APPROACH TO THE EMERGENCY PATIENT

Steve C. Haskins and Douglass K. Macintire

I. INITIAL PREPARATION AND MANAGEMENT

A. Life-threatening abnormalities should be stabilized without delay.

1. A secondary, more exhaustive physical and laboratory examination can be completed once the life-threatening problems have been stabilized.

2. Semicomprehensive tertiary examinations should be repeated at regular intervals throughout the day and on subsequent days to keep abreast of the progress of the underlying abnormalities and their response to therapy, as well as to identify new problems that may become apparent.

B. Adequate preparation of the facility, equipment, supplies, and personnel for the array of emergencies likely to be presented, cannot be overemphasized.

1. The emergency receiving area should be designated and set up in advance with all of the necessary equipment and supplies readily available. There should be easy access to the area for patients on gurneys and stretchers, and there should be enough room for the care-givers to move around and gain easy access to the patient.

2. Equipment must be immediately available and in good working order. Personnel should be well trained in its use.

3. Supplies must be immediately available and inventoried daily and after each emergency.

4. Personnel should be trained and retrained in the job that they will be expected to perform when the emergency arrives. Protocols and procedures are established in advance and placed in an easy-to-access notebook. Wall charts of procedures and dosages are an excellent way to facilitate the conduct of emergency patient resuscitation.

5. Reference books and sources of information (e.g., poison control hot lines) for situations that were not foreseen should be readily available.

C. Effective management of the emergent patient begins when the hospital receives the call from the owner stating that there is a problem. The owner may be able to provide some first-aid care that would be beneficial to the animal even though the individual is not medically trained. Owners should be instructed to bring such animals to the nearest veterinary facility.

1. If the animal is unconscious or seizuring, the owner should place the animal on a blanket and transport it immediately to the hospital.

2. If the animal is not breathing, owners can be instructed as to how to manually hold the mouth closed, extend the head, and blow into the nose to get the chest to rise slightly.

3. Animals that have severe respiratory distress should brought immediately to the hospital.

4. If the animal is conscious and has been hit by a car, owners should be warned that such animals are hurt and scared and may bite when handled. Such animals should be strapped to a flat transport board if possible to prevent further orthopedic or spinal cord injury.

5. Open wounds should be covered with clean, moist towels prior to transport.

6. Fractured lower leg bones could be splinted with rolled up newspaper and tape but animals with fractures of the humerus or femur should just be moved onto a blanket and transported to the hospital. Cover open wounds with a clean, moist towel.

7. Animals that have ingested a poison/toxin should be brought to the hospital along with the container of ingestate. If not contraindicated (see Toxin Chapter) vomition can be induced.

8. Burn wounds should be covered with a cool, wet compress.

9. Suspected heat stroke patients should be wet down with cool water before transport.

10. Counterpressure should be applied to sites of active hemorrhage during transport.

11. Proptosed eyeballs should be held in place with wet compresses.

12. Owners should obey all traffic laws during the transport of patients to the hospital.

II. THE PRIMARY SURVEY: INITIAL EXAMINATION TO IDENTIFY LIFE-THREATENING PROBLEMS (Tables 1.1 and 1.2)

A. Assess the general demeanor of the animal.

1. Seizures should be terminated immediately by the administration of anticonvulsants.

a. Benzodiazepines (diazepam [0.2–0.6 mg/kg IV], midazolam [0.1–0.4 mg/kg IV]) are common choices. Any rapidly acting general anesthetic will also terminate seizure activity.

b. Phenobarbital (5–25 mg/kg) has anticonvulsant properties in subanesthetic dosages but has a slow onset of action (20 min) making it inconvenient to use in the initial management of seizures but

ideal for preventing recurrence (4–6 mg/kg IV, IM q 12 hr).

c. Seizures in puppies may be due to hydrocephalus, hypoglycemia (glycogen storage disease), infection (distemper), toxemia, portocaval shunt, or may be idiopathic.

d. Seizures in adults may be idiopathic, infectious, neoplastic, traumatic, ischemic, or thromboembolic, or may be caused by hypoglycemia (insulinoma), hypocalcemia, or granulomatous meningoencephalitis.

e. Other monitoring/treatment considerations: hyperthermia, airway protection, aspiration, neurogenic pulmonary edema, hypoxemia, hypoventilation, considerations specific to the underlying cause.

2. Tremors and hyperexcitability are treated symptomatically with benzodiazepines (diazepam, midazolam) or methocarbamol (50–150 mg/kg IV).

Tremors and hyperexcitability can be caused by amphetamines, herbal ephedra, ma huang, guarana root, methylxanthines (caffeine), pseudoephedrine, LSD, phencyclidine, marijuana, cocaine, benzodiazepines, opioids (large dose), metaldehyde, mycotoxins (penitrem A), blue-green algae, strychnine, 1080, bromethalin, ivermectin, organochlorine insecticides, pyrethrins, permethrins, organophosphates, carbamates, lead, zinc phosphate, tricyclic antidepressants, 4-aminopyridine, 5-fluorouracil, chocolate (large dose).

3. Severe obtundation and coma have many causes; initial management strategies involve examination, monitoring, and support of cardiopulmonary function.

a. Apnea? Intubate and ventilate (rate 10–15/min; tidal volume 10–15 ml/kg; proximal airway pressure 10–15 cm H_2O).

b. Cardiac arrest? Institute cardiopulmonary resuscitation.

c. Traumatic head injury? Prevent hypoxemia, hypoventilation, hyperthermia, hypotension, or induced hypertension (spontaneous hypertension should be allowed) or hypervolemia. Hyperosmotic edema-reducing therapy may be indicated (mannitol, hypertonic saline).

TABLE 1.1
PROBLEMS IDENTIFIED AT TRIAGE THAT REQUIRE IMMEDIATE ATTENTION

Problem	Why it is an immediate care issue
Respiratory	
Cyanosis, severe hypoxemia	Hypoxic cell damage
Severe hypercapnia	Cerebral vasodilation, respiratory acidosis, hypoxemia
Apnea or shallow breathing effort	Hypoxemia, hypercapnia
Dyspnea[a]	Hypoxemia, hypercapnia
Severe upper-airway obstruction	Hypoxemia, hypercapnia
Severe pleural space filling problems	Hypoxemia, hypercapnia
Open pneumothorax, severe flail chest	Hypoxemia, hypercapnia
Severe pulmonary parenchymal disease	Hypoxemia, hypercapnia
Cardiovascular	
Cardiac arrest	No blood flow to the body
Hypotension	Insufficient blood flow to the brain and heart
Severe vasoconstriction	Insufficient peripheral blood flow
Hypovolemia	Hypotension, vasoconstriction
Severe dehydration	Hypovolemia
Severe edema	Impaired blood/tissue oxygenation
Severe bradycardia, tachycardia, or arrhythmias	Insufficient blood flow, hypotension, cardiac arrest
Heart failure	Insufficient blood flow, edema
Active hemorrhage	Hypovolemia
Shock	Inadequate cellular energy production
Hypertension	Edema, hemorrhage, retinal detachment
Neurologic	
Seizures	Brain damage, hyperthermia, hypoventilation, regurgitation/aspiration
Tremors, hyperexcitability	Seizures
Coma	Brain damage from underlying disease, hypothermia, hypoventilation, regurgitation/aspiration
Paralysis	Hypoventilation
Head trauma	Brain damage from underlying disease, hypo/hyperthermia, hypoventilation, regurgitation/aspiration, hypertension, bradycardia
Acute spinal cord injury	Hypoventilation, spinal cord damage from underlying disease
Abdominal	
Hemorrhage	Hypovolemia
Large, painful bladder	Bladder atony, pain, azotemia, hyperkalemia, bladder rupture
Urinary tract rupture	Azotemia
Dystocia	Maternal exhaustion, fetal death
Gastric distention	Impaired venous return, impaired ventilation, impaired visceral perfusion, visceral ischemia/necrosis
Dehiscence, prolapse	Impaired visceral perfusion, visceral ischemia/necrosis
Other	
Hyperthermia/heat stroke	Elevated metabolism and further heat production, tissue damage (hemolysis, DIC[b], renal failure)
Hypoglycemia	Seizures, hyperthermia, brain damage
Hypocalcemia	Seizures, hyperthermia, hypotension

(Continued)

TABLE 1.1
(CONTINUED)

Hyperkalemia	Reduced contractility and cardiac output, cardiac arrest
Anemia	Decreased oxygen content and delivery
Snake bite[c]	Pain, local necrosis, DIC, hemolysis, paralysis
Burns, frostbite	Dermal injury
Blunt or penetrating trauma	Internal organ injury, hemorrhage
Fire, smoke inhalation	Dermal injury, upper/lower airway injury, carbon monoxide
Electrocution	Local tissue necrosis, arrhythmias, pulmonary edema
Glaucoma	Pain, loss of sight
Proptosis	Pain
Poisonings/intoxications[d]	Tremors, seizures, coma, coagulopathy, hemolysis, renal/hepatic failure

[a]Difficult breathing heralded by openmouthed breathing or gasping, extended head and neck, asynchronous chest wall and abdominal movement.
[b]Disseminated intravascular coagulation.
[c]Consequences depend upon the specific kind of snake.
[d]Consequences depend upon the specific poison/toxin.

TABLE 1.2
TREATMENT PRIORITIES IN EMERGENCY PATIENT

A. Airway and arterial bleeding
Provide patent airway, 100% O_2
Apply pressure to areas of active hemorrhage
B. Breathing
Auscult chest, characterize breathing pattern
Perform thoracocentesis or emergency tracheostomy if indicated
C. Circulation
Place IV catheter, obtain samples for PCV/TS, BUN, glucose, Na, K, blood gases, ± coagulation tests and blood smear, urinalysis
Treat for shock if cardiac failure is ruled out as the cause of poor perfusion Fluid guidelines for shock
Crystalloid resuscitation-hypovolemic shock
  Dog, 90 ml/kg/h—Administer in 25% increments and assess patient response
  Cat, 60 ml/kg/h—Administer as above
Acute blood loss, FCV <20%
  20 ml/kg fresh whole blood transfusion
Shock with head trauma or pulmonary contusions
  Minimize crystalloid fluid, 10–20 ml/kg IV maximum
  7.5% hypertonic saline, 5 ml/kg IV
  Small-volume resuscitation with colloids
    Give 5 ml/kg hetastarch or voluven (a colloid brand name) q 5–10 minutes until HR, color, pulses, and BP improve (generally up to 20 ml/kg)
Monitor PCV/TS q 20–30 minutes in trauma patients
Place abdominal compression bandage if dropping PCV indicates internal hemorrhage.
Transfusion ± surgery for uncontrollable hemorrhage
D. Disability assessment
Neurologic examination
  Brain, spinal cord, peripheral nerves

(Continued)

TABLE 1.2
(CONTINUED)

    Rule out lesions with poor prognoses
    Treat head trauma or spinal cord injury
  Musculoskeletal examination
    Antibiotics, cleaning, debridement for open fractures
    Splint, stabilize distal limb fractures
    Bandage, clean lacerations
    Radiograph when stable
  Administer analgesia following initial examination
E. Evaluate for abdominal injuries, urinary tract trauma, oliguria
  Abdominocentesis, diagnostic peritoneal lavage, radiographs, ultrasound
  Radiographic contrast studies
  Monitor urine output

B. Evaluate respiratory status
  1. Apnea is a sign of a central nervous system (CNS) lesion or a peripheral problem with the neuromuscular axis.
    a. If there are no breathing efforts, intubate and ventilate with 100% oxygen.
    b. If unable to intubate because of pharyngeal/laryngeal obstructions and the obstruction is not a foreign body that can be removed, try to intubate with a smaller-than-normal endotracheal tube. If unable to intubate with a small endotracheal tube, pass a stiff urinary catheter into the trachea and use it
      1) to insufflate oxygen;
      2) for high-frequency jet ventilation (connect the common outlet tube from an anesthetic machine to the catheter and use very brief flushes with the oxygen flush valve at a rapid rate); or
      3) as a stylet to guide the introduction of an endotracheal tube.
    c. If none of the above techniques work or appear unlikely to work on initial examination, an emergency tracheostomy can be performed (see p. 143) to bypass the obstruction.
  2. Breathing effort without air movement may be caused by complete upper airway obstruction (remove or bypass the obstruction [see II.B.1.b and c above]) or a severe pneumothorax (perform a thoracentesis [see p. 155]).
  3. Minimal breathing effort with little air movement may be caused by intracranial disease (hematoma, neoplasia, edema), spinal cord disease (disc prolapse, fibrocartilaginous embolism, neoplasia, trauma), a neuromuscular junction problem (myasthenia gravis, botulism, polyradiculoneuritis, paralytic tick envenomation, paralytic snake envenomation), or toxin overdose (ivermectin, moxidexin, baclofen, or drug causing respiratory depression).
    a. Intubate and
    b. Ventilate
  4. Increased breathing effort without much air movement may be caused by (Table 1.3):
    a. Upper airway obstruction (inspiratory low-pitched snoring or high-pitched squeaking). Remove or bypass obstruction.
    b. Lower airway obstruction (inspiratory/expiratory midpitched wheezing). Bronchodilators if bronchoconstriction; nebulization if exudate.
    c. Loss of chest wall integrity: flail chest (if severe, anesthetize, intubate, and ventilate); open pneumothorax (place a tube in the hole, squeeze the tissues around tube to make an airtight seal, and aspirate the air; or anesthetize, intubate, and ventilate).
    d. Abdominal filling disease (gastric distension, ascites, neoplasia, pyometra). Evacuate the offending problem.
    e. Pleural space filling disease (muffled lung sounds) may be caused by pneumothorax, hydrothorax, pyothorax, chylothorax, hemothorax. Diagnose and treat with thoracentesis.

TABLE 1.3
CHARACTERIZATION OF BREATHING NOISES AND PATTERNS

	Typical breathing noises	**Typical breathing pattern**
Upper-airway obstruction	Inspiratory low-pitched snoring or high-pitched squeaking[a]	Slow inspiratory times and rate; large tidal volumes[b]
Lower-airway obstruction	Inspiratory/expiratory midpitched wheezing	Slow inspiratory times and rate; large tidal volumes[b]
Pleural space filling	Muted, muffled breath sounds	Rapid, shallow
Pulmonary parenchymal	Crepitation; bubbling sounds	Rapid, shallow
Nonpulmonary causes[c]	Normal quality; increased volume	Rapid, deep

[a]Extrathoracic.
[b]In a compensated state; in a crisis, it will be rapid and shallow.
[c]Fever, metabolic acidosis, hypotension, pain, anxiety, pulmonary thromboembolism.

Diaphragmatic hernia: thoracentesis negative, abdomen palpates empty, radiographic evidence. Oxygen support and positive pressure ventilation if necessary until surgical correction.

f. Pneumonia, edema, contusions, or neoplasia: oxygen therapy or positive pressure ventilation.

5. Blood oxygenation

a. Cyanosis (blue or grey discoloration of mucous membranes) represents unoxygenated hemoglobin. Cyanosis is usually attributed to hypoxemia and, as such, represents severe hypoxemia. It is a late sign of hypoxemia and may not be manifested in animals that are anemic. It may also be due to sluggish peripheral circulation (severe hypovolemic shock, cardiac arrest) and methemoglobinemia.

b. Pulse oximetry

1) Pulse oximeters have to be able to detect a pulse to work and therefore require perfusion to the tissue where the probe is placed.

2) Pulse oximeters calculate oxyhemoglobin saturation from light absorption in arterial blood. Normal is about 98%; hypoxemia is represented by a value of less than 95%, and severe hypoxemia by a value of 90%.

3) Pulse oximeters can generate inaccurately low values. The indicated value is more likely to be correct if the indicated pulse rate matches that of the patient. Move the sensor to (several) new positions; record the highest reading. If the indicated values respond positively to increasing the inspired oxygen concentration, they are more likely to be correct. Evaluate other indices of blood oxygenation to corroborate the pulse oximeter-derived value (e.g., arterial blood gas measurement).

C. Evaluate cardiovascular status

1. Mucous membrane color

a. Red = vasodilation (sepsis, hyperthermia), cyanide, carbon monoxide

b. Pale = vasoconstriction (hypovolemia, heart failure, hypothermia), anemia

c. Cyanosis = unoxygenated hemoglobin

d. Brown = methemoglobinemia

2. Capillary refill time (CRT) (normal = 1–1.5 sec).

a. Vasoconstriction prolongs CRT: hypovolemia, heart failure, hypothermia, pain, vasoconstrictor drugs. Vasoconstriction may impair perfusion of peripheral tissues.

b. Vasodilation shortens CRT: sepsis, hyperthermia, vasodilator drugs. Vasodilation may cause hypotension.

3. Pulse quality is a determination of the height and width of the pulse pressure wave form. It is not a measure of blood pressure and cannot be used to define blood pressure. Pulse quality estimates must be indexed to patient size.

a. Tall, wide pulse waveforms ("bounding pulses") are attributed to large stroke volumes in vasodilated patients (sepsis, hyperthermia)

b. Tall, narrow pulse waveforms ("water-hammer pulses") are attributed to large stroke volumes in patients with very rapid diastolic runoff (patent ductus arteriosus, aortic insufficiency).

c. Small, narrow pulse waveforms ("weak, thready pulses") are attributed to small stroke volumes in vasoconstricted patients (hypovolemia, heart failure, hypothermia).

d. The term "pulse deficit" denotes pulses that are intermittently diminished or absent in association with an ausculted heartbeat or ECG depolarization (ventricular arrhythmias, premature atrial depolarization, atrial fibrillation).

e. Absent palpable pulses associated with an auscultable heartbeat may be caused by thromboembolism or severe hypotension; if there is no auscultable heart beat, it may represent cardiac arrest.

4. Heart rate

a. Tachycardia (large-breed dog >160; small-breed dog >180; puppy >200; cat >220) may be caused by hypovolemia, pain, hypoxemia, hypercapnia, hyperthermia, sepsis, anemia, stress, hyperthyroidism, or heart failure.

b. Bradycardia (dogs <60; cats <80) may be caused by hyperkalemia (urethral obstruction, hypoadrenocorticism, iatrogenic), organophosphate toxicity, hypothermia, hypoxia, and drug overdose (opioids, alpha$_2$-agonists), head trauma, atrioventricular conduction disturbances, and excessive vagal tone.

5. Hydration status

a. Dehydration represents a deficit of an extracellular crystalloid (water with variable concentrations of sodium). All dehydrated patients are hypovolemic, but there may be poor correlation between the two (hypovolemia may occur without dehydration) and, therefore, hydration status and volemic status must be evaluated separately. Dehydrated patients usually need crystalloid fluid replacement (see Fig. 1.1).

b. Edema represents an excess of extracellular crystalloid. Such patients may be hypovolemic (hypoproteinemia; increased vascular permeability) or hypervolemic (heart failure, iatrogenic). Edematous patients do not need any additional crystalloid fluids.

6. Jugular vein distention is a clinical assessment of venous blood volume and preload pressure to the heart. At normal venous volume, jugular veins are easy to distend by occluding flow at the thoracic inlet. Jugular veins that are distended without occluding flow at the thoracic inlet may represent excessive venous volume (hypervolemia). Jugular veins that are difficult/impossible to distend by occluding flow at the thoracic inlet may represent hypovolemia.

7. Arterial blood pressure is the most important determinant of cerebral and coronary perfusion. Normal values: systolic 100–160 mmHg, mean 80–120 mmHg, and diastolic 60–100 mmHg.

a. Hypotensive values of concern: systolic <80–100 mmHg, mean <60–80 mmHg, and diastolic <40–60 mmHg. Mean pressure is the most important when available. Excessive hypotension may be associated with inadequate cerebral and coronary perfusion. Hypotension should be treated first with preload volume augmentation (fluids) (Fig 1.1)(p. 10) and, if necessary, sympathomimetics (p. 32).

b. Hypertensive values of concern: systolic pressure >180 mmHg, mean pressure >140 mmHg. Excessive hypertension may be associated with edema, hemorrhage (epistaxis, retinal, intracranial, intrapulmonary), retinal detachment. Hypertension that is not compensatory for increased intracranial pressure should be treated with vasodilators (p. 92).

D. Evaluate intracranial status

1. Mentation should be evaluated at entry and periodically thereafter to assess the progress of the disease and treatment.

a. Bright and alert

b. Mildly obtunded (the animal is spontaneously aware of environmental

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                        ┌─────────────────────────────┐
                        │  Is the animal hypovolemic? │◄────────────┐
                    ┌──►└─────────────────────────────┘             │
                    │      ↙              ↘                         │
                    │  ┌───────┐        ┌────┐                      │
                    │  │  Yes  │        │ No │                      │
                    │  └───────┘        └────┘                      │
                    │   ↙      ↘          ↙     ↘                   │
          ┌──────────────────┐ ┌──────────────────┐   ┌─────────────────────┐
          │ Is the PCV < 20%?│ │ Is the PCV < 20%?│   │                     │
          └──────────────────┘ └──────────────────┘   │                     │
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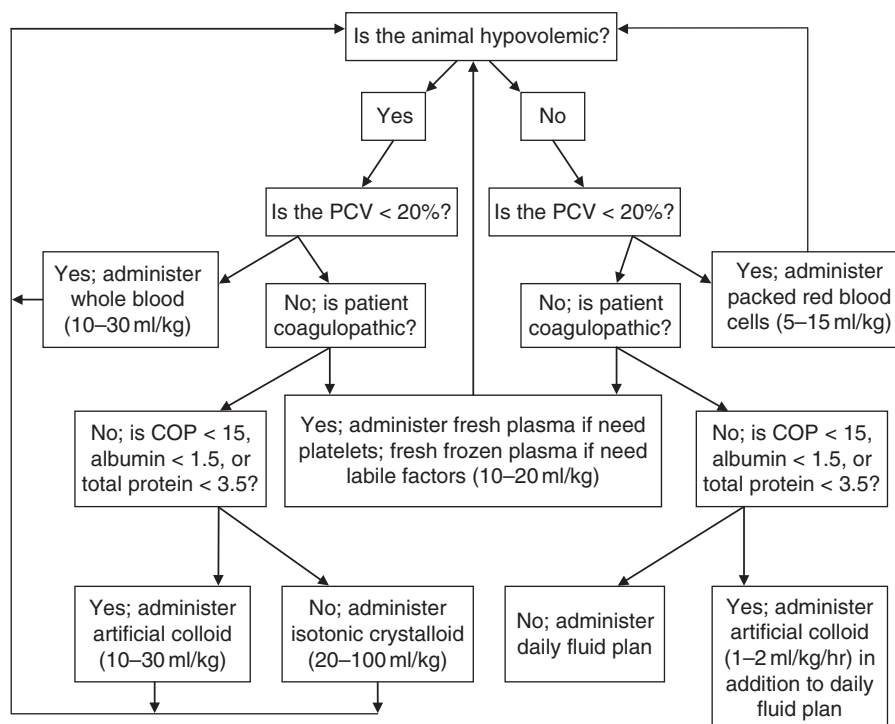

FIG. 1.1 Deciding which fluids to administer.

events but may "sleep" if there are no environmental stimuli). The judicious administration of tranquilizer drugs (phenothiazine, benzodiazepines) may, for example, produce this state.

c. Moderately obtunded (the animal mostly "sleeps" but will wake up and respond to loud noises or gentle physical stimulation). The judicious administration of sedative drugs (opioids) may, for example, produce this state.

d. Severely obtunded (the animal will only awaken to strong physical stimulation).

e. Comatose (the animal is unconscious and does not mentally respond to even the strongest of physical stimulations; withdrawal and hemodynamic reflexes may still be present).

f. Decreased mentation can be attributed to intracranial or extracranial causes. Intracranial causes are often associated with abnormal, localizing or lateralizing, cranial nerve signs. Extracranial causes are often associated with reduced cranial

nerve signs, which are usually bilaterally symmetrical.

2. Is there external evidence of trauma (skin abrasions or lacerations, epistaxis, cerebrospinal fluid (CSF) or blood in the external ear canal (hyphema or other hemorrhages, fractures)?

3. Eye signs: nystagmus, strabismus, bilateral miosis or mydriasis, anisocoria, hippus, menace reflex, palpebral reflex, pupillary light reflex, dazzle reflex?

4. Head tilt, torticollis, circling, head pressing, reluctance to be placed in some particular positions, positional nystagmus?

5. Sneeze reflex, gag reflex?

6. Brain stem involvement is characterized by unconsciousness; bilateral light-unresponsive miotic or mydriatic pupils; strabismus; absent physiologic nystagmus but present spontaneous or positional nystagmus, absent gag, swallow, and laryngeal reflexes; irregular breathing rhythms or apnea, decerebrate rigidity (extensor rigidity in all four appendages).

a. Cerebellar injury may also be associated with extensor rigidity but these animals are not comatose. Decerebellate rigidity carries a better prognosis than decerebrate rigidity.

b. Shiff-Sherrington syndrome is characterized by extensor rigidity of the forelimbs and flaccid paralysis of the rear limbs and denotes a severe spinal cord lesion between T2 and L4.

7. Treatment should be for the underlying disease process. Mannitol should be administered if the cranial nerve signs worsen or if the history suggests an event known to be associated with cerebral edema (hypoxia, hypoglycemia, trauma, prolonged seizures, neoplasia).

E. Evaluate spinal cord function

1. Check for pain or misalignment of vertebral column, spontaneous movement of front and hind legs, and tail. Evaluate front and hind pain perception, front and hind appendage reflexes (withdrawal, crossed-extensor), panniculus reflex, and anal tone and reflexes.

2. Animals with suspected traumatic spinal injuries should be taped to a solid transport stretcher suitable for radiographic imaging so that they cannot move or be moved prior to assuring an intact vertebral column.

F. Temperature

1. Hyperthermia

a. Fevers up to 40°C (104°F) could be an appropriate response to infection and should not be treated specifically. Hyperthermia in excess of 41°C (106°F) can be self-perpetuating and should be symptomatically treated. Hyperthermia in excess of 42°C (108°F) can be tissue damaging.

b. Common causes include infection, inflammation, excessive muscular activity (seizures or respiratory distress), and environmental (hot car, hot day, forced exercise, iatrogenic).

c. Active cooling is necessary in hyperthermia. Usually, wetting the animal down with room-temperature water (augments evaporative heat loss) and a fan (augments convective heat loss) is sufficient, along with room-temperature IV fluids. Stop active cooling when the temperature is about 1–1.5°C (2–3°F) above the desired goal. (Usually, stop cooling efforts when body temperature reaches 103°F.)

2. Hypothermia

a. Mild hypothermia (down to 35°C [95°F]) is of little medical concern although it may cause some discomfort and shivering for patients with normal mentation. It is usually adequate to cover the animal with blankets to prevent further heat loss, allowing the animal to warm itself. Moderate hypothermia (33–35°C [91–95°F]) may depress mentation (additive to drug sedative effects) and may require active rewarming. Severe hypothermia (30–33°C [86–91°F]) is associated with significant obtundation and requires active rewarming; such patients do not have sufficient metabolic activity to warm themselves. Very severe hypothermia (<30°C [<86°F]) may be associated with coma, arrhythmias, and coagulopathies.

b. Hypothermia is due to either excessive heat loss (environmental), insufficient heat production (impaired hypothalamic thermostasis in any critically ill patient), or both (general anesthesia).

c. Active rewarming applies a heat source to the patient.

1) Forced hot-air blankets

2) Circulating warm-water blankets

3) Infrared heat lamps (do not place too close to the patient because they will cause excessive skin heating)

4) Hot-water bottles placed around the patient (do not allow contact with the patient if water temperature exceeds 42°C [108°F]). Cover the patient and bottle to make a heat tent.

G. Stop or minimize external hemorrhage

1. Manual counterpressure should be applied immediately.

2. Compression bandages may work, but may only hide the hemorrhage until the bandage soaks through

3. Tourniquets, applied for too long, can cause ischemic damage of the appendage.

4. Pulsating arterial bleeders generally require surgical ligation to stop the hemorrhage.

H. Evaluate blood glucose

    1. Hypoglycemia is life threatening because the normal brain lives on glucose.

        a. Common causes include sepsis, glycogen storage diseases in toy breeds, insulinoma, iatrogenic.

        b. Severely hypoglycemic patients should receive a glucose bolus—0.5 g/kg diluted and given IV. Such patients may require a glucose infusion (0.1–0.25 g/kg/h or 2.5–5% added to IV fluids) to maintain acceptable blood glucose concentrations.

    2. Hyperglycemia is not, per se, life threatening but some human studies suggest that glycemic control (to near-normal levels) reduces septic complications and improves survival.

        a. Common causes include stress, diabetes mellitus, iatrogenic.

        b. Most important treatment consideration is effective management of the underlying disease process. Insulin may be necessary if the hyperglycemia does not respond to treatment of the underlying disease.

I. Packed cell volume

    1. Anemia reduces oxygen carrying capacity of the blood. In general, a packed cell volume of 20% has been used as a trigger for blood transfusion in animals with acute hemorrhage. Lower trigger thresholds can be used in animals with normal cardiac reserve, capable of increasing cardiac output to support oxygen delivery in the face of anemia (e.g., immune mediated anemia). Higher trigger thresholds should be used in animals with reduced cardiac reserve and reduced ability to increase cardiac output (sepsis, general anesthesia).

    2. Polycythemia increases blood viscosity and reduces cardiac output and tissue perfusion. Values above 60–70% in dehydrated patients should be treated with crystalloid fluid administration to rehydrate the patient and dilute the red blood cells. Values above 60–70% in nondehydrated patients should also receive clear fluids but a preemptive removal of whole blood may be necessary to prevent hypervolemia.

III. BRIEF HISTORY

A brief history should be obtained once initial lifesaving procedures have been implemented.

A. Obtain the reason for bringing the animal to the hospital. Ascertain (preferably in writing) whether the "bringing person" is the owner of the animal or not, and whether this person is willing to assume financial responsibility.

B. Upon completion of the primary survey, the owner should be informed of the current status of the animal, the immediate resuscitation plan, the expected outcome, and the estimated costs. Once informed, the owner must give consent to either continue or terminate the endeavor (preferably in writing).

C. Does the owner know what happened (what the owner knows for sure v. what the owner imagines might have happened)?

D. What signs have been manifested and how long have they existed?

E. Are any other animals showing similar clinical signs?

F. Are there any known medical conditions or medications?

G. To what poisons/toxins has the animal had access?

IV. THOROUGH SECONDARY SURVEY

Once initial life-saving procedures have been implemented, a more thorough secondary survey of the patient should be obtained. First, repeat the primary survey to determine what changes may have occurred as a consequence of the underlying disease process and the treatments so far implemented.

A. Respiratory

    1. Arterial partial pressure of oxygen (PaO_2) is a measure of blood oxygenation. Normal is 80–110 mmHg when the animal is breathing room air.

        a. Hyperoxemia is caused by enriched oxygen breathing.

        b. Hypoxemia is caused by low inspired oxygen (when attached to a malfunctioning anesthetic machine or breathing circuit), global hypoventilation (defined by $PaCO_2$), or lung dysfunction. Hypoxemia below about 60 mmHg should be treated.

            1) Hypoxemia is first treated symptomatically with oxygen. If oxygen therapy alone does not alleviate the hypoxemia, add positive pressure ventilation (PPV), positive end-expiratory

pressure (PEEP) or continuous positive airway pressure (CPAP).

   2) Once the hypoxemia has been alleviated, institute effective therapy of the underlying disease process. (See Mechanical ventilation, p. 185.)

2. Central venous partial pressure of oxygen (PvO_2) is a measure of tissue oxygenation. Peripheral venous PvO_2 is not reflective of central PvO_2 and cannot be interpreted in the context of this discussion. Central PvO_2 is determined by the balance between oxygen delivery and oxygen consumption. Normal is 40–50 mmHg.

   a. Values above 60 mmHg may indicate reduced oxygen consumption (hypothermia, sepsis) but may also occur with 100% oxygen breathing and general anesthesia.

   b. Values below 30 mmHg may indicate reduced oxygen delivery (poor cardiac output, low oxygen content).

3. Arterial partial pressure of carbon dioxide ($PaCO_2$) is a measure of alveolar minute ventilation. Normal is 35–45 mmHg (dog).

   a. Hypoventilation causes an increase in $PaCO_2$. Values above 60 mmHg should be treated with positive pressure ventilation.

   Hypercapnia causes cerebral vasodilation, which increases intracranial blood volume and intracranial pressure. In patients with intracranial disease, the $PaCO_2$ should be maintained below 45 mmHg.

   b. Hyperventilation causes a decrease in $PaCO_2$. Hypocapnia is usually not treated specifically; therapy is aimed at the underlying disease.

   c. $PvCO_2$ can be used as a surrogate marker of $PaCO_2$ because it is usually about 5 mmHg higher than $PaCO_2$. Although, $PvCO_2$ can be much higher than $PaCO_2$ in instances of poor tissue perfusion. Normal values are 40–50 mmHg.

   d. End-tidal PCO_2 can be used as a surrogate marker of $PaCO_2$ because it is usually about 5 mmHg lower than $PaCO_2$. Although end-tidal CO_2 values can be much lower with hypovolemia, hypotension, or pulmonary thromboembolism. Normal values are 30–40 mmHg.

4. Imaging procedures can be performed to further characterize abnormalities (radiography, computed tomography [CT], magnetic resonance imaging [MRI]).

5. Thoracentesis can be diagnostic or therapeutic. Cytologic examination should be performed on all aspirates.

6. Tracheal fluid aspiration when appropriate

   a. Cytologic examination will help differentiate exudates from transudates.

   b. Comparing total protein concentration or colloid osmotic pressure of undiluted samples with that of plasma may help differentiate a hydrostatic pulmonary transudate (congestive heart failure, volume overload) from a permeability pulmonary transudate (ARDS, sepsis, electric shock, noncardiogenic pulmonary edema).

B. Cardiovascular

1. Arterial blood pressure (ABP), if it was not done as part of the primary survey, should be done as part of the secondary survey.

   a. Indirect blood pressure can be measured using a circumferential cuff applied around an appendage to occlude blood flow, an aneroid manometer to measure the pressure in the cuff, and a blood flow detection device to measure blood flow beyond the cuff as the cuff pressure is decreased. The detection device could be a finger to palpate a pulse or a Doppler blood flow detector. Oscillometric devices interpret changes in intracuff pressure as the cuff is deflated and then display a value for systolic, diastolic, mean blood pressure, and heart rate.

   b. Direct blood pressure involves catheterization of an artery (dorsal metatarsal, femoral, tail, ear, ulnar, humeral), either percutaneously or via surgical exposure, which is then attached to a commercial transducer and a physiologic patient monitor.

   c. Normal values: systolic 100–160 mmHg, mean 80–120 mmHg, and diastolic 60–100 mmHg. The most important pressure is mean, which is the average area under the pulse pressure waveform and represents the average driving pressure to tissue perfusion.

   1) The primary determinants of systolic pressure are stroke volume and arterial compliance. The primary determinants of diastolic pressure are vascular resistance and heart rate.

   2) Directly measured systolic and diastolic pressures can be blunted by an

overdamped measuring system (a partial occlusion), and exaggerated by an underdamped measuring system (the harmonics of the measuring system match that of the patient's arterial system).

2. Central venous pressure (CVP) is a measure of preload pressure to the right heart. It is a surrogate marker of preload, which is end-diastolic (or presystolic) ventricular muscle stretch or end-diastolic ventricular volume.

    a. Normal CVP is 0–10 cm H_2O.

    b. The CVP can be up to midnormal range in a hypovolemic patient (representing venoconstriction around a reduced blood volume).

    c. A high-normal range CVP is a common endpoint of fluid loading; a high pressure being generally associated with a high preload.

    d. The CVP can be high in association with low preload in a heart with low compliance (hypertrophic cardiomyopathy, pericardial tamponade, endo/myo/epicardial fibrosis).

3. The base deficit/excess describes the metabolic component of the acid-base balance. Normal is 0 ± 4 mM/L.

    a. Surrogate markers of base deficit/excess include decreased standard bicarbonate concentration (bicarbonate concentration indexed to a PCO_2 of 40 mmHg), bicarbonate concentration, and total carbon dioxide concentration (most of which is bicarbonate).

    b. Metabolic acidosis can be caused by lactic acid (anaerobic glycolysis), ketoacids (insulin deficiency), phosphoric and sulfuric acids (renal failure), glycolic acid (ethylene glycol), bicarbonate losses (duodenal vomition, diarrhea), hypoadrenocorticism, renal tubular acidosis.

    c. Metabolic alkalosis can be caused by gastric vomition, furosemide administration, hypochloremia, hypokalemia, and metabolism of organic anions (lactate, acetate, gluconate, citrate).

4. Electrocardiogram should be obtained if arrhythmias (including bradycardia and tachycardia) were detected on the physical exam.

5. Ultrasound evaluation of cardiac and valvular performance.

C. Neurologic

    1. Determine the animal's ability to stand or walk (after spinal damage has been ruled out).

    2. Assess proprioception, and for lameness or abnormal gait.

    3. Serial neurologic exams should be performed on all animals with acute head or spinal cord injuries.

    4. Imaging procedures can be performed to further characterize abnormalities (radiography, computed tomography [CT], magnetic resonance imaging [MRI]).

D. The abdomen should be inspected for contusions, lacerations. It should be palpated for pain (which should be localized in so far as possible), fluid accumulation, and normal (urinary bladder, kidneys, spleen, liver, intestines) and abnormal masses (hematomas, neoplasias, GI foreign bodies, hernias).

    1. A diagnostic paracentesis (single site, four-quadrant thoracentesis, or a diagnostic peritoneal lavage) is indicated if free abdominal fluid is detected.

        a. If available, ultrasound guidance can be used to detect fluid pockets and obtain a diagnostic sample.

        b. If ultrasound is not available, the following procedure can be performed:

            1) Single site paracentesis (p. 227): Insert a 20/22 ga needle perpendicularly into the abdominal cavity at about the level of the midabdominal midline; gently aspirate.

            2) Four-quadrant paracentesis: If the single site paracentesis does not obtain a sample of fluid, one could attempt multiple site paracentesis, but avoid the area of the liver, stomach, spleen, and urinary bladder.

            3) Peritoneal lavage paracentesis (p. 228): If the multiple site paracentesis does not obtain a sample of fluid, one could instill approximately 20 ml/kg of a warmed isotonic crystalloid into the abdominal cavity via a preplaced intravenous catheter; role the animal to "wash" the peritoneal cavity; gently aspirate.

    2. All aspirates should be examined cytologically and cultured if infection is possible.

        a. High specific gravity and cell count suggest exudates.

b. High neutrophil counts suggest inflammation.

c. Intracellular bacteria denote septic peritonitis.

d. Bile salts and high bilirubin concentration suggest gall bladder/bile duct rupture.

e. High lactate and low glucose (compared to plasma) suggest sepsis.

f. High creatinine, urea nitrogen, potassium (compared to plasma) suggest urine.

3. Imaging procedures can be performed to further characterize abnormalities (radiography, ultrasound, computed tomography [CT], magnetic resonance imaging [MRI]).

E. Internal hemorrhage into a body cavity (hemothorax, hemoabdomen), into muscle or subcutaneous tissues (hematoma), or into organs (brain, lung contusions) may occur secondary to ruptured vessels or to coagulopathy.

1. Recognition of internal hemorrhage depends upon the site of hemorrhage.

a. Pleural space: increasing breathing effort; decreasing lung sounds; hypovolemia, which is poorly/transiently responsive to fluid therapy; decreasing hemoglobin concentration; decreasing blood oxygenation; increasing blood carbon dioxide. Diagnostics: auscultation, thoracentesis (p. 155), ultrasound, radiography. Therapy: chest drainage (p. 158), blood transfusions as necessary.

b. Abdomen: increasing abdominal girth and pressure; hypovolemia, which is poorly/transiently responsive to fluid therapy; decreasing hemoglobin concentration. Diagnostic paracentesis, ultrasound, and radiography may be indicated. Blood can be removed from the abdominal cavity if it has accumulated in sufficient quantity to cause impaired ventilation, if abdominal pressure exceeds 20–25 cm H_2O, or if the blood is needed for autotransfusion.

c. Hematoma: palpable enlarging mass. Therapy: apply counterpressure.

d. Brain contusion: deteriorating intracranial signs

e. Lung contusion: deteriorating pulmonary function

2. In all cases of hemorrhage, fluid therapy should be conservative but sufficient to reestablish a modest arterial blood pressure (60–80 mmHg mean) and tissue perfusion (decreasing lactate concentration or metabolic acidosis, capillary refill time of about 2 seconds). It is not necessary that cardiovascular parameters be normalized at this point. Aggressive fluid therapy could worsen the hemorrhage.

3. Coagulopathies should be ruled in or out in all cases of hemorrhage. Clotting problems can occur secondary to abnormalities of platelets, coagulation factors, and fibrinolysis.

a. Platelet counts are determined as part of a complete blood count and can be estimated from a blood smear by multiplying the number counted per high power field by 15,000. Bleeding secondary to thrombocytopenia is not expected until platelet numbers are far below 30,000.

b. Platelet function is determined by the presence or absence of petechiae or ecchymoses, by bleeding time, or by a commercial platelet function analyzer.

c. Problems within the coagulation cascade are determined by prolongation of prothrombin time, activated partial thromboplastin time, activated coagulation time, or whole blood coagulation time.

d. Fibrinolysis is assessed by D-dimer analysis. Primary fibrinolysis is thought to be very rare; it is normally associated with, and secondary to, activated coagulation.

e. Disseminated intravascular coagulation is a thrombotic syndrome heralded by thrombocytopenia, prolonged coagulation tests, and elevated fibrin degradation products and D-dimers.

F. Miscellaneous

1. Rectal examination to check for pelvic fractures, anal tone, or hemorrhage.

2. Debris should be removed from open wounds by copious lavage with sterile saline. Swabs for bacterial culture should be procured. Povidone iodine (1:9) or chlorhexidine (1:40) can be added to the lavage solution to aid decontamination. Dead or devitalized tissue should be removed from the wound as soon as possible to prevent further bacterial growth and contamination. Pack the wound with gauze pads moistened with one of the above solutions until definitive repair under general anesthesia can be accomplished.

3. Limb fractures should be immobilized to prevent further injury. Temporary splints can be made of rolled up magazines or newspaper, or

cardboard in the absence of more suitable materials. Heavily padded compressive bandages can be used for fractures below the elbow or stifle. A splint that extends over the shoulder or hip can be used to help stabilize a humeral or femoral fracture.

4. A ruptured urinary system (ureter, bladder, urethra) may be revealed by detection of free urine in the abdomen.

   a. A palpable bladder or a bladder visualized by radiography or ultrasound reinforces, but does not prove, the absence of a rupture.

   b. An excretory urogram (p. 272) or a positive contrast cystogram (p. 271) or urethrogram (p. 272) helps rule in/out rupture of a ureter, bladder, or urethra.

   c. Monitor urine output, patient demeanor, and plasma creatinine/BUN. A ruptured urinary system will be associated with abnormalities in 1–3 days.

G. Chemistry panel, complete blood count, and urinalysis should be measured as indicated.