

Septic Shock 101: An RT Guide to Recognition & Intervention

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Objectives

Pathophysiological foundations

Early clinical sepsis identification

Sepsis screening and biomarkers

Sepsis: ARDS interaction mechanics

Initial airway and respiratory support

Lung protective mechanical ventilation

Collaborative ICU sepsis bundles



SEPSIS & SEPTIC SHOCK OVERVIEW

Global healthcare challenge

Defining septic shock

Pulmonary integration

Primary source of infection

Cytokine storm



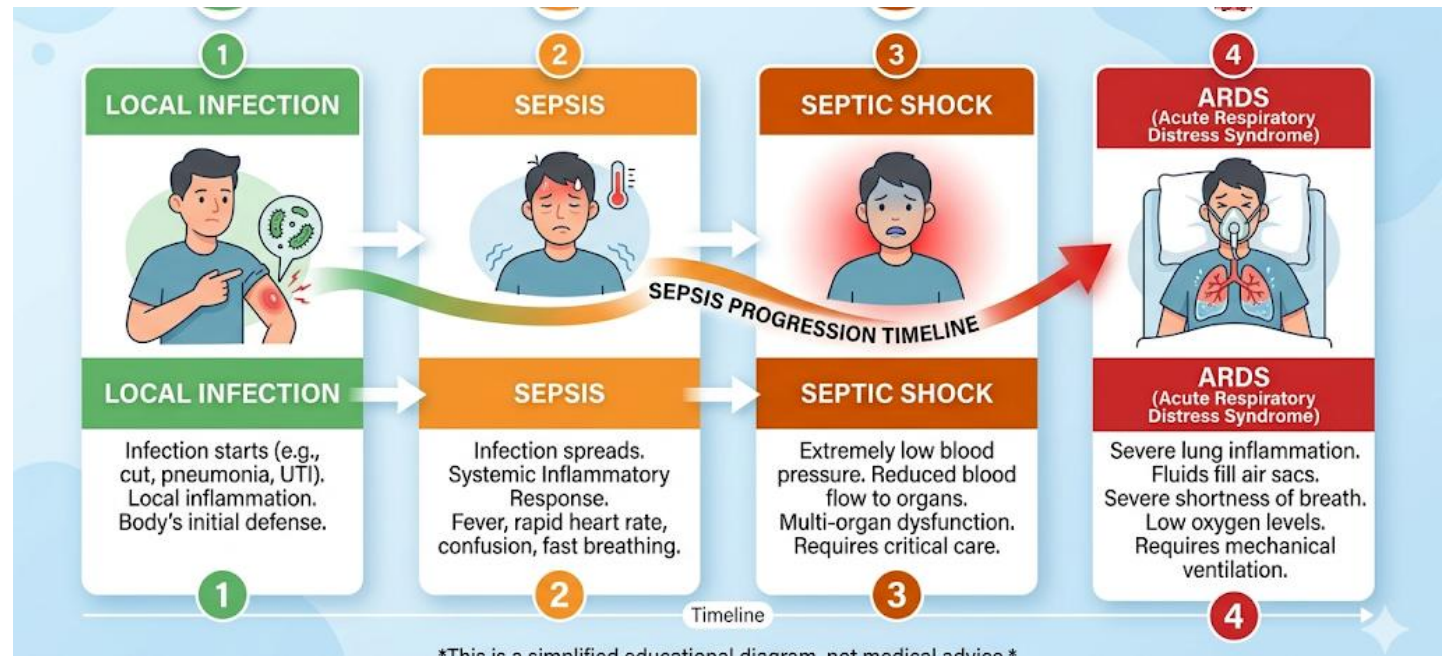
THE CLINICAL SEPSIS TIMELINE

Local infection

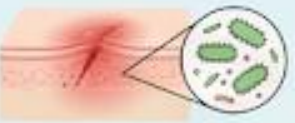
Sepsis

Septic shock

MODS/ ARDS



PROGRESSION FROM LOCAL INFECTION TO SEPSIS, SEPTIC SHOCK, AND ARDS

Stage 	Pathophysiology 	Common Symptoms 	Target Indicators / Lab Findings 
 LOCAL INFECTION	- Pathogens enter and multiply at a specific site (e.g., skin wound, UTI, pneumonia). - Local immune response.	- Redness (Rubor) - Swelling (Tumor) - Pain (Dolor) - Heat (Calor) - Impaired function.	- WBC count may rise. - Cultural confirmation of pathogen (e.g., skin swab, urine culture).
 SEPSIS	- Bacterial infection spreads to the bloodstream. Body launches a system-wide, dysregulated immune response (cytokine storm). - Severe inflammation.	- High or low temperature - Shivering - Tachypnea - Tachycardia - Altered mental state.	- Abnormal WBC. - Elevated lactate. - Multiple organ dysfunction score. - Blood cultures.
 SEPTIC SHOCK	Profound metabolic and circulatory disturbances. Sepsis leads to severe hypotension (low blood pressure) and hypoperfusion despite fluid resuscitation.	- Hypotension (e.g., MAP <65 mmHg). - Reduced urine output. - Cold, clammy skin. - Confusion.	- Serum lactate levels >2 mmol/L. - Refractory to initial treatment.
 ARDS (Acute Respiratory Distress Syndrome)	- Severe lung injury often driven by sepsis. - Damage to alveolar-capillary membrane leads to non-cardiogenic pulmonary edema (fluid in lungs). - Impaired gas exchange.	- Severe dyspnea (shortness of breath). - Tachypnea. - Cyanosis. - Hypoxemia despite oxygen therapy.	- Low PaO₂/FiO₂ ratio (e.g., <300 mmHg). - Chest X-ray/CT showing bilateral infiltrates (e.g., 'white out' appearance). - Mechanical ventilation required.

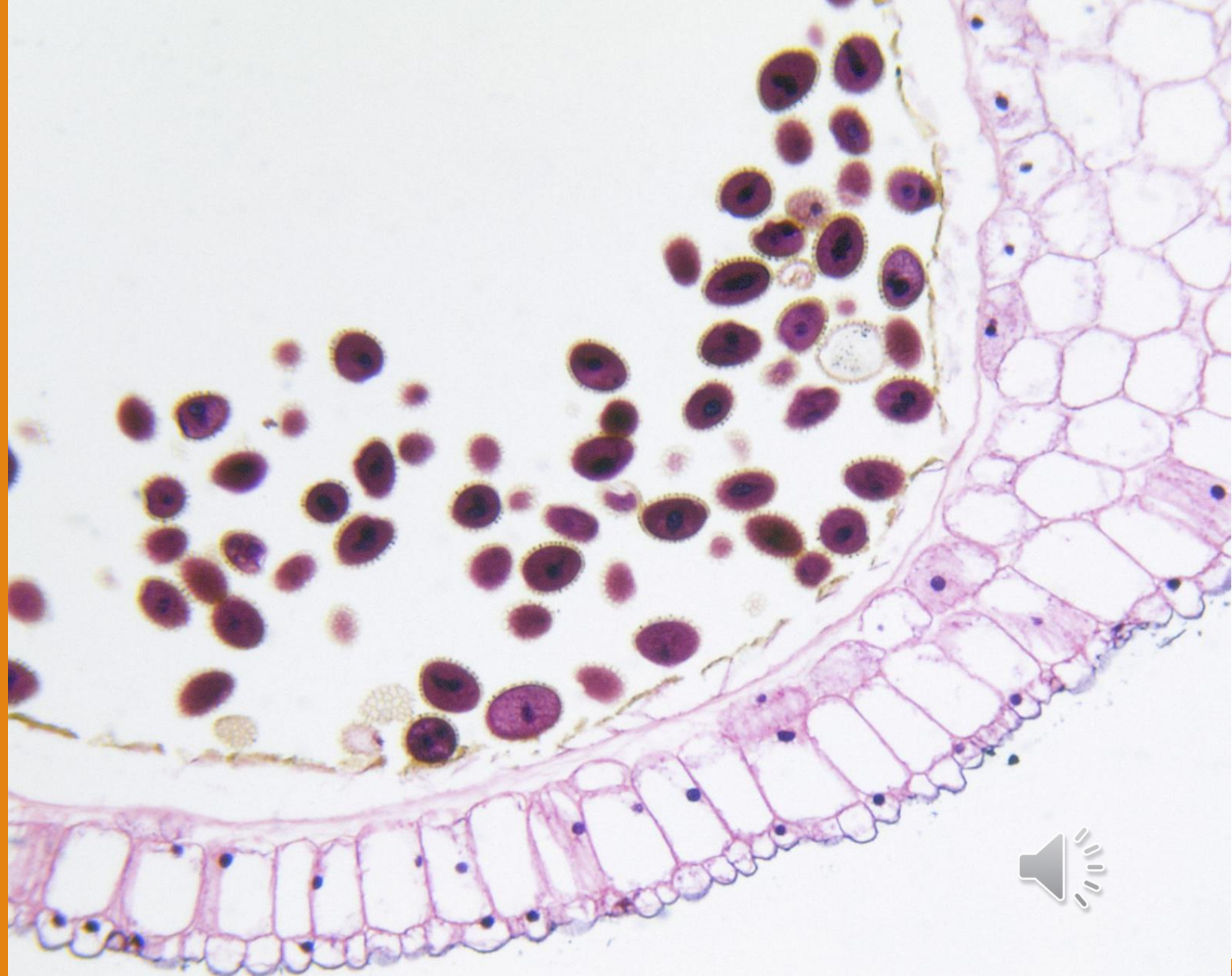
Pathophysiology of Septic Shock

Microvascular Dysfunction

- Widespread endothelial damage triggers microvascular coagulation. Fibrin deposits block capillary flow, causing profound tissue dysoxia. Organs fail to receive oxygen despite adequate system-level delivery.

Vasodilation & Leakage

- Nitric oxide overproduction causes massive systemic arterial vasodilation. Concurrently, hyper-permeable capillaries allow massive plasma leakage into the interstitial space, exhausting intravascular volume.



The RT's Frontline Sepsis Role

Tachypnea is often the Earliest Warning

- An elevated respiratory rate (RR >22) is frequently the body's primary compensatory mechanism for developing lactic metabolic acidosis, preceding hypotension.

Monitoring Cardiopulmonary Interaction

- RTs are uniquely positioned to assess sub-clinical signs of respiratory fatigue, accessory muscle use, and alterations in work of breathing (WOB).

Immediate Diagnostic Integration

- Assessing arterial blood gas (ABG) samples, evaluating shunt, and calculating oxygenation indices are crucial parts of early clinical grading.



Quick Sequential Organ Failure Assessment (qSOFA)

The qSOFA Criteria

A patient is assigned **1 point** for each of the following abnormal vital signs or clinical findings:

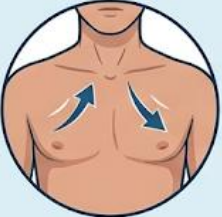
Interpreting the Score

- **Score of 0 or 1:** Indicates a lower immediate risk, though the patient must still be monitored closely if an infection is suspected.
- **Score of >2:** This is a critical threshold. A score of 2 or 3 indicates a **significantly higher risk of in-hospital mortality or a prolonged ICU stay.**

Criterion	Threshold	Clinical Significance
Altered Mentation	Glasgow Coma Scale (GCS) score less than 15	Any new confusion, disorientation, lethargy, or altered mental status from baseline.
Tachypnea (Rapid Breathing)	Respiratory rate > 22 breaths per minute	Indicates respiratory distress or metabolic compensation.
Hypotension (Low Blood Pressure)	Systolic blood pressure < 100 mmHg	Signs of cardiovascular compromise or poor tissue perfusion.



 **TACHYPNEA**
(Fast Breathing) 

 Respiratory rate **>22 breaths per minute** or new need for oxygen.

 **ALTERED MENTATION**

New **confusion, disorientation, drowsiness, or difficult to rouse.** 

CLINICAL SEPSIS RED FLAGS



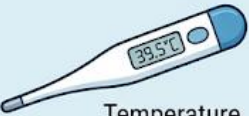
If you observe any of these red flags, **act immediately!**
SEPSIS is a medical emergency.

 **HYPOTENSION**
(Low Blood Pressure) 

Systolic blood pressure **<100 mmHg** or **dynamic drops.** 

 **THERMAL SPIKES**
(Fever or Hypothermia) 

 High or w shivering

Temperature **>38.3°C (101°F)** or **<36°C (96.8°F)**, often with **rigors** (shaking chills). 

Clinical Sepsis Red Flags

Tachypnea

Hypotension

Altered mentation

Thermal spikes



Parameter Evaluated	qSOFA (Quick SOFA Score)	NEWS (National Early Warning Score)
Target Clinical Utility	Fast bedside predictor of inpatient mortality risk.	Comprehensive early detection tool for sepsis.
Respiratory Parameters	RR $\geq$ 22 bpm (1 point)	Detailed tiering of RR, SpO ₂ , & supplemental O ₂ .
Cardiovascular Parameters	Systolic BP $\leq$ 100 mmHg (1 point)	Systolic BP tiered; Heart Rate tracked dynamically.
Neurological/Thermic Status	Altered mentation GCS $<$ 15 (1 point)	AVPU Scale (Alert, Voice, Pain, Unresponsive) & Temp.
Risk Stratification	High Risk: Score $\geq$ 2 points	Medium Risk: 5–6 points; High Risk: $\geq$ 7 points

Sepsis Screening Tools Compared qSOFA Versus NEWS



Lactate as a Perfusion Marker

Lactate Dynamics in Septic Shock

- Elevated blood lactate is a key surrogate marker for cellular dysoxia, tissue hypoperfusion, and anaerobic metabolism. Standard diagnosis of septic shock requires lactate levels greater than 2.0 mmol/L after fluid resuscitation.

Lactate Clearance Target

- Clinicians aim for a clearance of at least 10% every two hours during resuscitation. A failure to clear lactate indicates unresolved tissue hypoxia and carries high mortality risk.

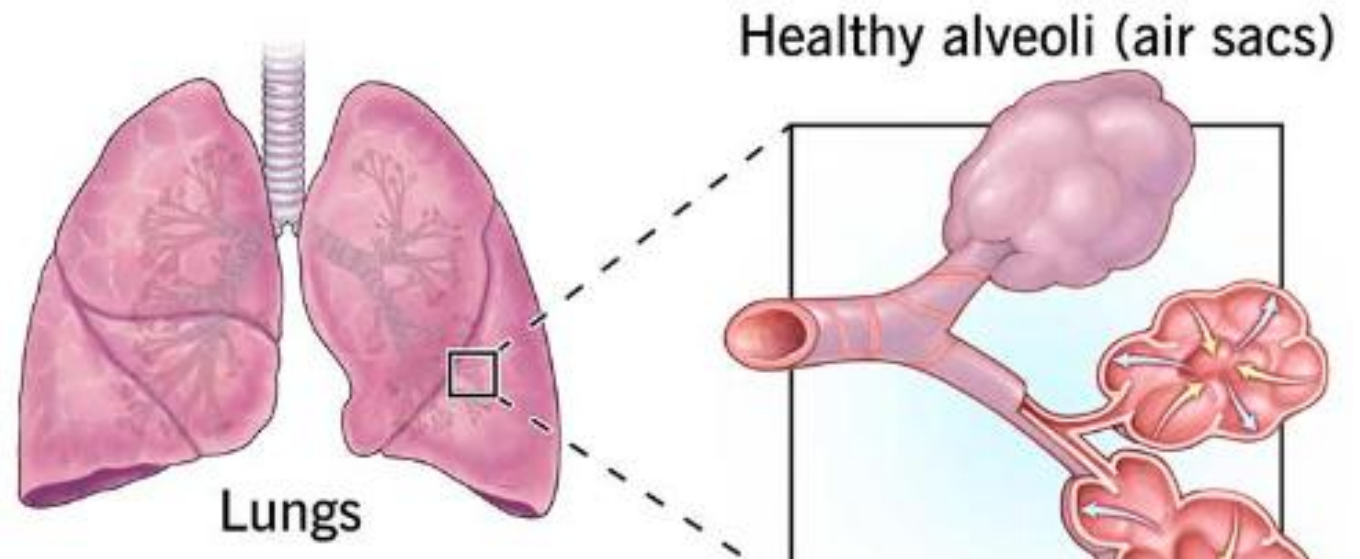


The Sepsis-ARDS Connection

Sepsis-Induced Lung Damage

- Sepsis is the most common cause of Acute Respiratory Distress Syndrome (ARDS), accounting for over 40% of cases in the ICU.
- Endotoxins destroy the capillary endothelium. Cytokines recruit neutrophils, causing massive alveolar flooding, protein leakage, surfactant degradation, and acute, refractory hypoxemia.

Acute respiratory distress syndrome (ARDS)



Fluid Resuscitation Pitfalls

Initial Crystalloid Volume

- The Surviving Sepsis guidelines recommend an initial crystalloid fluid bolus of 30 mL/kg within the first 3 hours. This volume is critical to restore mean arterial pressure and microvascular organ perfusion.

Pulmonary Complications

- Excessive fluids degrade the endothelial glycocalyx, leaking into the lungs and causing hydrostatic pulmonary edema. RTs must monitor lung sounds, worsening compliance, and escalating oxygen demands closely.



The Hour-1 Sepsis Bundle

Measure Blood Lactate Level

- Evaluate metabolic status instantly. Re-measure lactate if the initial reading is > 2.0 mmol/L to guide fluid therapy.

Obtain Key Blood Cultures

- Draw blood cultures (two distinct sets) before administering empirical antimicrobials to isolate the source.

Give Broad-Spectrum Antibiotics

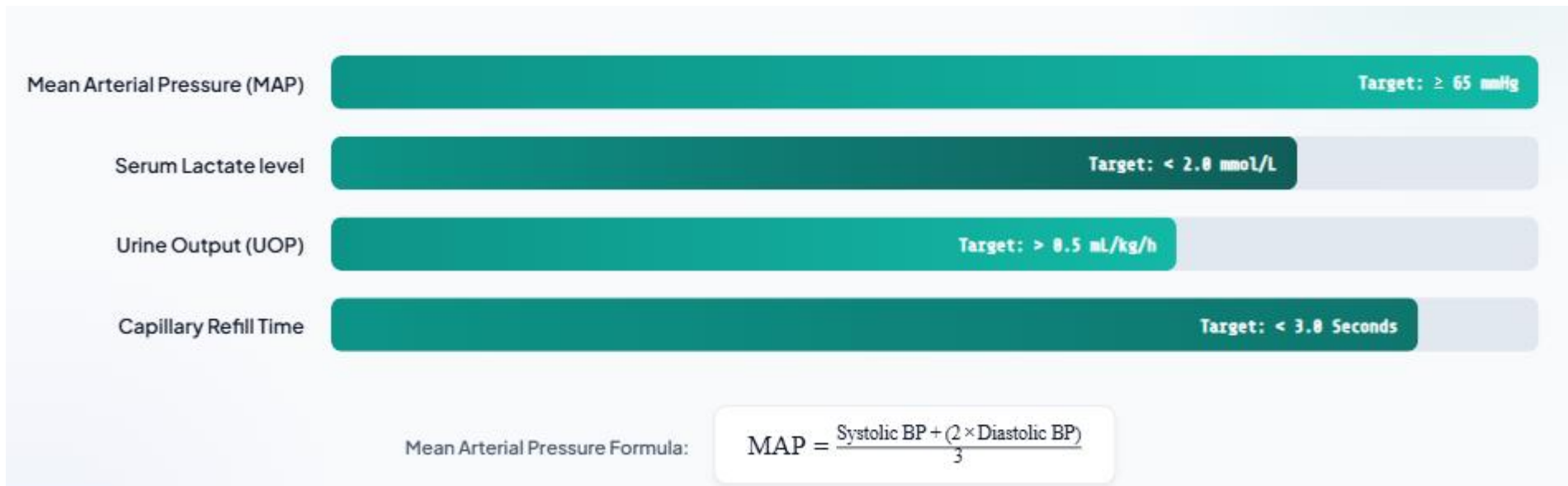
- Administer IV broad-spectrum empiric antibiotics immediately; do not delay for difficult vascular access or cultures.

Crystalloid and Vasopressor Infusion

- Infuse 30 mL/kg fluids for hypotension. Initiate vasopressors immediately if MAP remains below 65 mmHg.



Resuscitation Metric Targets





HIGH-FLOW NASAL CANNULA (HFNC)

Why HFNC is Preferred

- Surviving Sepsis guidelines suggest HFNC over Non-Invasive Ventilation (NIV) for acute hypoxemic respiratory failure. High flow rates (up to 60 L/min) provide precise FiO₂, generate mild positive pressure (3-5 cm H₂O PEEP), and match patient inspiratory demands, reducing intubation rates.

ROX Index Monitoring

- The ROX index combines respiratory rate and oxygenation parameters to predict success or identify when intubation is needed:
- $$ROX = \frac{SpO_2/FiO_2}{\text{Respiratory Rate}}$$

Score < 4.88 at 12 hours is associated with high risk of HFNC failure.



NON-INVASIVE VENTILATION LIMITS

Aspiration and Hemodynamic Collapse

- NIV is generally discouraged in septic shock. Severe encephalopathy leads to poor airway protection, high emesis risk, and severe aspiration pneumonitis.

Sustained High Transpulmonary Pressures

- Patients on NIV often generate vigorous inspiratory efforts, producing excessive transpulmonary pressures. This exacerbates Patient-Self Induced Lung Injury (P-SILI).

Delayed Intubation Risk

- Prolonging failing NIV leads to muscle exhaustion, abrupt respiratory arrest, and severe hypoxemia, which significantly increases mortality rates.



SEPSIS AIRWAY MANAGEMENT

Severe Post-Induction Collapse

- Induction agents and positive pressure ventilation acutely reduce venous return. In volume-depleted septic shock patients, this can trigger severe cardiac arrest.

The Pre-Oxygenation Mandate

- Ensure high-flow oxygenation is maintained. Delay induction if safe to optimize fluid volume and initiate early vasopressor support.

RT Preparedness Roles

- Assemble a bag-valve-mask (BVM) with PEEP valves, prepare colorimetric capnography, set up a functional suction unit, and pre-program emergency ventilator settings.



LUNG-PROTECTIVE VENTILATION

Low Tidal Volume Ventilation (LTVV)

- Initiate volume-control at 6 mL/kg predicted body weight (PBW) to prevent alveolar overdistension (volutrauma). Titrate down to 4 mL/kg if necessary.

Strict Plateau Pressure Limits (Pplat)

- Maintain Plateau Pressure ≤ 30 cm H₂O. Pplat reflects alveolar pressure and should be measured via a 0.5-second inspiratory pause.

Keep Driving Pressure ≤ 15 cm H₂O

- Driving pressure represents the tidal volume scaled to functional lung size. Keep ΔP within safe thresholds:

$$\Delta P = \text{Plateau Pressure} - \text{Total PEEP}$$



ARDSnet PEEP/FiO₂ TABLES: Guidance for ARDS Mechanical Ventilation

1. LOWER PEEP / HIGHER FiO₂ STRATEGY (Standard)

 FiO ₂ (Inspired O ₂ Fraction)	 PEEP (cmH ₂ O) ↓
0.3	5 ↑
0.4	5 ↓
0.4	8
0.5	8
0.5	10
0.6	10 ↑
0.6	11
0.7	14
0.7	16
0.7	18 ↑
0.8	14-14 ↓
0.8	16
0.9	18
0.9	18-20 ↓

2. HIGHER PEEP / LOWER FiO₂ STRATEGY (Alternative)

 FiO ₂ (Inspired O ₂ Fraction)	 PEEP (cmH ₂ O) ↓
0.3	12 ↑
0.3	14 ↓
0.4	14
0.4	16
0.5	16
0.5	17
0.6	18
0.6	20
0.7	20
0.7	24 ↓
0.8	16-16 ↑
0.8	18-20
0.9	20-21
0.9	22-22 ↑





ARDS RESCUE THERAPIES

Prone Positioning Protocol

- For patients with $\text{PaO}_2/\text{FiO}_2$ ratios < 150 , apply prone positioning for ≥ 16 hours daily. This redistributes ventilation toward healthier posterior lung segments, improves V/Q matching, and reduces ventilator-induced lung injury (VILI).

Neuromuscular Blockade

- Continuous infusion of neuromuscular blocking agents (NMBAs) for the first 48 hours eliminates patient-ventilator asynchrony and prevents harmful P-SILI, reducing overall oxygen consumption.



ACID-BASE BALANCE IN SEPSIS

Metabolic Acidosis Management

- Tissue hypoperfusion in septic shock leads to lactic metabolic acidosis. For patients with severe metabolic acidosis ($\text{pH} \leq 7.20$) accompanied by acute kidney injury (AKI Stage 2/3), Surviving Sepsis guidelines suggest using sodium bicarbonate therapy.

Respiratory Compensation

- Sepsis-induced metabolic acidosis drives high respiratory demands. The RT must ensure the ventilator settings permit adequate minute ventilation to compensate for severe metabolic acidosis.



Weaning and liberation

Standardized Daily Sedation Interruption

- Daily sedation holidays are critical to evaluate mental status, decrease ventilator days, and reduce the risk of developing ICU delirium.

Daily Spontaneous Breathing Trials (SBTs)

- Initiate SBTs once hemodynamic stability is restored, vasopressor demands are low, and oxygenation parameters are stable ($\text{FiO}_2 \leq 0.40$ and $\text{PEEP} \leq 8$).

Rapid Shallow Breathing Index (RSBI)

- Calculate RSBI during a brief trial of unsupported breathing to assess readiness for weaning:

$$\text{RSBI} = \frac{\text{Respiratory Rate (f)}}{\text{Tidal Volume (Vt in Liters)}}$$

An RSBI < 105 is predictive of a successful extubation.





THE SEPSIS CARE TEAM

Respiratory therapist

ICU physician

Critical care nurse

Clinical pharmacist



Early Suspicion and Resuscitation

- Recognizing early signs like tachypnea ($RR \geq 22$) and maintaining target perfusion ($MAP \geq 65$ mmHg, lactate < 2.0 mmol/L) are critical first steps. Promptly implement the Hour-1 bundle to stop systemic injury.

Lung-Protective Mechanical Support

- Limit mechanical ventilation to 6 mL/kg PBW, maintain $P_{plat} \leq 30$, and keep Driving Pressure ≤ 15 . Apply early high PEEP and prone positioning for sepsis-induced moderate-to-severe ARDS.

SUMMARY & TAKEAWAYS



EVIDENCE-BASED REFERENCES

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