1. GLOBAL APPROACH TO THE TRAUMA PATIENT
   a. Trauma is defined as a “wound or injury” and may occur secondary to motor vehicle accidents, falls from heights, animal interactions, human-animal interactions, etc. Severity may range from mild to fatal. Trauma may affect only one organ system or multiple organ systems, either directly or indirectly. Therefore, a global and thorough approach is required to improve survival and decrease morbidity in trauma patients.
   b. Initial assessment of the trauma patient occurs quickly but can theoretically be broken down into two major phases; primary survey and secondary survey:
      i. Primary survey is the assessment of the respiratory and cardiovascular systems, followed by assessment of the central nervous system and urinary tract system.
      ii. Secondary survey is assessment of all other systems once the animal’s most immediately life-threatening problems, identified during the primary survey, are dealt with.
   c. The primary goal in attending to a critically injured trauma patient is to optimize oxygen delivery to the tissues. In fact, during the initial approach and resuscitation of these patients, all procedures are oriented toward this goal. Oxygen delivery is dependent upon cardiac output, hemoglobin concentration, and oxygen saturation of hemoglobin. It is important to optimize these variables at all times.

2. BRIEF PATHOPHYSIOLOGY
   a. Traumatic shock results in a maldistribution of blood flow because of increased circulating catecholamines, hypovolemia, and increased vasoactive hormones.
   b. Persistent microcirculatory perfusion failure may lead to sludging of blood and increased cellular and platelet aggregation.
   c. Endothelial injury due to trauma may result in release of inflammatory mediators and stimulation of coagulation.
   d. Damaged cellular membranes, due to poor perfusion or direct trauma, result in release of phospholipids. The enzymes phospholipase, cyclooxygenase, and lipoxygenase produce thromboxane and leukotrienes:
      i. Thromboxane causes vasoconstriction, further compromising tissue perfusion and stimulating platelet aggregation.
      ii. Leukotrienes and activation of the complement cascade propagates the inflammatory response with mobilization and activation of neutrophils.
      iii. Neutrophil release of lysosomal enzymes and toxic oxygen metabolites cause further cellular damage, leading to edema with subsequent
increase of oxygen diffusion distance to the cells from the capillaries.

iv. Optimizing tissue oxygen delivery will minimize the perpetuation of these inflammation-producing cascades.

3. ASSESSING AND MAINTAINING OXYGEN SATURATION OF HEMOGLOBIN—THE RESPIRATORY SYSTEM

a. At presentation, supplemental oxygen should be provided to the severely affected trauma patient until assessment of an arterial blood gas or measurement of hemoglobin saturation confirms that oxygen supplementation is not required:
   i. Clinical signs of respiratory compromise include increased respiratory rate or effort, flail chest, pale or cyanotic mucous membranes, increased heart rate, increased upper airway sounds, or altered lower airway sounds (increased or decreased airway sounds). If these signs are present, oxygen supplementation is essential.

b. Physical assessment of the respiratory system (Figure 1.1):
   i. Look:
      1. Signs of respiratory compromise include increased respiratory rate and effort, restlessness, extended head and neck, abducted elbows, and paradoxical movement of the chest and abdominal walls (normally should move in and out together but with paradoxical movement they move in opposite directions).

   ii. Listen:
      1. Loud upper airway sounds heard without the stethoscope indicate upper airway compromise (from the carina forward).
      2. Traumatic causes of upper airway problems include cervical tracheal crush injury (bite wounds are most common), pharyngeal injury (swelling/bleeding), and tracheal avulsion (see Chapter 6: Trauma-Associated Thoracic Injury):
         a. Note that nasal airway injuries and frontal sinus injuries can result in bleeding into the nasal passages, causing upper airway sounds. Despite this problem, most animals do not suffer respiratory compromise from these injuries.

      3. Decreased air sounds on auscultation:
         a. Indicates pleural space disease.

      b. Traumatic causes of pleural space disease:
         i. Pneumothorax (most common)
         ii. Diaphragmatic hernia
         iii. Severe hemothorax (least common)

      c. Thoracocentesis:
         i. If the patient is in respiratory distress and you cannot hear the lung sounds well, then thoracocentesis is warranted.

      4. Increased lung sounds on auscultation (not the upper airway sounds referred to earlier):
         a. Indicate lower airway/pulmonary parenchymal injury:
            i. Pulmonary contusions:
               1. Therapy for pulmonary contusions is largely supportive with caution on overzealous fluid therapy.

      5. Open chest wound/"sucking" chest wound:
         a. Open wounds connecting to the pleural space should be immediately covered with a sterile bandage, which will
create a seal, and thoracocentesis should then be performed to remove the residual air.

b. If respiratory distress persists, intubation and positive pressure ventilation should be provided until the wounds can be surgically repaired.

6. Flail chest:
   a. Recognized by paradoxical movement of the chest wall. The flail segment moves in on inspiration and is a result of a “floating” segment of the chest wall because consecutive ribs are broken in more than one place.
   b. Primary physiologic consequences are pain and underlying lung damage and pleural space injury in most instances.

4. ASSESSING CARDIAC OUTPUT/TISSUE PERFUSION
   a. Initial assessment of adequate cardiac output and circulating volume includes mucous membrane color, capillary refill time, pulse rate, and quality:
      i. Abnormalities indicating poor tissue perfusion
         1. Pale or gray mucous membranes
         2. Prolonged capillary refill time (>1–2 seconds)
         3. Weak and rapid pulses
   b. Measurement of arterial blood pressure directly or indirectly by Doppler or oscillometric techniques should be performed when possible:
      i. A Doppler systolic blood pressure of <80 is suggestive of decreased tissue perfusion.
      ii. A mean arterial pressure of <60 mm Hg is suggestive of decreased tissue perfusion.
      iii. It should be kept in mind that blood pressure may be normal or even high and peripheral tissue perfusion could still be inadequate for the body’s requirements.
   c. More objective evaluation is obtained by pulmonary artery catheter placement and actual measurement of cardiac output, oxygen delivery, and oxygen consumption; although this is uncommonly done in veterinary emergency medicine and carries its own complications.
   d. The most common cause of poor tissue perfusion in the trauma patient is hypovolemia from blood loss:
      i. Most common areas for hemorrhage causing hypovolemia are in order of frequency; the peritoneal space, retroperitoneal space, thigh musculature, pleural space, external, or a combination of spaces (e.g., retroperitoneal and peritoneal spaces).
   e. The less common causes of poor tissue perfusion are cardiac arrhythmias, tension pneumothorax, diaphragmatic hernia, peritoneopericardial diaphragmatic hernia, and cardiac tamponade due to hemopericardium from traumatic atrial rupture.
   f. Therapy of poor tissue perfusion:
      i. Thoracocentesis if a tension pneumothorax is present (this is extremely rare).
   ii. Intravenous fluids/colloids/blood products/hemoglobin substitutes:
      1. Caution with fluids when pulmonary contusions are present.

5. ASSESSMENT OF THE NEUROLOGIC SYSTEM
   a. It is important to assess brain function at presentation to obtain a baseline for potential dynamic changes that may occur.
   b. Conclusions regarding brain and neurologic function (brain and spinal cord) should be withheld until perfusion is adequate and function is reassessed:
      i. Profound neurologic changes may be found with poor tissue perfusion and corrected once tissue perfusion is improved.
   c. Assessment of brain function:
      i. Mentation:
         1. Grades of mentation:
            a. Excited/agitated
            b. Normal
            c. Depressed
            d. Obtunded (markedly decreased level of consciousness, but arousable with auditory or tactile stimulation)
            e. Stupor (unconscious but responds to noxious stimuli)
            f. Coma (unconscious and does not respond to noxious stimuli)
   2. Cranial nerve assessment:
      a. Assessment of facial sensation, jaw tone (be careful with jaw fractures), eye position and movement, papillary light responses, menace reflex, eye blink, and gag reflex.
3. Evidence of potential brain injury:
   a. Obvious head trauma (skull fractures, bleeding from ear, jaw fractures, scleral, or ocular hemorrhage, etc.)
   b. Altered mentation
   c. Altered cranial nerve function
4. General principles of treating brain injury:
   a. Primary injury (direct trauma to tissues)—no specific treatment for this.
   b. Secondary injury (swelling, inflammation, reperfusion injury, increased intracranial pressure):
      i. Maintain good perfusion/blood pressure (MAP at least 80 mm Hg, systolic at least 90 mm Hg).
      ii. Maintain good oxygenation via oxygen supplementation, ventilation, etc. (PaO₂ > 80 mm Hg, SpO₂ > 95%).
      iii. Hyperosmotics:
         1. Mannitol
         2. Hypertonic saline/colloid combination to treat decreased blood pressure and poor tissue perfusion
      iv. Elevate head 30° from horizontal if increased intracranial pressure is suspected.
   d. Assessing spinal cord function:
      i. Ensure that tissue perfusion is adequate.
      ii. Palpate full length of the spine and pelvis.
      iii. Note the voluntary movement of all legs.
      iv. Assess the ability to stand/ambulate.
      v. Check anal tone and anal reflex.
      vi. Check proprioception of limbs.
      vii. Assess limb reflexes.
   v. Evidence of spinal cord injury:
      1. Spinal or sacral fractures/displacement
      2. Spinal pain
      3. Changes in limb function or sensation.
      4. Change in tail tone, bladder, or anal sphincter function
   vi. General principles of treating spinal cord injury:
      1. Stabilization of fractures/luxations
      2. Decompression surgery (e.g., hemilaminectomy for ruptured intervertebral disk)
      3. Corticosteroids (controversial)
      4. Time
6. ASSESSING THE RENAL SYSTEM
   a. Physical assessment of the urinary tract is limited to palpation of the urinary bladder and kidneys.
   b. Urinary tract injury seems to occur more commonly with pelvic fractures.
   c. BUN and serum creatinine may not have substantial increases until several hours after urinary tract rupture or injury.
   d. Hematuria indicates urinary tract injury.
   e. Manifestations of renal damage may not be immediately evident at presentation and may not be detected until several hours later, after continuous monitoring.
   f. Potential trauma-induced urinary tract injuries:
      i. Kidney contusions, rupture, or avulsion
      ii. Renal pelvic disruption
      iii. Urethral disruption
      iv. Urinary bladder rupture
      v. Urethral trauma/rupture
   g. General therapy for urinary tract trauma:
      i. IV fluid diuresis
      ii. Stabilization of cardiovascular manifestations of urinary tract injury (hyperkalemia)
      iii. Surgical repair and/or urinary diversion of injuries if necessary
7. SECONDARY SURVEY
   a. Full physical examination.
   b. Particular attention should be paid to the following:
      i. Musculoskeletal system:
         1. Observe limb function of all four legs.
         2. Observe ambulation.
         3. Complete palpation of appendicular and axial skeleton including rectal examination, palpation of skull, and manipulation of jaw.
      ii. Peripheral nervous system:
         1. Full evaluation of peripheral nerves.
         3. Examine the animal from the tip of the nose to the tip of the tail.
8. MONITORING
   a. Monitoring of all the above systems should be done for at least 24–48 hours for animals that have had significant trauma.
b. Generally, if significant injury has occurred, clinical signs will usually develop within 24–48 hours of the traumatic incident:
   i. Biliary tract rupture can sometimes result in clinical signs several days after the trauma.
   ii. Patients with diaphragmatic hernias may not develop respiratory distress for months to years.

c. The intensity of the monitoring should be proportional to the degree of compromise of the patient.