Heart Failure Certification Review Course
Part 1

Ashley Moore-Gibbs, MSN, AGPCNP-BC, CHFN

Objectives
♥ Review the current incidence and prevalence of heart failure (HF)
♥ Discuss the basic pathophysiology of heart failure (HF)
♥ Define HF and discuss various etiologies
♥ Outline the neurohormonal and hemodynamic changes in HF
♥ Discuss how to perform a history and physical exam on a HF patient

Heart Failure has Reached Epidemic Proportions

Prevalence
- 5.7 million Americans ≥ 20 years of age have HF
- HF will increase 46% from 2012 to 2030 with
  - Greater than 8 million people ≥ 18 years of age

Incidence
- Between 400,000 – 700,000 new cases/year
- 75% of HF cases have antecedent HTN

Morbidity
- > 1 million hospital discharges
  - [117% from 1979 to 2005]
  - Most frequent cause of hospitalization in elderly (> 65 y/o)
  - Accounts for 5% to 10% of all hospital admissions (> 1 million)

Mortality
- One in 9 deaths included HF as contributing cause
  - AHA Heart and Stroke Statistical Update 2015, Circulation.
Hospitalizations Due to HF Continue to Rise

- Progression of disease inevitable
- Incidence of HF rising
  - Population of US is aging
    - 36 million over age 65 years in 2003, 87 million in 2050
    - In 2010, “Baby Boomers” began reaching 65 years
- Survival has improved with acute myocardial infarction and revascularization
- HF is not treated appropriately during hospitalization
- Patients do not adhere to diet and drugs
- By 2030, total Cost of HF will be almost 127% ($69.7 billion from the total cost of $30.7 billion in 2012

Implications of a Diagnosis of HF

- Mortality rates at 1 to 2 years estimated at 35%-50% for advanced heart failure
  - 4-5 year mortality rate ranges 15% - 40% with mild to moderate symptoms
  - 5 year survival following hospitalization is ~ 42.3%; each re-hospitalization increases mortality by 20% - 22%
- Eighty percent of men and 70% of women younger than 65 years of age die within one year
- Re-hospitalization rates 6 months following discharge are as much as 50%

Problems with Conventional Management

- Reactive approach
- Urgency to discharge home – short term management of acute problem that is chronic in nature
- Problems with continuity of care
- Patients do not seek attention in a timely manner - 80% admitted when presenting to ED
- May not receive adequate care
Precipitating Factors for (Re-)Hospitalization

<table>
<thead>
<tr>
<th>Cause</th>
<th>Contributing Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary indiscretions &amp; medical nonadherence</td>
<td>excessive salt intake, nonadherence to medical therapy</td>
</tr>
<tr>
<td>Innovative cardiac dysfunction</td>
<td>progression of underlying dysfunction, cardiac toxins, right ventricular pacing</td>
</tr>
<tr>
<td>Other Cardiovascular Causes</td>
<td>cardiac dysrhythmias, uncontrolled hypertension, acute myocardial infarction, valvular disease (progressive mitral regurgitation)</td>
</tr>
<tr>
<td>Noncardiac Causes</td>
<td>pulmonary disease, anemia, systemic infection</td>
</tr>
<tr>
<td>Adverse Effects of Medications</td>
<td>negative inotropic medications, medications that increase sodium retention</td>
</tr>
</tbody>
</table>

What is Heart Failure?

Complex clinical syndrome that can result from any structural or functional cardiac disorder impairing the ability of the ventricle to fill with or eject blood.

Purpose of the Cardiovascular System

- **Oxygen Delivery:** distributes oxygen-rich blood to cells in different parts of the body.
- **Waste Removal:** transport of by-products of cellular metabolism including carbon dioxide and lactic acid.
- **Synthesis and Regulation of Hormones:** The heart produces several hormones, including atrial and B-type natriuretic peptide.
Heart Anatomical Position

- Positioned slightly to the left center of the chest with the right side rotated more anteriorly and its left side mostly posterior.
- Located in the mediastinum behind the lungs and between the 2nd to the 5th intercostal space and from the right border of the sternum to the left midclavicular line.
- The apex is the pointed end and is the area of the PMI.
- The base is the broad aspect and forms the upper border of the heart below the 2nd rib and primarily involves the left atrium, part of the right atrium, and proximal portion of the great vessels.

Heart Wall

The wall of the heart has three distinct layers of tissue.

1. **Epicardium**: thin, fibrous external surface where the coronary arteries lie.
2. **Myocardium**: thick, middle layer with involuntary cardiac muscle cells that contract to propel blood.
3. **Endocardium**: innermost layer, forms the smooth lining of the heart's chambers and also helps form the heart's valves.

Lymphatic System

- Most fluid is absorbed by tissue cells.
- Excess drains into lymphatic capillaries and is eventually returned to the cardiovascular circulation.
Defining and Describing Heart Failure

- Ischemic versus Non ischemic Cardiomyopathies
- Heart failure in the setting of Reduced Left Ventricular Ejection Fraction (LVEF) versus Preserved LVEF
- Left-sided, Right-sided and Biventricular Heart Failure
- Remodeling versus Reverse Remodeling
- Chronic versus Acute Decompensated Heart Failure
- Staging & Classifying Heart Failure

Etiology of Heart Failure

The loss of a critical quantity of functioning myocardial cells after injury to the heart:
- Ischemic Heart Disease
- Hypertension
- Idiopathic Cardiomyopathy
- Infections (e.g., viral myocarditis, Chagas’ disease)
- Infiltrative cardiomyopathy (e.g. amyloidosis, sarcoidosis, hemochromatosis)
- Toxins (e.g., alcohol, cancer therapies, radiation therapy, illicit drugs)
- Valvular Disease
- Genetic (e.g. Duchenne muscular dystrophy, certain hypertrrophic cardiomyopathies)
- Metabolic disorders (e.g. hyperthyroidism, diabetes mellitus)
- Incessant Arrhythmias, tachycardia-induced
- Congenital Heart Disease
- Peripartum cardiomyopathy
- Obesity
- Rare manifestation of systemic lupus erythematosus
- Stress (Takotsubo) cardiomyopathy
- Endocrine disease (Thyroid)

Etiology: Ischemic versus Nonischemic

<table>
<thead>
<tr>
<th>Ischemic in Origin</th>
<th>Nonischemic in Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive</td>
<td></td>
</tr>
<tr>
<td>Valvular Disease</td>
<td></td>
</tr>
<tr>
<td>Toxins (ETOH)</td>
<td></td>
</tr>
<tr>
<td>Chemotherapy agents</td>
<td></td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td></td>
</tr>
<tr>
<td>Autoimmune disorders</td>
<td></td>
</tr>
<tr>
<td>Endocrine disease (Thyroid)</td>
<td></td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
</tr>
<tr>
<td>Viral processes</td>
<td></td>
</tr>
</tbody>
</table>
Heart Failure: Is it Left-Sided, Right-Sided or is it Both?

- **Left-sided HF:** left ventricular dysfunction
  - Most common form of HF, disease progression leads to right-sided failure

- **Right-sided HF:** right ventricular dysfunction
  - Occurring due to pulmonary etiology or failure of the left side of the heart

**Left Ventricular Dysfunction**

**Systolic:** Impaired contractility/ejection
- HF with Reduced Ejection Fraction (HFrEF)
- Ejection Fraction <40%

**Diastolic:** Impaired filling/relaxation, increased left ventricular wall stiffness; reduced left ventricular compliance.
- Ejection Fraction >40%
- Estimates on incidence vary; now reported ~50%
- Heart failure with preserved ejection fraction (HFpEF)

**Chronic versus Acute Decompensated HF**

- **Chronic HF:**
  - more stable condition
  - organ function remains adequate

- **ADHF:**
  - unstable condition
  - immediate treatment is warranted to prevent injury to systemic organs
Case Study 1
Defining HF, HF symptoms, History & Physical Exam, Class/Stage of HF, ECHO, HF Medical Therapy

DF is a 72 year old male with a long-standing history of an ischemic cardiomyopathy. Prior echocardiogram performed in Jan 2012 showed LVEF 40%. He has been added on to the office schedule for cardiac evaluation for complaints of shortness of breath (SOB).

An echocardiogram is obtained the day of the office visit.

Heart Failure Pathophysiology

Myocardial injury → Fall in LV performance → Activation of RAAS, SNS, ET, and others → Myocardial toxicity → Change in gene expression → Peripheral vasoconstriction → Sodium & water retention → Remodeling and progressive worsening of LV function → Heart failure symptoms → Morbidity and mortality

RAAS (Renin-Angiotensin-Aldosterone System)

Activation of AT1 receptors by angiotensin
- Vasoconstriction
- Sodium retention
- Increased aldosterone release
- Increased cellular growth
- Increased sympathetic nervous activity

Natriuretic Peptide System

ANP, BNP
- Vasodilation
- Sodium excretion
- Decreased aldosterone levels
- Inhibition of RAAS
- Inhibition of sympathetic nervous activity
- Antiproliferation of vascular smooth muscle cells

ANP = atrial natriuretic peptide, AT1 = angiotensin 1, BNP = endogenous B-type natriuretic peptide
Physiology of Endogenous hBNP

- Hemodynamic  
  - (balanced vasodilation)
  - veins
  - arteries
  - coronary arteries

Neurohumoral  
- aldosterone
- endothelin
- norepinephrine

Cardiac  
- lusitropic
- antifibrotic
- antiremodeling

Renal  
- diuresis
- natriuresis

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)
  - Vasoconstrictor 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 the Immune System

- Heart failure causes immune activation of pro-inflammatory cytokines:
  - Tumor Necrosis Factor (TNF-α), Interleukin (IL-1, IL-6)

- These are over-expressed in the systemic circulation and myocardium. Sustained over-expression contributes to central and peripheral manifestations

- Effects of pro-inflammatory cytokines on left ventricular function:
  - Exert negative inotropic effect
  - Induce abnormalities in cardiac metabolism
  - Promote myocardial remodeling
  - Cardiomyocyte hypertrophy
  - Necrosis and Apoptosis (programmed cell death)
  - Endothelial dysfunction
Compensatory Mechanisms in HF

1. Tachycardia and increase contractility
2. Increase preload from the release of renin and aldosterone
3. Vasoconstriction
4. Ventricular hypertrophy
5. Remodeling

Cardiac Remodeling

- Damage/insult occurs to the muscle followed by inflammation
- Neurohormonal (NH) activation occurs increasing ventricular pressure, volume, and peripheral vasoconstriction
- Heart changes in structure (dimensions, mass, shape) and function
- NH activity continues causing further structural & functional changes

Cardiac Reverse Remodeling

- Many factors influence the time, course, & extent of remodeling:
  1. Severity of insult
  2. Secondary events
  3. NH activation
  4. Treatment therapies
- Improvement in ventricular mechanics & function influenced by:
  1. Controlling risk factors
  2. Optimal drug & device therapies
Cardiac Vocabulary

Chronotropy: RATE at which the heart contracts
   – Important when looking at cardiac output

Inotropy: STRENGTH at which the heart contracts

Normal Cardiac Function

Cardiac Output = HR x SV

Cardiac Output is the volume of blood pumped per minute, measured by the following equation:

\[
CO = SV \times HR
\]

- CO is expressed in L/min (normal ~5L/min)
- CI is the output indexed to body size
- SV is stroke volume ejected per beat
- HR is the number of beats per minute
Impacting Hemodynamic Components

Heart Rate is...

Increased:
• Beta stimulation
• Increase demand
  – Fever, exercise, volume depletion

Decreased:
• Beta blockade
• Vagal stimulation
• Decrease metabolic demand
  – Cooling, paralyze, sedation

Stroke Volume (SV)

Determined by 3 factors:

Preload, Afterload, and Contractility.

– Preload: the VOLUME of blood that the ventricle has available to pump
– Contractility: the FORCE that the muscle can create at it's given length
– Afterload is the arterial pressure or RESISTANCE against which the muscle will contract.

These factors establish the volume of blood pumped with each heart beat.

Cardiac Vocabulary

Preload: the muscle length prior to contractility, and it is dependent on diastole (or end diastolic volume...EDV)
  – Ventricle fills during this time

• The most important determining factor for preload is venous return (in other words your JVP or CVP).
  – Think of it in terms of volume but don't forget the ability of the heart to eject it!
### Frank-Starling Law

- Increased end-diastolic fiber (sarcomere) length, volume, and pressure (increased preload) yields increased cardiac output.
- This is a physiologic mechanism to allow for the CO to be responsive to increased activity.
- However, excessive preload (as found in HF) exhausts the Frank-Starling mechanism, such that further increase in preload no longer increases cardiac output.

### Cardiac Vocabulary

**Afterload:** the tension (or the arterial pressure) against which the ventricle must contract. (SVR)

- If arterial pressure increases, afterload also increases.
- Increased VASOCONSTRICTION also increases Afterload.
- Afterload for the left ventricle is determined by aortic pressure (systemic Blood pressure and Systemic vascular resistance or SVR).
- Afterload for the right ventricle is determined by pulmonary artery pressure. (hence the term pulmonary hypertension!)

### Impacting Hemodynamic Components

**Afterload is...**

**Increased:**
- Volume overload
- Hypertension
- Cardiogenic shock
- Vasoconstriction

**Decreased:**
- Vascular collapse
- Vasodilatation
- Blocking of angiotension receptor's
Impacting Hemodynamic Components

Contractility is...

Increased:
- Inotropy
- Decrease SVR and systemic BP

Decreased:
- Increase SVR along with systemic BP
- Hypovolemia

Cardiac Output

Determined by:
1. Stroke volume:
   - preload: force used to stretch heart muscle and directly related to contraction
   - afterload: resistance the heart must overcome
   - contractility: amount of myofibril shortening

2. Heart Rate: controlled by autonomic nervous system

Clinical Presentation

- Symptoms vary widely
- Symptoms may be vague to severely acute
- Symptoms of HF can be similar to those of other conditions, i.e. COPD exacerbation
HF Symptoms

- Fatigue or tiredness
- Rapid weight gain, 3 lbs overnight or 5 lbs in 2 days
- Shortness of breath
- Use more pillows to sleep (orthopnea)
- Wake up short of breath at night (PND)
- Sleeps in recliner
- Frequent coughing
- Increased abdominal girth
- Early satiety, lack of appetite and/or nausea
- LE edema, swollen ankles, legs, and/or abdomen
- Decreased exercise tolerance
- Increased heart rate

Other Symptoms of HF

- Nonproductive cough - worse when lying flat
- Dizziness
- Palpitations
- Chest pain
- Syncope
- RUQ pain/discomfort
- Nausea/early satiety
- Snoring/apneic episodes

Common Presenting Symptoms
Divided into 2 Categories

Excessive Fluid Accumulation:
- Dyspnea
- Edema
- Hepatic congestion
- Ascites

Decrease in Cardiac Output:
- Fatigue
- Weakness
Staging Heart Failure: Focus on Disease Progression

<table>
<thead>
<tr>
<th>Stage</th>
<th>Patient Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>High risk for developing heart failure (HF)</td>
</tr>
<tr>
<td>B</td>
<td>Asymptomatic HF</td>
</tr>
<tr>
<td>C</td>
<td>Symptomatic HF</td>
</tr>
<tr>
<td>D</td>
<td>Refractory end-stage HF</td>
</tr>
</tbody>
</table>

- Marked symptoms at rest despite maximal medical therapy (eg, those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions)
- Known structural heart disease
- Shortness of breath and fatigue
- Reduced exercise tolerance
- Previous MI
- LV systolic dysfunction
- Asymptomatic valvular disease
- Hypertension
- CAD
- Diabetes mellitus
- Family history of cardiomyopathy
- High risk for developing heart failure (HF)
- Asymptomatic valvular disease
- LV systolic dysfunction
- Asymptomatic HF
- Previous MI
- Hypertension
- CAD
- Diabetes mellitus
- Family history of cardiomyopathy

Classification of Heart Failure: Comparison Between HF Stages and NYHA Functional Class

<table>
<thead>
<tr>
<th>ACC/AHA HF Stage</th>
<th>NYHA Functional Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>None</td>
</tr>
<tr>
<td>B</td>
<td>I - Asymptomatic</td>
</tr>
<tr>
<td>C</td>
<td>II - Symptomatic with moderate exertion</td>
</tr>
<tr>
<td>D</td>
<td>III - Symptomatic with minimal exertion</td>
</tr>
<tr>
<td>D</td>
<td>IV - Symptomatic at rest</td>
</tr>
</tbody>
</table>

Evaluation of the HF Patient

- Patient history
- Physical Exam
- Diagnostic tests
A thorough history and physical examination should be obtained/ performed in patients presenting with HF to identify cardiac and noncardiac disorders or behaviors that might cause or accelerate the development or progression of HF.

In patients with idiopathic DCM, a 3-generational family history should be obtained to aid in establishing the diagnosis of familial DCM.

Volume status and vital signs should be assessed at each patient encounter. This includes serial assessment of weight, as well as estimates of jugular venous pressure and the presence of peripheral edema or orthopnea.

Components of the Health History

1. Biographical data
2. Reason for seeking care
3. Present health or history of present illness
4. Past history
5. Review of systems
6. Social history
7. Family history

1. Biographical Data

- Name, address, age, DOB, birthplace, gender, marital status, race, ethnic origin, occupation
- Language spoken
- Communication needs
- Source of history
  - Record who furnishes information
  - Record if the patient seems willing and reliable
  - Note any special circumstances
2. Reason for Seeking Care

- Brief statement in the person’s own words that describes the reason for the visit
  - “Title of the story”
- It states 2 things:
  - Symptom
  - Sign
- Things to watch for:
  - It is not a diagnostic statement
  - Watch for self-diagnosing
  - Patient’s that list reasons for seeking care – which one is the most important?

What is a Symptom?

- Subjective sensation the person feels from the disorder
- Example: chest pain, shortness of breath.

What is a Sign?

A sign is an objective abnormality that can be detected on physical exam or in lab reports
3. History of Present Illness

• Includes age, sex, race & occupation

• Well patient’s will have a short HPI
  – Short statement about their general health

• Ill patients require further description that reviews:
  – Chronologic record of reason for seeking care
  – Time symptom first started
  – Give details of all symptoms concerned in the illness including location, character, severity, duration, intermittency, and radiation of pain. Describe factors making pain worse of better

Characteristics of any Symptom

• Location
  – Specific

• Quality
  – Requires descriptive terms

• Severity
  – Quantifies the sign or symptom

• Timing
  – Onset, duration, frequency

Characteristics of any Symptom Continued

• Setting
  – Where was patient
  – What brings it on

• Aggravating or relieving factors
  – What makes it worse
  – What relieves it

• Associated factors
  – Are there any other symptoms

• Patient’s perception
  – Effecting ADL
PQRSTU: A Way to Remember how to Define the Characteristics of Symptoms

- P: Provocative or Palliative
- Q: Quality or Quantity
- R: Region or Radiation
- S: Severity Scale
- T: Timing
- U: Understanding patient perception

4. Past Medical (Health) History

- Childhood illnesses
- Accidents or injuries
- Chronic illnesses
- Hospitalizations
- Operations
- Obstetric history
- Immunizations
- Last Examination date
- Allergies
- Current medications

Relevant Past Medical & Surgical History

- Prior history of HF or myocardial infarction
- CABG/PCIs
- Valvular repairs/replacement
- Device therapy
- HTN
- OSA
- Anemia
- ETOH
5. Review of Systems

- ROS addresses the following:
  - Evaluates the past & present health state of each body system
  - Evaluates health promotion practices

6. Social History

- Place of birth and residence
- Marital status
- Habits including:
  - Self-esteem, self-concept
  - Activity/exercise:
    - Ability to perform self-care or activities of daily living (ADLs)
    - Activities needed for independence, ie grocery shopping, laundry, nutrition, managing finances, cooking (ADLs)
  - Sleep/rest
  - Nutrition/elimination
  - Interpersonal relationships/resources:
    - Intimate partner violence
  - Spiritual resources
- Occupation: past and present, service in military
- Environmental factors:
- Name/contact info of physicians

7. Family History

- Health or age of death & cause
- Close family members:
  - Contact with communicable diseases
Family History

- Specifically address the following:
  - Family history of heart disease
  - High blood pressure
  - Stroke
  - Diabetes
  - Blood disorders
  - Cancer
  - Sickle-cell anemia
  - Arthritis
  - Allergies
  - Obesity
  - Alcoholism
  - Mental illness
  - Seizure disorder
  - Kidney disease
  - Tuberculosis

Case Study 1

Defining HF, HF symptoms, History & Physical Exam, Class/Stage of HF, ECHO, HF Medical Therapy

Reason for Seeking Care (Chief Complaint):
"Shortness of breath is worse."

History of Present Illness:
(HPI is a chronological record of the reason for seeking care.)
DF has experienced a 20 pound weight gain over the past 2–3 months. During this time he has developed c/o worsening fatigue, orthopnea, PND, decreased appetite, abdominal distention and lower extremity edema. Prior to 3 months ago he was in his usual state of health. He denies any chest pain, presyncope or syncope. He was able to walk 1 mile daily but is only able to walk 2 blocks without exertional symptoms of SOB and worsening fatigue.

Past Medical History:
Dyslipidemia with high LDL & low HDL
HTN for 35 years
COPD
MI at age 64 and a 2nd MI at age 68.

Past:
CABGs x 3 in 2004
Cholecystectomy in 2010

Allergies:
No known drug allergies

Social History:
Widowed, supportive daughter, reformed tobacco user smoking 1PPD x 20 years, no ETOH or illicit drug use. 2-3 cups of coffee daily. Retired accountant. No routine exercise. Sleeps 6 hours.
Focused Physical Exam

• Vital Signs
• Neck
• Pulmonary
• Cardiovascular
• Abdominal
• Extremities

Vital Signs

• Blood pressure
• Orthostatic blood pressure
• Heart rate
• Respiratory rate
• Temperature

• Blood pressure
  – Measure of the heart’s ability to pump & indicator of degree of antihypertensive from medications
  – Orthostatic readings helpful in determining dehydration & over-diuresis

• Heart rate
  – Helpful in indicating arrhythmias
  – Resting tachycardia may be an indicator of poor prognosis
  – Bradycardia may indicate heart block or over medication
Vital Signs

• Blood pressure
  – Measure of the heart’s ability to pump & indicator of degree of antihypertensive from medications
  – Orthostatic readings helpful in determining dehydration & over-diuresis

• Heart rate
  – Helpful in indicating arrhythmias
  – Resting tachycardia may be an indicator of poor prognosis
  – Bradycardia may indicate heart block or over medication

Neck Examination

• Carotid bruits
• Jugular venous distention
• Abdominojugular reflux

Abnormal Lung Sounds

Rales or crackles are lung sounds that are brief, discrete, nonmusical sounds with a popping quality. Typically heard in the bases.
Focused Cardiac Exam

- Point of Maximal Impulse (PMI)
- Rhythm (Regular/Irregular)
- Rate (Fast/Slow)
- Extra heart sounds (S3, S4)
- Murmurs (new or existing)

Auscultation

Stethoscope dynamics:
- Bell: allows low frequency sounds to be heard when light pressure is applied
- Diaphragm accentuates higher frequency sounds when enough pressure is applied

Characteristics of S1

- First heart sound
- Beginning of systole; ventricular volume is maximum
- Events surrounding closure of the mitral and tricuspid valves
- Heard best with the diaphragm at the apex
Characteristics of S2

- Second heart sound in cardiac cycle
- End of systole
- Events surrounding the closure of aortic & pulmonic valves
- Best heard at the base of the heart using the diaphragm

Defining a Third Heart Sound

Third heart sound (S3)
- is an extra low-pitched sound
- heard after S2 resulting from decrease ventricular compliance with conditions of volume overload
- heard best at the apex or left lower sternal border. It resembles the rhythm of Ken-tuc-ky also described as lub-dub-da

S1- S2- S3

Defining a Fourth Heart Sound

Fourth heart sound (S4)
- heard late in diastole, heard immediately prior to S1
- occurs when the atria contract late in diastole
- soft sound, heard best at apex (left ventricular origin)
- caused by atrial systole ejecting blood into a non-compliant ventricle

S4- S1- S2
Heart Murmurs

- Originate from failure of heart valves to open adequately (stenosis) or failure to close (incompetence causing regurgitation)
- Divided into benign or pathologic
- Sounds that are heard as harsh (AS), rumble (MS), blowing (AR)

Grading Murmurs

- Grade I: very, very faint
- Grade II: quiet but immediately heard
- Grade III: moderately loud without a thrill
- Grade IV: loud with a thrill
- Grade V: very loud with a thrill
- Grade VI: audible without a stethoscope

Abdominal Exam

- Right upper quadrant tenderness
- Enlarged, palpable liver border > 3 fingerbreadths below the costal margin
- Pulsatile liver
- Ascites
Evaluation ofExtremities

- Decreased strength of arterial pulses
- Cyanosis
- Peripheral edema
- Muscle wasting

Summary of Common Objective Findings on a HF Physical Exam

**HEENT:** JVP, there are many wave forms in the neck and tricuspid regurgitation can alter it

**CV:** PMI displaced, S3 heard at the apex, (split S2 heard at sternum and can disappear with a deep breath), Holosystolic Murmur at the apex in the patient with dilated CM

**Resp:** May or may not be crackles, are they using accessory muscles, are they able to complete a sentence

**ABD:** HJR, Hepatomegaly, Ascites from RV failure

**Ext:** Edema or not, (don’t forget to look in sacral area) always feel don’t just look, what is the temperature of the extremity

Goals of Diagnostic Testing

- Establish Diagnosis
- Identify Pathology
- Treat Cause
Laboratory Data Evaluation

<table>
<thead>
<tr>
<th>Lab Test</th>
<th>Assessing for Possible Precipitating Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Troponin</td>
<td>Ongoing ischemic heart injury</td>
</tr>
<tr>
<td>Complete Blood Count</td>
<td>Anemia or infection</td>
</tr>
<tr>
<td>Liver Function Tests</td>
<td>Poor hepatic function</td>
</tr>
<tr>
<td>D-dimer</td>
<td>Pulmonary embolus</td>
</tr>
<tr>
<td>Thyroid Profile</td>
<td>Hyper- or hypothyroid disorders</td>
</tr>
<tr>
<td>Renal Function Studies</td>
<td>Renal dysfunction/failure</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>Urinary tract infection, prostatitis</td>
</tr>
<tr>
<td>Arterial Blood Gases</td>
<td>Hypovolemic, comorbid lung, pulmonary edema</td>
</tr>
</tbody>
</table>

Cardiac BNP Levels

- 32-amino acid peptide secreted primarily from the ventricles of the heart in response to stretch and increased volume in the ventricles
- BNP < 100pg/ml negative
- BNP > 500pg/ml positive
- BNP levels correlate with:
  - LV end-diastolic pressure and volume
  - NYHA classification
  - Trends in BNP more meaningful