Critical Care & Shock

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Introduction :

1. MONITORING OF THE CRITICALLY ILL PATIENT
2. RESPIRATORY FAILURE
3. CIRCULATORY FAILURE: SHOCK
4. SEPSIS
5. UPPER GASTROINTESTINAL HEMORRHAGE PROPHYLAXIS
6. ANEMIA
7. BLOOD GLUCOSE CONTROL

1.MONITORING OF THE CRITICALLY ILL PATIENT

A. Temperature Monitoring
B. Electrocardiographic (ECG) Monitoring
C. Arterial Pressure Monitoring
    1. Indirect
    2. Direct
D. Central Venous Pressure (CVP) Monitoring

    CVP, SCvO2, vasoactive medications, TPN

1.MONITORING OF THE CRITICALLY ILL PATIENT

E. Pulmonary artery (PA)

CVP, cardiac output (CO), PA pressures, SVR, SvO2

No mortality improvement in surgical patients.

Complications > Transient right-bundle-branch block

Balloon rupture

PA perforation

Malposition (e.g., coronary sinus)

Right ventricular rupture

Cardiac tamponade.

1.MONITORING OF THE CRITICALLY ILL PATIENT

F. Esophageal Doppler and Pulse Contour Analysis

Less invasive > descending aortic blood flow > CO

Pulse contour analysis > arterial pressure waveform > CO

G. Respiratory Monitoring

1. Pulse oximetry

2. Capnography > Quantitative, continuous assessment of expired CO_2 concentrations

ETCO2 ↑ > ↓ Alveolar ventilation, ↑ CO_2 production…Sepsis

ETCO2 ↓ > ↑ Alveolar ventilation or ↑ dead space…ET obs, PE

1.MONITORING OF THE CRITICALLY ILL PATIENT

H. Neurologic Monitoring

1. Intracranial pressure monitoring
2. Processed electroencephalogram monitors

2. RESPIRATORY FAILURE

A. Etiology: Inadequate gas exchange > ventilation/perfusion (V/Q) mismatch

Hypoventilation

Impaired systemic delivery/extraction

Dead space ventilation > COPD & PE.

B. **Diagnosis:** Signs or symptoms of respiratory distress > Pulse oximetry and an ABG.

Oxygen saturation <90% impaired tissue oxygenation.

↑ $PaCO_2$ > (respiratory acidosis)

History, physical examination a chest x-ray, EKG, and chest CT with PE protocol.

2. RESPIRATORY FAILURE

Inhaled oxygen

Noninvasive positive pressure ventilation (NIPPV)

Endotracheal intubation

Chest tube placement

Extracorporeal membrane oxygenation (ECMO)

3. CIRCULATORY FAILURE: SHOCK

A. Shock is defined by global tissue hypoxia and occurs when the supply of oxygen is insufficient to meet metabolic demands.

B. Classification and Recognition of Shock.

History, laboratory values, and physical examination are usually sufficient for determining the etiology

1. **Hypovolemic shock** results from loss of circulating blood volume caused by acute hemorrhage, fluid depletion, or dehydration. Patients are peripherally vasoconstricted, tachycardic, and have low jugular venous pressure.

3. CIRCULATORY FAILURE: SHOCK

2. **Distributive shock** is a hyperdynamic state consisting of tachycardia, vasodilation, decreased SVR, and increased CO. The most common causes include sepsis, neurogenic shock, adrenal insufficiency, and liver failure.

3. **Neurogenic shock** results from interruption of the spinal cord at or above the thoracolumbar sympathetic nerve roots, which produces loss of sympathetic tone, causing vasodilation. Patients are peripherally vasodilated and tachycardic. Jugular venous pressure is low.

3. CIRCULATORY FAILURE: SHOCK

4. **Obstructive shock** results from etiologies that prevent adequate CO but are not intrinsically cardiac in origin. This may be caused by PE, tension pneumothorax, or cardiac tamponade. Jugular venous pressure is elevated while the peripheral tissues demonstrate vasoconstriction

5. Cardiogenic shock results from inadequate CO due to intrinsic cardiac failure. Diagnosis may require echocardiography. These patients typically are peripherally vasoconstricted and tachycardic with an elevated jugular venous pressure

TABLE 7-3 Clinical Parameters in Shock

Shock Classification	Skin	Jugular Venous Distention	Cardiac Output	Pulmonary Capillary Wedge Pressure	Systemic Vascular Resistance	Mixed Venous Oxygen Content
Hypovolemic	Cool, pale	↓	↓	↓	↑	↓
Cardiogenic	Cool, pale	↑	↓	↑	↑	↓
Septic						
Early	Warm, pink	↑↓	↑	↓	↓	↑
Late	Cool, pale	↓	↓	↓	↑	↑↓
Neurogenic	Warm, pink	↓	↓	↓	↓	↓

3. CIRCULATORY FAILURE: SHOCK

- The goal of therapy is to ensure adequate oxygen delivery. Because oxygen delivery is proportional to SaO2, hemoglobin concentration, and CO, each should be optimized.

Specific Therapy:

Hypovolemic shock > Therapy focuses on control of ongoing loss and restoration of intravascular volume.

Neurogenic shock > The initial intervention is volume infusion. A peripheral vasoconstrictor, phenylephrine or norepinephrine, is administered to increase vascular tone if hypotension is refractory to volume infusion.

3. CIRCULATORY FAILURE: SHOCK

Obstructive shock > Tension pneumothorax is treated by needle decompression followed by tube thoracostomy.

Pericardial tamponade is treated by needle decompression, often with catheter placement for drainage.

Cardiogenic shock > Management is directed toward maintaining adequate myocardial perfusion and CO with volume expansion and vasoactive medications

4. SEPSIS

A. Definition:

Sepsis is defined as SIRS with a documented or presumed infection. The clinical definition of SIRS requires two of the following:

Body temperature >38¡C or <36¡C

Heart rate >90 beats/minute

Respiratory rate >20/minute or Paco2 <32

WBC count >12 or 10% bands

Severe sepsis is multiple-organ dysfunction or hypoperfusion (septic shock) resulting from infection.

4. SEPSIS

B. Diagnosis: Cultures

C. Treatment:

1. Infection
    a. Antibiotic therapy
    b. Source control
2. Circulatory support
    a. Volume resuscitation.
    b. Vasoactive medications.

5. UPPER GASTROINTESTINAL HEMORRHAGE PROPHYLAXIS.

Patients in the ICU are at increased risk for stress-induced mucosal ulceration and GI hemorrhage.

Risk factors: head injury (Cushing ulcers); burns (Curling ulcers); prolonged mechanical ventilation; history of peptic ulcer disease; NSAIDs or steroids; and the presence of shock, renal failure, portal hypertension, or coagulopathy

H2-receptor antagonist > Proton-pump inhibitors

Clostridium difficile-associated diarrhea

6. ANEMIA

Transfusing all patients to a hemoglobin of 10 mg/dL either has no effect or may actually decrease survival in the critically ill.

Restrictive transfusion strategy (hemoglobin <7 mg/dl)

7. BLOOD GLUCOSE CONTROL

Tight glucose control (blood glucose goal: 80 to 110 mg/dL)

Conventional control (blood glucose goal: 180 to 200 mg/dL)

In surgical patients > twofold decrease in mortality.