CHAPTER 199
Sepsis

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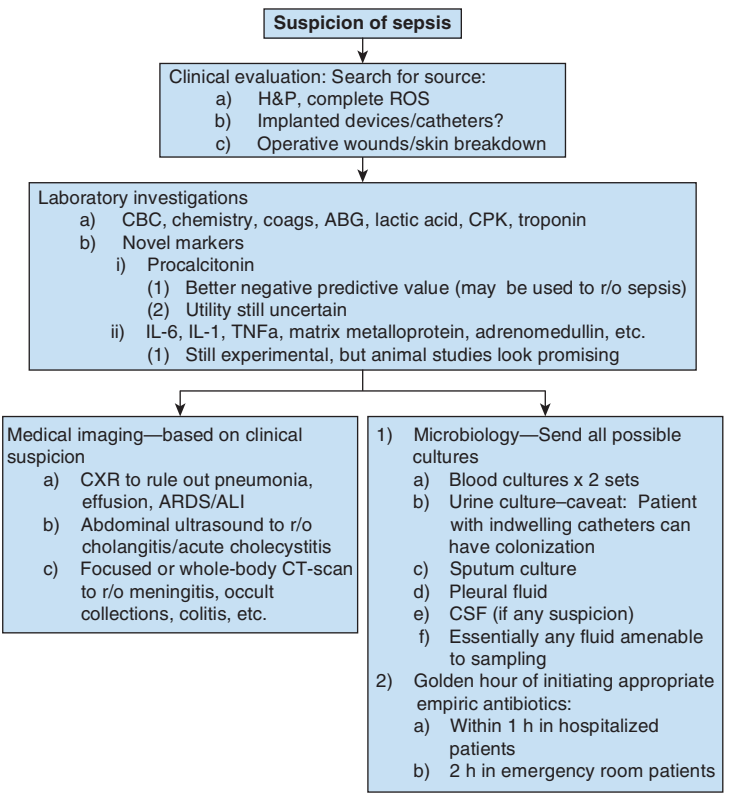
DEFINITIONS

- Systemic inflammatory response syndrome (SIRS): systemic response to any inflammatory/infectious etiology (see Table 199-1)

TABLE 199-1 SIRS Criteria (Need 2 Out of 4)

• WBC >12,000/mL, <4,000/mL, or >10% immature bands
• HR >90/min
• Temperature >38.5°C or <35°C
• RR >20 breaths/min, or $Paco_2$ <32 mm Hg

FIGURE 199-1. Diagnostic workup of sepsis



- Sepsis: definite infectious etiology with a resultant systemic response (at least two or more SIRS criteria)
- Severe sepsis → sepsis with acute organ dysfunction
- Septic shock → sepsis-induced hypotension refractory to fluid resuscitation and evidence of end-organ damage including lactic acidosis, oliguria, or altered mental status
- *Mortality ranges from 28% to 50%*

Management of Severe Sepsis or Septic Shock	
Initial resuscitation (first 6 h)	
Patients with hypotension, lactic acid >4 mmol/L require immediate resuscitation	
Goals	• Mean arterial pressure ≥65 mm Hg • Urine output ≥0.5 mL/kg/h • Central venous pressure (CVP) 8–12 mm Hg (controversial benefit) • Central venous oxygen saturation ≥70% or mixed venous ≥65% (controversial benefit)
Crystalloids and colloids equally effective	• Challenges of 1,000 mL of crystalloid or 300–500 mL of colloid over 30 min • May require larger volumes in patients with persistent hypotension with vasopressors • Bicarbonate therapy contraindicated in patients with hypoperfusion-induced lactic acidosis and pH ≥7.15
Blood product transfusion	• Transfuse packed red blood cells to target Hb ≥7 (may require higher targets in patients with special circumstances [myocardial ischemia, etc.]) • Avoid plasma or platelet administration unless active bleeding or planned procedure
Vasopressors	• Start if shock persists despite fluid resuscitation (20–30 mL/kg) to keep MAP ≥60–65 mm Hg • Vasopressors, including norepinephrine and dopamine, should be administered via central venous catheter • Vasopressin (0.03 U/min), phenylephrine, or epinephrine may be added if shock unresponsive to initial vasoactive medications • Arterial catheter use recommended for hemodynamic monitoring • Dobutamine recommended in patients with myocardial dysfunction
Source control and antibiotics	• Identify infectious etiology within 6 h of presentation • Evaluate and implement measures of source control (abscess drainage, tissue debridement, etc.) • Remove infected intravascular devices • Culture all available specimens • Start broad-spectrum antibiotics within the first hour as sepsis and septic shock recognized ▸ Combination therapy should be used in patients with suspected *Pseudomonas* infection or who are otherwise immunocompromised
Mechanical ventilation in patients with ALI/ARDS	• Tidal volume of 6 mL/kg (ideal body weight) • Maintain plateau pressures ≤30 cm H_2O • Increase PEEP as needed to avoid lung collapse at end-expiration and to avoid oxygen toxicity with high FiO_2 levels • Allow $Paco_2$ to rise to minimize plateau pressures and tidal volumes • Keep head of bed elevated to at least 30° (30–45°), unless contraindicated • Institute weaning protocols and daily assessment for SBT to liberate patients from mechanical ventilation • Use conservative fluid strategy • Some advocate against PAC in patients with ALI/ARDS: no survival benefit, increased risk of infection, health care costs, and risk of nonfatal cardiac arrhythmias

(*continued*)

Management of Severe Sepsis or Septic Shock (*continued*)	
Recombinant human activated protein C (drotrecogin alfa [activated]) withdrawn from the market in 10/2011 due to lack of efficacy in the PROWESS-SHOCK trial	
Steroids	• May consider starting in patients with shock refractory to fluid resuscitation and vasopressor therapy (refractory shock: increasing doses of vasopressors, add vasopressors to maintain MAP >65 mm Hg) • Start with hydrocortisone (preferred steroid) dose of 200–300 mg/day • ACTH stimulation test is not recommended prior to starting steroids • Do not treat sepsis with steroids unless hypotension present or patient's medical status necessitates the use of steroids (prior steroid use, endocrine disorder, etc.)
Glycemic control	• Intravenous insulin should be used for correcting severe hyperglycemia in critically ill patients • Most recent evidence suggests intensive insulin therapy may lead to worse outcomes • Monitor glucose closely and aim to maintain plasma glucose levels ≤180 mg/dL
Sedation/ analgesia	• For critically ill mechanically ventilated patients, use sedation protocols • Sedation may be intermittent or as a continuous infusion • Allow for daily sedation interruption to awaken patients • Neuromuscular blockers should be avoided unless refractory hypoxemia in ARDS. Monitor response using train-of-fours
Stress ulcer prophylaxis	Use H_2 blockers or PPI
VTE prophylaxis	• No difference between low-dose unfractionated heparin or low-molecular-weight heparin • If heparin is contraindicated, mechanical compression devices can be used

REFERENCES

For references, please visit **www.TheAnesthesiaGuide.com**.

CHAPTER 200
Acute Myocardial Infarction, Complications and Treatment

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DIAGNOSIS OF ACUTE MI

- *Clinical*: new angina, increasing angina, or angina at rest. Diaphoresis, hypotension, new MR murmur, pulmonary edema or rales, JVD
- *EKG (Figures 200-1 and 200-2)*: ST segment elevation (≥0.2 mV in men or ≥0.15 mV in women in leads V2–V3 and/or ≥0.1 mV in other leads) or depression (>0.05 mV in two contiguous leads), T-wave inversion, new blocks especially LBBB
- *Echo*: wall motion abnormality, new MR (papillary muscle dysfunction)
- *Lab*: cardiac enzymes (troponin) serially STAT (for baseline) and q6 hours × 3
- *DDx*:
  - Elicit cocaine use
  - PE, aortic dissection: CT angiography to rule out

NB: See chapter 5 for more details on EKG changes.