Module 02: Coronary Artery Disease, Myocardial Infarction and Heart Failure

- Coronary Artery Disease
- Myocardial Infarction
- Heart Failure

Introduction

Critical care nurses provide specialized care to patients experiencing a life threatening or potentially life threatening illness. The care provided is intensive, complex and continuous. The attributes required to practice critical care nursing include but are not limited to, advanced theoretical knowledge, critical thinking, advanced problem solving, responsible leadership, advocacy, judgment and sound communication skills.

Critical care nurses working with patients who have cardiovascular illness focus not only on the current event but optimization of cardiovascular health across the lifespan. This role requires extensive clinical knowledge and expertise to facilitate safe patient care and promote long term health and wellness.

Module Objectives

- Compare and contrast stable and unstable angina
- Discuss the pathophysiology of coronary artery disease
- State the common complications of a patient experiencing a myocardial infarction
- Identify the medical management and nursing interventions used to treat a patient experiencing a myocardial infarction
- Distinguish between the different types of heart failure.
- Identify the medical management and nursing interventions used to treat a patient experiencing heart failure

Module Checklist

1. Complete assigned readings
2. Watch the Panopto videos
3. Read the Professor Notes
4. Complete Module Quiz

Coronary Artery Disease

https://slcme.hosted.panopto.com/Panopto/Pages/Viewer.aspx?id=4974d381-d079-4cb8-aafb-aafe014dbe17

Matching Activity

Using the dropdown beside each of the factors that determine myocardial oxygen needs, identify whether this is a factor that results in decreased oxygen supply, or increased oxygen demand.

Aortic stenosis	
Anemia	
Anxiety	
Asthma	
Cardiomyopathy	
Cocaine use	
COPD	
Coronary artery spasm	
Coronary artery thrombosis	
Heart Failure	
Low blood volume	
Physical exertion	
Pneumonia	
Tachycardia	
Valve disorders	

Fibrinolytic Therapy

For a patient experiencing an acute ST-elevation myocardial infarction (STEMI), the use of fibrinolytic therapy is an important clinical intervention. The administration of a fibrinolyic agent such as streptokinase (SK) or tissue plasminogen activator (tPA) results in the lysis of the acute thrombus so that the obstructed coronary artery can be opened and blood flow to the affected tissue can be restored. Adjunctive measures, such as the initiation of heparin infusions are then taken to prevent further clot formation and obstruction reoccurrence. Lysis is achieved by converting inactive plasminogen to plasmin, an enzyme that is responsible for the degradation of fibrin.

Patients with recent onset of chest pain with persistent ST elevation and those with underlying bundle branch blocks are ideal candidates for therapy. To ensure maximum effectiveness the a goal of 30 minutes from the onset of the patient's pain to the administration of the agent, should be set.

Following the lysis of the thrombus, there are several phenomena that may be observed.

- Pain
    - as blood is restored to the ischemic myocardium, chest pain will stop abruptly
- Reperfusion dysrhythmias
    - while many different dysrhythmias may occur, accelerated idioventricular rhythms have been shown to be the most frequent with reperfusion
    - These dysrhythmias are usually limited do not require antidysrhythmic therapy unless they are sustained
- ST segment
    - rapid return of ST segments to the isoelectric line indicated restoration of blood flow to the previously ischemic myocardial tissue

- Cardiac biomarkers
    - Continued measurement of the biomarkers serves as evidence of successful reperfusion following administration of a fibrinolytic
- Residual coronary stenosis
    - Even after successful fibrinolysis, residual coronary artery stenosis from the underlying atherosclerotic process remains.
    - Prevention methods are crucial to preserving myocardial function and preventing the risk of late complications

Nursing responsibilities in fibrinolytic therapies include:

- establishing IV access and ensuring continued patency
- obtaining baseline laboratory values and vital signs
- assess and monitor for signs of reperfusion
- assess and monitor for signs of complications related to therapy
- assess and monitor for clinical manifestations of bleeding
- minimize potential for bleeding
- health teaching for patient and family

Interventions via Catheter for Coronary Artery Disease

Catheter procedures are used to open coronary arteries that have been blocked or narrowed by CAD. These interventions are collectively known as *Percutaneous Coronary Intervention (PCI).* While patients undergoing non-emergent/scheduled PCI once stayed in hospital overnight, they are now often discharged the same day as the procedure is performed.

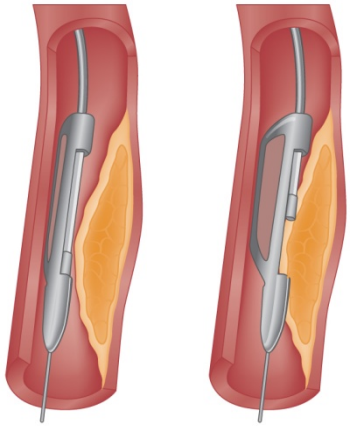
PCI is performed in a cardiac catheterization laboratory using fluoroscopy. Anti-platelet medications are often administered at the beginning of the procedures. An introducer is placed into the femoral, radial or brachial artery.

An example of a PCI via the femoral artery can be viewed here

While the incidence of serious complications after PCI have decreased considerably in recent years, there is still a possibility of coronary artery spasm, coronary artery dissection, renal failure induced by contrast, dysrhythmia, vasovagal response during manipulation of the sheaths and acute coronary thrombosis.

Commonly used interventions are:

- Atherectomy
    - the excision and removal of the atherosclerotic plaque by cutting, shaving or grinding
    - two types of catheters are used
        - directional coronary atherectomy (DCA)
            - rotating cup shaped blade within a windowed cylindrical chamber on one side and low-inflation balloon on the other
            - catheter is positioned in the lesion, balloon is inflated, forcing the plaque into the chamber window

        - cutting blade then used to shave the protruding plaque that is then collected into the chamber
        - 
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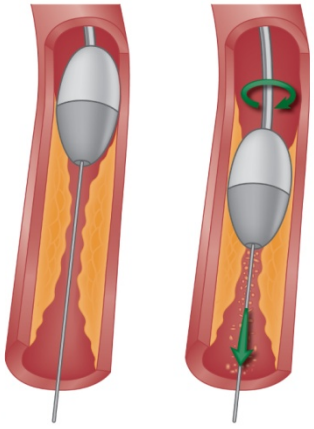
    - rotational ablation
        - high-speed, diamond-cated bur that drills through the plaque that creates tiny particles
        - the particles are carried via the blood stream and disposed of by the various cells in the body that function to remove dead or abnormal cells, tissues and foreign substances
        - most frequently used to debulk heavily calcified lesions that cannot be dilated by angioplasty or prevent stent placement
  - useful for removing plaque in calcified or fibrotic lesions
  - helps to increase wall compliance and facilitate angioplasty and stent placement
  - 
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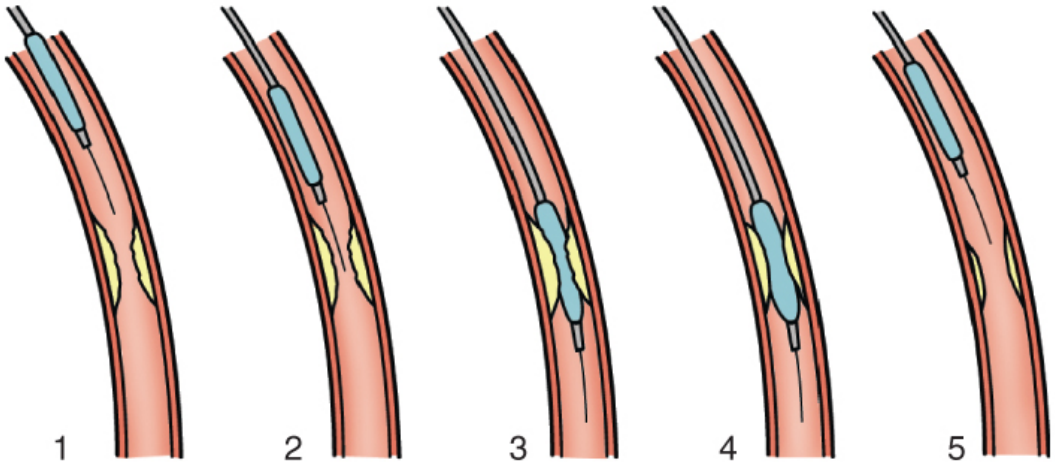
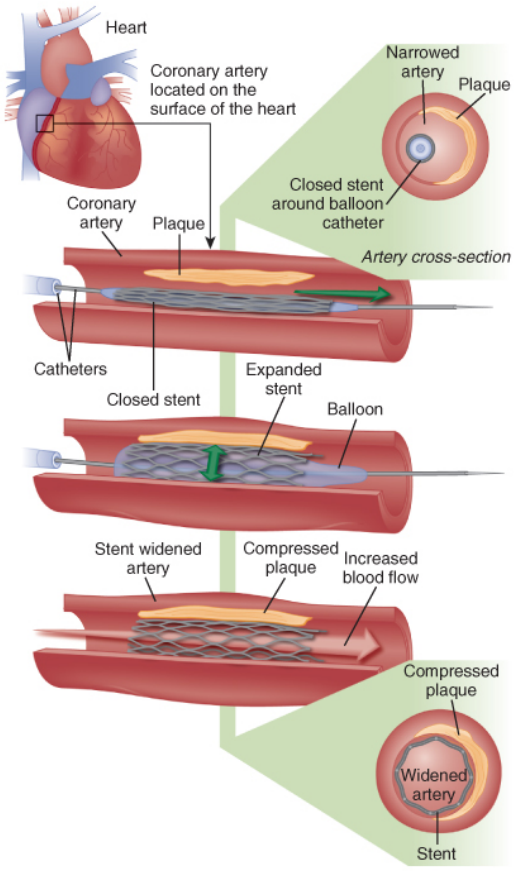
- Balloon Angioplasty
  - percutaneous transluminal coronary angioplasty (PTCA) was introduced in 1977 as an alternative to surgical intervention
  - patients with single or multi-vessel disease my be candidates for PCTA
  - while once impossible, it is now possible for vessels to be totally dilated
  - involves the use of a balloon-tipped catheter that, when advanced through the atherosclerotic lesion can be inflated intermittently for the purpose of dilating the stenotic area and improving blood flow.

- The higher inflation pressure of the balloon stretches the wall of the coronary artery, breaks the plaque into smaller fragments and enlarges the vessel.
- When the balloon is deflated, the vessel does exert its properties of elasticity and results in 30% residual stenosis.
- The procedure is considered successful if the initial stenosis is reduced to less than 50% of the vessel diameter
- The limitations of the treatment are risk of vessel occlusion and restenosis.
- PCTA is not often used in the absence of other treatment such as stent insertion
- 

- Stent Implantation
  - a stent is a metal structure that is introduced into the coronary artery over a guidewire and expanded into the vessel wall at the site of the lesion
  - stents can be placed in large vessels with prominent lesions and in smaller lesions with diffuse disease, in vessels with lesions at bifurcations and vessels with thrombi
  - multiple stents may be implanted sequentially to fully cover a large area of lesion
  - they comprise 90% of all interventional PCIs
  - They are made of many different materials and configurations
    - to prevent restenosis, drug-eluting stents that have polymer coatings that are impregnated with medications that are slowly released at the site of placement to prevent cellular proliferation are often used



Nursing responsibilities following a PCI procedure include care of the vascular access site, management of active closure devices if required, monitoring for any complications and providing patient education regarding post procedure care and risk factor modification.

The Cardiothoracic Learning Package produced by the Liverpool Hospital can be found here. It provides a comprehensive review of previously learned concepts and an introduction of new material regarding cardiac surgery and the care required of this patient population.

Myocardial Infarction

https://slcme.hosted.panopto.com/Panopto/Pages/Viewer.aspx?id=0d01f0c2-87d6-4071-9896-aafe014d988a

Matching Activity

Match the terms with the definitions:

Ischemia
Injury
Infarction

	Penumbra, composed of viable cells. Repolarization is temporarily impaired but is eventually restored to normal
	Still potentially viable tissue. Cells in the area do not fully repolarize because of deficient blood supply. Recorded on ECG as ST segment elevation
	Area of necrotic muscle in the myocardium. New pathologic Q waves are noted on ECG. Lack of depolarization on surface involved.

Heart Failure

Heart failure is not a specific disease, but a clinical syndrome characterized by dyspnea, activity intolerance, and fluid overload adversely affecting patients' functional status and quality of life. Dyspnea and activity intolerance are the two most common symptoms of heart failure, while manifestations of fluid overload, such as edema and jugular venous distention, are the most common signs. This clinical syndrome occurs as a consequence of structural or functional (or both) cardiac abnormalities that impair cardiac pumping or filling.

Heart failure is a clinical syndrome that commonly is the final manifestation of cardiac risk factors (e.g., untreated hypertension) and cardiac diseases or events (e.g., acute myocardial infarction [MI]). Patients with heart failure commonly report very poor quality of life because heart failure has a negative impact on all aspects of life, particularly symptom burden and ability to perform usual activities. Heart failure has an impact on quality of life comparable to, or worse than, that seen in other serious chronic conditions.

Given the enormous personal, social, and economic burdens of heart failure, preventing it from occurring in the first place is a major priority that all healthcare providers must embrace. Once heart failure develops, prevention of both rehospitalisation and progression of heart failure becomes a major priority.

Pathophysiology of Heart Failure

Heart failure is a response to cardiac dysfunction, a condition in which the heart cannot pump blood at a volume required to meet the body's needs. Any condition that impairs the ability of the ventricles to fill or eject blood can cause heart failure. CAD with resultant necrotic damage to the left ventricle is the underlying cause of heart failure in most patients. Other major conditions that lead to heart failure include valvular dysfunction, infection (myocarditis or endocarditis), cardiomyopathy, and uncontrolled hypertension. Hypertension is the precursor of heart failure in men and women.

When the heart begins to fail and the cardiac output is no longer sufficient to meet the metabolic needs of tissues, the body activates several major compensatory mechanisms:

The sympathetic nervous system:

The sympathetic nervous system compensates for low cardiac output by increasing heart rate and blood pressure. Levels of circulating catecholamines are increased, resulting in peripheral vasoconstriction and causing the shunting of blood from nonvital organs to vital organs. This mechanism, although initially

helpful, may become a negative factor if elevation of heart rate increases myocardial oxygen demand while shortening the amount of time for diastolic filling and coronary artery perfusion.

The Renin-angiotension-aldosterone system:

Activation of the RAAS in heart failure promotes fluid retention. A physiologic chain of events is then set in motion that leads to volume overload. To break the RAAS cycle of fluid retention in heart failure, medications are prescribed to interrupt the steps.

Ventricular Hypertrophy:

This is the final compensatory mechanism. Because myocardial hypertrophy increases the force of contraction, hypertrophy helps the ventricle overcome an increase in afterload. When this mechanism is no longer efficient for the ventricle, it will remodel by dilation.

Ventricular Remodeling:

In this final stage of heart failure, the shape of the ventricle is changed, or is remodeled, to resemble a round bowl. A dilated ventricle has poor contractility and is enlarged without hypertrophy.

Types of Heart Failure

All patients do not have the same type of heart failure. The type of heart failure/classification is based upon either the area of the heart affected or the type of dysfunction that occurs.

Left Ventricular Failure: is a disturbance of the contracile function of the left ventricle; resulting in low cardiac output. As a result, the arterial bed vasoconstricts, increasing afterload (systemic vascular resistance). This then, creates congestion and edema in the pulmonary circulation and alveoli. Patients with LV failure are often diagnosed when they present with one or more of the following:

a) Decreased exercise tolerance
b) Fluid retention
c) Discovery during examination of noncardiac problems

Clinical Manifestations of LV Failure	
Signs	Symptoms
Tachypnea Tachycardia Cough Bibasilar crackles S3 and S4 Increased pulmonary artery pressures Hemoptysis Cyanosis Pulmonary Edema	Fatigue Dyspnea Orthopnea Paroxysmal nocturnal dyspnea Nocturia

Right Ventricular Failure: is a disturbance of the contractile function of the right ventricle. While this type of heart failure may result from an acute condition such as a pulmonary embolus, it can be caused by LV failure.

Clinical Manifestations of RV Failure	
Signs	Symptoms
Peripheral edema Hepatomegaly Splenomegaly Hepatojugular reflux Ascites Jugular vein distention Increased central venous pressure Pulmonary hypertension	Weakness Anorexia Indigestion Weight gain Mental changes

3. Systolic dysfunction: the type of failure associated with the decreased contractility during systole, lessens the quantity of blood that can be ejected from the heart (decreased cardiac output).
4. Diastolic dysfunction: normally caused by LV dysfunction, this is when the heart muscle is unable to relax, stretch or filling during diastole.

The Treatment of Heart Failure

The treatment of heart failure is based upon the stage of heart failure they are exhibiting.

Stage A:

The patient is at risk for heart failure but without structural heart disease or symptoms of heart failure. These are patients diagnosed with hypertension, diabetes or metabolic syndrome, atherosclerosis, peripheral vascular disease and/or obesity.

The management for patients with stage A heart failure involves the treatment of risk factors for heart failure and its precursors. For example:

- Treat hypertension aggressively
- Treat dyslipidemias
- Control diabetes and metabolic syndrome
- Smoking cessation
- Promote regular exercise
- Obesity control
- Other measures to prevent atherosclerosis and coronary artery disease
- Discourage excess alcohol intake, illicit drug use
- Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers in at-risk patients

Stage B:

The patient has structural heart disease without heart failure symptoms. These are patients who may have previous myocardial infarction, left ventricular hypertrophy and low ejection fractions.

The management for patients with stage B heart failure involves all those in stage A, as well as the introduction of beta blockers unless contraindicated.

Stage C:

The patient has structure heart disease with prior or current heart failure symptoms such as; shortness of breath, fatigue or activity intolerance.

The management for patients with stage C heart failure involves all those in stages A and B, as well as:

- All stage A and stage B lifestyle measures
- Dietary sodium restriction
- Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers in at-risk patients
- Beta-adrenergic receptor blockers
- Diuretics
- Digoxin
- Aldosterone antagonists
- Hydralazine/nitrates
- Biventricular pacing or implantable defibrillators in select patients

Stage D:

The patient has refractory heart failure that requires specialized interventions because they are experiencing symptoms at rest despite optimal therapy.

These interventions include:

- All measures used in stages A, B and C
- Mechanical assist devices
- Heart transplantation
- Palliative or hospice care

Matching Activity

Put the following events that are the consequences of heart failure in sequence.

Heart Failure results in low CO
Angiotensinogen is manufactured and released by the liver
Low CO causes renal vasoconstriction, stimulating release of renin
Renin coverts angiotensinogen into angiotensin I
Angiotensin passes through the pulmonary vascular bed and converted to angiotensinogen II
Angiotensin II causes vasoconstriction
Angiotensin II causes release of aldactone by the adrenal glands
Na and H2O are retained due to the aldosterone secretion
SVR worsens, symptoms of heart failure increase because of increased preload

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Summary

The development of any acute coronary syndrome (unstable angina or myocardial infarction) results from the rupture or erosion of atherosclerotic plaque. The ruptures results in the adhesions of platelets to the plaque and the formation of fibrin clots and the activation of thrombin.

Patients with certain risk factors are at a greater likelihood of developing an acute coronary syndrome. The degree and duration of the blockage determine the type of occlusion and subsequent ischemia or infarction.

The responsibility of the critical care nurse is to have a comprehensive knowledge of the pathophysiology of these disorders and intervene rapidly to minimize long term sequelae of cardiac events.

References:

- Lewis, S.L., Bucher, L., Heitkemper, M., Harding, M.M., Barry, M., Lok, J., Tyerman, J., Goldsworthy, S., Kwong, J. & Roberts, D. (2019). Medical-Surgical Nursing In Canada. 4th ed. Elsevier Canada, Ltd. Milton, ON.
- Urden, L.D., Stacy, K.M. & Lough, M.E. (2018). Critical Care Nursing: Diagnosis and Management (8th edition). Maryland Heights, Missouri. Elsevier