



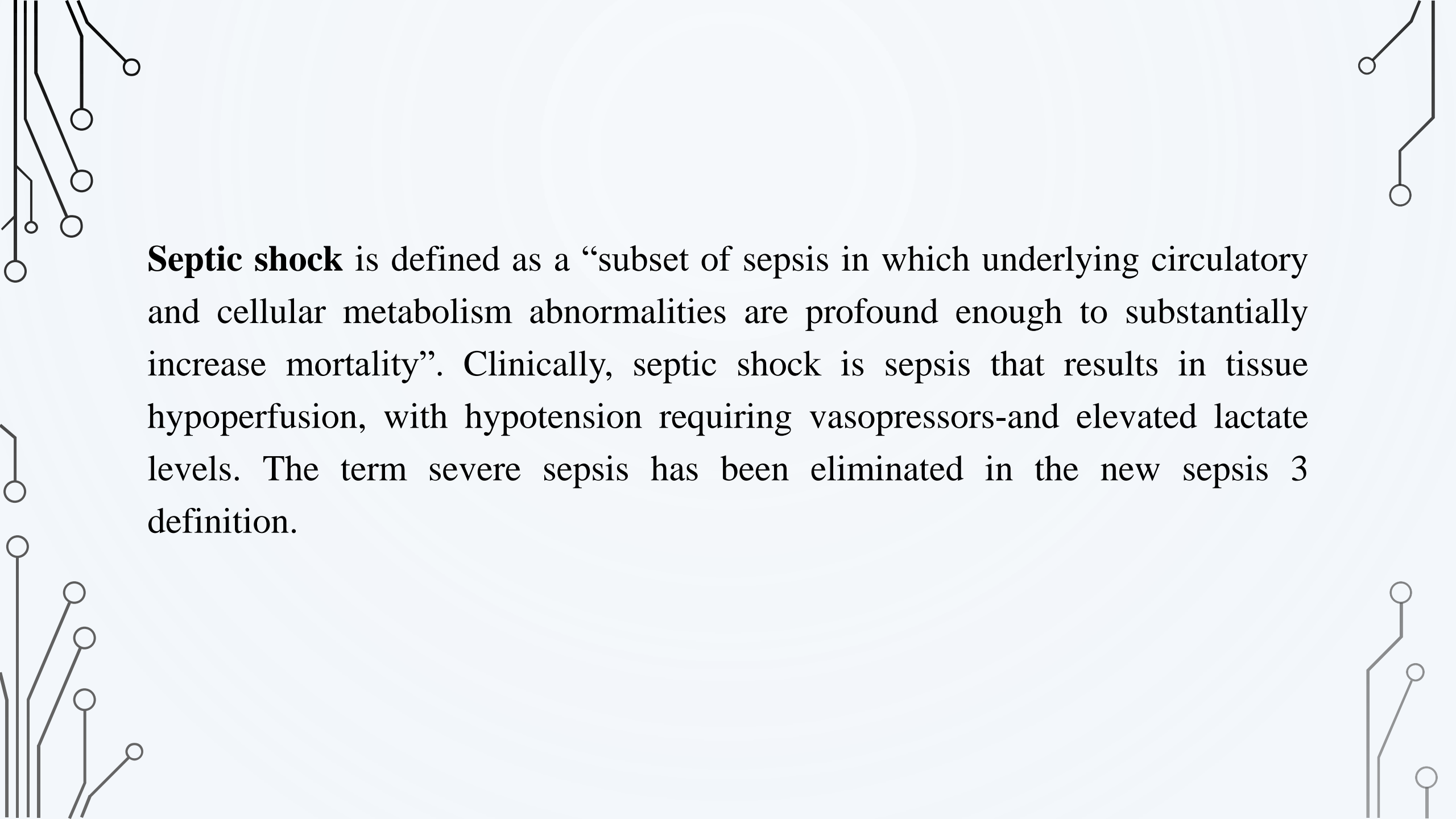
Septic shock

By
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Septic shock

A 60-year-old diabetic male patient presented with a history of dysuria and fever. His vital signs on admission were as follows: pulse 120/min, BP 80/50 mmHg, and respiratory rate 28/min. He was disoriented and agitated.

Sepsis is defined as “life-threatening organ dysfunction caused by a dysregulated host response to infection”. Organ dysfunction is assessed at the bedside by a change in SOFA score. Early identification of organ dysfunction with a quick SOFA score will help in early resuscitation with a resultant decrease in morbidity and mortality.

The image features a light blue background with a faint, large-scale circuit pattern. In the corners, there are more prominent, darker circuit-like line drawings. These lines are straight and angular, connecting small white circles that represent nodes or components of a network. The overall aesthetic is technical and modern.

Septic shock is defined as a “subset of sepsis in which underlying circulatory and cellular metabolism abnormalities are profound enough to substantially increase mortality”. Clinically, septic shock is sepsis that results in tissue hypoperfusion, with hypotension requiring vasopressors-and elevated lactate levels. The term severe sepsis has been eliminated in the new sepsis 3 definition.

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Step 1: take care of airway and breathing

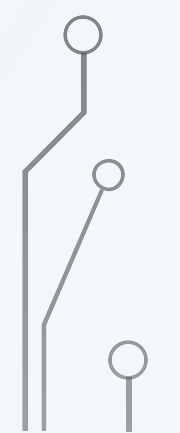

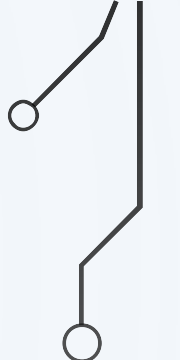
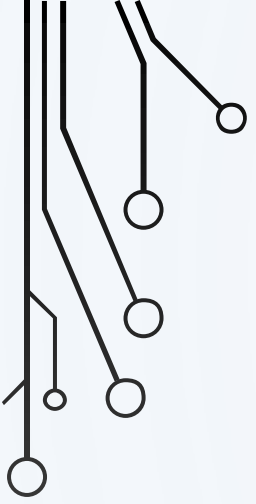
Proper airway care and, if needed, assisted ventilation should be promptly initiated in all patients with sepsis and shock. Taking early control of breathing decreases the oxygen consumption by the respiratory muscles and enables better perfusion of vital organs.

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Step 2: recognize sepsis and septic shock

Sepsis has a variable clinical presentation, depending on site of infection, causal organism, the predominant organ affected or the pattern of organ dysfunction, the presence of pre-existent chronic diseases and most importantly time interval before presentation. Since both infection and organ dysfunction can be subtle, a high index of suspicion is required to diagnose sepsis. SOFA scores and qSOFA are used to identify organ dysfunction, which should raise the suspicion of sepsis in patients with suspected infection (Table [52.1](#)).

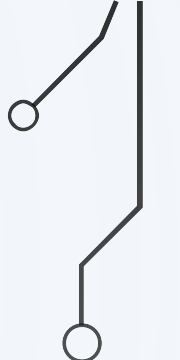
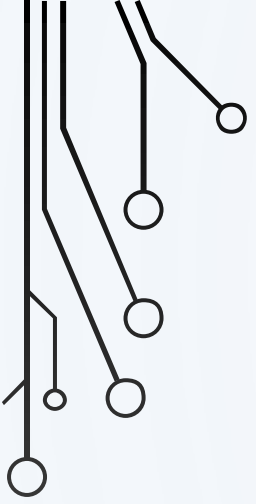
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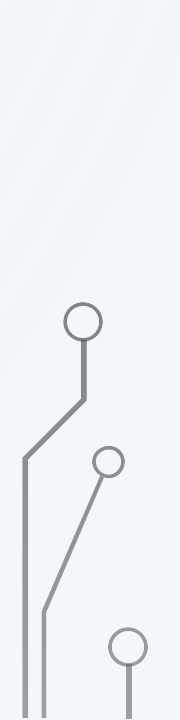

Since calculating SOFA requires blood tests, patients with suspected infection who are likely to have a prolonged ICU stay or to die in the hospital can be promptly identified at the bedside with qSOFA i.e. 2 or more of the following are present: Hypotension: SBP less than or equal to 100 mmHg, Altered mental status (any GCS less than 15) and Tachypnoea: RR greater than or equal to 22/mt.

Table 52.1 Sequential [Sepsis-Related] Organ Failure Assessment (SOFA) Score

Sequential [Sepsis-Related] Organ Failure Assessment Score					
System	Score				
	0	1	2	3	4
Respiration PaO ₂ /FiO ₂ , mm Hg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation platelets × 10 ³ /μL	≥150	<150	<100	<50	<20
Liver Bilirubin, mg/dL (μmol/L)	<1.2 (20)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	>12.0 (204)
Cardiovascular	MAP ≥70 mm Hg	MAP ≤70 mm Hg	Dopamine <5 or dobutamine (any dose) ^a	Dopamine 5.1–15 or epinephrine ≤0.1 or norepinephrine ≤0.1 ^a	Dopamine 15 or epinephrine >0.1 or norepinephrine >0.1 ^a
Central nervous system Glasgow Coma Scale score ^b	15	13–14	10–12	6–9	<6
Renal Creatinine, mg/dL (μmol/L)	<1.2 (110)	1.2–1.9 (110–170)	2.0–3.4 (171–299)	3.5–4.9 (300–400)	>5.0 (440)
Urine output, mL/ day				<500	<200



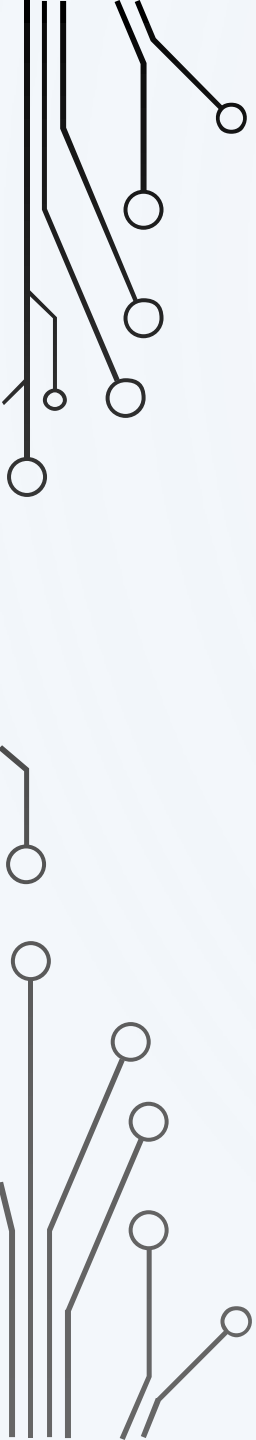
Sepsis can affect all organ systems acutely, especially the cardiovascular system and respiratory system manifesting as hypotension and acute respiratory distress syndrome (ARDS). Acute Kidney injury (AKI) manifests as oliguria and an increase in serum creatinine values. Obtundation and delirium are also very common CNS manifestations of sepsis. The other manifestations of sepsis include paralytic ileus, abnormal liver enzymes, hyperglycemia, thrombocytopenia and disseminated intravascular coagulation, adrenal dysfunction and the euthyroid sick syndrome.



Categorizing patients into sepsis and septic shock helps in triaging, prognostication, and choosing appropriate therapy.

Step 3: initial resuscitation

- Fluid resuscitation is of utmost importance in initial management of patients with sepsis induced tissue hypoperfusion and septic shock.
- Insert a wide-bore peripheral line and give initial fluid challenge of 1000 mL of crystalloids (normal saline or Ringer lactate) to achieve a minimum of 30 ml/kg of crystalloids over 3 hours with careful monitoring of vital signs. Following initial fluid resuscitation, further fluids should be guided by frequent reassessment of hemodynamic status.
- If type of shock is not clear, further hemodynamic monitoring is required.
- One can do initially echocardiography for assessment.

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- A decorative graphic consisting of thin black lines and small circles, resembling a circuit board or neural network, is positioned along the left and right edges of the slide.
- Fluid responsiveness should be assessed by using dynamic indices preferably over static indices. Dynamic variables have better diagnostic accuracy at predicting a response to a fluid challenge by increasing stroke volume. These techniques include passive leg raising while measuring stroke volume in a spontaneously breathing patient, or the variations in systolic pressure, pulse pressure, or stroke volume using heart lung interactions during mechanical ventilation.
 - Fluid responsiveness should always be assessed in conjunction with fluid tolerance. Fluid tolerance can be assessed clinically or using ultrasound of the lungs.
 - In septic shock requiring vasopressors, target means arterial pressure (MAP) of 65 mmHg.
 - Target Urine output of at least 0.5 ml/kg/h.

Fluid Responsiveness: dynamic tests



Not every patient with low blood pressure needs more fluids. Giving too much can cause harm (edema, lung injury). So we check if the heart will actually pump more blood (↑ stroke volume) after fluids. So instead of looking at *static numbers* (like CVP or a single BP reading), we use dynamic tests that show how the heart responds to changes in preload.

❖ Passive Leg Raising (PLR) Test (PT with spontaneous breathing)

- You lift the patient's legs (like giving a 300 ml “auto-fluid bolus”).
- If stroke volume or cardiac output increases → the patient is *fluid responsive*.

Example: A patient with septic shock → PLR test shows ↑ stroke volume by 15% → give IV fluids.

❖ Heart–Lung Interaction in Mechanical Ventilation

■ Inspiration (positive pressure):

❑ \uparrow Intrathoracic pressure \rightarrow \downarrow Venous return \rightarrow \downarrow RV preload

❑ After delay \rightarrow \downarrow LV filling \rightarrow \downarrow Stroke volume

■ Expiration (pressure release):

❑ \downarrow Intrathoracic pressure \rightarrow \uparrow Venous return \rightarrow \uparrow RV preload

❑ After delay \rightarrow \uparrow LV filling \rightarrow \uparrow Stroke volume

✓ Clinically  & 

Causes arterial line variations (PPV, SPV, SVV)

PPV > 12–13% \rightarrow fluid responsive

☞ Dynamic indices from heart–lung interaction are **more accurate than CVP** for predicting fluid responsiveness.

Formula for PPV (%):

$$PPV = \frac{PP_{max} - PP_{min}}{\left(\frac{PP_{max} + PP_{min}}{2}\right)} \times 100$$

Formula for SVV (%):

$$SVV = \frac{SV_{max} - SV_{min}}{\left(\frac{SV_{max} + SV_{min}}{2}\right)} \times 100$$

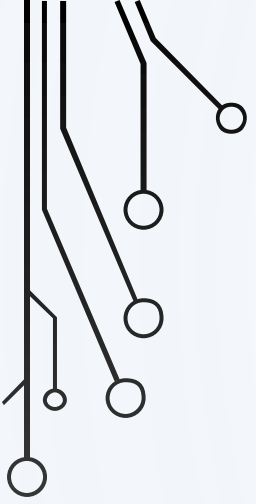
Where:

- PP_{max} and PP_{min} = maximum and minimum pulse pressures during the respiratory cycle.
- SV_{max} and SV_{min} = maximum and minimum stroke volumes during the respiratory cycle.

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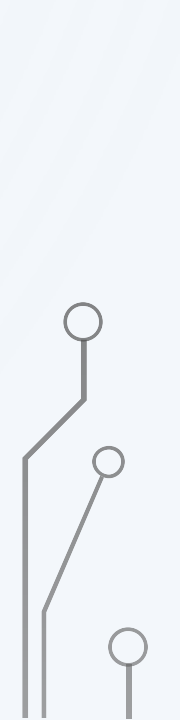

Step 4: send initial investigations

- As the patient is being resuscitated, send blood for complete hemogram, blood cultures (two sets), and other appropriate cultures depending on the clinical situation, urea, creatinine, electrolytes, liver function test, ECG, and chest X-ray.
- Send arterial blood for **arterial blood gas** and **lactate analysis**. Increased lactate is a feature of global hypoperfusion and needs urgent attention.
- Serial lactate measurement should be done frequently as it may be useful in monitoring response to resuscitation, when initial lactates are high due to tissue hypoperfusion



- If lactate is not available, **base deficit** (metabolic acidosis) in the absence of renal failure, can be taken as a surrogate marker of lactic acidosis.

- Bio-markers of infection such as procalcitonin are not diagnostic of sepsis. However they have a **very high negative predictive value** and can be used to rule out sepsis. Serial Procalcitonin may be helpful in de-escalation of antibiotics. Testing for procalcitonin should not delay fluid resuscitation and antibiotic administration in patients with sepsis and septic shock.



Step 5: start antimicrobial agent

⌚ TimingStart

- broad-spectrum antibiotics within 1 hour of recognizing septic shock.
- Send cultures first, but if delayed >45 min → start antibiotics immediately.

💊 Antibiotic Selection

- Choose agents active against likely pathogens (bacterial, fungal, viral).
- Ensure good tissue penetration at the infection site.
- Base dosing on pharmacokinetic/pharmacodynamic (PK/PD) principles.




⚠ **Multidrug-Resistant (MDR)**

Risk Factors

- Prolonged hospital stay or ICU admission
- Recent antibiotic use
- Prior hospitalization
- Previous colonization/infection with MDR organisms

⌚ **Duration**

- Typically 7–10 days
 - Longer if: slow response, difficult-to-access infection (e.g., joints), fungal/viral infection, or immunodeficiency.
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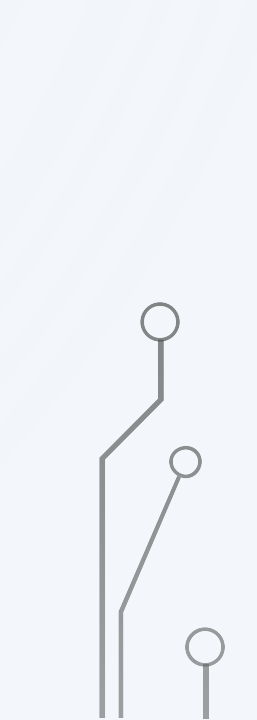


Antiviral Therapy

- Start early if viral sepsis suspected (e.g., severe influenza).

Empirical Antifungal Therapy

Start in high-risk patients with:

- Immunocompromised state (neutropenia, transplant, diabetes, liver/kidney failure)
 - Prolonged invasive devices (central line, dialysis catheter)
 - Recent surgery, especially abdominal
 - Prolonged ICU stay
 - Previous fungal infection or multisite colonization
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Step 6: source control

🎯 Goal

- Identify and eliminate the specific anatomic source of infection as early as possible.

🧠 Methods of Source Control

- Drainage of abscesses
- Debridement of infected or necrotic tissue
- Removal of infected devices (e.g., catheters, drains)

Step 6: source control

Timing

- Perform source control as soon as medically and logistically feasible.
- Ideally within 6–12 hours of diagnosis.
- Delays >12 hours are associated with worse outcomes.

Device Management

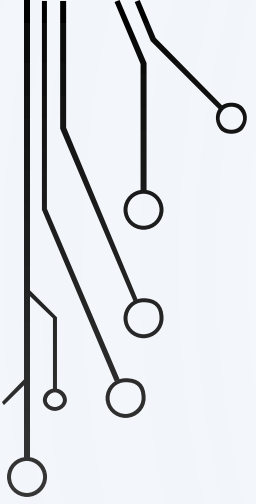
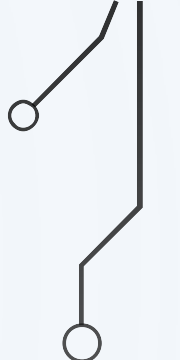

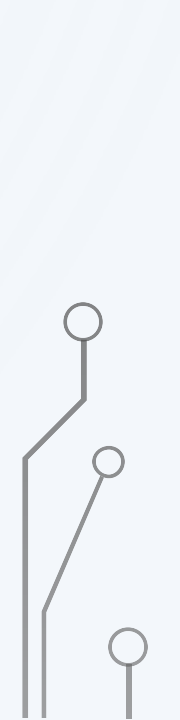
- Remove central lines and intravascular devices if they are a potential infection source — after establishing alternative vascular access.

Step 7: fluid therapy

- **Crystalloids** are the fluids of choice in sepsis and septic shock.
- Avoiding **hyperchloremia** may be an important issue with large volume resuscitation.
- Balanced salt solutions contain less chloride than normal saline and may be used as resuscitation fluid.
- **5% Albumin** When large volumes of crystalloids are needed to maintain perfusion. For initial resuscitation and ongoing volume replacement in severe sepsis/septic shock.
- **Hydroxyethyl starches have no role in resuscitation** of sepsis and septic shock due to its effects on renal function and the coagulation system and the attendant increase in mortality in clinical trials.

Step 8: optimize vasopressor use

- Vasopressors should be started as early as possible in septic shock as the duration and the degree of hypotension are associated with increased mortality.
- Vasopressor (norepinephrine) needs to be started to keep MAP more than **65 mmHg** as **a drug of choice**.
- **Intra-arterial line** should be placed in all these patients.
- Add low-dose **vasopressin** (0.03 unit/min) if the patient remains hypotensive on catecholamine.
- **Epinehrine** should be chosen **alternative** agents in septic shock that is poorly responsive to **norepinephrine**.
- Vasopressin should **not be used** as a first-line agent for hypotension.

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- **Dopamine** may be used as an alternative vasopressor agent to norepinephrine in highly selected patients at very low risk of arrhythmias and with low cardiac output and/or **low heart rate**.
 - High-dose vasopressors should always be given **through the central line**.
 - All attempts should be made to taper off vasopressors once blood pressure stabilizes.
 - Low-dose renal dopamine should **not be used** in managing these patients.
 - **Dobutamine** infusion should be administered or added to vasopressor (if in use) in the presence of **myocardial dysfunction** as suggested by elevated cardiac filling pressures and low cardiac output.

Step 9: de-escalation of fluids and vasopressors

For patients who demonstrate response to therapy, the rate of fluid administration should be reduced. Titrate the fluid therapy down to maintenance level, move to enteral feeds.

- Try to maintain neutral fluid balance
- If patient is not self diuresing, low dose diuretics in stable patient may achieve a neutral fluid balance

$$\text{Fluid Balance} = \text{Fluid Intake} - \text{Fluid Output} \approx 0$$



Step 9: corticosteroids

Indication:

- Septic shock unresponsive to fluids & vasopressors
- Hydrocortisone: 200 mg/day (continuous infusion or 50 mg IV q6h)

Duration:

- Continue while patient requires vasopressors.
- Gradually decrease over ~1 week once vasopressors are discontinued.

Chronic users: Continue replacement dose



Step 10: maintain glycemic control

- Frequent monitoring of blood glucose needs to be done.
- A protocolized approach to blood glucose management in ICU is recommended in patients with severe sepsis, commencing insulin infusion when two consecutive blood glucose levels are equal to or more than 180 mg/dL. **This protocolized approach should target an upper blood glucose less than or equal to 180 mg/dL rather than an upper target blood glucose greater than or equal to 110 mg/dL.**
- ❖ **Keep blood sugar between 140 and 180 mg/dL, preferably with intravenous insulin infusion.**

Step 11: other adjuncts

1. Blood Transfusion

- Maintain hemoglobin >7 g/dL (unless cardiac ischemia or special conditions).
- Avoid FFP unless there is active bleeding or an invasive procedure planned.

2. Mechanical Ventilation

- Use lung-protective strategy: Low tidal volume (≈ 6 mL/kg predicted body weight). Plateau pressure <30 cmH₂O. Adequate PEEP to prevent alveolar collapse.

3. Sedation


- Minimize sedation; prefer intermittent boluses over continuous infusion.
- Helps reduce ventilation duration and ICU stay.

4. Renal Replacement Therapy (RRT)

- For sepsis with AKI \rightarrow use continuous RRT for better hemodynamic stability.


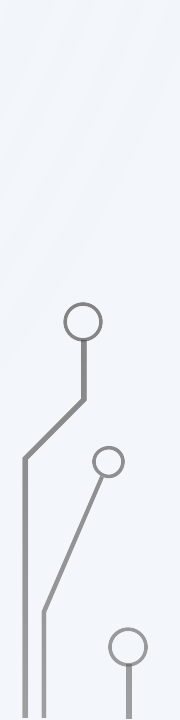


Step 12: following therapies are no more recommended in the management of severe sepsis

- Activated protein C.
 - Immunoglobulins.
 - Intravenous selenium.
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Step 13: general support

- General ICU support such as nutrition, stress ulcer prophylaxis, and deep vein thrombosis prophylaxis should be instituted.
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An illustration of an ICU setting. A patient is lying in a hospital bed on the left, wearing a blue t-shirt and a white oxygen mask. To the right, a healthcare worker in blue scrubs, a blue cap, and a white face mask stands next to a medical monitor displaying a heart rate line. In the background, there are IV stands with bags, another monitor, and a wall-mounted screen. The entire scene is rendered in a light blue color palette.

THANK YOU ICU STUDENTS

Have a good day!