Chapter 11

Critical Care

Introduction

The effective application of basic critical care concepts in a timely fashion is vital to the survival of the wounded warrior. At a fundamental level, most of the care required by patients in the combat care environment after a traumatic injury centers around the adequate delivery and utilization of oxygen. An organized system-based approach to care in the intensive care unit should focus on goals of resuscitation and the identification of factors that can threaten these efforts.

Shock/Endpoints of Resuscitation

Shock is an acute physiological state characterized by inadequate oxygen availability to support cellular metabolic needs. **Uncompensated shock** is easily identified at the bedside and is characterized by decreased urine output, altered mental status, hypotension, poor capillary refill, and tachycardia. **Compensated shock** is much more difficult to discern clinically because patients may look normal on examination, but may have organ hypoperfusion that is not appreciated. (See chapter 7 for further information on shock.) Resuscitation is not complete until oxygen delivery (DO_2) and uptake are adequate.

$$\mathbf{DO_2 = CO \times 1.34 \times Hgb \times SaO_2 + 0.0031 \times PaO_2,}$$

where CO = cardiac output, Hgb = hemoglobin, SaO_2 = percentage of oxygen saturation of hemoglobin, and PaO_2 = partial pressure of oxygen in the blood.

Hypovolemic shock is the most common form of shock in the combat casualty care setting and is characterized by decreased intravascular volume (IVV) as its primary abnormality. The resulting decrease in cardiac output leads to diminished DO_2. In

the case of hemorrhage, there is a concomitant decrease in red cell volume that further contributes to inadequate DO_2.

Distributive shock is produced by an inappropriate decrease in systemic vascular tone, leading to an abrupt decrease in mean arterial blood pressure (MAP) to a level that impairs adequate organ perfusion. Neurogenic shock, septic shock, and anaphylactic shock are examples of this process that may be encountered in the combat setting.

Cardiogenic shock results from a primary defect in cardiac output. Myocardial infarction leading to heart wall or valve function abnormalities and cardiac tamponade are classic causes.

Obstructive shock is a process that ultimately results in inadequate cardiac output. Pulmonary embolism (PE) and tension pneumothorax are two common etiologies.

Define Goals of Shock Resuscitation

- MAP > 65 mm Hg.
- Urine output > 0.5 mL/kg/h (in adults).
- Serum lactate <2.

Management of Uncompensated Shock

- Define the type of shock and its etiology; eliminate the cause of the shock if possible.
- Vigorously replete the IVV if MAP or urine output is inadequate.
- Use vasopressor agents to support the MAP after adequate volume restoration.
  - Norepinephrine is the first line agent in most nonhemorrhagic situations.
  - Administer epinephrine in anaphylaxis.
  - Consider dopamine in cardiogenic shock associated with low blood pressure.

Detection of Compensated Shock and Subsequent Management

- Inadequate DO_2 relative to oxygen uptake (VO_2) leads to **increased anaerobic metabolism**.
- Anaerobic metabolism leads to **increased lactate production**.
- Increased lactate may lead to the development of **anion gap metabolic acidosis**.

- An **increased base deficit > 10 mEq/L** suggests inadequate resuscitation.
- Central venous oxygen saturation ($ScvO_2$) **< 65% suggests inadequate resuscitation**.
  - The body should use <25%–35% of oxygen delivered.
  - Increased utilization by cells suggests inadequate DO_2.
  - $ScvO_2$ < 65% suggests inadequate DO_2 and an implied need to optimize SaO_2, hemoglobin, or cardiac output.
    - Optimize SaO_2 and IVV.
    - Consider transfusion > 10 mg/dL.
    - Consider inotropic therapy.

Fluid Management

Intravenous fluids are given to patients to either replete a deficit in IVV or prevent the development of such a deficit in a patient unable to accomplish these goals without assistance. The choice of fluid depends on which of these goals is being addressed and the overall clinical context.

- Total body sodium is directly proportional to extracellular fluid volume (ECFV).
- IVV generally represents 15%–20% of ECFV.
- IVV repletion, therefore, is dependent on sodium infusion.
  - **Lactated Ringer (LR) solution: 130 mEq/L sodium, pH** 5.5–6.0.
  - **0.9% normal saline (NS): 154 mEq/L sodium and chloride, pH 4.5–5.5.**
- In most clinical contexts, colloid infusion confers no benefit during resuscitation relative to isotonic crystalloid solutions, such as LR and NS.
  - However, equivalent IVV repletion can be accomplished using lower volumes of colloid solutions.
- A nonanion gap metabolic acidosis frequently results from the use of large volumes of NS during resuscitation; continued resuscitation can be then accomplished using other isotonic fluid combinations.
  - 0.5 L of ½ NS with 75 mEq sodium bicarbonate ($NaHCO_3$): approximately 152 mEq/L sodium.
  - 1 L of D5W (5% dextrose in water) with 150 mEq $NaHCO_3$: approximately 150 mEq/L sodium.

Special Fluid Considerations

- **Hypertonic saline** should be considered in patients with traumatic brain injury (TBI).
- ½ NS (±D5 [or 5% dextrose]) should be used for maintenance of IVV to counteract insensible losses.
- ½ NS (±D5) can be used to replete IVV for the rare patient with both hypernatremia and IVV depletion (postosmotic diuresis, etc).
- **Albumin** should be considered in the following patients:
  - Complicated burn resuscitation expected to result in hourly IV fluid rate exceeding 1,500 mL/h or if the projected 24-h total fluid volume approaches 250 mL/kg (see Burn CPG, March 2016).
    - ♦ Refer to Chapter 26, Burns, for further guidance.
  - Severely malnourished patients with serum albumin concentration <1.0.
  - Cirrhotic patients who present with spontaneous bacterial peritonitis.

Serum Electrolyte Management

Serum sodium management depends primarily on the recognition that the serum sodium concentration is not necessarily indicative of IVV status. Although IVV is directly proportional to ECFV and, therefore, total body sodium, abnormal serum sodium concentrations usually represent abnormalities in free water handling. Notable exceptions include hypovolemic hyponatremia (diuretics, etc) and hypervolemic hypernatremia (hypertonic saline administration, etc). Two key questions are important to consider in all patients with an abnormal serum sodium:

- **What is the IVV status of the patient?**
- **Is there free water excess (hyponatremia) or deficit (hypernatremia)?**

Hyponatremia (Na < 135 mEq/L)

- Euvolemic hyponatremia.
  - **Differential diagnosis (Ddx):** Antidiuretic hormone (ADH) release (syndrome of inappropriate ADH, pain, anxiety), adrenal insufficiency, hypothyroidism, and severe polydipsia.

- **Management:** Free water restriction, correct underlying cause.

- Hypovolemic hyponatremia.
  - **Ddx:** Diuretic use, cerebral salt wasting.
  - **Management:** IVV repletion with NS.
- Hypervolemic hyponatremia.
  - **Ddx:** Severe congestive heart failure (CHF), cirrhosis, or renal failure.
  - **Management:** Treat underlying condition; consider diuretic use.
- Relative "salt deficit" (mEq Na) = 0.6 × weight in kg × (140 – Na). Free water restriction for euvolemic and hypervolemic hyponatremia. 3% saline (5-6 mL/kg) infusion for central nervous system (CNS) symptoms (mental status changes, seizures, etc).

Rate of serum sodium correction should be <0.5 mEq/L/h and <10 mEq/L/24 h to prevent osmotic demyelination syndrome (formerly known as central pontine myelinolysis).

Hypernatremia (Na > 145 mEq/L)

- Euvolemic hypernatremia.
  - **Ddx:** Same as hypovolemic hypernatremia.
  - **Management:** Treat underlying cause, free water repletion.
- Hypovolemic hypernatremia.
  - **Ddx:** Renal water loss (osmotic diuresis [mannitol, hyperglycemia, etc]), impaired thirst/water intake, and central/nephrogenic diabetes insipidus.
  - **Management:** Treat underlying cause, replete IVV, and free water repletion.
- Hypervolemic hypernatremia.
  - **Ddx:** Iatrogenic (hypertonic saline administration).
  - **Management:** Discontinue NS infusion; free water repletion.
- Relative "free water excess" (in liters) = 0.6 × weight in kg × (Na – 140)/140.
  - Rate of serum sodium correction should be <0.5 mEq/L/h and <10 mEq/L/24 h.

Serum potassium concentration is frequently abnormal in critically ill patients. Similar to the case with serum sodium concentration disorders, the serum potassium level may not be indicative of total body potassium stores. In the case of potassium, the vast majority is contained in the intracellular fluid volume (ICFV) space, and only a small portion is found in the ECFV or intravascular spaces. Potassium shifts back and forth between the ECFV and ICFV with relative ease, leading to potentially large swings in serum concentrations. Total body potassium may be quickly depleted if lost through renal or nonrenal (diarrhea, sweat, and fasting) excretion.

Hypokalemia (K < 3.5 mEq/L)

Serum hypokalemia may be secondary to **redistribution of potassium** from the ECFV to the ICFV, as is commonly seen with significant alkalemia or increased beta-2 agonist utilization. Total body potassium **depletion** may also lead to a decrease in serum potassium concentration through increased renal losses (diuretic use, postobstructive diuresis, osmotic diuresis, alkalosis, and proximal/distal renal tubular acidoses) and nonrenal mechanisms.

- EKG changes consistent with hypokalemia include prominent U waves and T-wave flattening.
  - Clinically this can manifest as paralysis, respiratory muscle dysfunction, and rhabdomyolysis.
- Enteral supplementation is preferred when the patient is clinically stable because it is both safer and results in faster repletion relative to IV infusion.
- IV infusion rates are limited to 10 mEq/h through a peripheral IV and 20–40 mEq/h through a central line. In general, 10 mEq of potassium repletion increases serum levels by 0.1 mEq. **IV potassium replacement at rate >10 mEq/L/h requires continuous cardiovascular monitoring.**
- Use KCl for replacement in most situations; potassium citrate or potassium bicarbonate is more appropriate when hypokalemia is associated with metabolic acidosis (especially renal tubular acidosis).
- Oral repletion: KCl elixir or tablet 30–60 mEq qid until serum potassium concentration is normal.

- Emergent IV repletion: KCl via a central line 20–40 mEq/h until potassium > 3.0 mEq/L, then switch to oral as above or a lower infusion rate of 10–20 mEq/h until serum concentration is normal.
  - Avoid dextrose-containing solutions because the subsequent insulin release will cause intracellular redistribution of potassium (K), further complicating repletion efforts.
- If adequate potassium replacement does not result in appropriate rise in serum potassium, serum magnesium levels should be assessed and repleted

Hyperkalemia (K > 5.5 mEq/L)

- Hyperkalemia may present as a result of several different mechanisms. **Pseudohyperkalemia, or falsely elevated K,** is iatrogenic, arising from improper venipuncture (drawing directly from the IV line with LR) or hemolysis of specimen. The draw should be repeated if the lab value is not clinically correlated. An EKG should be obtained to assess for evidence of myocardial excitation, which may manifest in the following sequence:
  - Peaked T waves, flattened P waves, and prolonged PR interval.
- In more severe cases, this sequence may be followed by:
  - Idioventricular rhythm, widened QRS interval, sine wave pattern, and ventricular fibrillation.

Redistribution hyperkalemia is seen in the trauma critical care setting most frequently as a result of acidemia, succinylcholine utilization, or hypertonic states (hypertonic saline or mannitol use). Finally, hyperkalemia may result from **renal failure, hypoaldosteronism, and medications (salt substitutes and exogenous potassium supplementation).**

- Chronic hyperkalemia is better tolerated than the acute condition.
- Acute hyperkalemia should be regarded as a life-threatening medical emergency.
- Treatment options for hyperkalemia include:
  - 10 mL of 10% calcium chloride if central venous access is available (standard calcium chloride ampule) over 1–3 minutes; can repeat every 5 minutes, as long as severe EKG

changes persist. If peripheral access only, infuse 10 mL of 10% calcium gluconate over 1–3 minutes. **Remember, this will not alter the serum potassium level but will stabilize the myocardium and should be performed immediately upon recognition.**
  - 50 mEq of $NaHCO_3$ (1 standard ampule of a 7.5% $NaHCO_3$ solution). Repeat every 30 minutes until QRS is improved; often ineffective in renal failure.
  - Consider dialysis if QRS widening is present.
- Treatment with mild EKG changes (no evidence of QRS widening):
  - Beta-2 agonists (albuterol) 20 mg in 4 mL of saline nebulizer.
  - 50 mL of 50% dextrose/glucose, 10 U of regular insulin; follow glucose, repeat until EKG returns to baseline.
  - Loop or thiazide diuretic—use only in patients known to be intravascularly replete; will be ineffective in anuric renal failure.
  - Sodium polystyrene sulfonate (Kayexalate) 20 g orally every 6 hours or 50 g as an enema every 2–4 hours.
- Treatment with normal EKG consists of identification and correction of the cause, as well as 15 g of sodium polystyrene sulfonate (Kayexalate) orally every 6 hours or 30–60 g as an enema every 2–4 hours.
  - Intestinal necrosis can result, especially when given orally within a week of major surgery.

Serum magnesium is often not given significant priority in the care of the critical care patient. Similar to K, serum magnesium represents only a fraction of the total body stores. **Low serum magnesium levels indicate severe total body magnesium deficits. Normal serum magnesium levels do not correlate reliably with total body magnesium stores.**

Hypomagnesemia (Mg < 2.0 mEq/L)
Hypomagnesemia usually results from **inadequate intake (NPO status, malnutrition prior to admission)** or **excessive loss, usually via renal mechanisms (diuretics, osmotic diuresis).**
- Magnesium < 1.0 mEq/L may be associated with CNS excitability and torsades de pointes on EKG.

- Establishing and correcting the cause of hypomagnesemia is the ultimate key to the management of this disorder.
- Total body magnesium depletion (with or without serum hypomagnesemia) is frequently associated with both hypokalemia and hypocalcemia.
  - Successful repletion of potassium and calcium will not generally be possible until total body magnesium stores have been normalized.
- In the absence of CNS excitability or life-threatening hypokalemia or hypocalcemia, magnesium repletion should be given as 4 g magnesium sulfate IV every 24 hours for 72 hours before serum magnesium levels are rechecked.
- If CNS excitability or life-threatening hypokalemia or hypocalcemia is present, 2 g of magnesium should be given as an immediate IV push, followed by 4–6 g in 6 hours, and followed by 4–6 g IV each day for the next 2–3 days.
- Checking serum magnesium levels during repletion is not useful because mildly elevated magnesium levels do not indicate successful total body repletion, and clinically significant hypermagnesemia is not seen with the aforementioned rates of repletion unless severe renal failure exists.

Serum calcium disorders are seen frequently in the combat critical care setting. Hypocalcemia is seen with much greater frequency than hypercalcemia and will be given greater emphasis here. Serum calcium levels are often corrected for serum albumin levels because negatively charged proteins, such as albumin, bind positively charged calcium cations. Ionized calcium is the physiologically relevant portion of total calcium. Adjusting total calcium for measured albumin values is useful only if a measurement of ionized calcium is not available. In the combat casualty care setting, ionized calcium measurements can be obtained quickly using handheld point-of-care testing devices, such as the i-STAT Blood Gas Analyzer (with an EG7+ or EG8+ cartridge).

Hypocalcemia (iCa < 1.10)
Hypocalcemia in the combat setting is seen most frequently **after massive blood product transfusion** (calcium is bound by citrate used as an anticoagulant) or as a result of **associated total body hypomagnesemia**. QT interval prolongation can result from severe hypocalcemia, and its presence dictates the pace of repletion.

- 10% calcium chloride 10 mL vial contains 272 mg of elemental calcium.
- 10% calcium gluconate 10 mL vial contains 93 mg of elemental calcium.
- Administer one 10 mL vial of 10% calcium chloride in 50–100 mL of D5 in water for >10–15 minutes if QT prolongation is noted.
  - Follow this with 1–2 mEq/h of elemental calcium infusion until QT prolongation has been resolved or >1.00–1.10 g of calcium are corrected to within normal range. Supplementation should be performed via a central line, given the risk of venous thrombosis and subsequent tissue necrosis.
- Hypocalcemic patients without QT prolongation can be repleted as follows:
  - Oral supplementation of 1.5–2.5 g of elemental calcium per day.
  - If oral supplementation is not possible, initiate an infusion of 0.5 mg/kg/h of elemental calcium >1.10.
- If hypocalcemia is difficult to correct, consider total body magnesium depletion (with or without serum hypomagnesemia).

Pulmonary Medicine

Basics of Mechanical Ventilation
Patients are placed on invasive mechanical ventilation most commonly for airway protection, respiratory failure (hypoxemia), or ventilatory failure (hypercapnia leading to acidemia). Another relatively common indication is in the setting of shock to optimize DO_2. **Compliance** of the chest wall/lung unit is defined by the change in volume resulting from a change in pressure.

Volume control modes of ventilation (assist-control [A/C], synchronized intermittent mandatory ventilation [SIMV]) provide mandatory breaths at a specified volume.

Pressure control modes of ventilation (pressure control ventilation) provide mandatory breaths to a set pressure.

Ventilation (elimination of CO_2) is necessary to achieve a target $PaCO_2$ of 35–45.

- **$PaCO_2$ is regulated by altering respiratory rate (RR) or tidal volume (V_T) in order to change the minute volume (Ve).**

Oxygenation/respiration is necessary to support adequate DO_2 to the patient. Goal SaO_2 is 92%. **There is generally little physiological benefit from attempting to manipulate the ventilator to achieve values higher than 92%.**

Using positive pressure ventilation, increased oxygenation/respiration occurs by increasing the fraction of inspired oxygen (FiO_2) or increasing the mean airway pressure (positive end-expiratory pressure [PEEP]).

- A low PaO_2/FiO_2 (<300), in the absence of very severe hypercapnia, suggests shunt physiology as the most likely cause of hypoxemia in a patient.
- Increased mean airway pressure may be a useful adjunct (increase the PEEP).
- FiO_2 manipulation alone will be unlikely to correct hypoxemia in this setting.

Initial ventilator settings for most patients should **strive to optimize oxygenation and ventilation while at the same time serve to minimize barotrauma** (pneumothorax, subcutaneous emphysema, etc, due to excessive transalveolar pressures); **volutrauma** (lung damage due to excessive stretch); **atelectotrauma** (lung damage due to repetitive opening and closing of alveoli); and **biotrauma** (release of cytokines related to the application of positive pressure ventilation).

Mode: Volume Cycled (A/C or SIMV)

- SIMV is not recommended in the acute setting because it is associated with increased work of breathing when used for prolonged periods.

- When SIMV is used, it is best to use pressure support ventilation to augment any spontaneous breaths.
  - The standard military transport ventilator (Impact 754) does not allow pressure support ventilation to be used when the SIMV mode is used.
- FiO_2 = 100%; titrate down to lowest amount to keep SpO_2 or SaO_2 > 92%.
  - SaO_2 = saturation of hemoglobin as measured by arterial blood gas sampling.
  - SpO_2 = noninvasive pulse oximetry; a rough estimate of SaO_2.
- V_T = 5–7 mL/kg ideal body weight.
  - Ideal predicted body weight in kilograms in males = 50 + 2.3 × (height in inches – 60).
  - Ideal predicted body weight in females = 45.5 + 2.3 × (height in inches – 60).
  - Adjust to keep <8 mL/kg and plateau pressures < 30 cm H_2O.
- RR = 16.
  - Adjust to keep RR × V_T adequate to manipulate $PaCO_2$ to achieve goal pH.
- Inspiration:expiration (I:E) ratio = 1:2 to 1:3.
- PEEP = 5 cm H_2O.
  - Increase PEEP if PaO_2/FiO_2 < 300 (shunt physiology expected).
  - Increase PEEP to 10–12 cm H_2O if shunt physiology present.
    - Increase as necessary above this level to keep SpO_2 > 92%.
    - With increased PEEP, V_T may need to be decreased to keep plateau pressures < 30 cm H_2O.

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) can be caused by direct (inhaled toxins, aspiration) or indirect (trauma, burns, any cause of systemic inflammatory response syndrome) mechanisms, but the basic management is similar. To meet the definition, the following criteria must be met:

- Acute presentation of hypoxemic respiratory failure.
- Bilateral infiltrates on chest radiography.

- No clinical evidence of left heart volume overload; pulmonary capillary wedge pressure < 18 mm Hg if measured.

The severity of ARDS is determined by the PaO_2/FiO_2 (Berlin definition):

- **Mild ARDS**. The PaO_2/FiO_2 is >200 mm Hg, but ≤300 mm Hg, on ventilator settings that include PEEP or continuous positive airway pressure (CPAP) ≥5 cm H_2O.
- **Moderate ARDS.** The PaO_2/FiO_2 is >100 mm Hg, but ≤200 mm Hg, on ventilator settings that include PEEP ≥5 cm H_2O.
- **Severe ARDS.** The PaO_2/FiO_2 is ≤100 mm Hg on ventilator settings that include PEEP ≥5 cm H_2O.

Basic ventilatory strategies are designed to minimize barotrauma by avoiding excessive alveolar pressures, volutrauma by limiting delivered V_T, and atelectotrauma by keeping alveoli open using increased mean airway pressure ventilator strategies. A ventilator strategy encompassing these features was found by the ARDSNet investigators to lead to an improved mortality and should be followed where possible (Table 11-1).

Table 11-1. Mechanical Ventilation Protocol Summary

INCLUSION CRITERIA
Acute onset of the following:
1. PaO_2/FiO_2 ≤ 300 (corrected for altitude).
2. Bilateral (patchy, diffuse, or homogeneous) infiltrates consistent with pulmonary edema.
3. No clinical evidence of left atrial hypertension.

PART I: VENTILATOR SETUP AND ADJUSTMENT
1. Calculate Ideal Body Weight (IBW).
   Males = 50 + 2.3 (height [inches] – 60).
   Females = 45.5 + 2.3 (height [inches] – 60).
2. Select any ventilator mode.
3. Set ventilator settings to achieve initial V_T = 8 mL/kg IBW.
4. Reduce V_T by 1 mL/kg at intervals ≤2 hours until V_T = 6 mL/kg IBW.
5. Set initial rate to approximate baseline minute ventilation (not >35 bpm).
6. Adjust V_T and RR to achieve pH and plateau pressure goals below.

(**Table 11-1** *continues*)

(**Table 11-1** *continued*)

Oxygenation Goal: PaO_2, 55–80 mm Hg or SaO_2, 88%–95%
Use a minimum PEEP of 5 cm H_2O. Consider use of incremental FiO_2/PEEP combinations, such as shown below (not required) to achieve goal.

Lower PEEP/Higher FiO_2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12
FiO_2	0.7	0.8	0.9	0.9	0.9	1.0		
PEEP	14	14	14	16	18	18–24		

Higher PEEP/Lower FiO_2

FiO_2	**0.3**	**0.3**	**0.3**	**0.3**	**0.3**	**0.4**	**0.4**	**0.5**
PEEP	**5**	**8**	**10**	**12**	**14**	**14**	**16**	**16**
FiO_2	**0.5**	**0.5–0.8**	**0.8**	**0.9**	**1.0**	**1.0**		
PEEP	**18**	**20**	**22**	**22**	**22**	**24**		

Plateau Pressure Goal: ≤30 cm H_2O
Check Pplat (0.5-second inspiratory pause), at least q4h and after each change in PEEP or V_T.
- **If Pplat > 30 cm H_2O:** decrease V_T by 1 mL/kg steps (minimum = 4 mL/kg).
- **If Pplat < 25 cm H_2O and V_T < 6 mL/kg,** increase V_T by 1 mL/kg until Pplat > 23 cm H_2O or V_T = 6 mL/kg.
- **If Pplat < 30 and breath stacking or dyssynchrony occurs:** may increase V_T in 1 mL/kg increments to 7 or 8 mL/kg if Pplat remains ≤30 cm H_2O.

pH Goal: 7.30–7.45

Acidosis management: pH < 7.30.
(Consider other causes of acidemia, eg, hemorrhage).
- **If pH 7.15–7.30:** Increase RR until pH > 7.30 or $PaCO_2$ < 25.
  - Maximum set RR = 35.
- **If pH < 7.15:** Increase RR to 35.
  - If pH remains <7.15, V_T may be increased in 1 mL/kg steps until pH >7.15 (Pplat target of 30 may be exceeded).
  - May give $NaHCO_3$.

Alkalosis management: pH > 7.45 (decrease vent rate, if possible)

I:E: Ratio Goal
Recommend I:E = 1:2–1: 3

(**Table 11-1** *continues*)

(**Table 11-1** *continued*)

PART II: WEANING

A. Conduct a Daily Spontaneous Breathing Trial When:

- The cause of the respiratory failure has improved.
- The patient is oxygenating adequately.
- The arterial pH is >7.25.
- The patient is able to initiate an inspiratory effort.
- The patient is hemodynamically stable, without myocardial ischemia.

B. Spontaneous Breathing Trial

If all of the above criteria are met and the subject has been in the study for at least 12 hours, initiate a trial of UP TO 120 minutes of spontaneous breathing with $FiO_2 \leq 0.5$ and PEEP ≤ 5:

1. Place on T-piece, trach collar, or CPAP ≤ 5 cm H_2O with PS ≤ 5.
2. Assess for tolerance as below for up to 2 hours.
   a. $SpO_2 \geq 90$; and/or $PaO_2 \geq 60$ mm Hg.
   b. Spontaneous $V_T \geq 4$ mL/kg IBW.
   c. RR < 35/min.
   d. pH ≥ 7.3.
   e. No respiratory distress (distress = 2 or more).
      i. HR > 120% of baseline.
      ii. Marked accessory muscle use.
      iii. Abdominal paradox.
      iv. Diaphoresis.
      v. Marked dyspnea.
3. If tolerated for at least 30 minutes, consider extubation. A rapid shallow breathing index (RSBI = RR/ TV in liters) <105 has been proven to correlate with successful extubation.
4. If not tolerated, resume preweaning settings.

ARDS: acute respiratory distress syndrome; BP: blood pressure; bpm: breaths per minute; CPAP: continuous positive airway pressure; FiO_2: inspired oxygen; HR: heart rate; I:E: inspiration:expiration; IMV: intermittent mandatory ventilation; $NaHCO_3$: sodium bicarbonate; $PaCO_2$: partial arterial gas pressure (tension) of carbon dioxide; PaO_2: partial pressure of oxygen in the blood; PBW: predicted body weight; PEEP: positive end-expiratory pressure; Pplat: plateau pressure; PS: pressure support; q4h: every 4 hours; RR: respiratory rate; SpO_2: noninvasive pulse oximetry; trach collar: tracheostomy collar; V_T: tidal volume. Reprinted with permission and with minor changes from the ARDS Clinical Network website (www.ardsnet.org) and the National Institutes of Health and the National Heart, Lung, and Blood Institute.

Adjunctive therapies for ARDS have been studied for decades and have been demonstrated to have variable clinical benefit. Each can be considered in a given patient depending on the clinical scenario and availability of resources.

- High (>16 cm H_2O) vs moderate (10–16 cm H_2O) PEEP.
  - Possible benefit using higher levels in patients with more severe hypoxemia.
- Prone positioning.
  - Improves oxygenation in patients with severe hypoxemia.
  - No definitive mortality benefit.
  - Can be accomplished with a Stryker frame in the combat support setting.
- Conservative IVV management.
  - Improved outcomes relative to liberal strategy, as tolerated by physiology and injury pattern of the patient in question.
- Pulmonary artery catheter vs central venous pressure monitoring.
  - No benefit to using a pulmonary artery catheter to guide fluid management.
- Special dietary formulations.
  - No single proprietary formula has been demonstrated to improve outcomes.
- Corticosteroids.
  - Not recommended at this time
- Inhaled nitric oxide (if available).
  - Improved oxygenation noted.
  - No mortality benefit.
- Pressure control ventilation.
  - No significant outcomes benefit relative to volume control A/C mode.
  - If used, efforts must be made to continue to limit V_T as outlined in the ARDSNet protocol.
- Airway pressure release ventilation.
  - No significant outcomes benefit relative to volume control A/C mode.
  - Equivalent mean airway pressures can be obtained using lower amounts of sedation, and patients are less likely to require neuromuscular blockade.
  - If used, efforts must be made to continue to limit V_T as outlined in the ARDSNet protocol.
- High-frequency oscillatory ventilation.
  - No benefit to standard of care demonstrated in the 1990s.

  - Has not been directly compared with ARDSNet low V_T strategy.
  - Technology and expertise unlikely to be available in combat support operations.
- Extracorporeal membrane oxygenation.
  - Improved oxygenation.
  - No mortality benefit.
  - Technology and expertise unlikely to be available in combat support operations.
- Extracorporeal carbon dioxide removal.
  - Maybe a useful adjunct when carbon dioxide elimination is severely limited.
  - Has not been directly compared with ARDSNet low V_T strategy.

Pulmonary Contusion

Pulmonary contusion is frequently seen in the combat setting, most commonly associated with blunt trauma with or without rib fractures. The disorder is similar to ARDS, in that it may present with a significant degree of hypoxemia and decreased compliance. A significant distinction between the two clinical syndromes is the profoundly asymmetric nature of pulmonary contusion. **Excessive mean airway pressure delivery may lead to overdistension of healthy lung, which has the effect of shunting blood away from well-ventilated alveoli (increasing dead space fraction) and toward poorly ventilated contused regions (increasing shunt). If an increase in PEEP is associated with a significant fall in oxygen saturation, an increase in shunt physiology due to excessive mean airway pressure should be suspected, and PEEP should be decreased to its previous level.** Pulmonary contusion is generally managed in a supportive fashion using a low V_T strategy combined with aggressive pulmonary toilet.

Pulmonary Embolism

PE is part of a broader disease process known as venous thromboembolic disease that includes deep venous thrombosis (DVT). DVT is very common in the trauma setting, and may be a life-threatening if accompanied by PE.

Diagnosis of DVT

- Determine pretest clinical suspicion.
- If low, do not work up further.
- If moderate or high, perform duplex ultrasonography.
- If clinical suspicion is high, but ultrasonography is negative, consider empiric treatment with further testing at a higher level of care.
- **Treatment of DVT.**
  - Low molecular weight heparin (Lovenox, 1 mg/kg subcutaneously bid) or unfractionated heparin (initial bolus, 80 units/kg; followed by 18 units/kg/h with goal PTT 55–85).
  - Consider removable inferior vena cava filter placement if there is a contraindication to anticoagulation. Examples of contraindications to anticoagulation common to the combat casualty include TBI, solid visceral injury, and pelvic fracture.

Hemodynamically Significant PE

The majority of patients who die from PE die of right heart failure associated with acute pulmonary hypertension rather than hypoxemia. A high pretest clinical suspicion for PE in the setting of hypotension and evidence of right heart failure on exam should be considered a medical emergency. Patient instability may preclude making a formal diagnosis. In this case a bedside transthoracic echocardiogram can be obtained to assess for right heart failure. If present, the following should be considered:

- Start therapy immediately with low molecular weight heparin or unfractionated heparin.
  - The risks of starting anticoagulation should be carefully weighed in the multisystem trauma patient.
  - Protamine (1–1.5 mg protamine per 100 units heparin) can be used to reverse the effects of low molecular weight heparin, although dosing may be more difficult to predict than when used to reverse the effects of unfractionated heparin. **Note:** When heparin is given as a continuous IV infusion, only heparin given in the preceding 2 to 3 hours should be considered when administering protamine.
  - Do not give fluid boluses for hypotension if significant evidence of right heart failure exists.

- Support blood pressure (MAP > 60 mm Hg, DBP > 40–45 mm Hg) using epinephrine or dopamine.
- Norepinephrine is also acceptable, although reflex bradycardia may be seen.
- Consider the addition of Milrinone or Dobutamine if persistent shock noted.
  - Milrinone may be a superior choice due to an improved ability to directly lower pulmonary vascular resistance.
  - Consider the use of thrombolytic therapy if hypotension is persistent or cardiopulmonary arrest develops.

Prevention of Venous Thromboembolism

Given the high risk of venous thromboembolism complications associated with multisystem trauma patients (especially those with orthopedic and spine injuries), prevention remains the key to avoiding adverse consequences.

- All trauma patients should receive chemical prophylaxis for venous thromboembolism disease unless contraindicated (eg, TBI).
  - Low molecular weight heparin (Lovenox 30 mg subcutaneously bid) should be administered.
  - Highest-risk patients (spine injury, expected prolonged immobilization, and orthopedic injury) should also have intermittent pneumatic compression device therapy initiated.
- Trauma patients with clinical contraindications to chemical prophylaxis should be considered for IVC filter placement

Aspiration Pneumonitis

Patients with compromised pulmonary status secondary to aspiration should be managed supportively, with positive pressure ventilation and a lung protective strategy as described previously in this chapter. **Empiric antibiotics are NOT indicated for isolated aspiration.** Witnessed or clinically suspected aspiration usually results in a chemical pneumonitis and does not commonly lead to an infectious pneumonia. Aspiration pneumonitis generally presents with an infiltrate in a dependent portion of the lungs (especially the right lower lobe, left lower lobe, or the superior segments of the right or left upper lobes) and may be associated with a fever, moderate leukocytosis, worsening

oxygenation, and evidence of consolidation on physical exam. Failure to improve after 24–48 hours should initiate investigation for a secondary bacterial pneumonia infectious process. Cultures should be obtained before initiating empiric therapy with broad-spectrum antibiotics (see Chapter 10, Infections). Antibiotics should be stopped at 72 hours if cultures do not demonstrate a dominant organism. Duration of antibiotic therapy should be limited to 5–7 days. Bronchoscopy should be performed in cases of suspected foreign body aspiration (teeth, etc).

Combat-Associated Healthcare Pneumonia

Combat-associated healthcare pneumonia denotes a hospital-acquired pneumonia that is contracted while being treated in a combat medical facility. The distinction is important, because many combat medical facilities in Iraq and Afghanistan are associated with increased rates of patient colonization with multidrug-resistant bacteria. Patients who develop pneumonia after being in the combat medical system for at least 72 hours should be considered to be colonized with multidrug-resistant organisms, and empiric therapy should include vancomycin plus meropenem, doripenem, piperacillin/tazobactam, or cefepime. Ertapenem is not recommended due to poor coverage of *Pseudomonas aeruginosa*.

Cardiac Considerations

Cardiac Tamponade

Acute cardiac tamponade may occur as a result of either blunt or penetrating thoracic trauma and is a surgical emergency. Hemodynamically significant pericardial effusions may result from small volume collections of blood that result in a decreased ejection fraction. **Pericardial fluid in the setting of trauma requires immediate surgical evaluation**. Tamponade may be subtle, but cardiovascular collapse can quickly develop.

- Beck's Triad suggests the diagnosis of cardiac tamponade.
  - Hypotension, jugular venous distention, muffled heart sounds.
- The diagnosis can be confirmed with transthoracic echocardiogram.

- Assessment of cardiac enzymes has no role in the diagnosis of cardiac tamponade.
- Urgent pericardial drainage is necessary. In the setting of trauma, emergent pericardiocentesis may be performed as a temporizing measure in the absence of immediately available surgical care.
  - A needle is inserted subxyphoid at an angle of 20–30 degrees, and directed toward the left nipple. The needle should be aspirated as it is advanced. Ultrasound can be used to assist, if available.
- IVV may need to be aggressively supported to ensure adequate cardiac filling.
- Proximal aortic dissection should be strongly considered in patients with blunt trauma who develop acute cardiac tamponade.

Blunt Cardiac Injury

Blunt cardiac injury presents as a clinical consequence of blunt thoracic trauma in the combat setting. It is likely underdiagnosed because the vast majority of patients are asyptomatic, and significant consequences are uncommon. Diagnosis is suspected with PVCs or sinus tachycardia. If EKG abnormalities exist, cardiac enzymes are drawn to confirm the diagnosis. The patient should be managed in a monitored setting. A transthoracic echocardiogram should be obtained to assess for mechanical dysfunction (severe acute valve regurgitation, free wall rupture, and ventricular septal wall rupture).

Acute Coronary Syndrome

ST elevation myocardial infarction (STEMI) results from the occlusion of coronary vessels by an unstable plaque. This results in transmural cardiac muscle death. Management centers on revascularization, decreasing cardiac oxygen requirements, and monitoring closely for the development of mechanical complications, CHF, and potentially lethal arrhythmias such as ventricular tachycardia and fibrillation.

- Aspirin 81 mg PO, chewed as quickly as possible and daily thereafter.

- Beta blocker (Lopressor 5 mg IV initially) if no evidence of acute CHF. American Heart Association guidelines (Lopressor 5 mg IV incrementally or Esmolol drip) to target heart rate < 60–70 and SBP < 110.
- If heart rate target met with beta blocker, but SBP is >110, consider the following adjuncts:
  - Nitroglycerin gtt (dose may be limited by headache or the presence of right-sided disease).
  - Nicardipine gtt.
  - Nitroprusside gtt.
- Plavix 300 mg load followed by 75 mg PO daily.
- A glycoprotein 2B/3A inhibitor (Eptifibatide) should be considered.
- Supplemental oxygen to maintain SpO_2 > 96%–98%.
- Sublingual nitroglycerin (spray or tablet) as necessary for pain.
  - Rapid hypotension development with nitroglycerin suggests right-sided disease.
- Morphine IV as necessary for pain.
- Thrombolytic therapy (Tenecteplase, Reteplase) should be given ideally within 3 hours; 12 hours is acceptable.
- Cardiac catheterization is favored over thrombolytic therapy if available.
- **If evidence of CHF:**
  - Start nitroglycerin gtt.
  - Lasix q6h IV versus gtt to affect diuresis/preload reduction.
  - Consider nicardipine versus nitroprusside gtt to titrate blood pressure/afterload reduction, except in the setting of preload dependent inferior wall myocardial infarction.
  - Dopamine or Milrinone can be considered if SBP < 90.
  - Dobutamine can be considered; however, this agent will increase myocardial oxygen demand.
  - Aortic balloon pump is favored in this setting, if available.
- Continuous cardiac and hemodynamic monitoring (arterial line, central venous catheter with central venous pressure monitoring) should be continued until transfer to a higher level of care.
- An ACE (angiotensin-converting enzyme) inhibitor should be started within 24 hours of the index symptoms.
- A statin medication should be started as soon as possible.

Non-STEMI (NSTEMI) and unstable angina are closely related processes when cardiac demand exceeds oxygen supply. It should be regarded as a medical emergency. NSTEMI and unstable angina are physiologically the same process and are only distinguished by the presence of myocardial damage, as evidenced by cardiac enzyme elevation, in the setting of NSTEMI. Management is similar to STEMI; however, fibrinolytics play a less prominent role, and antiplatelet therapy plays a more prominent role due to the relative predominance of platelets over fibrin in coronary vessel clot associated with NSTEMI/unstable angina. Goals remain to improve coronary blood flow, decrease myocardial oxygen demand, and monitor for complications of the disease process. Progression to STEMI needs to be carefully monitored. Therapy should be directed as outlined for STEMI (see above).

Congestive Heart Failure

CHF is a clinical diagnosis in which cardiac output is inadequate relative to preload. Clinical signs and symptoms reflect left-sided heart failure (pulmonary edema, pleural effusions), as well as right-sided failure (jugular venous distention, dependent edema, liver and spleen engorgement). Systolic and diastolic dysfunction can both cause CHF when IVV is excessive (eg, acute or chronic valvular dysfunction). Goals of CHF management include **preload reduction, afterload reduction, and improved inotropic function**.

Preload Reduction

- Diuretic therapy.
  - Loop diuretic (Furosemide, Bumetanide).
    - Consider IV therapy for severe CHF; continuous gtt for refractory CHF.
  - Minimize salt intake (extracellular fluid volume is directly proportional to total body salt).
  - Total salt intake should be <1.5–2.0 g/d.
- Nitroglycerin drip.
  - Vasodilates venous system.
- Nitroprusside drip.
  - Relatively balanced arterial and venodilator.
- Atrial natriuretic peptide therapy (Nesiritide).

- Vasodilates arteries, but also affects significant natriuresis.
- For refractory CHF, no mortality benefit.

Afterload Reduction

- Goal SBP < 100–110 mm Hg.
- Beta-blocker therapy:
  - Carvediolol favored.
  - Lopressor, a longer acting agent, can also be considered.
  - Do not start a new beta blocker in the setting of acute CHF.
    - Patients already on a beta blocker who develop new CHF should have the dose dropped in half, **BUT NOT COMPLETELY DISCONTINUED**.
- Nicardipine gtt in the acute setting.
- ACE inhibitor therapy should be started early and titrated aggressively.
- Consider the addition of hydralazine, clonidine, or minoxidil if blood pressure is difficult to control.
- Nitroprusside or Nesiritide can be used transiently in the acute setting as described in the section on Preload Reduction.

Inotropic Therapy

- There is no mortality benefit to using inotropic therapy in the setting of acute CHF when complicating underlying systolic dysfunction exists.
  - However, it can be considered as a temporizing measure until more definite evaluation and care are available.
- Dobutamine or milrinone can be considered in acute CHF with SBP > 100 mm Hg.
- Dopamine should be considered if SBP < 90 mm Hg.
- An aortic balloon pump should be used, if available, when CHF complicates the period surrounding the presentation of an acute myocardial infarction or when aortic or mitral valve dysfunction is the cause of the CHF.

Other Aspects of Therapy

- Follow electrolytes closely.
  - Normalize serum magnesium and potassium.
  - Phosphorous levels below 1.0 mg/dL should be repleted.
  - Hyponatremia is a marker for increased mortality in the setting of CHF, but there is no benefit in correcting the hyponatremia as a specific therapeutic aim.

- ♦ It will correct on its own as CHF improves; the kidney sees better forward flow, and free water retention decreases.

- Watch for evidence of arrhythmias.
  - Patients with an ejection fraction < 30%–35% should be considered candidates for automated implantable cardioverter defibrillator placement unless life expectancy is <6–12 months.

Neurological Considerations

Traumatic Brain Injury

The medical management of TBI will be briefly reviewed in greater detail in Chapter 15, Head Injuries. Treatment is centered on the prevention of secondary brain injury resulting from hypotension and hypoxia.

Cerebrovascular Accident/Stroke Management

Two questions are vital to answer immediately when a patient presents with symptoms suggestive of a cerebrovascular accident (CVA), because they dictate the therapeutic approach:

- **When did the stroke occur?**
  - If fibrinolytic therapy is going to be considered, it should be delivered within 6 hours of symptom onset (better outcomes associated with early [<3 hour] therapy).
- **Is the stroke hemorrhagic or nonhemorrhagic**?
  - There is a risk of hemorrhagic conversion (may be seen in up to 10%–15% of patients with middle cerebral artery territory strokes). Document and follow serial neurological exams closely.
- Assess airway patency serially and have a low threshold for intubation.
- AVOID HYPOXEMIA (keep SpO_2 > 90% and PaO_2 > 60 mm Hg).
- Avoid hyperglycemia and hypoglycemia (keep glucose 90–140 mg/dL).
  - Utilize insulin drip if necessary.
- Keep head of bed flat unless aspiration risk is present, patient has been placed on mechanical ventilation, stroke territory is large, or there is evidence of elevated intracranial hypertension.

- If such relative contraindications to flat positioning exist, place patient in 30° head-of-bed elevation.

- Start therapy with aspirin within 24 hours if no evidence of intracranial hemorrhage.
- **<u>CAUTION</u>: THROMBOLYTICS SHOULD ONLY BE GIVEN IN ACCORDANCE WITH CURRENT AMERICAN HEART ASSOCIATION GUIDELINES REGARDING TIMING FROM THE ONSET OF SYMPTOMS AND STROKE SEVERITY.**
- Thrombolytics (Tenecteplase, Alteplase, Reteplase) should be given if no significant contraindications exist, the stroke is associated with significant clinical deficits, and there is no evidence of intracranial hemorrhage.
- Ensure that it is possible to lower SBP < 185 mm Hg and DBP < 110 mm Hg.
- Hypertension management.
  - Hypertension in the setting of CVA usually reflects either baseline blood pressure levels or a reaction to the stroke itself and may be dangerous to normalize in the acute setting.
    - SBP > 220 mm Hg or DBP > 140 mm Hg should be treated using short-acting, titratable IV medications, such as Labetalol or Nicardipine, with a goal of producing a 15% drop in blood pressure values.
    - Previously used outpatient antihypertensives should be initiated within 24–48 hours of the CVA, and goal blood pressures of SBP < 130 mm Hg and DBP < 80 mm Hg should be achieved slowly over days to weeks.
  - Other conditions that may coexist with the CVA and may dictate a more aggressive approach to rapid blood pressure titration (even normalization of blood pressure) using short-acting IV agents include:
    - Unclipped cerebral aneurysms associated with subarachnoid hemorrhage.
    - Aortic dissection.
    - Acute myocardial infarction.
- Body temperature regulation: MAINTAIN NORMOTHERMIA.
  - Efforts to normalize body temperature are appropriate.
  - Temperature regulation by the patients may not be normal.

- Hyperthermia is associated with worse outcomes and should be avoided.
- Acetaminophen PO or per rectum may be beneficial in this setting.
- Therapeutic hypothermia in the setting of CVA is not supported by the literature at this time outside of clinical research protocols.
- Other adjunctive agents.
  - Nimodipine has been associated with improved clinical outcomes when used in the management of acute subarachnoid hemorrhage.
  - Free radical scavengers have been suggested to have some benefit in CVA, but are not recommended for routine care at this time.

Gastrointestinal Considerations

Stress Gastritis

Indications for stress gastritis prophylaxis include several factors common in the combat critical care setting that predispose patients to develop stress gastritis. Of particular note are **coagulopathy, mechanical ventilation longer than 48 hours, shock, multisystem trauma, and burn >35% of the total body surface area.** Since most patients in the combat setting who need critical care support have at least one of these risk factors, **prophylaxis against stress gastritis should be considered** in all such patients.

- Suggested agents include pantoprazole 40 mg IV daily or ranitidine 50 mg IV or SQ every 8 hours.
- Sucralfate is not recommended in this setting.

Acalculous Cholecystitis

Trauma patients have several potential risk factors for the development of acalculous cholecystitis, significantly, multisystem trauma, hypotension, and burns. The diagnosis can be elusive but should be made in a timely fashion.

- Diagnosis suspected with new fever, vague abdominal discomfort, and leukocytosis.
  - Liver function tests are often normal

- Confirmation of the diagnosis can be made with RUQ (right upper quadrant) ultrasound.
- Empiric antibiotic therapy should be started when the diagnosis is suspected.
  - Imipenem, pipericillin/tazobactam, ampicillin/sulbactam, or a third-generation cephalosporin with metronidazole are all reasonable choices.
  - Vancomycin or Linezolid should be added only if the patient is known to be colonized with MRSA.
- Urgent percutaneous decompression should be considered in the unstable patient.

Renal Considerations

The most relevant forms of renal abnormalities in the combat setting include **prerenal azotemia, acute tubular necrosis (ATN), rhabdomyolysis, nephrolithiasis, and iatrogenic complications of medications.** Most of these entities are temporary if recognized and managed appropriately. For those that do develop significant azotemia, there usually exists a window of at least 24–36 hours to facilitate evacuation out of the theater of operation, because mechanisms to provide dialysis do not exist in the theater. **Early recognition of renal complications and appropriate early medical management are key to avoiding significant life-threatening complications**.

Prerenal Azotemia and Acute Tubular Necrosis

Although these two entities are separate clinical conditions, they are commonly related in the combat patient. **Prerenal azotemia represents the development of renal failure** (marked by a decreased CrCl and complications such as elevated BUN, acid-base abnormalities, hypervolemia, and electrolyte disturbances such as hyperkalemia) due to hypoperfusion of the kidneys. **ATN usually develops as a result of hypoperfusion with subsequent damage to renal tubule cells, especially in the region of the thick ascending loop of Henle.** Damaged tubule cells may form "muddy brown casts" that can be seen on urine microscopy and may obstruct tubules.

- **Prerenal azotemia diagnosis.**
  - Decreased urine output, elevated Cre, BUN/Cre > 20, UNa < 10 mg/dL.

- FeNa (%) = (UNa/SNa)/(SCre/UCre) × 100.
  - FeNa < 1% is consistent with a prerenal etiology of renal failure
    (where BUN = blood urea nitrogen, Cre = creatinine, UNa = urine sodium, FeNa = fractional excretion of sodium, SNa = serum sodium, SCre = serum creatinine, and UCre = urine creatinine).

- ATN diagnosis.
  - Decreased urine output, elevated creatinine, BUN/Cre 10–20, UNa >20 mg/dL.
  - Muddy brown casts on urine microscopy.
- Prerenal azotemia and ATN management.
  - Ensure adequate IVV.
  - There is no significant clinical benefit to converting anuric renal failure to oliguric failure, although patients who present with anuria generally do worse.
  - If IVV repletion is ensured and urine output is low, diuretic use can be considered in the patient with low urine output if IVV overload is a concern.
  - In the case of ATN, a period of 1–3 weeks may pass before renal recovery is noted.
    - An increase in urine volume occurs that precedes any true improvement in CrCl.
  - Watch closely for the development of hyperkalemia, acidemia due to anion gap metabolic acidosis, IVV overload, pericardial rubs, and extreme uremia.
    - These are indications for emergent hemodialysis.

Rhabdomyolysis

Rhabdomyolysis results in the setting of crush injury that causes significant destruction of skeletal muscle. CK_T (creatinine kinase), heme-pigmented myoglobin, and phosphate elevations are all released in significant amounts. **Heme-pigmented proteins may result in an ATN**. One unique feature of this form of renal failure is that it is associated with hypocalcemia. **Prevention of renal failure and its consequences is one of the fundamental priorities of the management of rhabdomyolysis**.

- Diagnosis: Red/brown low-volume urine, positive urine dipstick for myoglobin in the absence of red blood cells on urine microscopy, and CK_T elevation (may be >50,000–100,000).

- Aggressively ensure adequate IVV repletion.
  - Replete with isotonic crystalloid. (LR should be used with caution due to concern of hyperkalemia.)
- Goal urine output 150–300 mL/h; consider diuretic if IVV has been repleted.
- Bicarbonate therapy can be considered—titrate to a urine pH of 6.5–7.
  - Dose: 150 mEq $NaHCO_3$ (3 standard amps) in 1 L D5W at 100 mL/h initially.
  - No definite clinical benefit to this approach has been demonstrated.
- Mannitol diuresis is not recommended in the peritrauma setting due to possible IVV depletion.
- Follow serum electrolytes closely, especially potassium, phosphorous, and ionized calcium.

Nephrolithiasis

Nephrolithiasis represented one of the most common reasons for soldiers to be evacuated from the combat theater in both Operation Iraqi Freedom and Operation Enduring Freedom, and surgery of renal stones was the most common elective surgery performed in theater. Risk factors related to the combat environment include **low urine volume due to IVV depletion, as well as a high-protein diet.** The majority of stones are calcium based (approximately 80%) and are therefore easy to visualize with radiographic studies. The majority pass spontaneously, but patients with a history of recurrent stones, family history of stones, or complicating anatomical features leading to renal failure may necessitate surgical therapy by a urologist.

- Diagnosis of nephrolithiasis suggested by waxing/waning pain (radiating to the flank or scrotum, generally depending on level of obstruction) and microscopic hematuria.
- The stone may be visualized on KUB, CT/nephrolithiasis protocol, or ultrasound.
  - Start with KUB; subsequent studies based on availability.
- Adequate intravascular hydration is extremely important.
- Parenteral medications are frequently needed for pain control.
- Medical therapy can be considered with an alpha-blocking medication, such as Tamsulosin (0.4 mg PO daily).

- Consultation with a urologist early is important. Evacuation to a medical treatment facility for refractory symptoms should be considered.

Iatrogenic Complications of Therapy (Medications, Contrast Dye)

Several medications may cause or contribute to the worsening of renal function in the multisystem trauma patient. The most common offenders are medications such as diuretics that may be used before IVV repletion has been ensured, resulting in prerenal azotemia or even ATN. Nonsteroidal antiinflammatory medications used for pain management may result in renal failure by altering local glomerular perfusion pressure. Penicillin medications may be associated with acute interstitial nephritis. **The most important single agent to be aware of with respect to the kidneys is intravenous contrast dye, which may cause an associated ATN (contrast dye-associated nephropathy)**. These agents are iodinated and either ionic or nonionic. Most contrast dyes used currently are nonionic, which has decreased the rate of renal failure.

- ATN resulting from intravenous contrast dye generally resolves within days, in contrast to the 1–3 week recovery expected from other causes of ATN.
- Assurance of adequate IVV is most important for the prevention of contrast dye nephropathy.
- The most important aspect of contrast dye-associated nephropathy is aimed at prevention with precontrast hydration. No benefit has been shown with either bicarbonate therapy of *N*-acetylcysteine (Mucomyst).

Disseminated Intravascular Coagulation/Thrombotic Thrombocytopenic Purpura

Disseminated intravascular coagulation (DIC) usually identifies patients with a higher likely mortality due both to underlying injury and possibly DIC itself. The process results from a prothrombotic state wherein fibrin is deposited throughout the body, resulting in the **consumption of coagulation factors, hemolytic anemia, and thrombocytopenia**. This leads to an inability to clot blood effectively, and patients are noted to have petechiae and frank bleeding from IV sites, surgical wounds, and

mucosal barriers of the body. **Thrombotic thrombocytopenic purpura (TTP)** is caused by abnormal activity of von Willebrand's factor, resulting in activation and aggravation of platelets. Laboratory abnormalities include **thrombocytopenia and hemolytic anemia**. The classic clinical pentad includes **fever, anemia, renal failure, thrombocytopenia, and neurological abnormalities (especially seizures)**.

- DIC diagnosis:
  - Hemolytic anemia, thrombocytopenia, and fibrinogen decrease (usually <100).
  - INR elevation (KEY DISTINCTION FROM TTP—THERE IS NO INR ELEVATION WITH TTP).
- DIC management:
  - Largely supportive; correct the cause of DIC.
  - Cryoprecipitate, fresh frozen plasma, platelet, and red blood cell transfusion IF CORRECTABLE ETIOLOGY FOR DIC IS IDENTIFIED.
- TTP diagnosis:
  - Hemolytic anemia, thrombocytopenia, and fibrinogen decrease.
  - INR IS USUALLY NORMAL.
  - Clinical pentad of fever, anemia, thrombocytopenia, renal failure, and neurological abnormalities.
- TTP management:
  - Blood products are largely without benefit.
  - High-dose corticosteroids.
  - Plasma exchange transfusions.
- Unrecognized and untreated TTP can have an extremely high mortality.

Heparin-Induced Thrombocytopenia

Heparin-induced thrombocytopenia (HIT) is caused by antibodies directed at the heparin-platelet factor 4 complex. It usually presents approximately 4–5 days after the initiation of heparin products, but can present suddenly in susceptible patients who have received heparin within the previous 3 months. The risk of the development is 1%–5% with unfractionated heparin and <1% with low molecular weight heparin. **The diagnosis is suspected when the platelet count suddenly drops by 50% or to a value <100,000 (if platelet count was normal initially)**.

Confirmation of the diagnosis will generally not be possible in the combat care setting, but higher-level medical treatment facilities can confirm the diagnosis by sending HIT antibody studies in the appropriate clinical context.

- Suspected HIT should prompt immediate discontinuation of all heparin products (including low molecular weight heparin).
- Therapeutic anticoagulation should be initiated in full anticoagulation doses, if possible.
  - Thrombosis occurs in >50% of HIT patients.
  - Antithrombin agents that can be used in the combat environment require titration based on activated partial thomboplastin time levels:
    - Argatroban.
    - Hirudin.
  - Fondaparinux is an anti-Xa inhibitor that can be considered at Role 4 facilities that have access to onsite anti-Xa level measurement capability.
- Warfarin should NOT BE USED in the management of HIT patients unless an antithrombin agent is in use at full therapeutic anticoagulation doses.

Endocrine Considerations

The majority of endocrine emergencies that happen in the combat setting occur in patients with preexisting conditions (known or not) who undergo a clinical decompensation related to either a stress in the environment or lack of access to maintenance medical care (insulin in the case of the diabetic patient). While infrequently seen, the most likely endocrine emergencies to be aware of are diabetic ketoacidosis, hyperglycemic hyperosmolar syndrome, and adrenal insufficiency.

Diabetic Ketoacidosis/Hyperglycemic Hyperosmolar Syndrome

- Diagnosis of diabetic ketoacidosis (DKA):
  - Elevated glucose (200–600); long-standing DKA may have normal glucose.
  - Anion gap metabolic acidosis; elevated serum and urine ketoacidosis.
  - Glucosuria if serum glucose is elevated.
  - Dehydration (generally <6–8 L of total body water deficit).

- Diagnosis of hyperglycemic hyperosmolar syndrome (HHS):
  - Severely elevated glucose (600–1,500).
  - Severe intracellular dehydration due to extreme osmotic shifts.
  - Mild anion gap metabolic acidosis may be present, but is not a dominant clinical feature.
  - Severe glucosuria.
  - Severe dehydration (>8–10 L of total body water deficit).
- Management of DKA and HHS:
  - Correct the cause of DKA/HHS development (infection, trauma, etc).
  - Management is similar in many ways; differences will be highlighted.
  - Bolus 10 units of regular insulin IV; start insulin drip at 5 units of regular insulin IV per hour.
    - Hold on bolus if potassium < 3.0; do not give insulin until serum potassium > 3.0.
    - Do not correct glucose > 100 per hour or 1,200 in 24 hours.
  - Bolus 2 L of 0.9 NS in the first hour.
    - Repletion of volume is vital for both conditions; HHS will require substantially more isotonic crystalloid to accomplish this.
    - Give 4–6 L of 0.9 NS in the first 6 hours for DKA.
    - Give 6–8 L of 0.9 NS in the first 6 hours for HHS.
    - Subsequent 0.9 NS requirements will be determined by assessment of the adequacy of the IVV status.
  - After repletion of the IVV, change base fluid from isotonic crystalloid (0.9 NS) to hypotonic crystalloid (1/2 NS).
  - Check glucose hourly using point-of-care testing while adjusting insulin drip.
  - Measure serum electrolytes every 1–2 hours until potassium stable for >4 hours and glucose stable for >4 hours.
  - When potassium < 4.5 mg/dL, add 20 mEq KCl/L to current intravenous fluid.
    - Additional supplementation will be needed (orally as a KCl elixir) as well.
    - Potassium replacement needs are usually profound due to total body loss of potassium and magnesium due to diuresis, as well as transcellular shifts associated with insulin utilization.

- When serum glucose drops below 250 mg/dL, add D5 to whatever fluid is being utilized.
- **WHEN TREATING DKA, DO NOT STOP INSULIN INFUSION UNTIL THE ANION GAP IS CLOSED—HYPOGLYCEMIA IS TREATED WITH THE ADDITION OF DEXTROSE AND A DECREASE IN INSULIN DOSE, BUT CESSATION OF INSULIN WILL RESULT IN THE RETURN OF DKA.**

Adrenal Insufficiency

Adrenal insufficiency should be anticipated in patients requiring surgery who are taking corticosteroids at doses in excess of the equivalent of prednisone 10–20 mg daily. It is also seen clinically in patients who have taken such doses for more than 5–7 days at any point in the previous year. Rarely, adrenal insufficiency results from infarction of the bilateral adrenal glands associated with hypovolemic shock states. Unfortunately, there is no universal agreement on the laboratory diagnosis of adrenal insufficiency, so a high index of clinical suspicion should be present in patients with a known history of steroid use. **One clinical scenario that can be suggestive of adrenal insufficiency is a patient with a history of steroid use who is hypotensive (sepsis, hemorrhage, etc), who is not responsive to pressor therapy, and who does not have an appropriate tachycardia. The presence of hyponatremia and/or hyperkalemia may also suggest adrenal insufficiency**.

- Treatment of acute adrenal insufficiency: Hydrocortisone 200 mg IV, then 100 mg IV q8h.
- If hyponatremia and/or hyperkalemia persists despite hydrocortisone therapy, add fludrocortisone 0.1 mg PO every morning.

ICU Prophylaxis

Ventilator-Associated Pneumonia/Combat-Related Ventilator-Associated Pneumonia

- Assess daily the need for continued mechanical ventilation and discontinue as quickly as possible.
- Use a Hi-Lo Tracheal Tube to allow removal of subglottic secretions that collect above the endotracheal tube cuff in all patients expected to be intubated >96 hours.

- Provide oral care with chlorhexidine solution q4h.
- Do not routinely change out ventilator circuitry unless mechanical failure is present or visible contamination is noted.
- Keep head of bed 30°–45° at all times while intubated (unless absolute contraindication exists).
- Perform regular surveillance cultures of respiratory secretions in the ICU and regularly update the biogram describing organisms/susceptibilities that have been isolated.
- Minimize the empiric use of antibiotics.
- When treating a suspected combat-related ventilator-associated pneumonia (CRVAP):
  - Treat aggressively with broad-spectrum antibiotics based on local biogram (see section on Pulmonary Medicine).
  - Culture respiratory secretions, as well as blood; tailor antibiotic regimen based on culture results.
  - Discontinue all antibiotics if cultures are negative at 72 hours and patient is improving.
  - Continue CRVAP therapy for 7 days total if cultures demonstrate a dominant organism, and a Gram stain showed a significant number of leukocytes.
- When a multidrug-resistant organism is isolated, consider cohorting patients with similar isolates to one area of the ICU away from other patients.
- Consider terminal cleaning of a part of the ICU after a multidrug-resistant organism has been isolated and the patient treated.

Deep Venous Thrombosis Prophylaxis

See previous content within this chapter.

Glucose Control

- Most critical care patients in the combat setting should have glucose targeted between 140–200 mg/dL.
- Insulin drips should be initiated in any critically injured patient who has two or more consecutive glucose readings >180 mg/dL.

Nutrition

- Enteral nutrition is favored over venous routes, if possible.

- Duodenal tube placement is favored over gastric tube placement, but gastric is acceptable as long as residuals remain <500 mL/4 h.
  - Total parenteral nutrition may be available at some Role 3 facilities if full-dose enteral nutrition cannot be used by 72 hours.
  - The risk of infection related to total parenteral nutrition use may be driven more by the duration of central venous access and number of times the port is accessed than the actual content of total parenteral nutrition.
- Glutamine can be added to trauma patient nutrition regimens.
- Albumin should be given if the serum album is <1.
- Specialty formulas with specific additives generally offer little benefit in the acute setting.

For Clinical Practice Guidelines, go to http://jts.amedd.army.mil/index.cfm/PI_CPGs/cpgs