Shock

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Introduction

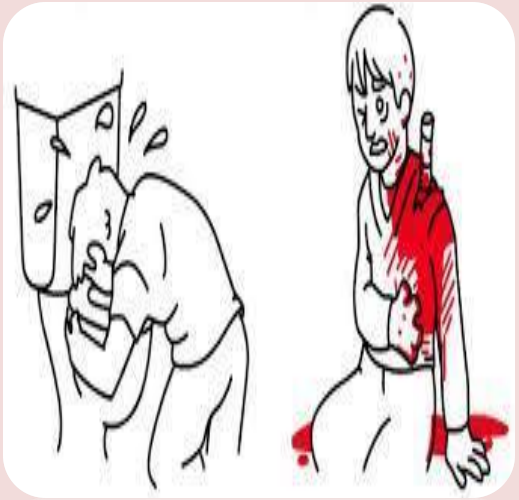
- Shock is a clinical syndrome characterized by inadequate tissue perfusion that results in cellular, metabolic, and hemodynamic derangements. Impaired tissue perfusion occurs when there is an imbalance between cellular oxygen supply and cellular oxygen demand.
- Shock is a complex pathophysiologic process that often results in MODS and death. All types of shock involve ineffective tissue perfusion and acute circulatory failure

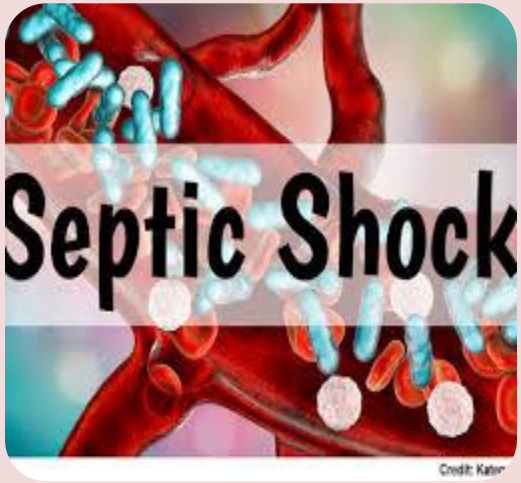
Introduction

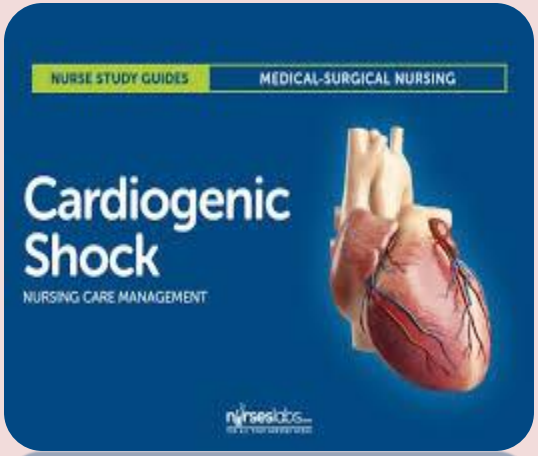
- The shock syndrome is a pathway involving a variety of pathologic processes that may be categorized as four stages: initial, compensatory, progressive, and refractory.
- Progression through each stage varies with the patient's prior condition, duration of initiating event, response to therapy, and correction of underlying cause

Classification of Shock

- Shock can be classified as hypovolemic, cardiogenic, or distributive, depending on the pathophysiologic cause and hemodynamic profile.







Type of Shock	Physiological Alteration
Hypovolemic	a loss of circulating or intravascular volume.
Cardiogenic	impaired ability of the heart to pump and Inadequate myocardial contractility
Obstructive	Obstruction of blood flow
Distributive **Neurogenic ,Anaphylactic, Septic**	maldistribution of circulating blood volume and Inadequate vascular tone

Stages of Shock

- Although the response to shock is highly individualized, a pattern of stages progresses at unpredictable rates. If each stage of shock is not recognized and treated promptly, progression to the next stage occurs. The pathophysiological events and associated clinical findings for each stage are summarized in table

Clinical Presentation	Physiological Events	Stage of Shock
I: Initiation	↓Tissue oxygenation caused by: ↓Intravascular volume (hypovolemic) ↓Myocardial contractility (cardiogenic) Obstruction of blood flow (obstructive) ↓Vascular tone (distributive) Septic (mediator release) Anaphylactic (histamine release) Neurogenic (suppression of SNS)	No observable clinical indications ↓CO may be noted with invasive hemodynamic monitoring
II:Compensatory	**Neural compensation by SNS** ↑Heart rate and contractility Vasoconstriction Redistribution of blood flow from nonessential to essential organs Bronchodilation **Endocrine compensation (RAAS, ADH, glucocorticoids release)** Renal reabsorption of Na, chloride, and water Vasoconstriction Glycogenolysis and gluconeogenesis Chemical compensation	↑Heart rate (except neurogenic) Narrowed pulse pressure Rapid, deep respirations causing respiratory alkalosis Thirst Cool, moist skin Oliguria Diminished bowel sounds Restlessness progressing to confusion Hyperglycemia ↑Urine specific gravity and ↓ creatinine clearance

Clinical Presentation	Physiological Events	Stage of Shock
III: Progressive	Progressive tissue hypo perfusion Anaerobic metabolism with lactic acidosis Failure of sodium-potassium pump Cellular edema	Dysrhythmias ↓BP with narrowed pulse pressure Tachypnea Cold, clammy skin Anuria Absent bowel sounds Lethargy progressing to coma Hyperglycemia ↑ BUN, creatinine, and potassium Respiratory and metabolic acidosis
IV: Refractory	Severe tissue hypoxia with ischemia and necrosis Worsening acidosis SIRS MODS	Life-threatening dysrhythmias Severe hypotension despite vasopressors Respiratory and metabolic acidosis Acute respiratory failure Acute respiratory distress syndrome Disseminated intravascular coagulation Hepatic dysfunction/failure Acute kidney injury Myocardial ischemia/infarction/failure Cerebral ischemia/infarction

Clinical Alert of Shock

Assessment	Significance
Change in vital signs, hemodynamic parameters, sensorium	Secondary to decreased tissue perfusion and initiation of compensatory mechanisms
Decreased urine output, rising BUN and creatinine levels	Secondary to initiation of compensatory mechanisms and decreased renal perfusion
Tachypnea, hypoxemia, worsening chest x-ray	Related to development of acute respiratory distress syndrome secondary to hypoperfusion
Petechiae, ecchymosis, bleeding from puncture sites, overt or occult blood in urine, stool, gastric aspirate, tracheal aspirate	Related to development of disseminated intravascular coagulation secondary to shock, SIRS
Hypoglycemia, increase in liver enzymes	Related to hepatic dysfunction secondary to hypoperfusion

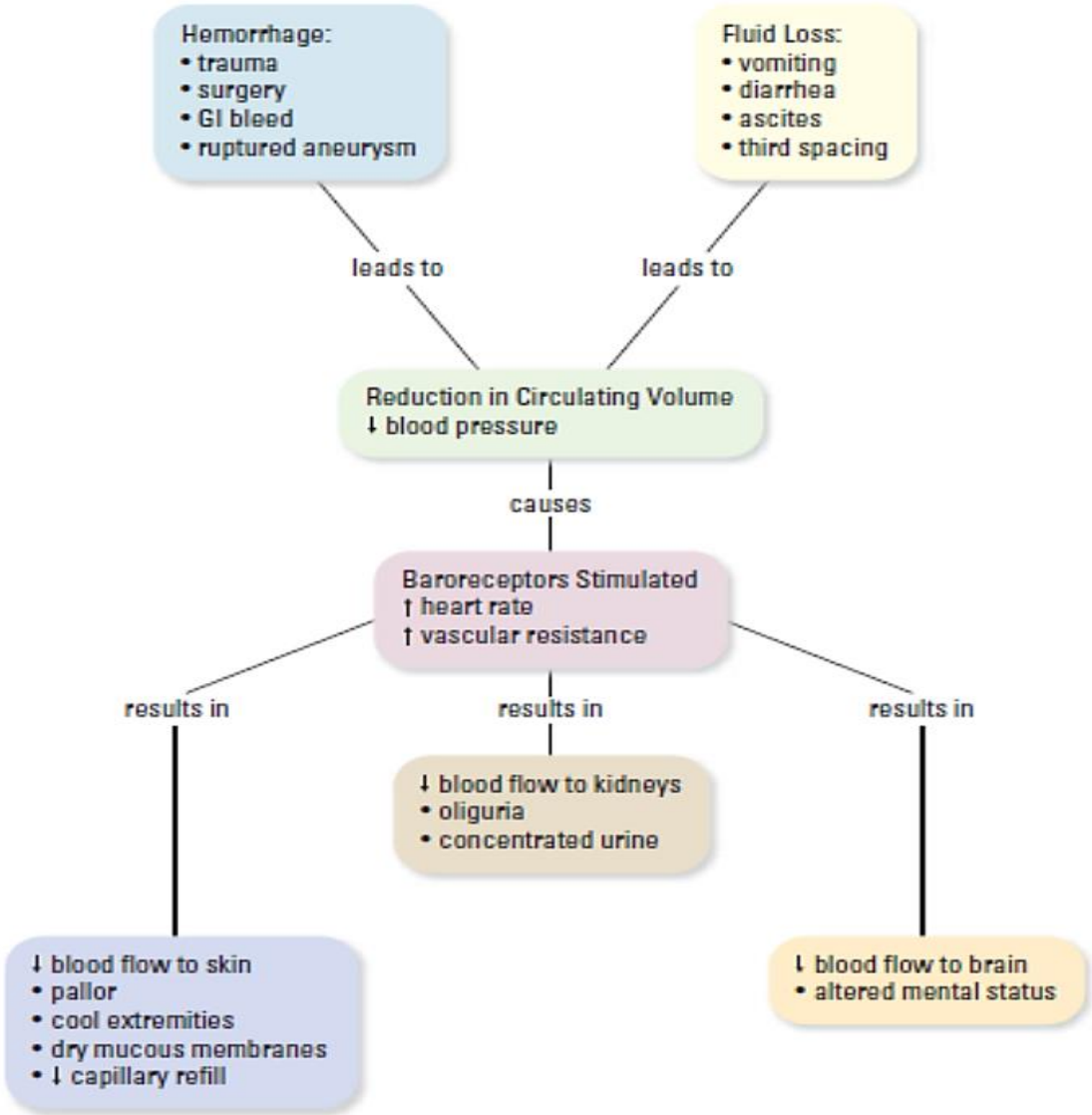
TABLE 11-3 HEMODYNAMIC ALTERATIONS IN SHOCK STATES

HEMODYNAMIC PARAMETER, NORMAL VALUE				DISTRIBUTIVE		
	HYPOVOLEMIC	CARDIOGENIC	OBSTRUCTIVE	SEPTIC	ANAPHYLACTIC	NEUROGENIC
Heart rate 60-100 beats/min	High	High	High	High	High	Normal or low
Blood pressure	Normal → Low	Normal → Low	Normal → Low	Normal → Low	Normal → Low	Normal → Low
Cardiac output 4-8 L/min	Low	Low	Low	High then low	Normal → Low	Normal → Low
Cardiac index 2.5-4.0 L/min/m^2	Low	Low	Low	High then low	Normal → Low	Normal → Low
RAP 2-6 mm Hg	Low	High	High	Low to variable	Low	Low
PAOP 8-12 mm Hg or PADP 8-15 mm Hg	Low	High	High if impaired diastolic filling or high LV afterload; Low if high RV afterload	Low to variable	Low	Low
SVR 770-1500 dynes/sec/cm^{-5}	High	High	SVR Low PVR High	Low to variable	Low	Low
SvO_2 60%-75%	Low	Low	Low	High then low	Low	Low

LV, Left ventricular; *PADP*, pulmonary artery diastolic pressure; *PAOP*, pulmonary artery occlusion pressure; *PVR*, pulmonary vascular resistance; *RAP*, right atrial pressure; *RV*, right ventricular; *SvO_2*, mixed venous oxygen saturation; *SVR*, systemic vascular resistance.

Hypovolemic Shock

- occurs from inadequate fluid volume in the intravascular space. The lack of adequate circulating volume leads to decreased tissue perfusion and initiation of the general shock response. Hypovolemic shock is the most commonly occurring form of shock (Fig. 35-2).



Visual Map 8-1 Hypovolemic Shock

Etiologic Factors in Hypovolemic Shock

Absolute Factors	Relative Factors
❑ Loss of whole blood ❑ Trauma or surgery ❑ Gastrointestinal bleeding ❑ Loss of plasma ❑ Thermal injuries ❑ Large lesions ❑ Loss of other body fluids ❑ Severe vomiting or diarrhea ❑ Massive diuresis ❑ Loss of intravascular integrity ❑ Ruptured spleen ❑ Long bone or pelvic fractures ❑ Hemorrhagic pancreatitis ❑ Hemothorax or hemoperitoneum ❑ Arterial dissection or rupture	❑ Vasodilation ❑ SIRS/sepsis ❑ Anaphylaxis ❑ Loss of sympathetic stimulation ❑ Increased capillary membrane permeability ❑ SIRS/sepsis ❑ Anaphylaxis ❑ Thermal injuries ❑ Decreased colloidal osmotic pressure ❑ Severe sodium depletion ❑ Hypopituitarism ❑ Cirrhosis ❑ Intestinal obstruction

Classification of hypovolemic shock:

- A simpler approach of classifying hypovolemic shock as mild, moderate, or severe is also commonly used.

1. Class I, or mild shock, indicates a fluid volume loss up to 15% or an actual volume loss up to 750 mL.
2. Class II hypovolemia occurs with a fluid volume loss of 15% to 30% or an actual volume loss of 750 to 1500 mL.
3. Hypovolemic shock that is class III occurs with a fluid volume loss of 30% to 40% or an actual volume loss of 1500 to 2000 mL.
4. Class IV hypovolemic shock is severe shock and usually refractory in nature. It occurs with a fluid volume loss of greater than 40% or an actual volume loss of more than 2000 mL.

Medical Management

The major goals of therapy for the patient in hypovolemic shock are to:

1. Correct the cause of the hypovolemia.
2. Restore tissue perfusion, and prevent complications.

- Identifying and stopping the source of fluid loss.
- Administering fluid to replace circulating volume. Fluid administration can be accomplished with use of a crystalloid solution, a colloid solution, blood products, or a combination of fluids.The type of solution used **depends on:**

- ✓ Fluid lost.
- ✓ The degree of hypovolemia.
- ✓ The severity of hypoperfusion.
- ✓ The cause of hypovolemia.

- Aggressive fluid resuscitation in trauma and surgical patients. The benefit of limited or hypotensive. Volume resuscitation in patients with uncontrolled hemorrhage is postulated to lessen
- bleeding and improve survival.

Nursing Management

1. Administering volume replacement.
2. Monitor vital signs.
3. Accurate monitoring of intake and output

and daily weights are essential components of preventive nursing care.

1. Early identification and treatment result in decreased mortality.
2. continuous evaluation of intravascular volume, tissue perfusion, and response to therapy
3. Providing comfort and emotional support.
4. Measures to minimize fluid loss include limiting blood sampling, observing lines for accidental disconnection, and applying direct pressure to bleeding sites.
5. Positioning the patient with the legs elevated, trunk flat, and head and shoulders above the chest.
6. Monitoring the patient for clinical manifestations of fluid overload or complications related to fluid and blood product administration is essential for preventing further problems.

Nursing diagnosis

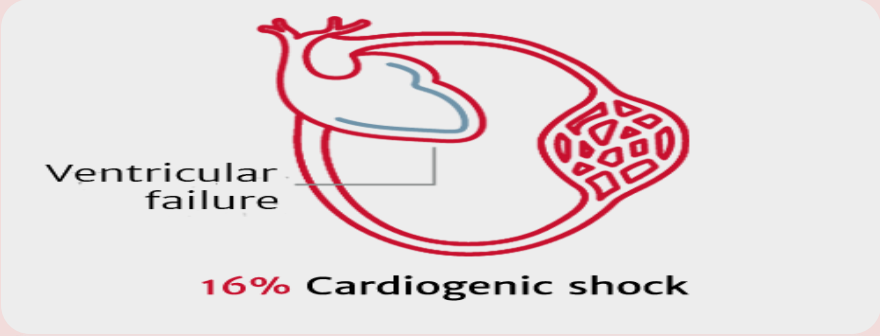
- Deficient Fluid Volume related to active blood loss
- Deficient Fluid Volume related to interstitial fluid shift.
- Decreased Cardiac Output related to alterations in preload.
- Imbalanced Nutrition: Less Than Body Requirements related to increased
- metabolic demands or lack of exogenous nutrients
- Risk for Infection.

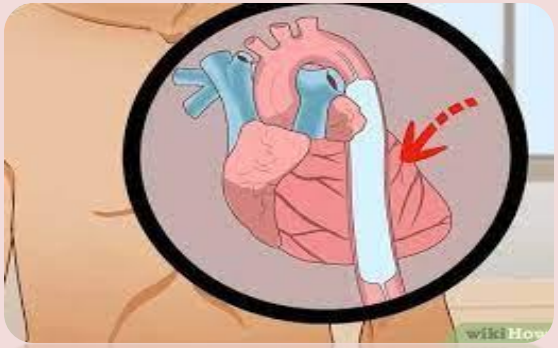
Cardiogenic shock

- It is the result of failure of the heart to effectively pump blood forward. It can occur with dysfunction of the right or the left ventricle, or both. The lack of adequate pumping function leads to decreased tissue perfusion and circulatory failure (Fig. 35-3).

Pathophysiology

- Cardiogenic shock results from the impaired ability of the ventricle to pump blood forward, which leads to a decrease in SV and an increase in the blood left in the ventricle at the end of systole. The decrease in SV results in a decrease in CO, which leads to decreased cellular oxygen supply and ineffective tissue perfusion.





Etiology

- Cardiogenic shock can result from problems affecting the muscular function or the mechanical function of the heart or the cardiac rhythm

Muscular	Mechanical	Rhythmic
▪ Ischemic injury ▪ Acute myocardial infarction ▪ Cardiopulmonary arrest ▪ Acute decompensated heart failure ▪ Cardiomyopathy ▪ Acute myocarditis ▪ Myocardial contusion ▪ Prolonged cardiopulmonary bypass ▪ Septic shock ▪ Hemorrhagic shock ▪ Medications (beta-adrenergic blockers, calcium-channel antagonists, cytotoxic agents)	✓ Valvular dysfunction ✓ Papillary muscle dysfunction or rupture ✓ Septal wall rupture ✓ Free wall rupture ✓ Ventricular aneurysm ✓ Obstructive hypertrophic ✓ Cardiomyopathy ✓ Pulmonary embolus ✓ Atrial thrombus ✓ Cardiac tamponade ✓ Massive pulmonary embolus ✓ Constrictive pericarditis	▪ Bradydysrhythmia ▪ Tachydysrhythmias

Clinical Manifestations of Cardiogenic Shock

- Systolic blood pressure <90 mm Hg
- Acute drop in blood pressure >30 mm Hg
- Heart rate >100 beats/min
- Weak, thready pulse
- Diminished heart sounds
- Change in sensorium
- Cool, pale, moist skin
- Urine output <30 mL/hr
- Chest pain
- Dysrhythmias
- Tachypnea
- Crackles
- Decreased cardiac output
- Cardiac index <2.2 L/min/m2
- Increased pulmonary artery occlusion pressure
- Increased right atrial pressure
- Variable systemic vascular resistance

Medical Management

The major goals of therapy are to treat the underlying cause, enhance the effectiveness of the pump, and improve tissue perfusion.

1. Identifying and treating the etiologic factors of heart failure and administering pharmacologic agents or using mechanical devices to enhance CO.
2. Inotropic agents are used to increase contractility and maintain adequate blood pressure and tissue perfusion. A vasopressor may be necessary to maintain blood pressure when hypotension is severe.
3. Diuretics may be used for preload reduction. After blood pressure has been stabilized, vasodilating agents are used for preload and afterload reduction.
4. Antidysrhythmic agents should be used to suppress or control dysrhythmias that can affect CO.
5. Intubation and mechanical ventilation are usually necessary to support oxygenation.

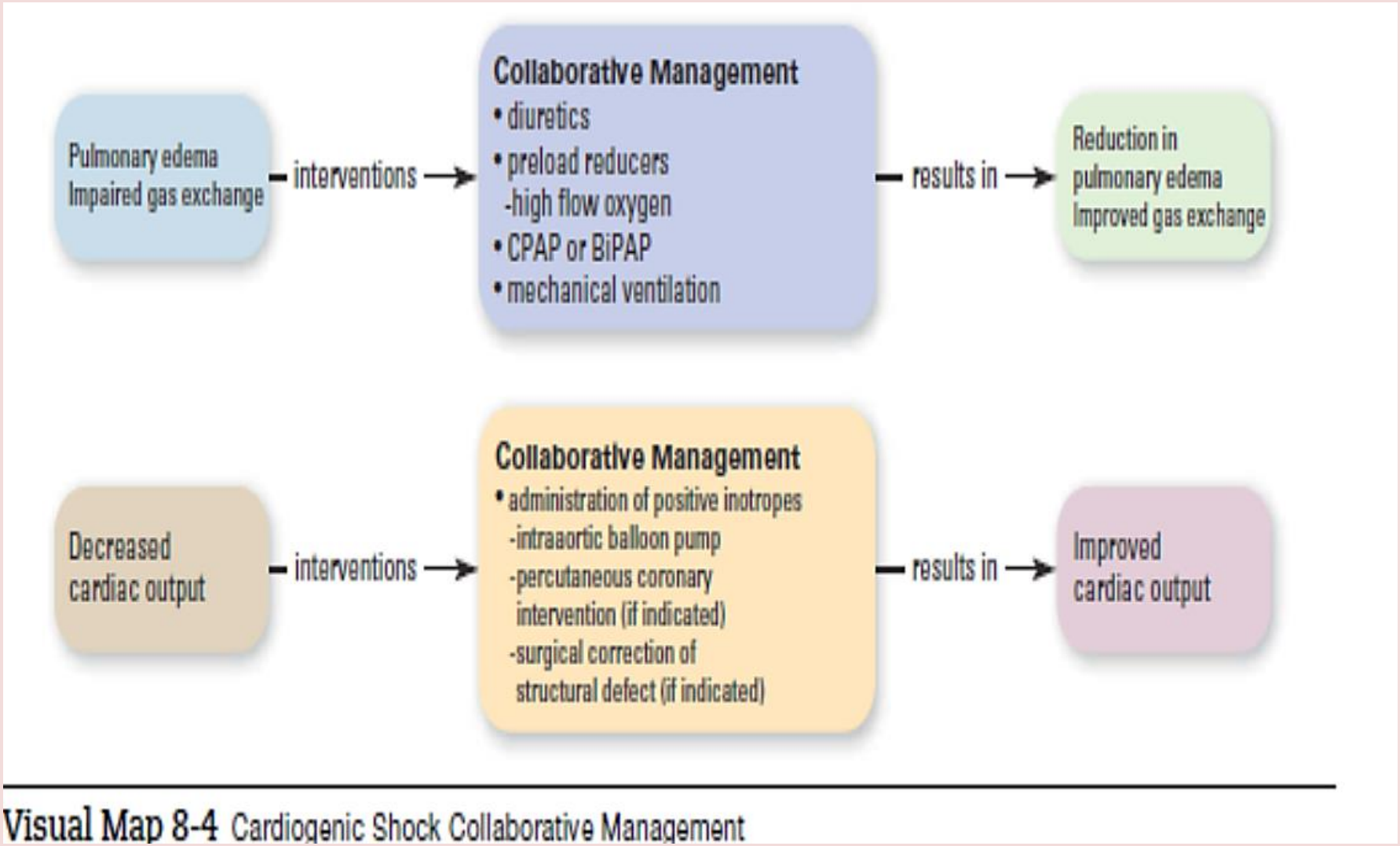
Nursing Management

1. Assess frequently blood pressure, heart rate, and respiratory rate and pulse oximetry.
2. Assess the patient closely signs of respiratory compromise from pulmonary congestion. Signs and symptoms indicating pulmonary congestion and inadequate oxygenation include tachypnea, crackles on auscultation, and decreased oxygen saturation.
3. Monitoring and management of heart rate, preload, afterload, and contractility.
4. Limiting myocardial oxygen demand.
5. Enhancing myocardial oxygen supply.
6. Maintaining adequate tissue perfusion.
7. Providing comfort and emotional support.
8. Measures to limit myocardial oxygen demand include:

- ✓ Administering analgesics, sedatives, and agents to control afterload and dysrhythmias.
- ✓ Positioning the patient for comfort; limiting activities; providing a calm and quiet environment and offering support to reduce anxiety; and teaching the patient about the condition.

9. Measures to enhance myocardial oxygen supply include administering supplemental oxygen monitoring the patient's respiratory status.

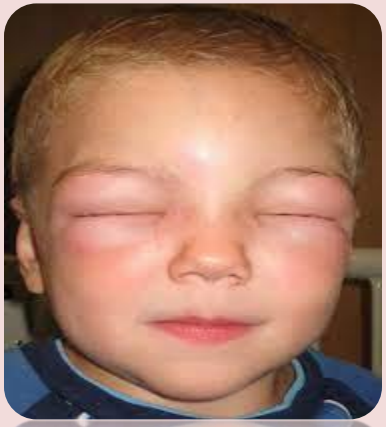
Collaborative management

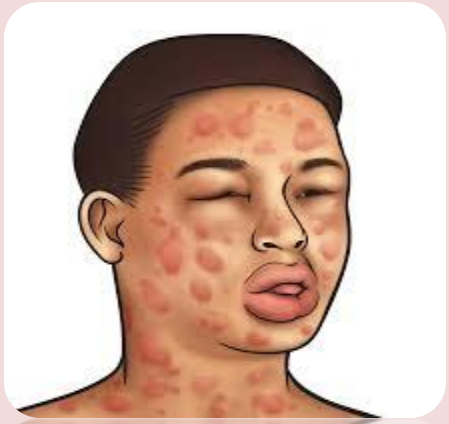


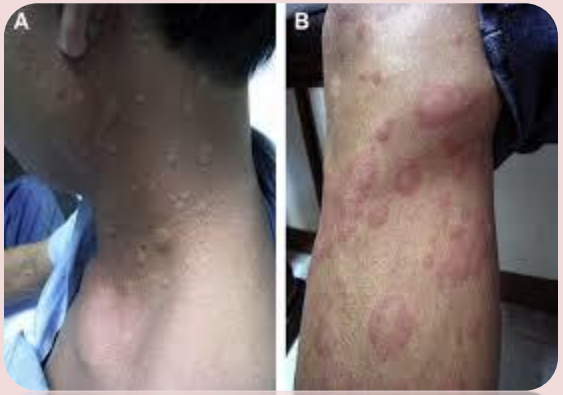
Visual Map 8-4 Cardiogenic Shock Collaborative Management

Anaphylactic Shock

A type of distributive shock, is the result of an immediate hypersensitivity reaction. It is a life-threatening event that requires prompt intervention. The severe and systemic response leads to decreased tissue perfusion and initiation of the general shock response (Fig. 35-4).







Etiology of Anaphylactic Shock

Foods

- ✓ Eggs and milk
- ✓ Fish and shellfish
- ✓ Nuts and seeds
- ✓ Legumes& cereals
- ✓ Soy
- ✓ Wheat
- ✓ Strawberries
- ✓ Avocados

Food Additives

- ✓ Food coloring
- ✓ Preservatives
- ✓ Diagnostic Agents
- ✓ Radio contrast media

Biologic agents

- ✓ Blood &blood components
- ✓ Insulin & other hormones
- ✓ Gammaglobulin
- ✓ Vaccines and antitoxins

Environmental Agents

- ➢ Pollens, molds, and spores
- ➢ Sunlight
- ➢ Cold or heat
- ➢ Animal dander
- ➢ Latex

Venoms

- ➢ Bees, hornets, yellow jackets,
- ➢ and wasps
- ➢ Snakes, jellyfish
- ➢ Deer flies
- ➢ Fire ants

Drugs

- ➢ Antibiotics
- ➢ Aspirin
- ➢ NSAIDs
- ➢ Opioids
- ➢ Dextran
- ➢ Vitamins
- ➢ Muscle relaxants
- ➢ Neuromuscular blocking agents
- ➢ Barbiturates
- ➢ Non barbiturate hypnotics

Clinical Manifestations of Anaphylactic Shock

Cardiovascular
- Hypotension
- Tachycardia
- Bradycardia
- Chest pain

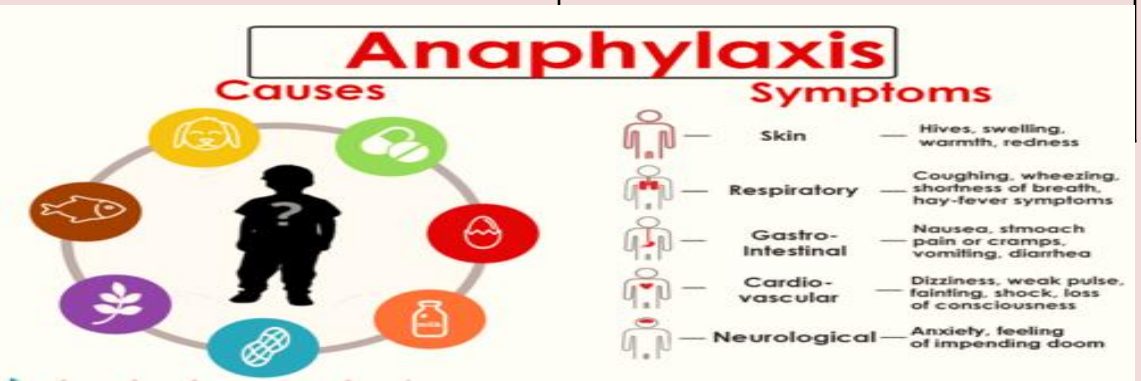
Respiratory
- Lump in throat
- Cough
- Dyspnea
- Dysphagia
- Hoarseness
- Stridor
- Wheezing
- Rhinitis
- Chest tightness

Cutaneous
- Pruritus
- Erythema
- Urticaria
- Angioedema
- Sense of warmth

Gastrointestinal
- Nausea
- Vomiting
- Diarrhea
- Cramping abdominal pain

Neurologic
- Restlessness
- Uneasiness
- Apprehension
- Anxiety
- Dizziness
- Headache
- Sense of impending doom
- Confusion
- Syncope or near syncope

Genitourinary
- Incontinence



Clinical Criteria For Diagnosing Anaphylaxis

- Acute onset of an illness (minutes to several hours) with involvement of the skin or mucosal tissue, or both (e.g., generalized hives; pruritus or flushing; swollen lips, tongue, and uvula) and at least one of the Following:

a. Respiratory compromise (e.g., dyspnea, wheeze [bronchospasm], stridor, reduced peak expiratory flow, hypoxemia)

b. Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence)

2.Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):

a. Involvement of the skin-mucosal tissue (e.g., generalized hives; pruritusor flushing; swollen lips, tongue, and uvula)

b. Respiratory compromise (e.g., dyspnea, wheeze [bronchospasm], stridor, reduced peak expiratory flow, hypoxemia)

Clinical Criteria For Diagnosing Anaphylaxis

c. Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence)

d. Persistent gastrointestinal symptoms (e.g., crampy abdominal pain,

vomiting).

3. Reduced blood pressure after exposure to known allergen for that patient (minutes to several hours):

a. Infants and children: low systolic blood pressure (age-specific) or greater than 30% decrease in systolic blood pressure*

b. Adults: systolic blood pressure of less than 90 mm Hg or greater than 30% decrease for the person’s baseline

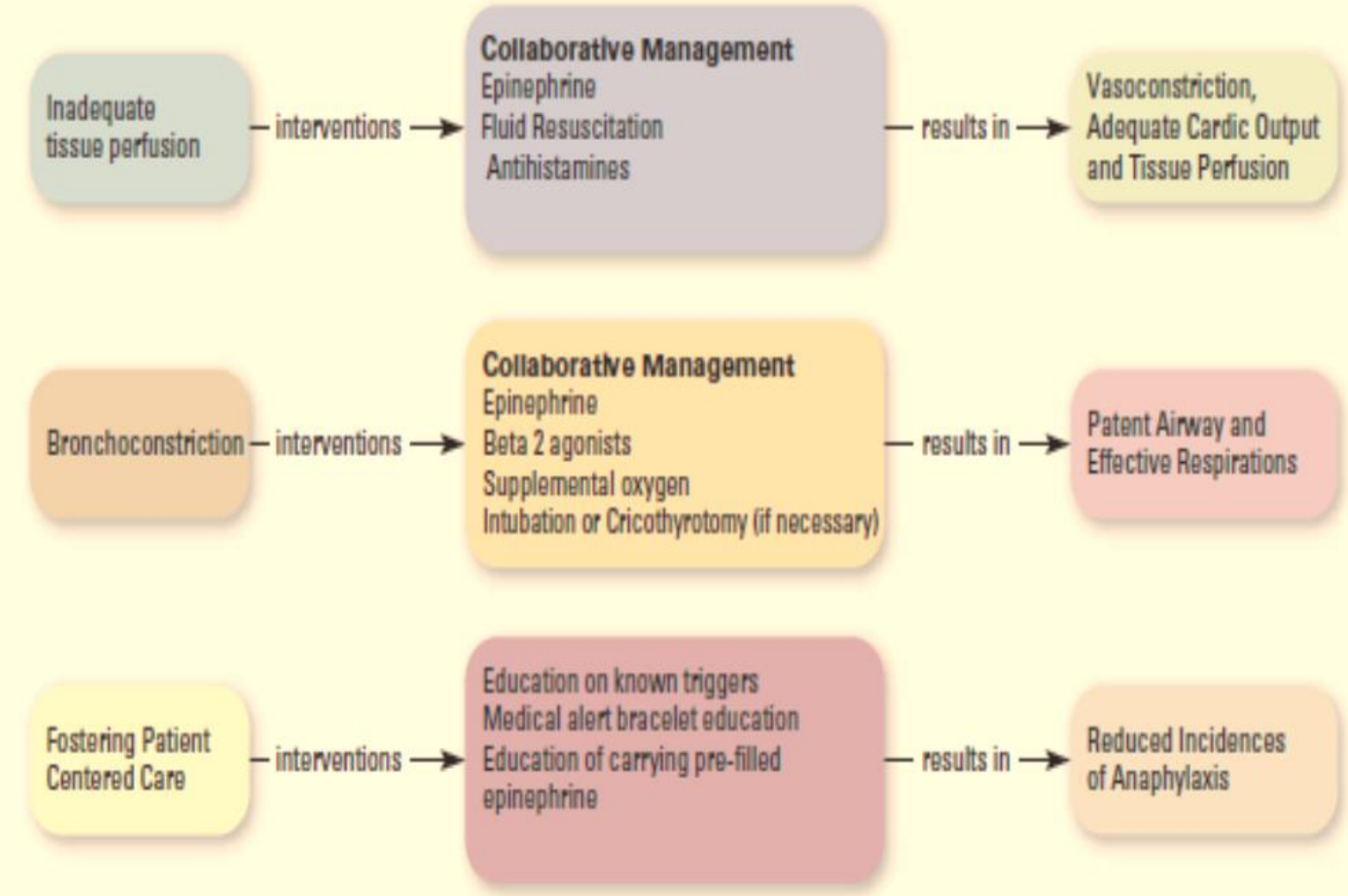
Medical Management

- The goals of therapy are to remove the offending antigen, reverse the effects of the biochemical mediators, and promote adequate tissue perfusion.
- When the hypersensitivity reaction occurs as a result of administration of medications, dye, blood, or blood products, the infusion should be immediately discontinued.
- Epinephrine is the first-line treatment of choice for anaphylaxis and should be administered when initial signs and symptoms occur.
- Oxygen therapy, intubation, mechanical ventilation, and administration of medications and fluids.
- Several medications are used as second-line adjunctive therapy. Inhaled beta-adrenergic agents are used to treat bronchospasm unresponsive to epinephrine.

Nursing Management

- Prevention of anaphylactic shock is one of the primary responsibilities of the nurse in the critical care area. Nursing interventions include:
- Administering epinephrine as order.
- Facilitating ventilation n include:
- Positioning the patient to assist with breathing and instructing the patient to breathe slowly and deeply.
- Airway protection through prompt administration of prescribed medications is essential.
- Administering volume replacement includes
- Inserting large-bore peripheral intravenous catheters.
- Rapidly administering prescribed fluids; and positioning the patient in a supine position with the legs elevated

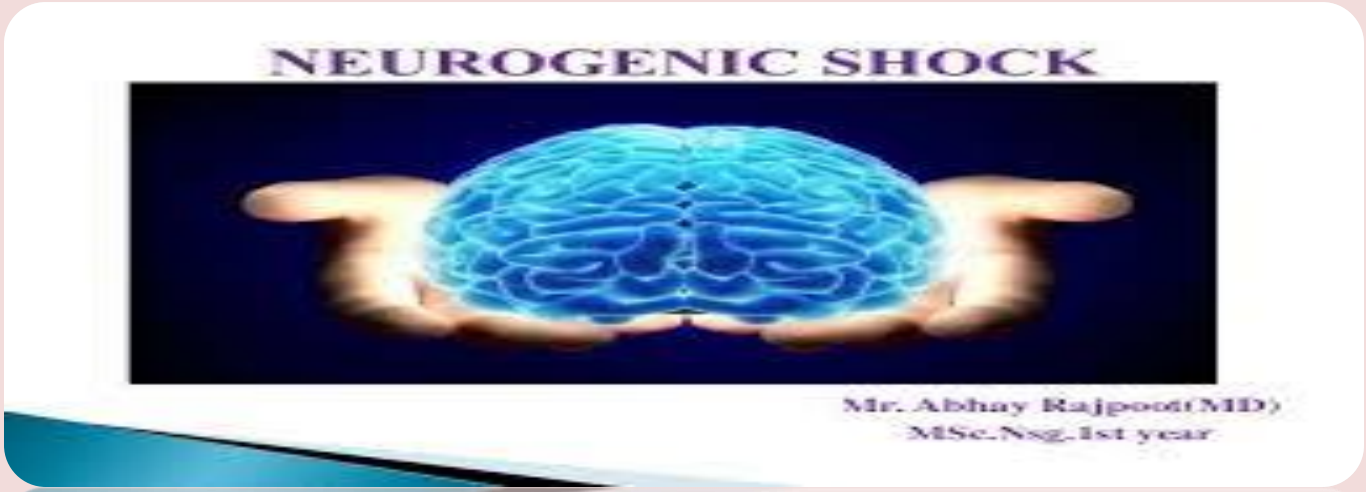
- Providing comfort includes administering medications to relieve itching, and applying warm soaks to skin and emotional support
- Maintaining surveillance for recurrent reactions and preventing and maintaining surveillance for complications.
- Patient education about how to avoid the precipitating allergen is essential for preventing future episodes of anaphylaxis.



Visual Map 8-8 Anaphylactic Shock Collaborative Management

Neurogenic Shock

- Neurogenic shock, another type of distributive shock, is the result of the loss or suppression of sympathetic tone. The lack of sympathetic tone leads to decreased tissue perfusion and initiation of the general shock response (Fig. 35-5).

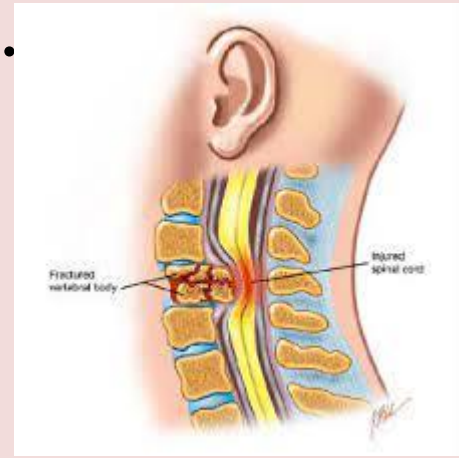


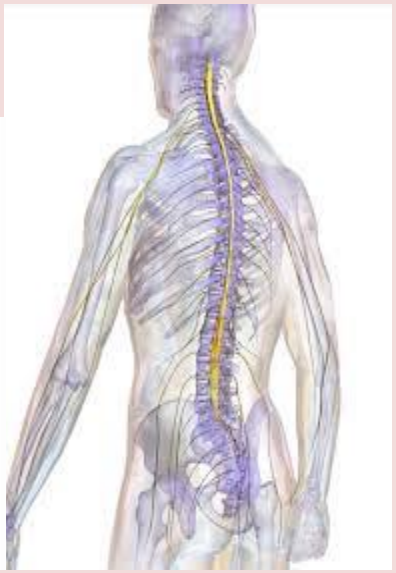
Etiology

Neurogenic shock can be caused by

1. Anything that disrupts the SNS. The problem can occur as the result of interrupted impulse transmission or blockage of sympathetic outflow from the vasomotor center in the brain.

2. Spinal cord injury (SCI).

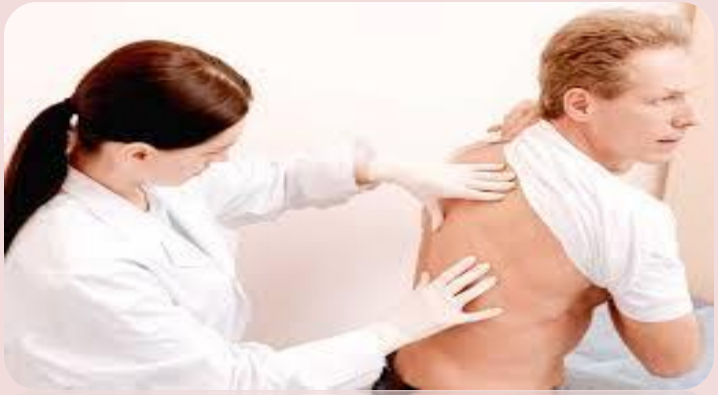


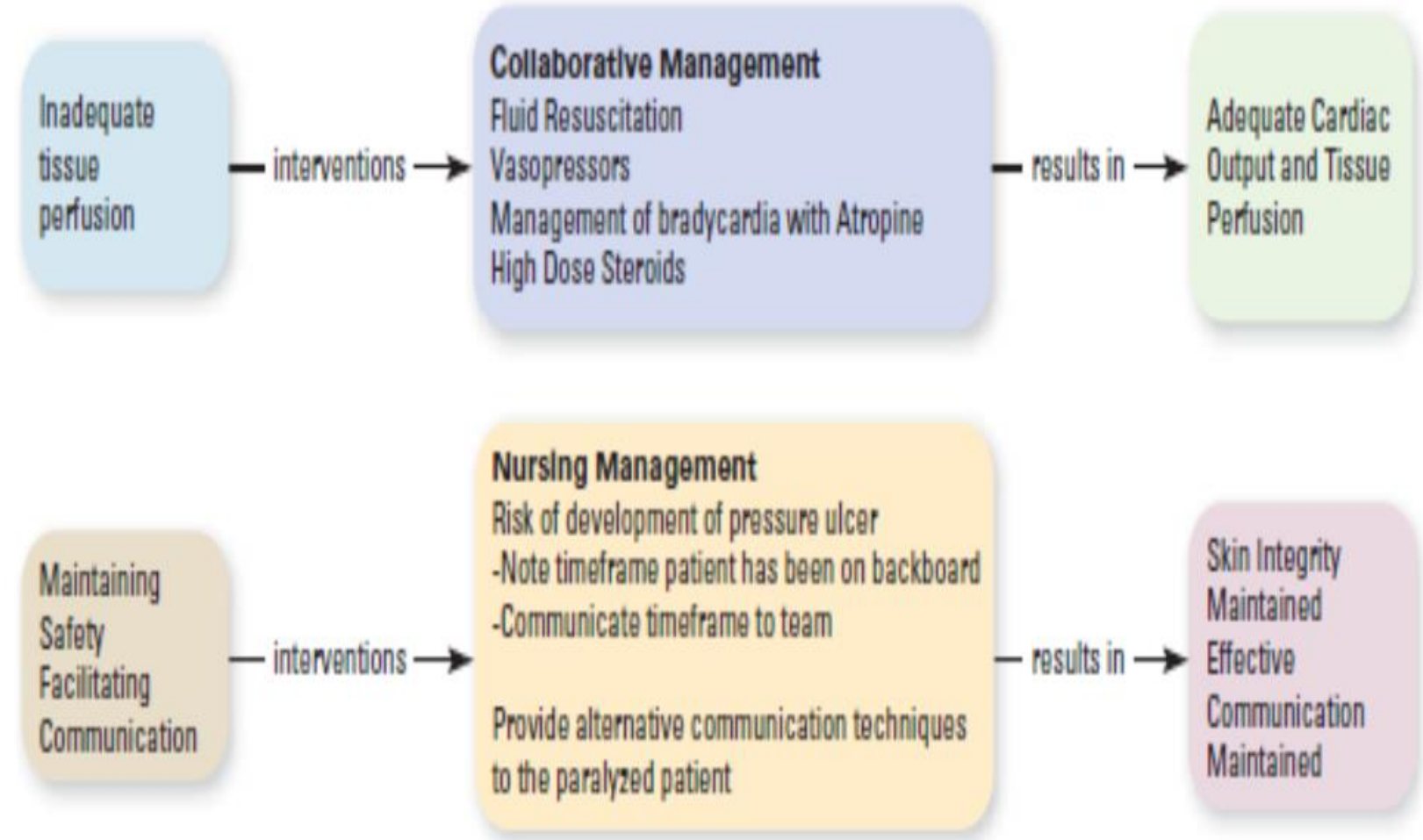


Medical Management

- cardiovascular instability, and promote optimal tissue perfusion. Cardiovascular instability can result from hypovolemia, bradycardia, and hypothermia.

1. Hypovolemia is treated with careful fluid resuscitation. The minimal amount of fluid is administered to ensure adequate tissue perfusion. Volume replacement is initiated for systolic blood pressure lower than 90 mm Hg or evidence of inadequate tissue perfusion.
2. The patient is carefully observed for evidence of fluid overload.
3. Vasopressors are used as necessary to maintain blood pressure and organ perfusion.
4. Hypothermia is treated with warming measures and environmental temperature regulation.





Visual Map 8-6 Neurogenic Shock Collaborative Management

Nursing Management

1. Treating hypovolemia and maintaining tissue perfusion,
2. Maintaining normothermia, monitoring for and treating dysrhythmias,
3. Providing comfort and emotional support, and preventing and maintaining surveillance for complications.
4. All patients at risk for DVT should be started on prophylaxis therapy. DVT-prophylactic measures include monitoring of passive range-of-motion exercises, application of sequential pneumatic stockings, and administration of prescribed anticoagulation therapy