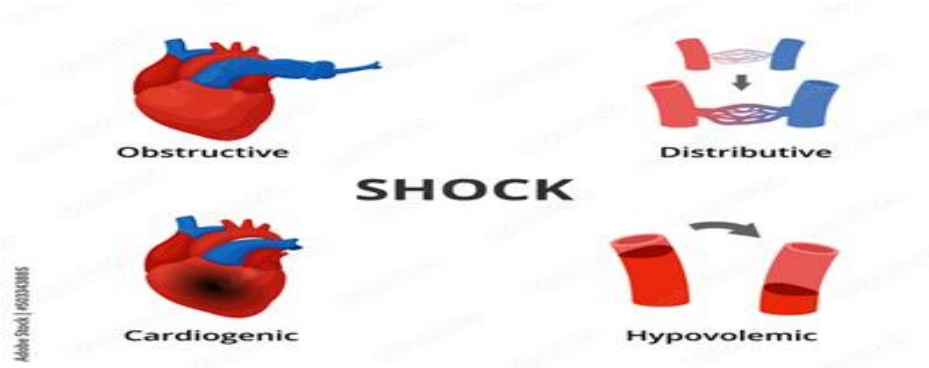
University of Al- Mustaqbal
College Of Nursing
Critical Care Nursing
4th stage
semester 1
lecture 5-6

Shock



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Introduction

Shock as a complex, life threatening condition or syndrome characterized by inadequate blood flow to the tissues and cells of the body. or

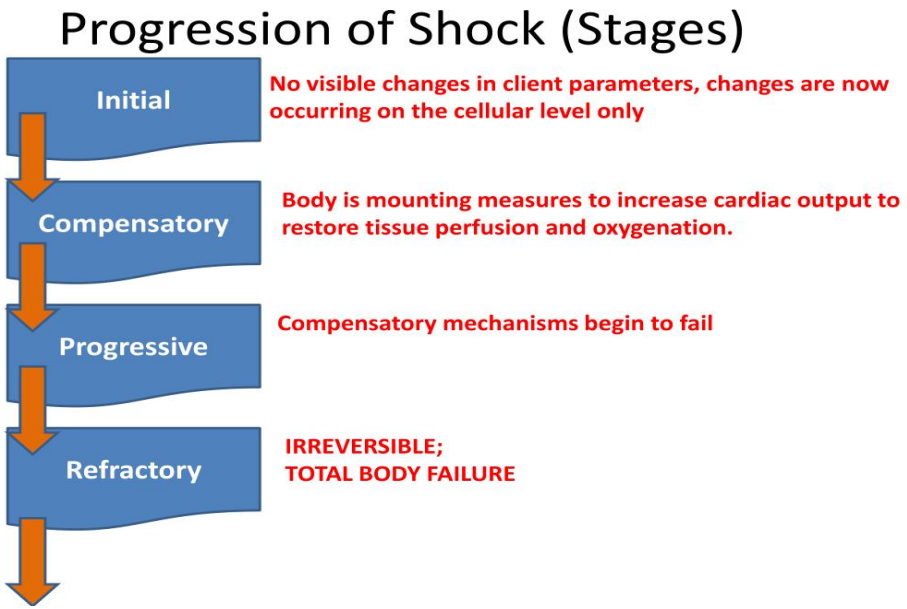
Shock is a condition of impaired tissue perfusion and oxygenation. When oxygen demands exceed oxygen supply, there is a progressive deterioration of cellular dysfunction and organ failure.

The nurse must be able to recognize the impending shock state and administer treatment in a timely fashion. Delays in recognition and treatment can lead to irreversible shock and multisystem organ failure.

What happens in shock.

1. Inadequate perfusion
2. Anaerobic metabolism
3. Buildup of lactic acid
4. Metabolic acidosis
5. Respiratory rate increases in response to metabolic acidosis.

Stages of Shock



Four stages of shock.

a)Initial Stage: the cardiac output is insufficient to supply the norm

nutritional needs of tissues but not low enough to cause serious sym

Cellular level problem, no major hemodynamic changes yet.

↓ oxygen delivery → cells shift from aerobic to anaerobic metabolism.

Anaerobic metabolism → less ATP (2 ATP per glucose instead of 36) → lactic acid accumulation → metabolic acidosis begins. ATP deficiency impairs Na^+/K^+ pump, leading to cell swelling and dysfunction.

- More subtle changes may also be detected in this stage. Decreased perfusion to the brain can result in agitation, and decreased flow to the kidneys can result in a slight decrease in urinary output.

Vital signs in the initial stage of shock:

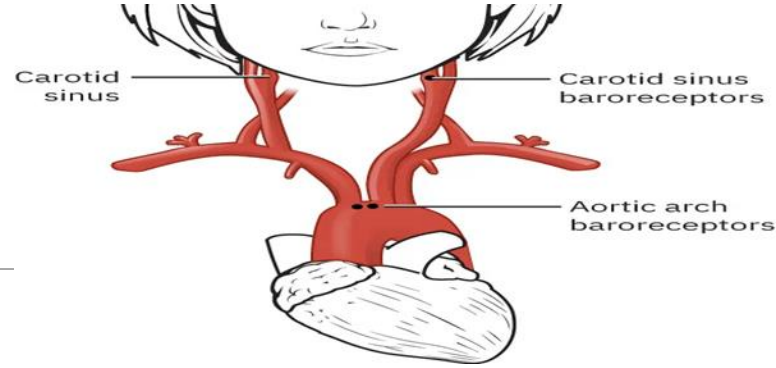
-normal to slightly elevated blood pressure due to stimulation of the sympathetic nervous system.

-normal to slightly elevated heart rate due to blood vessels constrict.

-normal respiratory rate -normal temperature

If adequate cell perfusion is not restored, shock progresses to the compensatory stage.

Stages of Shock



b)Compensatory Stage:

- The body activates compensatory mechanisms to maintain perfusion to vital organs (brain, heart).These mechanisms are primarily neuroendocrine:
- Sympathetic Nervous System (SNS) Activation: Baroreceptors sense ↓ MAP → stimulate SNS. Epinephrine & norepinephrine released → ↑ HR (tachycardia), ↑ contractility, and vasoconstriction. Blood is shunted from skin, GI tract, and kidneys → to heart & brain.
- Renin–Angiotensin–Aldosterone System (RAAS):Renin → angiotensin II → vasoconstriction + aldosterone release. Aldosterone → sodium and water retention → ↑ blood volume.
- Antidiuretic Hormone (ADH):Released from posterior pituitary → water reabsorption by kidneys.
- Respiratory Compensation: Hyperventilation to blow off CO_2 → partial compensation for metabolic acidosis.

Stages of Shock

- Despite these mechanisms, tissue hypoxia develops due to reduced perfusion. In the lungs, endothelial damage increases capillary permeability, causing fluid leakage into alveoli, which can lead to pulmonary edema, respiratory failure, or ARDS if compensation fails.
- **Vital signs in the compensatory stage of shock include:**
- low blood pressure
- high heart rate
- normal to slightly elevated respiratory rate
- normal temperature
- cool skin
- decreased urine output

If the cause of shock is identified and appropriate interventions are implemented, the situation is reversible with no permanent damage to cells and tissues. If the cause of shock is not corrected, the next stage of shock begins.

C)progressive Stage :

In the progressive stage , cellular perfusion drops severely , leading to increased capillary permeability . This causes proteins and fluids to leak from the intravascular space into the interstitial space , disrupting fluid balance and resulting in hypotension, electrolyte imbalance, and impaired tissue perfusion .

- As oxygen delivery declines , cells shift to anaerobic metabolism , producing lactic acid and **causing metabolic acidosis** . All body systems become hypoxic:

- **Skin:** Pale, cool, and clammy due to vasoconstriction.
- **Brain:** confusion or loss of consciousness .
- **Heart:** tachydysrhythmias , myocardial ischemia , or infarction , cardiovascular collapse.
- **Lungs:** pulmonary edema , impaired gas exchange , tachypnea , crackles , respiratory acidosis , and may progress to ARDS .
- **Gastrointestinal system:** Poor perfusion damages the mucosal barrier , leading to ulcers , bacterial translocation , and malabsorption .
- **Kidneys:** oliguria (<30 mL/hr) , elevated BUN and creatinine , and worsen metabolic acidosis .
- **Liver:** jaundice , elevated liver enzymes , reduced immune function , and loss of metabolic regulation .

- **If timely treatment restores perfusion and oxygenation, recovery is possible ; otherwise, multi-organ failure ensues.**

d) Stages of Shock :Irreversible Stage:

- The refractory stage of shock is characterized by extreme dysfunction of cellular processes in multiple body systems. The significant increase in capillary permeability, anaerobic metabolism, lactic acid buildup, metabolic acidosis, tachycardia, and profound hypotension continues despite treatment. Cells, tissues, and organ systems shut down because of irreversible cell and organ damage, and recovery is unlikely.

- vital signs in the refractory stage of shock include mottled skin, characterized by a bluish-red lace pattern under the skin, caused by the pooling of deoxygenated blood. Other manifestations include:

-continued low blood pressure despite treatment

-increased tachycardia

-low to no urine output

-weak to absent peripheral pulses

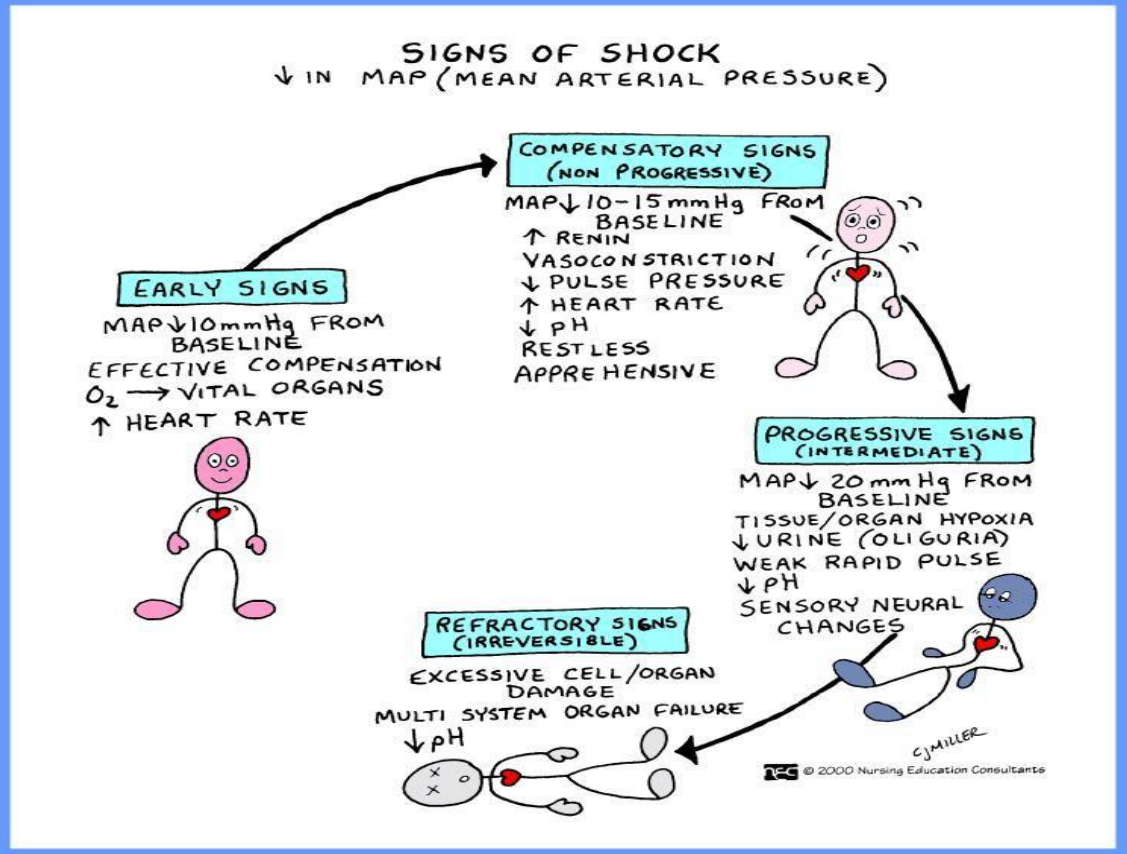
- increased respiratory rate

- cool skin

- low temperature

Summarizes vital sign changes in each stage of shock.

Stage	Initial	Compensatory	Progressive	Refractory
Blood pressure	Normal	Low	Low	Low despite treatment
Heart rate	Normal	Elevated	Elevated	Elevated
Respiratory rate	Normal	Elevated	Elevated	Elevated
Urinary output	Normal	Low	Low	Low to zero
Temperature	Normal	Normal	Low	Low
Skin	Normal	Cool	Cool	Cool
Distal pulses	Normal	Normal	Weak	Weak to absent

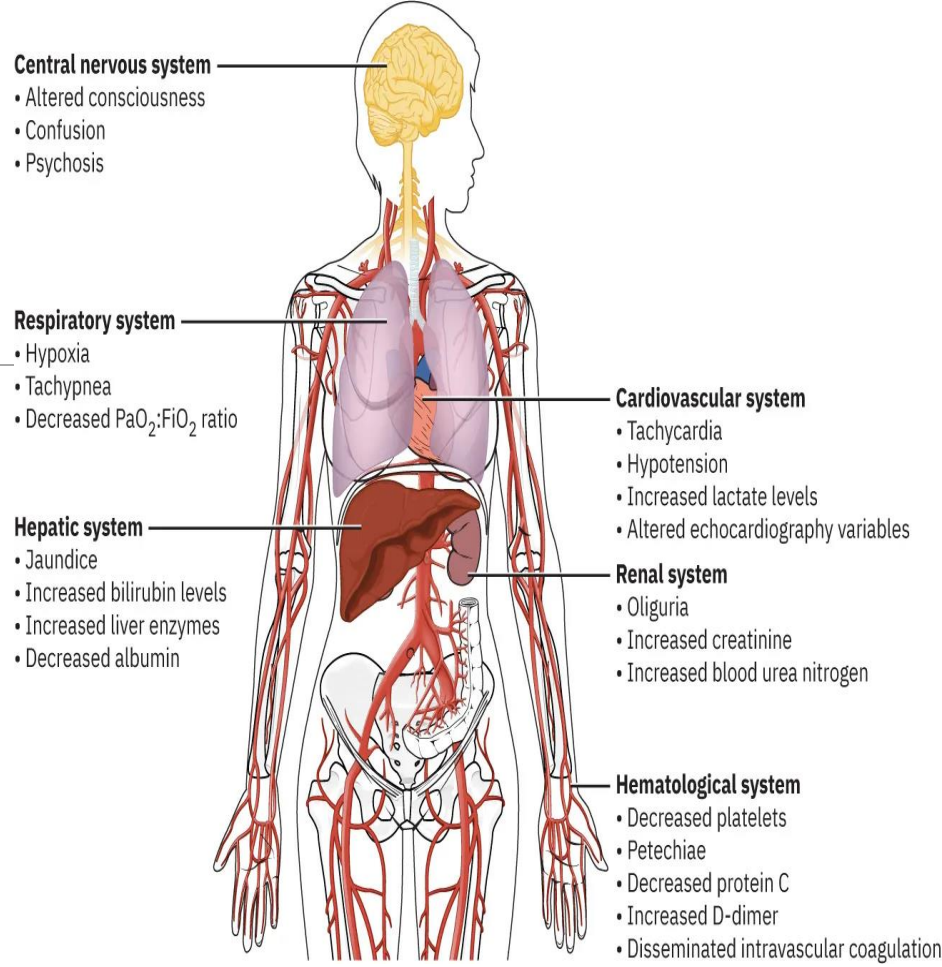


Multiorgan Failure

Multiorgan dysfunction syndrome (MODS), or multiorgan failure (MOF), results from prolonged cellular hypoperfusion. It is most often a result of hypovolemic and septic shock. The prognosis for MOF/MODS is dependent upon the number of organs affected and the body's response to treatment.

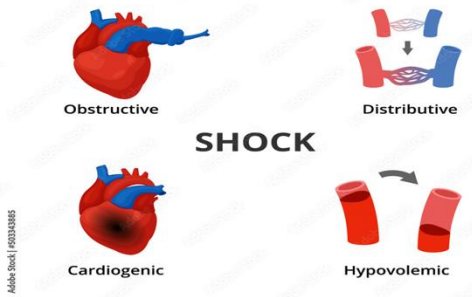
A normal inflammatory response to decrease cell perfusion and cellular injury facilitates healing. Part of the normal inflammatory response includes the release of cytokines, which are protein substances that regulate the inflammatory response.

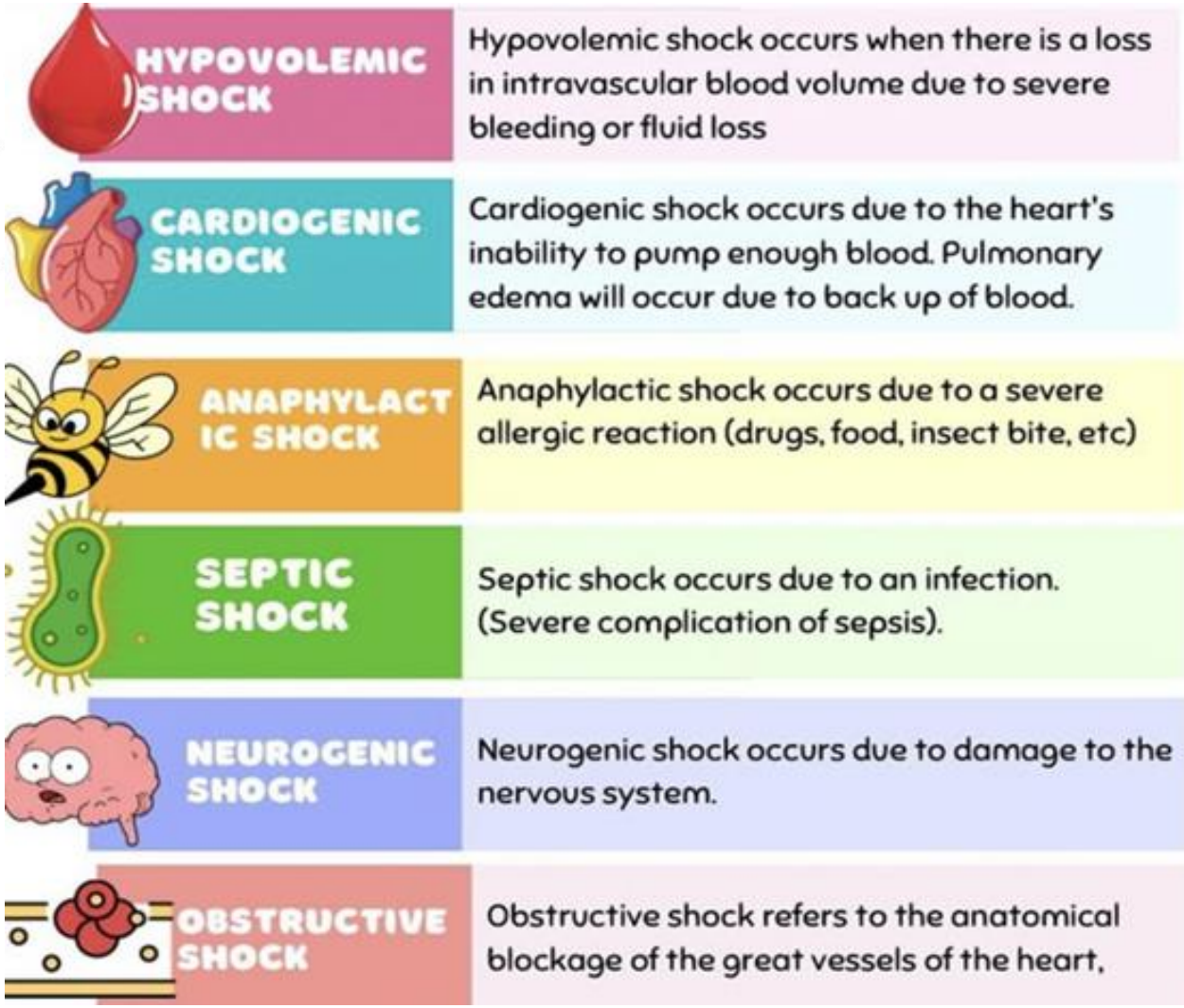
MOF and MODS results when the inflammatory response extends beyond the area of injury throughout the body for a prolonged period of time. The development of MOF/MODS in sepsis has been linked to the release of cytokines. This causes an exaggerated inflammatory response throughout the body, leading to a cascade of events that can ultimately result in cell hypoxia, cell death, and organ failure.

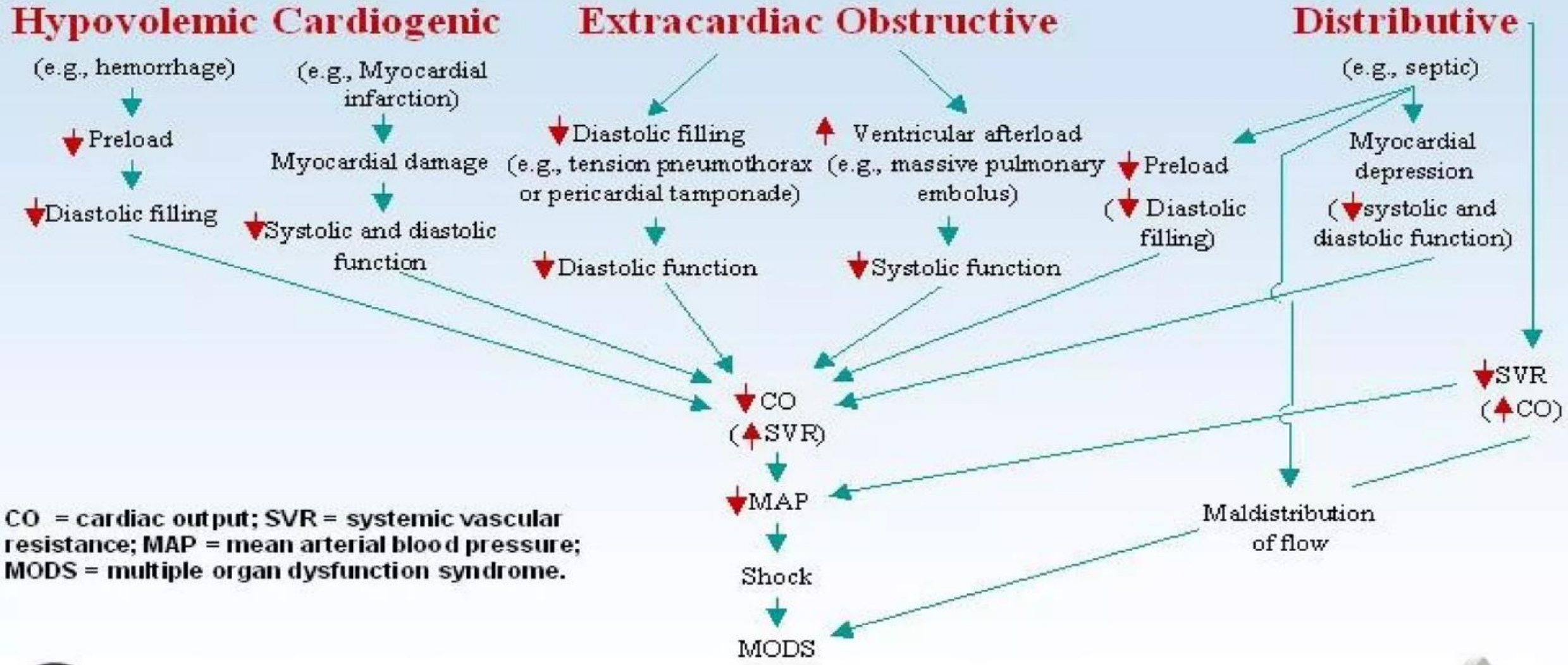


Type of shock

- **Low blood flow**
  - Hypovolemic
  - Cardiogenic
- **Maldistribution of blood flow**(Distributive Shock or Circulatory Shock)
  - Septic
  - Anaphylactic
  - Neurogenic
- **Obstructive shock**







CO = cardiac output; SVR = systemic vascular resistance; MAP = mean arterial blood pressure; MODS = multiple organ dysfunction syndrome.

Kumar and Parrillo, 2001

Society of Critical Care Medicine
The Intensive Care Professionals

I. Low blood flow

A .Hypovolemic shock

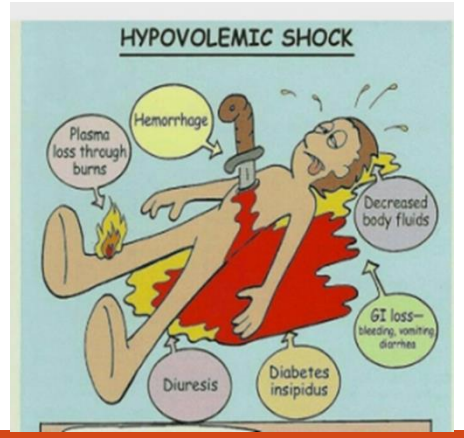
- ✓Hypovolemic shock results from a decreased circulating volume. It is the most common type of shock.
- ✓Body fluid is contained **in the intracellular** and **extracellular compartments**.
- ✓ Intracellular fluid accounts for about two thirds of the total body water.
- ✓The extracellular body fluid is found in one of two compartments: **intravascular** (inside blood vessels) or **interstitial** (surrounding tissues).
- ✓The volume of interstitial fluid is about three to four times that of intravascular fluid.
- ✓Loss of approximately 15% to 30% or more of the normal blood volume can produce hypovolemic shock ,which represents an approximate loss of 750 to 1500 mL of blood in a 70-kg person.

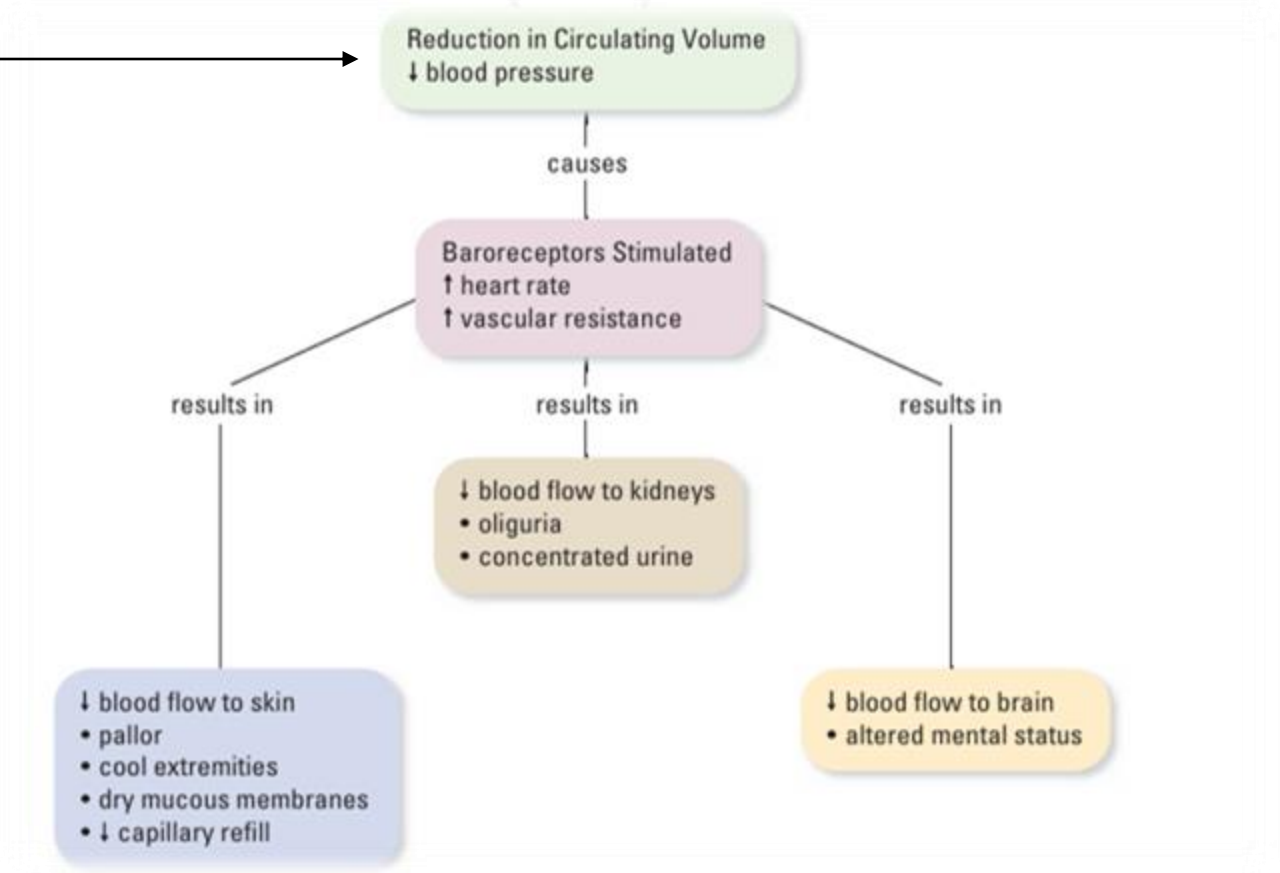
Etiology and Pathophysiology:

1. **EXTERNAL :**
   - Trauma
   - Surgery
   - Vomiting
   - Diarrhoea
   - Diuretics
   - Diabetes Insipidus
   - External bleeding

2. **INTERNAL:**
   - Haemorrhage
   - Burns
   - Ascites
   - Peritonitis
   - Dehydration
   - Internal bleeding

Diabetic ketoacidosis





Assessment

Clinical Manifestations:

- Restlessness, anxiety, Altered mental status
- Tachycardia (early compensatory)
- Hypotension (late sign)
- Dry mucous membranes, Poor skin turgor, Thirst, Weight loss
- Weak, thready pulse
- Cool, pale, clammy skin

- ↓ Urine output (<0.5 mL/kg/hr,)
- Rapid, shallow breathing

Lab Findings:

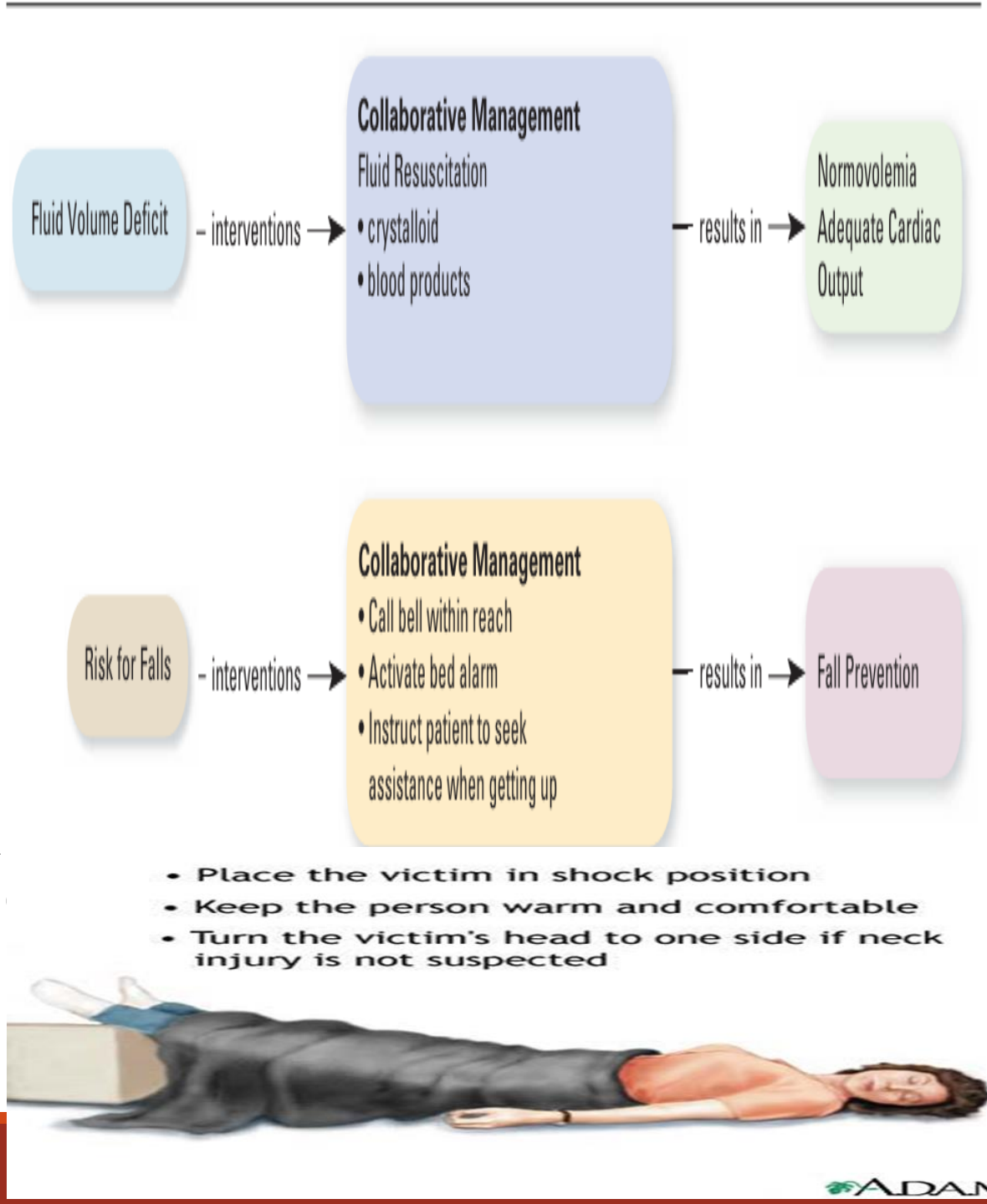
- ↑ Hematocrit (dehydration) or ↓ (hemorrhage)
- ↑ Lactate, metabolic acidosis
- Electrolyte imbalances (Hypernatremia)

Hemodynamic Findings

- the mean arterial pressure (MAP), central venous pressure/ right atrial pressure (CVP/RAP), and pulmonary artery wedge pressure (PAWP) are decreased, reflecting a reduced preload.
- Decrease in stroke volume (SV) and cardiac output (CO) or cardiac index (CI).
- An elevated systemic vascular resistance index (SVRI) due to compensatory mechanisms

Nursing Actions

- Ensure **airway and oxygenation (always 1)**
- Establish **IV access (2 large-bore IVs)**
- **Patient in Trendelenburg position** (lower extremities are elevated to an angle of about 20 degrees).
- Administer **crystalloids or blood products** per protocol
- Monitor **vital signs every 5 minute , urine output, mental status**
- Prevent **hypothermia** (wrap patient, warm fluids)
- Monitor fluid resuscitation parameters. A positive response to fluid therapy would be indicated by a MAP of ≥to 65 mmHg, UOP ≥ 0.5 to 1 mL/kg/hr, and a decreasing lactate level.
- assess the patient closely during fluid resuscitation for signs of respiratory compromise from pulmonary congestion.
- Monitoring for complications and side effects of treatment.



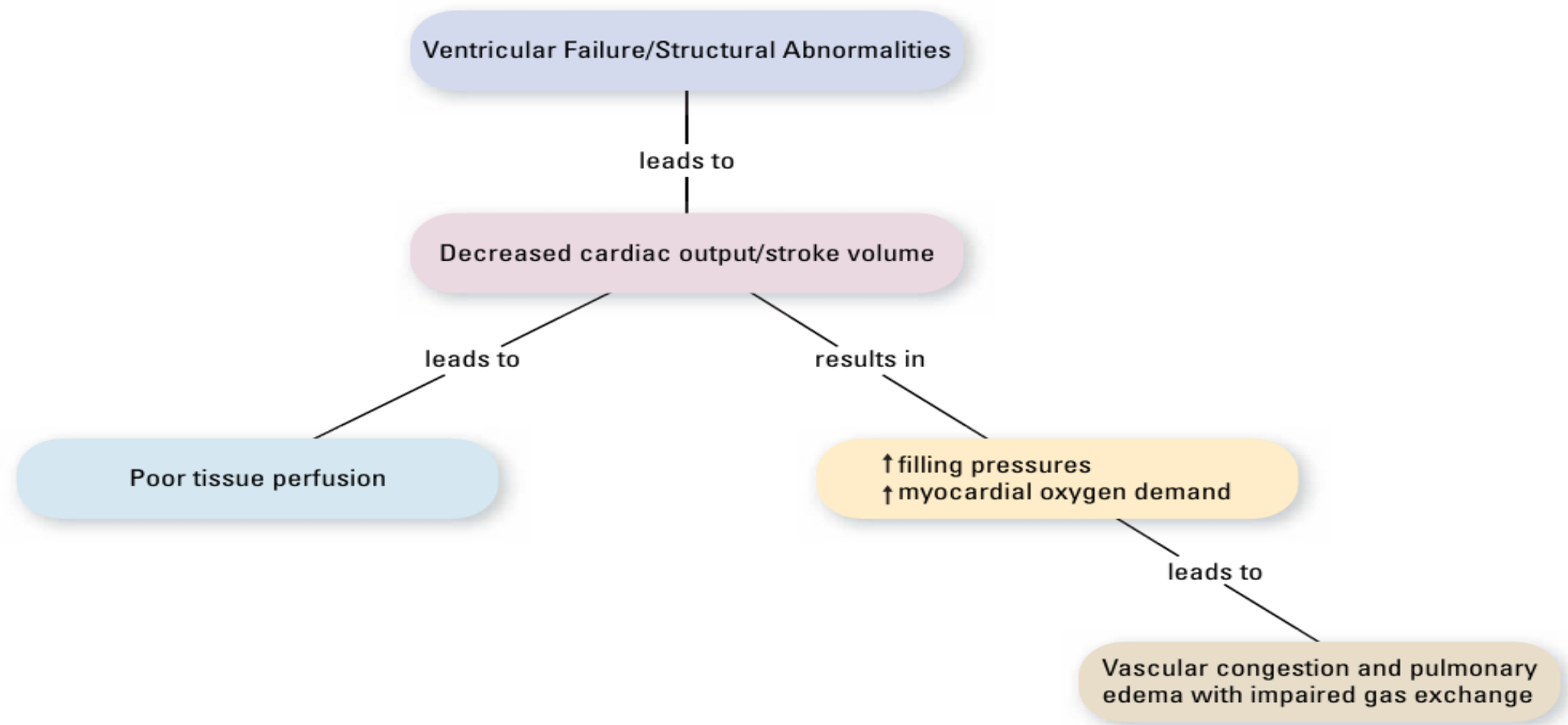
I. Low blood flow B. Cardiogenic Shock

Cardiogenic shock is a result of the heart's inability to deliver adequate circulation to the tissues due to cardiac pump failure. This occurs in the presence of adequate circulating volume.

Etiologies:

- **Damage to the myocardium: as myocardial infarction or cardiomyopathy,**
- **Structural defects to the cardiac values or septum,**
- **Arrhythmias .**
- **conditions that stress he myocardium** (e.g., severe hypoxemia, acidosis, hypoglycemia, hypocalcemia, tension pneumothorax)

Cardiogenic Shock pathophysiology



Pathophysiology

1. Cardiogenic Shock and Hemodynamics

1. In **cardiogenic shock**, the heart's ability to pump blood is **impaired**.
2. This leads to a **reduction in stroke volume (SV)** — the amount of blood ejected per beat — and consequently a **decrease in cardiac output (CO)**.
3. **Reduced CO → poor tissue perfusion**, causing hypoxia at the cellular level.

2. Increased Filling Pressures

1. When SV is reduced, **blood remains in the ventricles**, increasing **end-diastolic volume and pressure**.
2. The **left ventricle** faces higher filling pressures, which increases **myocardial wall stress** and **oxygen demand**, worsening ischemia.

3. Backward Transmission and Pulmonary Effects

1. Elevated **left ventricular pressures** are transmitted backward into the **left atrium** and **pulmonary veins**.
2. This leads to **pulmonary vascular congestion**, causing **pulmonary edema**.
3. Pulmonary edema **impairs gas exchange**, reducing oxygenation of blood and worsening tissue hypoxia.

Signs and symptoms of cardiogenic shock

- Hypotension (systolic blood pressure less than 90 mmHg)
- Tachycardia
- Delayed capillary refill
- Cool, Pallor; clammy , mottled extremities
- Elevated jugular venous distention
- If pulmonary edema is present, the patient will **exhibit dyspnea and crackles upon auscultation of the lungs**
- Oliguria. concentrated urine
- Altered mental status .Anxiety, confusion, agitation

Assessment finding:

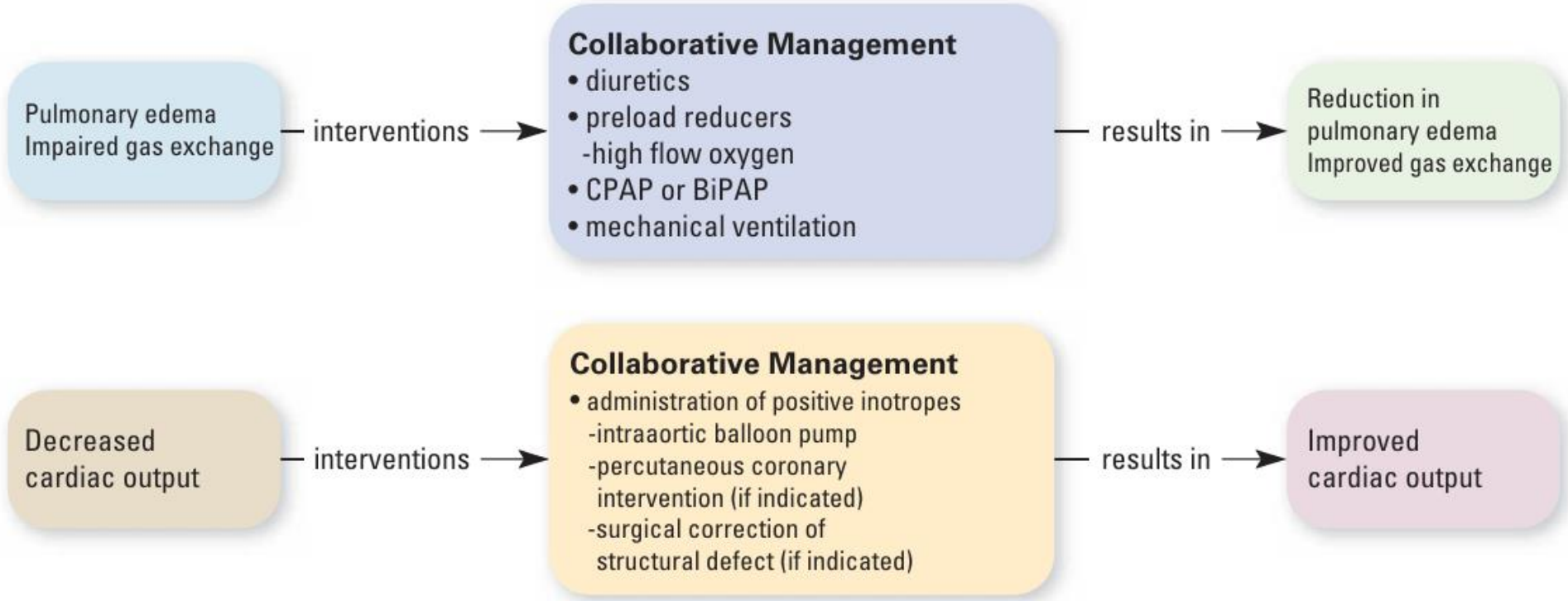
❑**Lab findings include:**

- Arterial blood gases: metabolic acidosis, hypoxemia
- ↑ Cardiac enzymes (if MI).
- Elevated serum lactate (serum lactate greater than 4 mmol/L)
- Reduced mixed venous oxygen saturation (SvO2 less than 60%; ScvO2 less than 70%)
- Pulmonary edema on chest X-ray

❑**Hemodynamic findings include:**

- ○ Reduced cardiac index
- ○ Reduced stroke volume
- ○ Elevated PAWP, CVP/RA, SVRI

Cardiogenic Shock Collaborative Management



Nursing Actions

- frequently monitoring the patient's vital signs every 5 minute ,cardiac status and electrocardiographic and pulse oximeter
- Assess cardiodynamics (cardiac index, pulmonary artery pressures).
- Administer medication (**inotropes** (dobutamine) and **vasopressors**) cautiously to support blood pressure, reduce chest pain and reduce pulmonary edema if present.
- Oxygen therapy (maintain $SpO_2 > 92\%$)
- Monitor fluid balance closely; and record in-put and out-put to avoid fluid overload
- Continuous cardiac monitoring
- Elevate head of bed to reduce preload
- Monitoring for signs of complications and side effects of treatment
- If indicated, prepare patient for percutaneous coronary intervention (PCI) including obtaining informed consent.

2. Maldistribution of Blood Flow (Distributive Shock or Circulatory Shock)

Distributive shock is characterized by vasodilation and redistribution of blood volume.

Distributive shock occurs when **intravascular volume pools in peripheral blood vessels**.

This abnormal displacement of intravascular volume causes a **relative hypovolemia** because not enough blood returns to the heart, which leads to inadequate tissue perfusion.

The ability of the blood vessels to constrict helps return the blood to the heart. Vascular tone is determined both **by central regulatory mechanisms, as in BP regulation**, and **by local regulatory mechanisms**, such as tissue demands for oxygen and nutrients.

Therefore, distributive shock can be caused by either **a loss of sympathetic tone** or **a release of biochemical mediators from cells** that causes **vasodilation**.

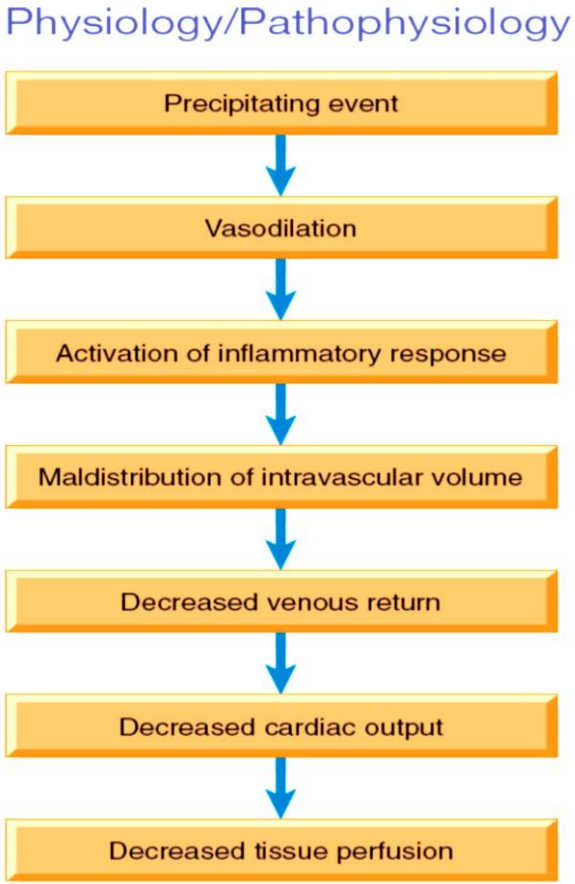


Figure 11-7 • Pathophysiologic sequence of events in distributive shock.

Septic Shock:

4. SEPTIC SHOCK

- The most common type of circulatory shock, is caused by widespread of infection.
- The incidence is more in ICU and is increasing day by day. Mortality rates as high as 50%
- The most common cause of death.
- The incidence can be reduced by using strict aseptic technique, thorough hand hygiene techniques.
- Interventions include prevention of central line infection, early debriding of wounds to remove the necrotic tissues, carrying out standard precaution, adhering to infection control practices, prompt cleaning and maintaining of equipment.

Septic Shock Risk Factors

- Immunosuppression
- Malnourishment
- Chronic illness
- Invasive procedures

Causes

- UTI , abortion
- RTA
- Severe burn
- Due to chronic diseases : diabetes, AIDS
- Indwelling lines and catheter
- Improper wound care and management

Septic Shock:

Sepsis: systemic inflammatory response to a documented or suspected infection

- Septic Shock: presence of sepsis with hypotension despite fluid resuscitation along with the presence of tissue perfusion abnormalities.

The body responds through both hyper-inflammatory and anti-inflammatory means.

- Endotoxins released by the invading organisms prompt release of hydrolytic enzymes from weakened cell lysosomes, which causes cellular destruction of bacteria and normal cells
- When the body is unable to control the proinflammatory mediators, it produces a systemic inflammatory response
- As a result, there is widespread cellular dysfunction to the endothelium, resulting in vasodilation, increased capillary permeability, and platelet aggregation and adhesions to the endothelium

Clinical manifestations of Septic Shock

- Early: warm, flushed skin, fever, bounding pulses
- Late: cool, pale skin, hypotension, altered LOC
  - Tachypnea/hyperventilation
  - Temperature dysregulation
  - ↓ Urine output
  - Altered neurologic status
  - GI dysfunction
  - Respiratory failure is common.
  - Hyperthermia
  - Multiple organ failure
  - Anuria

Nursing Management

- Identifying and eliminating the cause of infection. Primary prevention of shock is an essential focus on nursing intervention;
- Specimens of blood, sputum, urine, wound drainage, are collected for culture using aseptic technique.
- All invasive procedures must be carried out with aseptic technique and monitored for signs of infection in all patients.
- Antibiotic agents are started until culture and sensitivity reports are received, IV antibiotics within 1 hour Fluid resuscitation (crystalloids)Vasopressors if hypotensive after fluids,
- Monitor lactate, urine output, hemodynamics
- Nutritional supplementation within the first 24 hours of the onset of shock.
- The nurse identifies patients at particular risk for septic shock (ie, elderly , immunosuppressed trauma , burns and diabetes).

Neurogenic Shock Neurogenic shock

(also called spinal shock) occurs when a spinal cord injury (SCI) to the cervical and upper thoracic spinal cord above T5 causes vasodilation occurs as a result of a loss of balance between parasympathetic and sympathetic stimulation.

This loss of sympathetic nervous system (SNS) innervation leads to immediate loss of autonomic and motor reflexes below the level of the injury.

Specifically, the loss of the autonomic reflexes results in vasodilation and redistribution of the blood volume, loss of cutaneous control of sweat glands resulting in warm, dry skin, and parasympathetic regulation of the heart rate functioning unopposed resulting in bradycardia.

Neurogenic Shock Risk Factors:

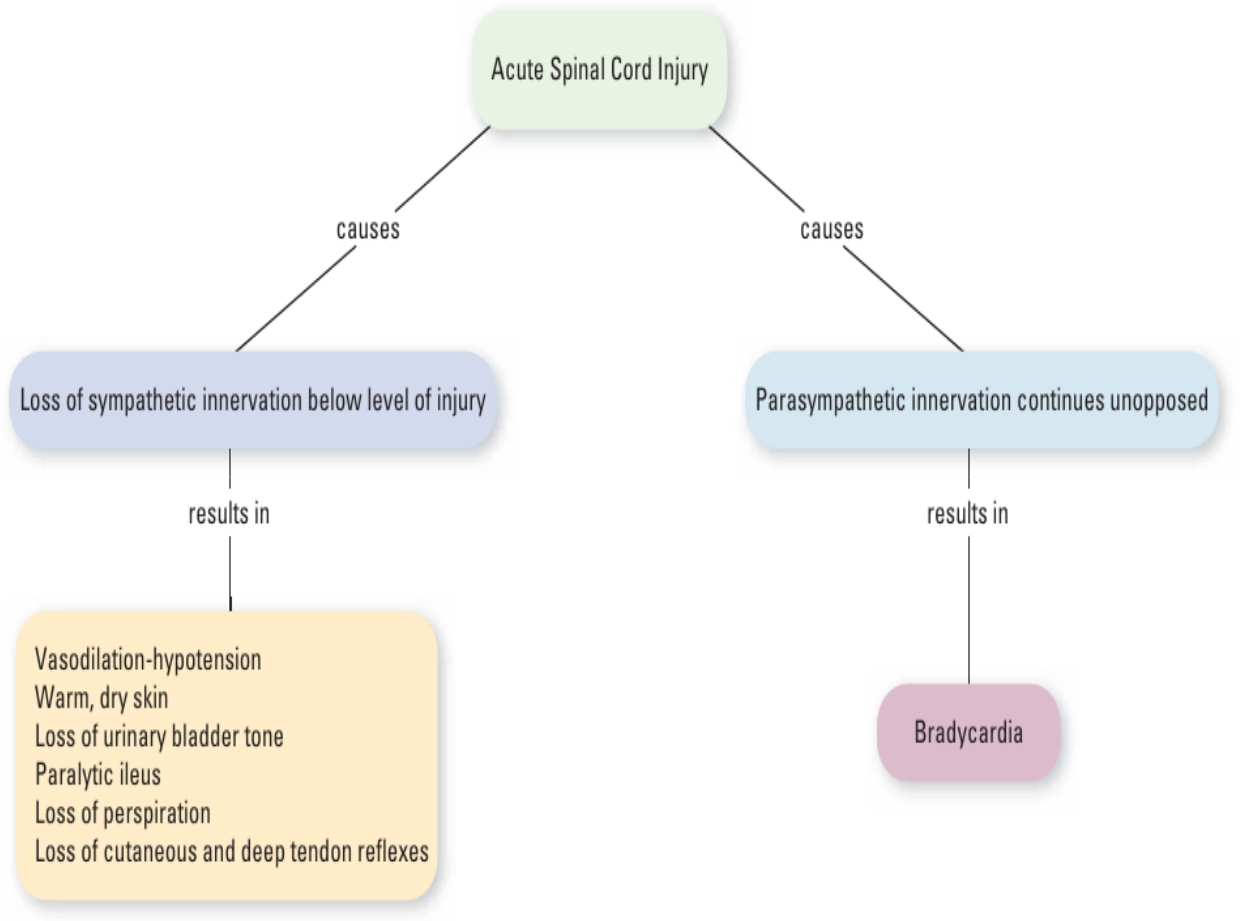
Spinal cord injury

Spinal anesthesia

Depressant action of medications

Glucose deficiency

Neurogenic Shock Neurogenic shock



Signs and Symptoms:

Hypotension without compensatory tachycardia , Bradycardia

• Warm, dry skin

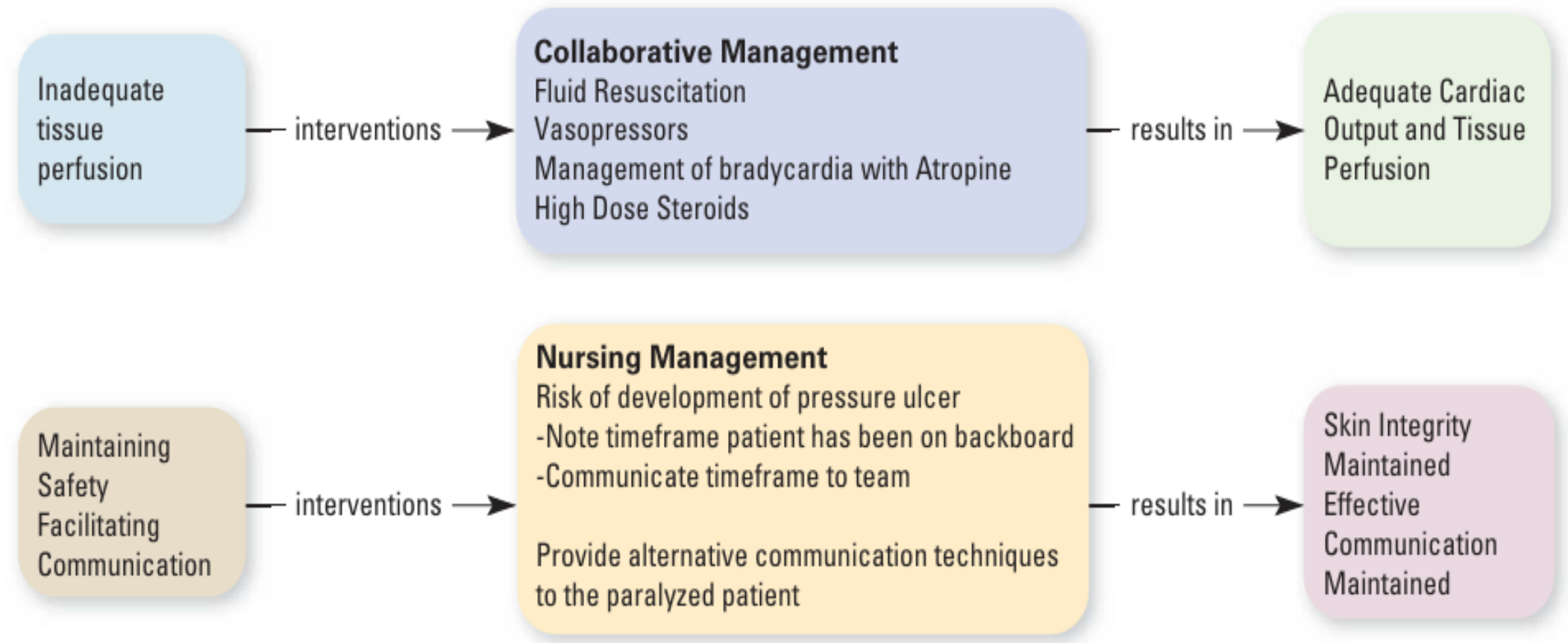
Associated physical findings include:

• Flaccid paralysis below level of injury.

• Loss of cutaneous and deep tendon reflexes below level of injury.

• Bowel and bladder dysfunction.

Nursing Interventions:



Nursing Interventions:

- IV fluids carefully
- Vasopressors (phenylephrine) . Infuse vasopressors through a central line if possible. If infusion through a peripheral line is absolutely necessary, inspect the site hourly for signs and symptoms of extravasation.
- Extravasation of vasopressor agents can be extremely damaging to the surrounding tissue
- Immobilization of spine
- Ensure an adequate airway and monitor for adequate respiration, assist if necessary.
- Monitor blood pressure and heart rate. A positive response would be a MAP greater than 65 mmHg and a heart rate within normal limits.
- Monitor for bradyarrhythmias, prophylaxis DVT
-

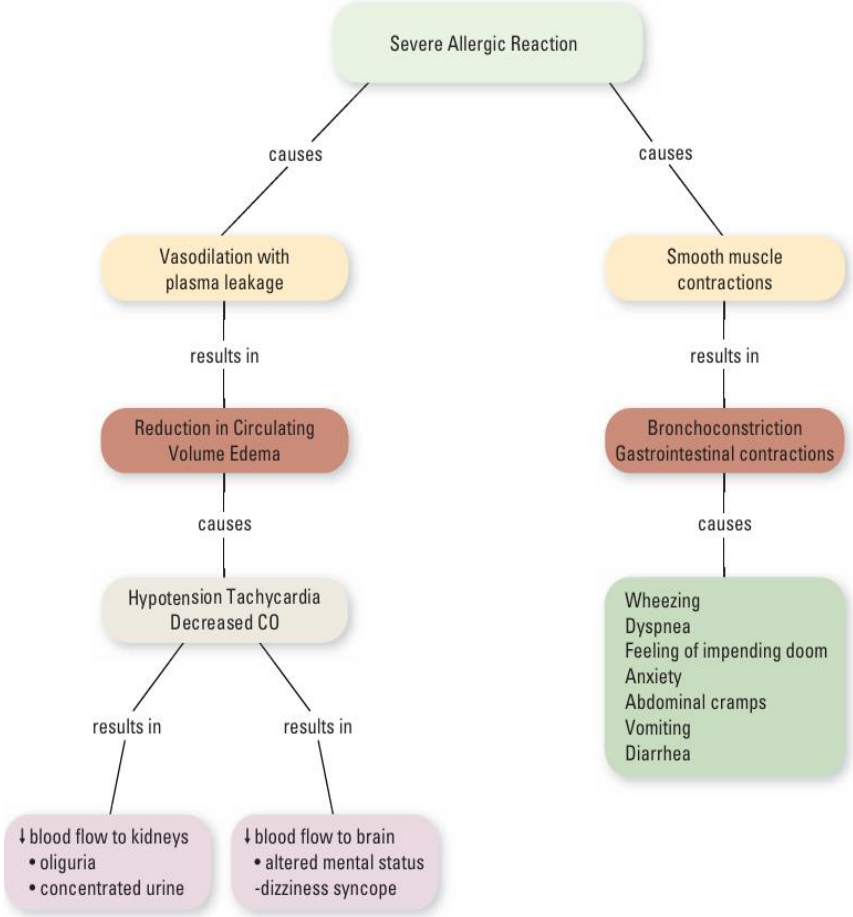
Anaphylactic Shock

❑**caused by a severe allergic reaction** when patients who have already produced antibodies to an antigen (foreign substance) develop a systemic antigen–antibody reaction; specifically, an immunoglobulin E (IgE)- mediated response.

➢ This antigen–antibody reaction **Neurogenic shock mast cells to release potent vasoactive** substances, such as **histamine** or **bradykinin**, and **activates inflammatory cytokines**, causing **widespread vasodilation** and **capillary permeability**.

Anaphylactic Shock Risk Factors

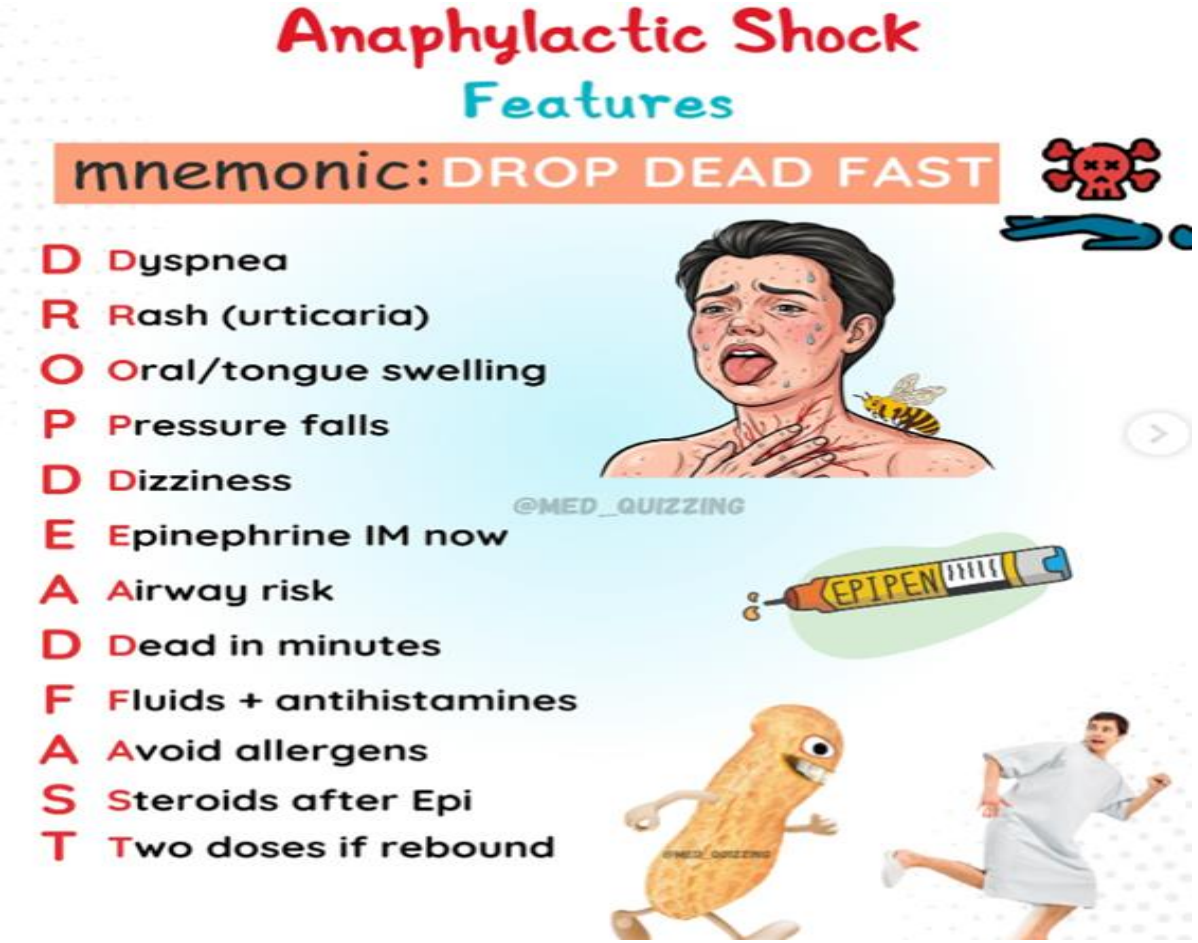
- ➢ Penicillin sensitivity
- ➢ Transfusion reaction
- ➢ Bee sting allergy
- ➢ • Foods.
- ➢ • Contrast for imaging.
- ➢ •Latex sensitivity



Signs of Anaphylactic Shock

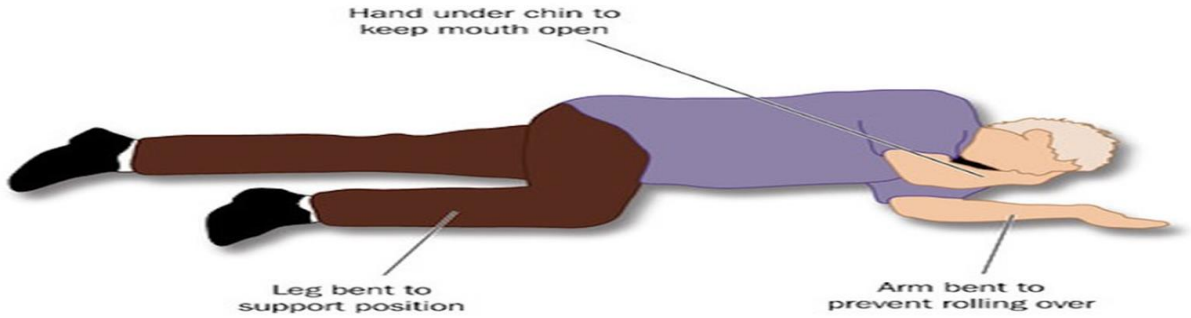
Often an anaphylactic response is not the first exposure for the patient. The re-exposure to the antigen triggers the overwhelming response.

- Itching
- Redness
- Swelling
- Progressing to:
  - Tightness in the chest
  - Anxiety
  - Difficulty breathing
  - Hypotension
  - Tachycardia
  - Syncope



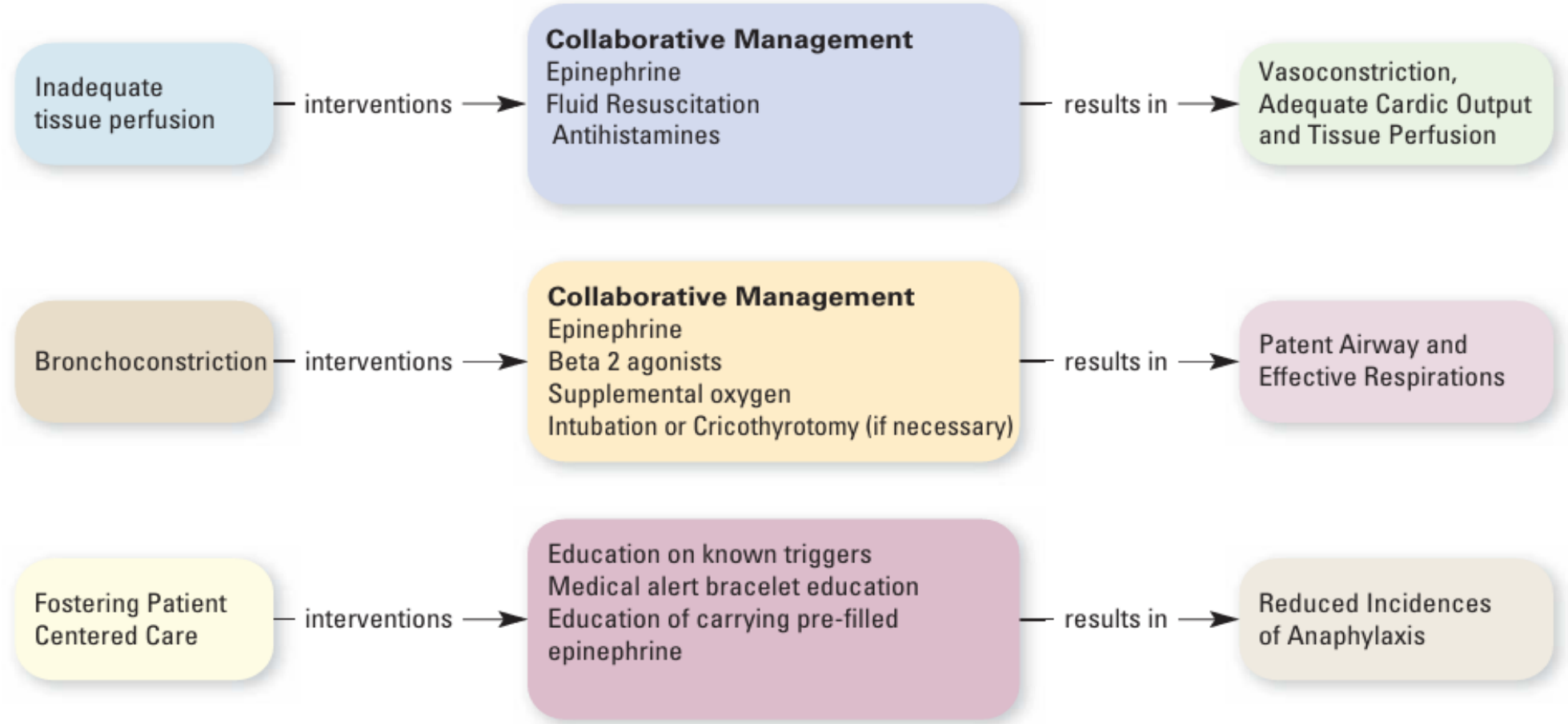
Nursing Actions

- Closely monitor for a patent airway and respiratory drive.
- Notify provider immediately with worsening respiratory status.
- Assist provider with intubation or cricothyrotomy if indicated.
- Monitor blood pressure and heart rate. A positive response would be a MAP greater than 65 mmHg and a heart rate within normal limits.
- Administer epinephrine as ordered without delay.
- Oxygen, IV fluids
- Antihistamines, corticosteroids
- Monitor airway and vital signs



- Help client sit up in position of easiest breathing - put unresponsive client who is breathing in recovery position.

Intervention



Obstructive Shock

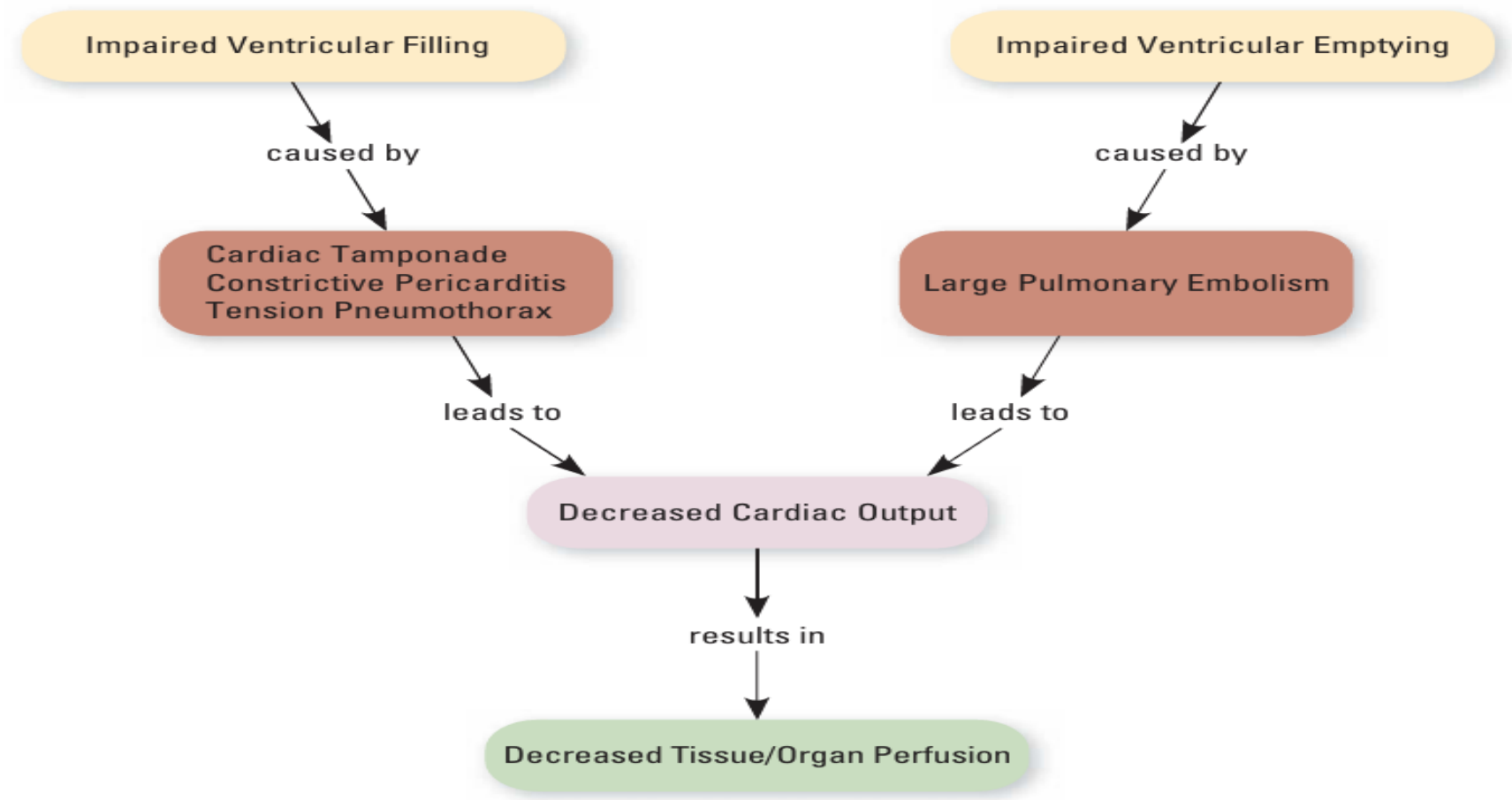
Patients at risk for developing cardiac tamponade include those who have experienced a blunt or penetrating trauma to the chest.

Acute pericarditis and the development of fluid accumulation in the pericardial space may result from viral or bacterial infection, autoimmune disorders like rheumatoid arthritis, uremia, post myocardial infarction, post cardiac surgery, irradiation, or tumors.

The development of fibrosis or constrictive pericarditis can occur as a result of any of the above conditions.

The diagnosis of cardiac tamponade is often through echocardiography. If the patient is very unstable, this will be done at the bedside.

Pathophysiology

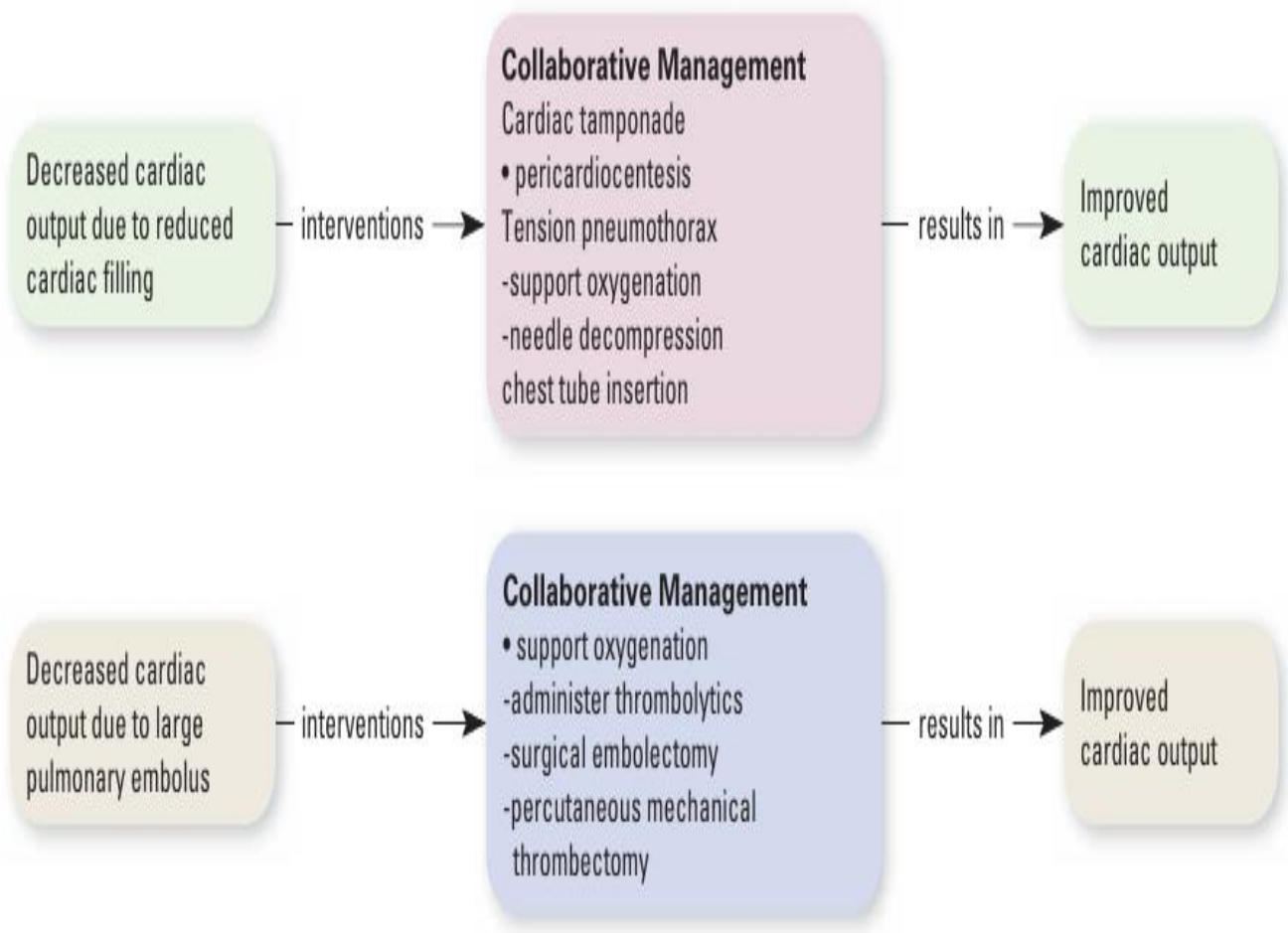


Assessment

- Hypotension
- Pulsus paradoxus (a drop on systolic BP greater than 10 mmHg on inspiration)
- Tachycardia
- Muffled heart tones (cardiac tamponade)
- Reduced capillary refill
- Tachypnea
- Elevated JVD (depending on volume status)
- Crackles
- Unilateral absence of breath sounds (tension pneumothorax)
- Tracheal deviation (tension pneumothorax)
- Oliguria
- Altered mental status

Hemodynamic Findings

- MAP is reduced.
- The CVP/RAP and PAWP are increased reflecting impaired atrial filling.
- A reduction in SV and CO/CI.
- An elevated SVR may be present reflecting the vasoconstriction due to compensatory mechanisms.



Nursing Actions

- Emergency intervention to relieve obstruction (chest tube, pericardiocentesis)
- Oxygen therapy
- Closely monitor heart rate, blood pressure, respiratory rate and pulse oximetry during procedures. Continuous hemodynamic monitoring
- Administer the thrombolytic without delay for patient with hemodynamically unstable pulmonary embolism.
- Closely monitor the patient for side effects such as external bleeding or signs and symptoms of cerebral bleeding, such as change in level of consciousness or change in neurological exam.
- Patients receiving thrombolytics must have a baseline neurological exam done prior to initiation of the medication.

Nursing Priorities

1. **Early recognition & rapid intervention**
2. **Airway & oxygenation**
3. **Restore tissue perfusion** (fluids, medications)
4. **Prevent complications:** organ failure, infection, pressure sores
5. **Patient & family support.**


Thank You