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 organ system 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, in fact, have organ hypoperfusion that is not appreciated. Resuscitation is not complete until adequate oxygen delivery (DO_2) and uptake have been ensured for all cells throughout the body.

$$\mathbf{DO_2 = C.O. \times 1.34 \times Hgb \times SaO_2 + 0.0031 \times PaO_2,}$$

where C.O. = 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 also often an accompanying decrease in hemoglobin that also contributes to inadequate DO_2.

Distributive shock is produced by an inappropriate decrease in systemic vascular tone, leading to an abrupt decrease in blood pressure to a level that cannot ensure adequate organ perfusion. Neurogenic shock, septic shock, and anaphylactic shock are examples of this process that may be seen with reasonable frequency in the combat setting.

Cardiogenic shock results from a primary defect in the generation of cardiac output. Myocardial infarction leading to heart wall or valve function abnormalities and cardiac tamponade are commonly seen examples. Many consider **obstructive shock** a related disorder. Processes that cause obstructive shock ultimately result in an inadequate cardiac output, although the mechanisms by which this occurs are variable. Pulmonary embolism (PE) and tension pneumothorax are two illustrative examples.

Define Goals of Shock Resuscitation

- Mean arterial pressure (MAP) > 60 mm Hg (assuming no traumatic brain injury [TBI]).
- Urine output > 0.5 mL/kg/h.
- Adequate DO_2 to meet the needs of organ function.

Management of Uncompensated Shock

- Define the type of shock and its etiology; eliminate the cause of the shock as possible.
- Vigorously replete the IVV if MAP or urine output is inadequate targeting central venous pressure 8–10 mm Hg.
  - Central venous pressure: 8–10 mm Hg.
  - Pulse pressure variation <13%.
    - Pulse pressure = systolic blood pressure (SBP) – diastolic blood pressure (DBP).
- Use vasopressor agents to support the MAP after adequate volume restoration.
  - Vasopressin is the first-line agent in burn resuscitation.
  - Norepinephrine is the first line in most other nonhemorrhagic situations.
  - Consider 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 an **anion gap metabolic acidosis**.
- An **increased base deficit** suggests inadequate resuscitation.
  - Base deficit = number of mmoL of bicarbonate that must be added to a liter of plasma to make the pH = 7.4, assuming the partial arterial gas pressure of CO_2 ($PaCO_2$) is normal.
- 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's (LR) solution: 130 mEq/L sodium, pH 5.5–6.0.
  - 0.9% normal saline (NS): 154 mEq/L sodium, 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 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 >6 mL/kg/24 h resuscitation.
    - ♦ 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).
  - Rate of serum sodium correction should be <1 mEq/L/h and <12 mEq/L/24 h.
  - Free water restriction for euvolemic and hypervolemic hyponatremia.
  - NS (154 mEq/L) or 3% saline (513 mEq/L Na) infusion.
    - Reserved for seizures, severe mental status changes, etc.

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:** Free water repletion.
- Relative "free water excess" (in liters) = 0.6 × weight in kg × (Na – 140)/140.
  - Rate of serum sodium correction should be <1 mEq/L/h and <12 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 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 acidemia or increased beta-2 agonist utilization. Total body potassium **depletion** may also lead to a decrease in serum potassium concentration through renal (diuretic use, postobstructive diuresis, osmotic diuresis, metabolic alkalosis, and proximal/distal renal tubular acidoses) and nonrenal (diarrhea, sweat, and fasting) mechanisms.

Total body potassium deficits range from 150 to 400 mEq for each 1 mEq/L decrease in serum:

- Potassium supplementation must be carefully monitored to avoid hyperkalemia development.
- Repletion of potassium is made more difficult if total body magnesium stores are low.
- The pace of potassium repletion depends on the presence or absence of clinical manifestations more than the absolute serum concentration.
  - Prominent U waves, T-wave flattening on EKG.
  - Paralysis, respiratory muscle dysfunction, and rhabdomyolysis.
- Supplementation is best accomplished with enteral supplementation and is preferred if possible 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, and these higher rates require continuous cardiac 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 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 IV fluids containing dextrose during emergent repletion, because the dextrose will result in the intracellular redistribution of potassium and complicate repletion efforts.

Hyperkalemia (K > 5.5 mEq/L)

Hyperkalemia may present as a result of several different mechanisms. **Pseudohyperkalemia** results when large amounts of potassium are spilled from the intracellular space during measurement and subsequently measured in the extracellular space. The measured serum potassium level is not indicative of true serum concentration in the patient (eg, severe thrombocytosis [>1,000,000] or leukocytosis [>200,000]). **Redistribution hyperkalemia** is seen in the trauma critical care setting most frequently as a result of academia, succinylcholine utilization, or hypertonic states (hypertonic saline or mannitol use). Finally, hyperkalemia may result from **renal failure, hypoaldosteronism, and medications (penicillin potassium, salt substitutes, and exogenous potassium supplementation).**

- Chronic hyperkalemia of a given value is generally better tolerated than acute presentations.
- Acute hyperkalemia should be regarded as a life-threatening medical emergency.
- Pace of treatment is generally dictated by EKG abnormalities (seen, in general order, as):
  - Peaked T waves, flattened P waves, and prolonged PR interval.
  - Idioventricular rhythm, widened QRS interval, sine wave pattern, and ventricular fibrillation.

- Treatment options for hyperkalemia include:
  - 50 mEq of $NaHCO_3$ (1 standard ampule of a 7.5% $NaHCO_3$ solution). Repeat every 30 minutes until QRS improved; often ineffective if renal failure has caused the hyperkalemia.
  - 10 mL of calcium chloride of a 10% solution (standard calcium chloride ampule) over 1–3 minutes; can repeat every 5 minutes, as long as severe EKG changes persist.
  - Consider dialysis as soon as possible if QRS widening has presented.
- 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 as needed EKG changes.
  - 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 grams orally every 6 hours or 50 grams as an enema every 2–4 hours.
- Treatment with normal EKG consists of identification and correction of the cause, as well as 15 grams of sodium polystyrene sulfonate (Kayexalate) orally every 6 hours or 30–60 grams 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. Serum magnesium represents only a fraction of the total body magnesium stores, similar to the case with potassium balance. A significant difference with respect to magnesium is that it does not transition readily from the ICFV to ECFV. **Low serum magnesium levels indicated 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 central nervous system (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 grams 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 grams of magnesium should be given as an immediate push, followed by 4–6 grams in 6 hours, and followed by 4–6 grams 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 in this setting and will be given greater emphasis here. Serum calcium levels are often corrected for serum albumin levels since 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 grams of calcium are corrected to within normal range.
- Hypocalcemic patients without QT prolongation can be repleted as follows:
  - Oral supplementation of 1.5–2.5 grams 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); an associated hypokalemia may be a clue to the presence of a trication deficiency.

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 associated with a given change in pressure. Inherent in this definition is the concept that a volume given to the patient by a ventilator will result in some change in pressure, whereas a pressure given will result in some change in volume.

Volume control modes of ventilation (assist-control [A/C], synchronized intermittent mandatory ventilation [SIMV]) provide mandatory breaths as a set volume (a set flow is given until a predefined volume is achieved) and generate some resulting pressure.

Pressure control modes of ventilation (pressure control ventilation) provide mandatory breaths as a set pressure, generating some resulting volume.

Ventilation (elimination of CO_2) is necessary to achieve a target pH that is physiologically acceptable to the body (7.35–7.45 in most patients).

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

Oxygenation/respiration (intake of oxygen) is necessary to support adequate DO_2 to the patient. Goal SaO_2 in most patients ranges between 92%–100%. **There is generally little physiological benefit from attempting to manipulate the ventilator to achieve values higher than 92%–94%**.

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 because it is associated with increased work of breathing when used for prolonged periods.
- In addition, 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 Lung Injury
Both acute respiratory distress syndrome (ARDS) and acute lung injury (ALI) represent the same disease process, and their definition differs only on the degree of shunt as estimated by the PaO_2/FiO_2:

- 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.
- PaO_2/FiO_2 < 200 (ARDS), PaO_2/FiO_2 200–300 (ALI).

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.

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 relative to standard of care in 2000 and should be followed where possible (Table 11-1).

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.

Table 11-1. Mechanical Ventilation Protocol Summary

INCLUSION CRITERIA

Acute onset of the following:

1. $PaO_2/FiO_2 \leq 300$ (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 PBW.
   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 PBW.
4. Reduce V_T by 1 mL/kg at intervals ≤2 hours until V_T = 6 mL/kg PBW.
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.

Oxygenation Goal: PaO_2, 55–80 mm Hg or SpO_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: ≤0 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.

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

Table 11-1 *continued*

pH Goal: 7.30–7.45

Acidosis management: pH < 7.30

- **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 that the duration of inspiration be less than or equal to the duration of expiration.

PART II: WEANING

A. Conduct a Spontaneous Breathing Trial Daily When:

1. $FiO_2 \leq 0.40$ and PEEP ≤ 8.
2. PEEP and FiO_2 less than or equal to the values of the previous day.
3. Patient has acceptable spontaneous breathing efforts. (May decrease vent rate by 50% for 5 minutes to detect effort.)
4. Systolic BP ≥ 90 mm Hg without vasopressor support.
5. No neuromuscular blocking agents or blockage.

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 PBW.
   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.
4. If not tolerated, resume preweaning settings.

DEFINITION OF UNASSISTED BREATHING

(Different from the Spontaneous Breathing Criteria Because PS Is Not Allowed)

1. Extubated with face mask, nasal prong oxygen, or room air

OR

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

Table 11-1 *continued*

2. T-tube breathing
OR
3. Tracheostomy mask breathing
OR
4. CPAP ≤ 5 cm H_2O **without PS or IMV assistance**.

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.

    - ♦ Device can be used in combat medical facilities, as well as ground and air transport vehicles.
- Conservative IVV management.
    - o 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.
    - o No benefit to using a pulmonary artery catheter to guide fluid management.
- Special dietary formulations.
    - o No single proprietary formula has been demonstrated to improve outcomes.
- Corticosteroids.
    - o No consistent benefit for the use of corticosteroids in ARDS.
- Inhaled nitric oxide.
    - o Improved oxygenation noted.
    - o No mortality benefit.
- Pressure control ventilation.
    - o No significant outcomes benefit relative to volume control A/C mode.
    - o If used, efforts must be made to continue to limit V_T as outlined in the ARDSNet protocol.
- Airway pressure release ventilation.
    - o 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 with carbon dioxide elimination is severely limited.
  - Has not been directly compared with ARDSNet low V_T strategy.

Patients with PEEP > 14 cm H_2O or who appear clinically unstable and who require immediate transport should be considered candidates for activation of specialized lung teams, where available. Such a team is based at Landstuhl Regional Medical Center to support EUCOM (US European Command), AFRICOM (Africa Command), and CENTCOM (US Central Command) missions.

Pulmonary Contusion

Pulmonary contusion is frequently seen in the combat setting, most commonly being associated with blunt, nonpenetrating trauma with or without rib fractures. The disorder is similar to ARDS, in that it may present with a significant degree of hypoxemia due to shunt physiology requiring increased mean airway pressure, as well as decreased compliance requiring limited V_T. 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). Each patient may have a different mean airway pressure where this happens that is clinically hard to predict. **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 and occasional bronchoscopy to facilitate pulmonary toilet.

Pulmonary Embolism

PE is part of a broader disease process that includes deep venous thrombosis (DVT) known as venous thromboembolic disease. DVT is very common in the trauma setting and associated PE may be a life-threatening result. Diagnosis of DVT can be made in the combat support setting using duplex ultrasound or CT chest/PE protocol with leg venous runoffs if available, but may need to be treated empirically if clinically suspected; however, technology is unavailable for confirmation. PE diagnosis is difficult in the best of circumstances, but it is vital to systematically define pretest probability before ordering any studies. Available studies to confirm PE in the combat support setting are largely limited to CT chest/PE protocol performance at higher echelon facilities. If pretest clinical suspicion (see next page) is moderate or high, treatment should be given until confirmatory testing has been accomplished.

Diagnosis of DVT

- Define pretest clinical suspicion.
- If low pretest clinical suspicion, do not work up further.
- If moderate or high pretest clinical suspicion, perform duplex ultrasonography.
- If clinical suspicion is high in the absence of ultrasonography high pretest clinical suspicion, but negative ultrasonography, consider empiric treatment with further testing at a higher echelon of care.

- Consider empiric treatment with further testing at a higher echelon.
  - Consider serial ultrasonography (a total of three times over 3–5 days).
- **Treatment of DVT.**
  - Low molecular weight heparin (Lovenox 1 mg/kg subcutaneously bid)
  - 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, pelvic fracture, etc.

Diagnosis of PE

- Define pretest clinical suspicion.
- If low clinical suspicion:
  - Obtain duplex ultrasonography of bilateral lower extremities (if available).
  - Perform portable chest X-ray (pCXR) (PA/LAT CXR [posteroanterior/lateral chest X-ray], if possible) to exclude easily identified mimics of PE (pneumothorax, hemothorax, ARDS, pulmonary contusion, and pneumonia).
  - Do not work up further if ultrasound is negative (or if study unavailable).
- If moderate or high clinical suspicion:
  - Initiate therapy with low molecular weight heparin (Lovenox 1 mg/kg subcutaneously bid).
  - Perform pCXR (PA/LAT CXR, if possible) to exclude easily identified mimics of PE (pneumothorax, hemothorax, ARDS, pulmonary contusion, and pneumonia).
  - Obtain duplex ultrasonography of bilateral lower extremities (if available).
    - If DVT identified, continue full-dose low molecular weight heparin and do not perform further diagnostic studies to evaluate for PE.
  - Obtain CT chest/PE protocol if ultrasound is negative (or unavailable).
  - If CT chest/PE protocol was performed and was negative for PE, therapy for PE can be discontinued, and no further diagnostic studies to evaluate for PE are necessary.

- Full-dose anticoagulation should be continued unless the CT chest/PE protocol was normal or another obvious source for the patient's symptoms is identified.
  - Further diagnostic evaluation should be performed at higher medical treatment facilities in this case.
- Removable inferior vena cava (IVC) filter placement should be considered in patients with PE pretest clinical suspicion who have DVT or PE diagnosed, or in whom PE cannot be excluded by CT chest/PE protocol, and in whom there is a significant contraindication to therapeutic anticoagulation.
  - Placement of such endovascular devices will not be possible at most combat support medical facilities.

- If high pretest clinical suspicion:
  - Initiate therapy with low molecular weight heparin (Lovenox 1 mg/kg subcutaneously bid).
  - Perform pCXR (PA/LAT CXR, if possible) to exclude easily identified mimics of PE (pneumothorax, hemothorax, ARDS, pulmonary contusion, and pneumonia).
  - Obtain duplex ultrasonography of bilateral lower extremities (if available).
    - If DVT identified, continue full-dose low molecular weight heparin and do not perform further diagnostic studies to evaluate for PE.
  - Obtain CT chest/PE protocol if ultrasound is negative (or available).
  - Full-dose anticoagulation should be continued regardless of the CT chest/PE protocol results in the setting of high pretest clinical suspicion unless another obvious source for the patient's symptoms is identified.
    - Further diagnostic evaluation should be performed at higher medical treatment facilities in this case.
  - Removable IVC filter placement should be considered in patients with high pretest clinical suspicion who have DVT or PE diagnosed; or in whom another obvious diagnosis is not provided by CXR, ultrasound, or CT chest/PE protocol; and in whom there is a significant contraindication to therapeutic anticoagulation.
    - Placement of such endovascular devices will not be possible at most combat support medical facilities.

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, because this defines a patient population with a very high rate of mortality. Patient instability may preclude making a formal diagnosis of hemodynamically unstable PE. Bedside transthoracic echocardiogram demonstrating evidence of right heart failure in the setting of a high pretest clinical suspicion for PE may assist in making a reasonable clinical diagnosis. The following should be considered:

- Start therapy immediately with low molecular weight heparin (Lovenox 1 mg/kg subcutaneously bid) or unfractionated heparin.
  - Use of this agent must be considered carefully in the multisystem trauma patient.
  - Protamine 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.
  - Do not give fluid boluses for hypotension if significant evidence of right heart failure exists.
  - Jugular venous pressure elevation noted or central venous pressure > 18 mm Hg if being transduced from a central venous catheter with a tip known to be in the superior vena cava.
- Support blood pressure (MAP > 60 mm Hg, DBP > 40–45 mm Hg) using epinephrine or dopamine.
- Norepinephrine is also acceptable, although reflex vagal stimulation may result in a decreased cardiac output relative to what is seen with epinephrine.
- 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.

- Absolute versus relative contraindications to the use of such agents must be considered carefully in the multisystem trauma patient.

Prevention of Venous Thromboembolism

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

- All trauma patients should receive chemical prophylaxis for venous thromboembolism disease.
  - Low molecular weight heparin (Lovenox 30 mg subcutaneously bid) should be administered.
  - Highest risk patients (spine injury, expected prolonged immobilization, and orthopaedic injury) should also have intermittent pneumatic compression device therapy initiated.
- Trauma patients with a significant clinical contraindication to chemical prophylaxis should receive intermittent pneumatic compression device therapy.
  - Highest risk patients (spine injury, expected prolonged immobilization, and orthopaedic injury) should also be considered for removable intravenous vena cava filter placement.

Aspiration Pneumonitis

In patients with compromised pulmonary status secondary to aspiration, they 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. Antibiotic therapy should be based on concomitant injuries. 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 an impressive fever, moderate leukocytosis, worsening oxygenation, and evidence of consolidation on physical exam. Antibiotics are not recommended

for this process in the first 24 hours after a suspected aspiration event. Failure to demonstrate some improvement after this time should prompt consideration of a secondary bacterial pneumonia infectious process.

Empiric antibiotic therapy with a broad-spectrum agent (meropenem, pipericillin/tazobactam, and cefepime) should then be initiated due to a high rate or oral colonization with multidrug-resistant organisms in the combat critical care setting. Specific coverage targeting anaerobic organisms is not necessary in the absence of poor dentition, although anaerobic coverage will be included with most of the empiric broad-spectrum agents discussed previously. Specific coverage for methicillin-resistant *Staphylococcus aureus* (MRSA) is not necessary unless the patient is believed to be previously colonized with this organism. If available, bronchoscopy with directed bronchoalveolar lavage or blind aspiration through an endotracheal tube can be used to determine the duration of antibiotic therapy. Bronchoscopy should be performed in any case where foreign body aspiration (teeth, etc) is suspected. Antibiotics should be stopped at 72 hours if cultures do not demonstrate a dominant organism. If a dominant organism exists, antibiotics can be discontinued at 5–7 days.

Combat-Associated Healthcare Pneumonia

Combat-associated healthcare pneumonia denotes a healthcare-associated pneumonia that is obtained by a patient 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 meropenem, doripenem, pipericillin/tazobactam, or cefepime. Ertapenem is not recommended due to poor coverage of *Pseudomonas aeruginosa*.

Vancomycin or Linezolid should be added if MRSA is clinically suspected (a known history of MRSA colonization), and double

coverage for *Pseudomonas* should be included if associated *Pseudomonas* bacteremia is suspected. Antibiotic coverage should be tailored to the narrowest spectrum possible based on respiratory and blood culture results, and duration of therapy should continue for 5–7 days if clinical improvement is noted. Failure to improve by 7 days should prompt a reconsideration of the diagnosis, repeated efforts to obtain cultures, consideration of other infectious organisms, and a search for defects in the immune system (neutrophil number/function, B-cell function, T-cell function).

Cardiac Considerations

Cardiac Tamponade

Acute cardiac tamponade is seen in the combat setting as a result of either blunt or penetrating thoracic trauma. Cardiac tamponade in the setting of trauma is a surgical emergency. Hemodynamically significant pericardial effusions associated with trauma generally may be small volume collections of blood that result in the collapse of the cardiac chambers; however, any pericardial effusion in the setting of trauma requires immediate surgical evaluation. Tamponade physiology, initially, may be subtle and vary with respiration, but eventual 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 percutaneous pericardial drainage may be considered as a temporizing method in the absence of immediately available surgical care.
  - Via an unguided subxyphoid needle directed toward the left nipple in an emergency.
  - Echocardiographically guided needle insertion for pigtail drainage, if available.
- IVV may need to be aggressively supported to ensure adequate cardiac filling.

- Inotropic therapy with Dobutamine may temporize the condition until elimination of the pericardial fluid collection has been accomplished outside the setting of trauma-associated tamponade.
- 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 with cardiac contusion have minimal-related symptoms, and significant consequences are uncommon. Severe blunt cardiac injury symptoms are usually those referable to musculoskeletal pain, although CHF may be present if the degree of injury was significant enough to result in myocardial wall or valve dysfunction. When valve dysfunction occurs, it usually represents improperly functioning chordae tendinae because of myocardial wall dysfunction. Diagnosis is usually made by demonstrating focal cardiac wall or valve dysfunction in a patient with recent blunt thoracic trauma. Cardiac enzymes do not have a role in the diagnosis or management of blunt cardiac injury. Management is supportive and centers around cardiac monitoring to detect the rare patient who develops significant arrhythmias or mechanical heart dysfunction (severe acute valve regurgitation, free wall rupture, and ventricular septal wall rupture).

Acute Coronary Syndrome

ST elevation myocardial infarction (STEMI) is usually caused by the rapid accumulation of fibrin at the site of a previously stable atherosclerotic plaque in a coronary artery that results in significant (often transmural) cardiac muscle death. To prevent further damage, management centers on opening the vessel as quickly as possible; decreasing oxygen demand by the heart; 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.
- Plavix 300 mg load followed by 75 mg PO daily.
- A glycoprotein 2B/3A inhibitor should be considered (Eptifibatide).
- 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 in <1 hour (within 3 hours is ideal, 12 hours is acceptable).
- If an invasive cardiac catheterization laboratory is available that is favored over thrombolytic therapy.
- Beta blocker (Lopressor 5 mg IV initially) if no evidence of acute CHF.
- Beta blocker per the current 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.
- **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.
  - 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 transferred to a higher medical treatment facility.

- An ACE (angiotensin-converting enzyme) inhibitor should be started within 24 hours of the index symptoms.
- A statin anticholesterol medication should be started as soon as possible.

Non-STEMI (NSTEMI) and unstable angina are closely related processes whereby a platelet-rich clot forms in the region of a previously existing atherosclerotic plaque. Symptoms associated with NSTEMI/unstable angina usually represent supply/demand mismatch in the setting of a slowly progressive clot, although the clot may progress quickly in some. 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 plays 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 rapidly, decrease myocardial oxygen demand, and monitor for complications of the disease process. Progression to STEMI needs to be carefully watched because it could affect therapy.

- Aspirin 81 mg PO, chewed as quickly as possible and daily thereafter.
- Plavix 300 mg load, followed by 75 mg PO daily.
- A glycoprotein 2B/3A inhibitor should be considered (Eptifibatide).
- 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 in <1 hour (within 3 hours is ideal, 12 hours is acceptable).
- Beta blocker per the current American Heart Association guidelines to target heart rate < 60–70 and SBP < 110.

- If heart rate target met with beta blocker, but SBP > 110, consider the following adjuncts:
  - Nitroglycerin gtt (dose may be limited by headache or the presence of right-sided disease).
  - Nicardipine gtt.
  - Nitroprusside gtt.
- If evidence of CHF:
  - Start nitroglycerin gtt.
  - Lasix q6h IV vs gtt to affect diuresis/preload reduction.
  - Consider nicardipine vs nitroprusside gtt to titrate blood pressure/afterload reduction.
  - Dopamine or Milrinone can be considered if SBP < 90.
- Continuous cardiac and hemodynamic monitoring (arterial line, central venous catheter with central venous pressure monitoring) should be continued until transferred to a higher echelon of care.
- An ACE inhibitor should be started within 24 hours of the index symptoms.
- A statin anticholesterol medication should be started as soon as possible.
- Aspirin 81 mg PO, chewed as quickly as possible and daily thereafter.
- Plavix 150 mg load followed by 75 mg PO daily.
- A glycoprotein 2B/3A inhibitor should be started (Eptifibitide).
  - Most important in patients with recurrent pain, ST segment depression or dynamic ST segment changes.
- Supplemental oxygen to maintain SpO_2 > 96%–98%.
- Sublingual nitroglycerin (spray or tablet) PRN pain.
  - Rapid hypotension development with nitroglycerin suggests right-sided disease
- Morphine IV PRN pain.
- Beta blocker (Lopressor 5 mg IV initially) if no evidence of acute congestive heart failure.
- Beta blocker (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 > 110, consider the following adjuncts:
  - Nitroglycerin gtt (dose may be limited by headache or the presence of right-sided disease).

- Nicardipine gtt.
- Nitroprusside gtt.

- If evidence of CHF:
  - Start nitroglycerin gtt.
  - Lasix q6h IV versus gtt to affect diuresis/preload reduction.
  - Consider nicardipine vs nitroprusside gtt to titrate blood pressure/afterload reduction.
  - Dopamine 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 transferred to a higher medical treatment facility.
- An ACE inhibitor should be started within 24 hours of the index symptoms.
- A statin anticholesterol medication should be started as soon as possible.

Congestive Heart Failure

CHF represents a clinical diagnosis describing the inability of the heart to pump adequately relative to a given preload. Resulting 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 becomes relatively excessive, as can acute or chronic valve dysfunction. Acute valve dysfunction can be seen in the setting of blunt cardiac contusion injury. Goals of CHF management center around **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 as 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.
  - Long active Lopressor 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 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.
  - 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 and can be explored in greater detail in Chapter 15, Head Injuries. There is nothing about medical management that can reverse primary brain injury caused by a traumatic event, but aggressive critical care management can greatly decrease the subsequent evolution of secondary brain injury. The critical care management of TBI casualties focuses upon the tenets of adequate oxygenation and adequate volume to minimize the risk of secondary brain injury.

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?**

- If 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 to place on mechanical ventilation if necessary.
- 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.
- **CAUTION: 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 achieved slowly over days to weeks.
- o Other conditions that may coexist with the CVA that 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.
  - o Efforts to normalize body temperature are appropriate.
  - o Temperature regulation by the patients may not be normal.
  - o Hyperthermia is associated with worse outcomes and should be avoided.
  - o Acetaminophen PO or rectum may be beneficial in this setting.
  - o Therapeutic hypothermia in the setting of CVA is not supported by the literature at this time outside of clinical research protocols.
  - o Other adjunctive agents.
  - o Nimodipine has been associated with improved clinical outcomes when used in the management of acute subarachnoid hemorrhage.
  - o 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 **coagulopathy, mechanical ventilation greater than 48 hours, shock, multisystem trauma, and burn >35% of the total body surface area.** Since most patients in the combat setting who have a need for critical care support have at least one of

these risk factors, **prophylaxis against stress gastritis should be considered necessary** in all such patients.

Pantoprazole 40 mg IV Daily or Ranitidine 50 mg IV or Subcutaneously 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, significant among them multisystem trauma, hypotension, and burns. The diagnosis can be very difficult to make at bedside, but is extremely important to make in a timely fashion, because a delay in therapy can result in significant morbidity or mortality.

- Diagnosis suspected with new fever, vague abdominal discomfort, and leukocytosis.
  - Mild alkaline phosphotase elevation.
  - Conjugated hyperbilirubinemia (elevated Tbili; Dbili/Tbili > 0.5).
- Confirmation of the diagnosis can be made with RUQ (right upper quadrant) ultrasound.
  - If normal, but condition suspected, laparoscopic or open laparotomy should be performed.
    - A HIDA (hepatobiliary iminodiacetic acid) scan can be performed at major medical centers prior to surgery if clinically stable, but this will not be available in the combat care setting.
- 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 consultation for operative management or interventional drainage of the condition should be obtained before frank necrosis and perforation of the gallbladder occurs.

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 do not involve permanent kidney damage if recognized and managed appropriately. For those that do develop significant azotemia (either transiently or permanently), there usually exists a reasonable window of at least 24–36 hours to facilitate evacuation out of the theater of operation. In general, a reliable mechanism for providing dialysis does not exist in the wartime environment until Role 4 is reached. **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 develops usually as a result of hypoperfusion with resultant 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, leading to several local hemodynamic consequences.

- **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.
  - FeNa (%) = (UNa/SNa)/(SCre/UCre) × 100.
    - FeNa >1% is consistent with a non-prerenal etiology of renal failure.
  - 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 in anuric renal failure do worse than those who are oliguric on presentation.
  - 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 an 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 (0.9% NS or LR may also be utilized, but consider the risk of hyperkalemia in the setting of rhabdomyolysis and associated renal failure).
- 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 represents 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 diet that may be high in protein**. The majority of stones are calcium based (approximately 80%) and are therefore easy to visualize with radiographic studies. Many will eventually pass on their own, 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 intravenous 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 and evacuation to a medical treatment facility where surgery can be performed if the stone does not pass can 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 dye 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 both due 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 of <100,000 (if platelet count was normal initially)**.

Confirmation of the diagnosis will generally not be possible in the combat care setting, but higher 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 (½NS).
  - 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 is not able to 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://usaisr.amedd.army.mil/clinical_practice_guidelines.html