BRIGHAM HEALTH

BRIGHAM AND WOMEN'S HOSPITAL

Department of Rehabilitation Services
Physical Therapy

Standard of Care: Cardiac
Inpatient Physical Therapy Management of the Surgical and Non-Surgical Patient with Cardiac Disease

Case Type / Diagnosis: This standard of care applies to patients with cardiac disease including, but not limited to: coronary artery disease (CAD), myocardial infarction (MI), valvular disease, cardiomyopathy (CMP), heart failure (HF), arrhythmias, pulmonary hypertension, pulmonary embolisms/deep vein thromboses, and congenital heart disease. It also applies to patients status post (s/p) cardiac surgical and non-surgical procedures including, but not limited to: coronary artery bypass graft (CABG), valve replacement or repair, percutaneous coronary intervention (PCI), percutaneous transluminal coronary angioplasty (PTCA), aortic aneurysm repairs, radiofrequency ablation (RFA), and transcatheter valve repairs. This standard of care does not specifically address patients who are s/p mechanical circulatory support device (MCSD) or s/p orthotopic cardiac transplant (OHT). For standards associated with Physical Therapy management of these patient populations, please refer to the respective standards of care.

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Cardiac Pathologies:

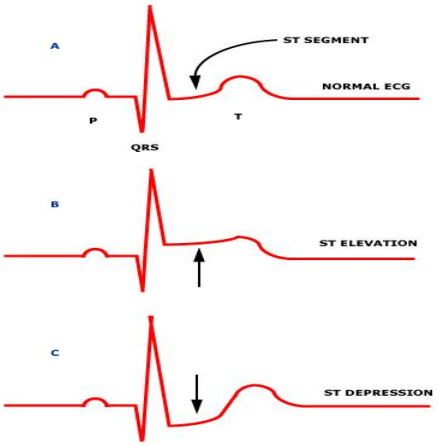
Coronary Artery Disease and Myocardial Infarction

Definitions:

- Coronary Artery Disease (CAD), also known as atherosclerotic heart disease, is a progressive disease resulting in lipid deposits in coronary arteries resulting in coronary artery stenosis and ischemia.
- Acute Coronary Syndrome: an umbrella term used to describe events and symptoms related to cardiac ischemia
  - Angina: typically presenting as chest pain, pressure, or discomfort. It can also present as jaw, back, neck, or left arm pain or stiffness. Can be mistaken for indigestion. Defined as stable, unstable, or variant angina. Symptoms can be masked by diabetes (silent MI) and can also present in atypical ways in women

- *Stable angina (angina pectoris):* presents during activity/exercise at predictable heart rates and blood pressures (known as rate pressure product) and is relieved by coronary vasodilators or rest. Typically managed by medication that reduces the cardiac workload (i.e. vasodilators), stress management, and activity pacing.
- *Unstable angina*: can occur at rest, spontaneously, or with reduced workloads, and may not be easily managed by rest or medication. Typically indicates a blockage with intervention required, either percutaneous or surgical
- *Variant angina (Prinzmetal's Variant Angina):* typically occurs in the younger population due to coronary artery vasospasm and most often occurs at rest or overnight.[1]

- Myocardial infarction (MI): typically classified as ST segment elevation (STEMI) or non-ST segment elevation (NSTEMI), determined by 12-lead ECG, related to elevation of the ST segment above the isoelectric line
  - *STEMI*: An ST-elevation myocardial infarction (STEMI) is a result of the complete blockage of a coronary artery, therefore typically has a higher risk of death or disability due to increased myocardial cell damage/death. Tissue damage tends to extend through the full depth of the cardiac wall local to the area of ischemia
  - *NSTEMI*: A non-ST elevation myocardial infarction (NSTEMI) is defined as when a partial blockage of a coronary artery, though severity within this group can vary pending the level of occlusion.[2]



A. Normal Sinus Rhythm
B. STEMI
C. NSTEMI

Chart Review:

- Treatment: can include pharmacologic therapy, percutaneous intervention (i.e. stenting), or coronary artery bypass grafting (see CABG below)
- MI rule out (R/O): Patients admitted with acute coronary syndrome (ACS), chest pain (angina), or suspected MI are not appropriate for PT until they have either been ruled out for a MI event, or until they are medically/surgically managed. During a R/O for MI, three sets (one every 8 hours) of cardiac enzymes (creatinine kinase [CK-MB isoform], troponin [Tn-I]) are drawn and electrocardiograms (ECGs) are performed.[1,3]
  - Creatinine Kinase (CK-MB isoform): Creatinine phosphokinase is an enzyme released after cell injury or death of cardiac muscle. CK-MB measurement can assist in the diagnosis of an MI, estimate the size of infarction and evaluate the occurrence of re-perfusion. An early peak and rapid clearance from the blood can indicate re-perfusion. Values may also be elevated due to other reasons. Communication with the medical team should occur when there is question of the appropriateness of PT.

- Brigham and Women's Hospital has started using a troponin assay which is more sensitive and specific for myocardial tissue necrosis. The numerical value associated with myocardial necrosis has changed.
  - Rule-in: troponin ≥ 52 ng/L, OR Δ >5 ng/L
  - Rule-out: troponin <10 ng/L for women and <12 ng/L for men, AND Δ <3 ng/L
  - If after q1h troponin neither rule-in or rule-out, obtain 3h troponin
- ECGs: Electrocardiography changes are almost always present in the event of an MI. Early tracings typically show peaked or "hyperacute" T waves. As the MI progresses, this is followed by ST-segment depression or elevation, Q-wave development and lastly, T-wave inversions. Presentation can vary, with this progression occurring over a few hours to several days, with Q-waves only developing in 30-50% of acute MIs.
  - It is important to note that ST-segment depression or elevation can also be caused by coronary artery spasm, electrolyte abnormalities, left ventricular hypertrophy, interventricular conduction delays (BBB), atrial fibrillation or flutter, Digoxin and pacemakers.

PT Examination:

- Determine medical stability:
  - If the patient rules in for a MI, care must be taken to determine when a patient is stable to participate in a PT examination or intervention. In general, it can be expected that the patient may resume progressive monitored activity once cardiac biomarkers have peaked and down trended for two sets and/or once the patient is hemodynamically stable at rest.
  - On occasion, once a patient has undergone intervention to treat the myocardial ischemia (i.e.: heart catheterization with stenting), no further biomarkers will be drawn, removing the ability to watch for down trending levels. In this event, communication with the team is vital to determine whether it is medically appropriate and safe to proceed with physical therapy evaluation and intervention.
  - Of note, in some instances, cardiac enzymes may be elevated e.g., cardiac stress related to volume overload (heart failure) or tachy-arrhythmias, or rise from previously lower values e.g., after a cardiac catheterization, and may not indicate a new MI event. Clarify appropriate activity orders with the responding clinician.
- Assessment of endurance
  - Following ACSM Guidelines for Exercise Testing and Prescription[4], it is appropriate to evaluate a patient's exercise tolerance soon after an MI occurs. Submaximal exercise tests (such as a 6MWT, see Appendix 1) are recommended before hospital discharge at 4-6 days after acute MI. Submaximal exercise testing can be used to determine an appropriate exercise prescription and guide the medical team on the effectiveness of their interventions, based on a patient's hemodynamic response.
  - See endurance/exercise testing.

PT Intervention: Exercise Testing and Prescription

- Refer to PT Intervention/Aerobic Exercise Training for information regarding exercise testing and prescription in the inpatient setting for a patient with cardiac dysfunction. A home walking program should be established based on results of submax exercise test prior to discharge.
- Post MI, a referral to a cardiac rehab program is highly recommended. Patients should be educated on purpose of cardiac rehab, locations near their home (found on T-drive) and provided with a referral prior to discharge.

Valvular Disease

Definitions:

- Any of the four valves of the heart can become diseased or dysfunctional for various reasons, but commonly seen reasons include congenital abnormalities, progressive calcification/atherosclerotic changes, rheumatic heart disease, infective endocarditis, connective tissue disorders, and changes associated with heart failure and ventricular dilation. Valve disease is typically diagnosed via echocardiogram which shows alterations in a person's blood flow.

Common terms of dysfunction/disease of heart valves:

- *Insufficiency (or regurgitation)*: valves do not close properly, allowing blood to flow in the reverse direction during ventricular systole or diastole. Consequences of insufficiency include chamber hypertrophy and retrograde chamber dilation.
    - Example: mitral insufficiency (mitral regurgitation): the mitral valve does not close properly during systole, allowing leakage of blood backwards from the left ventricle into the left atrium; it is the most common type of valvular heart disease.
        - Mitral Valve Prolapse: the cusps of the mitral valve become enlarged and floppy, bulging backward into the left atrium
- *Stenosis*: narrowing of the opening of the valve, often due to progressive calcification or from rheumatic heart disease. This narrowing reduces forward blood flow, leading to progressive dilation and hypertrophy of the chamber preceding the valve and potentially a reduction in cardiac output
    - Aortic valve stenosis is common and is classified in severity by a grading system based on velocity of blood flow, pressure gradients from the left ventricle to the aorta, and the aortic valve area. Cardiac output is preserved and the patient is often asymptomatic until the stenosis is severe

Aortic Stenosis	Area	Mean gradient	Jet velocity
Mild	>1.5cm^2	<25 mmHg	<3.0 m/s
Moderate	1.0 – 1.5cm^2	35-40 mmHg	3.0 – 4.0 m/s
Severe	<1.0cm^2	>40 mmHg	>4.0 m/s

    - Higher pressures on the left side of the heart lead to greater chance of valvular dysfunction in the mitral and aortic valves, increasing the workload on the heart. The heart can compensate for a time with chamber hypertrophy, dilation, and

systemic BP adjustments however ultimately symptoms of heart failure can develop.[3]

Chart Review:

- **Medical History:**
  - Etiology of valvular dysfunction
  - Associated comorbidities
  - History of management
- **Chart Review:**
  - Reason for admission: typically related to progression of symptoms of heart failure, admitted for medical management. Can be admitted for elective cardiac surgery (see below).
  - Type of valve dysfunction and severity. See grading system above
  - Recent imaging including echo or right heart catheterization to evaluate for progression
  - Management plan, including changes in medication and/or progression to valve repair or replacement.
    - Diuretics for volume management or other plans for pharmacologic management.
    - Valvuloplasty (for aortic stenosis) – use of femorally inserted balloon catheter to separate calcified leaflets or to stretch the annulus. This is a palliative measure for patients not eligible for valve replacement
    - Cardiac surgery
    - Percutaneous valve replacement
      - Aortic valves can be replaced transfemorally, particularly in older, frail patients, due to risks associated with cardiac surgery. See TAVR below. On occasion, patients undergo mitral valve replacements with a similar technique

PT Examination:

- Prior level of function including most recent limitations. It is important, as with patients with heart failure, to establish a baseline level of activity tolerance, both when a patient has been feeling well and when they are feeling poorly, most likely due to symptoms related to volume overload. See CHF section for further information
- Lung and heart sounds, listening for murmurs, pulmonary congestion, both pre and post activity to assess for response to exertion
- Refer to endurance/exercise testing

PT Intervention:

- Refer to PT Intervention/Aerobic Exercise Training for information regarding exercise testing and prescription

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Heart Failure/Cardiomyopathy

Definition/Pathology:

- The American Heart Association defines heart failure (HF) as a chronic, progressive condition in which the heart muscle is unable to pump sufficient blood to meet the body's metabolic demand. Heart failure is caused by conditions that damage or weaken the heart, including coronary artery disease, myocardial infarction, hypertension, valvular disease, cardiomyopathy, congenital heart defects, heart arrhythmias, myocarditis or other chronic conditions such as diabetes, HIV, hyperthyroidism, hypothyroidism, or a buildup of iron (hemochromatosis) or protein (amyloidosis).[5]
- There are four types of heart failure, categorized by either a structural perspective (Left-sided vs. Right-sided) or functional perspective (Systolic and Diastolic):[5-7]
    - ***Left-sided heart failure*** *is caused by failure* of the left ventricle to adequately pump blood to periphery, which can lead to a backup of fluid on the lungs and shortness of breath. Left ventricular failure and the resultant pulmonary congestion is referred to as Congestive Heart Failure (CHF)
    - ***Right-sided heart failure*** is caused by failure of right ventricle to pump deoxygenated peripheral blood to the lungs, which can lead to back of fluid into the abdomen, legs and veins. Right-sided heart failure can occur because of progressive left-sided heart failure, pulmonary hypertension, valvular disease or from pulmonary disease.
    - ***Systolic heart failure*** is caused by impaired contractile function of the ventricle resulting in reduced stroke volume, cardiac output and ejection fraction
    - ***Diastolic heart failure*** is caused by impaired ventricular relaxation during diastole leading to impaired filling and causes a reduced stroke volume and cardiac output but the ejection fraction remains the same. Impaired ventricular relaxation is often related to ventricular stiffness which is associated with older age and amplified by HTN, diabetes mellitus and kidney disease.
- When treating a patient with heart failure, it is important to understand the type and cause of heart failure to best design your education and treatment plan.
- Heart failure is typically classified based on the severity of the patient's symptoms and the effect the symptoms have on physical activity. The table below shows the most commonly used classification system, the New York Heart Association Functional Classification.[8] This classification places patients in stages I-IV based on their physical activity limitations.

NYHA Class	Level of Clinical Impairment
I	No limitation of physical activity. Ordinary physical activity does not cause undue breathlessness, fatigue or palpitations.
II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in undue breathlessness, fatigue or palpitations.

III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in undue breathlessness, fatigue or palpitations.
IV	Unable to carry on any physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.

Of note, *Cardiac amyloidosis* (CA) is a unique and rare form of heart failure that causes thickening of the heart walls and leads, most commonly, to diastolic heart failure. Due to the way amyloid affects the heart and other body systems, patients present differently than those with more common forms of heart failure and therefore, our physical therapy approach should be different. Patients with CA often have an impaired hemodynamic response to activity or exercise, including exercise-induced syncope.[9] If you are treating a patient with this diagnosis, be sure to consult a team lead or cardiopulmonary specialist when developing your plan of care. The remainder of this section will focus on the more common presentation of heart failure.

Chart Review:

- **Medical History**
  - Cause of heart failure and categorization based structure or function (e.g. - ischemic vs. non-ischemic, congenital, systolic vs. diastolic, preserved vs. reduced ejection fraction)
  - Onset and duration of HF symptoms and NYHA Stage
- **Hospital Course**
  - Reason for admission. Patients are often admitted due to symptoms related to increased volume status, e.g. - progressive dyspnea on exertion, increased lower extremity edema and decline in activity tolerance
  - Take note of any relevant lab values, imaging or tests. Below are common tests/measures done by the medical team in a patient with CHF:
    - **Chest X-ray:** Determines the level of pulmonary congestion, common findings include interstitial edema, atelectasis and pleural effusions.[10]
    - Echocardiogram
    - **Lab Values:** Many lab values and electrolytes are affected by fluid retention that occurs secondary to the inadequate cardiac output in CHF. Fluid overload can lead to impaired renal function, hepatic function and altered electrolytes.[10]
      - Below are common changes in lab values found in CHF:
        - ↑ BNP, protein in urine (Albumin), BUN/Cr, $PaCO_2$, liver enzymes
        - ↓ $PaCO_2$, SpO_2, Sodium
        - ↑/↓ Potassium- may be increased or decreased
          - Severe hypokalemia (< 2.5 mEq/L) or hyperkalemia (>5 mEq/L) should be a precaution to PT intervention due to the increased risk for cardiac

arrhythmias. Be sure to collaborate with medical team prior to intervention.
- Troponin can be elevated with heart failure, often due to increased demand on the heart rather than acute myocardial infarction. Be sure to trend troponin and look at clinical picture to determine appropriateness for PT intervention.

- Patients with heart failure are at risk for gout due to the fluid fluctuations, use of diuretics and potential for renal dysfunction.[11] Therefore, blood work may reveal hyperuricemia and the patient may present with joint pain and inflammation.

- Cardiac Catheterization**:** may be done to determine the cause of heart failure and potential for intervention (e.g. - valve repair/replacement, CABG). Additionally, cardiac catheterizations can provide the medical team with accurate information regarding ejection fraction, cardiac output, stroke volume and filling pressures to assist in prognosis and to optimize medical management.[12]

- Medications*[13]
  - The focus of drug therapy in heart failure is on optimizing preload, improving cardiac pumping performance and reducing afterload. Below are common medications seen in patients with heart failure:
    - **Pre-load reducing agents:** Diuretics
    - **Cardiac Pumping Enhancement:** Ionotropic Agents
    - **Afterload Reducers:** vasodilators (i.e. ACE inhibitors)
    - **Beta-Blockers (BB):** are first line treatment (along with ACE inhibitors) for patients with systolic heart failure, even when asymptomatic. Prescription of a BB improves symptoms, reduces hospitalization, and enhances survival.[14-15] The 3 FDA-approved BB to treat HF include Bisoprolol (Zebeta), Carvedilol (Coreg), Metoprolol (Toprol)[16]
  - The patient may also be on an anticoagulant if they have a comorbid arrhythmia, such as Atrial Fibrillation. Common anticoagulants seen in heart failure include: aspirin, warfarin, rivaroxaban (Xarelto)
  - *See the Appendix 2 for relevant medications, side effects and physical therapy considerations for the categories/medications above.
- Determine Medical Stability - Some factors to consider related to heart failure include: the patient's level of dyspnea, resting vital signs, EKG pattern and any significant changes in lab profile (CBC, electrolytes, kidney function)
  - Patients admitted to the hospital are often in **Acute (or Advanced) Decompensated Heart Failure (ADHF)**, which is defined as "the sudden or gradual onset of the signs or symptoms of heart failure requiring unplanned office visits, emergency room visits, or hospitalization."[17] Care must be taken as a therapist when designing exercise programs, endurance programs and plan of care for patients in ADHF. If patients are hospitalized with ADHF, they may be

undergoing medication changes or introduction of inotropes requiring close monitoring. These patients may have a PA line, A-line, PICC and/or CVC.
    - Much of the research is focused on those with stable heart failure, therefore, the therapist must consider the *cause* of the heart failure, *severity* of HF symptoms (e.g. dyspnea and fatigue) and *hemodynamic response* to exertion when designing interventions and plan of care for a patient in potentially decompensated heart failure. Any questions regarding the medical stability of your patient should be addressed with the medical team, team lead and/or cardiopulmonary specialist.

PT Examination:

- Determine prior level of function and impact of heart failure symptoms
    - It is important to understand a patient's lifestyle and past experiences with HF management. Below are some good questions to ask a patient:
        - How far can you walk before you need to rest? What causes you to rest? SOB? Muscle weakness?
        - Have you been to cardiac rehab?
        - Do you exercise regularly?
    - Objective Measurements of Quality of Life - *it may not be realistic to perform entire outcome measure in this setting but using the questions on these measures may help guide your treatment, plan of care and goals.*
        - Kansas City Cardiomyopathy Questionnaire - quantifies the degree of physical limitations associated with heart failure
        - Minnesota Living with Heart Failure Questionnaire - total 50 or greater may indicate a greater potential for successful rehabilitation.
- Vital Sign Assessment
    - Be sure to monitor HR, SpO2, RR and BP at rest and with activity. Patients with heart failure are at risk for oxygen desaturation and impaired hemodynamics due to increased stresses on the heart, fluid overload and medication side effects.
    - Be sure to check telemetry before, during (if possible) and after a physical therapy session. Patients with heart failure are at increased risk for arrhythmias.[18] Additionally, patients are at risk for more life threatening ventricular arrhythmias (such as VT and VFib). Be sure to know of baseline arrhythmias and any new arrhythmias from this admission. Alert the medical team if an arrhythmia is noted during physical therapy evaluation or treatment.
- Cognition/Mental Status
    - Advanced heart failure and ionotropic medical support may have a negative impact on cognition, specifically memory, motor response time and speed of processing.[21]
        - In this case, you may want to consider the Mini Mental Status Examination or consult Occupational Therapy
- Integumentary
    - Patients in ADHF often present with lower extremity edema, which can be quantified using the Pitting Edema Scale (0 to 4+), documented in EPIC by

nursing or PT staff. It is important to consider how lower extremity edema is affecting physical function and can be addressed by physical therapy intervention, notably via imbalance, lower extremity fatigue, or gait deviations.

- Strength and Endurance
  - Use of MMT strength assessment to identify any obvious weakness. Many patients with heart failure will present with > ⅘ MMT strength, but will fatigue quickly. Therefore, it is recommended to use an outcome measure to quantify muscular endurance, such as: the 30 second sit to stand test or 5 Times Sit to Stand test
  - Quantify aerobic capacity and endurance. The results of a 6MWT can be used to assist in determining discharge recommendations, exercise prescription and prognosis. *See appendix 1 for patient instructions and appropriate performance of this test.
  - Be sure to quantify fatigue and dyspnea during functional mobility and ambulation using the 0-10 RPE and DOE scales. These scales can help determine a patient's tolerance for activity and ADLs and can assist in discharge planning, goal-setting and plan of care.
- Balance
  - Patients at significant risk for balance impairments are older adults with ADHF. This is due to the higher incidence of frailty in this population, leading to severe deficits in all domains of physical function: balance, mobility, strength and endurance.[21]
  - Use of standardized measures and gross observations of static and dynamic balance to determine fall risk and balance impairments that can be addressed in the therapist's plan of care.

PT Intervention:

- Endurance Training
  - Refer to PT Intervention/Aerobic Exercise Training for information regarding exercise testing and prescription
- Strengthening
  - It is recommended that patients with heart failure participate in a strengthening program as an adjunct to aerobic exercise. Initial resistance training should begin at a lower intensity, especially in the acute care setting, with close monitoring of symptoms. Be sure to educate the patient on proper technique and breathing to avoid valsalva. Below are the recommendations based on the updated literature:[22]
    - Mode: dynamic exercises targeting major muscle groups
      - Be sure to include postural exercises to optimize positioning for improved breathing and lower extremity muscle groups that correlate to function
    - Frequency: 2-3x/week

        - Intensity/Repetitions/Sets: 1 set of 10-15 reps to volitional fatigue or rated 3-5/10
            - Initial resistance training should begin at a lower load and higher repetitions. As tolerated the load can be increased with light weights or resistance bands, which will decrease the number of repetitions.
- Pacing/Energy Conservation[10]
    - Pacing and energy conservation techniques should be taught to avoid max fatigue or shortness of breath with activity and to maximize participation in desired activities. An analysis of all activities an individual performs helps to develop an inventory to set priorities and organize the individual's day.
    - Attention should be paid to activities that create fatigue or dyspnea.
- Patient/Family Education
    - Self-management techniques should be taught to manage the disease including:[10]
        - Signs and symptoms of decompensation (excessive SOB, fatigue, peripheral swelling, waking at night with dyspnea/cough)
        - Adherence to regular exercise program
            - Create a home exercise program based on level of function at discharge
            - Include family member or caregiver on instruction of home exercise plan to ensure compliance
- Referral to Cardiac Rehab. Patients must demonstrate 6 weeks of stable management of heart failure to be deemed appropriate for initiating care.

Arrhythmias

Definitions and PT considerations:

- Cardiac arrhythmias represent an abnormal cardiac conduction that can potentially lead to a decrease in cardiac output with activity, or at rest. Therapists should look at their patient's telemetry regularly as part of their pre-evaluation assessment and during treatment to become familiar with patient responses. There are several factors that the therapist should consider when deciding whether a patient with an arrhythmia is appropriate for PT intervention, including:
    - Acuity (how long this arrhythmia has been present)
    - Symptoms
    - Plans for management (what medications are being used, plans for electrolyte replacements, cardioversion or ablation)
    - Presence of a pacemaker or AICD.
    - *New or progressive arrhythmias during therapy should be noted and this information relayed to the medical team.*
- The following are additional clinical considerations for some specific arrhythmias to aid in deciding whether the patient is appropriate for physical therapy. The therapist should be aware that this is a list of some common arrhythmias but is not exhaustive. The therapist should discuss appropriateness of PT intervention with the medical team, team

lead and/or cardiopulmonary specialist in cases where the patient may be experiencing any arrhythmia they are not familiar with.

- **Atrial Fibrillation (AF):** AF is a disorganized depolarization of the atria resulting in an irregularly irregular heart rate and lack of effective atrial contraction.[23]
  - Identify heart rate via telemetry or identify pulse rate by manual inspection for one minute. Auscultation can also be used
  - Considerations for PT intervention should include:
    - Is the AF new?
    - How high is the HR elevated due to AF at rest? With activity?
    - Is the patient's BP affected by the arrhythmia?
    - Is the patient having any symptoms due to an altered HR/BP such as lightheadedness, shortness of breath, fatigue, exercise intolerance?
- **Heart Block (HB):** Watch for progression of heart block or alteration in rhythm in response to activity.
  - First-degree heart block: Prolonged PR interval. Generally, there are no precautions if the patient's HR and BP are stable.
  - Second degree heart block
    - Type I (Mobitz I): PR interval increases until a ventricular complex is dropped. This arrhythmia is frequently noticed with increasing activity.
    - Type 2 (Mobitz II): Several atrial contractions are needed to propagate a single ventricular contraction (i.e. 2:1). This arrhythmia can follow a Mobitz I or be a progression in a person with first degree HB.
  - Third degree (Complete heart block): There is a complete lack of synchrony between atrial and ventricles observed on the EKG, resulting in a greatly reduced ejection fraction and potential for hemodynamic instability. PT is deferred until the patient has a PPM. If the patient progresses to this rhythm during treatment they may become lightheaded or syncopal, this is a medical emergency and the patient needs immediate medical attention.
  - First and second degree heart blocks may be stable and asymptomatic, pacing sometimes required. Monitor patient throughout session
- **Premature Ventricular Contraction (PVC):** A PVC is a ventricular contraction initiated by an abnormal focus within the ventricle rather than via the sinoatrial node. PVCs are common, with a higher incidence observed in patients with cardiac health conditions or after cardiac surgery.[23] PVCs can be palpated or observed via ECG.
  - While generally benign, considerations should include:
    - Acuity
    - Frequency at rest and with activity

- Hemodynamic response
- Patient subjective response
- In general, an increase in frequency of PVCs with activity or exercise should be noted in your documentation and reported to the medical team as they can indicate increasing irritability in the heart in response to exertion

- **Ventricular Tachycardia (VT):** VT is defined as four or more consecutive premature ventricular contractions. VT can be palpated or observed via ECG. Patients may also report a fluttering sensation in their chest or complain of feeling lightheaded or dizzy. Sustained VT is a very serious arrhythmia that can result in greatly decreased cardiac output due to ventricular contraction without time for adequate refilling. Runs of VT are frequently seen in patients with heart failure and may be considered baseline for certain patients.
  - Consideration should include:
    - Acuity, frequency, and duration of the runs of VT during the hospitalization, particularly in a patient admitted with recurrent VT
    - Patient symptomatology (i.e., lightheadedness/dizziness, syncope, diminished exercise tolerance and/or confusion)
    - Triggers of VT if known – electrolyte imbalance, exertion
    - Current or planned pharmacological interventions including addition of IV antiarrhythmic, beta blockers, electrolyte repletion.
  - New onset of VT, long runs of VT and patient symptoms may indicate that the patient may not be clinically appropriate for PT.
- **Ventricular Fibrillation (VF):** VF is defined as absence of organized ventricular activity and presents as irregular undulations of varying contour and amplitude on ECG. There is no cardiac output and the patient will usually die within 3-5 minutes if a more normal cardiac function is not restored.[23]

Pulmonary Hypertension

Definition:

- Pulmonary hypertension (PH) is defined as an increase in resting mean pulmonary artery pressure (mPAP) of $\geq$ 25mmHg, measured via right heart catheterization. Normal mPAP can range between 14-20mmHg at rest.[24]
- Increased pressure in the pulmonary arteries can be due to changes in endothelial lining of the vasculature, leading to arteries that become narrowed, blocked, or destroyed.[25,26] Increased pressure can also result from hypoxic vasoconstriction, occlusion of the pulmonary vascular bed, or parenchymal disease with loss of vascular surface area.[27]
- Over time, increasing resistance in the pulmonary vasculature can lead to right heart dysfunction and failure with its resultant clinical picture
- **Classification:** Pulmonary hypertension is classified into 5 types, dependent upon the underlying cause. They are grouped based on clinical presentation, pathological findings, hemodynamics, and treatments strategies.[24]

Group	Clinical Classification
Group 1: ***pulmonary arterial hypertension*** & Group 1[1]: pulmonary veno-occlusive and/or pulmonary capillary hemangiomatosis	Idiopathic (or primary) Heritable (genetic mutation) Drug or toxin induced Associated with: • Connective tissue disease • Portal hypertension • Congenital heart disease
Group 2: pulmonary hypertension due to left heart disease – ***pulmonary vascular hypertension***	Left ventricular systolic and/or diastolic dysfunction Valvular disease Congenital/acquired left heart inflow/outflow tract obstruction and congenital cardiomyopathy Congenital/acquired pulmonary vein stenosis
Group 3: pulmonary hypertension due to lung diseases and/or hypoxia	COPD Interstitial lung disease (ILD) Other pulmonary diseases with mixed restrictive and obstruction patterns Sleep-disordered breathing Alveolar hypoventilation disorders Developmental lung diseases
Group 4: chronic thromboembolic pulmonary hypertension (CTEPH) and other pulmonary artery obstructions	CTEPH Angiosarcoma or other intravascular tumors Arteritis Congenital pulmonary artery stenosis Parasites
Group 5: Pulmonary hypertension with unclear and/or multifactorial mechanisms	Hematologic disorders Systemic disorders: sarcoidosis, neurofibromatosis Metabolic disorders: thyroid disorders Chronic renal failure (w/ and w/out dialysis)

- Pulmonary hypertension associated with another pathology is more common than Group 1 or idiopathic PH
- Symptoms are generally non-specific and related more to the underlying pathology or the progression of right heart dysfunction and failure.
- WHO Pulmonary Hypertension Classifications[25,26]

Class I – No limits	Diagnosed, but no symptoms
Class II – slight/mild limits	No symptoms at rest, (+) shortness of breath, fatigue, chest pain during physical activity
Class III – noticeable/marked limits	Comfortable at rest, (+) symptoms during normal activity
Class IV – severe limits	Symptomatic at rest

Chart Review:

- **Medical History**
  - Etiology of PH and categorization based on above classifications
  - Other potentially associated cardiopulmonary conditions
  - Onset and duration of PH symptoms and symptom class, if noted
  - Take note of any recent or frequent hospitalizations. Information gathered here can include home medication management, use of home oxygen
- **Hospital Course**
  - Reason for admission. Patients are often admitted due to symptoms related to the underlying pathology, i.e. increased volume status, or change in symptomatic presentation
  - Take note of any relevant lab values, imaging or tests. Below are common tests/measures done by the medical team in a patient with PH:
    - **Chest X-ray:** Used to determine condition of the lungs in underlying pathology, i.e. the level of pulmonary congestion. Depending on the cause of PH, abnormalities on CXR may not correlate with the degree of PH.[21]
    - Echocardiogram**:** Used to evaluate the effects of PH on the heart, particularly the right sided anatomy, including chamber dilation, as well as to estimate pulmonary artery pressure.[24]
    - Right Heart Catheterization: allows for more direct visualization of the functioning of the right heart and can also be used to assess congenital or acquired intracardiac shunting
    - **Pulmonary Function Tests**: Used to identify the contribution of underlying airway disease to a patient's symptomatic presentation, and can be useful in diagnosing new PH. Patients with Group 1 PAH can show mild to moderate reduction in lung volumes and frequently show reduced lung diffusion capacity for carbon monoxide, or DLCO, while patients with PH secondary to COPD will show PFT changes consistent with that pathology, namely airway obstruction and increased lung volumes. A decreased DLCO can indicate an interstitial lung disease as an underlying pathology.[24]
    - **Lab Values:** many lab values are assessed in patients with known or suspected PH, depending on underlying etiology. During the work-up for new or existing PH, arterial blood gases are often evaluated and can show a variety of issues. Rates of O_2 diffusion vary depending on pathology, therefore PaO_2 can be reduced or normal, while $PaCO_2$ can be normal, reduced, or elevated. An understanding of a patient's blood gas readings can help determine likely functional status, need for supplemental O_2, and potential for CO_2 retention.[24]
- Medications
  - Treatment of PH depends upon etiology and includes management of the underlying condition in Groups 2-5, optimizing hemodynamics, lung function, and volume status.

    - Common medications used to treat PH*:
        - Pulmonary vasodilators: epoprostenol (Flolan, Veletri), Treprostinil (Tyvaso, Remodulin)
        - Phosphodiesterase inhibitors: increases lung production of intrinsic vasodilators. Examples include: sildenafil, tadalafil
        - High-dose calcium channel blockers: relax smooth muscle in vasculature. Examples include: amlodipine, diltiazem
        - Anticoagulants, digoxin (rate control), beta blockers, ACE inhibitors, O2 therapy, and diuretics can also be employed
    - *See the Appendix 2 for relevant medications, side effects and physical therapy considerations for the medications above.
    - Lung transplant is the only definitive cure for Group 1 PH (pulmonary arterial hypertension), considered only after failure of medical management
- Determine Medical Stability
    - Patients admitted to the hospital are often in an exacerbation of their underlying condition, including a heart failure or COPD exacerbation, and medical stability and appropriateness for PT will depend upon the management of those conditions.
    - Understanding the anticipated management plan can assist in determining appropriateness for PT. For instance, patients who are going to be initiated on IV pulmonary vasodilators may benefit from PT assessment following the start of drug therapy.
    - Additionally, patients with progressive disease in need of more advanced care can rapidly move from the stepdown floors to the ICUs depending on stability, with a progression of care that can include surgery (atrial septostomy, lung transplant) or mechanical support (VV ECMO). Patients being worked up for lung transplant may have PT consults placed for 6MWT. Discuss appropriateness of this with your team lead or cardiopulmonary specialist
    - Any questions regarding the medical stability of your patient should be addressed with the medical team, team lead and/or cardiopulmonary specialist.

PT Examination:

- Determine prior level of function and impact of PH symptoms
    - It is important to understand a patient's lifestyle and past experiences with medical management. Questions can include:
        - How far can you walk before you need to rest? What causes you to rest? SOB? Muscle weakness?
        - Do you exercise regularly?
        - Have you attended a formal rehab program, cardiac or pulmonary as appropriate?
- Vital Sign Assessment
    - Be sure to monitor HR, SpO_2, RR and BP at rest and with activity. Patients with PH are at risk for oxygen desaturation and impaired hemodynamics due to increased stresses on the heart, fluid overload and medication side effects.

- Lung auscultation: assessing for pulmonary congestion both before and after treatment in the case of left heart dysfunction.
- Signs and symptoms of progressive right heart failure, particularly in later stages of PH: jugular venous distension, peripheral edema, ascites, and systemic hypotension due to impaired cardiac output (note hemodynamic response to exertion).[28]
- Endurance
    - Exercise tolerance can be limited in these patients, as with increasing workloads, pulmonary pressures increase, further restricting cardiac output and limited oxygen supply to skeletal muscles. Patients can be subject to lactic acidosis at lower workloads
    - The 6MWT can be used to quantify aerobic capacity and endurance. *See appendix 1 for patient instructions and appropriate performance of this test.

PT Intervention:

- Exercise intolerance in patients with PH is associated with reductions in maximal O_2 uptake and other factors similar to those with advanced heart failure. Increased resting pressures in the pulmonary vasculature lead to more dramatic increases during activity which can result in reduced pulmonary blood flow and subsequently reductions in cardiac output, with output insufficient to meet increasing demands. Patients with PH have additionally been found with skeletal muscle abnormalities with impaired oxygen utilization, further exacerbating the intolerance.[29]
- Physical Activity recommendations:
    - Evidence has demonstrated that exercise is safe for patients with PH however it is suggested that patients should be encouraged to be active within symptom limits and should avoid excessive physical activity that leads to a progression or exacerbation of symptoms. Patients should be treated with the best standard of pharmacological treatment and in stable clinical condition before starting a supervised rehab program.[24]
    - Exercise training has been shown to improve 6MWT distances and quality of life, as well as several physiologic measures of aerobic capacity and cardiopulmonary health. [29,30] Presumed negative effects of exercise on the right ventricle have been demonstrated to be short-lived, with function normalizing within days.[30] Exercise training has been shown to be highly effective in patients with more severe symptoms, with patients able to improve their WHO classification.[31]
    - Training protocols include progressive endurance training, either walking or cycling, at low to moderate workloads, measured as % peak VO_2 or heart rate.[29] Successful programs have also included respiratory muscle training.
    - In this setting, it is often more practical to prescribe exercise based on the RPE, targeting workloads at less than 5/10 on the modified Borg scale, pending appropriate hemodynamic response as determined on exercise testing including the 6MWT.

PE/DVT

Definition:

- Venous thrombi are intravascular deposits composed mainly of fibrin and red blood cells, usually located in the deep veins. They can lead to complications, including deep vein thrombosis (DVT) or pulmonary embolism (PE). DVTs occur in regions of slow or disturbed blood flow, often in the lower extremity. The majority are confined to the calf and are asymptomatic, small and not associated with major complications. However, venous thrombi in the proximal veins (popliteal, femoral, iliofemoral), may break off, leading to a blockage in the pulmonary circulatory system, called a PE.[28]

Chart Review:

- **DVT**: The major signs and symptoms of LE DVT include pitting edema, pain, tenderness, swelling, warmth, redness or discoloration (erythema), and prominent superficial veins.
    - **Diagnosis:** The Wells criteria for LE DVT are the most commonly used tool to determine likelihood of DVT. The results of the Wells criteria should guide the selection of medical testing, including the D-dimer test to measure the breakdown or degradation of cross-linked fibrin (increases in the presence of a thrombosis) and then a duplex ultrasound.
        - If the ultrasound confirms an LE DVT, medical treatment should be initiated and mobilization is often postponed, however this should be considered on a case by case basis, as the risks of immobility have been shown often to outweigh the risks associated with mobilizing someone with a DVT.[32]
    - **Treatment**: Anticoagulation therapy is effective in the prevention of extension, embolization, and recurrence of DVT.
        - Options include subcutaneous low-molecular weight heparin (Lovenox, Fragmin), monitored IV or subcutaneous unfractioned heparin (Heparin Sodium), or fondaparinux (Arixtra)
        - Lab Values:
            - **Heparin** is measured by clotting times such as activated partial thromboplastin time (APTT or PTT).
              Warfarin (Coumadin) is measured using the International Normalized Ratio for Prothrombin Time (PT INR)
                - INR Reference Ranges
                    - 2.0-3.0 = desired range
                    - 2.5-3.5 = heart valve in place
                    - >4.0 = risk of hemorrhage
                - If the INR is between 4.0 and 5.0, resistive exercises should be avoided, with participation in light exercise only due to increased risk of bleeding. Ambulation should be restricted if gait is unsteady to prevent falls.

        - Placing a filter in the inferior vena cava is a treatment that is used when a patient has a PE or acute proximal DVT and anticoagulant therapy is not indicated because of the risk of bleeding.
- **Pulmonary Embolism (PE)**: major signs and symptoms include tachycardia, sudden change in breathing, chest pain, coughing
    - **Categories:**
        - Massive: Acute PE that causes hemodynamic instability including SBP <90 mmHg, pulselessness, bradycardia, tachycardia, tachypnea, bradypnea, respiratory failure, right ventricle failure. Damaged lung tissue, combined with less available area for pulmonary perfusion can cause high pulmonary artery pressures, dramatically increasing right ventricular work and leading to right-sided heart failure.[33]
        - Submassive: Acute PE, patient remains hemodynamically stable without systemic hypotension (systolic blood pressure >90 mm Hg) but with either RV dysfunction or myocardial necrosis.
        - Nonmassive: no signs of clinical instability, hemodynamic compromise, or right ventricular strain.[34]
    - **Diagnosis:** The most frequent diagnostic testing for PE is serial computed tomography, followed by ventilation/perfusion scans, or pulmonary angiography
    - **Treatment:** Heparin therapy is most commonly used to treat PE, however massive and submassive PEs can also be treated with endovascular catheter directed thrombolysis via an EKOS device. To maintain adequate tissue oxygenation, mechanical ventilation with supplemental oxygen may be required. In addition, if the patient is hypotensive or in shock, fluid therapy and vasopressors may be needed.
        - Pulmonary embolectomy may be indicated in patients who have large emboli and cannot receive heparin therapy or have overt right ventricular heart failure leading to cardiac arrest.[33]

PT Examination/Intervention:

- As physical therapists in the acute care setting, we play a role in the prevention of DVTs by educating patients on their risk, mobilizing and providing LE exercises
- We can also be a first line for screening patients for DVTs. If your patient presents with signs and symptoms of a DVT, be sure to communicate these with the medical team.
- A patient with a known DVT who develops unexplained breathlessness, desaturation, hemoptysis, pleuritic pain, arrhythmia, or fever should be suspected of having developed a PE and the medical team should be notified.[28]
- Early ambulation and compression stockings are recommended for patients with acute DVT once medical treatment has been initiated (anticoagulation or IVC filter) and may provide even faster improvement with less pain, swelling, and minimize the extension of a DVT.
- In patients with diagnosed massive and submassive PEs, mobility is only indicated when the patient is stabilized after all interventions other than anticoagulation therapy (ex:

thrombectomy or thrombolysis) have been performed, or when optimum symptom management and right heart function have been achieved.[32]

- If out of bed activity orders are written for someone with a known DVT or PE who is not being treated medically, be sure to clarify activity orders and document these in your note

Pericardial Effusion/Tamponade

Definition/Pathology:

- A pericardial effusion refers to excess fluid that develops between the pericardium and the heart itself. Injury to the pericardium can cause acute pericarditis (inflammation in the pericardial sac surrounding the heart), which can lead to a pericardial effusion. Some common causes of pericardial inflammation include: heart surgery, infection, inflammatory disorders such as RA or Lupus, metastatic cancer and kidney failure with excessive nitrogen levels.
    - A rapid or significant accumulation of fluid can lead to cardiac tamponade, which is a life-threatening condition characterized by elevated intracardiac pressures, progressively limited ventricular diastolic filling, reduced stroke volume and pulsus paradoxus.[10,35]

Chart Review:

- When reviewing a chart for a patient with a known pericardial effusion, it is important to note the cause and current or planned treatment. How the effusion is managed can help you determine the severity of the effusion and the patient's expected hospital course. The goal of medical treatment is to address the underlying cause of the effusion.
- Some common treatments of pericardial effusion include:[10,36]
    - Initiation of NSAIDs, corticosteroids, colchicine (commonly used to treat gout), diuretics or antibiotics
    - If the effusion is related to cancer, radiation or chemotherapy may be used
    - For larger pericardial effusions:
        - **Pericardiocentesis**: a needle and catheter is inserted into the pericardial space to drain the effusion. The catheter and drain may remain in place temporarily for ongoing drainage.[36]
            - Some indications for removal of catheter by nursing include:
                - Pericardial drainage less than 25-30 mL over 24 hours.
                - Hemodynamic stability: SBP greater than 100 mmHg, no pulsus paradoxus (< 10 mm Hg)
                - Absence of pericardial effusion.
        - **Pericardial Window:** a surgical procedure for recurrent effusions, involves removing a portion of the pericardium to allow the effusion to drain continuously into the peritoneum or chest.[37]
- Check activity orders. Activity may be restricted while monitoring for tamponade.
    - If a patient is without a procedure (e.g. with close monitoring, medications or chemo/radiation), be sure that the effusion is stable and the patient has been

hemodynamically stable. Check in with nursing, in addition to the chart, to ensure hemodynamic stability and updated activity orders.
    - If a pericardial drain was placed, the patient is often on bedrest to allow for frequent vital sign assessment by nursing.[38] Pt will be cleared for progression of activity when deemed stable by medical team. Be sure to get updated activity orders on a case-by-case basis depending on the duration of the drain.
    - When a pericardial drain is removed, the patient should remain on bed rest for 1 hour and defer physical activity/therapy for 2 hours.[38] Be sure to seek updated activity orders from medical team.

PT Examination:

- Careful monitoring of vital signs and patient response to activity is essential to monitor for progression to tamponade.
- *Be sure to look out for signs and symptoms of tamponade*, including: jugular vein distention, hypotension, tachycardia, fatigue/malaise, lightheadedness, rales at lung bases, shortness of breath and tachypnea. Alert nursing immediately if cardiac tamponade is suspected.[36]

PT Intervention:

- Be sure to look at the whole clinical picture and the reason for pericardial effusion when designing your intervention and plan of care.
    - For example, someone with recent heart surgery who developed a pericardial effusion may have a different plan of care then someone who known metastatic cancer who also developed a pericardial effusion
- Patient's should be educated on the signs and symptoms of Tamponade and on appropriate activity progression in order to achieve their goals
- Out of bed mobility and ambulation is not contraindicated in these patients and should be encouraged, just be sure to clarify all activity orders based on their treatment and hemodynamic stability as noted above

Congenital Heart Defects

Definition:

- Congenital heart defects include any structural abnormality of the heart, present at birth, and typically forming early in gestational development. They can cause a variety of signs and symptoms and are of varying severity and prevalence. They are typically classified into acyanotic and cyanotic lesions. Management is dependent upon the severity of the presentation.[39]
- ***Acyanotic lesions***: this group of defects result in increased flow of blood through the pulmonary vasculature through **left to right shunting**, with fully oxygenated blood circling back to the lungs as well as into systemic circulation. Signs and symptoms can include increased respiratory rate, low peripheral PO_2, low stroke volume, and increased work load on the heart leading to progressive heart failure.[39]

- **Septal defects**: Includes both atrial and ventricular septal wall abnormalities that allow mixing of blood between 2 chambers and decreasing the amount of fully oxygenated blood that that leaves as cardiac output.
    - Atrial septal defects (ASD)/Ventricular septal defects (VSD): small ones present at birth are generally asymptomatic and close with growth. Larger defects allow for greater mixing of deoxygenated blood, and in adults, increase the risk for stroke, heart failure, pulmonary hypertension, and endocarditis.[39,40,41]
        - The most common atrial septal defect is a patent foramen ovale, often going undiagnosed but placing a patient at increased risk for stroke, requiring surgical closure.[39,40]
        - In adults, small unrepaired defects or childhood repairs of larger ASDs rarely cause problems. Previously undiagnosed defects can lead to heart failure in adults and are often closed, even when asymptomatic.[40]
        - Late or adult complications of childhood VSD closures are rare. Large unrepaired defects can lead to pulmonary hypertension in adults, and small defects left open are at increased risk for endocarditis.[41]
- **Patent Ductus Arteriosus (PDA)** is a common heart defect, due to failure of the ductus arteriosus to close after birth, leading to mixing of oxygenated blood from the aorta with deoxygenated blood in the pulmonary artery with subsequent increased pressure and pulmonary hypertension. The defect can close without treatment, but is often managed with medication or minimally invasive surgical or catheter assisted closure.[39,42]Long term prognosis in childhood repairs is good, and cardiology follow up is usually not needed.[42]
- **Coarctation of the Aorta** is a narrowing of the aorta, creating a left ventricular outflow obstruction and leading to progressive left ventricular hypertrophy. Additionally, upper body hypertension can occur which can lead to progressive heart failure. There are typically normal to low blood pressures in the supply distal to the constriction.[39,43]
    - Surgery is required to correct the defect and is performed as soon as the coarctation is diagnosed, which can happen in adulthood.
    - Patients with early repair will require long-term cardiology follow up for BP management and monitoring due to the risk of recoarctation or aneurysm.[43]

- ***Cyanotic lesions***: this classification of defects involves **right to left shunting** whereby most of the blood bypasses the lungs and enters systemic circulation without being fully oxygenated. Chronic hypoxemia leads the body to increase its red blood cell production, leading to polycythemia and an increase in blood viscosity, increasing the risk for stroke.[39]
    - **Tetralogy of Fallot:** This complex CHD includes pulmonary valve stenosis, a large ventricular septal defect, an overriding aorta (aorta leaves the heart between the right and left ventricles, directly over the septal defect), and right ventricular hypertrophy (due to the narrowing of the pulmonary valve). This defect requires early surgical management to redirect blood flow and surgical techniques are continuously evolving.[39,44]

        - Patients admitted at BWH as adults are often admitted to the heart failure service for management of the long-term consequences of the abnormal/repaired blood flow, including right sided heart failure, arrhythmia, and repair of continued areas of stenosis.[44]
    - **Hypoplastic left-heart syndrome**: this syndrome includes an underdeveloped or hypoplastic left ventricle, aortic and mitral valve stenosis or atresia, and coarctation of the aorta. This syndrome is largely asymptomatic until the ductus arteriosus closes. Medications can be used to keep the ductus arteriosus open and surgery or transplant is required for survival.[39] Surgical correction includes staged procedures that allow for the right ventricle to pump systemically and to redirect blood flow to the lungs.[45]
    - **Transposition of the Great Arteries (TGA)**: the key component of this defect is a reversal of the positions of the pulmonary artery and the aorta, leading to separate pulmonary and systemic circulatory systems with variable communication via PDA, ASD, or VSD. Without communication to allow mixing of the blood inside the heart, severe cyanosis occurs and the defect is incompatible with life. Prostaglandin E_1 can be given to maintain a PDA however surgical treatment is required early.[39]
        - Adults with a history of repaired TGA require lifelong cardiology follow up due to risk of heart failure, tachy and bradyarrhythmias, endocarditis and for blood pressure management. Surgical baffles can also become obstructed requiring stenting or further surgery for management.[46]

Chart Review:

- **Medical History:**
    - Review to gain an understanding of the patient's CHD and surgical history or management of the defect. This should include an understanding of the patient's current medication management such as beta blocker therapy, inotropic therapy, diuretic use, anti-arrhythmics, etc
    - Number of recent admissions and reason, as this can indicate a decline in cardiac function and the potential for progression to advanced therapies
- **Hospital Course:**
    - Reason for admission, often a progression of symptoms associated with heart failure (refer to CHF)
    - Planned surgical procedures, if any.
    - Changes to current medical management including pharmacologic and potential for progression to advanced therapies such as mechanical circulatory support and/or transplant

PT Examination: refer to heart failure

PT Intervention: refer to heart failure and PT Intervention/Exercise Training

Aortic Dissection

Definition:

- Aortic dissections involve a tearing of the intimal lining of the aorta, allowing for blood to enter the lining, leading to further separation of the walls of the vessel, which at high pressures, can cause an aortic rupture.
- Dissections are typically classified as:
    - *Type A*: occurring in the ascending aorta, distal to the aortic valve, and along the arch. Medical emergency that requires surgery, via sternotomy, to manage given the acuity and risk for neurologic compromise
    - *Type B*: occurring in the descending aorta past the bifurcation of the left subclavian artery. Often medically managed, however may also require surgery[3,47]

Chart Review:

- **Medical History:**
    - Presenting symptoms of an acute or chronic dissection can mimic those of an MI, so be sure that medical work up is complete before seeing these patients. Symptoms can include sudden chest or back pain, change in pulse, shortness of breath, or syncope.[3]
- **Hospital Course:**
    - Diagnostic work up to rule in dissection can include CT angiography, MRI and/or transesophageal echo (TEE).
    - If an MI is suspected, check lab values (see above)
    - Plan for management:
        - Typically, patients with Type A dissection will be referred for urgent surgical management to the cardiac surgery team. Please refer to cardiac surgery below
        - Management of Type B dissections often involves aggressive pharmacologic management for BP and HR control to prevent progression of the tear. Type B dissections that have ruptured, are causing severe pain, or have led to distal organ ischemia may also require surgical management
    - Review of vitals to determine how well controlled the patient's HR and BP have been under current medical management

PT Examination:

- It is extremely important to establish hemodynamic parameters for patients with medically managed aortic dissections prior to initiating mobility
    - Heart rate and BP parameters are often provided by the managing medical team; however, those parameters may not take response to activity into consideration
    - A conversation should be had with the responding clinician or other member of the medical team to establish reasonable parameters for HR and BP in response to functional mobility
- Establish baseline and current level of functional mobility with frequent VS monitoring throughout examination

PT Intervention:

- Restore baseline level of mobility as appropriate given hemodynamic response to activity
- Education, specifically around limitations and expectations for return to prior level of function.
    - These patients are often counseled to minimize exertion and while basic functional mobility is acceptable, a return to a high-level exercise program may not be recommended
    - Avoid valsalva

Cardiogenic Shock

Definition/Pathology:

- Cardiogenic shock (CS) is a clinical condition of inadequate end organ perfusion due to cardiac dysfunction.[48] It can present across a spectrum of severity from mild hypoperfusion to profound shock,[49] with the reduction in tissue perfusion resulting in decreased oxygen and nutrient delivery to the tissues and, if prolonged, leading to multi-organ failure. Acute myocardial infarction (AMI) is the most common cause of CS, usually associated with severe ventricular dysfunction (anterior wall STEMI). There are multiple other causes of CS as well.
- Clinical criteria for diagnosing CS include:[48]
    - Persistent hypotension
        - Systolic blood pressure (SBP) <80-90 mmHg for 30 minutes OR mean arterial pressure (MAP) <65 mmHg for 30 minutes OR vasopressor required to achieve a SBP ≥90 mmHg OR MAP 30 mmHg or lower than baseline
    - Severe reduction of cardiac index
        - 1.8 L/min/m^2 without support
        - 2.0-2.2 L/min/m^2 with support
    - Elevated filling pressures of left, right, or both ventricles
    - Signs of impaired organ perfusion with at least one of the following criteria:
        - Altered mental status
        - Cold extremities
        - Oliguria
        - Increased serum-lactate
- The diagnosis of CS is usually made with clinical signs and symptoms as well as help from monitoring hemodynamics via pulmonary artery catheter (PAC), electrocardiogram (ECG), chest x-ray, blood tests (ABG, electrolytes, CBC, and troponin), echocardiogram, and cardiac catheterization.[48]

Chart review:

- It is important to note the cause, or suspected cause, as well as the current and/or planned treatment. If an intervention such as PCI, CABG, valve surgery, VAD placement, or heart

transplant is planned, it should be noted. If the patient is pending an intervention check the activity orders and speak with the responding clinician as deferring mobility until after the intervention may or may not be indicated. Patients with CS are often critically ill and in the ICU.

- Trends and current vitals and lab values should also be noted to ensure a patient is hemodynamically appropriate for mobility. There are often hemodynamic parameter goals that are individualized to each patient given their clinical presentation.
- Patients diagnosed with CS have increased likelihood of requiring mechanical ventilator support as well as increased likelihood of worsening renal failure, which could require renal replacement therapy.
- While reviewing the chart, therapists should also take notice of:[48,50]
    - Lines, including central venous catheter, arterial line, and pulmonary artery catheter. The therapist should note the location of the line as frequently lines are placed via femoral access and may alter or impair a patient's ability to participate
    - Pharmacologic Support*
        - IV fluids
        - Vasoactive Medications including vasopressors and inotropes
        - Antiarrhythmics
        - *See the Appendix 2 for relevant side effects and physical therapy considerations for the medications above.
    - Mechanical ventilation – level of support, recent changes to indicate improvement or worsening of condition, and presence of sedating medications
    - Renal replacement therapy, continuous versus intermittent
    - Mechanical circulatory support devices (left ventricular, right ventricular, or biventricular devices) including IABP, Impella, VA ECMO (see support devices below for therapy considerations for each device). The ProtekDuo Tandem Heart or CentriMag can also be used for temporary support as a bridge to recovery or more durable support, see Mechanical Circulatory Support Devices Standard of Care for further information.

PT Examination:

- Once physical therapy is indicated it is essential to monitor the patient's hemodynamic response to exercise and mobility. ROM testing or mobility may be limited if the patient has certain lines or mechanical circulatory support devices present. Beyond a traditional examination a therapist should focus on:
    - Cognition
        - Increased likelihood of altered mental status and decreased command following (reference?)
    - Skin integrity: note change in color and/or temperature, presence and severity of edema
    - Peripheral pulses: likely to be irregular, rapid, and/or faint
    - Jugular vein distension

- Pulmonary assessment including auscultation of lung sounds as able, assessing for presence of inspiratory crackles indicating pulmonary edema, and ventilatory pattern and effort
- Auscultation of the heart: distant or faint heart sounds, presence of S3 or S4 heart sounds

PT Intervention

- Treatment should be based on the patient's impairments and hemodynamic response to mobility with the goal of maximizing functional mobility, independence, and safety prior to discharge. As a patient recovers, formal exercise testing and prescription may be indicated, however functional mobility assessment can be used to provide guidance for an exercise program. See PT Intervention/Exercise Training for further information.

Cardiac Tests and Procedures:

Common Cardiac Diagnostic Tests

Echocardiogram

- An echocardiogram is one of the most common diagnostic tests to visualize the heart, aorta, and other blood vessels. The device emits ultrasound waves to create a single or two-dimensional image.[51,52] It allows for examination of the chambers of the heart, blood flow, valve function, and volume status to provide information about heart function including cardiac output, ejection fraction, and diastolic function or to diagnose conditions such as a blood clot or mass in the heart, pericardial effusion, congenital heart diseases, or active infections of the heart valves.[51-53]
- Types of Echocardiograms include:
  - *Transthoracic Echocardiogram (TTE)*[52] -Transducer is placed on the chest and ultrasound must travel through the chest wall and lungs to reach the heart. This is the most common type of echocardiogram utilized and can be performed at bedside.
  - *Transesophageal Echocardiogram (TEE)*[53]- under sedation, a transducer is passed through the esophagus, which sits posterior to the heart. This allows for less interference, superior image quality and visualization.

Stress Test -MIBI

- A sestamibi or MIBI is a nuclear perfusion scan that looks at myocardial blood flow via IV injection of technetium sestamibi, a pharmacological agent which contains a type of radioactive isotope. Sestamibi distributes in the myocardium proportional to the myocardial blood flow.[54]Single photon emission computed tomography (SPECT) imaging of the heart is completed by detecting the gamma rays emitted by the isotope as it decays.[55]
- Two sets of images are taken:
  - One set of images is taken when the patient is at rest.

    - The second set is taken once the patient's heart is stressed. This is done by having the patient exercise on a treadmill or pharmacologically (with use of a vasodilator or dobutamine).
- The images of the heart at rest and during peak stress are compared. From the comparison one can distinguish ischemic versus infarcted areas of the myocardium with high sensitivity.[54]

Cardiac MRI

- A cardiac magnetic resonance imaging (MRI) is used to create a detailed image of the heart's structures including chambers, muscle, valves, as well as how blood is flowing through the heart and vessels. It can help diagnose conditions including atherosclerosis, cardiomyopathy, congenital heart disease, heart failure, aneurysm, heart valve disease, cardiac tumor, or infiltrative disorders like cardiac sarcoid or cardiac amyloid.[56]

Cardiac PET Scan

- A cardiac positron emission tomography (PET) scan is a test where radioactive tracers are injected intravenously into a patient and a gamma detector picks up images from the tracer. A computer converts the signals into an image that shows the size, shape, position, and some function of the heart.
- A cardiac PET scan show if the heart is getting enough blood flow, if coronary artery disease is present, and can identify areas of damage or ischemia.[55]

Cardiac Catheterization

- A catheter is inserted into the left or right side of the heart for diagnostic and interventional purposes. It is important to determine whether only a cardiac catheterization was performed or if additional procedures were performed as well e.g., percutaneous transluminal coronary angioplasty (PTCA) or insertion of coronary artery stents, prior to PT examination or intervention. Activity precautions are aimed at protection of the incision site and are as follows:
    - **Left heart catheterization** (LHC) is used to diagnose left ventricular, atrial, pulmonary vein, and coronary artery impairments.
        - Due to the arterial incision site via the femoral artery, these patients are on bed rest for 6-8 hours with involved LE straight. Patient may have a knee immobilizer donned to minimize hip flexion. The patient is monitored for groin hematomas, and pain.
        - Use of the radial artery is also common and the patient can ambulate with assistance if vital signs are stable and there is no signs of bleeding or other complications, or if not otherwise stated in the physician orders.
        - Complications associated with a left heart catheterization include groin hematoma and retroperitoneal or intramuscular bleeding in the LE or intramuscular bleeding in the forearm. Also, consider a patient's response to the general anesthesia while examining functional mobility.

- **Right heart catheterization** (RHC) is used to diagnose right atrial, ventricular, and pulmonary artery impairments including flow rates and pressures. The incision site is generally via the external jugular vein and there are no activity restrictions. RHC can also be used to estimate left heart pressures

Arrhythmia Management

Cardioversion

- Electrical Cardioversion [Direct-Current Cardioversion (DCCV)] or Pharmacological Cardioversion
  - Medical interventions, either by cardiac electrical shock (DCCV) or with medication (e.g. Ibutilide) are aimed at restoring normal sinus rhythm to optimize cardiac output.
  - Due to potential return of arrhythmias, or patient fatigue associated with anesthesia for DCCV, PT is generally held the day of cardioversion. Make sure to check for changes to activity orders and monitor for resumption or progression of cardiac arrhythmia.

Ablation

- Catheter ablation is a procedure that uses radiofrequency energy, laser light or extremely cold temperatures (cryoablation) to destroy a small area of heart tissue that is causing arrhythmias when medicines are not tolerated or effective.
  - Venous access (either femoral, subclavian, or internal jugular) is achieved through cardiac catheterization.
- Most commonly used in treating supraventricular tachycardia, atrial flutter and atrial fibrillation.
- If patient had femoral access, they are on bed rest with leg straight for 6-8 hours.

Cardiac Surgery and Sternal Precautions[1,3]

Common Cardiac Surgeries requiring median sternotomy

- **Coronary Artery Bypass Graft:** CABG is a surgical intervention used to treat a completely occluded coronary artery that cannot be treated by, or has failed treatment with a percutaneous intervention. It uses a vascular graft (typically the saphenous vein or left internal mammary artery) to revascularize the myocardium. CABG can be performed for one or more vessels as typically documented in a patient's chart by CABG x4 or 4vCABG.
- **Valve Repair/replacement:** Valve repair or replacement surgery is a treatment for valvular disease. Valves can be replaced by mechanical valves (bileaflet, tilting disc) which are known for a longer life span and durability, but need lifelong anticoagulation, or with biological valves (cadavers, porcine or bovine) which carry a benefit of not needing life time anticoagulation therapy. Minimally invasive valve replacements via a partial sternotomy (aortic and mitral valve), and mitral valve repair/replacement via a thoracotomy approach can be utilized in appropriate patients
- **Aortic arch repair:** An aortic arch repair can be done using synthetic material to replace the diseased or damaged portions of the vessel. A minimally invasive approach would

entail a metal wire mesh stent being inserted percutaneously via an artery. This minimally invasive approach is typically seen in pts who would not tolerate an open surgical approach, see TAVR below.

- **Maze:** The Maze procedure is performed to treat atrial fibrillation. It is an open procedure that entails the etching of lines by heat (radiofrequency energy), cold (cryoablation) or a scalpel to the atria to create patterns of scar tissue (maze) to block the abnormal electrical impulses associated with atrial fibrillation. This procedure is often combined with other open-heart procedures and rarely performed on its own.
- **ASD/VSD:** Atrial and ventricular septal defects are often repaired surgically. Small defects are closed via internal sutures while larger defects may require patching that may be created from the patient's own tissue or use synthetic material. These repairs can be performed on their own but are often performed in conjunction with other procedures during the open sternotomy surgery.

Sternal Precautions

- The exact origin of sternal precautions is not known and current literature does not support all the imposed restrictions, many of which are anecdotal "or based on expert opinion". Multiple recommendations and protocols exist across institutions, with some limited consensus.
- Current recommendations for sternal precautions at Brigham and Women's Hospital to be followed for up to 12 weeks:
    - No lifting more than 10 lbs
    - Avoid excessive pushing/pulling (such as closing/opening heavy doors, vacuuming, dog walking, shoveling snow, carrying groceries or laundry baskets)
    - Avoid excessive twisting through your trunk, such as reaching behind you
    - Avoid excessive coughing, laughing or sneezing; be sure to hug a pillow across your chest when performing these activities.
    - Avoid sit-ups. Log roll to get in and out of bed.
    - Avoid driving or sitting in the front seat of a car with an airbag for 3-4 weeks
    - Avoid excessive overhead or lateral arm movements, however patients are encouraged to perform functional arm movements and active ROM exercises in a pain free range
- Consideration should be taken to identify those patients who have multiple risk factors for sternal wound dehiscence and these patients should be encouraged to strictly adhere to sternal precautions:[59,60]
    - Use of internal mammary artery (IMA) in the bypass graft
    - Females with pendulous breasts
    - Morbid obesity
    - Barrel chest
    - History of poorly controlled diabetes mellitus
    - Osteoporosis
    - Redo operation for bleeding or repeat cardiothoracic surgery
- Signs of impaired sternal healing:

  - A crunch, pop or click in your sternum
  - Bleeding that does not stop even with pressure applied
  - Signs of infection, such as fever, redness or warm skin around the incision
- The therapist should use sound clinical judgment to allow for safe progression of mobility to optimize functional return, and individual patient limitations should be considered and discussed with the medical team and the patient. At this time, patients s/p full sternotomy without signs of sternal infection should follow sternal precautions for up to 12 weeks, pending follow up with the surgeon and potentially earlier liberalization of precautions. Patients with minimally invasive (partial) sternotomies should follow sternal precautions until surgeon clearance.
- Current literature has shown that restrictive sternal precautions may in fact worsen functional limitations due to impairments in muscle performance, poor healing of connective tissues across the sternal incision, and long-term impairments in pulmonary function and thoracic cavity mobility.[58,61]

TAVR

- Transfemoral/Transcatheter Aortic Valve Replacement (TAVR) is a less invasive method for replacing the aortic valve in patients with aortic stenosis when compared to a standard cardiac surgery for replacement of the valve. This method is currently approved for use in patients with severe symptomatic aortic stenosis (AS) who are at intermediate to high surgical risk or are otherwise inoperable.[62]
- Performed via femoral artery catheter access to deploy a mechanical valve across the pre-existing diseased valve. Imaging is required to ensure the vascular pathway is clear without stenosis, calcification, or tortuosity
- A balloon is expanded to seat the valve in position before the catheter is withdrawn
- PT implications:
  - Monitor patients for post-op bleeding and/or tamponade
  - This technique is performed without a sternotomy, therefore there are no sternal precautions post-procedure. However, because the technique is performed via a femoral approach, the patient should avoid any heavy lifting (>5-10lbs) for 1 week following the procedure.
  - Activities promoting sudden or extreme hypertension should be avoided
  - Lower incidence of arrhythmias as compared to open approach
  - As this procedure is performed in a higher risk population, baseline frailty should be noted and may significantly impact functional performance post procedure.
  - Length of stay is typically very short following the procedure and patients may be discharged the following day

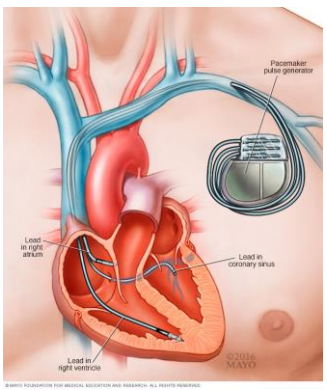
Cardiac Support Devices:

Chest Tubes

- Chest tubes (CT) on the cardiac service are primarily placed post-operatively in the pleural or mediastinal cavity to remove excess fluid or blood associated with surgery. The chest tube unit provides negative pressure to assist with drainage.[63]
- Maintaining the integrity of this closed system by keeping the chest tube upright and the water seal trap in place prevents fluid buildup and potential lung compression and ensuing respiratory distress. Use of low wall suction may be employed to maintain a closed system when there is a large air leak from the lung to the thoracic cavity. When the air leak is small or when the necessity of continued chest tube use is being evaluated, the CT may not require suction but remain on water seal or have temporary surgical clamps in place on the tubing.
- Prior to PT examination or treatment, clarify if the chest tube needs to remain to suction. If continuous suction is necessary, either use a portable suction unit if walking in hall, or extended tubing to allow increased patient activity in room.
- Chest PT, shoulder ROM, and deep breathing exercises to patient tolerance are beneficial in the management of ventilation and airway clearance.[63]
- Removal of a CT may result in the development of a pneumothorax or exacerbation of the initial air leak related to lung tissue injury. A follow-up chest x-ray (CXR) is performed and read by a physician assistant (PA) or physician (MD) to rule out a pneumothorax following CT removal.
    - If a PT works with a patient before the CXR has been read, activity orders should be clarified with the PA. If the decision is made to treat the patient, monitor for signs of acute pneumothorax (acute shortness of breath, new or increased palpable crepitus felt anywhere in the thorax, precipitous drop in SPO2 from baseline or < 80%).

Permanent Pacemaker and/or Implantable Cardioverter-Defibrillator

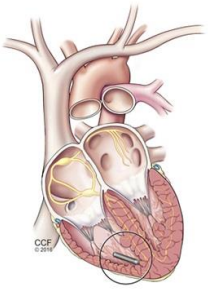
- The insertion of a permanent pacemaker (PPM) or implantable cardioverter-defibrillator (ICD) may occur to maintain appropriate cardiac conduction or prevent cardiac arrhythmias.
- A PPM or ICD has three main components: a pulse generator, leads, and electrodes. Generators are usually described as being either single-chamber or dual-chamber.
    - Single-chamber systems have one lead, which is usually placed in the right ventricle or right atrium.
    - Dual-chamber systems have two leads, one of which is implanted in the right atrium and the other in the right ventricle. [64]



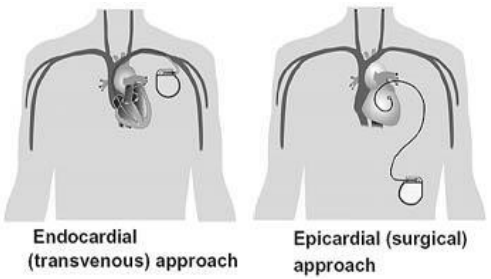
- The device is usually inserted under the skin in the left subclavian pocket, with leads inserted into the right side of the heart via the left subclavian vein to the superior vena cava. In instances where insertion

via a left subclavian pocket is not possible or after an infection of a left pocket, the new device may be inserted via the same routes on the right side.[65]

- Precautions in movement following placement are focused on incision healing and lead fixation. Although current literature has not investigated the time frame for the leads to firmly attach into the myocardium, current recommendations from physicians indicate that 4-6 weeks allows incision healing and firm adhesion of pacemaker leads.[65]
- Current **pacemaker precautions** at BWH are:
    - Keeping the involved UE in a standard sling for 24 hours
    - No therapeutic exercise to involved shoulder for 4-6 weeks unless approved by the Electrophysiology Service (EPS).
    - Involved UE may be used functionally but limit shoulder flexion and abduction to 90 degrees for 4-6 weeks.
    - Patient may use minimal weight bearing (10-15 lbs.) into UE while using an ambulatory assistive device, however use of axillary crutches are not recommended due to venous pressure in the axilla region and incisional stress. In rare instances, use of axillary crutches to safely negotiate stairs may be indicated.
    - No lifting greater than 5 lbs. with involved UE for 4-6 weeks.
- **Leadless pacemakers** are small self-contained devices that are inserted in the right ventricle of the heart. A leadless pacemaker does not require leads or a generator, or the creation of a surgical pocket on the chest. Currently, the device is available for patients who need single-chamber pacing only. Because there are no wires or generator, there are no upper body activity limitations after the implantation so typical pacemaker precautions do not apply.[66]



- **Semi-permanent pacemakers**
    - Generally used as a bridge therapy when a patient is not clinically able to tolerate a permanent pacemaker placement (e.g. infection with positive blood cultures). The device wires are placed transcutaneously with the pacemaker external and secured with subcutaneous sutures and a dressing. Given the location and lack of permanent fixation, mobility of the ipsilateral shoulder is generally contraindicated. Seek clarification of activity orders out of bed with MD and EPS.
- **Temporary Cardiac Pacing**
    - Used to treat bradyarrhythmias and rarely a tachyarrhythmia, until it resolves or until long-term therapy can be initiated.[67]
    - Two types are typically seen at BWH:
        - Transvenous pacing wires (typically used more emergently in the CCU): Introduced through the internal jugular or subclavian veins, therefore no ROM assessment or therapeutic exercise to the involved shoulder is allowed.
            - More comfortable and more durable in patients in whom the



anticipated duration of temporary cardiac pacing may be several days to weeks.
    - Most patients will be restricted to bed or chair and cannot ambulate.[64]
- Epicardial pacing wires (typically seen post-cardiac surgery): often left in place after cardiac surgery when the risk of cardiac arrhythmias is most great.[68]
    - Patients may be dependent on this pacemaker for cardiac rhythm, or the device may be in place to take over in the event of an adverse cardiac rhythm (usually bradycardia) and just sensing (a.k.a. "back-up mode")
    - Leads may be attached to the atrium, ventricle, or both chambers, with the wires tunneled and externalized.
    - No UE ROM restrictions as the wires are transthoracic.[67]
    - Mobilization of a patient with temporary epicardial pacing wires is considered appropriate, however caution should be taken to avoid dislodging the wires. The therapist should be aware of the patient's underlying cardiac rhythm, and activity orders should be clarified. Most importantly, orders should be clarified in patients who have more serious arrhythmias (ex: heart blocks) who are dependent on the pacing for adequate cardiac function.
    - When no longer needed, the epicardial pacing wires may also be detached from the temporary pacing device, capped with insulating wire caps, and taped to patient's chest. When epicardial pacing wires are removed, they are either cut at the skin level or pulled through the skin. When removed through the skin, there is a risk of bleeding where they were attached to the myocardium and therefore these patients are monitored for signs of cardiac tamponade (i.e., tachycardia, lightheadedness, dyspnea) and follow this progression of activity to allow for monitoring:
        - Patient may be out of bed with RN in room after 1 hour of bed rest.
        - Patient may participate with PT after 2 hours.
        - Patient may initiate or resume stair training after 4 hours.

Pulmonary Artery catheter (or Swan-Ganz catheter)

- Pulmonary artery catheters (PAC) are the most commonly used tool for in-depth hemodynamic assessment. They allow for a rapid evaluation of the effectiveness of interventions used to manipulate circulatory volume and pressure, including the administration of fluids and diuretics as well as vasoactive and inotropic medications.[69]
- The catheter is flexible, balloon tipped, and is most commonly inserted in the jugular or subclavian vein. It travels to the superior vena cava, right atrium, right ventricle, and

pulmonary artery. The catheter is connected to a transducer that measures many hemodynamic parameters including:
    - Direct measurement of pulmonary artery pressure, pulmonary capillary wedge pressure (during balloon inflation), central venous pressure, and mixed venous oxygen saturation
    - Estimates stroke volume, cardiac output, and cardiac index.[63]
    - See Appendix 3 for full description of PAC values and interpretation
- Potential complications with PAC insertion are
    - Ventricular arrhythmias, right bundle branch block, pneumothorax.
- Potential complications with the ongoing use of PACs are
    - Pulmonary artery infarction, pulmonary artery rupture, line infection, movement within the pulmonary artery causing injury to the vessel or accidental dislodgement into the right ventricle, or dysrhythmias.[70]
- Therapists treating patients with PACs should be aware of the normal waveforms for atrial, pulmonary artery and pulmonary capillary wedge pressure measurement, although artifact is commonly seen when mobilizing the patient due to changes in leveling of the transducer. Physical therapy is contraindicated if the catheter is in the wedged position.[63] See Appendix 3 for a picture of various waveforms
- The actual risk of potential PAC complications arising from positional changes is not known, as there are limited reports directly measuring the effects of positional changes and mobility however one 2015 study did demonstrate safe mobility, including ambulation, without PAC complications.[70]
- At Brigham & Women's Hospital, when the patient is medically appropriate for PT and continues to have a PA line in place, activity orders are often for therapeutic exercise and/or bed to chair transfers. However, patients at times are liberalized to ambulation. The therapist should seek appropriate activity orders. To minimize movement in a locked PA line, ipsilateral shoulder flexion and abduction is limited to 90 degrees.
- Patients being admitted for management of acute decompensated heart failure or pending orthotopic heart transplant often have PA lines placed long-term for tailored medical therapy. In these instances, a standing exercise program and/or seated stationary biking is often utilized for optimization of the patient's aerobic capacity.

IABP

- Intra-aortic balloon pump (IABP) are temporary devices used to support cardiac pump function and improve blood flow to the myocardium. A flexible catheter with a balloon mounted on its end is inserted, usually through the femoral artery, and passed into the descending thoracic aorta. Rapid inflation and deflation of the balloon in synchrony with cardiac contraction causes counterpulsation due to volume displacement and pressure changes within the aorta.[71] Balloon deflation augments forward flow, balloon inflation redirects flow into the coronary arteries.[63]
- Patients with IABPs are typically hemodynamically unstable and inappropriate for therapeutic exercise and mobility programs. Protection of the catheter's integrity is of

utmost importance.[71] In specific cases, the therapist may receive a consult from the MD for therapeutic exercise to uninvolved extremities. In these instances, movement and activity should be limited to avoid disruption of the catheter, balloon rupture, or incision site infection.

- Minimize extreme joint range of motion where the IABP is present.[63]
- Avoid hip flexion greater than 70 degrees on the side where the catheter is inserted.[63]
- Other institutions have demonstrated safe mobility with patients with femoral IABP placements, however current practice at BWH is for these patients to remain on bedrest. Use of this type of support is often transient, as either a bridge to recovery or transition to a more durable level of support

Impella

- The Impella devices are a continuous flow axial pumps contained within a pigtail catheter that are designed to offload pressure from the ventricle by augmenting forward blood flow and increasing cardiac output.
- They are most commonly used as a left ventricular support device with the catheter placed in a retrograde fashion, with the inlet pump sitting in the left ventricle and outlet in the ascending aorta. This creates a continuous flow of blood from the left ventricle to the proximal ascending aorta.[72]
  - The three most common versions available are the Impella 2.5, CP, and 5.0 which have a maximum flow of 2.5 L/min, 3-4 L/min, and 5.0 L/min respectively.
    - The Impella 2.5 and CP devices can be deployed percutaneously via the femoral artery and the 5.0 device requires a surgical cutdown but can be placed either via the femoral or axillary artery.[73]
  - The external controller system allows for manual adjustments in rotational speed to alter flow and cardiac output, and houses the infusate of heparin and dextrose, designed to prevent clotting and contamination of the motor by blood.
- This device is employed in situations of clinical deterioration or progressive cardiogenic shock not otherwise managed pharmacologically. It has been documented for use in patients with cardiogenic shock for a variety of reasons including acute and post MI, ischemic cardiomyopathy, myocarditis, and acute cardiac transplant graft failure, used as either a bridge to recovery or a transition to another more durable device. Impella can also be employed as a transition from IABP or EMCO, in cases where longer recovery time is needed. The device is approved for 6 hours of use, however has been used off-label for days to weeks.[74]
- Mobilizing patients who require Impella support with an axillary cannulation has been documented as safe and feasible with benefits to the patient.[73,75,76] However, it is recommended that a patient with a femorally inserted Impella maintain bedrest.[77]
- At BWH, patients with femorally inserted devices are on bedrest; however, if the device is thought to be needed long term, they have been converted, when able, to proximal cannulation to allow for mobility.

- There are no standard mobility protocols or precautions for these devices however typically ipsilateral shoulder movement is kept to a minimum, maintained below 90°. Monitoring vital signs for response to exercise and device flow is important.
- Working with the Impella specialists can help identify changes to device settings that may be important during PT sessions in order to support adequate cardiac output with potentially increased venous return due to exercise

ECMO

- Extracorporeal Membrane Oxygenation (ECMO): Utilized in cases of severe acute respiratory failure or cardiac failure/cardiogenic shock, when other forms of life support are insufficient for sustaining body functioning. ECMO can be placed either venoarterial (VA) and venovenous (VV) for support. Both methods of cannulation provide respiratory support (i.e. support for gas exchange), but only VA ECMO provides hemodynamic support by supporting both the lungs and the heart.[78] This standard of care will focus on VA ECMO cannulation for support of significant cardiac compromise.
- Indications for VA ECMO include:
    - Cardiac/circulatory failure and refractory cardiogenic shock
    - Massive PE with cardiac compromise
    - Cardiac arrest
    - Failure to wean from cardiopulmonary bypass after surgery
    - As a bridge to recovery, cardiac transplant, or placement of durable VAD support.[78]
- With VA ECMO support, venous blood will enter the external device to be oxygenated and then recirculated back into the arterial circulation to supply all body tissues. As stated above, this system augments the heart and lungs to provide respiratory and hemodynamic support. In the oxygenator, hemoglobin is saturated with O_2 and CO_2 is removed. Oxygenation of the blood is determined in part by the flow rate through the device. CO_2 elimination is determined by the sweep gas, typically 100% oxygen, and the sweep flow rate.[78]
- VA ECMO cannulation:
    - Venous cannula is placed in either the inferior vena cava or the right atrium, preferably via the femoral vein.
    - Arterial cannulation: The blood is returned to arterial circulation typically via the right femoral artery to the left ventricle or aorta.
        - Although femoral access is preferable due to ease of insertion, the primary complication associated with this is the risk for ipsilateral lower extremity vascular compromise and ischemia. A reperfusion cannula can be used, placed distally to the femoral artery cannula, redirecting part of the infused blood directly back to the leg.[78]
    - Additional complications of VA ECMO, with the blood return to the aorta potentially competing with native, antegrade circulation, are a separation of upper

and lower body perfusion (watershed phenomenon), left ventricular distention, and pulmonary edema.[79]

- VA ECMO cannulation can occur proximally as well, via the R common carotid or subclavian arteries, allowing increased ease of mobility.[78]
- Literature supports mobilization on VA ECMO, including ambulation, even with femoral cannulation,[80] however current practice at BWH is still largely on a case by case basis. It has been demonstrated as feasible and safe in a small population of patients.
- PT considerations for patients on ECMO:
    - Please note, treating a patient on ECMO support is an advanced skill and requires consultation by a team lead or clinical specialist. Please work in consultation with respiratory therapy as well for documentation of sweep, SvO_2, and flow rates
    - Sweep: gas exchange measured in flow (L/min) and FiO2. The flow is indicative of rate of gas exchange (removal of CO2) and the fraction of delivered O2 is FiO2.
    - SvO_2: measure of mixed venous oxygen saturation, normal values are ~75%. Drops in SvO_2 can be seen with activity and increasing exertion however care should be taken to minimize the drop. Seek parameters with the ECMO team. Often changes to the sweep can help maintain appropriate levels.
    - Arterial BP: can be low and relatively static with minimal to no difference between systolic and diastolic readings, depending upon the flow rate through the ECMO circuit (i.e. how much is the device unloading the heart).
    - Flow: monitoring flow rates through the circuit is important with all activity to ensure adequate functioning of the device and perfusion of the body
    - Range of motion for femoral cannulation: although no formal guidelines have yet been developed, PT intervention has maintained a hip flexion angle of less than 90° due to risk of compressing the cannula and creating a permanent kink in the line.
    - Collaborate with the ECMO specialists to make appropriate adjustments to ECMO settings to support mobility and exercise. This can include increasing the sweep for improved SvO_2 with activity
    - Ensure safety and security of the ECMO cannulas to the patient with an appropriate device (including but not limited to: Foley strap, drain secure, headband, abdominal binder) prior to and during mobilization

Medications

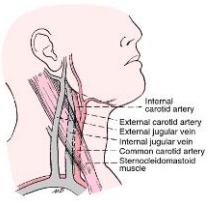
Therapists should review and be familiar with a patient's current medication list. The therapist should especially be aware of any intravenous medications being used, and become familiar with their actions and potential side effects. The administration of certain medications generally indicates that the patient is hemodynamically unstable and/or has complex medical issues. Discussion regarding the goals of PT intervention on a case-by-case basis should occur prior to PT treatment session with staff mentor, clinical specialist, or PT supervisor and/or a member of the physician team. Clarify with the MD an appropriate activity level for patients on vasoactive

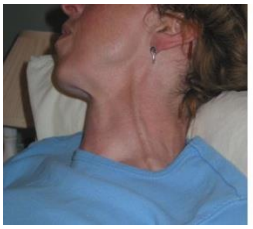
medications: vasodilator, inotropic/vasopressor, antiarrhythmic, and antianginal. Common cardiac medications are listed in Appendix 2.

Cardiac Physical Exam

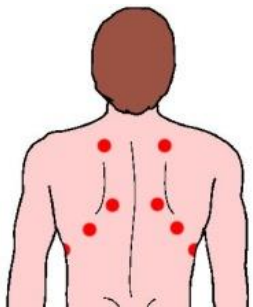
The outline below is designed to guide the therapist through relevant components of a physical exam on a patient with cardiac dysfunction, or to assist with interpretation of a chart review. Note that components may not apply to all patients.

- General appearance
  - Body type
  - Posture- tripod, semi-fowler
  - Skin tone
  - Equipment, lines, and support devices
- Neck
  - JVD
    - Assessment for distention of the right Internal Jugular vein (IJ) is a difficult skill. Its importance lies in the fact that the IJ is in straight-line communication with the right atrium. The IJ can therefore function as a manometer, with distention indicating elevation of Central Venous Pressure (CVP). This in turn is an important marker of intravascular volume status and related cardiac function.
      - Position: Sitting or recumbent in bed with HOB elevated at least 45 degrees, head turned to left. https://www.youtube.com/watch?v=NH8YM8DDhkk



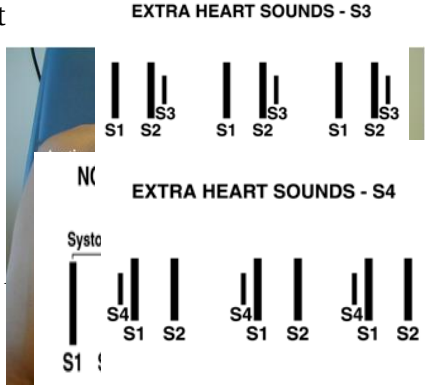


  - Accessory muscle use, SCM hypertrophy
- Chest
  - AP diameter (i.e. barrel chest), scoliosis/kyphosis, pectus excavatum vs pectus carinatum
  - Hands on lower lobes to assess excursion, respiratory rate, and breathing pattern
    - Common breathing patterns
      - Tachypnea: increased respiratory rate
      - Hyperpnea: normal rate but deeper, seen with emotional stress, diabetic ketoacidosis
    - Fremitus: vibration that is produced by the voice or by presence of secretions in airways and is transmitted to the chest wall and palpated by hand.
  - Cough (and how to document)
    - Types: productive, non-productive, dry, congested, moist, persistent, strong, weak, spontaneous
    - Sputum: color, amount, smell
  - Auscultation of lungs (using diaphragm of stethoscope)
    - For examples of general abnormal sounds: https://www.youtube.com/watch?v=gOB0nM0PRTc

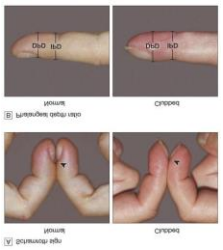


- Listen for normal, abnormal or adventitious sounds
  - Diminished, caused by:
    - Hyperinflation: COPD
    - Hypoinflation: acute lung disease- atelectasis, pneumothorax (PTX), pleural effusion
  - Absent: Pleural effusion, PTX, severe hyperinflation, obesity
  - Adventitious:
    - Bronchial: Consolidation, atelectasis with adjacent patent airway
    - Crackles/Rales/Ronchi: https://www.youtube.com/watch?v=5wEMGd1PRrg
      - Secretions if biphasic
      - Deflation or edema if monophasic
    - Wheezes
      - Diffuse airway obstruction if polyphonic
      - Localized stenosis if monophonic
- Extrapulmonary adventitious sounds
  - Crunch: mediastinal emphysema
  - Pleural rub: pleural inflammation or reaction: https://www.youtube.com/watch?v=36t0vMrcrKU
  - Pericardial rub: pericardial inflammation

- Auscultation of heart (bell of stethoscope)
  - Normal sounds: S1/S2 "lub-dub"
    - *S1* is the sound which marks the approximate beginning of systole, and is created by the *closing of the tricuspid and mitral* due to increased intraventricular pressure during systole. The ventricles continue to contract throughout systole, forcing blood through the aortic and pulmonary, or semilunar valves. At the end of systole, the ventricles begin to relax, the pressures within the heart become less than that in the aorta and pulmonary artery, and a brief back flow of blood *causes the semilunar valves to snap shut, producing S2*.
  - Abnormal sounds: S3/S4
    - An *S3* is most commonly associated with left ventricular failure and is caused by blood from the left atrium rushing into an already overfilled ventricle during early diastolic filling, "S3, Slosh-ing-in". The *S4* is a sound created by blood trying to enter a stiff, non-compliant left ventricle during atrial contraction (beginning of systole). It is most frequently associated with left ventricular hypertrophy



that is the result of long standing hypertension, "S4 A-Stiff-Wall".
        - Murmurs and mechanical valves
            - https://www.youtube.com/watch?v=6YY3OOPmUDA
- Extremities – assess for:



    - Digital clubbing
    - Coloration
    - Cyanosis – Perioral, distal extremities
    - PAD: Pale, loss of hair, shiny, diminished pulse, cold, painful (claudication), ulceration (heel, malleoli, anterior shin), numbness/tingling
    - PVD: Brownish coloration, edema, ulceration (above medial malleolus, calf, dorsum of foot), sometimes numbness/tingling
    - Edema
        - None
        - Non-pitting
        - 1+ Mild pitting, barely perceptible impression when finger is pressed into the skin
        - 2+ Moderate pitting, slight indentation, skin rebounds <15 seconds
        - 3+ Deep pitting, deeper indentation, skin rebounds 15-30 seconds
        - 4+ Very deep pitting indentation, skin rebounds >30 seconds
    - Pulses: Brachial, radial, PT, DP

Exercise/endurance testing:

- Assess baseline level of function, both just prior to admission and when last feeling well, as appropriate
    - Take note of any recent or frequent hospitalizations. Be sure to check in "Chart Review" in EPIC to see PT notes from previous admissions, if applicable, to help guide understanding of functional status and assist in determining a change in status.
- Use of standardized endurance testing is encouraged for all patients as clinically stable and able.[81-84]
    - **6MWT**: The 6MWT can be used to quantify aerobic capacity and endurance. Results of the test can be compared to age-predicted norms using an equation developed by Gibbons et al[82] for all patients 22-79. A spreadsheet for calculating this information is located on the T drive. The spreadsheet additionally calculates average gait speed and average METs.
        - Documentation should include: distance walked in feet and meters, percent predicted, ability of the patient to complete the test, adverse events, and vital signs per/during/post including rate of perceived exertion (RPE).
        - Gait speed and METs can be used to help with the development of goals and patient education regarding return to activity.

    - For patients unable to tolerate or otherwise complete a 6MWT, the 2-minute walk test is an option. See Appendix 1
- Monitor patient for significant tachy- or bradycardia that is new or changed from baseline as this may indicate an abnormal response to the activity being performed or identify a person not ready to participate in activity with PT.
- Use of HR for exercise prescription in patients with cardiac health conditions is not always effective given the effects of cardiac medications, specifically Beta Blockers[85], on heart rate. Use of the RPE scale to monitor exertion levels is the best way to determine safe exercise parameters in the acute care setting, since peak HR cannot always be determined by a pharmacological or exercise stress test.
    - In general, however, the ability for the heart rate to respond to changing activity is considered a good prognostic factor after cardiac event, while a decrease in heart rate (>5 beats/min) with increased activity *or* abnormal HR increase for the level of work being done is considered a poor prognostic indicator.[86]

PT Intervention/Aerobic Exercise Training:

Endurance Training:

- Whenever exercise testing is utilized, it is important to monitor the patient closely for symptoms of exercise intolerance and hemodynamic response. Data extracted from exercise testing will then assist the therapist in providing the patient with an appropriate and safe exercise prescription, likely in the form of a home exercise program.
- Designed by choosing the mode, intensity, frequency and duration based on patient presentation and previous tests and measures, such as the 6MWT or 2MWT. Be sure to include a warm-up and cool-down. The recommendations below are based on the ACSM recommendations for an inpatient exercise program for patients with cardiac dysfunction.[87]
    - *Mode*: Walking
        - You could consider a stationary bike or restorator depending on the patient's prior level of function or symptom burden
    - *Intensity/Duration*: Determined by patient's symptoms (DOE) and RPE during exam or during a 6MWT in combination with HR response. It is recommended to start an exercise program at a lower intensity (RPE </= 5/10).
        - Consider using interval training with multiple short bouts of exercise with rest breaks in between to allow for longer duration of exercise without excessive symptoms. If using interval training, the goal would be to increase the time of the exercise bout and decrease the rest time. You should attempt to achieve a 2:1 ratio of exercise/rest. A good starting point would be 3-5 min of work, followed by 1-3 min of rest (slower walk or complete stop, at the patient's discretion).
        - If a patient can walk for 6 minutes with a low RPE, then intervals of time greater than 6 minutes are indicated. If the patient is unable to complete the 6-minute interval, intervals based on 6MWT results can be used to

achieve an aerobic training effect (may only be 1-2 or 5-6 intervals to achieve this aerobic training effect). Rest intervals can also be determined using the 6MWT and should be included in the exercise prescription.
    - *Frequency*: 2-4x/day, 5-7x/week
    - *Progression*: When continuous exercise duration reaches 10-15 min, increase intensity as tolerated within the recommended RPE and HR limits.
- Be sure to quantify fatigue and dyspnea during functional mobility and ambulation using the 0-10 RPE and DOE scales. These scales can also help determine a patient's tolerance for activity and ADLs and can assist in discharge planning, goal-setting and plan of care.

Cardiac Rehab:

- Cardiac rehab (CR) is a comprehensive outpatient program offered to patients with a variety of diagnoses of cardiac dysfunction, designed to improve physical, psychological, social and vocational functioning. The emphasis is on monitored exercise training, however programs additionally offer education on smoking cessation, diet and nutrition, weight loss, stress management, and management of diabetes, lipids, and hypertension.
- CR programs have been shown to reduce all-cause mortality and cardiovascular mortality, decrease symptoms, reduce recurrent MI, improve adherence with medication management, increase exercise tolerance, mood and health-related quality of life, and to reduce hospitalizations.
    - CR programs additionally show great benefits on risk factor modification, including lipid profile, obesity, and psychosocial stress.[88-91]
- Thirty-six sessions are covered, however MI risk reduction has been seen with fewer sessions attended.
- ACCF/AHA clinical guidelines:[92]
    - Class IA recommendation for management of stable ischemic heart disease
    - Class IIA recommendation for chronic stable heart failure
- Covered diagnoses:
    - MI/ACS
    - CABG
    - s/p PCI
    - stable angina
    - Heart valve repair/replacement
    - Heart failure (EF $\leq$ 35%),
    - Heart or heart/lung transplant
    - PAD
- Patients and their families should be educated on the purpose and benefits of cardiac rehab and referred prior to discharge as appropriate.
- A list of certified cardiac rehab centers in Massachusetts, New Hampshire, Maine, Connecticut and Rhode Island can be found on the T-drive and printed for patients to reference based on where they live. Centers are certified by the American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR), https://www.aacvpr.org/.

BRIGHAM HEALTH
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Appendix 1:

Six-Minute Walk Test

Purpose: The purpose of the 6MWT is to have patients perform a standardized test to assess aerobic capacity and to assist in the prescription of an exercise program based on test results. The results of the 6MWT have been shown to aide in the prognosis of medical conditions such as CHF and COPD

- The test consists of standardized instructions to the patient (available on the T drive), with the patient allowed to self-pace the walk based on the instructions.
- The therapist measures distance walked, vital signs pre/during/post walk, and the number of rests required. For practical purposes, HR, SpO_2, and RPE are collected during the walk at 2 minute intervals, with BP measurements taken prior to the test, just after the test, and after a period of recovery if indicated. The test is performed without benefit of a warm-up.
- Document distance ambulated by recording the number of completed laps on a pre-measured hallway. Pace off the additional distances as needed (2ft floor and ceiling tiles in Shapiro)
- Conditions for stopping the test include:
    - Drop in SpO_2 < 80% or if other signs/symptoms of significant desaturation are present (i.e. confusion, stupor)
    - Lightheadedness
    - Level III/IV angina
    - Marked dyspnea or fatigue
    - Severe musculoskeletal pain or vascular insufficiency such as LE claudication
    - Greater than moderate discomfort from any cause
    - Patients on telemetry who demonstrate:
        - Increasing multifocal premature ventricular contractions (PVCs), coupled PVCs, or ventricular tachycardia (3 consecutive PVCs)
        - Rapid atrial dysrhythmias

Normative Values/Prognosis:

- Patient's with chronic heart failure walked an average distance of 310-427 meters depending on the severity of the disease[94]
- Distance walked on the 6MWT has an inverse correlation with the NYHA functional class[94]

- A recent study by McCabe et al. found that 6MWD in patients with HF performed in the inpatient setting prior to discharge can predict hospital readmission in 30 days. Patients with a 30-day readmission walked less than 536ft. Subjects who walked >984ft had a probability of readmission in 30 days < 13%. It has been suggested that persons who ambulate less than 300 meters have significantly increased mortality and morbidity regardless of sex.[20]

Two Minute Walk Test

Purpose: The 2-minute walk test exists as an alternative to the 6MWT for patients who are otherwise unable to complete the longer time/distance due to symptomatic presentation, level of aerobic conditioning, or time constraints. Normative values have recently been established in a small population and work is ongoing to build a robust comparative dataset.[93] Instructions for performing the test are similar to the 6MWT, as are the values collected by the therapist and the ability to utilize the results as an objective starting point for creating an exercise prescription. Age matched norms for healthy, community dwelling adults age 18-85.[93]

Age	Women's Distance (m)	Men's Distance (m)
18-54	183	200.9
55-59	176.4	191.0
60-64	166.4	179.1
65-69	155.2	184.2
70-74	145.9	172.4
75-79	140.9	157.6
80-85	134.3	144.1

Modified Borg RPE

Modified Borg Scale
Rate of Perceived Exertion (RPE)

0 Nothing at all
0.5 Very, very slight
1 Very slight
2 Slight
3 Moderate
4 Somewhat hard
5 Hard
6
7 Very hard
8
9
10 Very, very hard, Maximal

METs[87]

If a patient's functional capacity is >/4 METs, here is an example of how to progress exercise in setting of recent cardiac hospitalization or surgery:

Week	%FC	Total exercise time (min) at %FC	Exercise bout (min)	Rest bout (min)	Number of exercise/rest bouts
1-2	40-50%	10-20	3-7	3-5	3-4
3-4	50-60%	15-30	7-15	2-5	2-3
5	60-70%	25-40	12-20	2	2

- METs reference points:
  - 1 MET: self-care, ADLs, household ambulation, walk 1-2 blocks on level ground, stationary bike very low resistance
  - 2-5: carrying up to 2-5 lbs, cleaning windows, raking leaves, golf, tennis, biking up to 8 mph
  - <4 METs: do light housework
  - >/4 METs: climb a flight of stairs, walk uphill, walk at 4 mph, heavier housework, moderate recreational activities

Appendix 2:
Common Cardiac Medications

Class	Drug	Indication	Mechanism	Admin Route	Onset	PT Implications
ACE Inhibitors (-pril)	Captopril Lisinopril	-CHF -HTN	Decrease cardiac workload Decrease peripheral vascular resistance. Also prevent Na+ and water retention Promote vasodilation	PO	15-60 minutes	Hypotension Skin rash Dry Cough
Beta Blockers (-lol)	Metoprolol* Atenolol* Esmolol* Labetalol Carvedilol *Indicates cardio-selective*	-Arrhythmias -CHF -Angina -HTN	Decrease cardiac workload Decrease heart rate and contractility. Some may also produce peripheral vasodilation	PO IV (see IV meds below)	15-60 minutes	Bradycardia Hypotension Arrhythmias Excessive negative inotropic effect
Calcium Channel Blockers	Amlodipine Diltiazem Verapamil	-Arrhythmias -Angina -HTN	Decrease rate of discharge of SA node. Inhibit conduction velocity through AV node. Causes coronary vasodilation by blocking Ca+ from entering vascular smooth muscle, which increases O2 supply	PO IV (see IV meds below)	30-60 minutes	Cough Hypotension Dry mouth Edema
Diuretics	Hydrochlorothiazide Furosemide	-CHF -HTN	Decrease cardiac workload. Decrease volume of fluid that heart	PO	30-60	Hypotension

	Torsemide Spironolactone		must pump. Decreases fluid accumulation	IV (see IV meds below)	minutes	Electrolyte imbalance
Vasodilators	Nitrates (ie Nitroglycerin) Hydralazine Milrinone Nifedipine	-CHF -HTN -Angina	Decrease cardiac workload. Promote dilation of peripheral vasculature – decreased cardiac preload and afterload Decreases peripheral and vascular resistance	PO IV (see IV meds below)	Nitro: 1-3 minutes 20-45 minutes	Tachycardia Hypotension Palpitations Fluid Retention
Angiotensin II Receptor Antagonist	Losartan Valsartan	-HTN -CHF	Block effects of angiotensin II on vasculature, which causes blood vessels to dilate	PO	1-2 hours	Hypotension Cough (less likely than with use of ACE inhibitors)
Positive Inotropic Agents	Digoxin Milrinone	-CHF -Afib	Increase myocardial contractility. May also help normalize autonomic effects on the heart	PO IV (see IV meds below)	30-120 minutes	Hypotension Arrhythmias Toxicity (Digoxin)
Anti-arrhythmic	Amiodarone	-Arrhythmias	Delay repolarization of cardiac cells – slows and stabilizes heart rate	PO IV (see IV meds below)	3-7 hours	Increase in arrhythmias Pulmonary toxicity
Pulmonary Vasodilators	Sildenafil Tadalafil Remodulin Flolan/Veletri	-Pulmonary HTN	Dilates pulmonary arteries	PO IV (see IV meds below)	1-2 hours	Dry Mouth Irregular HR Hypotension
Lipid Management	Atorvastatin Lovastatin	-HLD	Increased LDL-receptor activity Inhibits cholesterol Synthesis		1-2 hours	Myopathy

(-statins)	Pravastatin Simvastatin			PO		
Anti-platelet agents	Aspirin Clopidogrel (Plavix) Ticagrelor	-unstable angina -s/p MI -s/p CVA	Decreased platelet aggregation and inhibit thrombus formation	PO	Within two hours	Increased risk for bleeding
Anti-coagulants "Blood thinners"	Coumadin Apixaban Xarelto	-DVT/PE -s/p Mechanical heart valve replacement -Afib	Prolong time it takes for blood to clot and inhibits blood clot formation.	PO	12-24 hours	For Coumadin: monitor INR levels Increased risk for bleeding For Heparin: Monitor PTT
	Lovenox			Subcutaneous Injection	3-5 hours	
	Heparin Bivalirudin			Subcutaneous Injection IV (see IV meds below)	20-60 minutes	

Common Cardiac/Critical Care IV Medications

Class	Drug	Indication	Mechanism	Dosing (gtt)	Onset	Half Life	PT Implications
Vasopressors /Inotropes	Dobutamine	Hypotension Shock Low cardiac output CHF	Increases the strength and force of the heartbeat, causing more blood to circulate through the body. Does not cause	2-20 mcg/kg/min	1-10 min	2 min	Trend BP Can be administered on step down floor

		Augment diuresis	vasoconstriction or tachycardia				
	Dopamine	Hypotension Bradycardia	Low doses increase myocardial contractility to increase cardiac output. High doses cause vasoconstriction to increase BP.	2-20 mcg/kg/min	5 min	2 min	Trend BP Can be administered on step down floor
	Epinephrine	Decreased atrial & ventricular contractility Bradycardia	Increases the coronary artery pressure thereby promoting increased coronary blood flow	0.05-1 mcg/kg/min	Rapid, w/in mins		Trend BP*
	Levophed (Norepinephrine)	Decreased cardiac output Bradycardia Hypotension Decreased coronary artery flow	Causes heart muscle vasodilation and peripheral muscle vasoconstriction	0.01-3 mcg/kg/min	Rapid, w/in secs		Trend BP*
	Neosynephrine (Phenylephrine)	Bradycardia Decreased cardiac output	Exhibits rapid and extended vasoconstrictor actions	10-220 mcg/min	Rapid, w/in secs	1.5 hours	Trend BP*
	Vasopressin	Hypotension	Increases peripheral vascular resistance to increase arterial BP	0.01-0.1 Units/min	Rapid, w/in mins	<10 minutes	Trend BP*

	Primacor (Milrinone)	Acute CHF	Inotropic vasodilator, increases contractile force in the heart muscles	0.375-0.75 mcg/kg/min	5-12 min	2.4 hours	Trend BP
Vasodilators	Nipride (Nitroprusside)	Acute CHF Hypertensive crisis Hypotensive induction (for surgery)	Hypotensive agent relaxes smooth muscle of blood vessels, dilate peripheral arteries and veins; promotes peripheral pooling and decreases venous return to reduce preload (left ventricular end-diastolic pressure and pulmonary capillary wedge pressure); decrease in afterload (systemic vascular resistance, systolic and MAP)	0.3-4 mcg/kg/min	<2 min	2 min	Trend BP
	Nitroglycerin	Angina CHF HTN Hypotension induction (intraoperative)	Vasodilating agent that relieves tension on vascular smooth muscle and dilates peripheral veins and arteries; improves the contractile state in smooth muscle which results in vasodilation	1-200 mcg/min	Rapid, w/in secs	~ 3 minutes	Exercise tolerance may be blunted; angina; HR, BP, wedge pressure

Antiarrhythmic	Amiodarone (Similar meds: Sotalol)	Supraventricular arrhythmias (afib/flutter)	lengthen the cardiac action potential	initial 300 mg IV over 1 hour, f/b 10-50 mg/hr IV over 24 hours, then switch to p.o. For CV of Afib or for vent arrhythmias: 150 mg IV over 10 min, then 1 mg/min IV for 6 hours, then 0.5 mg/min IV for 18 hours or switch to p.o	-	10-50 days	HR, SpO2
	Digoxin	Afib Heart Failure	Reduced sympathetic response	8 to 12 mcg/kg		1.5-2 days	Renal function, electrolyte levels
	Brevibloc (Esmolol)	-Angina -Acute MI -CHF -Hypertension	Beta Blocker; Block sympathetic activity; reduces rate and conduction	50-200 mcg/kg/min	2-10 min	~9 mins	BP, HR
	Lidocaine	-Ventricular arrhythmias (a/w MI/ischemia) -Ventricular fibrillation	Depresses diastolic depolarization and automaticity in the ventricles. Has little effect on atrial tissue.	1-4 mg/min	45-90 seconds	~2 hours	ECG

	Quinidine	-Afib/flutter	Sodium-channel blockade	0.25 mg/kg/min		6-8 hours	ECG, BP
	Procainamide	-Ventricular arrhythmias	increases effective refractory period and reduces impulse conduction velocity and excitability in the atria, His-Purkinje fibers and ventricular muscle of the heart	20 to 50 mg/min		3-4hours	BP, ECG
	Cardizem (Diltiazem)	-Atrial Arrhythmia -HTN -Paroxysmal supraventricular tachycardia -Stable angina	Ca^{2+} Channel Blocker; most effective at SA & AV nodes; reduce rate and conduction	5-20 mg/hr	minutes	3.4-4.9 hours	Vitals
	Cardene (Nicardipine)	HTN Stable angina, chronic	Ca^{2+} Channel Blocker; affects the contractile functions of cardiac and vascular smooth muscle	2.5-15mg/hr	10 min	14.4 hours	Vitals
	Isoproterenol (Isoprel)	Bronchospasm Cardiac Arrest Heart Block	Beta Agonist; lowers peripheral vascular resistance and diastolic pressure; prevents bronchoconstriction	2-10 mcg/min	seconds		BP, HR, ECG, CVP, ABG

Diuretic	Bumex (Bumetanide)	Edema (CHF)	Potent loop diuretic, inhibits reabsorption of sodium and chloride, enhances excretion of potassium; increases serum uric acid and reduces uric acid excretion	1mg/hr	2-3 min	1-1.5 hours	Ototoxicity may occur, Check labs (electrolytes)
	Lasix (Furosemide)	Edema (CHF, renal failure) HTN Pulmonary Edema	Potent diuretic blocks the absorption of sodium and chloride; increases urine output	10-20 mg/hr	5 min	~2 hours	Ototoxicity may occur, Check labs (electrolytes)
Anticoagulant	Heparin	Afib PE/DVT	Inhibits blood clotting	Weight or PTT based	Seconds	1.5 hours	Increased risk of bleeding; check PTT
	Bivalirudin	Angina PCI Can be used when patients have HIT	Helps prevent the formation of blood clots	0.75 mg/kg as an IV bolus dose, followed immediately by 1.75 mg/kg/hr	seconds	25 minutes	Increased risk of bleeding
Pulmonary Vasodilators	Remodulin	Pulmonary HTN	Dilates arteries and decreases amount of blood clotting platelets	1.25 ng/kg/min		4 hours	Hypotension Arrythmias
	Flolan/Veletri	Pulmonary HTN	Synthetic prostaglandin that dilates blood vessels and decreases stickiness of platelets	2 ng/kg/min	Within a minute	3-5 minutes	Hypotension Tachycardia Chest pain

Sedatives	Diprivan (Propofol)	Procedures requiring sedation Mechanically vented adults	Short acting. Decreases level of consciousness	5-50 mcg/kg/min	seconds	3-12 hr	Consider dose and ability to follow commands/ participate in PT
	Precedex (Dexmedetomdine)	Procedures requiring sedation Mechanically vented adults	Can provide semi-arousal sedation. Provides sedation without high risk of respiratory depression	0.2-0.7 mcg/kg/hr	1 min	~2 hours	Consider dose and ability to follow commands/ participate in PT
	Versed (Midazolam)	Used to produce amnesia Anxiety	Depresses the CNS. Used commonly for procedures that do not require general anesthesia; causes relaxation	1-30 mg/hr	1-5 min	~3 hours	Consider dose and ability to follow commands/ participate in PT
	Nimbex (Cisatracurium)	Surgical procedures Mechanically assisted breathing, or insertion of breathing tube.	Skeletal muscle relaxant that blocks the effects of acetylcholine.	1-10 mcg/kg/min	2-3 min	22 minutes	Hold PT, pt paralyzed and unable to participate

Appendix 3

Parameter	Normal Range	Clinical Significance
Pulmonary Artery Pressure (PAP)	20-30 mmHg/0-6 mmHg	Indicates state of resistance in pulmonary vasculature and right ventricle function ↑: pulmonary artery hypertension, COPD or emphysema, PE, pulmonary edema
Pulmonary Artery Wedge Pressure (PAWP)	4-12 mmHg	Indicates left ventricle function ↑: LV failure with pulmonary congestion ↓: LV compliance (hypertrophy, infarction)
Central Venous Pressure (CVP)	0-6 mmHg	Indicates volume status and right ventricle function ↑: overhydration, increased venous return, RV failure ↓: hypovolemia, decreased venous return
Stroke Volume (SV)	60-80 mL/beat	Amount of blood ejected during systole ↓: impaired cardiac contractility, valve dysfunction
Cardiac Output (CO) = Stroke Volume (SV) x Heart Rate (HR)	4-8 L/min	Amount of blood ejected from the left ventricle in 1 minute ↓: decreased volume, decreased strength of ventricular contraction
Cardiac Index (CI)	2.5- 4 L/min	Relates cardiac output (CO) to body surface area (BSA); CI = CO/BSA ↑: high-output failure due to fluid overload, renal disease, septic shock ↓: CHF, hypovolemia, cardiogenic shock
Mixed Venous Oxygen Saturation (SvO2)	60-75%	Index of oxygenation status that measures that relationship between O2 delivery and O2 demand; reflects cardiovascular tissue perfusion[66]

PAC wave forms

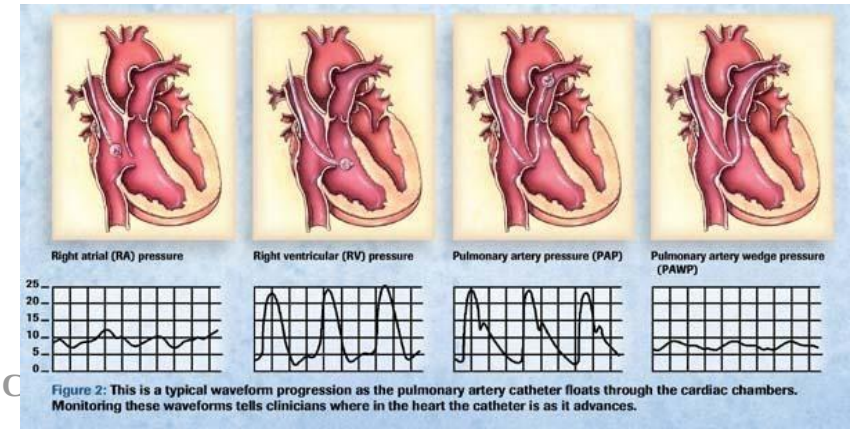

Figure 2: This is a typical waveform progression as the pulmonary artery catheter floats through the cardiac chambers. Monitoring these waveforms tells clinicians where in the heart the catheter is as it advances.

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