CARDIOVASCULAR PHYSIOLOGY

SHOCK

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LECTURE OUTCOMES

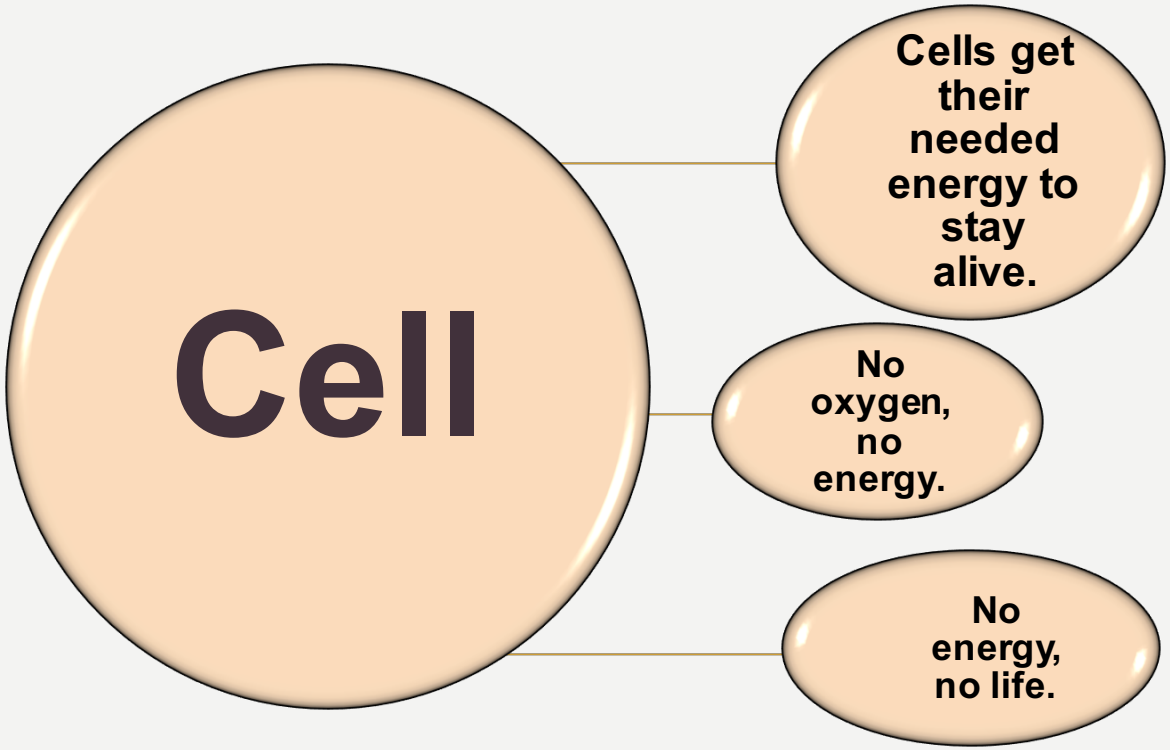
Define circulatory shock.

Types & causes of shock.

Body compensatory mechanisms during reversible phases of hemorrhagic shock.

Mechanisms responsible for the irreversible phase of hemorrhagic shock.

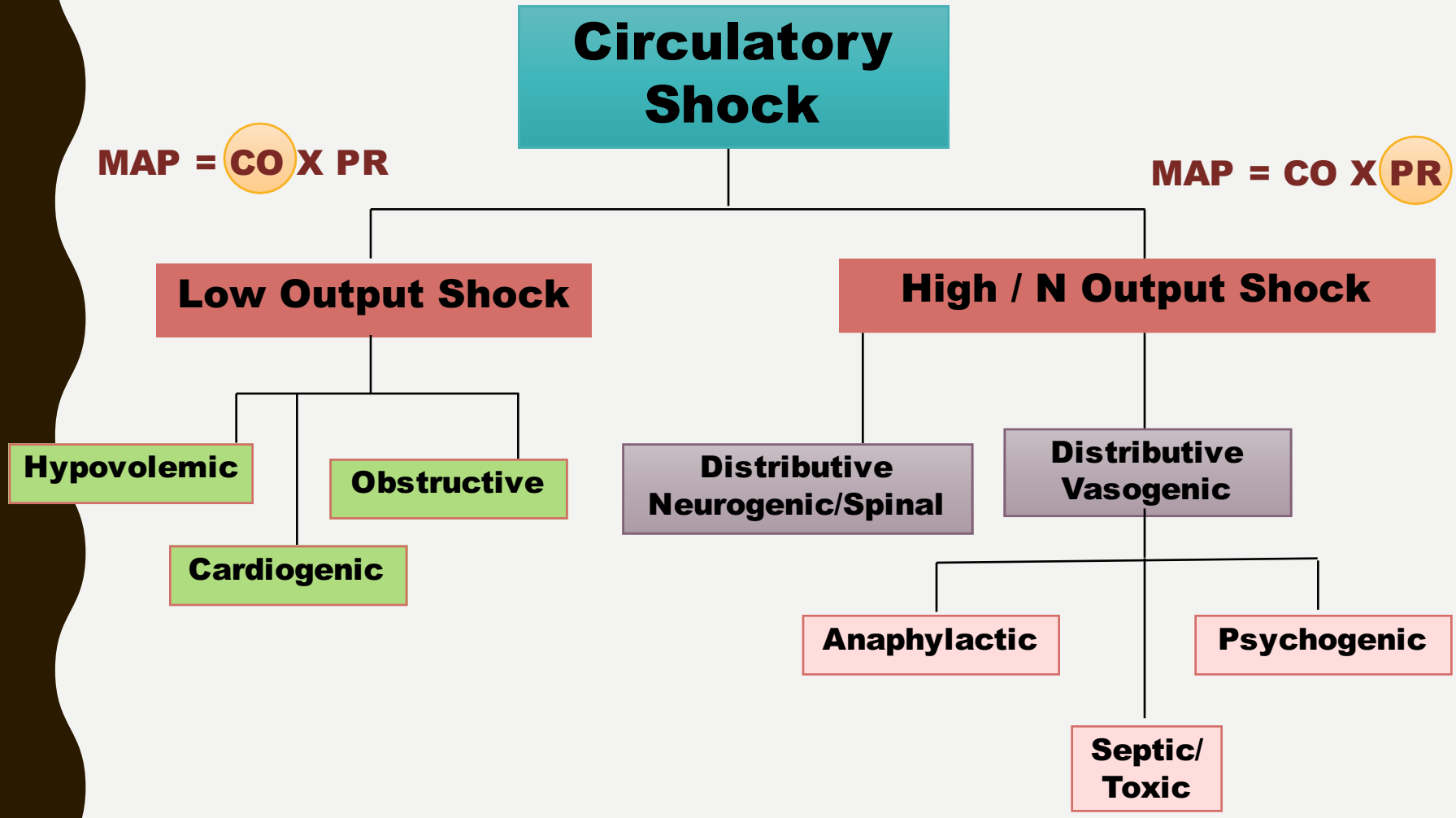
BASIC UNIT OF LIFE



WHAT IS SHOCK ?

- Any condition in which the circulatory system is unable to provide adequate circulation & tissue perfusion, resulting in failure to deliver oxygen to the tissues & vital body organs relative to its metabolic requirement.
- Defined as **Circulatory Shock**.
- Results in organ dysfunction & cellular damage.
- If not quickly corrected, it may lead to irreversible shock & death.

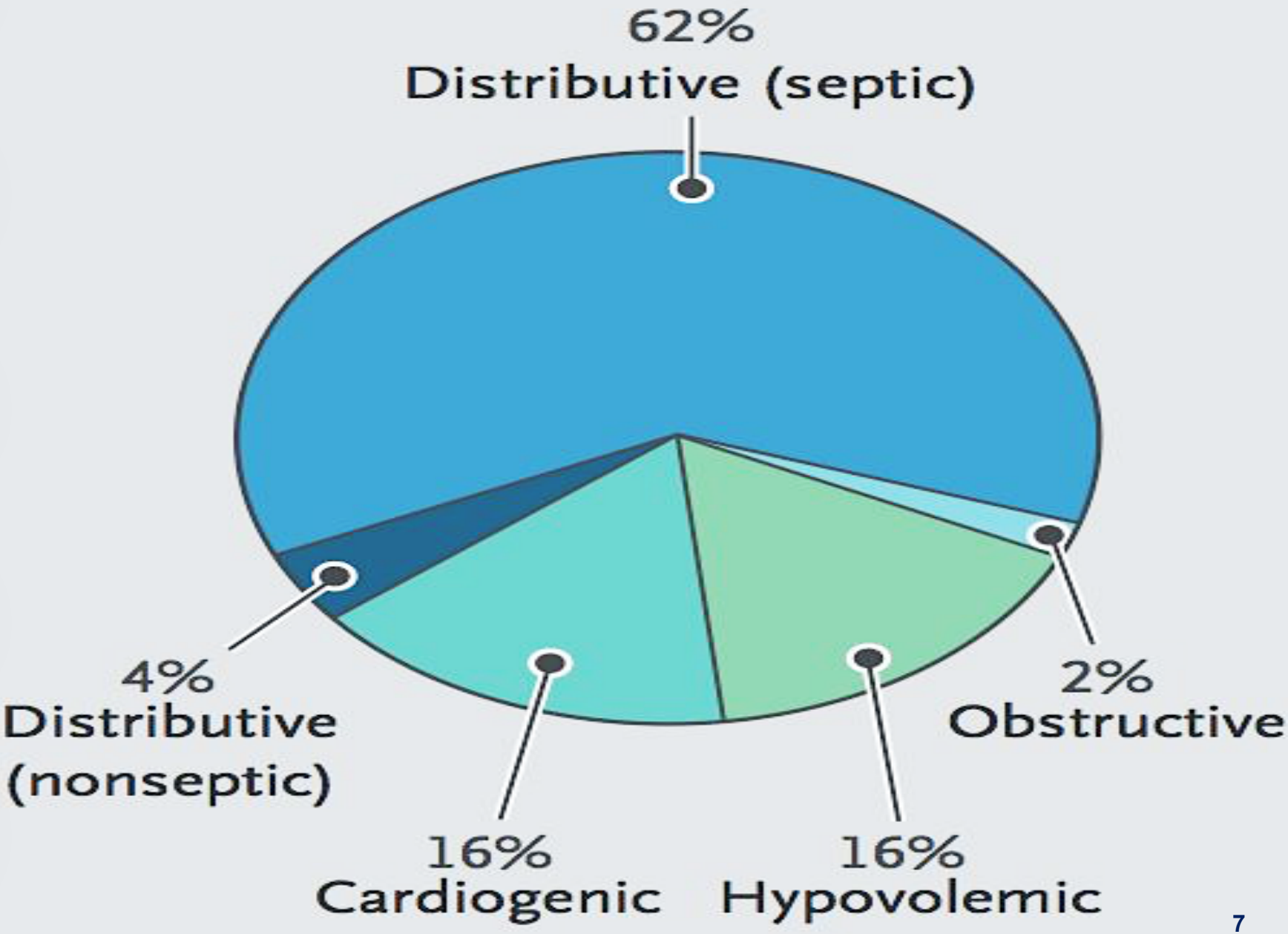
TYPES OF CIRCULATORY SHOCK



Types of Shock

- Hypovolemic – most common
  - Hemorrhagic, occult fluid loss
- Cardiogenic
  - Ischemia, arrhythmia, valvular, myocardial depression
- Distributive
  - Anaphylaxis, sepsis, neurogenic
- Obstructive
  - Tension pneumo, pericardial tamponade, PE

B Types of shock



HYPOVOLEMIC SHOCK

- **Low CO due to:**

MAP = CO X PR

  - Inadequate blood/plasma volume (loss of 15-25% / 1-2 L).
  - Reduced venous return (preload.).

- **Causes:**

  - Blood loss: Hemorrhage.. internal or external. (commonest.)
  - Fluid/plasma loss: Vomiting, diarrhea, burn, excess sweating, dehydration, trauma.

Hypovolemic shock

- Loss of blood (hemorrhagic shock)
  - External hemorrhage
    - Trauma
    - Gastrointestinal tract bleeding
  - Internal hemorrhage
    - Hematoma
    - Hemothorax or hemoperitoneum
- Loss of plasma
  - Burns
  - Exfoliative dermatitis
- Loss of fluid and electrolytes
  - External
    - Vomiting
    - Diarrhea
    - Excessive sweating
    - Hyperosmolar states (diabetic ketoacidosis, hyperosmolar nonketotic coma)
  - Internal ("third-spacing")
    - Pancreatitis
    - Ascites
    - Bowel obstruction

CARDIOGENIC SHOCK

- **Low CO due to:** **MAP = CO X PR**
  - Failure of myocardial pump, despite adequate ventricular filling pressure.
- **Causes:**
  - Myocardial Infarction.. (Most common.)
  - Myocarditis.
  - Cardiomyopathy.
  - Cardiac tamponade.
  - Acute valvular dysfunction, e.g. rupture of papillary muscle post MI.
  - Congestive heart failure.
  - Sustained Arrhythmias, e.g. heart block, ventricular tachycardia.
  - Pulmonary embolism.
- Is associated with loss of > 40% of LV myocardial function.
- Mortality rate is high (60-90%).

OBSTRUCTIVE SHOCK

- **CO is reduced by vascular obstruction:**
  - **Obstruction of venous return:**
    - e.g. Vena Cava Syndrome (usually neoplasms).
  - **Compression of the heart:**
    - e.g. hemorrhagic pericarditis → cardiac tamponade.
  - **Obstruction of the outflow of the heart:**
    - Aortic dissection.
    - Massive pulmonary embolism.
    - Pneumothorax.

MAP = CO X PR

DISTRIBUTIVE SHOCK: HIGH/ NORMAL OUTPUT

- CO is normal or elevated.
- Distribution is inappropriate.
- Shock is due to loss of vascular resistance.

MAP = CO X PR

DISTRIBUTIVE SHOCK: HIGH/ NORMAL OUTPUT

- **Septic/ Toxic/ Endotoxic Shock:** **MAP = CO X PR**
  - Bacterial endotoxin triggers peripheral vasodilatation & endothelial injury.
  - Hyperdynamic state.




- **Anaphylactic shock:**
  - Massive & generalized allergic reaction.
  - IgE- mediated hypersensitivity.
  - Histamine triggers peripheral vasodilation & ↑ capillary permeability.
  - Can lead to low output distributive shock.
- **Psychogenic shock:**
  - Simple fainting (syncope.)
  - Caused by stress, pain, or fright.
  - ↓ HR & vessels dilate.
  - Brain becomes hypoperfused.
  - Loss of consciousness.

NEUROGENIC/ SPINAL SHOCK .. (VENOUS POOLING)

- **Circulatory failure:**
  - Loss or drop in vasomotor (vascular) tone/ spinal cord injury.
  - Generalized peripheral vasodilation.
  - Blood volume remains normal.
  - CO is severely reduced as blood is pooled in peripheral veins.. (Capacity of blood ↑, & venous return ↓.)
  - Behaves like hypovolemic shock.

PATHOPHYSIOLOGY OF SHOCK

- Reduce capillary perfusion.
- Inadequate tissue oxygen.
- Shift to anaerobic metabolism.
- Metabolic acidosis.
- Release of free radicals & oxidative stress.
- Tissue damage.
- Apoptosis.

METABOLIC CHANGES & CELLULAR RESPONSE TO SHOCK

1. Spasm of pre/post capillary sphincters:

→ **reduced capillary perfusion.**

→ hypoxic tissue damage, (oxidative stress.)

→ anaerobic metabolism (anaerobic glycolysis.)

→ lactic acid production.

→ **metabolic acidosis (intracellular acidosis)**.

→ **Failure of Na+/K+ pump** (inc [Na+] & [C++]).

→ **Lysosomes, nuclear membranes & mitochondrial breakdown**.

METABOLIC CHANGES & CELLULAR RESPONSE TO SHOCK

2. After 3 - 5 hrs of shock → precapillary sphincters dilate (venules are still constricted) → blood stagnation in capillaries → hypoxia continue + fluid leaves to extra vascular compartment → further reduction in circulating blood volume.

3. Granulocytes accumulation at injured vessels → **free radicals release** → further tissue damage.

METABOLIC CHANGES & CELLULAR RESPONSE TO SHOCK

4. Damage in **GIT mucosa** → allows bacteria into circulation.
5. **Cerebral ischemia** → depression of VMC → vasodilation + ↓ HR

(vasomotor center .. sympathetic)

further decrease in blood pressure.

6. **Myocardial ischemia** → depressed contractility + myocardial damage

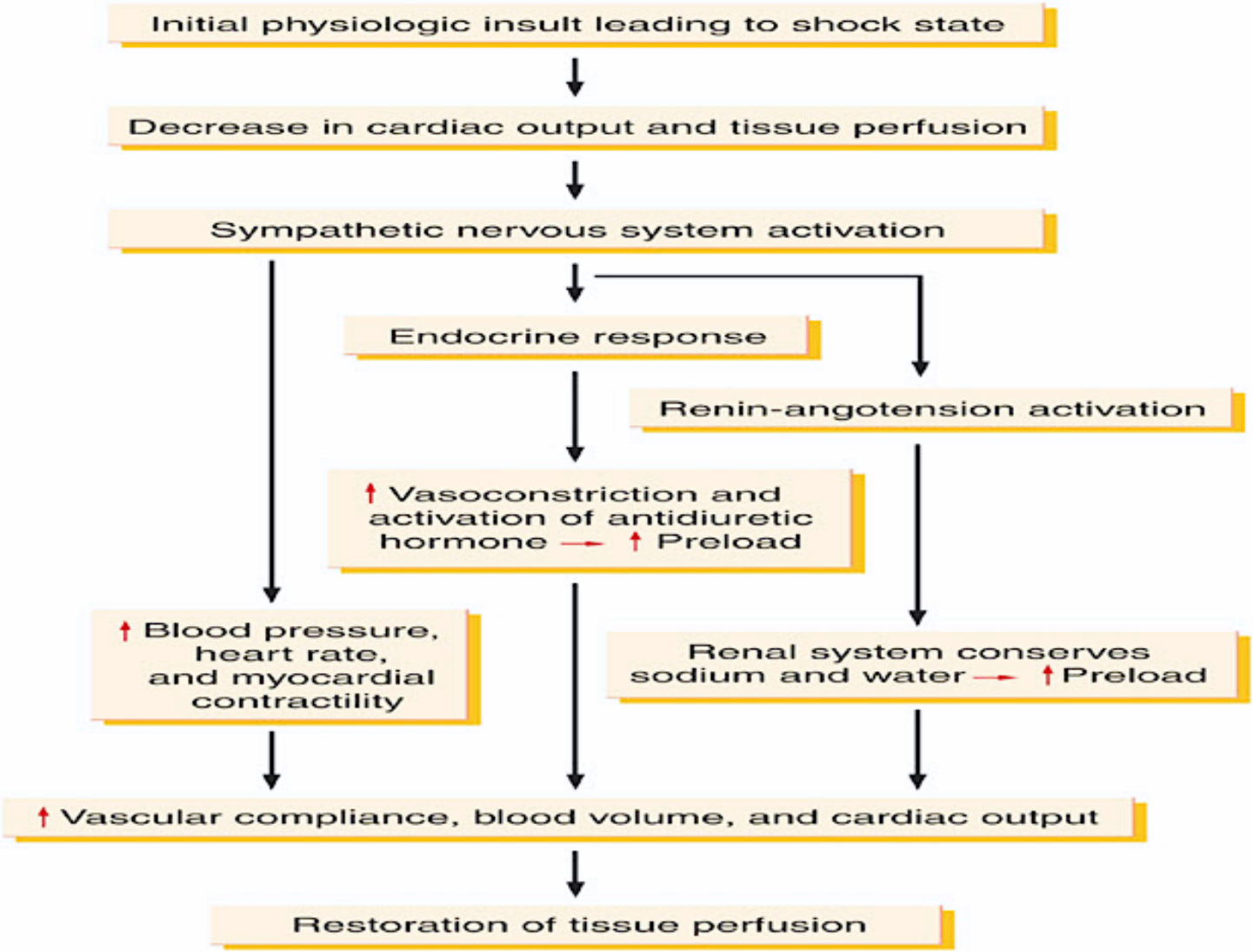
more shock & acidosis.

7. **Respiratory distress** syndrome occurs due to damage of capillary endothelial cells & alveolar epithelial cells, with release of cytokines.
8. Multiple organ failure & death.

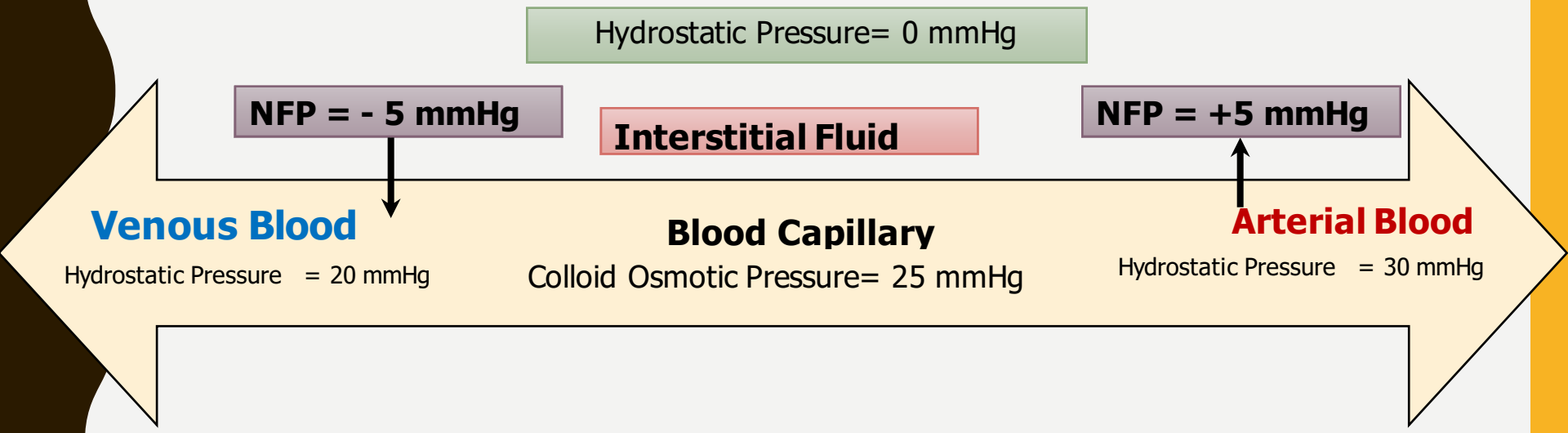
COMPENSATORY MECHANISMS

- ↓ BP stimulates **baroreceptors reflex** → sympathetic stimulation.
- Acidosis stimulates **chemoreceptors reflex** → sympathetic stimulation.
- **Renin-Angiotensin Mechanism:**
  - Angiotensin II & III: powerful vasoconstrictors.
  - Aldosterone: Na+ & water retention.
- **ADH (vasopressin):**
  - Water retention, vasoconstriction & thirst stimulation.
- Plasma proteins synthesis.
- Fluid- shift mechanism.

Compensatory Mechanisms



IN NORMAL MICROCIRCULATION



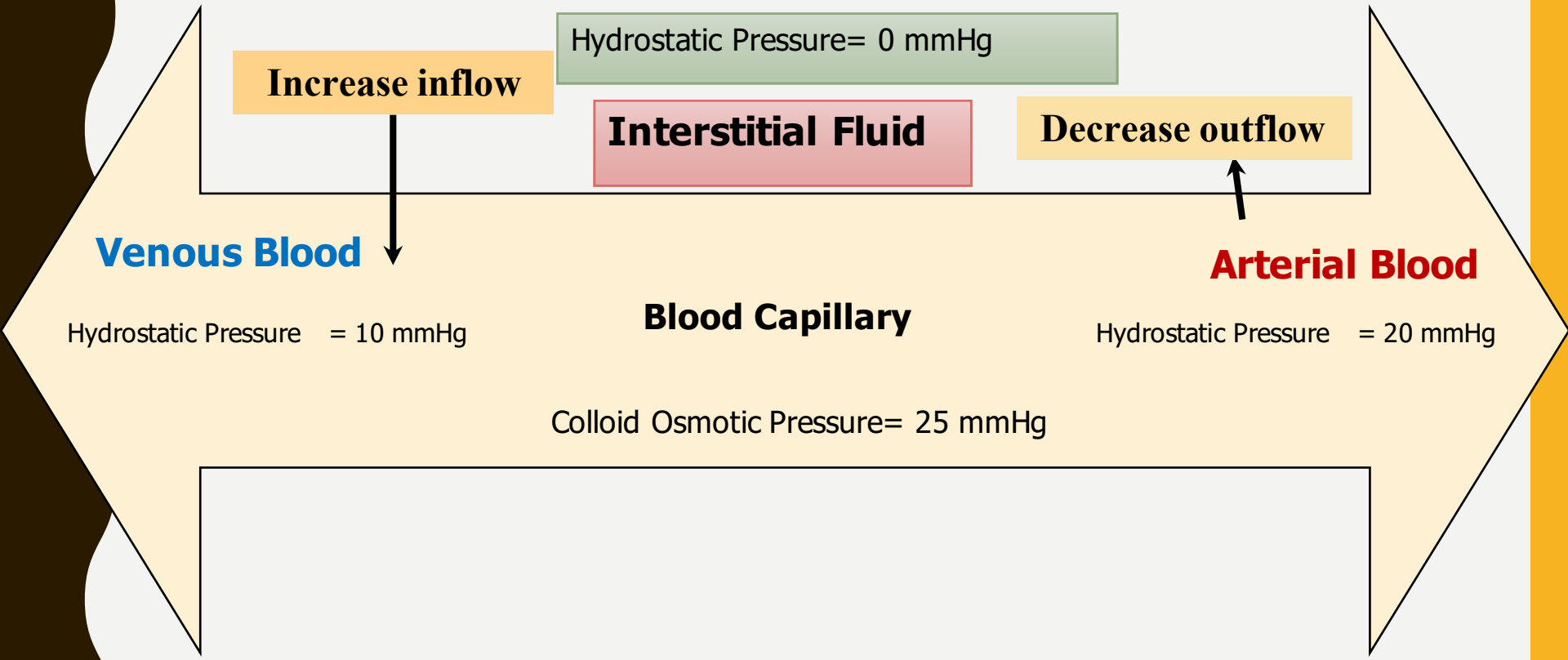
- **At arterial end:**
  - Water moves **out** of the capillary with a NFP of +5 mmHg.
  - Hydrostatic pressure dominates at the arterial end & net fluid flows out of the circulation.
- **At venous end:**
  - Water moves into the capillary with a NFP of -5 mmHg.
  - Oncotic pressure dominates at the venous end & net fluid will flow into the bloodstream.

FLUID- SHIFT MECHANISM IN SHOCK

- In shock, the hydrostatic pressure decreases & oncotic pressure is constant, as a result:
  - The fluid exchange from the capillary to the extracellular space decreases.
  - The fluid return from the extracellular space to the capillary increases.

That will increase the blood volume & will increase BP helping to compensate shock situations.

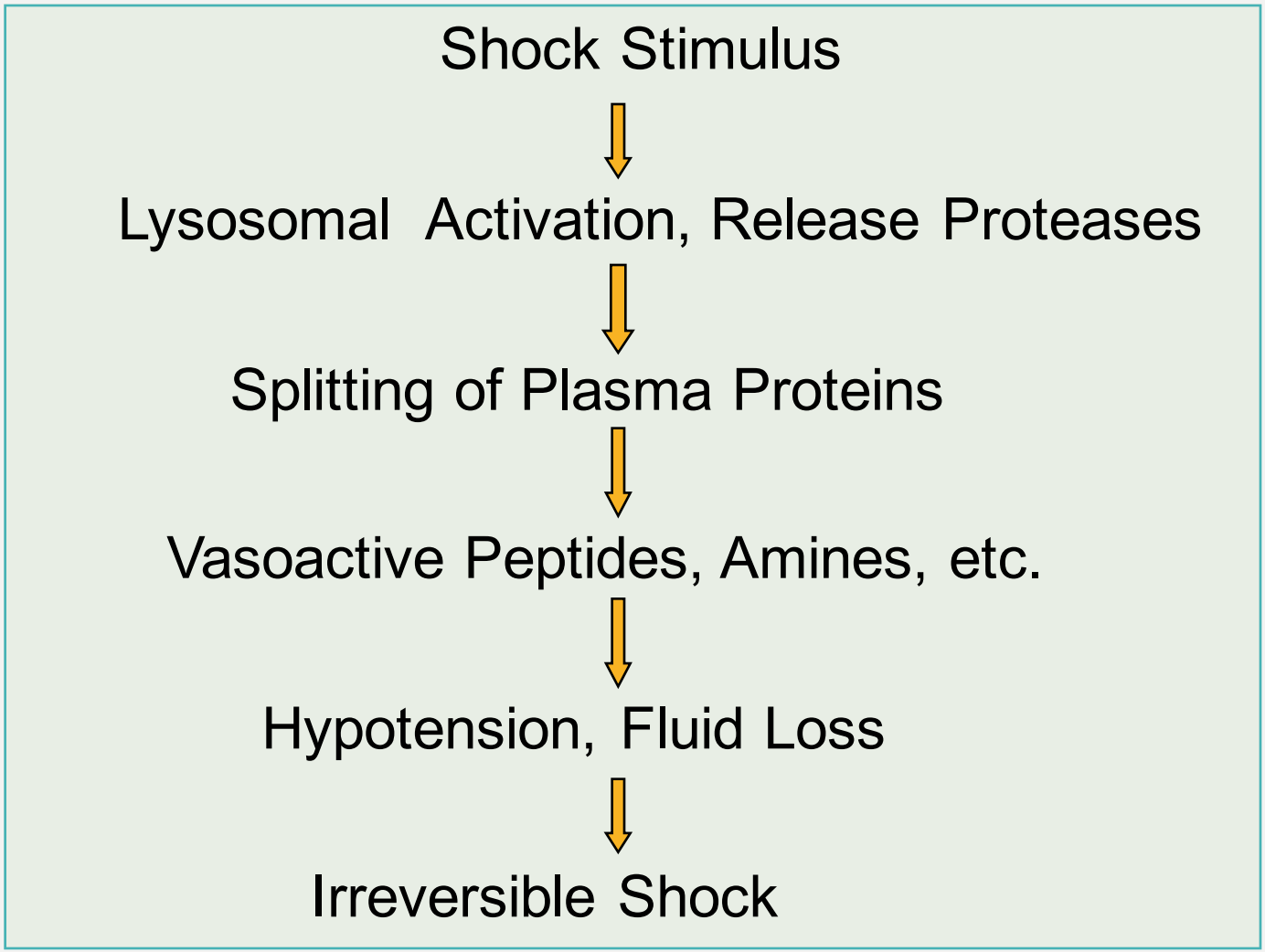
Fluid – Shift Mechanism In Shock



STAGES OF SHOCK

- **Reversible shock: (Compensated)**
  - Changes can be reversed by compensatory mechanism (neurohormonal activation) or by treatment.
  - Defense mechanisms are successful in maintaining perfusion.
  - Non-progressive.
- **Progressive:**
  - Defense mechanisms begin to fall.
  - Multi-organ failure.
- **Irreversible shock:**
  - Complete failure of compensatory mechanisms.
  - Can lead to death.

POSSIBLE MECHANISM IN DEVELOPMENT IRREVERSIBLE SHOCK



SIGNS/SYMPTOMS: HYPOVOLEMIC SHOCK

- Hypotension… (? ≤ 85/40 mmHg)
- Tachycardia… Compensation for ↓ MAP sensed by Baroreceptors.
- Rapid, weak, & thready pulse… (? 140/min).
- Intense thirst.
- Tachypnea (rapid respiration)… Compensation for hypoxia sensed by Chemoreceptors.
- Restlessness… due to hypo-perfusion.
- Cold, pale skin… due to hypo-perfusion.
- Oliguria (low urine output)/ Anuria (no urine output).
- Blood test: Lactic acidosis.

SIGNS/SYMPTOMS: CARDIOGENIC SHOCK

- Similar signs & symptoms to that of hypovolemic shock.
- Congestion of lungs & viscera: (CXR)
  - Interstitial pulmonary oedema.
  - Alveolar edema.
  - Cardiomegaly.

SIGNS/SYMPTOMS: SEPTIC SHOCK

- Patient flushed & warm due to his hyperdynamic state.

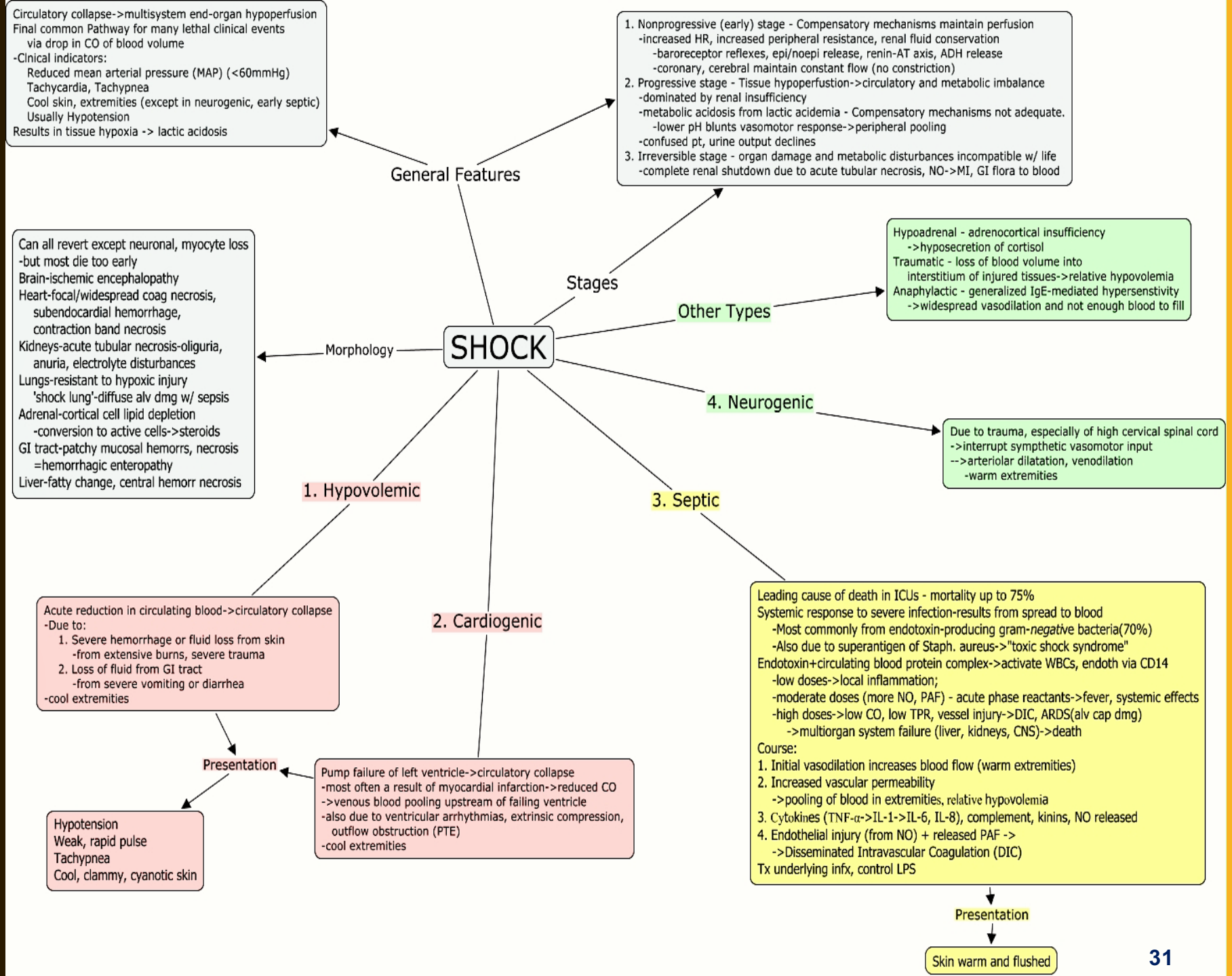
To Summarize

Type of Shock	Insult	Physiologic Effect	Compensation
Cardiogenic	Heart fails to pump blood out	↓CO	BaroRc ↑SVR
Obstructive	Heart pumps well, but the outflow is obstructed	↓CO	BaroRc ↑SVR
Hemorrhagic	Heart pumps well, but not enough blood volume to pump	↓CO	BaroRc ↑SVR
Distributive	Heart pumps well, but there is peripheral vasodilation	↓SVR	↑CO

Hemodynamics of Shock

Red arrow indicates primary abnormality	PCWP (preload)	Cardiac Output	SVR (afterload)	Treatment
Hypovolemic shock	↓	↑	↑	IV fluids
Cardiogenic shock	↑	↓	↑	Inotropes Revascularization
Distributive shock (septic, neurogenic)	↓	↑	↓	Pressors IV fluids

PCWP = pulmonary capillary wedge pressure SVR = systemic vascular resistance


Circulatory collapse->multisystem end-organ hypoperfusion
Final common Pathway for many lethal clinical events
via drop in CO of blood volume
-Clnical indicators:
Reduced mean arterial pressure (MAP) (<60mmHg)
Tachycardia, Tachypnea
Cool skin, extremities (except in neurogenic, early septic)
Usually Hypotension
Results in tissue hypoxia -> lactic acidosis
General Features
1. Nonprogressive (early) stage - Compensatory mechanisms maintain perfusion
-increased HR, increased peripheral resistance, renal fluid conservation
-baroreceptor reflexes, epi/noepi release, renin-AT axis, ADH release
-coronary, cerebral maintain constant flow (no constriction)
2. Progressive stage - Tissue hypoperfustion->circulatory and metabolic imbalance
-dominated by renal insufficiency
-metabolic acidosis from lactic acidemia - Compensatory mechanisms not adequate.
-lower pH blunts vasomotor response->peripheral pooling
-confused pt, urine output declines
3. Irreversible stage - organ damage and metabolic disturbances incompatible w/ life
-complete renal shutdown due to acute tubular necrosis, NO->MI, GI flora to blood
Stages
Can all revert except neuronal, myocyte loss
-but most die too early
Brain-ischemic encephalopathy
Heart-focal/widespread coag necrosis,
subendocardial hemorrhage,
contraction band necrosis
Kidneys-acute tubular necrosis-oliguria,
anuria, electrolyte disturbances
Lungs-resistant to hypoxic injury
'shock lung'-diffuse alv dmg w/ sepsis
Adrenal-cortical cell lipid depletion
-conversion to active cells->steroids
GI tract-patchy mucosal hemorrs, necrosis
=hemorrhagic enteropathy
Liver-fatty change, central hemorr necrosis
Morphology
SHOCK
Hypoadrenal - adrenocortical insufficiency
->hyposecretion of cortisol
Traumatic - loss of blood volume into
interstitium of injured tissues->relative hypovolemia
Anaphylactic - generalized IgE-mediated hypersenstivity
->widespread vasodilation and not enough blood to fill
Other Types
4. Neurogenic
Due to trauma, especially of high cervical spinal cord
->interrupt sympthetic vasomotor input
-->arteriolar dilatation, venodilation
-warm extremities
1. Hypovolemic
Acute reduction in circulating blood->circulatory collapse
-Due to:
1. Severe hemorrhage or fluid loss from skin
-from extensive burns, severe trauma
2. Loss of fluid from GI tract
-from severe vomiting or diarrhea
-cool extremities
Presentation
Hypotension
Weak, rapid pulse
Tachypnea
Cool, clammy, cyanotic skin
2. Cardiogenic
Pump failure of left ventricle->circulatory collapse
-most often a result of myocardial infarction->reduced CO
->venous blood pooling upstream of failing ventricle
-also due to ventricular arrhythmias, extrinsic compression,
outflow obstruction (PTE)
-cool extremities
3. Septic
Leading cause of death in ICUs - mortality up to 75%
Systemic response to severe infection-results from spread to blood
-Most commonly from endotoxin-producing gram-negative bacteria(70%)
-Also due to superantigen of Staph. aureus->"toxic shock syndrome"
Endotoxin+circulating blood protein complex->activate WBCs, endoth via CD14
-low doses->local inflammation;
-moderate doses (more NO, PAF) - acute phase reactants->fever, systemic effects
-high doses->low CO, low TPR, vessel injury->DIC, ARDS(alv cap dmg)
->multiorgan system failure (liver, kidneys, CNS)->death
Course:
1. Initial vasodilation increases blood flow (warm extremities)
2. Increased vascular permeability
->pooling of blood in extremities, relative hypovolemia
3. Cytokines (TNF-α->IL-1->IL-6, IL-8), complement, kinins, NO released
4. Endothelial injury (from NO) + released PAF ->
->Disseminated Intravascular Coagulation (DIC)
Tx underlying infx, control LPS
Presentation
Skin warm and flushed


Thank You