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# CORRECTION OF PATHOPHYSIOLOGICAL PROCESSES IN SEPSIS

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**Abstract.** This article discusses the pathophysiological mechanisms of sepsis and modern approaches to their correction. Sepsis is a life-threatening condition characterized by a dysregulated host response to infection, leading to systemic inflammation, endothelial dysfunction, microcirculatory failure, and multiple organ dysfunction syndrome (MODS). The study analyzes inflammatory cascades, cytokine storm, coagulation disorders, metabolic disturbances, and mitochondrial dysfunction. Special attention is given to therapeutic strategies aimed at correcting hemodynamic instability, restoring microcirculation, modulating immune response, and supporting organ function.

**Keywords:** sepsis, cytokine storm, microcirculation, MODS, inflammation, hemodynamic support, immune modulation.

**Introduction.** Sepsis is one of the leading causes of mortality in intensive care units worldwide. It develops when the body's response to infection becomes dysregulated, resulting in systemic inflammation and progressive organ dysfunction. Despite advances in intensive care medicine, sepsis remains a major clinical challenge due to its complex and multifactorial pathophysiology.

The pathological process in sepsis involves activation of immune cells, excessive release of pro-inflammatory cytokines, endothelial injury, coagulation abnormalities, and profound metabolic disturbances. Understanding these mechanisms is essential for effective correction and treatment strategies.

**Aim of the Study.** To analyze the pathophysiological mechanisms of sepsis and evaluate modern methods of correcting systemic dysfunctions.

**Objectives**

1. To investigate inflammatory and immune responses in sepsis.
2. To analyze microcirculatory and endothelial dysfunction.
3. To evaluate coagulation disorders in septic conditions.
4. To study metabolic and mitochondrial changes.
5. To assess modern therapeutic approaches for correction.

**Main Body. Inflammatory Response and Cytokine Storm.** Sepsis begins with the activation of the innate immune system in response to pathogens. This leads to excessive production of inflammatory mediators such as TNF-α, IL-1β, and IL-6, resulting in a "cytokine storm." This uncontrolled inflammatory response causes:

- endothelial damage
- capillary leakage
- tissue edema
- systemic hypotension

**Microcirculatory Dysfunction**

One of the key features of sepsis is microcirculatory failure. Endothelial injury leads to impaired vasoregulation and reduced tissue perfusion. O2 delivery↓⇒ tissue hypoxia↑O_2\

delivery \downarrow \Rightarrow \ tissue \ hypoxia \uparrowO2 delivery↓⇒ tissue hypoxia↑ This results in cellular hypoxia and organ dysfunction despite normal or increased cardiac output in early stages.

**Coagulation Disorders.** Sepsis induces activation of the coagulation cascade, leading to disseminated intravascular coagulation (DIC).

Key mechanisms include:

- increased tissue factor expression
- platelet activation
- suppression of fibrinolysis

This leads to microthrombi formation and impaired organ perfusion.

**Metabolic and Mitochondrial Dysfunction**

Sepsis is associated with cellular metabolic failure. Mitochondrial dysfunction reduces ATP production, shifting metabolism toward anaerobic glycolysis. ATP↓⇒anaerobic metabolism↑⇒lactate↑ATP \downarrow \Rightarrow anaerobic\ metabolism \uparrow \Rightarrow lactate \uparrowATP↓⇒anaerobic metabolism↑⇒lactate↑ Elevated lactate is an important marker of disease severity.

**Immune Dysregulation.** Sepsis is characterized by both hyperinflammation and immunosuppression. After the initial cytokine storm, patients may develop immune paralysis, increasing susceptibility to secondary infections.

**Correction of Pathophysiological Processes. Hemodynamic Support.** Early goal-directed therapy includes fluid resuscitation with crystalloids and vasopressors (e.g., norepinephrine) to maintain mean arterial pressure (MAP $\geq$ 65 mmHg).

**Antimicrobial Therapy.** Early administration of broad-spectrum antibiotics is critical to control infection and reduce inflammatory stimulus.

**Immunomodulatory Therapy.** Targeted therapies aim to regulate excessive inflammation, including:

- corticosteroids in selected cases
- cytokine adsorption techniques
- monoclonal antibodies (experimental approaches)

**Microcirculation Restoration**

Improving tissue perfusion is essential in sepsis management. This includes:

- fluid optimization
- vasopressor titration
- oxygen therapy
- inotropic support when needed

**Metabolic Support**

Correction of metabolic disturbances includes:

- glucose control
- lactate monitoring
- nutritional support
- mitochondrial protection strategies (experimental)

**Results.** Clinical analysis shows that early correction of hemodynamic and microcirculatory disturbances significantly reduces mortality in sepsis. Elevated lactate levels correlate with disease severity and poor prognosis. Timely antibiotic therapy and fluid resuscitation improve survival rates.

**Discussion.** Sepsis is a complex syndrome involving immune dysregulation, endothelial injury, and metabolic failure. Modern treatment strategies focus not only on infection control but also on restoring physiological homeostasis. Future approaches include personalized medicine, biomarker-guided therapy, and advanced extracorporeal blood purification techniques.

**Conclusion**

1. Sepsis is characterized by dysregulated systemic inflammation and multiorgan dysfunction.
2. Microcirculatory failure is a key determinant of tissue hypoxia.
3. Metabolic and mitochondrial dysfunction contribute to disease severity.
4. Early hemodynamic and antimicrobial therapy is essential.
5. Future treatment strategies focus on personalized and immunomodulatory approaches.

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