SHOCK

Historical considerations

- LeDran in 1743 was the first to use the French term "choc" in medicine. It means "push" or "impact"
- Belief – symptoms denoting fear or some other form of altered cerebral function
- Crile in 1899 showed that replacement of blood volume decreased mortality experimentally

Definition of Shock

- **A physiological state characterized by a significant, systemic reduction in tissue perfusion (hypoperfusion), resulting in decreased tissue oxygen delivery and insufficient removal of cellular metabolic products, resulting in tissue injury.**

Classification of Shock

- **Hypovolemic**
- **Septic/Inflammatory**
- **Cardiogenic (Intrinsic, compressive & Obstructive)**
- **Neurogenic**
- **Anaphylactic**

Main Classes of Shock

- **Hypovolemic Shock**
- **Distributive Shock**
- **Cardiogenic Shock**
- **Obstructive Shock**

Distributive Shock

- **Septic**
- **Anaphylactic/ Anaphylactoid**
- **Neurogenic**

Obstructive Shock

- Pulmonary Embolism
- Cardiac Tamponade
- Pneumothorax

Pathophysiology of Shock

- **Shock develops with inadequate capillary perfusion by decreased Cardiac Output following heart attack (cardiogenic shock) or blood/volume loss (hypovolemic shock)**

Clinical Markers of Shock:

- **Brachial systolic blood pressure: <110mmHg**
- **Sinus tachycardia: >90 beats/min**
- **Respiratory rate: <7 or >29 breaths/min**
- **Urine Output: <0.5cc/kg/hr**
- **Metabolic acidemia: [HCO3]<31mEq/L or base deficit>3mEq/L**
- **Hypoxemia: (0-50years: <90mmHg);**
  (51-70yearsr: <80mmHg;)
  (>71years<70mmHg;)
- **Cutaneous: vasoconstriction vs. vasodilation.**
- **Mental Changes: anxiousness, agitation, indifference, lethargy, obtundation**

Determinants of Shock:

- Inadequate tissue perfusion
- Sustained loss of effective circulatory blood volume
- Breakdown of cellular metabolism and microcirculatory homeostasis
- Hypoperfusion of peripheral tissue that leads to a diminutive transcapillary exchange function
- Disproportion between VO_2 and DO_2

Hemodynamic States of Shock

- **Hyperdynamic State**
- **Hypodynamic State**
- **Related to:**
  - Cardiac Output (CO)
  - Systemic Vascular Resistance (SVR)

Comparison of Different Forms of Shock

	Cardiogenic Shock	Hemorrhagic Shock	Septic Shock
CV Origin	Cardiac	Volume	Vascular
Cardiac Output	↓	↓	↑↓
Vascular Resistance	↑	↑	↓
Blood Volume	↑	↓	↓
Management	Mechanical Inotropes Vasopressors Vasodilators	IV Fluids/Blood Vasopressors	IV Fluids Antibiotics Vasopressors Inotropes

Hypovolemic Shock

Aims:

- Describe ***how acute blood loss*** leads to hypotension.
- Describe the ***compensatory mechanisms*** that operate to restore arterial pressure following hemorrhage.
- Describe the ***decompensatory mechanisms*** that lead to ***irreversible shock***.
- Describe the ***rationale for different medical interventions*** following hemorrhage.

General Definition of Hemorrhagic Shock:

- A clinical syndrome resulting from decreased blood and oxygen perfusion of vital organs resulting from a loss of blood volume.

Hypovolemic Shock

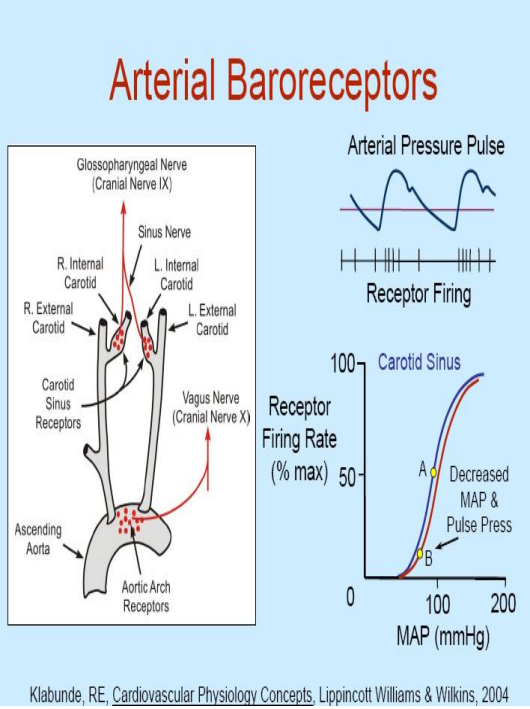
- Hemorrhagic/Traumatic
- Dehydrative
- Burn

Compensatory Mechanisms

- ***Baroreceptor*** reflexes
- Circulating ***vasoconstrictors***
- ***Chemoreceptor*** reflexes
- ***Reabsorption*** of tissue fluids
- ***Renal reabsorption*** of ***sodium and water***
- Activation of ***thirst mechanisms***
- ***Cerebral ischemia***
- ***Hemopoiesis (included in the metabolic response to trauma)***

Cardiopulmonary Baroreceptors

- **Location**: Venoatrial Junction
  Tonically active
- Receptor firing decreases **ADH** (vasopressin) release
  leading to diuresis and vasodilation
- **Hemorrhage** → **increase ADH** (reduced urine formation
  and increased vasoconstriction)
- Location: Atria and Ventricles
  Tonically active
- affect vagal and sympathetic outflow similar to arterial baroreceptors
- reinforce arterial baroreceptor responses during hypovolemia



Klabunde, RE, Cardiovascular Physiology Concepts, Lippincott Williams & Wilkins, 2004

Autonomic Responses to Baroreceptor Activity

- **Normally : Arterial baroreceptor firing inhibits sympathetic outflow and stimulates parasympathetic outflow**

- Therefore, **reduced firing, which occurs during hemorrhage,** leads to **sympathetic activation and parasympathetic inhibition**

Redistribution of cardiac output

– **Intense vasoconstriction** in skin, skeletal muscle, renal (during severe hemorrhage) and splanchnic circulations increases systemic vascular resistance, which attenuates the fall in arterial pressure

– **Coronary and cerebral circulations *spared***
Therefore, cardiac output is shunted to essential organs

• **Redistribution of blood volume**

– Strong venoconstriction in GI, hepatic and skin circulations

– Partial restoration of central venous blood volume and pressure to counteract loss of filling pressure to the heart

- Baroreceptor Reflexes Cont.

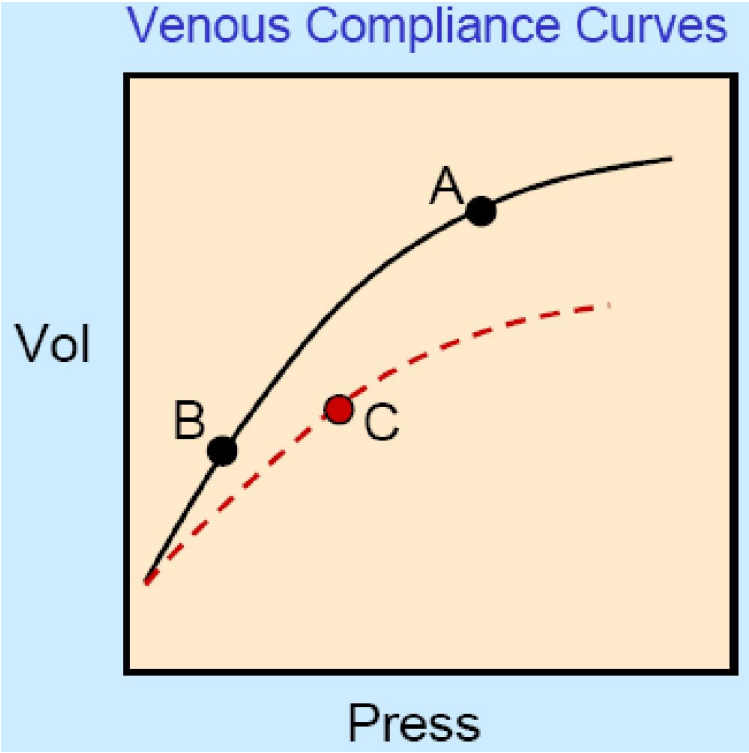
Central Venous Pressure (CVP) During Hge

• Hemorrhage decreases **blood volume and decreases CVP (A→B)**

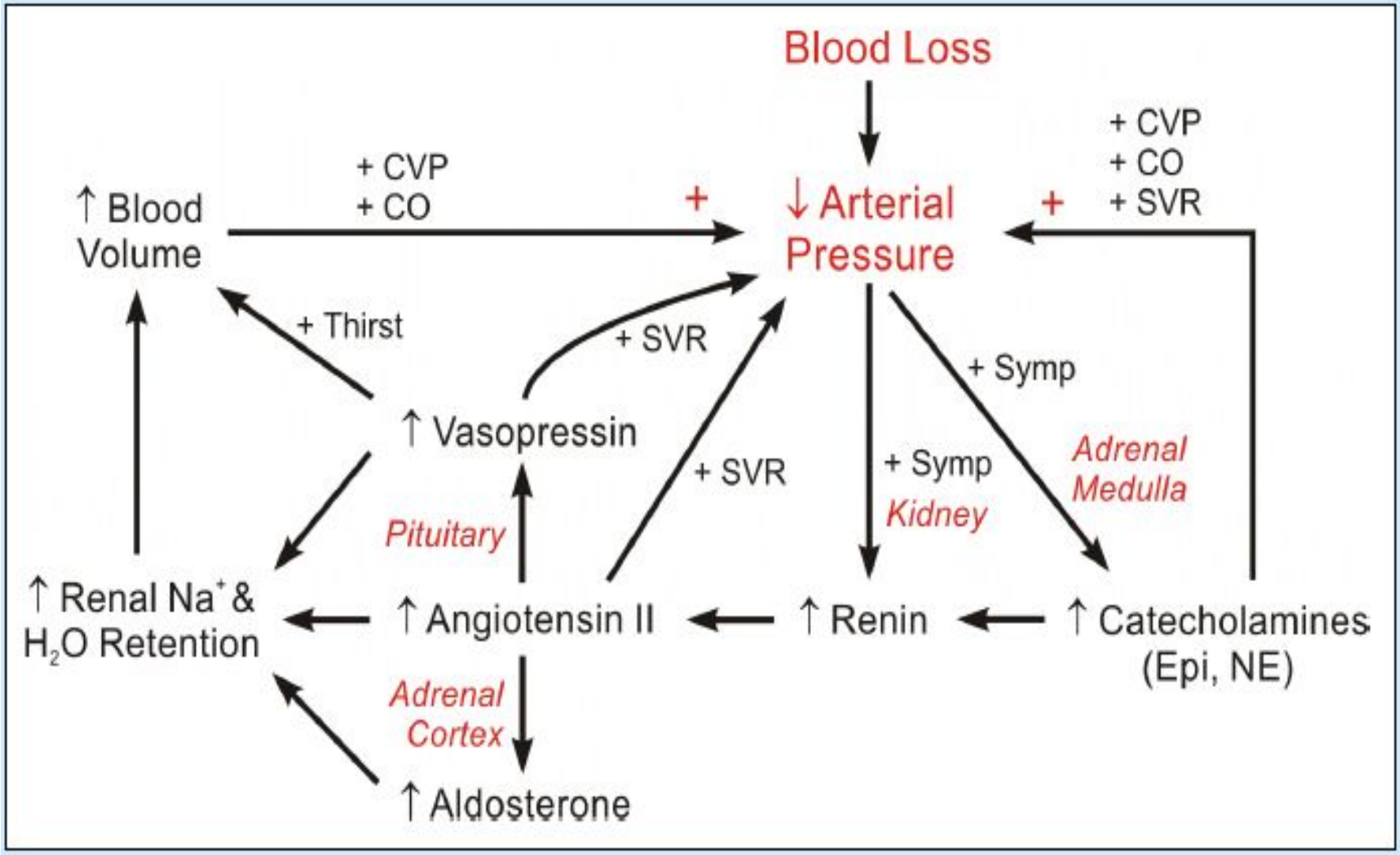
• Peripheral venous constriction decreases **venous compliance (B→C),**

*this increases CVP and shifts blood volume towards the **heart***

Increased CVP increases ventricular **preload and **force of contraction** (**Frank-Starling mechanism**)*



Humoral Compensatory Mechanisms



Importance of Humoral Compensatory Mechanisms

- **Angiotensin II, vasopressin** and **catecholamines** reinforce ***sympathetic mediated vasoconstriction*** to help maintain arterial pressure by:
  - increasing systemic vascular resistance
  - decreasing venous compliance, which increases ventricular preload and enhances stroke volume
- **Renal role:** Angiotensin II, aldosterone and vasopressin act on the kidneys to increase blood volume

Chemoreceptor Reflexes:

- **Peripheral** chemoreceptors:
  - **Carotid** bodies
  - **Aortic** bodies
- **Central** chemoreceptors:
  - **Medulla** (associated with ***Cardiovascular control "centers")***

Chemoreceptor Reflexes (cont.)

- when mean arterial pressure (MBP) falls below 60 mmHg (i.e., when arterial baroreceptor firing rate is at minimum)

- Acidosis resulting from decreased organ perfusion stimulates central and peripheral chemoreceptors → sympathetic activation

- Stagnant hypoxia in carotid bodies enhances peripheral vasoconstriction

- Respiratory stimulation may enhance venous return (VR); (abdominothoracic pump)

Reabsorption of Tissue Fluids:

- **Capillary pressure falls:**
  - Reduced arterial and venous pressures
  - Increased precapillary resistance
  - **Transcapillary fluid reabsorption (up to 1 liter/hr autoinfused)**

- **Capillary plasma oncotic pressure can fall from 25 to 15 mmHg due to autoinfusion there by limiting capillary fluid reabsorption**

- **Hemodilution causes hematocrit to fall which decreases blood viscosity**

Cerebral Ischemia:

- When mean arterial pressure falls **below 60** mmHg, **cerebral perfusion decreases**

  because the pressure is below the autoregulatory range

- Cerebral ischemia produces very intense

  sympathetic discharge that is several-fold

  greater than the maximal sympatheticactivation caused by the baroreceptor reflex

Decompensate Mechanisms "Progressive Shock"

- Cardiogenic Shock:
  - Impaired coronary perfusion causing myocardial hypoxia, systolic and diastolic dysfunction, arrhythmias

- Sympathetic Escape:
  - Loss of vascular tone (↓SVR) causing progressive hypotension and organ hypoperfusion
  - Increased capillary pressure causing increased fluid filtration and hypovolemia

- Cerebral Ischemia
  - Loss of autonomic outflow due to severe cerebral hypoxia

"Progressive Shock",(cont.)

- Metabolic Acidosis:

– Increased microvascular viscosity
– Microvascular plugging by leukocytes and platelets
– Intravascular coagulation

- Systemic Inflammatory Response

– Endotoxin release into systemic circulation
– Cytokine formation – TNF, IL, etc.
– Enhanced nitric oxide formation
– Reactive oxygen-induced cellular damage
– Increased capillary permeability
– Multiple organ failure

Mediators of Shock

- **Toxins**
  - Endotoxins
- **Oligo- and polypeptides**
  - Complement Factors
  - Opiods
  - TNF, Interleukins
- **Fatty Acid Derivatives**
  - Arachidonic acid metabolites
- **Variables:**
  - Calcium

Management of Shock

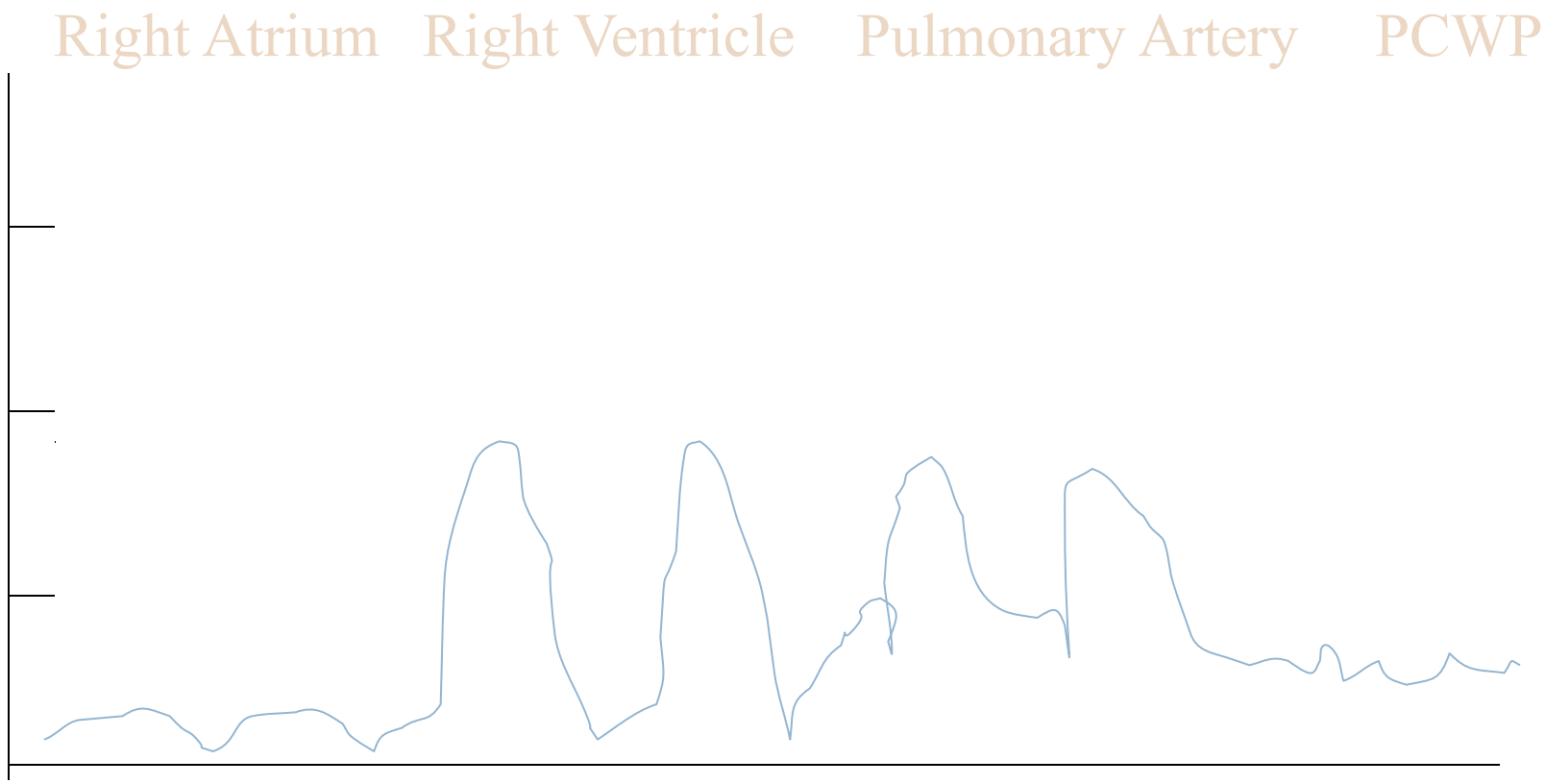
- Shock begins when DO2 to the cells is inadequate to meet metabolic demand
- The major therapeutic **goals** in shock therefore are sufficient tissue perfusion and oxygenation
- Early diagnosis remains a major problem

PULMONARY ARTERY CATHETERIZATION

Allows accurate and continuous hemodynamic monitoring in shocked patients:

1. Evaluate Fluid Resuscitation
2. Titration of Vasoactive Medications
3. Allows for Assessment of Cardiovascular Performance.
4. Monitor the Effects of Changes in Mechanical Ventilation.

Pulmonary Artery Catheter Waveforms



Classes of Hemorrhagic Shock

(according to amount of blood loss)

.Class I hemorrhage (loss of 0-15%)
- Little tachycardia
- Usually no significant change in BP, pulse pressure,or respiratory rate

• Class II hemorrhage (loss of 15-30%)
- HR >100 beats per minute, tachypnea, decreased pulse pressure

• Class III hemorrhage (loss of 30-40%)
- Marked tachycardia and tachypnea, decreased systolic, BP, oliguria

• Class IV hemorrhage (loss of >40%)
- Marked tachycardia and decreased systolic BP, narrowed pulse pressure, markedly decreased (or no) urinary output
- Immediately life threatening

Resuscitation Issues

- **Reducing reperfusion injury & systemic inflammatory response syndrome (SIRS):**
  - Anti-inflammatory drugs
  - NO scavenging and antioxidant drugs
- **Resuscitation fluids:**
  - **Crystalloid** vs. non-crystalloid solutions
  - I**sotonic** vs. hypertonic solutions
  - **Whole blood** vs. packed red cells
  - **Hemoglobin-based solutions**
  - **Perfluorocarbon**-based solutions
  - **Fluid volume-related issues**

Resuscitation Issues cont.

- Efficacy of pressor agents
- Hypothermic vs. normothermic resuscitation
- Tailoring therapy to conditions of shock
  - Uncontrolled vs. controlled hemorrhage
  - Traumatic vs. atraumatic shock

Septic Shock

Definitions: SIRS, sepsis, shock, MODS

- Morbidity/mortality of Sepsis/Shock
- Pathogenesis of septic shock
- Microbial triggers (endotoxin,…)
- Cytokine and non-cytokine mediators of SIRS and shock
- Pathophysiology of shock
- Therapy

Systemic Inflammatory Response Syndrome (SIRS)

- Systemic inflammatory response to a variety of severe clinical insults manifested by ≥ 2 of the following conditions:
  - Temperature >38°C or <36°C
  - Heart rate ≥90 beats/min
  - Respiratory rate ≥20 breaths/min or PaCO2 ,<32 torr (<4.3 kPa)
  - White blood cell count ≥12,000 cells/mm3, ≤4000 cells/mm3, or >10% immature (band) cells

Sepsis:

- **The presence of SIRS associated with a confirmed infectious process.**

Severe Sepsis:

- **Sepsis with either hypotension or systemic manifestations of hypoperfusion,**
  Lactic acidosis, oliguria, altered mental status

Septic Shock:

- **Sepsis with hypotension despite adequate fluid resuscitation, associated with hypoperfusion abnormalities**

Multiple Organ Dysfunction Syndrome (MODS)

- **Progressive distant organ failure (initially uninvolved) following severe infectious or noninfectious insults (severe burn, multiple trauma, shock, acute pancreatitis)**

Pathogenesis of Shock

Infectious or noninfectious triggers

↓

Cytokine and inflammatory mediator cascade

↓

Cardiac dysfunction and microvascular injury

↓

Hypotension and shock

Some Characteristics of Septic Shock

- Systemic vasodilation and hypotension
- Tachycardia; depressed contractility
- Vascular leakage and edema; hypovolemia
- Compromised nutrient blood flow to organs
- Disseminated intravascular coagulation (DIC)
- Abnormal blood gases and acidosis
- Respiratory distress and multiple organ failure (MOF)

Cardiogenic Shock

- Characterized by high preload (CVP) with low CO
- Signs: Dyspnea, rales, loud P2 gallop, low BP, oliguria
- Monitor/findings: CXR pulm venous congestion, elevated
- CVP, Low CO.
- TRT: CHF– diuretics & vasodilators +/- pressors.
  LV failure – pressors, decrease afterload,
  intraaortic ballon pump &
  ventricular assist device.

Anaphylactic Shock

- **A type of distributive shock that results from widespread systemic allergic reaction to an antigen**
- **This hypersensitive reaction is LIFE THREATENING**

Pathophysiology Anaphylactic Shock

- **Antigen exposure**
- **body stimulated to produce IgE antibodies specific to antigen:**
  - *drugs, bites, contrast, blood, foods, vaccines*
- **Reexposure to antigen**
  - *IgE binds to mast cells and basophils*
- **Anaphylactic response**

Anaphylactic Response

- Vasodilatation
- Increased vascular permeability
- Bronchoconstriction
- Increased mucus production
- Increased inflammatory mediators recruitment to sites of antigen interaction

Clinical Presentation Anaphylactic Shock:

Almost immediate response to inciting antigen

- ***Cutaneous manifestations***
  - ***urticaria, erythema, pruritis, angioedema***
- ***Respiratory compromise***
  - ***stridor, wheezing, bronchorrhea, resp. distress***
- ***Circulatory collapse***
  - ***tachycardia, vasodilation, hypotension***

Management Anaphylactic Shock

- *Early Recognition, treat aggressively*
- **AIRWAY SUPPORT**
- **IV *EPINEPHRINE* (open airways), NE**
- *Antihistamines, diphenhydramine 50 mg IV*
- *Corticosteroids*
- **IMMEDIATE WITHDRAWAL OF ANTIGEN IF POSSIBLE**
- **PREVENTION**

Management Anaphylactic Shock (cont.)

- **Judicious crystalloid administration**
- **Vasopressors to maintain organ perfusion**
- **Positive inotropes**
- **Patient education**

NEUROGENIC SHOCK

NEUROGENIC SHOCK

- **Definition:**

  A type of distributive shock that results from the loss or suppression of sympathetic tone

- **Haemodynamics: massive vasodilatation in the venous vasculature leading to :**

  - **venous return to heart,**
  - **cardiac output.**

- **Most common cause : *Spinal cord injury above T6***
- **Neurogenic origin is the rarest form of shock!**

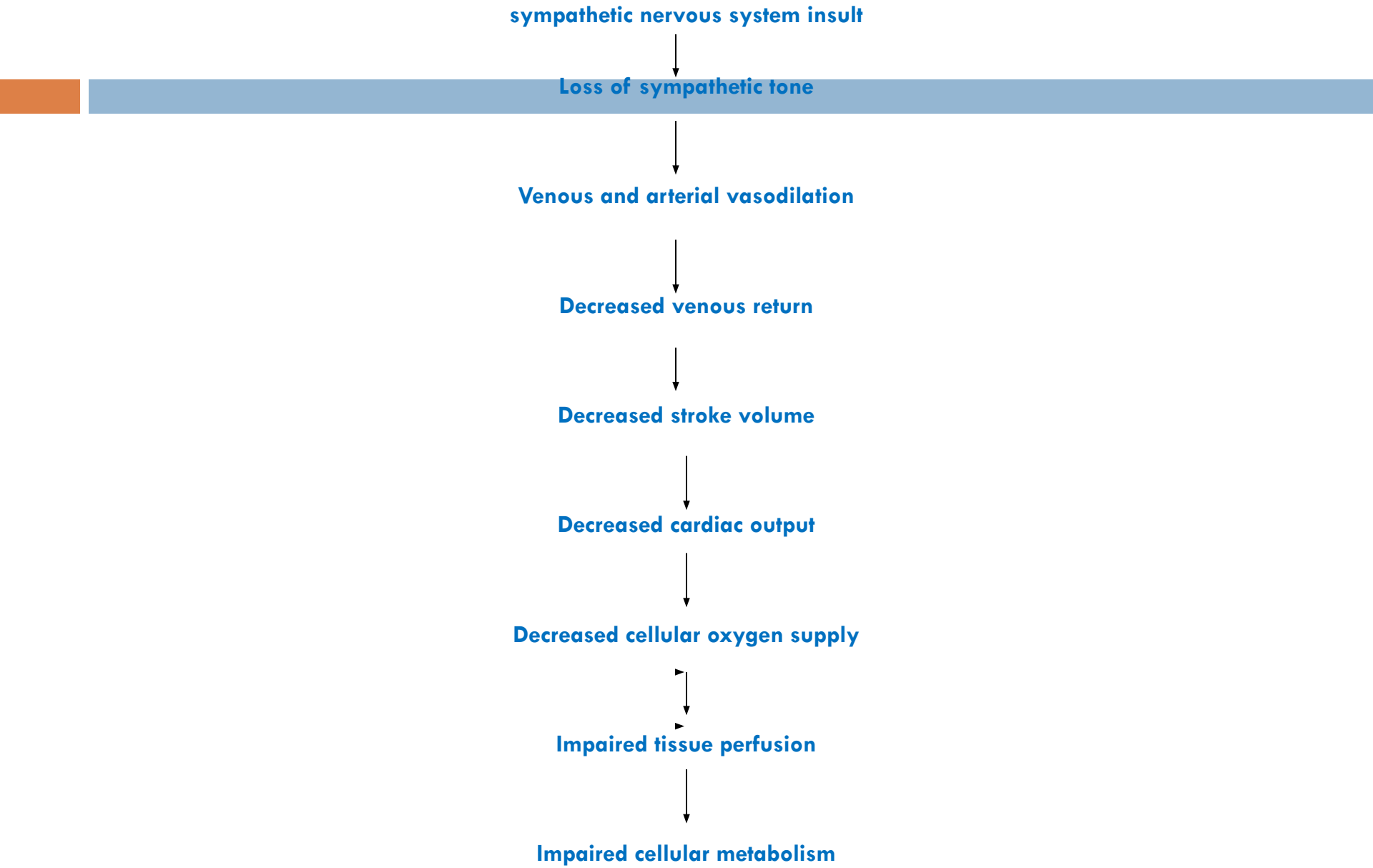
NEUROGENIC SHOCK: (CONT.)

<u>Mechanism:</u> Loss of autonomic innervation of the cardiovascular system (arterioles, venules, small veins, including the heart)

Causes:

1. **Spinal cord injury**
2. **Regional anesthesia**
3. **Drugs**
4. **Neurological disorders**

Pathophysiology of Neurogenic Shock



Assessment, Diagnosis and Management of Neurogenic Shock

PATIENT ASSESSMENT

- Hypotension
- Bradycardia
- Hypothermia
- Warm, dry skin
- RAP □
- PAWP □
- CO □
- Flaccid paralysis below level of the spinal lesion

MEDICAL MANAGEMENT

- Goals of Therapy are to treat or remove the cause & prevent cardiovascular instability, & promote optimal tissue perfusion

MANAGEMENT OF NEUROGENIC SHOCK

- Hypovolemia- tx with careful fluid replacement for BP<90mmHg, UOP<30cc/hr
- Observe closely for fluid overload
- Vasopressors may be needed
- - Hypothermia- warming txs
- - avoid large swings in pts body temperature
- Treat Hypoxia

- Maintain ventilatory support

Management Neurogenic Shock(cont,)

- **Alpha agonist** to augment tone if perfusion still inadequate

  - **Dopamine at alpha doses (> 10 mcg/kg per min)**

  - **Ephedrine (12.5-25 mg IV every 3-4 hour)**

  Treat bradycardia with atropine 0.5-1 mg doses to maximum 3 mg

  - **may need transcutaneous or transvenous pacing temporarily**