inability to cough, aspiration</td>
<td>Possibly</td>
<td>Unchanged</td>
<td>Up</td>
<td>Up</td>
<td>Down</td>
</tr>
<tr>
<td>COPD</td>
<td>Gradual</td>
<td>Possibly</td>
<td>Bilateral wheezing</td>
<td>Retractions, tripod position, pursed lips</td>
<td>Smoking</td>
<td>Late</td>
<td>Unchanged or up</td>
<td>Up</td>
<td>Up</td>
<td>Down</td>
</tr>
<tr>
<td>Myocardial Infarction</td>
<td>Sudden or gradual</td>
<td>No</td>
<td>Clear</td>
<td>Pale, diaphoretic</td>
<td>Chest, abd, back discomfort</td>
<td>If BP is up</td>
<td>Up or down</td>
<td>Up or down</td>
<td>Up or down</td>
<td></td>
</tr>
<tr>
<td>Heart Failure</td>
<td>Sudden or gradual</td>
<td>Possibly dry or frothy</td>
<td>Bilateral rales or wheezing</td>
<td>Dependent edema Ascites</td>
<td>Dyspnea on exertion, orthopnia, previous AMI</td>
<td>If BP is up</td>
<td>Up or down</td>
<td>Up</td>
<td>Up</td>
<td>Down</td>
</tr>
</tbody>
</table>
1) Medications can improve the signs and symptoms of heart failure, but these lifestyle changes will help as well.

2) Swelling in the legs, ankles and feet is called ___________________.

3) List 4 symptoms of Acute Heart Failure
   1) 
   2) 
   3) 
   4) 

4) The ventricles of the heart will stretch or dilate and cause this.

5) The term congestive heart failure comes from.

6) Describe the following heart failures:
   Left sided heart failure:
   Right sided heart failure:
   Systolic heart failure:
   Diastolic heart failure:
7) Cardiomyopathy can be caused by:

8) Myocarditis is:

9) Name 6 risk factors that can cause heart failure:
   1)
   2)
   3)
   4)
   5)
   6)

10) The first two medications given by system protocol for a patient in congestive heart failure are?

11) What medication in our protocol for CHF can be given for severe anxiety?

12) A patient in CHF that has been placed on CPAP, must maintain a SBP > than ____________, and we would like to get SpO2 > than ________________.

For the following patients write your field impression, include any additional information you would want to obtain to support your decision.

13) A 34 year old women with a sudden onset of trouble breathing. She is awake and alert, only able to get one or two words out at time. Lung sounds bilateral wheezing. She is sitting in a tripod position. No swelling or rashes noted. She has a history of allergies to shrimp, asthma and hypertension. She uses and albuterol inhaler when needed. BP 124/60, HR 112, RR 26 SaO2 90 on room air.
14) An 84 year old patient with dementia who is acutely dyspneic. You note no JVD or pedal edema and he is occasionally coughing. Lung sounds crackles and wheezing in the RLL. Vital signs BP 134/80 HR 108 RR 32. SaO2 88% on room air. He also feels warm to touch.

15) A 54 year old gentleman with acute sudden onset of respiratory distress. He has a productive cough of frothy sputum. Lung sounds are bilateral wheezes and rales. You note dependent edema and JVD are present. His color is pale, skin is warm and dry. Vital signs BP 188/110 HR 106 RR 28 SaO2 90% on room air.

If you are NOT a member of the McHenry Western Lake County EMS System, Please include your address on each optional quiz turned into our office. Our mailing address is: Northwestern Medicine – McHenry Hospital EMS, 4201 Medical Center Drive, McHenry, Illinois 60050. We will forward to your home address verification of your continuing education hours.

If you ARE a member of our EMS System, your credit will be added to your Image Trend record. Please refer to Image Trend to see your current list of continuing education credits. Any questions regarding this can be addressed to Cindy Tabert at 224-654-0160. Please fax your quiz to Cindy Tabert at 224-654-0165.