Heart Failure

Objectives

At the end of this lesson, the student should be able to:

1. Review cardiac anatomy and physiology with a focus on heart failure.
2. Discuss the relationship between fluid retention, fluid shifting, and the signs and symptoms seen in the heart failure patient.
3. Review patient history and physical assessment for heart failure patients.
4. Discuss prehospital treatment options for acute decompensating heart failure.

Case Study

It is a warm, sunny afternoon in September when you and your paramedic partner are dispatched to a nearby residence for a male who called 911 complaining of shortness of breath. When you arrive at the one story house on Main Street a neighbor ushers you in the front door where you find an obese man who appears to be in his 70s sitting tripod on a chair in his kitchen. His entire upper body heaves with each breath and he appears tired, barely looking at you when you enter his house. He is oriented and is able to tell you his name, the date including the day of the week, and he is able to provide what appears to be an accurate medical history.

Speaking in only four or five word sentences he manages to tell you he has a long history of diabetes and hypertension, but hasn't been to the doctor in at least a year and takes his medication only sporadically. He used to visit the cardiologist and states that during his last evaluation over a year ago he was told his ejection fraction was around 30%. He does not check his blood glucose levels on a regular basis but states he can normally tell when his sugars are too high or too low because he starts to feel bad.

On the counter you note a few insulin syringes, a sharps container, and a few prescription bottles including losartan, furosemide, Colace, and aspirin. You see a home continuous positive airway pressure (CPAP) machine sitting on the table along with a variety of empty wrappers and containers from nearby fast food restaurants. The patient tells you he has sleep apnea and uses the machine at night. He states he has been feeling more short of breath and tired over the last few days but noticed that he has been breathing better at night with the machine on so he had started using it periodically during the day. He denies having any chest pain or chest discomfort. The patient readily admits he hasn't been following a low salt diet and says this is because he doesn't really cook. He is a widower and tends to eat only fast food since his wife passed away about eight months ago. He weighs himself on a daily basis and points to his log secured by a magnet to the fridge; the log shows he has gained about 13 lbs (almost 6 kg) over the past three weeks His current weight is recorded as 208 lbs. The handwritten information scrawled on the top of the page lists his height as 5'7".

The patient's skin is pale, he is diaphoretic, and his extremities are slightly cool to the touch. Pupils are equal, round, and reactive to light. His lips and nail beds are cyanotic. On auscultation you hear coarse bilateral rales in both the right and left pulmonary bases anteriorly and posteriorly. Air entry is clear but diminished in both apexes. The patient abdomen, which heaves with each breath, is rounded and protruding with noticeable stretch marks throughout the right and left lower quadrants. Radial pulses feel regular, strong and equal when palpated bilaterally. The skin on both lower extremities appears taunt and shiny with an absence of hair growth below the knee. You note bilateral pretibial edema and 3+ pitting pedal edema which the patient states has been normal for him over the last week or more. Bot dorsalis pedis pulses feel weak and thready when you are able to locate them. The edema makes them difficult to palpate and your fingers leave pits in the skin when removed. The patient's heart rate is 132, respiratory rate is 28, SpO2 84%, temperature 97.0 F, blood pressure 206/138 mmHg and serum glucose is 268 mg/dL. You obtain an ECG strip which shows sinus tachycardia with an abnormally wide Q wave and no ST elevation in Lead 2.

Statistics

According to the Centers for Disease Control and Prevention (2012), around 5.7 million Americans are diagnosed with heart failure.[1] The disease is a primary cause of over 55,000 deaths every year and listed as a contributing factor in over 250,000.[1] The incidence of heart failure has been on the rise for many years and this trend shows no evidence o slowing down.[2] Heart failure is a disease of the elderly; about 80% of people hospitalized with heart failure are over the age of 65.[2] The significant improvements in the treatment of myocardial infarct (MI) have increased survival rate this means people who have myocardial damage are living longer with damaged and weakened hearts. The American College of Cardiology (ACC), American Heart Association (AHA) and Heart Failure Society of America all considered improved MI treatment to be one of the main reasons for the increase in people living with heart failure.[2,3]

Etiology

Anatomy of the Heart

The heart is the body's pump moving oxygen, nutrients and waste products through the system. It is positioned in th thoracic cavity with the most superior edge (called the base) around the level of the second intercostal space and the inferior tip (called the apex) along the left midclavicular line around the 4th or 5th intercostal space. It is separated into four chambers: the right atrium, right ventricle, left atrium, and left ventricle. Four different, strategically located valves keep the blood moving in the correct direction and prevent back flow. Atrioventricular valves (also called AV valves or cuspid valves) are located between the atria and the ventricles; semilunar valves are located between the ventricles and the major arteries associated with that ventricle.

The circulatory system can be divided into two separate branches; the pulmonary circulation and the systemic circulation. Likewise, the heart can be divided into right and left sides based on the circulatory system it supplies with blood. The right side of the heart receives deoxygenated blood from the body and pumps it into the lungs where it picks up oxygen. The left side of the heart receives oxygenated blood from the lungs and pumps it to the body. Specif movement, including movement through valves, is as follows. Deoxygenated blood enters the right atrium from the superior and inferior vena cava, moves through the tricuspid valve into the right ventricle, and is then pumped throug the pulmonary artery into the pulmonary circulation. In the pulmonary circulatory system it picks up oxygen and ther enters the left atria through the pulmonary vein, moves through the bicuspid (or mitral valve) into the left ventricle and is then pushed into the aorta. The aorta is the largest artery in the body and all blood leaving the heart travels through it.

The heart wall consists of three layers - from superficial to deep these are the epicardium, myocardium and endocardium. The heart's job is to pump blood to all of the cells in the body so the majority of the heart wall is the thick, muscular myocardium. The myocardium is much thicker in the ventricles than it is in the two atria. The left ventricle must pump blood throughout the body and has to overcome any pressure in the systemic circulation. Due to this increased work load, the myocardium is thickest in the left ventricle in a healthy heart.

Frank-Starling's Law of the Heart

Besides the thickness of the myocardial wall, there are a number of other factors which determine the strength of the ventricular contraction including serum calcium levels and the oxygen/nutrient supply. Frank-Starling's Law, also calle Starling's Law, addresses how muscle fibers stretch and preload affect contraction strength. Blood dumping into the ventricles causes these chambers to fill, the wall stretches to accommodate the fluid, and muscle fibers stretch accordingly. The more the muscle fibers stretch the stronger the resultant contraction. The ventricles fill with blood during diastole when the heart is at rest, just prior to contraction or systole, and is referred to as the preload or the end-diastolic volume (EDV). The direct relationship between preload volumes and the force of contraction is the essence of the law. Increases in preload result in increased contraction for a period of time, but more is not always better and if preload continues to increase the heart becomes overwhelmed and contraction strength decreases.

A common analogy used to describe this mechanism and how it fails in heart failure involves an elastic or rubber banc Picture putting one end of the rubber band on your thumb and pulling back on the other end like you were aiming it at someone; the further you want the rubber band to travel the further you pull back on it and stretch it out. However, if you pull the rubber band too far or stretch it too much too often it will break or not bounce back to its regular size. In heart failure an increased preload strains damaged muscles and does not result in a stronger or more forceful contraction.

Shock, a life threatening medical condition, occurs when there is an insufficient amount of oxygen and nutrients reaching the body's cells; this can be due to pump failure, hypovolemia or massive vasodilation like that seen in an anaphylactic reaction or sepsis. In heart failure, the heart is not pumping enough blood to meets the body's metabolic demands. This type of shock is called cardiogenic shock.

Cardiac Output and Stroke Volume

Cardiac output (CO) is the volume of blood the heart ejects in one minute. It is determined by the amount of blood ejected in one contraction, stroke volume (SV) and the number of contractions per minute (HR). This is represented b the equation CO=SV X HR. In a normal, healthy heart the stroke volume should be the same for both the right and le ventricles. When cardiac output falls the body will try to compensate by manipulating this equation. For example, if stroke volume is decreased due to a decrease in fluid volume (hemorrhage) or impaired contractions, the heart rate increases to try and maintain the same CO.

Many different factors can affect stroke volume. These include the strength of the contraction, the amount of blood returning to the heart, and the amount of resistance the heart must pump against. The left ventricle is forcing blood into the aorta and must overcome the pressure in this system with each contraction. The amount of force the ventricl

must overcome is called afterload.

What is Heart Failure?

The AHA defines heart failure as "any structural or functional impairment of ventricular filling or ejection of blood" (reduced stroke volume) and divides heart failure into two categories based on a patient's ejection fraction (EF).[4] The two divisions used by the AHA when discussing heart failure are preserved ejection fraction or reduced ejection fraction.[4] According to the AHA it is important for EF to be determined in heart failure patients because it plays a role in determining treatment plans, patient prognosis and clinical trials.[4] On account of this significance it may not be unusual for a patient to offer you his or her most recent EF number; therefore, knowledge of what constitutes "norma may assist in your patient assessment. An in-depth discussion about ejection fraction is beyond the scope of this review.

Ejection fraction is the percentage of blood pumped by the heart in one beat. The heart chambers are never completely empty which means the heart never pumps 100% of the blood in the chambers. It only pumps or ejects a portion of the blood. This portion that is ejected is the ejection fraction. According to the AHA, a normal EF is between 55% and 70%.[5] A patient that reports a reduced EF is at significantly greater risk of hospitalization or death than a heart failure patient with a normal EF.[6]

Heart failure may be classified a few different ways. It may be discussed as being either left-sided heart failure or right-sided heart failure and refers to the ventricle that has impaired contraction or impaired filling. It can also be classified as systolic or diastolic dysfunction, terminology that refers to the ejection fraction (EF). Systolic dysfunction refers to a reduced EF and reduced stroke volume while in diastolic dysfunction the EF is normal but the stroke volum is decreased.

Possible Causes of Heart Failure

A number of medical conditions can lead to this impairment and many affect both structure and function of the heart. Myocardial infarct causes scarred heart muscle which is stiff and does not contract. Valvular heart disease causes hardened valves that allow for regurgitation or the restriction of blood flow and cause the heart to work harder. Cardiomyopathy causes damage to the heart's myocardium affecting its ability to contract and/or fill. Dilated cardiomyopathy, a condition found in 30% to 40% of heart failure patients who are enrolled in randomized control trials, decreases the heart's ability to contract due to a weak ventricular wall and enlargement of the ventricular chamber.4 Chronic hypertension can cause hypertrophy of the left ventricle; the ventricle has to work harder to push blood against the increased afterload, and eventually, the myocardial muscle wall enlarges to accommodate the increased workload. The muscle wall gets bigger at the expense of the ventricular chamber which gets smaller; there is less blood flowing into the chamber so less blood is ejected out on contraction (decreased stroke volume). Also, the thickened myocardium becomes more prone to ischemia. This is because the coronary arteries run along the surface the heart over the epicardium and when the myocardium enlarges, the nutrients and oxygen have further to travel through the thickened muscle to reach the inner most layers.

My Own Worst Enemy

The body has a number of feedback mechanisms that spiral into themselves and actually make the heart failure patient's condition worse and not better. During heart failure the ineffective contractions or filling of the heart causes a decrease in stroke volume. The patient's heart rate will increase in an effort to compensate for the decreased amount of blood being ejected with each contraction. Like all of the body's muscles the myocardium also needs adequate bloo flow and an adequate oxygen supply to function properly. It receives this blood flow when the heart is relaxing during diastole and when heart rate increases, diastolic time decreases. The decreased time in diastole results in decreased blood flow and decreased oxygen supply to the cardiac muscles, but the increased heart rate means the heart is working harder and its oxygen demand is increasing. This mismatch in oxygen supply and oxygen demand causes more myocardial ischemia, which further decreases the heart's ability to pump blood.

The Kidneys

This is not the only way our systems work against each other during heart failure. It is important to remember that th decrease in cardiac output is because the pump isn't working correctly and not due to a decrease in fluid volume. Actually, the opposite happens and fluid volume is increased because the kidneys start retaining water. The kidneys ar bean shaped organs located in the retroperitoneal space around the level of the 12th thoracic vertebrae or 1st lumbar vertebrae. The large, solid liver in the right upper abdominal quadrant causes the right kidney to sit slightly lower tha the left kidney. The kidneys are part of the urinary system and play a vital role in regulating fluid volume, controlling blood solute concentrations (like sodium) and filtering waste products which the blood eliminates in the form of urine. To function properly the kidneys require a constant blood flow and consistent blood pressure so they contain millions o

specialized cells that monitor blood pressure. When these cells sense a decrease in blood flow they secrete a substanc called renin which triggers a widespread, systemic response called the renin-angiotensin-aldosterone system or the RAAS. This response includes the secretion of antidiuretic hormone from the pituitary gland in the brain which also promotes fluid retention. The end result is vessel constriction, an increased thirst sensation, increased sodium absorption (water follows salt), and increased water absorption all in an effort to increase blood volume. Preload to th heart increases, end diastolic volume increases, afterload increases, and collectively, these actions increase the work load on the already failing heart. The kidneys only know they are not getting enough blood so they increase the volume; they don't know the volume is fine but the pump is defective. This turns into a vicious cycle with the kidneys attempting to increase cardiac output in response to decreased renal blood flow, and putting more strain on the heart in the process.

In the initial stages of heart failure the excess fluid load on the heart causes secretion of substances called natriuretic peptides that encourage vasodilation and diuresis, which is increased urine formation, in an effort to decrease fluid volume. In this way the cardiac system attempts to decrease cardiac workload by lowering intravascular volume and causing vasodilation while the RAAS attempts to increase volume and cause vasoconstriction; these systems compete against each other. As heart failure progresses the vasodilation and diuresis compensatory mechanisms decline. The RAAS becomes the major player and vasoconstriction with fluid retention ensues.

Congestion and Heart Failure

Although the term congestive heart failure is still widely used, the term heart failure is preferred by the AHA and ACC because congestion implies fluid retention, a symptom that may not be present in all heart failure patients.[3] In the prehospital setting, many paramedics associate heart failure patients with fluid retention because this is the emergent patient presentation; rales and hypoperfusion indicate a more acute onset of heart failure.[2] Also, the main reason for hospitalization in heart failure patients is dyspnea due to congestion which is also indicative of poor patient outcomes patients hospitalized with congestion have higher mortality rates post discharge than heart failure patients without congestion.[7] An exacerbation of heart failure symptoms requiring urgent care may be referred to as acute heart failur or acute decompensated heart failure (ACDH).[3,7]

If a patient has acute decompensated right sided heart failure, the right ventricle is not pumping blood effectively. Thi fluid backs up into the right atria and into areas that normally empty into the right atria. This means the fluid overloa will manifest itself in the jugular veins, abdominal area, and peripheral extremities. In acute decompensated left side heart failure the left ventricle is not pumping blood effectively which causes the fluid backup into the left atrium and, subsequently, the lungs. In their 2013 practice guidelines, the AHA lists dyspnea, fatigue, and fluid retention as the three cardinal manifestations of heart failure.[4]

Physical Assessment

During patient assessment a regular, sequential pattern that is followed every time helps prioritize findings, prevents the medical professional from missing important information, and keeps the assessment on track. In the field this pattern usually runs head to toe and starts with level of consciousness, airway, breathing, and circulation. The heart failure patient is no exception. Depending on the severity of the exacerbation, this patient may present with severe dyspnea, anxiety and an altered level of consciousness. Hypoxia can also cause combativeness and irrational behavio by the patient. A mismatch between myocardial oxygen demand and myocardial oxygen supply can lead to acute myocardial infarct, and the patient may be complaining of chest pain. The regular assessment should be performed, life threats addressed immediately, vital signs monitored, and an ECG obtained. The basic primary and secondary assessments are not expounded on here; instead, assessment techniques and findings that are more specific to heart failure patients are covered in greater detail.

Edema

The failing heart cannot pump blood effectively and this leads to a fluid backup in the vessels. The vessels are unable accommodate the excess amount of blood accumulating in the system and fluid starts to leak out into the surrounding tissues. The excess fluid being pushed out of the vasculature causes swelling and will be identifiable on physical assessment in specific locations depending on gravity and patient positioning. Just like a plastic bottle half filled with water, fluid collects in the most dependent areas. For patients sitting upright or standing fluid will collect in the lower extremities; swelling in the lower legs is called pretibial edema, and in the feet, it is pedal edema. It may also cause ascites which is fluid buildup in the abdominal (peritoneal) cavity. A large amount of fluid can gather in the peritoneal space and cause the abdomen to appear distended, and an excess of peritoneal fluid can cause or exacerbate shortne of breath because it mechanically impedes the movements of the diaphragm. Patients who are bed ridden or supine may have sacral edema.

Mild ascites is typically not noticeable on physical assessment, and sacral edema is not commonly assessed by EMT's; however, pretibial and pedal edema are easily evaluated and monitored in the prehospital field. In order to assess

edema in the lower legs and feet press the tips of your fingers firmly into both lower extremities for about five seconds. When you remove your fingers assess the skin for pits or dimples in the locations where pressure had been applied. If present, this is pitting edema and is caused when the pressure displaces the fluid. If there are no noticeab pits or dimples in the skin but edema is still present, this is called non-pitting edema.

Pitting edema is graded on a scale from 1+ to 4+ depending on severity which is determined by the depth of the pits that remain noticeable after removing the pressure. Pitting edema rated as 1+ leaves indents that are approximately 2 mm deep and disappear quickly after the pressure is removed. With each move up the grading scale the depth of th pits increases by a factor of 2 mm, and they remain noticeable for a longer period of time. Pitting edema with a ratin of 4+ would leave indents that are at least 8 mm deep and remain visible for a noticeable period of time after the pressure was removed (at least 20 seconds).[8] In addition to evaluating the severity of the edema, assess the extent a well. Bilateral 3+ pitting pedal and pretibial edema that extends to a couple inches below the knee paints a clear picture of your patient's fluid volume excess and helps indicate the severity of that excess.

Lower extremity edema is often accompanied by noticeable changes in the texture and appearance of the skin. The excess fluid causes skin to look taunt and shiny. The increased pressure caused by the interstitial fluid compresses vasculature in the area and causes decreased circulation which will also affect skin color. If the circulation has been compromised for an extended period of time hair growth can be diminished or absent. Peripheral pulses should be assessed. The dorsalis pedis artery travels along the dorsal aspect of the foot and can normally be palpated midfoot between the great toe and the second toe; this may be difficult to locate in severely edematous feet.

Traditionally, another method of assessing circulation has been capillary refill time (CRT). This involves compressing the fingernail until it blanches. When pressure is released color should return to the area in two seconds or less. CRT assessments which are compared from day to day may be useful for hospital patients in a controlled environment, bu CRT is generally considered an unreliable method for assessing perfusion in the prehospital setting.[9,10]

Pulmonary Edema

When the heart is not pumping effectively, fluid may back up from the left side of the heart into the pulmonary interstitial spaces. Gravity will affect fluid in all the interstitial spaces and the pulmonary space is no exception. The rate and effectiveness of gas exchange between the alveoli and the pulmonary capillaries will be decreased because this fluid creates a barrier between these two surfaces. As gases try to move from the alveoli to the blood they must travel through the fluid creating adventitious (abnormal) lung sounds that resemble bubbling or crackling noises often compared to the sounds made by Rice Krispies. These lung sounds are called crackles or rales and can be described a being fine or coarse. Fluid that gathers in the pulmonary spaces or alveoli is referred to as pulmonary edema.

When auscultating lung field, note the position your patient is in during assessment. Patients in an upright position wil have fluid collect at the pulmonary bases and this will manifest as crackles on auscultation. If the patient is supine this fluid will be dispersed through a wider area and may not be as noticeable on auscultation. For consistency it is important to document the position the patient was in during the pulmonary assessment and, if adventitious sounds were noted, the amount of pulmonary field those sounds occupied. Due to fluid shifting, coarse crackles in both bases that extends halfway up through the pulmonary field may not be noted in the same patient when he or she moves from his or her chair into a supine position on a stretcher.

Other signs and symptoms associated with pulmonary edema include increased dyspnea, increased respiratory rate and coughing. If the fluid backup and pulmonary edema is severe enough, a patient may be coughing up red-tinged frothy sputum. The fluid can also irritate the pulmonary tissues causing congestion and bronchoconstriction which may be heard as expiratory wheezing on auscultation also called rhonchi.

Heart Sounds and Jugular Vein Distention

During physical examination of the heart failure patient, the AHA recommends paying particular attention to jugular venous pressures and the presence of a third heart sound due to their "prognostic significance."[2] They also list jugular vein distention (JVD) as the "most reliable sign of volume overload." [2] Unless working in the critical care transport field, EMTs do not evaluate jugular venous pressures in the field, and this skill is beyond the scope of this article.

JVD is assessed with the patient lying in the semi-Fowler's position. The jugular vein travels obliquely across the sternocleidomastoid muscle on both sides of the neck. To approximate its path, imagine a line from the angle of the mandible, where you grasp to perform a jaw thrust, to the midclavicular area. Turning the patient's head slightly to th opposite side may help visualize a distended jugular vein; if examining the right side of the neck, turn the patient's head slightly to the left.

Patient Medical History

A thorough patient history can help guide the working diagnosis. Patients with a history of myocardial infarct are eight to 10 times more likely to develop heart failure.[3] As previously mentioned, other medical history that is associated with heart failure includes hypertension, coronary artery disease, diabetes, obesity, dilated cardiomyopathy, and valvular disease. Dilated cardiomyopathy is a common cause of heart failure and idiopathic dilated cardiomyopathy ca have a genetic component, meaning a person may be more likely to develop it if they have a family history of the disease.[11] Asking about family medical history may offer an additional clue to the diagnosis if the patient has no know history of heart failure. However, this is not the most likely scenario; only 15% to 20% of patients presenting with acute decompensated heart failure have new onset.[12] Other clues include activity intolerance associated with activitie of daily living and new onset or a worsening of previous symptoms. Remember that fatigue is commonly associated with heart failure and is one of the AHA three cardinal manifestations.[4] Determine the patient's medication regime, compliance with medications, fluid restriction, diet, and weight monitoring schedule. Exacerbation of heart failure symptoms can be caused by a patient not taking his or her medications on a regular basis, not taking them as prescribed, or failing to follow a low sodium diet. Heart failure patients should weigh themselves on a regular basis as a means to monitor for increased fluid retention; some of these patients may have their logs available and these records should be transported to the hospital with the patient. The relationship between weight gain and fluid retentio is as follows: 1 kg (2.2 lbs) equals 1 liter of fluid.

Are You Sleeping

Inquire about the patient's shortness of breath, specifically what makes it better or worse and if it is associated with certain times of the day. The heart failure patient may have a history of orthopnea and paroxysmal nocturnal dyspnea (PND); PND is dyspnea that wakes the patient up from sleep while orthopnea is shortness of breath when lying supine A history of sleeping in an upright position, in a chair or with a number of pillows on the bed, may be indicative of increased shortness of breath when supine. If a patient is on a CPAP machine at night he or she may report decreased shortness of breath when sleeping. The increased airway pressure in the lungs from the machine pushes the fluid out

the interstitial pulmonary tissues allowing for better gas exchange.

Heart failure patients may have a history of nocturia which means they will report getting up at night to urinate more frequently. When the patient is supine some of the fluid that gathered in the interstitial spaces during the day moves back into the circulation. Renal blood flow increases and the kidneys attempt to remove some of the excess fluid through filtration. This results in an increase in urine formation and may manifest as nocturia.

Treatment – The Old Way

Supplement oxygen, intubation during severe exacerbations, nitroglycerine, morphine, and a diuretic (Lasix, typically were once the mainstays of prehospital treatment for patients with acute decompensating heart failure. This paradigm has changed as the field of prehospital emergency medicine has moved away from the idea that what is good in the emergency room must be good in the prehospital field, too. Also, technology has offered superior treatment options in the form of CPAP machines that became compact and ambulance-friendly. There is now evidence that the prehospital administration of diuretics and morphine were causing more negative patient outcomes than positive ones.[13,14]

Basic Level Treatment

Complete your patient assessment, place patient in sitting or upright position allowing the legs to drop/hang over edg of stretcher, if possible. Administer oxygen, as condition warrants and consider ALS support, if available.

Sample Basic Protocol

1. Place patient in position of comfort, typically sitting up, loosen tight clothing and reassure.
2. Maintain oxygenation with cannula or mask if oxygen saturations are below 94% titrate to 94% - 99%.
3. Transport immediately if the patient has any of the following:
    a. No history of cardiac problems.
    b. Systolic blood pressure of less than 100.
    c. A history of cardiac problems, but does not have nitroglycerin.
4. If capability exists, obtain a 12-lead EKG and transmit it to the receiving facility and/or medical control for interpretation prior to patient's arrival.
5. Contact medical direction for orders.
6. If the patient has been prescribed nitroglycerin (patient's nitro only) and systolic blood pressure is 90 mmHg or above, give one dose. If patient is taking erectile dysfunction drugs such as Viagra, contact medical direction prio to giving nitroglycerin.
7. Repeat one dose of nitroglycerin in 3-5 minutes if pain continues if systolic blood pressure is 90 mmHg or above and authorized by medical direction, up to a maximum of three doses Reassess patient and vital signs after each dose of nitroglycerin.
8. Further assess the patient and evaluate possible causes (unless other treatment priorities exist).[20]

CPAP

In their 2010 guidelines for the treatment of heart failure, the HFSA assigns recommendation levels to treatment options based on supporting evidence with "A" being the most valid. The only A level recommended treatment option the guidelines for acute decompensated heart failure is continuous positive airway pressure (CPAP).[3] CPAP relieves dyspnea during pulmonary congestion by applying continuous pressure to the alveoli, keeping alveoli from collapsing, and increasing functional residual capacity (FRC). It does not protect from aspiration and should not be used on a patient with an altered level of consciousness who is unable to protect his or her airway.

Possible complications associated with CPAP and increased pressures include pulmonary barotrauma, aspiration, abdominal discomfort or bloating, and allergies or irritation from the mask. Pulmonary barotrauma can manifest as a pneumothorax, a life-threatening emergency caused by air accumulating in the space between the lung and the thora wall. As more air accumulates the increased pressure causes the lung to compress and collapse – a condition known a a tension pneumothorax. If a patient on CPAP whose condition was previously improving suddenly develops increased shortness of breath, immediately auscultate breath sounds bilaterally. This sudden change is indicative of a pneumothorax. Diminished or absent breath sounds on one side indicate a tension pneumothorax which is treated by chest tube insertion or emergency needle decompression. Another possible complication with CPAP is hypotension. To achieve continuous positive airway pressure oxygen is pushed into the thoracic cavity to keep the alveoli open, which also increases the pressure in the thoracic cavity. This is called intrathoracic pressure. The increased pressure around the heart can impede blood return, which flows from high pressure to low pressure, and cause cardiac output to decrease. Continuously assess your patient and auscultate the pulmonary fields on a regular basis.

It is important to remember that CPAP allows for better oxygenation but does not ventilate the patient; he or she mu have an intact respiratory drive and be breathing on his or her own. If the patient goes into respiratory failure he or

she will need assisted ventilations. You will need to immediately remove the CPAP machine and start assisting ventilations; have airway adjuncts and a bag valve mask (BVM) readily available. If CPAP is not available positive pressure ventilation (PPV) with a non-invasive device can provide adequate ventilator support in the emergency settir and has been shown to reduce intubations and hospital mortality rates.[12]

Administration of supplemental oxygen, if hypoxemia is present, carries a level C recommendation while routine oxygen administration to patients who are not short of breath and have no signs of hypoxia is not recommended.[3] CPAP is the preferred treatment modality and should be considered before supplemental oxygen; however, oxygen should not be withheld from a patient who is short of breath. Any patient that has signs and symptoms indicative of hypoxemia (for example low SpO_2, pale, diaphoretic, tachypneic, adventitious pulmonary sounds) should be given supplemental oxygen.

Diuretics

The emergency administration of diuretics is a level B recommendation and this is only in the presence of fluid overload until the signs and symptoms of congestion are relieved.[3] The recommendation advises avoiding a "rapid reduction" in intravascular volume due to the complications involved with excessive fluid depletions and electrolyte imbalances. Serum potassium levels and serum magnesium levels should be frequently monitored as well as vital signs, congestion symptoms, and body weight.[3] These guidelines imply that diuretic administration would be more suitable in the emergency room instead of the prehospital field where an intravenous bolus of furosemide is often given, and many services do not have the capability to monitor serum electrolyte levels or patient body weight. Also, the diagnosis of acute decompensating heart failure can be challenging without the diagnostic tests and blood work results available in the hospital. Repeatedly, studies continue to show that paramedic success rates when diagnosing heart failure in the field are low, and the number of patients treated with furosemide that later experience adverse effects and/or require fluid replacement is unacceptably high.[15,16,17,18] In total, these factors and considerations ma lend credence to the idea that furosemide administration is more appropriate to the hospital setting. Perhaps with continuing education that focuses on diagnosing acute, decompensating heart failure, and technologies that allow relevant and accurate point of care testing in the field, the rate of correct diagnosis will increase.

Nitroglycerine and Morphine

Nitroglycerin, a venodilator more potent than morphine, treats the fluid shifting and overload in acute heart failure by reducing preload (the amount of blood returning to the heart) and reducing end diastolic volume (the amount of blooc at the end of diastole before contraction occurs). This decreases the work load on the heart without having the undesirable respiratory depression effects that can be seen with morphine administration. Morphine, an opioid, can cause respiratory depression due to its actions on the respiratory centers in the brain stem. This manifests as a decreased respiratory rate and respirations that are shallower than normal. The tachypnea and dyspnea seen in patients with acute decompensating heart failure is a compensatory mechanism; they are breathing faster due to cardiogenic shock and pulmonary edema. If these two issues are addressed, their respiratory rate and rhythm will return to their normal state. Morphine administration to acute heart failure patients has been associated with increase

admissions into the intensive care unit (ICU).[14]

Nitroglycerin administration can cause transient hypotension but the drug has fewer side effects than morphine administration, and patients treated with nitroglycerin appear to have better outcomes than those treated with morphine.[8,14]

Captopril and Dopamine

Reduced ICU admission rates and the need for fewer intubations have been demonstrated with the prehospital administration of sublingual captopril, adding credence to the role ACE inhibitors may play in the treatment of acute decompensated heart failure in the field.[14] Intravenous enalapril has also been shown to be beneficial.[19] ACE inhibito help block the excess fluid retention and vasoconstriction caused by the RAAS system by preventing inhibiting angiotensin converting enzyme – the enzyme required for the conversion of angiotensin I to angiotensin II. It inhibits the secretion of antidiuretic hormone (ADH) and also promotes vasodilation by preventing the enzyme's breakdown o a substance called bradykinin. Positive inotropic medications are medications that increased cardiac contractility and may be indicated during cardiogenic shock. Positive inotropic medications, like dobutamine, may be warranted during acute heart failure exacerbations if cardiogenic shock is present (depending on paramedic capabilities and protocols).

Conclusion

You administer one 0.4 mg spray of sublingual nitro and provide noninvasive airway support by applying CPAP. You obtain intravenous (IV) access by placing an 18 gauge needle with a saline lock in the right forearm. The patient is given 25 mg of sublingual captopril and a repeat 0.4 mg sublingual nitro five minutes after the first dose. While you ar obtaining another set of vital signs and printing out a 12-Lead ECG the patient reports a decrease in his shortness of breath and you note a more normal, less distressed sentence structure and speech pattern. He does not appear as tired and accessory muscle use during breathing decreases. The ECG shows sinus tachycardia at a rate of 130 with pathologic Q waves 5 milliseconds long, indicative of the patient's self-reported history of a previous MI. His blood pressure decreases to 178/108 mmHg, respiratory rate is 24, and SpO2 increases to 92%. At the emergency room, testing confirms the diagnosis of acute decompensated heart failure. The patient continues to improve, does not require intubation, and is admitted for further monitoring and evaluation.

References

1. Centers for Disease Control and Prevention. (2012, Oct. 17). Division for Heart Disease and Stroke Prevention. Heart Failure Fact Sheet. http://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_heart_failure.htm
2. Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, Jessup M, Konstam MA, Mancini DM, Michl K, Oates JA, Rahko PS, Silver MA, Stevenson LW, Yancy CW. ACC/AHA 2005 guideline update for the diagnosis ar management of chronic heart failure in the adult: a report of the American College of Cardiology/American Hear Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluatic and Management of Heart Failure). American College of Cardiology Web Site. Available at: http://www.acc.org /clinical/guidelines/failure/index.pdf
3. Lindenfeld J, Albert NM, Boehmer JP, Collins SP, Ezekowitz JA, Givertz MM, Klapholz M, Moser DK, Rogers JG, Starling RC, Stevenson WG, Tang WHW, Teerlink JR, Walsh MN. Executive Summary: HFSA 2010 Comprehensive Heart Failure Practice Guideline. J Card Fail 2010;16:475e539.
4. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, Fonarow GC, Geraci SA, Horwich T, Januzzi JL Johnson MR, Kasper EK, Levy WC, Masoudi FA, McBride PE, McMurray JJV, Mitchell JE, Peterson PN, Riegel B, Sar F, Stevenson LW, Tang WHW, Tsai EJ, Wilkoff BL. 2013 ACCF/AHA guideline for the management of heart failure: report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013;128:e240–e327
5. American Heart Association. (2012, Aug 20). Ejection Fraction Heart Failure Measurement. http://www.heart.org /HEARTORG/Conditions/HeartFailure/SymptomsDiagnosisofHeartFailure/Ejection-Fraction-Heart-Failure-Measurement_UCM_306339_Article.jsp
6. Pocock, S.J. et al. (2006). Predictors of mortality and morbidity in patients with chronic heart failure. European Scoiety of Cardiology. European Heart Journal; 27, 65-75
7. Gheorghiade, M et al. (2010). Assessing and grading congestion in acute heart failure: a scientific statement from the Heart Failure Association of the European Society of Cardiology and endorsed by the European Society off Intensive Care Medicine. European Journal of Heart Failure; 12, 423-433. http://eurjhf.oxfordjournals.org/conter /12/5/423.full.pdf
8. Hogan, M (2007). Medical-Surgical Nursing, 2nd ed. Salt Lake City: Prentice Hall
9. Anderson, B., Kelly, A.M., Kerr, D., Clooney, M., Jolley, D. (2008, Jan). Impact of patient and environmental factors on capillary refill time in adults. American Journal of Emergency Medicine; 26(1):62-65

10. Monija, M., Rytter, D., Brabrand, M. (2013). Capillary refill time is a predictor of short-term mortality for adult patients admitted to a medical department: an observational cohort study. Emergency Medicine Journal. doi:10.116/emermed-2013-202925
11. National Heart, Lung and Blood Institute. (2011, Jan 1). What Causes Cardiomyopathy? http://www.nhlbi.nih.go /health/health-topics/topics/cm/causes.html
12. Dumitru,I., Baker, M. (2013, Jan25). Heart Failure Treatment and Management. *Emedicine Referrence.* http://emedicine.medscape.com/article/163062-treatment
13. Kosowsky, J.M., Chan, J.L. (2006, Dec). Acutely decompensated heart failure: diagnostic and therapeutic strategies. Emergency Medicine Practice. EBMedicine.net; 8(12)
14. Mosesso, V.N., Dunford, J., Blackwell, T., Griswell, J. (2003). Prehospital Therapy for Acute Congestive Heart Failure: State of the Art. Prehospital Emergency Care;7:13-23. http://www.reepl.ru/_user_files /PrehospTherACHF.pdf
15. Dobson, T., Jensen, J.L., Karim, S., Travers, A.H. (2009). Correlation of paramedic administration of furosemide with emergency physician diagnosis of congestive heart failure. Journal of Emergency Primary Health Care; Vol.7 (3), Article – 990378. http://ro.ecu.edu.au/cgi/viewcontent.cgi?article=1333&context=jephc
16. Eckstein, M., Suyehara, D. (2002, Jan). Ability of paramedics to treat patients with congestive heart failure via standing field treatment protocols. American Journal of Emergency Medicine; Vol. 20 (1):23-5. http://www.ncbi.nlm.nih.gov/pubmed/11781907
17. Jaronik, J., Mikkelson, P., Fales, W., Overton, D.T. (2006, Apr-Jun). Evaluation of prehospital use of furosemide ir patients with respiratory distress. Prehospital Emergency Care; 10(2): 194-7
18. Stiell, I., Pan, A., Dionne, R., Maloney, J. (2010). The USE of furosemide in the prehospital setting for the treatment of heart failure. Canadian Journal of Emergency Medicine; Vol 14 (S1). http://www.cjem-online.ca /v14/s1/the-use-of-furosemide-in-the-prehospital-setting-for-the-treatment-of-heart-failure
19. Bosomworth, J. (2008). Rural treatment of acute cardiogenic pulmonary edema: applying the evidence to achiev success with failure. Canadian Journal of Rural Medicine 2008; 13. http://www.cma.ca/multimedia/staticContent /HTML/N0/l2/cjrm/vol-13/issue-3/pdf/pg121.pdf
20. Iowa Department of Public Health BUREAU OF EMERGENCY MEDICAL SERVICES Statewide EMS TREATMENT PROTOCOLS ADULT & PEDIATRIC January 2014