Objectives

1. Describe the pathophysiologic changes that occur in heart failure
2. List the elements of a patient history
3. Explain the components of a cardiac-focused patient assessment
4. Identify appropriate diagnostic tests for a patient with heart failure
Purpose of the Heart

• Pump blood
• Send oxygenated blood around to the body
  – Oxygen enters into aerobic cellular metabolism to produce adenosine triphosphate molecules (ATP) the power house of cell function
• Neurohormonal biofeedback loop

Normal Heart Structure

• Muscle
• Blood flow
  – Great Vessels
  – Coronary arteries
• Electrical stimulation
  – Automaticity
• Unidirectional flow
  – Four chambers separated by septum
  – Competent valves
Cardiac Output

- Cardiac output = Stroke volume \times \text{Heart rate}
- Stroke volume: blood in ventricle end diastole
  - Dependent on Preload
- Heart rate: number of beats per minute
  50 \text{ to } 150

Frank Starling Law

- Ernest Starling and Otto Frank late 1800’s early 1900’s looked at animal models
- The more the heart is stretched the more it squeezes
- This is a physiologic mechanism to allow for the CO to be responsive to increased activity

Preload

- Preload is pressure of filling
- End diastolic pressure that stretches the ventricle to its greatest geometric dimensions during diastole
- Increased LVEDP in a failing heart
Afterload

- Afterload is resistance to emptying
- Arterial pressure against which the ventricle must generate in order to eject blood out of the heart during systole; reflective of aortic pressure for the left ventricle and pulmonary artery pressure for the right ventricle
- As afterload increases cardiac output decreases

Vascular Resistance

- The resistance to blood flow that must be overcome to push blood from the heart through the circulatory system
- Systemic vascular resistance (SVR) is the resistance the left ventricle must overcome to pump blood through the systemic, or peripheral, circulation
- Pulmonary vascular resistance (PVR) is the resistance the right ventricle must overcome to pump blood through the vasculature of the lungs

Pressure Volume Curves

Big Volume \(\rightarrow\) Pressure build up \(\rightarrow\) Valves opening
Increases CO

- Target heart rate
- Increased atrial kick
- Adequate filling
- Reduced after load
- Good myocardial stretch

Impact Cardiac Performance

- Preload
- After load
- Cardiac output
- Vascular resistance
- Intra-cardiac pressures
- End diastolic pressure that stretches the ventricle to its greatest geometric dimensions during diastole.
- Increased LVEDP in a failing heart

Decrease CO

- Arrhythmias
  - Tachycardia or bradycardia
- Reduced atrial kick
- Inadequate filling
- Increased after load
- Stiff myocardial wall
Causes of Poor CO

- Coronary Artery Disease (CAD)
- Hypertension
- Myopathies
  - Viral
  - Valvular
  - Idiopathic
- Incessant arrhythmias
  - Atrial fibrillation
  - Ventricular arrhythmias

Causes of Poor CO

- Toxins
  - Alcohol
  - Chemotherapy
  - Radiation therapy
  - Illicit drugs
- Medications
  - Calcium agonists
  - Antiarrhythmics
  - Cytotoxic agents
  - NSAIDS

Causes of Poor CO

- Infiltrative
  - Sarcoidosis: inappropriate immune response resulting in granulomas
  - Amyloidosis: abnormal deposition of a protein, amyloid, in tissues
  - Hemochromocytosis: excessive iron absorption and deposition in organs
  - Connective tissue diseases
- Other causes
  - Chiagas
  - HIV
  - Peripartum
  - End stage renal disease
Causes of Poor CO

- Metabolic disorders
  - Hyperthyroidism
  - Diabetes mellitus
  - Septic shock
- Nutritional
  - Thiamine deficiency: Beriberi: Seen in sailors deprived of Vitamin B or alcoholics
  - Selenium: trace mineral
  - Obesity
  - Cachexia

Causes of Poor CO

- Genetic
  - Duchenne muscular dystrophy
  - Certain hypertrophic cardiomyopathies
  - Hypertrophic obstructive CM (HOCM)
- Congenital abnormalities
  - Tricuspid atresia
  - Septal defects

Acute Decompensated Heart Failure (ADHF)

HF, a complex clinical syndrome, can result from any structural or functional cardiac disorder that impairs ability of ventricle to fill with or eject blood.
Cardinal symptoms are fatigue and dyspnea, and clinical signs are fluid retention and exercise intolerance
Heart Failure is a progressive clinical syndrome that involves the interaction of multiple systems in the body.

Hunt SA et al. Circulation. 2001;104:2996
Neurohormonal Activation

- Vasopressin (Anti-diuretic Hormone)
  - Released in response to arterial underfilling/reduced volume via high pressure baroreceptors
  - Regulates retention of water by stimulating renal tubular reabsorption
  - Causes congestion and hyponatremia

- Endothelin (ET)
  - Vasconstrictor peptide expressed in the myocardium stimulated by hypoxia, ischemia, neurohormones
  - Increased levels contribute to progression of left ventricular dysfunction and secondary pulmonary hypertension

Dysregulation of Immune System

- Untoward effects of pro-inflammatory cytokines on ventricular function
- Immune activation of pro-inflammatory cytokines
- Negative inotropic effect
- Induce abnormalities in cardiac metabolism
- Promote myocardial remodeling and cardiomyocyte hypertrophy
- Apoptosis

Types of Heart Failure
Heart Failure with Preserved Ejection Fraction (HFpEF)

- Normal or near normal EF > 50%
- Hypertrophic CMP, HTN, Ischemia, Age
- Restrictive filling impaired relaxation

Heart Failure with Reduced Ejection Fraction (HFrEF)

- Depressed contractility EF < 40%
- CAD, Valve disease, Ischemic and Idiopathic CMP
- Impaired contraction, forward or backward failure

Intra-cardiac Pressures Guide Diagnosis and Treatment of HF

- Compensatory mechanisms increase blood flow as well as cardiac filling pressures and heart rate to maintain pumping function of the heart and supply redistribution of blood flow
- Damaged myocardium stretches out of shape causing increased filling pressures
Normal Pressures

- Central venous pressure (CVP) 0-5 mm Hg
  - Pressure of blood in the vena cava, near the right atrium
  - Reflects the amount of blood returning to the heart
  - Good approximation of right atrial pressure

- Right ventricular pressure
  - Systolic – 15-30 mm Hg
  - Diastolic – 0-5 mm Hg

- Pulmonary artery pressure
  - Systolic – 15-30 mm Hg
  - Diastolic – 2-10 mm Hg
  - Pulmonary capillary wedge pressure (PCWP) 6-12 mm Hg

- Pulmonary vascular resistance (PVR)
  - \( \frac{PAP-PCWP}{CO} \) \* 80 \( \frac{\text{dynes-sec/cm}^5}{\text{mL}} \)
  - An increase in PVR can result in RV failure
  - Abnormally high PVR can be a contraindication to cardiac transplantation as it could cause RV dysfunction in the transplanted heart

- Pulmonary capillary wedge pressure (PCWP) 6-12 mm Hg
  - Indirect pressure of left atrial pressure
  - Pulmonary edema reflective of PCWP >25 mm Hg

- Left ventricular systemic pressure (systolic) 100-140 mm Hg

- Pulmonary vascular resistance (PVR)
  - \( \frac{PAP-PCWP}{CO} \) \* 80 \( \frac{\text{dynes-sec/cm}^5}{\text{mL}} \)
  - An increase in PVR can result in RV failure
  - Abnormally high PVR can be a contraindication to cardiac transplantation as it could cause RV dysfunction in the transplanted heart

- Systemic vascular resistance (SVR)
  - \( 80 \times \frac{PAP-CVP}{CO} \) \( \frac{\text{dynes-sec/cm}^5}{\text{mL}} \)

Perils of Elevated PVR

Elevated PVR is a possible contraindication to cardiac transplant
Elevated PVR causes RV dysfunction/failure and death in heart transplant recipients especially in the first month after transplant

Caution for Patients in the Coffin Corner

Rapid Assessment of Hemodynamic Status

- Low perfusion at rest
  - Warm & Dry
  - Cold & Dry
  - Warm & Wet
  - Cold & Wet

- Possible evidence of low perfusion
  - Cyanosis
  - Mottled skin
  - Cool extremities
  - Weak or thready pulse
  - Hypotension
  - Bradycardia

- Treat with interventions to improve perfusion
  - Infusion
  - Vasopressor
  - Inotropic support
  - Imaging

- Monitor response to interventions

- If no response, consider other diagnoses or complications
Long Term Affects of HF

Myocardial Remodeling

- Alteration in the structure (dimensions, mass, shape) of the heart muscle in response to the hemodynamic load and/or cardiac injury in association with neurohormonal activation
- Remodeled heart is less elliptical and more spherical
- Left ventricular dilatation
- Left ventricular hypertrophy
- Occurs with pressure overload
  - aortic stenosis
  - hypertension
  - volume overload
  - valvular regurgitation
Cardio Renal Syndrome

- Cardio renal syndrome occurs when reduction in cardiac output results in disproportionate reduction in renal perfusion
- Leads to diminished glomerular filtration rate (GFR) and increased serum creatinine levels
- Worsening renal function:
  - Change in serum creatinine >0.3 mg/dL or >25% over baseline (normal range: adult male: 0.8 to 1.4 mg/dL, adult female: 0.6 – 1.4 mg/dL)
  - >70% of patients will experience increase in creatinine during hospitalization
  - 20% to 30% of patients experience increase of >0.3 mg/dL
  - Any increase of >0.3 mg/dL is associated with longer LOS and increased mortality

Cardio Renal Syndrome

Cardio Renal Syndrome is a complex interaction between the heart and kidneys resulting in bidirectional injury and dysfunction.

There are 5 subcategories
- ADHF leading to ARF
- Chronic HF leading CKD
- ARF leading to cardiac event
- CRD leading to worsening CV disease
- Acute illness (i.e. sepsis) leading to simultaneous heart and kidney failure
Hyponatremia

- Hyponatremia defined as serum sodium concentration <136 mmol/L
- Mild hyponatremia is seen in approximately 25% of patients with acute HF
- Hyponatremia poses significantly greater risk of death after discharge
- Reduction in cardiac output triggers hyponatremia

Hyponatremia as a Prognostic Tool

- Hyponatremia is one of the strongest predictors for poor outcomes
- Hyponatremia may be caused by multiple factors:
  - RAAS: Aldosterone causes sodium and water retention.
  - Antidiuretic Hormone, Vasopressin, ADH
  - Impaired renal function leads to a decreased ability of the kidneys to excrete sodium and water.
- Efforts should be made to limit nephrotoxic medications and therapies such as
  - Dye loads
  - NSAIDS
  - Antibiotics


Osmotic Movement of H2O
Identifying Heart Failure

What Patients Tell You is Important

• How can we help patients report symptoms appropriately
• HFS FACES

The Art of Taking a History

A history is
• past events
• subjective complaints
• symptoms
• current state
How Does the History Help?

- Documenting the presence or the progression of signs and symptoms is important in determining:
  - The degree of heart failure
  - Whether treatment or medical interventions are therapeutic and achieving the desired goal for the patient

Systematic Method to Obtain an Accurate History

- Patients do not always recognize symptoms as evolving from HF, so it is important to ask specific questions to obtain the information necessary to develop the plan of care
- Early detection of signs and symptoms can result in more effective treatment and decrease mortality
- An accurate history sets the stage for appropriate education and make timely changes in patient’s plan of care

Elements of a History Include

- Chief complaint
- History of present illness (HPI)
- Past Medical History
- Family history
- Social history and habits including past or present history
- Nutritional history
- Review of Systems
- Drug/blood history
- OB/GYN
Chief Complaint

• Reason for the visit in the patient’s own words
• Put the reason in quotes

History of present illness (HPI)

• Concise description of the patient’s current health status focusing on current HF signs and symptoms
  – Each positive symptom should be explored with the patient using the “PQRST” mnemonic
  – Document current treatment modalities or plan of care including the patient’s stated adherence to the prescribed plan of care – medications, dietary and fluid restrictions
  – Document recent clinic visits, hospitalizations or emergency department visits for similar signs and symptoms, and any new prescriptions or change in prescriptions

“PQRST”

If a patient acknowledges a specific symptom, the following questions using the mnemonic “PQRST,” to gain more specific details
P = Provocation and Palliation

• What causes the symptom
• What makes it worse
• What makes it better

Q = Quality and Quantity

• How does it feel, look or sound
• How much of it is there
• Try to let patient describe the pain, sometimes they say what they think you would like to hear
  - Sharp
  - Dull
  - Stabbing
  - Burning
  - Crushing
• If describing a discharge:
  - Thick
  - Runny
  - Clear
  - Color
• If describing a psychological problem: Do the voices drown out other sounds
  - Whose voice does it sound like

R = Region and Radiation

• Where is it?
• Does it spread?
• Where does the pain radiate?
• Is it in one place?
• Does it go anywhere else?
• Did it start elsewhere and is now localized to one spot?
• In the case of pain, does it travel:
  - Down your back
  - Down your arms
  - Up your neck
  - Down your legs
  - Jaw pain
**S = Severity and Scale**

- Does it interfere with activities
- How does it rate on a scale of 1 to 10
- How bad is it when it's at its worst
- Does it force you to sit down, lie down, slow down
- How long does an episode last

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**T = Timing and Type of Onset**

- When did it begin
- How often does it occur
- Is it sudden or gradual
- How long does it last
- What time did it begin
- When was the first date it happened
- What were you doing when you first experienced or noticed it
- How often do you experience it: hourly? daily? weekly? monthly?
- When do you usually experience it: daytime? night? in the early morning?
- Are you ever awakened by it
- Does it lead to anything else
- Is it accompanied by other signs and symptoms
- Does it ever occur before, during or after meals
- Does it occur seasonally

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**Past Medical History**

- History of cardiac-related conditions
- History of non-cardiac conditions that may increase morbidity in HF
- Allergies Surgical history/procedures
- Medications
- Immunization history
Family and Social History

- Focus on family members having:
  - Coronary artery disease
  - Stroke or transient ischemic attack
  - Cardiomyopathies
  - Sudden death
  - Hypertension
  - Hyperlipidemia
- Social history and habits including past or present history of:
  - Tobacco abuse (documented as pack/year history)
  - ETOH, drug use
  - Current level of physical activity
  - Education level
  - Pets
  - Travel
- Nutritional history including:
  - Weight loss or weight gain
  - Summary of average dietary intake focusing on fluid status and sodium intake

Review of Symptoms

Since patients do not always recognize all of their symptoms as evolving from HF, each of the following categories should be explored with the patient using the “PQRST” mnemonic:

- Shortness of breath (SOB) – the most common heart failure symptom
- Orthopnea can be very suggestive of HF
- Paroxysmal nocturnal dyspnea (PND)
- Cough
- Wheezing
- Symptoms
- Chest pain or angina
- Gastrointestinal Problems
- Fluid Overload
- Anoxia
- Palpitations
- Mental status changes or cerebral hypoperfusion
- Sleep disturbances
- Skin alterations

Shortness of Breath (SOB)

- SOB or DOE may occur early in the disease process before other symptoms of HF are evident
- As HF becomes progressively worse, less exertion is required before the patient develops a sense of dyspnea
- Have patient’s normal activity level changed or been curtailed
Orthopnea

- Occurs due to the redistribution of fluid from the lower extremities into the central circulation during recumbency
- Results in increased pulmonary capillary pressure
- Can occur rapidly with change in positioning and is usually relieved with sitting upright
- Appropriate questions to ask:
  - Number of pillows used beneath the patient's head while sleeping in order to breathe comfortably
  - Degree of elevation of the head of the bed
  - If the patient must sleep in a recliner in order to breathe comfortably

Paroxysmal Nocturnal Dyspnea (PND)

- Sudden onset of SOB that wakes a patient from sleep
- PND is caused by fluid accumulation in the lungs entering the alveoli during sleep
- During the day, the fluid is retained in the legs, but at night, while sleeping, the body reabsorbs this fluid resulting in an increase in total blood volume leading to pulmonary edema
- Associated symptoms can include anxiety and a sense of suffocation
- What relieves the symptom of PND
- Frequency of episodes

Cough

- Can be due to pulmonary, cardiac, gastric causes or a side effect of medications
- A dry hacking cough can be associated with angiotensin converting enzyme inhibitors (ACEI) as the result of increased bradykinin levels
- Frequently overlooked symptom is a chronic non productive cough that is worse in the recumbent position. This is frequently associated with pulmonary congestion
- Associated symptoms to explore when asking about a cough include presence of hemoptysis, wheezing, rhinorrhea, or sputum production
- Exposure to illnesses that cause chronic cough like tuberculosis
Wheezing

- May be caused by congestion of bronchial mucosa and compression of small bronchi
- May be related to pulmonary disease
- May be present at rest or with exertion

Fatigue

- Decreased exercise tolerance - comparison of what the patient can do compared to what they have been able to do in the past
- Comparison between what a patient could do in the past (3-6 months ago) compared to their present level of activity
- Fatigue may occur due to a decreased cardiac reserve
- As patient’s activity increases, the heart cannot maintain a cardiac output sufficient to sustain the activity
  - Leg fatigue/heaviness due to edematous extremities and poor peripheral blood flow may develop as the result of:
  - A low cardiac output
  - Venous congestion in the lower extremities
- Neurohormonal suppression of vasodilatory responses

Chest Pain or Angina

- May be the result of a progression of HF with a subsequent reduced cardiac output
- May be the associated with progression of CAD
- May occur due to myocardial stretch in fluid volume overload
- Important points to remember are:
  - Some will only use the term discomfort or pressure, not chest pain
  - Women may have an angina equivalent and complain of back pain, abdominal pain or neck pain
  - Because of neuropathy, diabetics may not experience chest pain or angina, but may have other symptoms such as shortness of breath
Gastrointestinal Problems

- May be due to ascites and passive liver congestion
  - Abdominal discomfort or tenderness
  - Bloating
  - Episodes of nausea and vomiting
- Early satiety
- Change in bowel habits including constipation, due to fluid shifting from intravascular space into tissues
- Anorexia or a loss of appetite
- Dysgeusia (alteration in the sense of taste) may be caused by some medications such as ACEI or angiotensin receptor blockers

Fluid Overload

- Edema is extravascular fluid sequestered in the body's tissues, usually a symptom of right HF, either in combination with left HF or by itself
- Generalized throughout the body, or for male patients, scrotal edema
- Edema may also be related to medications such as calcium channel blockers or NSAIDs, increased sodium intake, or venous stasis

Ascites

- Extra fluid within the peritoneal cavity caused by high pressure in the hepatic veins and portal circulation
- Symptoms may include:
  - Pants or clothes that feel tighter around the waist
- Increase in weight one kilogram increase in weight (or 2.2 pounds), equals one liter of retained fluid
Palpitations

- The most common dysrhythmia with HF is atrial fibrillation (AF)
- Patients may note fast irregular heartbeat and a fluttering sensation, or they may not feel any different when AF occurs
- AF may be either chronic or paroxysmal
- Palpitations may also be accompanied by symptoms of SOB, dizziness or near-syncope

Mental Status Changes

- Can occur as a result of hypoxia as a result of low output
- Related to worsening HF manifested as
  - Forgetfulness
  - Change in attention span (or inattentiveness)
  - Confusion
  - Poor judgment
  - Uncoordinated movements
- Mental status changes may also indicate that the patient has had a stroke or a transient ischemic attack
  - Document the etiology, timing and frequency of these events

Sleep Disturbances and SDB

- Nocturia may be causing patient to awaken frequently to urinate, and decrease deep sleep intervals
- Snoring and day time sleepiness may be indicative of sleep apnea
  - Does the patient feel well rested in the morning when awakening?
  - Do they nap often or fall asleep easily during the day?
  - Have they had a positive sleep study?
  - Do they have either CPAP or BiPAP and do they use it?
- Insomnia may be related to stress, anxiety, depression, or napping excessively during the day
Skin Alterations

Skin alterations may be present because of:
- Poor peripheral perfusion due to low cardiac output
- Venous stasis
- Peripheral arterial disease

Peripheral edema
- Development of deep vein thromboses (DVT)
- Mottling usually seen in poor perfusion, includes blue or grayish coloring, particularly in the extremities and is typically accompanied by a prolonged capillary refill
- Temperature changes may indicate poor perfusion
- Skin cool, cold, or clammy to the touch can indicate poor perfusion
- Hot reddened skin can indicate a DVT which may be related to bed rest or limited mobility

Exam

Look Listen Feel
Physical Assessment

• The physical exam begins the moment the patient is in sight
• Heart failure focused cardiac exam should be performed starting from the head and progressing down to the toes
• Important to remove the patient’s clothing, shoes, and socks to observe assessment findings
• Examine first while sitting up, then while lying supine at 45 degree angle
• Always examine from patient’s right side; follow the same exam procedure for every patient in order to easily note abnormal signs indicating HF

General Appearance

• Does patient look ill, well or malnourished?
• Note general build and appearance
• Malnutrition and cachexia
• Skin color and presence of pallor or cyanosis
• Shortness of breath or orthopnea

Signs or Symptoms of Pain

• Sitting quietly without moving
  – Consider asking about angina
• Moving about to get comfortable
  – Visibly uncomfortable consider acute myocardial infarction
• Sitting upright
  – indicative of HF
• Leaning forward
  – indicative of pericarditis or HF
Vital Signs

- Heart rate and rhythm
- Blood Pressure (BP)
- Respiratory rate and character
- O2 saturation
- Weight
- Height

Neurologic Assessment

- Assess responses to questions and ability to recall past events in their medical history
- Confusion and altered mental status
  - Decreased cerebral perfusion
  - Hypotension
  - Liver congestion or cirrhosis
  - Poly-pharmacy
  - Dementia
- Anxiety, depression
  - Increased incidence in HF patients, exceeding general population
  - May decrease adherence to HF regimen
  - Associated with increased mortality
- Cognitive impairment
  - Impacts patient’s ability for self-care management
  - May lead to medication errors

Neck Assessment

- Jugular venous pressure (JVP)
- Hepatojugular Reflux or abdominojugular test
Chest Assessment

• Point of maximum impulse (PMI)
  – Normally located in the 5th intercostal space (ICS) on the left anterior chest, but may be displaced laterally in the presence of cardiomegaly
  – PMI is not palpable in some patients

• Precordial lift or heave
  – An anterior systolic movement along the left parasternal border
  – May be felt with the finger pads. It may be caused by right ventricular pressure overload or volume overload
Cardiac Assessment

- Proper patient positioning is important. Auscultate over intercostal spaces (ICS), not over ribs
- Start at the apex of the heart (5th ICS midclavicular line). S1 most pronounced here
- Move diaphragm to left lower sternal border at 5th ICS
- Listen at the base of the heart at the 2nd ICS left sternal border. S2 is most prominent here
- Final location is right sternal border at 2nd ICS
- Switch to bell and reverse the order of listening

Abnormal Heart Sounds Indicate

- Fluid overload
- Ventricular dilatation
- Valvular abnormalities that contribute to, or result from, heart failure

Types of Abnormal Heart Sounds

- S3 ventricular gallop may be earliest sign of HF
- S4 atrial gallop
- Aortic insufficiency: failure of the aortic valve to close during diastole
- Mitral stenosis: failure of the mitral valve to open appropriately
- Mitral regurgitation: failure of the mitral valve to close appropriately
- Tricuspid regurgitation: failure of the tricuspid valve to close appropriately as the right ventricle dilates
Pulmonary Assessment

- Crackles:
  - Do not clear with coughing, caused by excessive fluid within the airways
  - Occur mid to late inspiration
  - Bibasilar crackles often present with HF

- Wheezes: Also known as cardiac asthma, caused by bronchial edema from worsening HF
  - May occur during inspiration or expiration
  - Occurs due to narrowing or obstruction of some part of the pulmonary tree
  - More prominent when the patient is recumbent
  - May be mistaken for bacteriaviral infection
  - May clear when the patient coughs

- Rhonchi: Low-pitched, caused by passage of air through thick mucus secretion
  - Heard in COPD and acute severe bronchitis
  - May clear when the patient coughs

- Cough: Usually not productive, may be mistaken for bacterial/viral infection
  - More prominent when the patient is recumbent
  - Dry hacking cough may be reflective of an adverse effect of ACEI

- Hemoptysis: Pink blood or pink tinged
  - Pink frothy sputum is indicative of pulmonary edema, etiology is the rupture of engorged bronchial veins
  - May be worse if the patient is anticoagulated

Abdominal Assessment

- Use Auscultation, Percussion, Palpation:
  - Abdominal distension
  - Ascites
  - Hepatomegaly
    - Palpation of the liver should be done slowly and carefully
    - Percuss the liver

Peripheral Exam

- Assess for Edema
  - 1+ is 2mm depression and disappears rapidly
  - 2+ is 4mm depression and disappears in 10 to 15 seconds
  - 3+ is 6mm depression and may last a minute or longer
  - 4+ is 8mm depression may last 2 to 5 minutes
  - Other causes of peripheral edema include renal failure, cellulitis, pregnancy, venous stasis, cirrhosis

- Assess capillary refill in all 4 extremities
- Normal capillary refill is less than 3 seconds
- Prolonged capillary refill (greater than 3 seconds) may be due to:
  - Vasoconstriction
  - Poor perfusion (low cardiac output)
  - Peripheral arterial disease
  - Cold, mottled extremities
  - Due to peripheral arterial vasoconstriction
  - Can be present with or without cyanosis

- Cold, clammy, moist skin signifies poor perfusion
Assessment of Functional Class

• New York Heart Association (NYHA) Functional Class
  – Relates to symptoms of everyday activities and the patients quality of life

ACC/AHA Stages of Heart Failure

• Classification system that reflects the level of structural changes of the heart
• Four stages help define the goals of treatment and specific therapies recommended for each stage
• Patients progress in one direction (Stage A to D) due to cardiac remodeling and the progressive nature of LV dysfunction.
Testing

• Identify appropriate diagnostic tests for a patient with heart failure

Rhythm and Synchrony

• Electrocardiogram and monitoring
  – Assess for past myocardial infarcts
  – Rhythm
  – Width of QRS

Imagining and Function

• Chest X-ray
  – Cardiomegaly
  – Pneumonia
• Echocardiogram
  – Ejection Fraction
  – Valve competence
  – Wall movement
• Coronary Arteriography
  – Need for revascularization
Functional Capacity

- Cardiopulmonary Stress Test
  - Objective measure of oxygen consumption as a segregate for CO
  - MVO2 < 14 should be a trigger for AHF referral
  - VE/VO2 slope also helpful
- Exercise stress test may help to assess for CAD

Laboratory Tests

- CBC
- Urinalysis
- BMP
- Fasting Blood Sugar
- Lipid Profile
- Liver Function tests
- BNP for patients with SOB
- Pre-albumin
- T4 TSH
- Iron studies

Case Study

HISTORY
- 63 year old white male, under the care of cardiologist and primary care physician
- Frequent readmissions for decompensated HF/ER visits
- Recently underwent a cardiac catheterization - found to have no evidence of obstructive CAD
- Admits to occasional high dietary sodium intake, several instances within the last week
- Has been drinking more than the recommended 2 liters of fluid a day
- Has not been adherent to medication regimen as determined by his daughter who picks up his prescriptions
SYMPTOMS
• Weight today was up 9 pounds over the last 2 months
• Increasing dyspnea with exertion over the last 2 days as well as abdominal distention and early satiety, easy at rest, mild orthopnea,
• No PND, lightheadedness, or coughing
• Has noticed mild lower extremity edema

DIAGNOSTICS
• BMP included a sodium of 141, potassium of 3.7, BUN of 15, and creatinine of 1.0
• Chest x-ray showed mildly increased pulmonary vasculature
• LVEF of 28% documented via myocardial perfusion within last year
• Echo within last year shows global hypokinesis with elevated left ventricular filling pressures

CURRENT MEDICAL DIAGNOSES
• Hypertension
• Cardiomyopathy Idiopathic
• Atrial Fibrillation
• COPD
• Hyperlipidemia

ALLERGIES
• NKA
MEDICATIONS
• Coumadin 5 mg tablet, Takes 1 tab daily, or as directed
• Digoxin 250 mcg tablet, 1 by mouth daily
• Furosemide 20 mg tablet, 1 by mouth daily
• Lisinopril 10 mg tablet, 1 by mouth daily
• Metoprolol tartrate 100 mg tablet, 1 by mouth twice daily
• Mirtazapine 30 mg tablet, 1/2 tab
• Pravachol 20 mg tablet, 1 by mouth daily

PAST HISTORY
• Past Medical Illnesses: Arthritis (osteo), hypertension, hyperlipidemia
• Past Cardiac Illnesses: Atrial fibrillation, cardiomyopathy(dilated)
• Surgical Procedures: No previous surgical procedures

SOCIAL HISTORY
• Alcohol Use: Does not use alcohol
• Smoking: Current non-smoker
• Diet: Low sodium diet and caffeine use-2-3 per day
• Lifestyle: Divorced
• Exercise: No regular exercise, minimal basic ADL’s
• Occupation: Unemployed and on disability
• Resources: Unemployed, limited financial resources
• Social support: Limited, one daughter living in area who checks on him weekly. Has a weekly Home Health nurse monitoring failure and has a telemonitoring system installed. No close friends
• Education: Completed up to 6th grade, diagnosed as functionally illiterate
• Illicit Drug Use: Denies substance abuse
• Residence: Lives alone
HEART FAILURE CLINIC DATA
• HF Clinic Enrollment Date: 6/25/12
• Etiology: Dilated Cardiomyopathy
• Comorbidities: Atrial Fibrillation, Hypertension, Hyperlipidemia
• Education with Successful Teach back: Needs Reinforcement
• Medication Reconciliation Accurate: Yes

PHYSICAL EXAMINATION
• Blood Pressure: 130/80 sitting, left arm, regular cuff; Pulse: 72/min; Respirations: 16/min.
• Weight: 206.00 lbs.
• Height: 61”
• BMI: 39
• Constitutional: Well developed, well nourished, in no acute distress, 63 year old male arriving ambulatory
• Skin: Warm and dry to touch
• Head: Normocephalic, normal male hair pattern
• ENT: Ears, nose and throat unremarkable
• Neck: No JVD, no bruits, non-tender
• Chest: Diminished breath sounds, fine rales in bases
• Cardiac: Irregularly irregular rhythm with variable 1st heart sound, normal 2nd heart sound, no murmur, positive S3, no S4
• Abdomen: Abdomen slightly firm, non-tender, moderately obese
• Peripheral Pulses: Pulses full and equal in all extremities
• Extremities & Back: 1+ bilateral calf edema, 1+ bilateral ankle edema
• Psychiatric: Mood appropriate, no difficulties with speech or language
• Neurological: Oriented to time, person and place

QUESTIONS FOR PART 1:

1. Name patient’s NYHA class/stage
   a. Stage A, Class 2
   b. Stage C, Class 2
   c. Stage B, Class 3
   d. Stage D, Class 3
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1. Name patient’s NYHA class/stage
   a. Stage A, Class 2
   b. Stage C, Class 2
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QUESTIONS FOR PART 1:

2. What type of HF does patient have?
   a. HFpEF
   b. Combined
   c. Restrictive
   d. HFrEF

QUESTIONS FOR PART 1:
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3. From exam, list the clinic signs of HF exacerbation:
   a. S3, edema, fine rales in bases, edema
   b. Elevated blood pressure, edema, firm abdomen
   c. Elevated BMI, abnormal lung sounds, weight gain
   d. S3, elevated blood pressure, edema

QUESTIONS FOR PART 1:

4. Describe the co-morbidities the patient has for potential worsening of his HF and symptoms:
   a. Atrial fibrillation
   b. COPD
   c. Hypertension
   d. All of the above
4. Describe the co-morbidities the patient has for potential worsening of his HF and symptoms:
   a. Atrial fibrillation
   b. COPD
   c. Hypertension
   d. All of the above