Part II: Cardiovascular Disorders:

- Acute coronary syndromes:
  a. Angina pectoris.
  b. Myocardial infarction.

- Heart failure
- Cardiogenic shock.
- Cardiac surgery.
- Nursing management for client undergoing Cardiac Surgery.

Learning Objectives

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

1. Define Concepts

2. Describe the pathophysiology, clinical manifestations, and treatment of acute coronary artery.

3. Use the nursing process as a framework for care of patients with angina pectoris.

4. Describe the pathophysiology, clinical manifestations, and treatment of myocardial infarction.

5. Describe the management of patients with heart failure (HF).

1. Use the nursing process as a framework for care of patients with HF.
2. Incorporate assessment of functional health patterns and cardiac risk factors into the health history and physical assessment of the patient with cardiovascular disease.
3. Describe the causes, clinical manifestation, diagnostic test, medical management and care of cardiogenic shock.
4. Describe the nursing care of a patient who has undergone cardiac surgery.

Acute Coronary Syndrome

- Acute coronary syndrome (ACS) refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non–ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina.
- It is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery.
- Unstable angina is distinguished from stable angina by the new onset or worsening of symptoms in the previous 60 days or by the development of post-MI angina 24 hours or more after the onset of MI.
- When the clinical picture of unstable angina is accompanied by elevated markers of myocardial injury, such as troponins or cardiac isoenzymes, non–ST segment elevation MI is diagnosed.
- The distinction between non–ST segment elevation MI and MI with ST segment elevation is clinically important because acute recanalization therapy is critical for improving the outcome in ST elevation MI but is less urgent in non–ST segment elevation MI.
- Secondary unstable angina should resolve with successful treatment of the precipitating condition.
Classification of Unstable angina

<table>
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<tr>
<th>Class</th>
<th>Severity</th>
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<tbody>
<tr>
<td>Class I</td>
<td>New-onset, severe or accelerated angina (angina &lt;2 months in duration, severe or occurring &gt;3 times/day, or angina that is distinctly more frequent and precipitated by distinctly less exertion; no rest pain within 2 months)</td>
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<tr>
<td>Class II</td>
<td>Angina at rest, sub-acute (angina at rest within the preceding month but not within the preceding 48 hours)</td>
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<tr>
<td>Class III</td>
<td>Angina at rest, acute (angina at rest within the preceding 48 hours)</td>
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Pathobiology

- Plaque rupture or erosion with overlying thrombosis is considered to be the initiating mechanism of ACS, including unstable angina and non-ST segment elevation MI.
- Mechanical factors contribute to plaque disruption. A thin fibrous cap is more likely to rupture than a thick one is, and plaque rupture occurs commonly where the plaque joins the adjacent vessel wall.
- Plaque erosion and plaque rupture can initiate an ACS.
- Erosion usually occurs centrally through a thinning cap rather than at the lateral edge of the plaque.
- The cytokine interleukin-6, which is the main producer of C-reactive protein in the liver, similarly is elevated in unstable angina but not instable angina.
- The stimulus that initiates the acute inflammatory process in ACS has not been identified. *Chlamydia pneumoniae*, cytomegalovirus, and
*Helicobacter pylori* have been identified within human atherosclerotic lesions.

**Causes of ACS**

Atherosclerosis is the primary cause of ACS, with most cases occurring from the disruption of a previously non-severe lesion.

**Signs and symptoms**

1. Chest Pain, which is usually described as pressure, squeezing, or a burning sensation across the precordium and may radiate to the neck, shoulder, jaw, back, upper abdomen, or either arm
2. Dyspnea on exertion.
3. Decreased exercise tolerance
4. Nausea from vagal stimulation
5. Palpitations
6. Diaphoresis from sympathetic discharge

**Diagnosis**

**A: electrocardiography (ECG),** is the most important diagnostic test for angina. ECG changes that may be seen during anginal episodes include the following:
1. Transient ST-segment elevations.
2. Dynamic T-wave changes: Inversions, normalizations, or hyperacute changes.
3. ST depressions: These may be junctional, downsloping, or horizontal.

**B: Laboratory studies**, that may be helpful include the following:

1. Creatine kinase isoenzyme MB (CK-MB) levels.
2. Cardiac troponin levels.
3. Myoglobin levels.
5. Basic metabolic panel.

**C: Diagnostic imaging modalities**, that may be useful include the following:

1. Chest radiography
2. Echocardiography
3. Myocardial perfusion imaging
4. Cardiac angiography
5. Computed tomography, including CT coronary angiography and CT coronary artery calcium scoring

**Management**

**The goals of urgent management include:**

- Stabilizing the patient’s condition.
- Relieving ischemic pain.
- Providing antithrombotic therapy.
1. **Anti-ischemic therapy includes the following:**

   a. Nitrates (for symptomatic relief).
   b. Beta blockers (e.g., metoprolol): These are indicated in all patients unless contraindicated.

2. **Antithrombotic therapy includes the following:**

   a. Aspirin.
   b. Clopidogrel.
   c. Prasugrel.

3. **Anticoagulant therapy includes the following:**

   a. Unfractionated heparin (UFH).
   b. Low-molecular-weight heparin (LMWH; dalteparin, nadroparin, enoxaparin).
   c. Factor Xa inhibitors (rivaroxaban, fondaparinux).

4. **Percutaneous coronary intervention** (preferred treatment for ST-elevation MI)

**Complications of ACS**

- Ischemia: Pulmonary edema
- Myocardial infarction: Rupture of the papillary muscle, left ventricular free wall, and ventricular septum

**Nursing Care Plan for patients with ACS**
Acute pain related to myocardial ischemia as evidenced by severe chest pain and tightness, radiation of pain to the neck and arms.

**Nursing Interventions**

1. Evaluate chest pain (e.g., intensity, location, radiation, duration, and precipitating and alleviating factors) in order to accurately evaluate, treat, and prevent further ischemia.
2. Monitor effectiveness of oxygen therapy to increase oxygenation of myocardial tissue and prevent further ischemia.
3. Administer medications to relieve/prevent pain and ischemia to decrease anxiety and cardiac workload.
4. Obtain 12-lead ECG during pain episode to help differentiate angina from extension of MI or pericarditis.
5. Monitor cardiac rhythm and rate and trends in blood pressure and hemodynamic parameters (e.g., central venous pressure and pulmonary artery wedge pressure) to monitor for hypotension and bradycardia, which may lead to hypoperfusion.

**Ineffective tissue perfusion (cardiac)** related to myocardial injury and potential pulmonary congestion as evidenced by decrease in BP, dyspnea, dysrhythmias, peripheral edema, and oliguria

**Nursing Interventions**

1. Monitor vital signs frequently to determine baseline and ongoing changes.
2. Monitor for cardiac dysrhythmias, including disturbances of both rhythm and conduction, to identify and treat significant dysrhythmias.
3. Monitor respiratory status for symptoms of heart failure to maintain appropriate levels of oxygenation and observe for signs of pulmonary edema.

4. Monitor fluid balance (e.g., intake/output, daily weight) to monitor renal perfusion and observe for fluid retention.

5. Arrange exercise and rest periods to avoid fatigue and decrease the oxygen demand on myocardium.

**Anxiety** related to perceived or actual threat of death, pain, possible lifestyle changes as evidenced by restlessness, agitation, and verbalization of concern over lifestyle changes.

**Nursing Interventions**

1. Observe for verbal and nonverbal signs of anxiety.

2. Identify when level of anxiety changes since anxiety increases the need for oxygen.

3. Use a calm, reassuring approach so as not to increase patient’s anxiety.

4. Instruct patient in use of relaxation techniques (e.g., relaxation breathing, imagery) to enhance self-control.

5. Encourage family to stay with patient to provide comfort.

6. Encourage verbalization of feelings, perceptions, and fears to decrease anxiety and stress.

7. Provide factual information concerning diagnosis, treatment, and prognosis to decrease fear of the unknown.

**Activity intolerance** related to fatigue secondary to decreased cardiac output and poor lung and tissue perfusion as evidenced by fatigue with
minimal activity, inability to care for self without dyspnea, and increased heart rate.

**Nursing Interventions**

1. Assist patient to understand energy conservation principles (e.g., the requirement for restricted activity) to conserve energy and promote healing.
2. Teach patient and significant other techniques of self-care that will minimize oxygen consumption (e.g., self-monitoring and pacing techniques for performance of activities of daily living) to promote independence as well as minimize O2 consumption.
3. Monitor patient’s response to antiarrhythmic medications since these medications will affect BP and pulse prior to activity.
4. Arrange exercise and rest periods to avoid fatigue and to increase activity tolerance without rapidly increasing cardiac workload.

**Ineffective therapeutic regimen management** related to lack of knowledge of risk factors, disease process, rehabilitation, home activities, and medications as evidenced by frequent questioning about illness, management, and care after discharge.

**Nursing Interventions**

1. Appraise the patient’s current level of knowledge related to myocardial infarction to obtain information on patient’s teaching needs.
2. Explain the pathophysiology of the disease and how it relates to anatomy and physiology to individualize the information and to increase understanding.
3. Discuss lifestyle changes that may be required to prevent further complications and/or control disease process to get the cooperation of the patient’s significant support system.

4. Refer the patient to local community agencies/support groups so that the patient and family have resources and support available.

5. Instruct the patient on the purpose and action of each medication.

6. Instruct the patient on the dosage, route, and duration of each medication so that patient understands the reason for taking the medication and will be less likely to refuse to take medications.

**Patient education**

The following mnemonic may useful in educating patients with ACS regarding treatments and lifestyle changes necessitated by their condition:

A = Aspirin and antianginals.

B = Beta blockers and blood pressure (BP).

C = Cholesterol and cigarettes.

D = Diet and diabetes.

E = Exercise and education.

**Myocardial Infarction**

- Myocardial infarction (MI) is a critical emergency that requires timely management to save heart muscle and limit damage that may evolve over several hours.

- Blood flow is abruptly decreased or stopped through the coronary arteries and results in ischemia and necrosis to the myocardium if not treated.
- Cardiac dysrhythmias, mainly ventricular fibrillation, is usually the cause of death in individuals with I.
- MI is usually a disease involving the left ventricle but the damage may extend to other areas, such as the atria or right ventricle.
- A right ventricular myocardial infarction usually has high right ventricular filling pressures and often has severe tricuspid regurgitation.

  a. **Transmural infarcts**, involve the entire thickness of the myocardium and are characterized by Q waves on the electrocardiogram.
  b. **Nontransmural infarcts**, are characterized by S-T segment and T wave changes.
  c. **Subendocardial infarcts**, usually involve the inner portion of the myocardium where wall tension is highest and the blood flow is most vulnerable to circulatory problems.

**Diagnostic tests**

5. **Laboratory:**
   a. leukocyte count, ESR and blood glucose may be elevated.
   b. creatinine phosphokinase (CK, CPK) will normally increase within 4-6 hours, peak between 12-24 hours, and last 2-3 days but should not be used as sole indicator due to possibility of elevation with other problems such as surgery or trauma.
   c. lactate dehydrogenase (LDH) will normally increase within 8-12 hours, peak between 2-4 days, and last 10-14 days but should not be used as sole indicator due to possibility of elevation with
other problems such as liver failure; serum glutamic oxaloacetic transaminase.

d. (SGOT) is occasionally used as an infarct indicator

6. **Chest x-ray**: shows any enlargement of the heart and pulmonary vein.

7. **Electrocardiography**
   - shows indicative changes associated with sites of acute infarcts using Q waves, S-T segment elevation, and T wave inversion.
   - *Also* reveals changes with atrial and ventricular enlargement.
   - rhythm and conduction abnormalities.
   - ischemia, electrolyte abnormalities, drug toxicity, and presence of dysrhythmias.

8. **Echocardiography**: used to study structural abnormalities and blood flow through the heart.

9. **Magnetic resonance imaging (MRI)**: provides a three-dimensional view that can detect changes in tissues before structural damage is done and is safe for pregnant women and children.

10. **Cardiac catheterization**, used to:
    - assess Pathophysiology of the patient’s cardiovascular disorder.
    - provide left ventricular function information.
    - allow for measurement of heart pressures and cardiac output.
    - evaluate stenotic lesions, and
    - measure blood gas content.
Medical Management

The main goals in treating myocardial infarction are to increase blood flow to the coronary arteries which lead to:

a. decrease infarction size.

b. increase oxygen supply.

c. decrease oxygen demand to prevent myocardial death or injury,
   and,

d. control or correct dysrhythmias.

1. **Oxygen**: to increase available oxygen supply

2. **Analgesics**: morphine is the drug of choice, given in incremental doses IV every 5 minutes as needed; IM injections are avoided because they can raise the enzyme levels and do not act as quickly.

3. **Thrombolytic agents**: Streptokinase, Urokinase, or Tissue Plasminogen Activator (tPa) given either intracoronary or intravenously to activate the body’s own fibrinolytic system to dissolve the clot and resume coronary blood perfusion.

4. **Cardiac glycosides**: digitalis to increase force and strength of ventricular contractions and to decrease the conduction and rate of contractions in order to increase cardiac output; usually not used in the acute phase.

5. **Diuretics**: furosemide (Lasix) to promote excess fluid removal, to decrease edema and pulmonary venous pressure by preventing sodium and water reabsorption.

6. **Vasodilators**: hydralazine (Apresoline), nifedipine (Procardia, Adalat), nitroglycerin (Nitropaste, Nitrodur, Nitrostat, Tridil, Nitroglycerine), prazosin (Minipres), captopril (Capoten)-used to relax
venous and/or arterial smooth muscle to decrease preload, decrease afterload, and decrease oxygen demand.

7. **Beta-adrenergic blockers**: used to decrease blood pressure, decrease elevated plasma renins.

11. **Aspirin**: used to decrease platelet aggregation and helps with vasodilation of peripheral vessels

12. **Thrombolytics**: used in the treatment of acute MI; acts by activating mechanisms for conversion of plasminogen to plasmin which is able to dissolve the clot; commonly used are streptokinase, urokinase, alteplase, or anistreplase.

13. **Heparin**: used with thrombolytic protocols, and in the treatment of MI; prevents conversion of fibrinogen to fibrin and prothrombin to thrombin by its action on antithrombin.

14. **Pacemakers**: either temporary or permanent, used in anticipation of lethal dysrhythmias or conduction problems.

15. **Surgery**: coronary artery bypass grafting to reroute the coronary blood flow around the diseased vessel to enable coronary perfusion.

16. **Intra-aortic balloon pump (IABP)**: used to:
   - decreases the workload on the heart.
   - decreases myocardial oxygen demand.
   - increases coronary perfusion, decreases afterload, decreases preload, and helps to limit infarct size if quickly initiated,

**Congestive Heart Failure**
- Heart failure is the inability of the heart to supply blood flow to meet physiologic demands, without utilizing compensatory changes.
There may be failure involving one or both sides of the heart, and over time, causes the development of pulmonary and systemic congestion and complications.

Congestive heart failure, or CHF, is a common complication after myocardial infarction.

Usually following MI, the heart failure is left-sided since most infarctions involve damage to the left ventricle.

**Classification of CHF**

- acute or,
- chronic.

- In chronic heart failure, the body experiences a gradual development as the heart becomes unable to pump a sufficient amount of blood to meet the body’s demands.
- Chronic heart failure can become acute without any overt cause.

**Diagnostic tests**

6. **Laboratory**
   a. electrolyte levels to monitor for imbalances; renal profiles to monitor for kidney function problems.
   b. digoxin levels to monitor for toxicity.
   c. platelet count to monitor for thrombocytopenia from amrinone

7. Chest x-ray: shows any enlargement of the heart and pulmonary vein, presence of pulmonary edema or pleural effusion
8. **Electrocardiography**: used to monitor for dysrhythmias which may occur as a result of the heart failure or as a result of digitalis toxicity

9. **Echocardiography**: used to study structural abnormalities and blood flow through the heart

10. Intra-aortic balloon **pump**: decreases the workload on the heart, decreases myocardial oxygen demand, increases coronary perfusion.

### Medical management

<table>
<thead>
<tr>
<th>Medication</th>
<th>Example</th>
<th>Action</th>
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<tbody>
<tr>
<td>Oxygen</td>
<td></td>
<td>to increase available oxygen supply</td>
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<tr>
<td>Morphine</td>
<td></td>
<td>- induce vasodilation, decrease venous return to the heart.</td>
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<tr>
<td></td>
<td></td>
<td>- reduce pain and anxiety, and</td>
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<td></td>
<td></td>
<td>- decrease myocardial oxygen consumption.</td>
</tr>
<tr>
<td>Cardiac glycosides</td>
<td>digitalis (Digoxin, Lanoxin) PO or IV</td>
<td>- increase the force and strength of ventricular contractions .</td>
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<tr>
<td></td>
<td></td>
<td>- decrease rate of contractions in order to increase cardiac output.</td>
</tr>
<tr>
<td>Diuretics</td>
<td>- furosemide (Lasix) PO or IV.</td>
<td>- promote excess fluid removal.</td>
</tr>
<tr>
<td></td>
<td>- chlorothiazide (Diuril) PO.</td>
<td>- decrease edema and pulmonary venous pressure by preventing sodium and water reabsorption.</td>
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</tbody>
</table>
| Vasodilators | PO or IV | - Relax vascular smooth muscle.  
|--------------|---------|-------------------------------------------------------------------  
|              | - **hydralazine** (Apresoline) | - Decrease preload and afterload.  
|              | (PO or IV) | - Decrease oxygen demand.  
|              | - **isosorbide dinitrate** (Isordil) SL or PO. | - Decrease systemic vascular resistance, and.  
|              | - **prazosin** (Minipress) PO. | - Increase venous capacitance.  
|              | - **minoxidil** (Loniten) PO. |                      
|              | - **diazoxide** (Hyperstat) IV, |                      
|              | - **sodium nitroprusside** (Nipride) IV. |                      
|              | - **nitroglycerine** (Nitrostat, Tridil) PO, SL, IV |                      
| Renin-angiotensin system inhibitors | captopril (Capoten) PO | - Inhibit angiotensin converting enzyme to reduce the production of angiotensin II  
|              | | - To enable the decrease in vasoconstriction and to reduce afterload.  
| Inotropic agents | - **dopamine**, dobutamine (Dobutrex) IV, | - Increase myocardial contractility, without increasing the heart rate.  
|              | - **amrinone** (Inocor) IV | - Produce peripheral vasodilation and decrease preload and afterload.  
|              | | **Critical Care Nursing**
**Cardiogenic Shock**

- The clinical definition of cardiogenic shock is decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume.
- Cardiogenic shock occurs when decreased CO leads to inadequate tissue perfusion and initiation of the shock syndrome.
- Cardiogenic shock may occur following MI when a large area of myocardium becomes ischemic, necrotic, and hypokinetic.
- It also can occur as a result of end-stage HF, cardiac tamponade, pulmonary embolism, cardiomyopathy, and dysrhythmias.
- Cardiogenic shock is a life-threatening condition with a high mortality rate.

**Pathophysiology**

The heart muscle loses its contractile power

resulting in a marked reduction in SV and CO

The decreased CO in turn reduces arterial blood pressure and tissue perfusion in the vital organs (heart, brain, lung, kidneys).
Flow to the coronary arteries is reduced
decreased oxygen supply to the myocardium, which increases ischemia
and further reduces the heart's ability to pump
Inadequate emptying of the ventricle
leads to increased pulmonary pressures, pulmonary congestion, and
pulmonary edema, exacerbating the hypoxia, causing ischemia of vital
organs, and setting a vicious cycle in motion

Causes

1. Coronary artery disease
2. Cardiac arrhythmias
3. Systolic dysfunction
4. Diastolic dysfunction
5. Valvular dysfunction
6. Mechanical complications

Signs and symptoms

1. Hypotension
2. Absence of hypovolemia
3. Clinical signs of poor tissue perfusion (i.e., oliguria, cyanosis, cool extremities, altered mentation)

**On Physical examination**

1. Skin is usually ashen or cyanotic and cool; extremities are mottled.
2. Peripheral pulses are rapid and faint and may be irregular if arrhythmias are present.
3. Jugular venous distention and crackles in the lungs are usually (but not always) present; peripheral edema also may be present.
4. Heart sounds are usually distant, and third and fourth heart sounds may be present.
5. The pulse pressure may be low, and patients are usually tachycardiac.
6. Patients show signs of hypo perfusion, such as altered mental status and decreased urine output.
7. Ultimately, patients develop systemic hypotension (i.e., systolic blood pressure below 90 mm Hg or a decrease in mean blood pressure by 30 mm Hg).

**Diagnostic tests**

2. **Laboratory studies**

   - Biochemical profile.
   - CBC.
   - Cardiac enzymes (e.g., creatine kinase and CK-MB, troponins, myoglobin, LDH).
   - Arterial blood gases.
   - Lactate.
• Brain natriuretic peptide

3. Imaging studies

• Echocardiography should be performed early to establish the cause of cardiogenic shock.
• Chest radiographic findings are useful for excluding other causes of shock or chest pain (e.g., aortic dissection, tension pneumothorax, pneumomediastinum).
• Ultrasonography can be used to guide fluid management.
• Coronary angiography is urgently indicated in patients with myocardial ischemia or MI who also develop cardiogenic shock.

4. Electrocardiography.

5. Swan-Ganz catheterization is very useful for helping exclude other causes and types of shock (e.g., volume depletion, obstructive shock, and shock).

Management

1. Fluid resuscitation to correct hypovolemia and hypotension, unless pulmonary edema is present.
2. Pharmacologic therapy to maintain blood pressure and cardiac output.
3. Early restoration of coronary blood flow.
4. Correction of electrolyte and acid-base abnormalities (e.g., hypokalemia, hypomagnesaemia, acidosis).
5. Provide vascular access for multiple infusions, and allow invasive monitoring of central venous pressure.
6. An arterial line may be placed to provide continuous blood pressure monitoring.

7. An intra-aortic balloon pump may be placed as a bridge to percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG).

**Pharmacologic therapy**

1. Aspirin and heparin for Patients with MI or acute coronary syndrome.

2. Inotropic and/or vasopressor drug therapy to maintain mean arterial pressure (MAP) of 60 or 65 mm Hg for patients with inadequate tissue perfusion and adequate intravascular volume.

3. Diuretics to decrease plasma volume and peripheral edema.

4. Dopamine is the drug of choice to improve cardiac contractility in patients with hypotension.

5. If the patient remains hypotensive despite moderate doses of dopamine, Norepinephrine is started at a dose of 0.5 mcg/kg/min and titrated to maintain an MAP of 60 mm Hg.

Phosphodiesterase inhibitors (eg, inamrinone [formerly amrinone], milrinone) are inotropic agents with vasodilating properties that are beneficial in patients with cardiac pump failure.

**PCI and CABG**

- Either PCI or CABG is the treatment of choice for cardiogenic shock.
- PCI should be initiated within 90 minutes after presentation.
• PCI remains helpful, as an acute intervention, within 12 hours after presentation.
• Thrombolytic therapy is second best but should be considered if PCI and CABG are not immediately available

Complications of cardiogenic shock

1. Cardiopulmonary arrest
2. Dysrhythmia.
3. Renal failure
5. Ventricular aneurysm.
6. Thromboembolic sequelae.
7. Stroke.
8. Death.

Nursing Care Plan for patients with cardiogenic shock

Assessment

Appearance

• Restlessness progressing to unresponsiveness.
• Chest pain.
• Dysrhythmias.

Vital signs
• HR: >100 beats/min.
• BP: <80 mm Hg.
• RR: > 20 breaths/min.

Neurologic

• Agitation.
• Restlessness progressing to unresponsiveness, and changes in level of consciousness.

Cardiovascular

• Weak thready pulses.
• Rhythm may be irregular.

Pulmonary

• Orthopnea
• Crackles
• Cough with increased secretions.

Nursing Diagnosis: Impaired gas exchange related to increased left ventricular diastolic pressure (LVEDP) and pulmonary edema associated with severe left ventricular (LV) dysfunction.

Nursing Interventions

1. Continuously monitor oxygenation status with pulse oximetry.
3. Monitor ECG for dysrhythmias caused by hypoxemia, electrolyte imbalances, or ventricular dysfunction.
5. Obtain HR, RR, and BP every 15 minutes to evaluate the patient’s response to therapy and detect cardiopulmonary deterioration.

6. Assess the patient’s respiratory status. The use of accessory muscles and inability to speak suggest worsening pulmonary congestion.

7. Assess for excess fluid volume, which can further compromise myocardial function.

8. Review ABGs for decreasing trend in Pao2 (hypoxemia) or pH (acidosis). These conditions can adversely affect myocardial contractility.

9. Review serial chest radiographs to evaluate the patient’s progress or a worsening lung condition.

10. Provide supplemental oxygen as ordered. If the patient develops respiratory distress, be prepared for intubation and mechanical ventilation.

11. Administer low-dose morphine sulfate as ordered to reduce preload in an attempt to decrease pulmonary congestion.

12. Minimize oxygen demand by maintaining bed rest and decreasing anxiety, fever, and pain.

13. Position the patient for maximum chest excursion and comfort.

14. Administer diuretics and/or vasodilators as ordered to reduce circulating volume and decrease preload.

**Cardiac Surgery**

- Coronary artery disease treatment requires the maximization of cardiac output and this can be accomplished by improvement in heart muscle
function and increase of blood flow through coronary artery bypass grafting and/or valvular replacements.

- Open heart surgery is commonly performed for three-vessel disease, valve dysfunction and congenital heart defects and requires blood to be diverted from the heart and lungs to facilitate a bloodless operative field.

- In coronary artery bypass graft (CABG) surgery, a graft from the arms or legs is anastomosed to the aorta with the distal portion to the involved coronary artery to bypass the diseased obstruction and supply adequate blood flow to the heart.

- The internal mammary artery is also being utilized for CABG surgery because the patency rate is 90-95% over a 5-10 year time period, and there are less problems with differences in lumen size since an artery is then anastomosed to an artery without the need for routing from the aorta.

- In valvular surgery, incompetent or leaking valves are replaced with prosthetic ones.

**CABG surgery recommended for patients with**

1. intractable angina.
2. signs of ischemia, or ,
3. an increased risk of coronary ischemia/infarction as a result of angiographical studies.

**The major indications for CABG:**

1. Alleviation of angina that cannot be controlled with medication or PCI.
2. Treatment of left main coronary artery stenosis or multivessel CAD.
3. Prevention and treatment of MI, dysrhythmias, or heart failure.
4. Treatment for complications from an unsuccessful PCI

**Steps of Cardiac Surgery (Procedure)**

1. The surgery is performed via a median sternotomy incision which provides exposure of the heart and avoids the pleural spaces.
2. A cannula is placed in a vein and an artery and then attached to the cardiopulmonary bypass machine whereby the diverted blood is mechanically oxygenated and circulated to the other parts of the body.
3. The machine, which is operated by a trained perfusionist, substitutes for left ventricular pumping and creates a blood-gas exchange.
4. After the patient’s body temperature has been cooled to around 86 degrees, the aorta is cross-clamped and a cold cardioplegic solution, usually containing dextrose, potassium, magnesium and Inderal, is placed around the heart and injected into the coronary arteries.
5. This causes an electromechanical arrest and provides an inert operative site.
6. Cross-clamp durations longer than 3 hours usually result in severe complications for the patient.
7. After the grafts have been completed or valves replaced, perfusion is slowly discontinued and cannulas are removed when arterial blood pressure and cardiac functioning are adequate.
8. Two atrial and ventricular pacing wires are placed, as well as arterial lines, pulmonary artery catheter, left atrial line, and mediastinal or pleural chest tubes.

**Common complications of CABG surgery**

1. perioperative MI.
2. vein graft closure.
3. Hemorrhage.
4. Blood trauma,
5. Complement activation, coagulation abnormalities, fluid shifts, increased catecholamine levels. Fat emboli, microemboli, dysrhythmias, pericarditis, embolism, pneumonia.
7. Cardiac tamponade, cardiogenic shock, endocarditis, gastrointestinal bleeding, mediastinitis, and paralytic ileus.

Medical Care
Pulmonary function studies: used to ascertain baseline pulmonary function:

Laboratory
11. Hemoglobin/hematocrit used to monitor oxygen-carrying capability, need for blood replacement, and to monitor for dehydration status; electrolytes used to monitor for imbalances which can affect cardiac function.
12. BUN and creatinine used to monitor renal function.
13. Liver profile used to monitor liver function and perfusion.
14. Glucose used to monitor for presence of diabetes, nutritional alterations, or organ dysfunction.
15. Cardiac enzymes and isoenzymes used to monitor for presence of acute or perioperative myocardial infarction.
16. Coagulation profiles used to determine baseline and monitor for coagulation problems.
17. Antibody or complement levels used to monitor for post-pericardiotomy syndrome or Dressler’s syndrome.
18. ACT used to monitor heparinization.
19. Arterial blood gases, used to monitor oxygenation and assess acid-base balance and ability to wean off mechanical ventilation

20. Electrocardiography: used to observe for changes in cardiac function, presence of conduction problems, dysrhythmias, or ischemic changes

21. Echocardiography: used to evaluate wall motion of the heart.

22. Cardiac catheterization: used to evaluate abnormal pressures preop, to assess for pressure gradients across the valves, and to locate and measure coronary lesions.

**Nursing Care Plan for patient undergoing cardiac surgery.**

**A: Assessment**

**Health History**

- The functional status of the cardiovascular system is determined by reviewing the patient's symptoms, including past and present experiences with chest pain, hypertension, palpitations, cyanosis, breathing difficulty (dyspnea), leg pain that occurs with walking (intermittent claudication), orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema.

- The patient's history of major illnesses, previous surgeries, medication therapies, and use of drugs, alcohol, and tobacco is also obtained.

**Physical Assessment**

A complete physical examination is performed, with special emphasis on the following:

- General appearance and behavior
- Vital signs
- Nutritional and fluid status, weight, and height
- Inspection and palpation of the heart, noting the point of maximal impulse, abnormal pulsations, and thrills
• Auscultation of the heart, noting pulse rate, rhythm, and quality; S₃ and S₄, snaps, clicks, murmurs, and friction rub
• Jugular venous pressure
• Peripheral pulses
• Peripheral edema

B: Diagnosis

• Fear related to the surgical procedure, its uncertain outcome, and the threat to well-being
• Deficient knowledge regarding the surgical procedure and the postoperative course
• Ineffective cardiac tissue perfusion related to reduced coronary blood flow

C: Planning and Goals

• reducing fear.
• learning about the surgical procedure and postoperative course, and,
• avoiding perioperative complications.

Risk for decreased cardiac output Related to:

• myocardial depression,
• Dysrhythmias.
• electrolyte imbalances, hypovolemia, hypervolemia,
• myocardial infarction, coronary artery spasm, vasoconstriction, impaired contractility.
• alteration in preload, alteration in afterload, hypo perfusion,
• hypoxia, damaged myocardium, use of PEEP while on ventilator
Nursing Interventions

1. Monitor vital signs, especially heart rate and blood pressure.
2. Notify MD of abnormalities.
3. Blood pressure should be taken/monitored every 15 minutes until stable, or every 5 minutes during active titration.

E: Evaluation

1. Demonstrates reduced fear
2. Learns about the surgical procedure and postoperative course
3. Shows no evidence of complications
   a. Reports anginal pain is relieved with medications and rest
   b. Takes medications as prescribed