Chapter 11

Critical Care

Introduction

Each battlefield ICU should have a dedicated intensive care physician, due to the severity and lethality of blast and high-velocity wounds, and the need for ongoing resuscitation of casualties requiring damage control.

> **Damage control is the initial control of hemorrhage and contamination followed by intraperitoneal packing and rapid closure, then resuscitation to normal physiology in the intensive care unit and subsequent definitive re-exploration. This places large logistic requirements on the ICU. This may include rewarming, large-volume resuscitation, blood products, vasoactive drugs, and mechanical ventilation.**

The ICU physician should observe the following guidelines:

- **Reexamine** (possibly, retriage) the patient, using detailed primary and secondary surveys, with attention to the "ABCs," potential life-threatening injuries, and other injuries missed during the ER and OR phases of resuscitation (tertiary survey).

> **Trust no one's examination before your own because the patient's condition may have changed, or prior examinations may be inaccurate or incomplete.**

- Provide necessary available **monitoring** of physiology, with periodic assessment of pain control, level of consciousness, and intake and output.
- **Resuscitate** from shock, using appropriate endpoints.
- **Provide organ-specific support**, as is done for CNS injury, pulmonary failure, cardiovascular collapse, and renal dysfunction.

- **Ensure adequate pain control.**
  - Use IV (not IM) narcotic agents in sufficient doses to alleviate pain.
  - Patients on mechanical ventilation require **both** narcotics (morphine, fentanyl) and sedatives (propofol, lorazepam, midazolam).
- Prepare the patient for **transport** out of theater.
- Important caveats for the intensivist.
  - **"Patients don't often suddenly deteriorate; healthcare providers suddenly notice!"**
  - The **organ system approach**, in which each organ system in turn is addressed in a mini-SOAP format, ensures that each of the body's physiologic systems is addressed in a complete, comprehensive, and integral fashion.
  - The **systemic inflammatory response** (SIRS) is a common metabolic sequela of severe injury, not always associated with infection.

Fever or leukocytosis should prompt a thorough search for infection. Antibiotic discipline must be enforced, saving these medications for short-course prophylaxis, documented infection, or empiric treatment of rapid deterioration due to sepsis.

Resuscitation From Shock

Shock can be defined as an acute state of cardiovascular insufficiency resulting in life-threatening global hypoperfusion. Hemorrhagic shock is the most common form of shock following major trauma. Therefore, initial efforts should be directed toward correction of hypovolemia.

Hypoperfusion implies inadequate delivery of oxygen to the body's cells. Oxygen delivery is a function of cardiac performance, arterial hemoglobin content, and arterial oxygen saturation. All attempts to correct shock involve optimizing these three variables.

- Shock resuscitation is approached in two phases, based on endpoints of resuscitation:
  - In the **first phase**, resuscitate to a mean arterial pressure of > 60 mm Hg, a urine output of 0.5 cc/kg/h (at least 30 ccs/h), and arterial oxygen saturation of > 92%.
  - Pursue endpoints aggressively to eliminate hypoperfusion, ideally within 1 hour (see Chapter 7, Shock and Resuscitation).
  - In the **second phase**, resuscitation is continued primarily with fluid, to eliminate metabolic acidosis (restore lactate to normal) within 24 hours.
  - The resuscitative fluid of choice is a warmed, balanced crystalloid solution (normal saline or lactated Ringer's) and is preferable to colloid.
  - Rate of infusion for resuscitation should be 500 mL to 1,000 mL bolus over 15–20 minutes and repeated as necessary.
  - After 3 L of crystalloid, blood products should generally follow at similar rates.

Vasopressor agents should **only** be considered for achieving minimal acceptable blood pressure **after** fluid boluses and confirmation of adequate intravascular volume.

  - Dopamine, norepinephrine, and phenylephrine are the preferred vasoactive agents, starting in the lower dose range.
  - Dobutamine should only be considered for demonstrated cardiac dysfunction, which may be seen in sepsis, the elderly, or myocardial infarction (MI).

Specific Organ Systems

Traumatic Brain Injury/CNS

Transient hypoxemia or hypotension in the patient with significant traumatic brain injury doubles the probability of death or poor neurologic outcome. The goal of treatment is to maintain cerebral perfusion pressure (CPP) and oxygenation.

- Identify potential intracranial surgical lesions for possible emergent craniotomy.
- Prevent hypoxemia: Maintain O_2 sat > 92%, PaO_2 > 100, and intubate for GCS ≤ 8.
- Prevent hypotension.
  - Maintain SBP > 100 mm Hg, MAP > 80.
    - MAP = DBP + ⅓ (SBP - DBP).
- Prevent, monitor, and treat intracranial hypertension.
  - Maintain intracranial pressure (ICP) = 5–15 mm Hg.
  - Maintain CPP = 70–90 mm Hg.

CPP = MAP – ICP

- **Measures to treat intracranial hypertension** include:
  - Elevation of head of bed 30° may be helpful.
  - Recognize that high levels of PEEP may raise ICP.
  - Control **serum osmolarity**.
    - Normal saline is the preferred IV solution.
    - Check serum sodium twice daily, and keep in the range of 145–150 mEq/dL.
    - IV **mannitol** (not in anuric patients), 0.25–1.0 g/kg, every 6–8 hours to keep serum osmolarity optimal.
  - Control **$PaCO_2$**.

Hypercarbia should always be prevented. Modest therapeutic hyperventilation may be used ($PaCO_2$ 30–35 mm Hg) for brief periods in the deteriorating patient.

    - Beneficial effects of hyperventilation/hypocarbia must be balanced: it reduces ICP through vasoconstriction, but also reduces cerebral blood flow.
    - **Prophylactic hyperventilation should not be used.**
  - Removal of **cerebrospinal fluid** by placement of an intraventricular catheter.
  - Barbiturates have unproven benefit but may be considered in extreme cases.
  - **Craniotomy** with bone and brain removal is a drastic, lifesaving procedure of last resort in the moribund patient.

Steroids have <u>no role</u> in traumatic brain injury treatment.

- **Avoid hyperthermia,** because this raises ICP.

- General Considerations.
  - Appropriate precautions should be taken (H_2 blocker, heparin, and oral care) to prevent development of stress gastritis, deep venous thrombosis, and aspiration pneumonitis.
  - If **coagulopathy** develops, use blood products as necessary to correct an elevated prothrombin time.
  - Prevent and aggressively treat pain, agitation, shivering, and fever to avoid increased cerebral metabolism and oxygen consumption.
  - **Hyperglycemia** has an adverse effect on outcome and should be monitored and treated aggressively to keep glucose levels between 100–150 mg/dL.
  - Seizure prophylaxis. Phenytoin/phosphenytoin should be administered to therapeutic levels in the penetrating head-injured patient, and to blunt head-injury patients with seizure.

Pulmonary System and Ventilators

General Considerations

- **Supplemental oxygen** in the early phase of resuscitation is imperative. The maximum fraction of inspired oxygen (FIO_2) delivered by:
  - Nasal cannula approaches 0.35.
  - Venturi mask is 0.50.
  - Non-rebreathing reservoir mask approaches 0.90.
- **Monitoring**: May include portable chest radiographs, periodic ABGs, regular assessments of level of sedation, airway pressures, and functioning of ventilator alarms.

Airway Considerations

- **Indications for endotracheal intubation and mechanical ventilation include:**
  - Airway obstruction due to trauma, edema, excess secretions.
  - Apnea.

- Excessive work of breathing (eg, flail chest), as indicated by accessory muscle use, fatigue, diaphoresis, or tachypnea when respiratory failure is imminent.
- Decreased level of consciousness: GSC ≤ 8.
- Hypoxia: $SaO_2 < 90\%$, $PaO_2 < 60$ mmHg on $FIO_2 > 50\%$.
- Hypercarbia: $PaCO_2 > 60$ mm Hg acutely (lower threshold with tachypnea).
- Shock.
- **Caution**: Patients not meeting the above criteria may still require airway protection and mechanical ventilation preceding prolonged transport.

- **Field Ventilator.**
  - Impact Uni-Vent Eagle 754.
    - Basic settings.
      - Turn the ventilator on and set the mode using the **Mode Selector Switch** (lower right). Most patients can be well ventilated using SIMV (synchronized intermittent mandatory ventilation).
      - Set FIO_2 using the **Air/Oxygen Mixer Control**, above the Mode Selector Switch. Generally, ICU patients should be started at an FIO_2 of 1.0, and weaned as appropriate to a level of 0.40.
      - Set minute ventilation using **Tidal Volume** and **Ventilation Rate** controls. The tidal volume is set at 6–10 mL/kg. Initial rate is set at 10–14 breaths/minute and titrated to normalize $PaCO_2$.
      - Set positive end-expiratory pressure (PEEP) using the **PEEP Control** located on the upper left of the control panel. Initial PEEP is usually set at 5 cm H_2O. Higher values can be set in severe respiratory failure such as adult respiratory distress syndrome (ARDS), although generally not higher than 15 cm H_2O.

Summary of typical initial settings for mechanical ventilator: FIO_2 1.0, SIMV mode, rate 12, tidal volume 800 mL, PEEP 5 cm H_2O.

ARDS

- ARDS can start within days of injury, and should be suspected in any casualty with:
  - Acute hypoxemia (PaO_2/FIO_2 ratio < 200).
  - Progressive fall in pulmonary compliance (stiff lungs, increasing airway pressures).
  - Bilateral alveolar infiltrates on chest radiograph, with no clinical evidence of volume overload.

Mechanical Ventilation Priorities in ARDS

- Maintain patient analgesia and sedation to prevent agitation and ventilator/patient asynchrony.
- Keep $SaO_2 > 90\%$ by increasing FIO_2 and/or PEEP (maximum 15–18 cm H_2O).
- Avoid prolonged $FIO_2 > 0.60$ due to O_2 toxicity.
- Avoid respiratory acidosis. Keep $PaCO_2$ 35–45 mm Hg, and arterial blood pH > 7.25.
- Keep peak inspiratory pressure (PIP) < 40 cm H_2O to prevent iatrogenic pneumothorax and destruction of normal lung tissue.
  - Decrease tidal volume to 5–7 mL/kg.
  - Increase ventilator rate.
  - If other measures are unsuccessful, allow **permissive hypercapnia** by accepting a respiratory acidosis ($PaCO_2$ 55–70 mm Hg). Use bicarb to maintain pH > 7.2.

Respiratory acidosis is less dangerous than ventilator-induced lung injury caused by high PIP and high tidal volumes.

Cardiovascular System

- The patient who exhibits **cardiovascular deterioration** after a period of apparent stability should be evaluated to rule out the following:
  - Hypoxia or loss of airway.
  - Tension pneumothorax.
  - Recurrent bleeding from sites of injury or surgery.
  - Cardiac tamponade or direct myocardial injury.

- Tachyarrhythmia.
- Fluid loss due to "third-spacing," burns, fever, diarrhea, or vomiting.
- Undiagnosed injury: intestinal injury, pancreatitis, or infection.
- Vasodilatation due to spinal shock, epidural anesthesia/analgesia, and sepsis.
- Side effect from medication.
- GI bleeding.
- Pulmonary embolus.
- Abdominal compartment syndrome.
- Excessive airway pressures from mechanical ventilation can directly decrease cardiac ventricular function and decrease venous preload.

- Management.
  - Support cardiovascular system by monitoring end-organ perfusion (urine output, capillary refill) and using four parameters of hemodynamic performance:
    - **Preload** (Best index: pulmonary capillary wedge pressure – PCWP).
    - **Afterload** (systemic vascular resistance [SVR] = [MAP – CVP]/ CO • 80).
    - **Heart rate**.
    - **Cardiac contractility** (best index: stroke volume; SV = CO/h).
    - "Make do" with the best information available—use CVP when PA catheter unavailable.
    - For hypovolemia and cardiovascular instability due to sepsis:
      - Assure adequate preload by volume repletion before adjusting other variables (eg, adding inotropes for low cardiac output).
      - In other states of cardiovascular instability, the variable manipulated is the one indicative of the major problem.

Sinus tachycardia may be a sign of an underlying problem (eg, hypoxia, hypovolemia, infection, or pain). Seek and treat the primary problem, not the tachycardia.

- **Myocardial ischemia/infarction (MI)** is an uncommon battlefield problem.
  - Suspicion is aroused when the patient exhibits angina-like chest pain or unexplained cardiac instability (arrhythmias or hypotension).
  - The diagnosis of an acute myocardial event is made by the presence of ST segment elevation or depression on 12-leak ECG and/or an abnormal elevation of serum markers of myocardial injury (myoglobin [MB] fraction of creatine phosphokinase, Troponin I).
- **Emergency treatment for MI.**
  - Supplemental O_2.
  - Morphine for pain, rest.
  - Aspirin 325 mg tablet chewed and swallowed (then one tablet PO qd).
  - NTG SL (0.4 mg tablet every 5 minutes until pain relieved, maximum 3 doses) or IV infusion depending on severity of condition.
  - Beta-blocker such as metoprolol (5–15 mg IV slowly q6h or 50–100 mg PO q12h) or atenolol (50–100 mg PO) on diagnosis and daily.
  - As resources and patient condition permit for MI with diagnostic ECG: Optimal therapy would also include, within 6 hours of symptoms, IV heparin and a thrombolytic such as tissue plasminogen activator.

Renal System and Electrolytes

- Monitor urine output, blood urea nitrogen (BUN), serum creatinine, and serum electrolytes.
- Acute renal failure (ARF) is manifested by oliguria (< 0.5 cc/kg/h) and a rise in BUN and creatinine. The most frequent causes for ARF are:
  - Hypovolemia.
  - Acute tubular necrosis (ATN) due to:
    - Hypovolemia.
    - Sepsis, IV contrast agents, aminoglycoside antibiotics, or NSAIDs.
  - Crush, massive, soft-tissue injury or compartment syndrome, with resultant rhabdomyolysis and myoglobinuria.

- ♦ In ARF due to rhabdomyolysis, consider administering large volumes of IV fluid (300–800 mL/h), combined with 50 mEq $NaHCO_3$/L, to alkalinize the urine with the goal of achieving a urine output of 2.0 cc/kg/h.
- ♦ Bilateral renal or ureteral trauma.

Two hours of oliguria (< 20cc/h) in an ICU patient (almost always due to inadequate resuscitation) warrants aggressive, immediate action.

- Algorithm for hemodynamically stable ICU patient with profound **oliguria** or **anuria**:
  - o **Irrigate or replace Foley** catheter to ensure function.
  - o After ensuring no signs of intravascular volume overload (diffuse pulmonary crackles, S3 heart sound), administer **bolus** of 1–2 L IV saline over 30 minutes.
  - o Review medication list and medical history to elicit potential factors causing ARF; **stop any agents** that could contribute.
  - o Send any urine to lab with serum sample to calculate fractional excretion of sodium (**FENA**) = $(U_{NA} \bullet P_{CR})/(P_{NA} \bullet U_{CR})$; FENA < 1.0 indicates prerenal cause (eg, hypovolemia); FENA > 2.0 points to renal insult (ATN; myoglobinuria) or postrenal cause (obstruction).
  - o Consider **sonogram** of kidneys to rule out bilateral renal obstruction.
  - o Consider **pulmonary artery catheter** to optimize preload (PCWP).
  - o Once PCWP > 16–18 mm Hg, and urine output minimal/nonexistent, administer **furosemide** in escalating doses IV bolus: 40, 80, 160, 240 mg max (> 100mg = ototoxic). Combine last dose with single dose 1.0 g chlorothiazide IV, or administer 10 mg metolazone PO given 30 minutes before last dose. Also consider furosemide drip or metolazone drip.
- If ineffective or if other complications of ARF occur, arrange for **dialysis** as a temporizing renal support until spontaneous renal recovery occurs. This means the physician must optimize the casualty for transport out of theater, with special attention to volume status, potassium, and acid base status.

- **Indications for dialysis in the casualty with ARF:**
  - Anuria beyond 8–12 hours.
  - Hypervolemia.
  - Hyperkalemia.
  - Acidosis.
  - Complications of uremia: mental status changes, pericardial rub.
  - Toxic levels of drugs/medications (eg, digoxin).

- **Hyperkalemia.**
  - Verify again **hyperkalemia (serum K > 6 mEq/L) and serum pH.**
  - Give IV calcium chloride, 10 mL of 10% solution over 5 minutes.
  - Give IV $NaHCO_3$, 50 mEq over 5 minutes.
  - Give IV Dextrose (50g D50) 50 grams + 10 units regular insulin IV over 10 minutes.
  - Recheck K^+.
  - Give beta-agonist albuterol 10–20 mg over 15 minutes by inhalation.
  - Consider enteral K-binding with enema of sodium polystyrene sulfonate, 25–50 g, in sorbitol.
- **Hypokalemia**: Treatment: 10–20 mEq KCL IV/hour in monitored setting; difficult to treat hypokalemia unless concomitant hypomagnesemia is first corrected.
- **Hypernatremia**: Usually indicative of free water deficit. Water deficit (L) = 0.6 • weight (kg) • [(measured serum Na)/(normal serum Na of 140) – 1]. Half of this deficit should be replaced over first 12–24 hours, and the remainder over the next 1–2 days.
- **Hyponatremia**: Indicative of excess of free water or vasopressin (SIADH). Levels of serum sodium < 125 mEq/L are associated with mental status changes or seizures. Treatment should involve **free water restriction** or use of IV normal saline, with goal of correction of sodium level no more than 15 mEq/L over 24 hours, to prevent complication of central pontine myelinolysis.
- **Hypophosphatemia**: Phosphate is important as an energy source, and should be repleted to level of 2.5 mg/dL with IV KPO_4 or $NaPO_4$, 30 mMol over 1 hour.

- **Hyperphosphatemia** (usually associated with ARF): Phosphate levels over 6.0 mg/dL should be treated by enteral binding agents, such as calcium acetate or sucralfate.
- **Hypomagnesemia**: Administer 2 g magnesium sulfate IV in solution over 60 minutes to goal of serum level of 2.0 mEq/dL.
- **Metabolic acidosis**: Primarily lactic (**most commonly due to hypovolemia**) and ketoacidosis. Neither should be treated with sodium bicarbonate (it is contraindicated in lactic acidosis). **Sodium bicarbonate has very limited role** in ICU disorders: hyperkalemia, alkalinization of urine in myoglobinuria, bicarbonate-responsive renal tubular acidosis (RTA), and for massive gastrointestinal losses of bicarbonate (profound diarrhea, enterocutaneous fistula).
- **Metabolic alkalosis**: NG **suction of stomach acid** causes a hypochloremic alkalosis, responsive to replacement of NG losses with crystalloid. **Excessive loop diuretic use** can also cause metabolic (contraction) alkalosis. If further diuresis is needed, use a carbonic anhydrase inhibitor (acetazolamide 250 mg IV every 6 h) for 1–2 days.

Hematologic System

- Most common coagulation disorder: **dilutional coagulopathy**.
  - Others include heparin-induced thrombocytopenia, disseminated intravascular coagulation, coagulopathy due to hypothermia or diffuse hepatic damage, and thrombocytopenia.
  - Most require replacement transfusion of appropriate blood products.
- To prevent trauma-related deep venous thrombosis (DVT) and pulmonary embolism, prophylactic measures (subcutaneous heparin or sequential compression devices) are required.

Gastrointestinal System and Nutrition

- Prolonged shock can lead to GI dysfunction.
  - **Stress gastritis**: Increased risk of severe head injuries or burns, mechanical ventilation, systemic anticoagulation therapy, or sepsis. Prevention: sucralfate, histamine-2 receptor antagonist (eg, ranitidine) or a proton pump inhibitor (eg, omeprazole).

- **Acalculous cholecystitis**: Suspect with right upper quadrant abdominal pain, abnormalities in liver function tests, or fever/leukocytosis of unclear cause. Ultrasound shows gallbladder inflammation with wall thickening or pericholecystic fluid. Treatment: broad-spectrum antibiotics and ultrasound-guided percutaneous drainage or operation.
- **Hepatic failure** portends a dire prognosis. Initial signs include hyperbilirubinemia, elevation of the prothrombin time, hypoalbuminemia, profound hypoglycemia, obtundation. Massive amounts of fresh frozen plasma are required to prevent exsanguination from coagulopathy.

- **Nutrition** can prove problematic in the battlefield ICU patient.
  - Systemic inflammation induced by severe injury often results in catabolism and protein wasting, making early nutritional support imperative.
  - Nutrition should commence within 24–48 hours of injury.
  - Enteral feedings are superior to parenteral nutrition (TPN), offering a lower infection rate and shorter ICU stays.
  - The following goals serve to guide nutritional management:
    - Caloric requirement: 25–30 kcal/kg/d.
    - Protein requirement: 1.0–1.5 g/kg/d.
    - 30%–40% of total caloric intake per day should be as fat.
  - Nutrition should include a balanced electrolyte solution containing supplemental potassium, calcium, magnesium, phosphate, multivitamins and trace elements (zinc, copper, manganese, and chromium).
  - The two most common problems associated with enteral nutrition are diarrhea and aspiration.
    - Aspiration can be associated with severe pneumonitis, but can be prevented by:
      - Keeping the head of the bed up.
      - Feeding into the jejunum or duodenum rather than the stomach.
      - Checking gastric residuals every 4 hours (feedings should be stopped if residual greater than 200 mL).
    - Diarrhea can be alleviated by:
      - Decreasing the osmolarity of the enteral solution.
      - Adding fiber.
      - Agents such as loperamide in small doses.

Immune System and Infections

- Differential diagnosis of ICU infections.
  - Pneumonia (nosocomial or aspiration).
  - Central venous catheter infection – if considered, remove catheter.
  - UTI.
  - Wound or soft-tissue infection.
  - Intra-abdominal abscess (especially following laparotomy).
  - Systemic fungal infection.
  - Sinusitis.
  - Acalculous cholecystitis.
  - Pancreatitis.
- Prophylactic antibiotics.
  - A short course of prophylactic antibiotics (24–48 h) is warranted after penetrating injury on the battlefield.
  - After this, antibiotics should be withheld unless a documented infection is confirmed, or a severe deterioration in clinical status suggestive of sepsis is encountered.
  - Sepsis warrants a short course of broad spectrum IV antibiotics, but they must be stopped in 72 hours if no microbiologic pathogens are confirmed by culture.
  - **Fever and leukocytosis, by themselves, are not sufficient justification for antibiotics.**

Endocrine System

- Hyperglycemia.
  - Control, to prevent ketoacidosis, hyperosmolar coma, and intravascular volume loss due to osmotic diuresis.
  - The two most common causes are uncontrolled or unrecognized infections and the use of TPN.
  - The best technique for control of hyperglycemia is a constant IV infusion of insulin, usually 1.0–10 units per hour.
    - Due to frequent problems with patient perfusion, subcutaneous injections are less reliable in the ICU patient.
    - Patients with profound hyperglycemia (serum glucose > 800 mg/dL) and volume depletion, due to osmotic diuresis, should receive fluid resuscitation with crystalloid

before receiving insulin, to prevent further shifts in intravascular volume as the glucose shifts intracellularly.
    - ♦ Limit correction rate to 100 mg/dL per hour (700 mg/dL takes 7 h to correct) and assess for resultant hypokalemia.
- Corticosteroids are rarely indicated after major trauma.
    - o There is no proven benefit to steroid treatment for closed head injury or sepsis.
    - o Steroids are indicated for proven adrenocortical deficiency (a rare occurrence among battlefield casualties) and spinal cord injury with neurologic deficit.

Musculoskeletal System

- Monitor for the development of compartment syndrome, vascular ischemia, and rhabdomyolysis.
- Distal extremities should be assessed regularly for neurovascular status: **presence of pulses**, sensation, motor function, warmth, and skin color.

Preparation for Evacuation

- Optimally, the combat casualty will be medically stabilized before transport out of theater.
    - o Native or mechanical airway is maintained.
    - o Sufficient blood pressure, to allow organ perfusion, that has been stable for at least 8 hours.
    - o Both primary and secondary phases of shock resuscitation have been completed.
    - o All sources of bleeding have been identified and controlled.
    - o Life-saving or definitive surgery not required for the next 24 hours.
- Transfer out of a battlefield ICU requires a USAF Critical Care Air Transport Team (CCATT) with physician-to-physician and nurse-to-nurse communication to summarize condition of the patient, operations performed, treatment being given, and support required during flight (in particular, need for oxygen, mechanical ventilation, suction, blood products, and monitoring).
- Copies of medical records, radiographs, 3 days of IV fluid, and all medications should accompany the patient.