

SHOCK

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13-12-2023.

SHOCK

- ▶ Shock is a state in which diminished cardiac output or reduced effective circulating blood volume impairs tissue perfusion and leads to cellular hypoxia

Three Major Types of Shock

- ▶ 1. Cardiogenic shock :
 - results from low cardiac output as a result of myocardial pump failure.
- ▶ It may be caused by:
 - ▶ myocardial damage (infarction).
 - ▶ ventricular arrhythmias.
 - ▶ extrinsic compression (cardiac tamponade).
 - ▶ outflow obstruction (e.g., pulmonary embolism).



cardiac tamponade: fluid or blood builds up between the heart and the pericardium.



▶ 2. Hypovolemic shock:

- results from low cardiac output due to loss of blood or plasma volume (e.g., resulting from hemorrhage or fluid loss from severe burns).

▶ 3. Neurogenic shock:

- result from a loss of vascular tone.

▶ 4. Anaphylactic shock:

- results from systemic vasodilation and increased vascular permeability that is triggered by an immunoglobulin E-mediated hypersensitivity reaction.

5. Septic shock

➤ life-threatening organ dysfunction due to dysregulated host response to infection, and organ dysfunction.

▶ Caused by massive outpouring of inflammatory mediators from innate and adaptive immune cells that produce:

▶ arterial vasodilation.

▶ vascular leakage.

▶ venous blood pooling.

LEAD TO

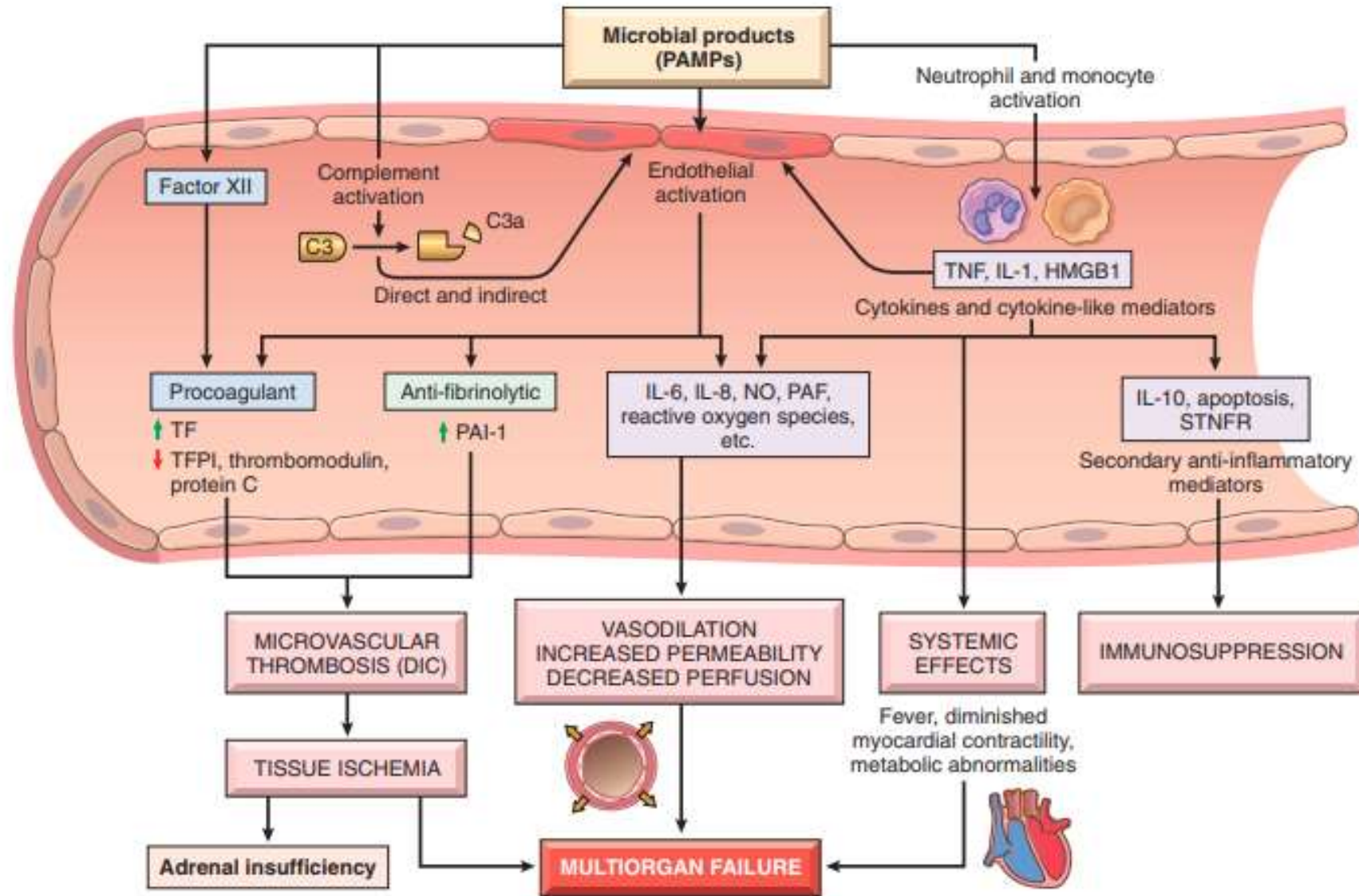
➤ tissue hypoperfusion

➤ cellular hypoxia

➤ metabolic derangements

Table 4.3 Three Major Types of Shock

Type of Shock	Clinical Examples	Principal Pathogenic Mechanisms
Cardiogenic	Myocardial infarction Ventricular rupture Arrhythmia Cardiac tamponade Pulmonary embolism	Failure of myocardial pump resulting from intrinsic myocardial damage, extrinsic pressure, or obstruction to outflow
Hypovolemic	Hemorrhage Fluid loss (e.g., vomiting, diarrhea, burns, trauma)	Inadequate blood or plasma volume
Septic	Overwhelming microbial infections Gram-negative sepsis Gram-positive septicemia Fungal sepsis Superantigens (e.g., toxic shock syndrome)	Peripheral vasodilation and pooling of blood; endothelial activation/injury; leukocyte-induced damage; disseminated intravascular coagulation; activation of cytokine cascades



Pathogenesis of Septic Shock

- ▶ 1. Inflammatory and counterinflammatory responses:
 - microbial cell wall constituents engage receptors , e.g: (TLRs).
 - innate immune cells produce numerous cytokines, including TNF, IL-1, IFN- γ .
 - the complement cascade is also activated by microbial components resulting in the production of anaphylotoxins (C3a, C5a).
 - microbial components can activate coagulation directly through factor XII and indirectly through altered endothelial function.

The hyperinflammatory state, triggers counterregulatory immunosuppressive mechanisms

- ▶ As a result, septic patients may range between hyperinflammatory and immunosuppressed states during their clinical course.
- mechanisms for the immune suppression include:
 - shift from proinflammatory (TH1) to anti-inflammatory (TH2).
 - production of anti-inflammatory mediators (e.g., soluble TNF receptor, IL-1 receptor antagonist, and IL-10).
 - lymphocyte apoptosis



▶ 2. • Endothelial activation and injury:

- Cytokines loosen endothelial cell tight junctions, making vessels leaky and resulting in the accumulation of protein-rich edema fluid throughout the body.
- Activated endothelium also upregulates production of nitric oxide (NO), C3a, C5a, and PAF, which may contribute to vascular smooth muscle relaxation and systemic hypotension

▶ 3. Induction of a procoagulant state:



▶ 4. Metabolic abnormalities:


- Septic patients exhibit insulin resistance and hyperglycemia.
- TNF and IL-1, glucagon, growth hormone, glucocorticoids, and catecholamines all drive gluconeogenesis.*

▶ 5. Organ dysfunction:

- ▶ Systemic hypotension, interstitial edema, and small vessel thrombosis all decrease the delivery of oxygen and nutrients to the tissues.

Stages of Shock

- ▶ 1. An initial nonprogressive stage:
 - during which reflex compensatory mechanisms are activated and vital organ perfusion is maintained.
- ▶ 2. A progressive stage :
 - characterized by tissue hypoperfusion and onset of worsening circulatory and metabolic derangement, including acidosis.
- ▶ 3. An irreversible stage :
 - in which cellular and tissue injury is so severe that even if the hemodynamic defects are corrected, survival is not possible.

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- ▶ The cellular and tissue effects of shock are:
 - those of hypoxic injury and are caused by a combination of hypoperfusion and microvascular thrombosis.

Clinical Features

- ▶ The primary threat to life is the underlying initiating event.
 - ▶ Hypotension.
 - ▶ a weak rapid pulse.
 - ▶ tachypnea,
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- ▶ If patients survive the initial period, worsening renal function can provoke a phase dominated by :
 - progressive oliguria.
 - Acidosis.
 - electrolyte imbalances