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Three heads of severe sepsis: hypotension, hypoperfusion and organ dysfunction

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Sepsis

- **Sepsis, severe sepsis, and septic shock are inflammatory states resulting from the systemic response to bacterial infection.**
- **In severe sepsis and septic shock, there is critical reduction in tissue perfusion.**

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review

- **Common causes include gram-negative organisms, staphylococci, and meningococci.**
- **Symptoms often begin with shaking chills and include fever, hypotension, oliguria, and confusion. Acute failure of multiple organs, including the lungs, kidneys, and liver, can occur.**

- Sepsis **is infection accompanied by an acute inflammatory reaction with systemic manifestations associated with release into the bloodstream of numerous endogenous mediators of inflammation. Acute pancreatitis and major trauma, including burns, may manifest with signs of sepsis. The inflammatory reaction typically manifests with ≥ 2 of the following:**

- **Temperature > 38°C or < 36°C**
- **Heart rate > 90 beats/min**
- **Respiratory rate > 20 breaths/min or $PaCO_2$ < 32 mm Hg**
- **WBC count > 12,000 cells/µL or < 4,000 cells/µL or > 10% immature forms**

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Symptoms and Signs

- With sepsis, the patient typically has fever, tachycardia, and tachypnea; BP remains normal.
- As severe sepsis or septic shock develops, the first sign may be confusion.
- BP generally falls, yet the skin is paradoxically warm.
- Oliguria (< 0.5 mL/kg/h) is likely to be present. Later, extremities become cool and pale, with peripheral cyanosis.
- Organ failure causes additional symptoms and signs specific to the organ involved.

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Diagnosis

- Appropriate cultures should be obtained from all potential sites of infection.
- In addition, a minimum of two blood cultures, of which at least one should be percutaneous and one from each vascular access site should be obtained.

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Diagnosis

- **Sepsis is suspected when a patient with a known infection develops systemic signs of inflammation or organ dysfunction.**
- **Similarly, a patient with otherwise unexplained signs of systemic inflammation should be evaluated for infection by history, physical examination, and tests, including urinalysis and urine culture (particularly in patients who have indwelling catheters), serial blood cultures, and cultures of other suspect body fluids.**
- **Blood levels of procalcitonin is elevated in severe sepsis and may facilitate diagnosis**

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Diagnosis

- Hyperventilation with respiratory alkalosis (low $PaCO_2$) occurs
- Serum HCO_3 is usually low, and serum and blood lactate levels increase.
- As shock progresses, metabolic acidosis worsens.
- Early respiratory failure leads to hypoxemia with $PaO_2 < 70$ mm Hg.
- Diffuse infiltrates may appear on the chest x-ray.
- Urea and creatinine usually increase progressively as a result of renal insufficiency.
- Bilirubin and transaminases may rise, although hepatic failure is uncommon.

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Severe sepsis

- **is sepsis accompanied by signs of failure of at least one organ.**
- **Cardiovascular failure is typically manifested by hypotension,**
- **respiratory failure by hypoxemia, renal failure by oliguria, and**
- **hematologic failure by coagulopathy.**

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Septic shock

- **is severe sepsis with organ hypoperfusion and hypotension that are poorly responsive to initial fluid resuscitation.**

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Septic shock Etiology

- **Most cases of septic shock are caused by hospital-acquired gram-negative bacilli or gram-positive cocci and often occur in immunocompromised patients and patients with chronic and debilitating diseases. Rarely, it is caused by *Candida* or other fungi.**
- **Form of shock caused by staphylococcal and streptococcal toxins is called toxic shock**

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Septic shock Pathophysiology

- An bacterial toxin triggers production of proinflammatory mediators, including tumor necrosis factor and IL-1.
- These cytokines cause neutrophil-endothelial cell adhesion, activate the clotting mechanism, and generate microthrombi.
- They also release numerous other mediators, including histamine, bradykinin, serotonin, and IL-2.
- They are opposed by anti-inflammatory mediators, such as IL-4 and IL-10

Septic shock Pathophysiology

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- **Initially, arteries and arterioles dilate, decreasing peripheral arterial resistance; cardiac output typically increases.**
- **This stage has been referred to as warm shock.**
- **Later, cardiac output may decrease, BP falls (with or without an increase in peripheral resistance), and typical features of shock appear.**

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Septic shock Pathophysiology

- **Even in the stage of increased cardiac output, vasoactive mediators cause blood flow to bypass capillary exchange vessels (a distributive defect).**
- **Poor capillary flow from this shunting along with capillary obstruction by microthrombi decreases delivery of O_2 and impairs removal of CO_2 and waste products.**

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Septic shock Pathophysiology

- **Decreased perfusion causes dysfunction and sometimes failure of one or more organs, including the kidneys, lungs, liver, brain, and heart.**
- **Coagulopathy may develop because of intravascular coagulation with consumption of major clotting factors, excessive fibrinolysis in reaction thereto, and more often a combination of both.**

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Diagnosis

- **Hemodynamic measurements with a central venous catheter can be used when the specific type of shock is unclear or when large fluid volumes are needed.**
- **Unlike in hypovolemic shock, cardiac output in septic shock is more likely to be normal or increased, and peripheral resistance is decreased. Central venous pressure is likely to be abnormal, unlike cardiogenic shock .**

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The following should be monitored frequently

- **systemic pressure;**
- **central venous pressure;**
- **pulse oximetry;**
- **blood glucose,**
- **electrolyte levels;**
- **renal function;**
- **PCO_2;**
- **Urine output, a good indicator of renal perfusion, should be measured, usually with an indwelling catheter.**

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Prognosis

- **Overall mortality in patients with septic shock is decreasing and now averages 40% (range 10 to 90%, depending on patient characteristics).**
- **Poor outcomes often follow failure to institute early aggressive therapy (eg, within 6 h of suspected diagnosis).**
- **Once severe lactic acidosis with decompensated metabolic acidosis becomes established, especially in conjunction with multiorgan failure, septic shock is likely to be irreversible and fatal.**

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Principles of Treatment

- Fluid resuscitation with 0.9% normal saline
- O_2
- Broad-spectrum antibiotics (modified by culture results)
- Drainage of abscesses and excision of necrotic tissue
- Normalization of blood glucose levels
- Replacement-dose corticosteroids

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Fluid resuscitation

- **Fluid resuscitation with 0.9% saline should be given until central venous pressure reaches** 10 cm H_2O
- **The quantity of fluid required often far exceeds the normal blood volume and may reach 10 L over 4 to 12 h.**
- **If a patient with septic shock remains hypotensive after central venous pressure has been raised to target levels, dopamine may be given to increase mean BP to at least 60 mm Hg. If dopamine dose exceeds 20 µg/kg/min, another vasopressor, typically norepinephrine, may be added.**

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antibiotics

- **Parenteral antibiotics should be given after specimens of blood, body fluids, and wound sites have been taken for Gram stain and culture.**
- **Empiric therapy, started immediately after suspecting sepsis, is essential and may be lifesaving.**
- **Antibiotic selection based on the suspected source, clinical setting, knowledge or suspicion of causative organisms.**
- **It is important to consider microorganisms susceptibility patterns in the community and hospital, which may vary from region to region.**

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antibiotics

- **One regimen for septic shock of unknown cause is 3rd-generation cephalosporin (ceftriaxone 2 g once/day or, if *Pseudomonas* is suspected, ceftazidime) plus a fluoroquinolone (eg, ciprofloxacin) may be used.**
- **Monotherapy with maximal therapeutic doses of ceftazidime or imipenem may be effective.**
- **Vancomycin must be added if resistant staphylococci or enterococci are suspected.**
- **If there is an abdominal source, a drug effective against anaerobes (eg, metronidazole) should be included.**
- **When culture and sensitivity results are available, the antibiotic regimen is changed accordingly.**
- **Antibiotics are continued for at least 5 days after shock resolves and evidence of infection subsides.**

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Surgical treatment

- **Abscesses must be drained, and necrotic tissues (eg, infarcted bowel, gangrenous gall-bladder) must be surgically excised.**
- **The patient's condition will continue to worsen despite antibiotic therapy unless septic foci are eliminated.**

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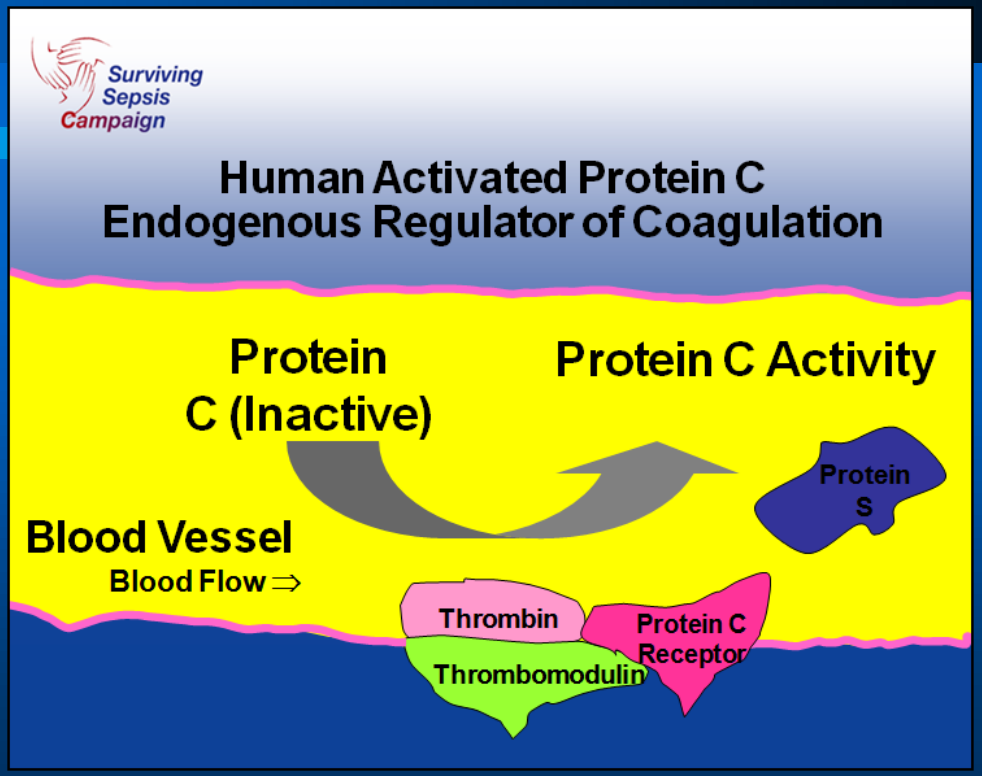
Activated protein C

- **Activated protein C, a recombinant drug with fibrinolytic and anti-inflammatory activity, seems beneficial for severe sepsis and septic shock if it is begun early.**

•Benefit has been shown only in patients with significant risk of death as defined by an APACHE II score of > 25



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Symptomatic therapy

- **Other therapies for severe sepsis include cooling for hyperthermia and early treatment of renal failure**