**AORTIC ANEURYSM**

**INTRODUCTION** The aorta is the largest artery and is responsible for supplying oxygenated blood to essentially all vital organs in the body. The most common vascular problems that affect the aorta are aneurysms, aortoiliac occlusive disease, and aortic dissection.

**DEFINITION**

    An aneurysm is a localized sac or dilation formed at a weak point in the wall of the aorta.

                                                             OR

     Aneurysms are outpouchings or dilations of the arterial wall.

**INCIDENCE AND PREVALENCE**

  Aneurysms occur in men more often than in women, and their incidence increases with age. Aortic aneurysms  occur in   4.1% to 14.2% of men and 0.35% to 6.2% of women over age age 60.

**CLASSIFICATION**

Aneurysms generally are divided into two basic classifications:

* **True aneurysm**: Is one in which the wall of the artery forms the aneurysm, with at least one vessel layer still intact.

  True aneurysms can be further subdivided into fusiform and saccular.

* **A fusiform aneurysm:** is circumferential and relatively uniform in shape.

* **A saccular aneurysm:** is pouch like with a narrow neck connecting the bulge to one side of the arterial wall.

* **A false aneurysm or pseudoaneurysm**:  is not an aneurysm but a disruption of all layers of the arterial wall resulting in bleeding that is contained by surrounding structures.

**ETIOLOGY**

* Atherosclerosis
* Trauma
* Infection
* Heredity
* Arterial punctures

**Contributing factors**

* Hypertension
* Arteriosclerosis
* Congenital weakness of vessels

**PATHOPHYSIOLOGY**

                                                            Etiological factors

*
* Collection of atherosclerotic plaque on the intimal surface of the aorta
*

                                                 Degenerative changes in the medial layer

*
* The destruction of the medial layer of a segment of the aorta leads to loss of elasticity  weakening

                                                          Dilation of the aorta

**CLINICAL MANIFESTATIONS**

* Pulase and blood pressure difference in upper extremities if aneurysm interferes with circulation in left subclavian artery
* Pain
* Intermittent and neuralgic pain because of impingement on nerves
* Dyspnea
* Hoarseness, voice weakness
* Dilated superficial veins on chest
* Cyanosis because of vein compression of chest vessels

**DIAGNOSTIC EVALUATION**

* History collection
* Physical examination
* Chest x-ray-may show calcification  that outlines aneurysm
* Computed tomography and ultrasonograghy- are used to detect and monitor size of aneurysm
* MRI
* Arteriography- allows visualization of aneurysm and vessel.

**COMPLICATIONS**

* Fatal hemorrhage
* Myocardial ischemia
* Sroke
* Acute renal failure

**MEDICAL MANAGEMENT**

Early treatment and detection is imperative. If aneurysm is larger than 5-6cm or increasing  aneurysm is 0.5cm over a six month period surgical repair is the treatment. For individuals with small aneurysm less than 4cm conservative therapy is initiated

**DRUG THERAPY**

* Beta-blokers -e.g: propranolol
* Anti-hypertensives

**SURGICAL THERAPY**

**Endovascular  grafting**

Repair of aneurysm using a stent graft , which is deployed through the femoral artery.

**NURSING MANAGEMENT**

**Pre operative**

* Preoperative assessment must include detection of concurrent coronary artery disease and cerebrovascular disease
* Assess all peripheral pulses for baseline comparison postoperativelyIf emergent surgery is required, time for preoperative care and teaching may be limited
* Implement measures to reduce fear and anxiety:
* Orient to the intensive care unit, if appropriate
* Describe and explain the reason for all equipment and tubes, sucgh as cardiac monitors, ventilators, nasogastric tubes, urinary catheters, intravenous lines and fluids and intra-arterial lines
* Explain what to expect following surgery (sights, sounds, frequency of taking vital signs, dressing, pain relief measures, communication strategies)

**Nursing assessment**

* A thorough nursing history and physical asessment should be performed, because atherosclerosis is a systemic desease process
* It is important for the nurse to watch for signs of cardiac, pulmonary, cerebral and lower extrimity vascular problems
* The patient should be monitored for indications of rupture of the aneurysm such as diaphoresis, paleness, weakness, tachycardia, hypotension, abdominal, back, groin or periumbilcal pain, changes in sensorium or a pulsating abdominal mass
* Attention to the character and quality of the peripheral pulses and the neurologic status
* Pedal pulse sites (dorsalis pedis and posterial tibial) and skin lesions on the lower extrimities should be marked and documented before surgery

**NURSING DIAGNOSIS**

1. Ineffective tissue perfusion related to aneurysm, aneurysm rupture,.
2. Acute Pain related to pressure of aneurysm on nerves , surgical procedure
3. Risk for infection related to surgery

**CONCLUSION**

 An aneurysm is a distention of an artery brought about by a weakening of the arterial  wall. The aorta is the most common site for aneurysms. It should be treated immediately, if left untreated ,they may rupture , causing a fatal hemorrhage.

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                                                 **CARDIAC TAMPONADE**

**INTRODUCTION**

Pericardial effusion refers to the accumulation of fluid in the pericardial sac. This occurrence may accompany pericarditis advanced HF, metastatic carcinoma, cardiac surgery, trauma, or non traumatic hemorrhage. Normally, the pericardial sac contains less than 50 mL of fluid, which  is needed to decrease friction for the beating heart. An increase in pericardial fluid raises the pressure within the pericardial sac and compresses the heart. This has the following effects:

• Increased right and left ventricular end-diastolic pressures

• Decreased venous return

• Inability of the ventricles to distend adequately and to fill

Pericardial fluid may accumulate slowly without causing noticeable symptoms. A rapidly developing effusion, however, can stretch the pericardium to its maximum size and, because of increased pericardial pressure, reduce venous return to the heart and decrease CO. The result is cardiac tamponade (compression of  the heart)

**DEFINITION**

Cardiac tamponade is caused by accumulation of fluid or blood between the two layers of the pericardium. It is the most serious complication of pericarditis.

**ETIOLOGY**

**Trauma to the chest**: Cardiac contusion may occur. (Bruising of the heart muscle.) Blood and fluid leak into the pericardial sac.

**Myocardial infarction:** Inflammation at the site of the infarction leads to increased capillary permeability. Fluid can leak into the pericardial sac resulting in a tamponade.

**Cardiac bypass surgery:** Normally blood and fluid accumulate around the heart after heart surgery. Sometimes, though, one of the sutures to a graft may burst. This may cause sudden accumulation of blood in the mediastinum, resulting in a cardiac tamponade

**PATHOPHYSIOLOGY**

Normally, the pericardial sac contains less than 50 mL of fluid, which is needed to decrease friction for the beating heart. An increase in pericardial fluid raises the pressure within the pericardial sac and compresses the heart. This has the following effects:

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• Inability of the ventricles to distend adequately and to fill

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**CLINICAL MANIFESTATIONS**

* **Jugular vein distension (JVD):** Heart is “squeezed” so blood cannot fill heart. Instead blood backs up into venous system, causing distension of jugular vein

* **Drop in blood pressure:** The heart squeezes → CO drops → decreased forward flow of volume.

* **Muffled heart sounds:** Fluid accumulates around the heart muffling heart sounds

* **Pulsus paradoxus:** Blood pressure drops more than 10 mm Hg with inspiration. This is because with inspiration there is even more pressure “squeezing” down on heart

* **Change in level of consciousness:** Decreased head perfusion due to drop (LOC) in CO

* **Increased HR** : Compensation for drop in CO

* **Edema** :  Blood backing up into the venous system

**DIAGNOSTIC EVALUATION**

* History and physical assessment
* **Chest x-ray**: widened mediastinum due to blood accumulation.
* **Echocardiography**: detects compression of the heart, variation in blood flow in heart that occurs with breathing; shows fluid accumulation.
* **Electrocardiography**: fast, slow, or normal HR with no pulse.

**COMPLICATIONS**

* Cardiogenic shock.
* Death.

**MEDICAL MANAGEMENT**

* Oxygen therapy: increases oxygenation and tissue perfusion.
* Inotropic agents: controls heart rate and decreases atrial fibrillation.

**PERICARDIOCENTESIS**

If cardiac function becomes seriously impaired, pericardiocentesis (puncture of the pericardial sac to aspirate pericardial fluid) is performed to remove fluid from the pericardial sac.

**PERCUTANEOUS BALLOON PERICARDIOTOMY**: drains fluid using a balloon-tipped catheter inserted through the skin.

 **SUBXIPHOID LIMITED PERICARDIOTOMY**: drains fluid using a balloon-tipped catheter inserted through a small incision in the chest.

**NURSING DIAGNOSIS**

* Decreased cardiac output related insuffient blood supply as evidenced by  hypotension.
* Ineffective tissue perfusion related to circulatory insufficiency
* Excess  fluid volume related to cardiac tamponade as evidenced by edema.

**CONCLUSION**

Cardiac tamponade is an complication of an pericarditis which results in accumulation of fluid in the pericardial sac, which results in compression of the heart. As a nurse she has to immediately prepare the articles for pericardiocentesis.

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 **CARDIOGENIC SHOCK**

**INTRODUCTION**

Shock is a complex physiologic entity representing a diverse group of life- threatening  circulatory conditions. Every cell in the body needs adequate tissue perfusion to provide the necessary supply of oxygen and nutrients and to remove metabolic byproducts . In shock , blood flow to  vital organs becomes inadequate , or the cells become unable to extract and use oxygen and substrates. If left untreated , this functional impairement of cells, tissues, organs and body system progress to multiple organ dysfunction and death.

**DEFINITION**

Cardiogenic shock occurs when the heart’s ability to contract and to pump blood is impaired and the supply of oxygen is inadequate for the heart and tissues.

**ETIOLOGY**

**Coronary Factors**

Myocardial infarction

**Non-coronary Factors**

Cardiomyopathies

Valvular damage

Cardiac tamponade

Dysrhythmias

**PATHOPHYSIOLOGY**

In cardiogenic shock, cardiac output, which is a function of both stroke volume and heart rate, is compromised. When stroke volume and heart rate decrease or become erratic, blood pressure drops and tissue perfusion is compromised. Along with other tissues and organs being deprived of adequate blood supply, the heart muscle itself receives inadequate blood. The result is impaired tissue perfusion. Because impaired tissue perfusion weakens the heart and impairs its ability to pump blood forward, the ventricle does not fully eject its volume of blood at systole. As a result, fluid accumulates in the lungs. This sequence of events can occur rapidly or over a period of days

**CLINICAL MANIFESTATIONS**

* Tachycardia
* Hypotension
* anginal pain
* dysrhythmias
* hemodynamic instability.

**DIAGNOSTIC EVALUATION**

* History and physical assessment
* Renal function test
* Urinalysis

**MEDICAL MANAGEMENT**

The goals of medical management are to,

* Limit further myocardial damage and preserve the healthy myocardium
* Improve the cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both

In general, these goals are achieved by increasing oxygen supply to the heart muscle while reducing oxygen demands.

**INITIATION OF FIRST-LINE TREATMENT**

First-line treatment of cardiogenic shock involves the following actions:

• Supplying supplemental oxygen

• Controlling chest pain

• Providing selected fluid support

• Administering vasoactive medications

• Controlling heart rate with medication or by implementation of a transthoracic or intravenous

   pacemaker

• Implementing mechanical cardiac support (intra-aortic balloon counterpulsation therapy,

   ventricular assist systems, or extracorporeal cardiopulmonary bypass)

**OXYGENATION.**

In the early stages of shock, supplemental oxygen is administered by nasal cannula at a rate of 2 to 6 L/min to achieve an oxygen saturation exceeding 90%. Monitoring arterial blood gas values and pulse oximetry values helps to indicate whether the patient requires a more aggressive method of oxygen delivery.

**PAIN CONTROL.**

If the patient experiences chest pain, morphine sulfate is administered intravenously for pain relief. In addition to relieving pain, morphine dilates the blood vessels. This reduces the workload of the heart by both decreasing the cardiac filling pressure (preload) and reducing the pressure against which the heart muscle has to eject blood (afterload). Morphine also relieves the patient’s anxiety. Cardiac enzyme (CPK-MB) levels are measured, and serial 12-lead electrocardiograms are obtained to assess the degree of myocardial damage.

**PHARMACOTHERAPY**

Medications commonly combined to treat cardiogenic shock include dobutamine, dopamine, and nitroglycerin .

**Dobutamine** (Dobutrex):

 Produces inotropic effects by stimulating myocardial beta receptors, increasing the strength of myocardial activity and improving cardiac output. Myocardial alpha-adrenergic receptors are also stimulated, resulting in decreased pulmonary and systemic vascular resistance (decreased afterload). Dobutamine enhances the strength of cardiac contraction,

improving stroke volume ejection and overall cardiacoutput

**Nitroglycerin.**

Intravenous nitroglycerin (Tridil) in low doses acts as a venous vasodilator and therefore reduces preload. At higher doses, nitroglycerin causes arterial vasodilation and therefore reduces afterload as well. These actions, in combination with medium-dose dopamine, increase cardiac output while minimizing cardiac workload. Additionally, vasodilation enhances blood flow to the myocardium, improving oxygen delivery to the weakened heart muscle

**Dopamine.**

Dopamine (Intropin) is a sympathomimetic agent that has varying vasoactive effects depending on the dosage. It may be used with dobutamine and nitroglycerine to improve tissue perfusion.

Low-dose dopamine (0.5 to 3.0 ⎧g/kg/min) increases renal blood flow, thereby preventing ischemia of these organs because shock causes blood to be shunted away from the kidney.. This dosage, however, does not improve cardiac output. Medium-dose dopamine (4 to 8 ⎧g/kg/min) has sympathomimetic properties and improves contractility (inotropic action) and slightly increases the heart rate (chronotropic action). At this dosage, dopamine increases cardiac output and therefore is desirable. High-dose dopamine (8 to 10 ⎧g/kg/min) predominantly causes vasoconstriction, which increases afterload and thus increases cardiac workload. Because this effect is undesirable in patients with cardiogenic shock, dopamine dosages must be carefully titrated. Once the patient’s blood pressure stabilizes, low-dose dopamine may be continued for its effect of promoting renal perfusion in particular.

**DIURETICS**

Such as furosemide (Lasix) may be administered to reduce the workload of the heart by reducing fluid accumulation

**Antiarrhythmic Medications.**

Antiarrhythmic medication is also part of the medication regimen in cardiogenic shock. Multiple factors, such as hypoxemia, electrolyte imbalances, and acid–base imbalances, contribute to serious cardiac dysrhythmias in all patients with shock. Additionally, as a compensatory response to decreased cardiac output and blood pressure, the heart rate increases beyond normal limits. This impedes cardiac output further by shortening diastole and thereby decreasing the time for ventricular filling. Consequently, anti-arrhythmic medications are required to stabilize the heart rate.

**Fluid Therapy.**

In addition to medications, appropriate fluid is necessary in treating cardiogenic shock. Administration of fluids must be monitored closely to detect signs of fluid overload. Incremental intravenous fluid boluses are cautiously administered to determine optimal filling pressures for improving cardiac output. A fluid bolus should never be given quickly because rapid fluid administration in patients with cardiac failure may result in acute pulmonary edema.

**MECHANICAL ASSISTIVE DEVICES**

If cardiac output does not improve despite supplemental oxygen, vasoactive medications, and fluid boluses, mechanical assistive devices are used temporarily to improve the heart’s ability to pump.

Intra-aortic balloon counterpulsation is one means of providing temporary circulatory assistance (A polyurethane balloon catheter is inserted percutaneously through the common femoral artery and advanced into the descending thoracic aorta. The balloon catheter is connected to a console containing a gasfilled pump. The timing of the balloon inflation is synchronized electrocardiographically with the beginning of diastole, and the balloon deflation occurs just before systole. The goals of intraaortic balloon counterpulsation include the following:

• Increased stroke volume

• Improved coronary artery perfusion

• Decreased preload

• Decreased cardiac workload

• Decreased myocardial oxygen demand

.

 **Human heart transplantation:**

 May be the only option remaining for a patient who has cardiogenic shock and who cannot be weaned from mechanical assistive devices.  Another short-term means of providing cardiac or pulmonary support to the patient in cardiogenic shock is through an extracorporeal device similar to the cardiopulmonary bypass (CPB) used in open-heart surgery. The CPB system requires systemic anticoagulation, arterial and venous cannulation of the femoral artery and vein, and connection to a centrifugal, oxygenated pump. The catheter tip is advanced into the right atrium. This system lowers left and right ventricular pressures, reducing the workload and oxygen needs of the heart.Complications of CPB include coagulopathies, myocardial ischemia, infection, and thromboembolism. CPB is used only in emergency situations until definitive treatment, such as heart transplantation, can be initiated.

**NURSING MANAGEMENT**

**PREVENTING CARDIOGENIC SHOCK**

In some circumstances, identifying patients at risk early and promoting adequate oxygenation of the heart muscle and decreasing cardiac workload can prevent cardiogenic shock. This can be accomplished by conserving the patient’s energy, promptly relieving angina, and administering supplemental oxygen. Often, however, cardiogenic shock cannot be prevented. In such instances, nursing management includes working with other members of the health care team to prevent shock from progressing and to restore adequate cardiac function and tissue perfusion.

**MONITORING HEMODYNAMIC STATUS**

A major role of the nurse is monitoring the patient’s hemodynamic and cardiac status. Arterial lines and electrocardiographic monitoring equipment must be maintained and functioning properly. The nurse anticipates the medications, intravenous fluids, and equipment that might be used and is ready to assist in implementing these measures. Changes in hemodynamic, cardiac, and pulmonary status are documented and reported promptly. Additionally, adventitious breath sounds, changes in cardiac rhythm, and other abnormal physical assessment findings are reported immediately.

**ADMINISTERING MEDICATIONS AND INTRAVENOUS FLUIDS**

The nurse has a critical role in safe and accurate administration of intravenous fluids and medications. Fluid overload and pulmonary edema are risks because of ineffective cardiac function and accumulation of blood and fluid in the pulmonary tissues. The nurse documents and records medications and treatments that are administered as well as the patient’s response to treatment. The nurse needs to be knowledgeable about the desired effects as well as the side effects of medications. For example, it is important to monitor the patient for decreased blood pressure after administering morphine or nitroglycerin. The patient receiving thrombolytic therapy must be monitored for bleeding. Arterial and venous puncture sites must be observed for bleeding and pressure must be applied at the sites if bleeding occurs. Neurologic assessment is essential after the administration of thrombolytic therapy to assess for the potential complication of cerebral hemorrhage associated with the therapy. Intravenous infusions must be observed closely because tissue necrosis and sloughing may occur if vasopressor medications infiltrate the tissues. Urine output, BUN, and serum creatinine levels are monitored to detect decreased renal function secondary to the effects of cardiogenic shock or its treatment.

**MAINTAINING INTRA-AORTIC BALLOON COUNTERPULSATION**

The nurse plays a critical role in caring for the patient receiving intra-aortic balloon counterpulsation The nurse makes ongoing timing adjustments of the balloon pump to maximize its effectiveness by synchronizing it with the cardiac cycle. The patient is at great risk for circulatory compromise to the leg on the side where the catheter for the balloon has been placed; therefore, the nurse must frequently check the neurovascular status of the lower extremities.

**ENHANCING SAFETY AND COMFORT**

Throughout care, the nurse must take an active role in safeguarding the patient, enhancing comfort, and reducing anxiety. This includes administering medication to relieve chest pain, preventing infection at the multiple arterial and venous line insertion sites, protecting the skin, and monitoring respiratory function. Proper positioning of the patient promotes effective breathing without decreasing blood pressure and may also increase the patient’s comfort while reducing anxiety. Brief explanations about procedures that are being performed and the use of comforting touch often provide reassurance to the patient and family.

**NURSING DIAGNOSIS**

* Decreased cardiac output related insuffient blood supply as evidenced by tachycardia,dysrhyyythmias
* Ineffective tissue perfusion related to circulatory insufficiency
* Deficient fluid volume related to shock
* Impaired gas exchange related to inadequate oxygen supply,.

**CONCLUSION**

Shock is a complex physiological entity. shock may develop in any patient, in any setting. Early recognition of clinical manifestations and initiation of therapeutic measures may halt the progression of shock and prevent death.

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                                  **RESPIRATORY FAILURE**

**INTRODUCTION**
       Respiratory failure is a broad ,non specific clinical diagnosis indicating that the respiratory sysytem is unable to supply the oxygen necessary to maintain metabolism or cannot eliminate sufficient C02 .Respiratory failure is not a disease; it is a condition that occurs as a result of one or more diseases involving the lungs or other body systems.

**DEFINITION**

 Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions. That is oxygenation and carbon dioxide elimination. In practice, (respiratory failure is defined as a : - PaO2 value of less than 60 mm Hg while breathing air - PaCO2 of more than 50 mm Hg.)

         Respiratory failure is a syndrome of inadequate gas exchange due to dysfunction of one or more essential components of the respiratory system Chest wall ,(including pleura and diaphragm) Airways, Alveolar– capillary units, Pulmonary circulation ,Nerves supply to respiratory organs, CNS or Brain Stem.

**CLASSIFICATION**

* **Type I or Hypoxemic (PaO2 <60 mm hg )**: Failure of oxygen exchange
* **Type II or Hypercapnic (PaCO2 >45 mm hg** ): Failure to exchange or remove carbon dioxide
* **Type III Respiratory Failure** : Perioperative respiratory failure Can be resulted by anesthetic or operative technique, incentive spirometry, post-operative analgesia,
* **Type IV Respiratory Failure** :  Type IV describes, patients who are intubated and ventilated in the process of resuscitation for shock. Goal of ventilation is to stabilize gas exchange and to unload the respiratory muscles, lowering their oxygen consumption. Respiratory failure can also due to shock.

**Hypoxemic respiratory failure (type I):**

It is characterized by a PaO2 of less than 60 mm Hg. This is the most common form of respiratory failure, and it can be associated with virtually all acute diseases of the lung which generally involve fluid filling or collapse of alveolar units. Some examples of type I respiratory failure are pulmonary edema, pneumonia, and pulmonary hemorrhage

**Hypercapnic respiratory failure (type II):**

It is characterized by a PaCO2 of more than 50 mm Hg. Hypoxemia is common in patients with hypercapnic respiratory failure who are breathing room air. Common etiologies include drug overdose, neuromuscular disease, chest wall abnormalities, and severe airway disorders (eg, asthma, chronic obstructive pulmonary disease [COPD].

**Perioperative respiratory failure (Type III):**

Peri-operative respiratory failure is usually caused by atelectasis. Effective means of preventing or treating atelectasis include incentive spirometry.

**CLASSIFICATION:**

Respiratory failure may be ,

Acute and Chronic E.g.: acute exacerbation of advanced COPD

**DISTINCTIONS BETWEEN ACUTE AND CHRONIC RESPIRATORY FAILURE:**

**ACUTE RESPIRATORY FAILURE:**

* Acute hypercapnic respiratory failure develops over minutes to hours.
* Acute respiratory failure can develop quickly and may require emergency treatment .
* Acute respiratory failure usually is treated in an intensive care unit

**CHRONIC RESPIRATORY FAILURE:**

* Chronic respiratory failure develops over several days or longer
* . Chronic respiratory failure develops more slowly and lasts longer.
* Chronic respiratory failure can be treated at home or at a long-term care center.

**ETIOLOGY OF RESPIRATORY FAILURE :**

**Type I respiratory failure (hypoxic) :**

Pneumonia ,pulmonary edema, Acute lung injury (ALI), Acute respiratory distress syndrome (ARDS) ,Pulmonary embolism ,Atelectasis, and Pulmonary fibrosis.

**Type II Respiratory Failure (hypercapnic) :**

 Hypoventilation, Asthma, Chronic obstructive pulmonary disease (COPD), Hypoxemia and hypercapnia often occur together.

**Type III Respiratory Failure (pre-operative) :**

 Inadequate post- operative analgesia, upper abdominal incision, Obesity, ascites Pre- operative tobacco smoking  Excessive airway secretions.

**Type IV Respiratory Failure (shock):**

* **Cardiogenic shock**: is based upon an inadequate circulation of blood due to primary failure of the ventricles of the heart to function effectively
* **Septic shock :** Septic shock is a medical emergency caused by decreased tissue perfusion and oxygen delivery as a result of severe infection and sepsis , though the microbe may be systemic or localized .
* **Hypovolemic shock** :  Hypovolemic shock is a particular form of shock in which the heart is unable to supply enough blood to the body

**CLINICAL MANIFESTATION :**

* Dyspnea
* Tachypnea
* Cyanosis
* Agitation
* Disorientation
* Restless
* Decreased level of consciousness
* Tachycardia
* Hypertension
* Cool skin, clammy,and diaphoretic
* Dysrhythmias
* Hypotension
* Fatigue
* Tremors
* seizures

**PATHOPHYSIOLOGY:**

Respiratory failure can arise from an abnormality in any of the components of the respiratory system, including the airways, alveoli, CNS, peripheral nervous system, respiratory muscles, and chest wall. Patients who have hypoperfusion secondary to cardiogenic, hypovolemic, or septic shock often present with respiratory failure.

**Hypoxemic respiratory failure**: The pathophysiologic mechanisms that account for the hypoxemia observed in a wide variety of diseases are ventilation-perfusion (V/Q) mismatch and shunt. These 2 mechanisms lead to widening of the alveolar-arterial oxygen difference, which normally is less than 15 mm Hg. With V/Q mismatch, the areas of low ventilation relative to perfusion (low V/Q units) contribute to hypoxemia. An intrapulmonary or intracardiac shunt causes mixed venous (deoxygenated) blood to bypass ventilated alveoli and results in venous admixture. The distinction between V/Q mismatch and shunt can be made by assessing the response to oxygen supplementation or calculating the shunt fraction following inhalation of 100% oxygen. In most patients with hypoxemic respiratory failure, these 2 mechanisms coexist.

**Hypercapnic respiratory failure**: At a constant rate of carbon dioxide production, PaCO2 is determined by the level of alveolar ventilation (Va), where VCO2 is ventilation of carbon dioxide and K is a constant value (0.863).

(Va = K x VCO2)/PaCO2

A decrease in alveolar ventilation can result from a reduction in overall (minute) ventilation or an increase in the proportion of dead space ventilation. A reduction in minute ventilation is observed primarily in the setting of neuromuscular disorders and CNS depression. In pure hypercapnic respiratory failure, the hypoxemia is easily corrected with oxygen therapy.

**DIAGNOSTIC EVALUATION**

* **History and physical assessment**
* **Physical Findings**: Hypotension usually with signs of poor perfusion suggests severe sepsis. Hypertension usually with signs of poor perfusion suggests pulmonary edema .Wheezing suggests airway obstruction.
* **Laboratory studies**
* **ABG analysis**  - Quantifies level of gas exchange, abnormality Identifies type and chronicity of respiratory failure
* **Complete blood count**:  Anemia may cause cardiogenic pulmonary edema ,Leukocytosis, or leukopenia suggestive of infection
* **Microbiology Respiratory cultures**: sputum/tracheal aspirate
* **Blood, urine and body fluid (e.g. pleural) cultures**
* **Chest radiography** - Identify chest wall, pleural and lung parenchymal with opacities present
* **Electrocardiogram** - Identify arrhythmias, ischemia, ventricular dysfunction
* **Echocardiography** - Identify right and/or left ventricular dysfunction
* **Pulmonary function tests/bedside spirometry**:  Identify obstruction, restriction May be difficult to perform if critically ill
* **Bronchoscopy**

**TREATMENT :**

* Mechanical Ventilator Emergency treatment  follows the principles of cardiopulmonary resuscitation .
* Treatment of the underlying cause is required
* Endotracheal intubation and mechanical ventilation may be required.
* Respiratory stimulants such as doxapram may be used.

**Management of Respiratory Failure:**

* Management of Respiratory Failure ABC’ s or resuscitation
* Ensure airway is adequate
* Ensure adequate supplemental oxygen and assisted ventilation, if indicated
* Support circulation as needed

**DRUG THERAPY**

Relief of bronchospasm:  e.g;albuterol

Reduction of airway inflammation: corticosteroids

Reduction of pulmonary congestion:  Lasix(furosemide)

Treatment of pulmonary infections:  Antibiotics

Reduction of severe anxiety, pain and agitation: Lorazepam

**Oxygen therapy** : Humidified oxygen during administration, preventing dehydration of the mucous membranes and pulmonary secretions.

**MECHANICAL VENTILATION:**

**Non-invasive**: (if patient can have patent airway and is stable)

 Mask: usually orofacial to start

**Invasive** - Endotracheal tube (ETT)

 **Tracheostomy** – if upper airway is obstructed

**INDICATIONS FOR MECHANICAL VENTILATION:**

* Cardiac or respiratory arrest
* Tachypnea or bradypnea with respiratory fatigue
* Acute respiratory acidosis hypoxemia (when the PaO2 could not be maintained above 60 mm Hg)
* Inability to clear secretions with impaired gas exchange or excessive respiratory work

**Goals of mechanical ventilation :**

* Improve ventilation by stabilizing respiratory rate and tidal volume.
* Assistance for neural or muscle dysfunction Sedated, comatose or paralyzed patient
* . Intra-operative ventilation
* Rest respiratory muscles
* Correct hypoxemia, improve cardiac function

**NURSING MANAGEMENT :**

* Patients with acute respiratory failure should be closely observed for potential deterioration
* Monitoring may involve intermittent/continual pulse oximetry .
* Any changes in physiological signs should be reported promptly to the senior practitioner
* Pulmonary secretions: oropharyngeal/nasopharyngeal suction helps to clear secretions.

* **NURSING DIAGNOSES**
	+ Impaired gas exchange related to alveolar hypoventilation, as evidenced by hypoxemia/hypercapnia.
	+ Ineffective airway clearancerelated to excessive secretions, decreased level of consciousness as evidenced by presence of rhonchi or crackles, ineffective cough.
	+ Ineffective breathing pattern related to  respiratory muscle fatigue, bronchospasm as evidenced by irregular breathing pattern, use of accessory muscles.
	+ Risk for fluid volume imbalance related to sodium and water retention
	+ Anxiety related to disease condition
	+ Imbalanced nutrition: Less than body requirements related to shortness of breath, poor appetite as evidenced by weight loss, poor muscle tone.

**CONCLUSION**

Respiratory failure is an life threatening disease because many different problems cause respiratory failure, specific care of these patients varies. Immediate treatment is necessary to save the life of the patient .  As a critical care nurse she should know the management of patients with respiratory failure and also regarding the mechanical ventilation.

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