Supportive therapy

ACID–BASE DISTURBANCES

Evaluation of the blood pH, bicarbonate (HCO₃⁻), Pₒ₂ and base excess may be very helpful in the assessment of a critically ill or injured animal. Arterial blood samples are necessary to evaluate the respiratory status and are the preferred sample, but venous blood samples are helpful to evaluate the status of the patient at the cellular level.

The normal arterial blood gas reference ranges for dogs and cats inspiring room air are listed in Table 1.1.

Evaluation of blood gas results is a process involving many steps.

1. Determine whether the pH is normal.
2. Evaluate the partial pressure of carbon dioxide (PₐCO₂) – the respiratory component.
3. Evaluate the bicarbonate concentration [HCO₃⁻] – the nonrespiratory component.
4. What is the primary disorder? The direction of the expected changes are shown in Table 1.2.
5. Is the secondary or adaptive response as expected? The expected responses are shown in Table 1.3.
6. Which disease process(es) is(are) responsible for the acid–base disorder?

RESPIRATORY ACIDOSIS

Respiratory acidosis or primary hypercapnia indicates hypoventilation and hypoxemia. The blood pH is decreased, the PₐCO₂ is increased and, with compensation, the HCO₃⁻ increases.

Sympathetic activation, increased cardiac output and possibly tachyarrhythmias may occur in the presence of moderately elevated PₐCO₂. As the PₐCO₂ increases, intracranial pressure and cerebral blood flow increase. Disorientation, narcosis, and coma may occur at extremely high PₐCO₂ levels (60–70 mm Hg).

Causes of respiratory acidosis include:

- Depression of the respiratory center
  - Associated with medications (inhalant anesthetics, opioids, barbiturates)
Table 1.3 Expected compensatory response in simple acid–base disturbances in dogs and cats*

<table>
<thead>
<tr>
<th>Disturbance</th>
<th>Primary change</th>
<th>Clinical guide for compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Dogs</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Each 1 mEq/L ↓ HCO₃⁻</td>
<td>P_{CO₂} ↓ by 0.7 mm Hg. P_{CO₂} does not change</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Each 1 mEq/L ↑ HCO₃⁻</td>
<td>P_{CO₂} ↑ by 0.7 mm Hg</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>Each 1 mm Hg ↑ P_{CO₂}</td>
<td>HCO₃⁻ ↑ by 0.15 mEq/L</td>
</tr>
<tr>
<td>Chronic</td>
<td>Each 1 mm Hg ↑ P_{CO₂}</td>
<td>HCO₃⁻ ↑ by Unknown</td>
</tr>
<tr>
<td>Long-standing‡</td>
<td>Each 1 mm Hg ↑ P_{CO₂}</td>
<td>HCO₃⁻ ↑ by 0.55 mEq/L</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>Each 1 mm Hg ↓ P_{CO₂}</td>
<td>HCO₃⁻ ↓ by 0.25 mEq/L</td>
</tr>
<tr>
<td>Chronic</td>
<td>Each 1 mm Hg ↓ P_{CO₂}</td>
<td>HCO₃⁻ ↓ by 0.55 mEq/L</td>
</tr>
</tbody>
</table>


†Data from cats are derived from a very limited number of cats.

‡More than 30 days.

§Exact degree of compensation has not been determined, but in cats with chronic respiratory alkalosis maintain normal arterial pH.
- Neurologic disease
  - Cervical spinal cord lesion
  - Brainstem lesion
- Neuromuscular disease
  - Myasthenia gravis
  - Botulism
  - Tetanus
  - Tick paralysis
  - Severe hypokalemia
  - Associated with medications or chemicals (organophosphates, aminoglycosides)
- Obstruction of large airways
  - Aspiration
  - Kinked or plugged endotracheal tube
  - Tracheal collapse
  - Brachycephalic syndrome
  - Laryngeal paralysis
  - Mass lesion (intraluminal or extraluminal)
  - Infiltrative lower airway disease (chronic obstructive pulmonary disease (COPD), asthma).

Treatment of respiratory acidosis is aimed at treatment of the underlying disorder, discontinuation or reversal of possible pharmacologic causes, establishment of an airway if needed, ventilation, and oxygenation.

**RESPIRATORY ALKALOSIS**

Respiratory alkalosis or primary hypocapnia indicates hyperventilation. The blood pH is increased, the $P_{aCO_2}$ is decreased and, with compensation, the $HCO_3^-$ decreases.

The $P_{aCO_2}$ can be falsely decreased by excessive dilution of the blood sample with heparin and by the presence of air bubbles in the sample. Arteriolar vasoconstriction occurs when $P_{aCO_2}$ decreases to less than 25 mm Hg and when arterial pH increases to 7.6. This results in a reduction in myocardial and cerebral blood flow.

Clinical signs are usually those of the underlying condition, although tachypnea may be noticed. Disorientation, seizures and cardiac arrhythmias may occur.

Causes of respiratory alkalosis include:

- Fear, excitement, anxiety, or pain
- Hyperventilation (either by the patient or by mechanical ventilation)
- Decreased inspired partial pressure of oxygen
- Pulmonary diseases such as pneumonia, pulmonary edema, pulmonary fibrosis, pulmonary thromboembolism
- Congestive heart failure
- Severe anemia
- Severe hypotension
- Central nervous system disease
- Stimulation of pulmonary stretch receptors or nociceptors
- Activation of central respiratory centers
- Medications such as xanthines (aminophylline), corticosteroids, salicylates
- Hepatic disease
- Hyperadrenocorticism
- Sepsis
- Heat stroke
- Exercise
- Following metabolic acidosis.

Treatment of respiratory alkalosis is aimed at the underlying disorder, decreasing fear or pain, decreasing mechanical ventilation, discontinuation of possible pharmacologic causes, and correcting hypoxemia.

### NONRESPIRATORY ACIDOSIS (METABOLIC ACIDOSIS)

Nonrespiratory or metabolic acidosis is based on the presence of decreased blood pH, decreased HCO$_3^-$, decreased base excess (BE), and with compensation the $P_{CO_2}$ decreases. The condition is considered severe if blood pH exceeds 7.1 or HCO$_3^-$ is less than 8 mEq/L. Although depression can occur with severe nonrespiratory acidosis and tachypnea may be observed, the clinical signs are usually associated with the underlying cause. Treatment is directed at the underlying disorder.

Causes of nonrespiratory acidosis include:

- Strong ion difference (SID) acidosis
  - Organic acidosis
    - Toxicities such as ethylene glycol or salicylate
    - Lactic acidosis
    - Uremic acidosis
    - Diabetic ketoacidosis
  - Dilutional acidosis (increased free water, associated with hyponatremia)
    - Due to a gain of hypotonic fluid (hypervolemia)
    - Congestive heart failure
    - Severe hepatic failure
    - Due to a gain of water (normovolemia)
    - Infusion of hypotonic fluids
    - Psychogenic polydipsia
    - Due to a loss of hypertonic fluid (hypovolemia)
    - Administration of diuretics
    - Hypoadrenocorticism
- Hyperchloremic acidosis
  - Renal failure
  - Total parenteral nutrition
  - Fluid therapy with 0.9% NaCl, 7.2% NaCl, or potassium chloride (KCl) supplemented fluids
  - Hypoadrenocorticism
  - Diarrhea
• Nonvolatile ion buffer acidosis
  – Hyperphosphatemic acidosis
    – Renal failure
    – Urethral obstruction
    – Uroabdomen
    – Intravenous phosphate supplementation
    – Administration of phosphate-containing enemas.

NONRESPIRATORY ALKALOSIS (METABOLIC ALKALOSIS)
Nonrespiratory or metabolic alkalosis is based on the presence of increased blood pH, increased $\text{HCO}_3^-$, increased base excess (BE), and with compensation the $P_{\text{CO}_2}$ increases. The condition is considered severe if blood pH exceeds 7.6. The clinical signs of nonrespiratory alkalosis are associated with the underlying cause. Treatment is directed at the underlying disorder.
Causes of nonrespiratory alkalosis include:
• Strong ion difference (SID) alkalosis
  – Hypochloremic alkalosis
    – Vomiting of stomach contents
    – Administration of loop diuretics, thiazides, or sodium bicarbonate
  – Chloride-resistant alkalosis
    – Hyperaldosteronism
    – Hyperadrenocorticism
  – Concentration alkalosis (from pure water loss, associated with hypernatremia)
    – Water deprivation
    – Vomiting or diarrhea
• Nonvolatile ion buffer alkalosis
  – Hypoalbuminemia
    – Protein-losing nephropathy
    – Protein-losing enteropathy
    – Hepatic failure.
Mixed disorders may occur and may involve combinations of two or three of any of the above disorders. If respiratory acidosis occurs simultaneously with nonrespiratory acidosis, the resulting pH tends to be much lower than with a solitary disorder. If respiratory acidosis occurs simultaneously with nonrespiratory alkalosis, the resulting pH may be within normal range.

STRONG ION APPROACH
According to this method of acid–base evaluation, three independent variables determine plasma pH.
1. $P_{\text{CO}_2}$ – an increase causes respiratory acidosis; a decrease causes respiratory alkalosis.
2. Strong ion difference (SID) – the sum of the completely dissociated plasma anions does not equal the sum of the completely dissociated plasma cations. These strong ions act together as one positively charged unit (SID) to affect plasma pH. The strong ions of importance in plasma
are sodium (Na⁺), potassium (K⁺), calcium (Ca²⁺), magnesium (Mg²⁺), chloride (Cl⁻), lactate, ketoacids (β-hydroxybutyrate, acetoacetate), and sulfate (SO₄²⁻). An increase of the SID, caused by an increased [Na⁺] or a decreased [Cl⁻], results in strong ion or metabolic alkalosis. A decrease of the SID, caused by a decreased [Na⁺] or an increased [Cl⁻], [SO₄²⁻], or organic anion concentration, results in strong ion or metabolic acidosis.

3. $A_{TOT}$ – the effect of nonvolatile buffer ions, which act as weak acids at physiologic pH, are albumin, globulin, and inorganic phosphate. An increased $A_{TOT}$ causes metabolic or nonvolatile ion buffer acidosis. A decreased $A_{TOT}$ causes metabolic or nonvolatile ion buffer alkalosis.

A gamblegram is usually used to evaluate the strong ion approach (Fig. 1.1).

There are six primary acid–base disturbances revealed by the strong ion approach:

1. Respiratory acidosis – increased $P_{CO_2}$ as seen with hypoventilation
2. Respiratory alkalosis – decreased $P_{CO_2}$ as seen with hyperventilation
3. Strong ion difference acidosis
   A. Dilutional acidosis (decreased [Na⁺])
      I. Hypervolemia (congestive heart failure, nephrotic syndrome, severe hepatic disease)
      II. Normovolemia (administration of hypotonic fluids, psychogenic polydipsia)
   III. Hypovolemia (third space losses, administration of diuretics, hypoadrenocorticism, diarrhea, or vomiting)
B. Hyperchloremic acidosis (increased [Cl⁻])
   I. Excessive loss via diarrhea of sodium
   II. Gain of chloride (administration of total parenteral nutrition, fluid
       therapy with KCl, NaCl (0.9%, 3%, 5%, or 7.5%))
C. Retention of chloride (hypoadrenocorticism, renal failure)
D. Organic acidosis (uremia, diabetic ketoacidosis, lactic acidosis, and
   ethylene glycol or salicylate intoxication)

4. Strong ion difference alkalosis
A. Concentrational alkalosis (increased [Na⁺])
   I. Hypotonic fluid loss (postobstructive diuresis, nonoliguric renal
       failure, vomiting)
   II. Pure water loss (diabetes insipidus, water deprivation)
B. Hypochloremic alkalosis (decreased [Cl⁻])
   I. Excessive gain of sodium relative to chloride as with the
      administration of sodium bicarbonate
   II. Excessive loss of chloride relative to sodium (thiazide or loop
       diuretic administration or vomiting of stomach contents)

5. Nonvolatile buffer ion acidosis (increased [ATOT])
A. Hyperalbuminemia (dehydration, water deprivation)
B. Hyperphosphatemia (tumor cell lysis, rhabdomyolysis or tissue
   trauma, administration of phosphate solutions or enemas, or
   decreased loss as occurs with renal failure, uroabdomen or urethral
   obstruction)

6. Nonvolatile buffer ion alkalosis (decreased [ATOT] due to
   hypoalbuminemia)
A. Excessive loss of albumin via protein-losing enteropathy or protein-
   losing neuropathy
B. Sequestration of albumin into inflammatory effusions or into tissues
   via vasculitis
C. Decreased albumin production due to malnutrition, starvation,
   chronic hepatic disease or the acute phase response of inflammation.

**OXYGEN THERAPY**

**HYPOXEMIA**

There are five causes of hypoxemia:

1. Hypoventilation
   A. Central nervous system disease (cervical spinal cord lesion above the
      fifth cervical vertebrae or a brainstem lesion)
   B. Neuromuscular disease (tetanus, botulism, tick paralysis,
      polyradiculoneuritis, myasthenia gravis, neuromuscular blocking
      agents)
   C. Medications that depress respiration (inhalant anesthetics,
      barbiturates, narcotics)
   D. Chest wall injury (fractured ribs, flail chest, pleural space disease,
      postoperative thoracotomy)
   E. Upper airway obstruction (aspiration, brachycephalic syndrome, tracheal
      collapse, laryngeal paralysis, mass, occluded endotracheal tube)
2. Decreased partial pressure of inspired oxygen (fraction of inspired oxygen (FiO₂))
   A. High altitude
   B. Faulty technique for inhalation anesthesia administration (inadequate minute ventilation, failure of the carbon dioxide filter or removal system)

3. Ventilation – perfusion mismatch (V/Q mismatch)
   A. Asthma
   B. Bronchitis
   C. Chronic obstructive pulmonary disease
   D. Pulmonary embolism

4. Diffusion impairment
   A. Diffuse pulmonary interstitial disease
   B. Vasculitis
   C. Emphysema
   D. Pneumonia
   E. Severe pulmonary edema

5. Right-to-left shunt
   A. Atelectasis
   B. Anatomic shunt with right-to-left blood flow (patent ductus arteriosus, ventricular septal defect, atrial septal defect, tetralogy of Fallot)

**ASSESSMENT OF HYPOXEMIA**

Along with adequate cardiac output, the oxygen content of arterial blood must be adequate to deliver sufficient oxygen to cells. Hypoxemia is the condition in which there is an inadequate oxygen level in the blood. Hemoglobin transports the majority of oxygen to cells. The normal hemoglobin level is 13–15 g/dL. Ideal oxygen delivery is thought to require a hemoglobin level of at least 10 g/dL, which corresponds to a packed cell volume (PCV) of about 30%, although 22–25% is usually adequate in healthy patients. One gram of hemoglobin (Hb) binds 1.34 mL of oxygen (98% of the oxygen content of blood) when it is fully saturated. \( SaO₂ \) is the saturation of hemoglobin with oxygen; \( PaO₂ \) is the partial pressure of oxygen dissolved in arterial plasma. The solubility coefficient of oxygen in plasma at body temperature \( = 0.003 \).

By plugging these numbers in the following equation, the patient’s arterial blood total oxygen content (\( CaO₂ \)) can be calculated:

\[
CaO₂ = ([Hb] \times SaO₂ \times 1.34) + (0.003 \times PaO₂)
\]

\( PaO₂ \) can be measured by a blood gas analysis machine from an arterial blood sample obtained in an appropriate manner. Excessive heparin volume and air exposure to the sample should be avoided. Hypoxemia (inadequate oxygen level in blood) is a partial pressure of oxygen in arterial blood (\( PaO₂ \)) less than 80 mm Hg. Visible cyanosis occurs at \( PaO₂ \) levels of 40 mm Hg and requires an [Hb] of 5 g/dL of deoxygenated Hb.

The alveolar air equation allows assessment of the quantity of oxygen in the alveolus. The alveolar air equation is:

\[
PAO₂ = (FiO₂ \times [PB - P_{H₂O}]) - PaCO₂/RQ
\]
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\[ PA_{O_2} \] = partial pressure of oxygen in alveolar air
\[ FiO_2 \] = fraction of inspired oxygen (21% [0.21] on room air)
\[ PB \] = barometric pressure (about 760 mm Hg at sea level, decreases with increases in altitude)
\[ P_{H_2}O \] = pressure of water vapor (about 50 mm Hg)
\[ RQ \] = respiratory quotient = CO₂ production/oxygen consumption, usually 0.8 or 0.9
\[ PaO_2 \] = measured value which has a limited impact on \( PaO_2 \) and will only cause hypoxemia in an animal breathing room air.

**A–a gradient**

Abnormalities of ventilation and perfusion can be assessed by determining the difference of the PO₂ of alveolar gas and arterial blood, known as the “A–a gradient”. \( PaO_2 \) should always be less than \( PAO_2 \).

\[
\text{Alveolar–arterial (A–a) gradient} = \text{PAO}_2 - \text{PaO}_2
\]

- <10–15 = normal
- >15 = impaired pulmonary oxygenation of blood
- >30 = severely impaired pulmonary gas exchange

Hypoxemia with a normal A–a gradient occurs with decreased \( FiO_2 \) and hypoventilation. If a patient has hypoxemia due to hypoventilation, an increase of the \( FiO_2 \) to 30% or more will usually remedy the situation. Hypoxemia with an increased A–a gradient occurs with conditions that cause diffusion impairment, V/Q mismatch, and shunts.

**\( PaO_2 \): \( FiO_2 \) ratio (or P:F ratio)**

Comparing the ratio of these values allows for a quick evaluation of oxygenation. Because \( PaCO_2 \) is not measured, it is less accurate than the A–a gradient.

\[
\text{\( PaO_2 \): \( FiO_2 \) = 500 is normal}
\]

- 300–500 indicates mild disease
- 200–300 indicates moderate disease
- <200 indicates severe disease

\( FiO_2 \times 5 \)

A rapid estimation of pulmonary oxygenation ability can be made by multiplying the \( FiO_2 \) by 5, which should approximate the \( PaO_2 \) in a normal patient at sea level. \( FiO_2 \) of 20% (room air) × 5 = \( PaO_2 \) of 100 mm Hg; \( FiO_2 \) of 100% (with 100% oxygen supplementation) × 5 = 500 mm Hg.

**PULSE OXIMETRY**

The saturation of hemoglobin with oxygen (\( SaO_2 \)) is the main factor affecting the amount of oxygen presented to the tissues in a patient with normal cardiovascular function. \( SaO_2 \) measured by a pulse oximeter is \( SpO_2 \). Two wavelengths of light are emitted by the probe, which differentiates oxygenated from deoxygenated hemoglobin. The measurement is inaccurate in the
presence of hypoxemia, poor perfusion, hypothermia, vasoconstriction, cardiac arrhythmias, increased pigmentation, abnormal hemoglobin and movement. Jaundice has no significant effect on \( \text{SpO}_2 \).

An \( \text{SpO}_2 \) of 98% corresponds to a \( \text{PaO}_2 \) of 100–500 mm Hg.

An \( \text{SpO}_2 \) of 95% corresponds to a \( \text{PaO}_2 \) of 80 mm Hg and is mild hypoxemia.

An \( \text{SpO}_2 \) of 90% corresponds to a \( \text{PaO}_2 \) of 60 mm Hg and is severe hypoxemia.

Carboxyhemoglobin is read as oxyhemoglobin, resulting in a falsely high \( \text{SpO}_2 \).

Methemoglobin causes the \( \text{SpO}_2 \) to read 85% regardless of the real \( \text{SpO}_2 \).

**VENOUS \( \text{P}_2 \)**

Venous partial pressure of oxygen (\( \text{PV}_2 \)) can provide an approximation of tissue oxygenation. A \( \text{PV}_2 \) of <30 mm Hg in a sample obtained from a central vein indicates increased oxygen consumption or decreased oxygen deliver. An estimation of ventilatory status can be made from a venous \( \text{PCO}_2 \) (\( \text{PV}_2 \)), which is usually 3–6 mm Hg higher than \( \text{PaCO}_2 \).

**SUPPLEMENTAL OXYGEN THERAPY**

Oxygen supplementation is needed if \( \text{PaO}_2 \) is less than 60–80 mm Hg, \( \text{SpO}_2 \) is less than 92% or the patient exhibits signs of hypoxemia. There are several methods to enrich the oxygen in the patient’s environment. Some of the more common ways are listed. To avoid oxygen toxicity, an \( \text{FiO}_2 \) of >60% should not be administered for more than 24–72 hours.

1. Flow-by
   - By holding oxygen tubing within 2 cm of a patient’s nostril or mouth, and running the oxygen at 2–3 L/min, the \( \text{FiO}_2 \) can often be increased to 25–40%.
   - Placing a loose plastic bag over the patient’s head like a tent and positioning the oxygen tubing under the bag may improve patient compliance and provide similar results.

2. Mask
   - An \( \text{FiO}_2 \) of up to 60% can be obtained by applying a tight-fitting face-mask onto a patient’s muzzle and an oxygen flow rate of 8–12 L/min.
   - Carbon dioxide, humidity and temperature may increase within the mask to hazardous levels, requiring frequent venting of the closed system.
   - Loose-fitting face-masks may require an oxygen flow rate of 2–5 L/min.
   - Many conscious patients will not accept a face-mask, especially for prolonged periods. Struggling and patient resistance will compound hypoxemia and should be avoided.

3. Hood or Elizabethan collar (Fig. 1.2)
   - An Elizabethan collar can be modified to allow supplemental administration of oxygen by covering the majority of the front of the collar with clear plastic wrap.
**FIG 1.2 Elizabethan collar hood oxygenation** Apply an Elizabethan collar that extends past the patient’s nose about 1.5–2 inches (4–5 mm). Thread a 5–12 Fr. red rubber catheter into the collar. Secure the end of the catheter inside the Elizabethan collar with tape so that it lies near the patient’s nose. Apply clear plastic food wrap over the front of the Elizabethan collar, leaving a 1.5–2 inch gap at the top to allow carbon dioxide to escape. A single strip of porous tape may be used to secure the top of the plastic wrap if desired. Secure the plastic wrap to the outside of the Elizabethan collar with tape. Attach an oxygen line to the outer end of the red rubber catheter and administer oxygen.

B. It is important to leave a small space open in the front to allow carbon dioxide and moisture to escape.

C. After the collar is applied to the patient, oxygen tubing is inserted from the back so that the tip of the tubing is near the nose and the tube is secured with tape.

D. An $F_{I\text{O}_2}$ of 30–40% can be obtained with an oxygen flow rate of 0.5–1 L/min.

E. Commercial hoods are also available.

F. Most patients will tolerate hood oxygenation, especially if the collar is long enough to keep the plastic a comfortable distance from the nose.

4. Nasal (Figs 1.3, 1.4)
A. Depending upon whether the patient is panting or mouth breathing, respiratory rate, and patient size, an oxygen flow rate of 50–150 mL/kg/min can provide an $F_{I\text{O}_2}$ of 30–60%.

B. Nasal oxygen should be humidified to avoid excessive irritation to nasal tissues.

C. Contraindications to nasal oxygenation include
   I. Increased intracranial pressure or an intracranial mass lesion
   II. Nasal or facial trauma
FIG 1.3 **Equipment for administering nasal oxygen** Intravenous tubing can be attached to an infusion bottle or other sterile bottle that is filled with sterile water. Oxygen is administered through water and IV tubing connected to the nasal catheter. An oxygen flow rate of 50–75 mL/kg will provide an oxygen concentration of 40–60%.

FIG 1.4 **Placement of a nasal oxygen catheter** Note that the tubing is brought up over the head between the eyes and sutured to the skin above the nose, on the forehead, and then draped behind the ears. The tubing is also secured with a ‘Chinese fingerlock’ suture.

III. Epistaxis
IV. Nasal mass
V. Laryngeal obstruction
VI. Coagulopathy.

D. Methods of nasal oxygenation
   I. Human cannulas
      a. The small two-pronged nasal cannulas commonly used in human hospitals may be tolerated by many patients.
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b. Placement is easy and the supplemental oxygen provided via this route may be beneficial.

II. Single nasal catheter
   a. A nasal catheter is quick and easy to place.
   b. Position the patient in either sternal or lateral recumbency.
   c. Instill several drops of dilute 2% lidocaine or proparacaine into the nostril while holding the patient’s nose upward.
   d. A 5-10 Fr. red rubber or polypropylene catheter is commonly used.
   e. Measure the catheter by placing the tip of the catheter at the medical canthus of the eye then mark the length on the catheter with a permanent marker at the lateral aspect of the nostril.
   f. Lubricate the catheter tip with water-soluble lubricant.
   g. Hold the catheter as close to the tip as possible with one hand as close to the nostril as possible; hold the patient’s nose with the other hand.
   h. Direct the catheter ventrally and medially into the nostril, to the mark on the catheter.
   i. Place a stay suture through the lateral aspect of the nares to secure the catheter.
   j. Place the catheter up the center of the muzzle between the eyes for dogs and to the side of the face for cats, avoiding the whiskers, and suture in place with 2–3 more sutures.
   k. Place an Elizabethan collar on the patient.
   l. Administer oxygen at flow rates of 50–100 mL/kg/minute.
   m. If additional oxygen is required, place another nasal catheter in the other nostril.

III. Bilateral nasal catheters enable the $F_{iO_2}$ to be increased to a higher level than at lower oxygen flow levels through a single nasal catheter.

5. Nasopharyngeal catheterization may enable the $F_{iO_2}$ to be increased to a higher level than with nasal catheterization.
   A. The procedure for placement is the same as nasal catheterization, except the catheter is introduced to the level of the ramus of the mandible rather than the medial canthus of the eye.
   B. For placement of a nasopharyngeal catheter, it is helpful to pinch the dorsolateral portion of the external nares medially while pushing the nasal philtrum dorsally.

6. Endotracheal intubation
   A. Use a laryngoscope to perform the procedure as gently as possible and avoid inducing laryngeal trauma or a vagal response.
   B. Use a low-pressure high-volume cuffed endotracheal tube if possible.
   C. For cats, a small volume of lidocaine may be squirted onto the larynx to decrease laryngospasm.
   D. Secure the endotracheal tube in place. A section of IV line, a piece of gauze, rubber bands and other items have been used.
   E. Ensure proper placement of the endotracheal tube by palpation of a single tube in the neck, visualization of the tube entering the trachea through the oropharynx, or assessment of end tidal carbon dioxide.
F. The endotracheal tube should be connected to an oxygen source such as an anesthetic machine circuit and oxygen should be administered.

G. The cuff should be gently inflated until the airway exterior to the tube is occluded.

H. An $F_{\text{I}O_{2}}$ of 100% may be provided to the patient with a sealed system.

I. Maintenance of an endotracheal tube requires sedation to obliterate the gag reflex. If the patient requires continued intubation to maintain a patent airway, a tracheostomy may be indicated.

J. Complications of endotracheal intubation include:
   I. Airway obstruction from kinking or clogging of the tube resulting in hypoxemia
   II. Pressure-induced tracheal necrosis, which may lead to pneumomediastinum and subcutaneous emphysema
   III. Tracheal laceration, which may lead to pneumomediastinum and subcutaneous emphysema
   IV. Intubation of a mainstem bronchus rather than the trachea resulting in overinflation of one lung lobe, impaired gas exchange, and possible airway rupture
   V. Pressure necrosis of the gingiva, lips and tongue
   VI. Laryngeal trauma
   VII. Excessive lingual swelling
   VIII. Increased intracranial pressure
   IX. Increased intraocular pressure.

7. Tracheostomy tube placement (Fig. 1.5)

A. Indications
   I. Severe upper airway obstruction
   II. Severe upper airway trauma

**FIG 1.5** Procedure for performing an emergency tracheostomy (A) With the patient in dorsal recumbency, quickly clip and prep the neck just distal to the larynx. With a scalpel, make a 2-3 cm incision through the skin, subcutaneous tissue and fascia on the ventral midline. Separate the muscles with scissors and forceps to expose the trachea. (B) Make an incision between tracheal rings that is one-quarter to one-third the circumference of the trachea. With the scalpel handle, separate the tracheal rings and insert a tracheostomy or endotracheal tube, directed caudally down the trachea. Secure the tube in place with umbilical tape tied around the neck.
III. Laryngeal paralysis
IV. Long term positive pressure ventilation
B. Contraindications
   I. Tracheal injury
   II. Tracheal obstruction distal to the tracheostomy site
C. Procedure
   I. Ideal situations allow for general anesthesia and placement of an endotracheal tube.
   II. The patient should be positioned in dorsal recumbency with the neck extended and elevated from the table with a towel or other padding.
   III. The ventral cervical region is clipped and routine surgical preparation is performed, time allowing.
   IV. A ventral cervical midline incision is made from the cricoid cartilage toward the sternum for 2-5 cm in length.
   V. With lateral retraction and blunt dissection, the sternohyoid muscles are separated along midline.
   VI. A transverse incision less than 50% of the tracheal circumference is made in the annular ligament between the fourth and fifth or third and fourth tracheal rings.
   VII. A tracheostomy tube should be inserted through the incision into the trachea, with the end directed ventrally.
      a. A cuffed tracheostomy tube with an inner cannula is ideal, but an uncuffed tube, tube without an inner cannula, or a modified endotracheal tube may be used.
      b. The tube size can be estimated by measurement of the inner tracheal diameter on a lateral cervical radiograph.
   VIII. A long suture loop should be placed around a tracheal ring above and below the incision to facilitate future replacement of the tube.
   IX. The muscle and skin should be sutured to decrease the size of the incision.
   X. The tube should be secured by tying around the neck with umbilical tape or suturing the tube in place.
   XI. The patient may be provided with supplemental oxygen by either increasing the oxygen concentration of the surrounding air, as in an oxygen kennel, or anesthetic tubing may be connected directly to the tracheostomy tube.
   XII. Daily tracheostomy tube care is essential to maintain a functional tube.

8. Oxygen kennel
   A. In addition to commercial oxygen kennels, which control humidity, temperature, and oxygen concentration, many other items can be substituted including a human pediatric incubator, a Plexiglas box, a Plexiglas door tightly fit onto a regular kennel, and, in the absence of anything else in an emergency situation, a regular kennel door can be covered with plastic wrap and an oxygen line can be run into the kennel to increase the $FiO_2$. 
B. Depending upon the kennel, patient size, frequency of opening the kennel door, and oxygen flow rate, an $F_iO_2$ of 40–60% can be maintained.

C. An oxygen kennel allows improvement of the $F_iO_2$ with minimal patient stress. When a dyspneic, distressed cat or small dog is brought in on emergency, it is often helpful to place the patient into an oxygen kennel, with oxygen supplementation, for several minutes to allow the patient to calm down, even if ultimately another method of oxygen supplementation will be provided to the patient.

D. Problems associated with oxygen kennels include
   I. They are expensive to purchase and to maintain.
   II. They require more oxygen than other methods.
   III. Hyperthermia commonly occurs.
      a. Placement of ice packs in the kennel but not on the patient may be helpful.
      b. Ice packs may also be placed in or around the humidifier.
   IV. Respiratory sounds such as stridor or stertor are muffled to the outside observer.
   V. Hands-on assessment of the patient requires opening of the oxygen kennel door and subsequent decrease in $F_iO_2$.

9. Hyperbaric oxygen
   A. The administration of hyperbaric oxygen requires a specialized commercial chamber and supra-atmospheric pressure (>760 mm Hg).
   B. The chamber generates an $F_iO_2$ of 100% and allows oxygen to diffuse readily into tissues.
   C. The indications for hyperbaric oxygen administration include:
      I. Osteomyelitis
      II. Burns
      III. Severe soft tissue infections.
   D. Hyperbaric chambers are usually not used in the treatment of patients with hypoxemia and are not often used in veterinary medicine.

**FLUID THERAPY**

There are many things to consider when evaluating the need and method of fluid therapy. Questions that should be answered include:

1. Does the animal require fluid therapy?
2. What type of fluid should be used?
3. Which route should be used?
4. How much should be given and over what period of time?
5. For how long should therapy continue?

Fluid therapy is beneficial for patients in shock or suffering from dehydration, or as a carrier for dilute intravenous medications. The signs of shock include:

1. Tachycardia
2. Pale oral mucous membranes
3. Weak or bounding peripheral pulses
4. Prolonged capillary refill time
5. Altered mentation.

Dehydration occurs when fluid losses exceed fluid intake. The signs of dehydration include:

1. Decreased skin turgor – although subjective, this assessment provides a rough idea of fluid losses in the patient. The skin over the lumbar region is pinched into a fold and the time required for it to return to normal position is evaluated. Obese animals may appear well hydrated. Emaciated and older animals may appear more dehydrated.

2. Retraction of the eye
3. Dry or tacky oral mucous membranes
4. Urine specific gravity should be high (>1.045) in a dehydrated dog or cat with normal renal function.

5. Serial evaluation of body weight is the best indicator of hydration status.
   A. A loss of 1 kg of body weight = 1 liter fluid deficit.
   B. Fluid ‘lost’ to third spacing (ascites, pleural effusion) doesn’t decrease body weight.
   C. Approximately 60% of total body weight in normal animals is fluid. This varies with age, metabolic status and the percentage of body fat:
      I. 10 kg dog = 6 kg of water
      II. 40 kg dog = 24 kg of water.

The packed cell volume and total solids (TS) increase with all types of fluid losses excluding hemorrhage (See Table 1.4.). The normal canine PCV = 38–55%, normal feline PCV = 29–45% and normal TS = 6.0–8.0 g/dL.
FLUID TYPES

1. Crystalloids are water-based salt and/or sugar solutions with electrolytes added that resemble the extracellular fluid (ECF) compartment. Sodium ions \([\text{Na}^+]\) are the major component. Within 30 minutes of administration, 75% of crystalloids leave intravascular space. Their primary effect is on the interstitial space, therefore they are recommended for replacing interstitial fluid losses (i.e. dehydration). Examples include lactated Ringer’s solution (LRS), Normosol R or M, Plasmalyte, 0.9% NaCl (saline) and dextrose 5% in water (D5W).

Maintenance solutions (Normosol M) have less sodium and more potassium than replacement (Normosol R) solutions (See Table 1.5.).

Selection of the crystalloid fluid should be based on \([\text{Na}^+]\) and \([\text{K}^+]\), osmolality, and pH. Patients should be infused with fluids that match the volume and electrolyte composition of fluid that has been lost.

A. Hypotonic fluids have an osmolality less than serum. Examples include 0.45% NaCl, 2.5% dextrose in 0.45% saline, and D5W. Since dextrose is rapidly oxidized to water and CO₂, giving 5% dextrose is equivalent to administering ‘free water’.

### Table 1.5 Crystalloid solutions

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Osmolality (mOsm/L)</th>
<th>pH</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>Mg</th>
<th>Glucose (g/L)</th>
<th>Buffer (mOsm/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>300</td>
<td>7.4</td>
<td>145</td>
<td>145</td>
<td>5</td>
<td>5</td>
<td>3</td>
<td>24</td>
<td>bicarbonate</td>
</tr>
<tr>
<td>5% Dextrose in water (D5W)</td>
<td>252</td>
<td>4.0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>2.5% Dextrose in 0.45% NaCl</td>
<td>280</td>
<td>4.5</td>
<td>77</td>
<td>77</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>0.9% NaCl</td>
<td>308</td>
<td>5.0</td>
<td>154</td>
<td>154</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>LRS</td>
<td>272</td>
<td>6.5</td>
<td>130</td>
<td>109</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>28 lactate</td>
<td></td>
</tr>
<tr>
<td>Plasmalyte 148</td>
<td>294</td>
<td>5.5</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>27 acetate/23 gluconate</td>
<td></td>
</tr>
<tr>
<td>Normosol R</td>
<td>296</td>
<td>6.4</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>27 acetate/23 gluconate</td>
<td></td>
</tr>
<tr>
<td>Normosol M in D5W</td>
<td>364</td>
<td>5.5</td>
<td>40</td>
<td>40</td>
<td>13</td>
<td>0</td>
<td>3</td>
<td>50 acetate</td>
<td></td>
</tr>
<tr>
<td>3% NaCl</td>
<td>1026</td>
<td>513</td>
<td>513</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>5% NaCl</td>
<td>1712</td>
<td>855</td>
<td>855</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>7.2% NaCl</td>
<td>2400</td>
<td>1232</td>
<td>1232</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>
I. These fluids should not be administered subcutaneously (could cause electrolyte imbalance).

II. Caloric needs cannot be maintained with 5% dextrose except in very small animals, as 1 liter of 5% dextrose supplies only 200 kcal.

B. Isotonic fluids have an osmolality closest to serum (extracellular fluid), about 290–310 mOsm/L, so they do not change in cell volume. These fluids are recommended for maintenance needs and shock therapy. Examples: LRS, 0.9% NaCl, whole blood, synthetic colloids.

C. Hypertonic fluids have greater osmolality than extracellular fluid, and create a large osmotic gradient – they cause rapid movement of water into the vascular space. These fluids can be used to treat shock, since they draw fluid from the interstitial space, which then redistributes out of the ECF compartment just as quickly as other crystalloids.

I. Hypertonic saline is available as NaCl 3%, 7.2%, 7.5% and 23%. The 23% solution should always be diluted prior to administration.

II. The advantage is the small volume required to resuscitate, which allows rapid resuscitation.

III. The recommended dose for the treatment of shock is hypertonic saline (7.2% NaCl), 3–5 mL/kg in dogs or 2–4 mL/kg in cats IV over 10 minutes in shock.

IV. Rapid administration of hypertonic saline may cause vagal mediated hypotension and bradycardia.

V. Hypertonic saline administration may provide rapid restoration of arterial blood pressure, higher blood pressure, a greater increase in cardiac contractility and cardiac output, improved blood flow and oxygen delivery to tissues. Hypertonic saline reduces endothelial swelling, microvascular permeability, and tumor necrosis factor levels. It also reduces intracranial pressure.

VI. Hypertonic saline is recommended for the treatment of circulatory shock, head and spinal trauma.

VII. Contraindications for hypertonic saline administration include dehydration, volume overload, hypernatremia, hyperosmolality, ventricular arrhythmias, and uncontrolled hemorrhage.
2. Colloids are fluids with large molecular weight particles that cannot readily cross capillary membranes, thus they are restricted to plasma, where they contribute to onctic pressure and expand the intravascular volume. Colloids contain negatively charged molecules (anions), which, by electrostatic attraction, retain cations (positively charged sodium ions) within the intravascular space. Water molecules follow the sodium ions, which results in expanded intravascular volume. The osmotic pressure generated by plasma proteins or by colloids in solution is called the colloid onctic pressure (COP). Osmotic pressure is proportional to the number of molecules present, rather than their size. RBCs, WBCs and platelets do not contribute to COP. Albumin makes up 60–70% of the COP with the remainder comprised of globulins and fibrinogen.

Natural colloids include: fresh whole blood, fresh frozen plasma (primarily contribute albumin).

Synthetic colloids include: Hetastarch, Pentastarch, Dextran 70, Oxyglobin (See Table 1.6.).

A. Albumin

I. Albumin is the primary component in plasma responsible for maintaining intravascular onctic pressure.

II. Normal albumin values for the dog and cat are 2.9–4.3 g/dL.

III. Hypoalbuminemia is a poor prognostic indicator in human medicine. If albumin values decrease below 1.5 g/dL, endothelial leakage of intravascular fluids occurs, resulting in peripheral edema and edema of multiple organs, which leads to organ dysfunction, respiratory distress, and death.

IV. Other consequences of hypoalbuminemia include altered homeostasis, altered delivery and metabolism of drugs and endogenous products, coagulation disorders, and increased inflammation via many routes.

V. Canine albumin has recently been introduced to the veterinary market in the USA.

VI. One unit of canine plasma contains about 6–7.5 g of albumin, equal to about 25 mL of human serum albumin (HSA) 25%.

VII. There are two human serum albumin concentrations commercially available: 5% and 25%. The 5% concentration has not been shown to be more beneficial than LRS. The use of the 25% HSA solution is controversial, has not been proven to improve survival, may increase mortality and may have the following beneficial effects:

a. May increase intravascular volume by as much as 4–5 times the administered volume
b. May improve oxygen delivery
c. May improve organ function
d. May reduce peripheral edema and the accumulation of pleural and peritoneal fluid
e. May increase blood pressure when administered as a bolus followed by an IV continuous rate infusion (CRI) over 4 hours.

VIII. The volume recommended for the treatment of hypotension in dogs is 4 mL/kg IV slow push or bolus followed by a CRI of
0.1–1.7 mL/kg/h. The maximum volume recommended for an individual dog is 25 mL/kg given over 72 hours.

IX. Although a vented delivery set is required for administration, a blood filter is not.

X. Adverse effects of human serum albumin administration include death from immediate or delayed immune responses, facial edema, polyarthritis, vasculitis, dermatitis, type III hypersensitivity reactions, and enteropathic polyarthritis. Normal dogs should not be given 25% HSA as there is increased morbidity and mortality in dogs with normal serum albumin levels.

XI. Because the administration of 25% HSA is controversial, and the complications can be severe, it is important to evaluate the risks versus the benefits for the individual patient and fully inform the owner of the potential risks.

B. Hetastarch
   I. Hetastarch is a complex carbohydrate polymer made from amylopectin, a highly branched polysaccharide.
   II. The molecular weight is 480 kilodaltons (kDa), with a COP of 32 mm Hg.
   III. The administration of 20 mL/kg causes the intravascular volume to expand 70–200% of the volume administered.
   IV. The total solids of Hetastarch = 4.5 g/dL.
   V. The dosage is 10–20 mL/kg/day in the dog and 10–15 mL/kg/day in the cat. It is recommended to start with 5 mL/kg/day in the cat and carefully reassess to avoid volume overload. The bolus administration of 5 mL/kg is often called a shock bolus. After the initial IV dose has been administered, another daily dose may be administered as a slow IV as a CRI. It is recommended that one should avoid exceeding 40 mL/kg/day in dogs.
   VI. Hetastarch may be associated with a dose related coagulopathy. The PTT may be prolonged, but there have been no clinical reports of excessive bleeding. There is no interference with platelet function.
   VII. Hetastarch is recommended for colloidal support, hypovolemia, and hypotension due to hypovolemia or decreased systemic vascular resistance.
   VIII. The hemodynamic benefits and colloidal support improve when Hetastarch is administered on successive days.

C. Pentastarch
   I. Pentastarch is an analogue of Hetastarch and has similar qualities.
   II. The COP is approximately 40 mm Hg and the molecular weight is 264 kDa.
   III. The intravascular volume expands by 150% of the administered volume.
   D. Dextran 70 is produced by bacteria from sucrose. The molecular weight is 70 kDa (similar to albumin).
I. It expands intravascular volume by 80–100% of the administered volume.

II. It causes a dose-related coagulopathy by coating platelets, decreasing von Willebrand’s factor and factor VIII activity, precipitation and dilution of clotting factors, and increasing thrombolysis. It also can interfere with the cross matching of blood after it is absorbed onto red blood cell membranes.

III. Another Dextran product, Dextran 40, causes acute renal failure and should not be used.

IV. Dextran molecules <20 kDa are excreted by renal glomerular filtration. The larger molecules are degraded by the reticuloendothelial system.

V. The dosage of Dextran 70 is 10–20 mL/kg/day.

VI. The total solids of Dextran 70 are 4.5 g/dL.

E. Oxyglobin (no longer available at time of publication)
   I. Oxyglobin was a synthetic oxygen-carrying stroma-free hemoglobin solution that had colloidal and vasopressor properties. It was made from bovine red blood cells.
   II. Oxyglobin improved oxygen delivery to tissues by expanding intravascular volume, increasing arterial blood pressure, and carrying more oxygen per volume because it was less viscous than blood. It was better able to penetrate tissues that have low blood flow.
   III. Oxyglobin had a molecular weight of 200 kDa and provided expansion of intravascular volume. It could cause volume overload if cautious dosing and monitoring was not provided. The colloidal pull of Oxyglobin was greater than that of Hetastarch, which is greater than that of plasma.
   IV. Oxyglobin was an effective and useful vasopressor; it elevated arterial blood pressure.
   V. Oxyglobin had multiple species compatibility (ferrets, dogs, cats, etc.) and caused no immune reactions.
   VI. It had a long shelf life (2 years) but had to be used within 24 hours of opening.
   VII. No refrigeration was needed, no filtration was needed during administration, and no cross match was needed.
   VIII. Each 125 mL unit of Oxyglobin contained the same amount of hemoglobin as one 450 mL unit of fresh whole blood.
   IX. The recommended dosage for Oxyglobin in dogs was 5–30 mL/kg IV. In cats, the dose was 2–15 mL/kg IV. For cats, Oxyglobin needed to be administered slowly and the patient needed to be monitored very carefully for signs of volume overload.
   X. The effective half-life varied with the dosage, with a half-life of 30–40 hours when dosed at 30 mL/kg.
   XI. Administration of Oxyglobin could raise a patient’s hemoglobin levels without raising the patient’s PCV. The Oxyglobin dose of 30 mL/kg could increase hemoglobin as if PCV has increased 12%. A rough estimate of the PCV can be made by multiplying the hemoglobin by three.
XII. Disadvantages of Oxyglobin include:
   a. Limited availability (no longer manufactured)
   b. High cost
   c. The yellow orange discoloration of the patient’s mucous membranes, sclera, skin, plasma and urine
   d. Interference with chemistry analysis.

F. Blood components

I. Commonly available products include:
   a. Fresh whole blood
   b. Stored whole blood
   c. Packed red blood cells (packed RBCs or pRBCs)
   d. Fresh plasma: donor must be available, contains all plasma components including coagulation factors and albumin
   e. Fresh frozen plasma: rapidly frozen within 8 hours of collection, frozen less than 1 year, contains all plasma components including coagulation factors and albumin; the dose is 10–40 mL/kg
   f. Frozen plasma: plasma which was frozen more than 8 hours following collection or fresh frozen plasma that has been stored more than 1 year; maximum length of storage = 5 years; contains factors II, VII, IX, X and albumin
   g. Platelet-rich plasma or platelet concentrate: indicated for the treatment of thrombocytopenia in patients with life-threatening hemorrhage; dose = 1 unit/10 kg
   h. Cryoprecipitate: dose = 1 unit or more/10 kg
   i. Cryoprecipitate (Cryo)-poor plasma: contains albumin and decreased amounts of factors II, VII, IX and X.

II. Dosages
   a. Standard formula:
      \[
      \text{Body wt (kg)} \times \frac{40 \text{ mL (dog) or } 30 \text{ mL (cat)}}{30} = \text{vol of blood (mL) to be administered}
      \]
      \[
      \left( \frac{\text{desired PCV} - \text{patient PCV}}{\text{PCV of donor blood}} \right) \times \text{PCV of donor blood} = \text{vol of blood (mL) to be administered}
      \]
   b. Short cuts:
      i. 20 mL/kg fresh whole blood or 10 mL/kg packed RBCs will usually increase the PCV by 10%.
      ii. 1 mL/lb will usually increase the PCV by 1%.
      iii. Volume of whole blood (mL) to be administered = \[
      \left( \frac{\text{PCV % increase}}{\text{desired}} \right) \times \text{body wt (kg)} \times 2
      \]

III. Administration
   a. All stored or frozen products should be gently brought to room temperature by use of immersion in a warm water bath prior to administration. If the patient is in urgent need of RBCs quicker than a unit can be warmed, 50–100 mL of warm sterile 0.9% NaCl can be added to the RBC unit and the IV line can be placed through a warm water bath or warming coil.
   b. A unit that has been thawed should be used within 24 hours.
c. A unit should not be at room temperature longer than 4 hours. If the patient is small, the portion of the unit to be administered within 4 hours should be removed for administration and the remainder of the product should be returned to refrigerated storage.
d. All blood component products should be administered through a blood filter. Depending upon the filter used, some filters require a new filter for each unit administered.

IV. Anaphylactic or allergic reactions
a. Monitor for transfusion reactions with blood components: urticaria, fever, vomiting, dyspnea, hemoglobinemia, hemoglobinuria, hematuria, restlessness, or pulmonary edema.
b. Treatment of transfusion reactions
   i. Stop the transfusion.
   ii. Establish an airway, ensure adequate oxygenation.
   iii. Administer epinephrine, 0.01–0.02 mg/kg IV, IM, or SC.
   iv. Administer diphenhydramine, 0.5 mg/kg, IV or IM.
   v. Administer crystalloid IV fluids and treat for shock.

ROUTES OF FLUID THERAPY

1. Oral
   A. This route is the most physiologic and allows for administration of high-caloric-density and hypertonic solutions.
   B. The oral route is very safe, unless the patient struggles or displacement of a feeding tube occurs. Passing of a feeding tube can be very stressful.
   C. Contraindications to oral fluid administration include vomiting, diarrhea, other gastrointestinal dysfunction, and patients with acute or excessive losses of fluid or electrolytes.

2. Subcutaneous
   A. Useful in mild dehydration.
   B. Must use isotonic fluid. LRS is preferred as it is reported to sting the least.
   C. The flow rate is governed by patient comfort, may need to use multiple sites (usually administer 10–20 mL/kg per site).
   D. All of the fluids should be absorbed within 6–8 hours.
   E. Potassium chloride may be added in concentrations up to 35 mEq/L.

3. Interosseous
   A. This route is useful in pediatric and exotic patients in urgent situations and in adults when peripheral vascular access cannot be obtained.
   B. The most commonly used sites include:
      I. The medial surface of the proximal tibia (direct the needle slightly distally to avoid the proximal growth plate)
      II. The tibial tuberosity
III. The trochanteric fossa of the femur (with the hip joint rotated internally and in a neutral position, walk the needle off the medial aspect of the greater trochanter to avoid the sciatic nerve)

IV. The wing of the ilium

V. The ischium

VI. The greater tubercle of the humerus.

C. Aseptic preparation of the selected site is required.

D. Local anesthetic should be infiltrated into the area, especially into the periosteum.

E. A small skin incision should be made to facilitate passage of the needle.

F. Depending upon the patient size, an 18- to 30-gauge hypodermic needle, an 18- to 22-gauge spinal needle, bone marrow needle, or intraosseous infusion needle should be used. A commercial bone injection gun is also available.

G. Correct placement of the interosseous needle involves local palpation and the needle should move with the bone when the limb is moved, without being dislodged. When flushed with sterile saline, there should be no distension indicating leakage. Bone marrow may be aspirated from younger patients.

H. The needle entry site should be covered with antiseptic or antimicrobial cream or ointment.

I. A piece of tape may be placed in a butterfly fashion around the needle hub and sutured to the skin. Suture may also be secured directly to the needle hub with cyanoacrylate glue.

4. Intraperitoneal
   A. Only warm isotonic fluids may be administered.
   B. Patient discomfort may occur.
   C. Peritonitis is a possible complication.
   D. This route is not commonly used in practice.

5. Intravenous
   A. For many patients, this is the preferred route of fluid administration, especially for those with acute or severe fluid loss.
   B. Presence of an intravenous catheter also facilitates administration of intravenous medication, repeated blood sample collection, and provides vascular access for emergencies.
   C. The most commonly used veins include the jugular, cephalic, femoral, and lateral saphenous veins.
   D. Contraindications of jugular vein catheterization include:
      I. Coagulopathies, thrombocytopenia, thrombocytopenia, vitamin K antagonist rodenticide intoxication
      II. Hypercoagulable states as occur with hyperadrenocorticism, immune-mediated hemolytic anemia, protein-losing enteropathy and protein-losing neuropathy
      III. Elevated intracranial pressure as with head trauma, intracranial mass lesions, and intractable seizures.
   E. Complications of intravenous fluid therapy include phlebitis, thrombosis, embolism, electrolyte abnormalities from inappropriate therapy, volume overload, mechanical difficulties with fluid
administration including kinking of the catheter or fluid lines, localized infections, and sepsis.

F. Sterile procedure must be followed during placement of an intravenous catheter and while providing the daily maintenance care.

G. Explanation of various placement techniques and available intravenous catheters can be easily found in many of the references.

HOW MUCH FLUID?

1. Hydration deficit
   A. Usually replace hydration deficit over 8–24 hours with isotonic crystalloids.
   B. % dehydration $\times$ body weight in kilograms (BW kg) = fluid deficit (in liters). Example: an 11 kg dog that is 7% dehydrated needs $770 \text{ mL}$ of fluid to replace deficit ($0.07 \times 11 = 0.77$).

2. Maintenance phase of fluid therapy begins once shock and/or dehydration has been corrected.
   A. $(30 \times \text{BW kg}) + 70 = \text{mL/24 h}$ for animals which weigh between 2 kg and 50 kg.
   B. For any dog or cat of any body weight, $70 \times \text{BW kg}^{0.75} = \text{mL/24 h}$.
   C. Traditionally, maintenance volumes are estimated at 60 mL/kg/day for adult patients.
   D. Neonatal patients (0–2 weeks of age) require 80–120 mL/kg/day for maintenance.
   E. Infants (2–6 weeks of age) and pediatric patients (6–12 weeks of age) require 120–200 mL/kg/day for maintenance.
   F. Shortcut version: multiply body weight (pounds) by 1.25 $\rightarrow$ hourly fluid rate (mL/h) or administer 1–2 mL/lb/h.
   G. 60 mL/kg/day for cats and small dogs; 40 mL/kg/day for large dogs.
   H. Depending upon the needs of the patient, it is common to administer 2–3 times the maintenance requirement and adjusting for losses as they occur.
   I. Fever will increase maintenance fluid requirements up to 15–20 mL/kg/day.

3. INS and OUTS
   A. It is important to monitor the volume of fluid administered or taken in by the patient (INS) in relationship to the amount of fluid lost by the patient (OUTS).
      I. If INS > OUTS, there is a risk of fluid overload.
      II. If INS < OUTS, there is a risk of dehydration.
   B. INS: fluids obtained by food and water intake, created during metabolism of carbohydrates and fats, includes intravenous fluids, blood products, liquid diets.
   C. OUTS: caused by sensible losses and insensible losses.
      I. Sensible losses include urine, feces, saliva, vomiting, 3rd space losses (losses into body cavities) = 27–40 mL/kg/day.
II. Insensible losses include evaporation, respiratory fluids, metabolic processes, sweat = about 12 mL/kg/day for cats and 20 mL/kg/day for dogs.

III. Normal body function losses = 40–60 mL/kg/day.

IV. Fever will increase maintenance fluid requirements up to 15–20 mL/kg/day.

V. Vomiting, diarrhea, polyuria, excessive salivation will increase fluid losses.

VI. May need to estimate OUTS or try to measure accurately if volume is critical using such methods as a closed urinary collection system and weighing soiled bedding (1 mL of urine = 1 gram).

D. Cannot start balancing INS and OUTS until initial dehydration deficit is corrected.

4. Administration
   A. IV fluid infusion pumps are very helpful in assuring the desired volume of fluid is administered at the desired rate and within the desired time.
   B. In the absence of an infusion pump, it is helpful to be able to determine the number of drops that need to be administered per minute.
      I. Administration sets are either standard size (10 drops = 1 mL) or micro drip (60 drops = 1 mL).
      II. Standard drip set: __ mL/h × 1 h/60 min × 10 drops/mL or __ mL/h ÷ 6 = __ drops/min.
      III. Micro drip set: __ mL/h × 1 h/60 min × 60 drops/mL or __ mL/h = __ drops/min.
   IV. To determine drops per minute, calculate the total volume needed and divide by number of minutes. Example: patient needs 700 mL/24 h or 0.48 mL/min. If using a 60 drop/mL micro drip set, 0.48 mL/min = 29 drops/min or 1 drop/2 s.
   C. Burette drip chambers can be used to control the total amount administered and can also be used to administer medications via a portion of the IV bag being infused.

5. Monitoring
   A. Serial physical exams (increased respiratory rate, nasal discharge, chemosis, cough)
   B. PCV/total solids
   C. Body weight (evaluate every 12–24 hours)
   D. Urine output and urine specific gravity
   E. Central venous pressure

6. Cessation of fluid therapy
   A. Resuscitate shock to end points.
   B. Dehydration has been corrected.
   C. The patient is stable, eating, and drinking.
   D. The patient is not vomiting and has no or minimal diarrhea.
   E. The BUN and creatinine are near normal and stable.
   F. Also stop fluid therapy if fluid overload has occurred.
   G. It is recommended to wean the patient off of IV fluid therapy over 12–24 hours.
Fluid rate during anesthesia

1. Fluids are administered during anesthesia to prevent hypotension, hypovolemia and to maintain perfusion of kidneys.
2. Basal rate is 5–10 mL/kg/h and for major exploratory surgery is 10–15 mL/kg/h.

BLOOD PRESSURE ASSESSMENT

Blood pressure is the force exerted by blood against any unit area of the blood vessel wall. It is usually measured in mm Hg, but may be measured in cm H₂O. To convert between units, 1 mm Hg = 1.36 cm H₂O. The ways to measure blood pressure include:

1. Direct arterial blood pressure measurement via an intravascular catheter that is connected to an electronic pressure transducer.
   A. The commonly used vessels include the metatarsal and dorsal pedal arteries. The femoral artery may be used in some cases.
   B. The site should be clipped and disinfected.
   C. A 21–23G indwelling venous or arterial catheter should be inserted via a small skin incision or percutaneously. The catheter is first introduced perpendicularly to the skin then advanced while being held flatly along the limb.
   D. Heparinized saline should be flushed through the catheter immediately upon placement and then intermittently via bolus fashion or via constant infusion.
   E. Specialized semirigid tubing is used.
   F. A bag of 0.9% NaCl with heparin 1 unit/mL added is connected to the tubing and pressurized to 300 mm Hg, monitored by a pressure transducer. The pressure transducer should be mounted level with the patient’s heart.
   G. The pressure changes are converted into an electrical signal by the pressure transducer. The signal is carried by a transducer cable to a monitor where it is displayed as a pressure waveform.
   H. Common problems associated with direct blood pressure measurement include difficulty in placing an intra-arterial catheter, kinking of the catheter or tubing, the catheter positioned against the wall of the artery, clotting of the catheter or tubing, air bubbles in the catheter or tubing, and the use of compliant tubing. These problems tend to result in a dampened waveform.
   I. Complications of direct blood pressure measurement include hematoma formation, thrombosis, phlebitis, infection, and necrosis of distal tissues.

2. Indirect blood pressure measurement
   A. Doppler ultrasound – a sound frequency change (Doppler shift) is created by the re-entry of red blood cells into an occluded artery.
      I. A cuff that is connected to a manometer is applied around the limb. The cuff width should be about 38–40% of the circumference of the limb. The measurements will be falsely
low if the cuff is too wide and falsely high if the cuff is too narrow.
a. Commonly used sites include the radial artery on the medial and proximal aspect of the carpus, the median caudal artery at the ventral base of the tail, the brachial artery on the upper forelimb of cats and small dogs, and the saphenous artery on the medial and proximal aspect of the tarsus.
b. The limb should be level with the body.

II. An ultrasound transducer that is connected to an amplifier is placed distally to the cuff. The site must usually be clipped and conductive gel applied. The transducer should be held perpendicularly to the artery and should not be pressed too tightly against the artery.

III. The cuff is inflated until the artery is occluded then the air in the cuff is gradually released until the artery reopens.

IV. The systolic blood pressure corresponds to the sound made by the first pressure wave of blood passing through the artery.

V. The use of a headset may be helpful to avoid patient distress and to assist hearing.

VI. It is recommended to take 3–5 separate measurements, prior to performing a physical exam, in the least stressful environment, then average the measurements.

B. Oscillometry

I. A pneumatic cuff that contains the sensor is placed at the same sites as used for the Doppler method. The same principles apply regarding the width of the cuff.

II. The cuff is automatically inflated and deflated by the unit.

III. Systolic blood pressure (SAP), diastolic blood pressure (DAP) and pulse rate are determined. Mean arterial pressure (MAP) is often also displayed.

IV. Clipping of the measurement site is not necessary.

V. Multiple measurements should be taken.

C. Plethysmography, either photo or pressure, is difficult to utilize in practice because of the interference of pigmentation and cuff size limitations.

D. Manual palpation

I. Commonly used sites include the femoral, saphenous, radial and lingual arteries.

II. The pulse pressure difference between systolic and diastolic pressure is what is palpable. A large difference in pressure results in a strong pulse and a small difference results in a weak pulse. It cannot be relied upon as an accurate measure of blood pressure.

III. The lowest systolic blood pressure at which femoral artery pressure is usually palpable is about 80 mm Hg.

E. Pulse oximetry – does not measure blood pressure but when pulse waves are too small to detect or absent, or the systolic blood pressure is less than 70 mm Hg, a pulse oximeter may malfunction.

3. Central venous pressure corresponds to right atrial pressure in the absence of vascular obstruction.
A. A jugular catheter is advanced so that the tip of the catheter lies in the anterior portion of the vena cava.

B. A bag of IV fluids is connected to an IV line that is connected to a three-way stopcock. A water manometer is connected to a second port of the three-way stopcock, and another IV line is connected from the last port of the three-way stopcock to the jugular catheter.

C. The manometer is filled to above 20 cm H₂O with IV fluids, then the system is turned off to the fluid bag and opened between the patient and the manometer.

D. The zero point of the manometer should be level with the right atrium, which is the manubrium of the sternum when the patient is in lateral recumbency and the point of the shoulder if the patient is in sternal recumbency.

E. The fluid is allowed to equilibrate. The fluid level should move slightly along with respiration. The central venous pressure (CVP) is then read off of the manometer, the result being in mm H₂O.

F. The normal CVP = 0–5 cm H₂O. Monitoring of trends is more important than one individual reading.

G. A low CVP (less than 0 cm H₂O) indicates hypovolemia or vasodilation.

H. A high CVP (greater than 10 cm H₂O) indicates volume overload, pleural effusion, pericardial effusion and tamponade, restrictive pericarditis, right-sided myocardial failure or increased intrathoracic pressure as occurs with positive end-expiratory pressure (PEEP), during positive pressure ventilation, and in patients with pneumothorax.

I. A CVP greater than 16 cm H₂O is associated with the development of edema and effusions.

J. The contraindications to evaluation of CVP include coagulopathy, increased risk of thromboembolism, increased intracranial pressure, or infection at the catheter placement site.

4. Blood pressure abnormalities

A. Normal systolic arterial pressure (SAP) in dogs is 90–140 mm Hg; in cats is 80–140 mm Hg. Normal diastolic arterial pressure (DAP) in dogs is 50–80 mm Hg; in cats is 55–75 mm Hg. Mean arterial pressure (MAP) in dogs is 60–100 mm Hg; in cats is 60–100 mm Hg.

B. Hypotension is MAP <60 mm Hg.

I. Hypotension is caused by:

- Hypovolemia
- Myocardial fibrosis
- Low venous return to the heart
- Positive pressure ventilation
- Gastric distension
- Tachycardia
- Outflow tract obstruction
- Vasodilating effects of anesthetic or other drugs
- Negative inotropic effects of anesthetic drugs, beta blockers, or calcium channel blockers.

- Poor diastolic or systolic cardiac function
- Cardiomyopathy
- Ventricular arrhythmias
- Pericardial tamponade
- Patent ductus arteriosus
- Bradycardia
- Low systemic vascular resistance

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II. The clinical signs of hypotension include tachycardia (cats usually have bradycardia), slow capillary refill time, pale mucous membranes, either weak or bounding peripheral pulses, hypothermia, cold extremities, decreased urine output, mental dullness, and weakness. Dogs with sepsis or SIRS may have injected mucous membranes rather than pallor.

III. The adverse effects of hypotension include acute renal failure, arrhythmias, mentation changes, coagulopathies, tachypnea, vomiting, and melena.

IV. Treatment is directed at identifying and remediying the underlying cause and aggressive fluid therapy for patients without cardiac disease as the primary etiology.
   a. Shock boluses (90 mL/kg in the dog and 60 mL/kg in the cat) and additional IV fluid therapy are administered as needed, with crystalloids and colloids.
   b. Inotropic support with β-adrenergic agonists, vasopressive support with α-agonists, or vasopressin may be needed in patients that fail to respond to adequate fluid therapy.
      i. β-adrenergic agonists are commonly used in patients with refractory hypotension, cardiogenic shock, congestive heart failure and oliguric renal failure and are usually the safest choice if poor cardiac contractility cannot be ruled out as the underlying cause. There is no one clear choice for therapy. Some studies recommend norepinephrine in the treatment of severe hypotension, with the second choice varying between dopamine and dobutamine.
         – Dopamine 5–10 μg/kg/min IV CRI increases cardiac contractility and heart rate along with causing a slight increase in systemic vascular resistance. Lower doses may cause splanchnic vasodilatation and changes in renal and gastrointestinal blood flow. Higher doses may cause ischemia of the gastrointestinal tract, kidneys, or heart.
         – Dobutamine 2–20 μg/kg/min IV CRI in dogs (1–5 μg/kg/min IV CRI in cats) increases cardiac contractility but does not affect systemic vascular resistance or heart rate. Higher doses in cats may cause tremors or seizures.
         – Epinephrine 0.005–1 μg/kg/min IV CRI can cause an increase in systemic vascular resistance, cardiac contractility, and heart rate but also causes an increase in oxygen consumption. The use of epinephrine is reserved for patients with resistant hypotension or during cardiopulmonary resuscitation.
         – Isoproterenol 0.04–0.08 μg/kg/min IV CRI increases heart rate and contractility but may decrease systemic vascular resistance. It is usually reserved for the treatment of patients with third-degree heart block.
EMERGENCY PROCEDURES FOR THE SMALL ANIMAL VETERINARIAN

Norepinephrine 0.05–2 μg/kg/min IV CRI increases systemic vascular resistance without causing much change in heart rate.

Vasopressin 0.5–2 mU/kg/min IV CRI in dogs has been shown to cause a significant increase in mean arterial blood pressure. The side effects are minimal although high doses can result in hypercoagulability and excessive coronary and splanchnic vasoconstriction.

Combinations of these medications are often utilized. It is recommended to start with a low dose, gradually titrate to a higher dose and if the desired response is not achieved, to add on an additional medication.

C. Hypertension is defined as >160/95 mm Hg. Hypertension may be primary or secondary to systemic illnesses or medications (glucocorticoids, erythropoietin) including:

Renal disease (renal failure, glomerulopathy)
Hyperadrenocorticism
Pheochromocytoma
Hepatic disease
Chronic anemia

I. The adverse effects of hypertension include:

a. Ocular changes associated with hypertension include retinal hemorrhage and/or detachment, hyphema, retinal vessel tortuosity, perivascular edema, papilledema, glaucoma, and acute blindness

b. Cardiac changes associated with hypertension include left ventricular hypertrophy, gallop rhythm, and arrhythmias

c. The neurological signs associated with hypertension include depression, stupor and seizures.

II. The treatment of hypertension

a. Treatment of secondary hypertension is directed at the underlying etiology. If the patient is exhibiting clinical signs of hypertension, additional therapy should be provided for hypertension.

b. Hypertensive emergencies (SAP >200 mm Hg) require continuous blood monitoring and intensive care. It is recommended to decrease blood pressure less than 25% in the first hour and repeat in 2–6 hours if the patient remains stable. Recommended medications include:

i. Fenoldopam 0.1–0.6 μg/kg/min IV CRI in dogs and cats

ii. Enalaprilat 0.1–1 mg IV q6h in dogs

iii. Sodium nitroprusside 1–3 μg/kg/min IV CRI in dogs and 1–2 μg/kg/min IV CRI in cats

iv. Hydralazine 0.5–3 mg/kg PO q12h or 0.25–4 mg/kg IM or SC q8–12 h in dogs, or 0.1–0.2 mg/kg/h IV CRI in dogs; or 2.5 mg (total dose) titrated up to 10 mg (total dose) PO q12h in cats or 0.25–2 mg/kg IM or SC q8–12 h in cats
v. Amlodipine 0.05–0.2 mg/kg PO or per rectum q24h in dogs or 0.625–1.25 mg PO or per rectum q24h in cats.

c. Cats
   i. Amlodipine 0.625–1.25 mg PO or per rectum q24h is usually the drug of choice for hypertension secondary to renal disease in cats.
   ii. Benazepril 0.25–0.5 mg/kg PO q12–24 h in cats is often the second choice.
   iii. Enalapril 0.25–0.5 mg/kg PO q12–24 h is an alternative ACE inhibitor that is usually less effective in cats.
   iv. Prazosin 0.25–0.5 mg/cat PO q24h has been shown to be more useful to cause urethral smooth muscle relaxation in cats with micturition disorders in cats or pheochromocytoma in dogs.

d. Dogs
   i. Amlodipine 0.05–0.2 mg/kg PO every 12–24 hours, adjusted up to 0.25 mg/kg as needed.
   ii. Enalapril 0.25–0.5 mg/kg PO q12–24 h, benazepril 0.25–0.5 mg/kg PO q12–24 h, lisinopril 0.75 mg/kg PO q24 h or other ACE inhibitors are often the first therapeutic agents recommended in the management of hypertension in dogs.
   iii. Prazosin 0.5–2 mg PO q8–12 h may be useful in the treatment of pheochromocytoma or to cause urethral smooth muscle relaxation in dogs with micturition disorders.
   iv. Propanolol 0.5–1 mg/kg PO q8–12 h may be used in dogs but should be avoided in asthmatic cats.
   v. Spironolactone 1–2 mg/kg PO q12h is useful in patients with hyperaldosteronism or in conjunction with other diuretics.

**INTRA-ABDOMINAL PRESSURE**

1. Values
   A. The normal intra-abdominal pressure for dogs is 0–5 cm H$_2$O. The normal intra-abdominal pressure following abdominal surgery in the dog is 0–15 cm H$_2$O. The normal values for cats have not been reported.
   B. 10–20 cm H$_2$O is mild intra-abdominal hypertension. The patient should be monitored, and fluid therapy should be reevaluated, often volume resuscitation will be beneficial.
   C. 20–35 cm H$_2$O is moderate to severe intra-abdominal hypertension. Attempt to identify the cause, provide volume resuscitation, and consider decompression.
   D. >35 cm H$_2$O is severe intra-abdominal hypertension and may cause abdominal compartment syndrome. Decompression is necessary.

2. Common causes of intra-abdominal hypertension include:
   A. Abdominal surgery
B. Abdominal effusion or fluid accumulation
   I. Hemoperitoneum / hemoretroperitoneum
   II. Peritonitis, including bile peritonitis
   III. Pancreatitis
   IV. Ruptured urinary bladder
C. Ileus or gastric distension
D. Pneumoperitoneum
E. Intra-abdominal mass
F. Urinary obstruction
G. Massive fluid resuscitation
H. Blunt or penetrating abdominal trauma
   I. Pelvic fractures with retroperitoneal hemorrhage
J. Abdominal packing or management of an open abdomen
K. Mechanical ventilation.

3. Clinical signs include:
   A. Short, shallow, rapid respiration
   B. Tense abdomen
   C. Decreased urine output
   D. Vomiting
   E. Obtundation, cranial nerve reflex deficits and seizures if increased intracranial pressure occurs.

4. Adverse effects
   A. Decreased cardiac output
   B. Decreased abdominal blood flow and visceral perfusion, increased blood lactate level
   C. Decreased renal function, decreased glomerular filtration rate and urine output, azotemia
   D. Decreased pulmonary compliance, increased pulmonary artery pressure and pulmonary capillary wedge pressure
   E. Increased central venous pressure
   F. Increased intracranial pressure.

5. Measurement
   A. Position the patient in lateral recumbency.
   B. A urethral catheter, preferably a Foley catheter, should be placed so that the tip is just inside the trigone of the urinary bladder.
   C. Using two three-way stopcocks, a closed urinary collection system should be connected to the urethral catheter, a water manometer, and a bag or 60 mL syringe of sterile 0.9% NaCl.
   D. After emptying the urinary bladder, 0.5 – 1 mL/kg of 0.9% NaCl should be instilled into the bladder.
   E. The manometer system should be zeroed at the patient’s midline at the symphysis pubis, then the manometer should be filled with 0.9% NaCl.
   F. The stopcock to the fluids should be closed and the pressure in the system should be allowed to equilibrate. The meniscus will fluctuate with respiration.
   G. The difference between the meniscus and the zero point is the intra-abdominal pressure.
6. Depending upon the situation, the following treatments have been utilized:
   A. Volume resuscitation including colloid administration if indicated
   B. Repositioning of the patient’s body
   C. Sedation
   D. Neuromuscular blockade
   E. Abdominal paracentesis
   F. Rectal decompression via the administration of enemas, placement of a drainage catheter or tube
   G. Gastric decompression via nasogastric suction
   H. Surgical decompression
   I. Administration of prokinetic agents such as cisapride, metoclopramide, pantoprazole, erythromycin, domperidone, or prostigmin
   J. Administration of diuretics
   K. Venovenous hemofiltration or ultrafiltration.

NUTRITIONAL SUPPORT

ENTERAL NUTRITION

1. Oral intake
   A. Critically ill patients often have metabolic disturbances that put them into a catabolic state and predispose them to malnutrition. It is important for the patient to ingest sufficient calories and nutrients on a daily basis to meet its needs.
   B. Nausea, pain, and the anxiety associated with hospitalization often interfere with the appetite of veterinary patients.
   C. Many things may be tried to encourage ingestion of adequate nutrients, including:
      I. Offer a more palatable diet.
      II. Warm the food (but avoid overheating and potentially burning the patient’s mouth).
      III. Gravies and taste enhancers are commercially available.
      IV. Offer the food by hand, rather than from a dish.
      V. Provide a quiet environment.
      VI. Have the owner hand-feed.
      VII. Appetite stimulants such as diazepam 0.2 mg/kg IV, oxazepam 2.5 mg/cat PO, or cyproheptadine 2 mg/cat PO two to three times a day may be tried, but are not often successful in emergency patients. Mirtazapine (Remeron ®) may be more successful. The dosage is 3.75 mg PO q72h in cats and 0.6 mg/kg PO q24h in dogs.
   D. If the patient will not ingest sufficient calories and nutrients on its own, supplemental nutritional support is needed. Force-feeding with a syringe is not recommended as it is very stressful.
   E. Utilize as much of the functional gastrointestinal tract as possible.
F. The benefits of enteral feeding include:
   I. Maintains gastrointestinal mucosal integrity
   II. Prevents intestinal villous atrophy
   III. Decreases risk of bacterial translocation
   IV. Maintains gastrointestinal immune function
   V. It is safer, cheaper and more physiologic than parenteral feeding.

G. The contraindications of enteral feeding include:
   I. Inability to protect the airway
   II. Uncontrolled vomiting
   III. Malabsorption or maldigestion
   IV. Gastrointestinal obstruction
   V. Ileus.

H. The daily water requirements of 50–100 mL/kg/day may be administered via the enteral feeding tube also. The amount of water used to dilute food for tube feeding and the amount of water used to flush the tubes should be subtracted from the daily water requirement, and the remaining amount should be given to the patient daily.

2. Nasoesophageal or nasogastric tubes (NE- or NG-tubes)
   A. Contraindications include those listed above plus facial trauma.
   B. Nasoesophageal tubes are preferred unless gastric suctioning is desired.
   C. Administer light sedation to the patient if desired or needed.
   D. Position the patient in sternal recumbency.
   E. Instill several drops of dilute 2% lidocaine or proparacaine into the nostril while holding the patient’s nose upward.
   F. A 3.5–8 Fr. silicone or polypropylene catheter is commonly used.
   G. Premeasure the catheter by placing the tip of the catheter along the patient’s side to the caudal edge of the scapula then mark the length on the catheter with the permanent marker.
   H. Lubricate the catheter tip with water-soluble lubricant.
   I. Hold the catheter as close to the tip as possible with one hand as close to the nostril as possible; hold the patient’s nose with the other hand. Try to hold the head in a neutral position and avoid sticking the nose straight up. Lowering the nose makes it easier for the patient to swallow the tube.
   J. Direct the catheter ventrally and medially into the nostril. It may be helpful to push the nasal planum of the dog upward while initially directing the catheter. Advance the catheter to the mark on the catheter.
   K. Take a lateral thoracic radiograph to confirm proper placement within the thoracic segment of the esophagus. Do not pass the tip into the stomach as that increases the incidence of gastric reflux.
   L. Place a stay suture through the lateral aspect of the nares to secure the catheter.
   M. Place the catheter up the center of the muzzle between the eyes for dogs and to the side of the face for cats, avoiding the whiskers, and suture in place with 2–3 more sutures.
   N. Place an Elizabethan collar on the patient.
O. Only liquid diets may be administered via a nasoesophageal or nasogastric tube.

P. The complications of nasoesophageal or nasogastric tube feeding include sneezing, rhinitis, dacryocystitis, epistaxis, sinusitis, esophagitis, gastroesophageal reflux, misplacement of the tube or movement of the tube into the trachea, removal of the tube by the patient and obstruction of the tube.

3. Esophagostomy tubes (E-tubes)

A. Indications include:
   I. Inappetence
   II. Maxillofacial trauma
   III. Severe dental disease
   IV. Severe stomatitis secondary to infectious disease, ingestion of potpourri oil or ingestion of an alkali
   V. Orofacial or pharyngeal masses
   VI. Orofacial surgery.

B. Contraindications include:
   I. Vomiting or regurgitation
   II. Esophageal stricture or esophagitis
   III. Megasphagus
   IV. Inability to protect the airway
   V. Severe cough
   VI. Pneumonia.

C. A 12–14 Fr. silicone feeding tube or red rubber catheter is usually used in cats. In dogs, depending upon the size of the patient, a tube up to 20 Fr. can be used.

D. General anesthesia is required.

E. The patient is positioned in lateral recumbency.

F. The tube should be premeasured by holding it alongside the patient and marking the distance on the tube with a permanent marker from the midcervical esophagus to just caudal to the caudal aspect of the scapula, about the 6th or 7th intercostal space. Avoid placement of the tip of the tube through the lower esophageal sphincter into the stomach to decrease the incidence of gastric reflux.

G. Placement of a mouth speculum facilitates oral manipulation but is not absolutely essential.

H. The tip of a long curved Carmalt forceps or Kelley hemostat is placed into the esophagus to the midcervical level. The curve of the instrument should follow the curve of the patient’s neck.

I. The handle of the forceps is gently lowered towards the table so that the tip of the forceps causes the esophagus to tent up, making it readily apparent and differentiated from surrounding structures. The handle can be held in place by an assistant if needed.

J. Identify the location of the carotid artery and jugular vein, then avoid those and other vital structures and carefully incise through the skin over the tented esophagus with a number 15 scalpel blade. The incision should be extended through the skin and into the esophagus but should be as short as possible in length (1–2 cm) to allow passage of the tube.
K. The tips of the forceps should be advanced through the incision and the tip of the esophagostomy tube should be grasped by the forceps. The forceps is withdrawn into the mouth, carrying the tube with it.

L. Without pulling the tube entirely through the skin incision, the tip of the tube is looped around in the mouth then redirected down the esophagus.

M. The tube is pushed down the esophagus; once the tip has passed the incision, the tube will flip around so that the entire tube is directed down the esophagus.

N. The position of the tube in the esophagus should be confirmed with a lateral thoracic radiograph and the oropharynx should be evaluated to remove any excessive length of tube that may be remaining.

O. The esophagostomy tube should be sutured in place. If the incision was excessive, the skin may be partially closed. Caution must be used in the placement of a purse-string suture, to avoid skin necrosis. A simple interrupted suture may be sufficient to secure the tube to the neck. Then a Chinese-finger-trap knot may be placed to secure the tube to the suture in the neck and decrease sliding of the tube.

P. Antimicrobial ointment should be placed over the incision and then the tube should be bandaged to the neck.

Q. Feeding may commence immediately once the patient has recovered from anesthesia.

R. In addition to liquid diets, there are many commercial diets such as Hills a/d and Iams Max Cal that facilitate tube feeding. Also, most other canned food diets may be combined with water, liquidized in a blender, and filtered for tube feeding.

S. Complications include:
   I. Cellulitis at the stoma site
   II. Infection at the stoma site
   III. Displacement or removal of the tube by the patient
   IV. Gagging or vomiting of the tube
   V. Gastroesophageal reflux
   VI. Clogging of the tube.

4. Gastrostomy tubes (G-tubes)

A. Indications include:
   I. Inappetence and the other indications for esophagostomy tube placement
   II. Esophageal stricture or dysfunction.

B. There are various ways of placement of a gastrostomy tube, including surgical placement, endoscopy-guided placement, and a blind technique using a placement device.


D. Usually a 14–28 Fr. mushroom tipped feeding tube is used.

E. General anesthesia is required for placement.

F. The tube should not be used for the first 24 hours following placement.
G. The tube should not be removed for at least 10 days following placement.
H. Complications include:
   I. Peritonitis
   II. Dehiscence
   III. Cellulitis at the stoma site
   IV. Infection at the stoma site
   V. Damage to intra-abdominal organs
   VI. Pyloric outflow obstruction
   VII. Clogging of the tube.
I. Blender-liquidized diets and liquid diets can be fed through the tube.
J. Long-term maintenance of the tube is relatively easy. A T-shirt may be applied over the patient to distract the patient from the tube.
K. An Elizabethan collar should be applied to any patient that licks or chews at the tube.

5. Jejunostomy tubes (J-tubes)
A. Indications include:
   I. Uncontrolled vomiting
   II. Pancreatitis
   III. Inability to protect the airway.
B. The procedure for jejunostomy tube placement is provided in detail in surgery texts.
C. A 5–8 Fr. feeding tube is placed directly into the proximal jejunum.
D. General anesthesia is required for placement.
E. The tube should not be used for the first 24 hours following placement.
F. Only a liquid diet can be administered.
G. Patient tolerance appears to be improved with constant infusion rather than bolus feeding.
H. Complications include:
   I. Peritonitis
   II. Dehiscence
   III. Cellulitis at the stoma site
   IV. Infection at the stoma site
   V. Migration of the tube resulting in intestinal obstruction
   VI. Abdominal cramping
   VII. Clogging of the tube.
I. A T-shirt may be applied over the patient to distract the patient from the tube.
J. An Elizabethan collar should be applied at least initially to all patients.

6. Diet options
A. The tube diameter will dictate the type of diet that may be fed, with NE-, NG-, and J-tubes requiring a liquid diet such as CliniCare®.
B. The resting energy requirements (RER) should be fed. ‘Illness factors’ are no longer used. Obese patients should be fed based upon their optimal body weight.
   I. \((30 \times \text{BW kg}) + 70 = \text{kcal/day}\)
   II. For animals that weigh <2 kg or >50 kg, \(70 \times \text{BW kg}^{0.75} = \text{kcal/day}\)
C. Protein requirements
   I. Cats require 6 or more grams of protein per 100 kcal; 25–35% of the total energy provided to cats should be in the form of protein.
   II. Dogs should be fed 4–6 grams of protein per 100 kcal; 15–25% of the total energy provided to dogs should be in the form of protein.

D. CliniCare® Canine/Feline and CliniCare® RF feline renal solutions supply 1 kcal/mL. Hills Prescription Diet a/d supplies 1.3 kcal/mL. Iams® Veterinary Formula™ Maximum-Calorie™ supplies 1.5 kcal/mL.

E. Divide the RER for the patient by the kcal supplied per mL in the chosen food, then divide this amount by 4 to determine the volume to be fed four times per day. For the first day of enteral feeding, administer \(\frac{1}{3}\) of the required volume (and about 33% of the RER), divided into four feedings. For the second day, increase to \(\frac{2}{3}\) of the required vol, divided into four feedings. For the third and subsequent days, feed the entire volume, divided into four feedings.

F. The gastric volume of a pediatric patient = 50 mL/kg.

G. Before feeding, evaluate the tube placement, flush the tube with 3–5 mL of tap water, then slowly feed the patient warm or room temperature food. The patient should be sternal or sitting when fed to decrease aspiration.

H. After feeding, flush the tube with 3–5 mL of tap water. Cap and replace the cover over the tube.

I. Do not administer medications, especially crushed tablets, down a feeding tube.

7. Complications of enteral feeding
   A. Refeeding syndrome may occur in any patient after a period of prolonged anorexia. Rapid movement of electrolytes from the intravascular to the intracellular space causes severe hypokalemia, hypomagnesemia and hypophosphatemia. It is recommended to reintroduce susceptible patients to feeding conservatively with close monitoring, then gradually increase the amount being fed.
   B. Aspiration may occur.
   C. Inflammation and infection may occur around stromal sites. Basic hygiene and daily care of the tubes will usually minimize this complication.
   D. Vomiting, diarrhea, and ileus are signs of gastrointestinal intolerance. The administration of antiemetics or prokinetics or changing the diet may allow feeding to continue.
   E. If the tube becomes clogged, gently flushing repeatedly with warm water or cola may help to relieve the clog. The tube should be replaced if it is still needed and the tube cannot be unclogged.

PARENTERAL NUTRITION (PN)

1. Indications
   A. When nutritional support is necessary but the patient cannot tolerate enteral feeding
   B. Patients with intractable nausea and vomiting
   C. Patients unable to protect their airways
2. Requirements
   A. Vascular access must be able to be obtained and maintained aseptically. Either a dedicated venous catheter or a dedicated port of a multilumen catheter should be available.
      I. Blood sample collection should not be performed through this catheter or port.
      II. Other IV fluids or medications should not be administered through this catheter or port. Potentially fatal interactions between nutrients and medications may occur.
      III. Hemodynamic monitoring should not be performed using this catheter or port.
      IV. A catheter placed in a central vein is preferred and is essential for the administration of hyperosmolar total parenteral nutrition solutions.
   B. Nursing care must be available 24 hours a day, along with the ability to monitor basic serum chemistry results in house.
      I. Most of these patients require critical care monitoring for their illness.
      II. The central venous catheter needs close monitoring and daily care. Phlebitis is common.
      III. The preferred method of administration of PN is as a constant rate infusion.
      IV. The patient’s albumin, glucose, blood urea nitrogen (BUN) and electrolytes usually need to be monitored at least once per day.
      V. The patient’s hydration status should be assessed daily.
   C. The PN prescription must be able to be formulated and compounded.
      I. A sterile environment is essential to prevent microbial contamination.
      II. To avoid precipitation of nutrients, the solutions must be combined in the proper order.
3. Nutritional requirements (See Table 1.7 and Table 1.8)
   A. The resting energy requirements (RER) should be fed. ‘Illness factors’ are no longer used. Obese patients should be fed based upon their optimal body weight.
      I. RER (kcal/day) = (30 × BW kg) + 70 kcal/day (for animals which weigh 2–45 kg)
      II. RER (kcal/day) for animals which weigh <2 kg or >45 kg = 70 × BW kg^0.75
   B. Protein requirements
      I. Cats require 6 or more grams of protein per 100 kcal; 25–35% of the total energy provided to cats should be in the form of protein.
      II. Dogs should be fed 4–6 grams of protein per 100 kcal; 15–25% of the total energy provided to dogs should be in the form of protein.
      III. Animals with renal disease and cats with hepatic disease should be fed 50% of their normal protein requirements.
   C. On the first day of providing PN and once weekly thereafter, administer Vitamin K₁ SC.
4. Complications
   A. Catheter complications include:
      I. Phlebitis
      II. Loss of vascular access due to malposition of the catheter
      III. Thrombosis
      IV. Catheter-related infections.
   B. Nutrient solution complications include:
      I. Microbial contamination

---

**Table 1.7 Sample calculation of total parenteral nutrition for a 22 lb (10 kg) dog with pancreatitis**

<table>
<thead>
<tr>
<th>Step</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Calculate the BER</td>
<td>$30 \times (10 \text{ kg}) + 70 = 370 \text{ kcal/day}$</td>
</tr>
<tr>
<td>2. Calculate the TER</td>
<td>In this case, the illness factor is 1.0</td>
</tr>
<tr>
<td>3. Determine the daily protein requirement</td>
<td>$4 \text{ g/100 TER} \times 370 \text{ kcal/day} = 14.8 \text{ g of protein/day}$</td>
</tr>
</tbody>
</table>
| 4. Determine the volume of nutrient solutions required | Dextrose: The patient will receive 60% of its daily energy requirement as dextrose $222 \text{ kcal/day} \div 1.7 \text{ kcal/mL of 50% dextrose}$
Lipids: The patient will receive 40% of its daily energy requirements as lipids $0.40 \times \text{TER} = 0.40 \times 370 \text{ kcal/day} = 148 \text{ kcal/day as lipids}$
$148 \text{ kcal/day} + 2 \text{ kcal/mL of 20% lipid solution} = 74 \text{ mL/day of 20% lipid solution}$
Amino acids: $14.8 \text{ g of protein/day} + 85 \text{ mg/mL of 8.5% amino acid solution} = 174.1 \text{ mL/day (rounded to 174 mL/day) of amino acids}$
| 5. Determine the total volume and hourly rate of TPN solution administration | $121 \text{ mL} + 74 \text{ mL} + 174 \text{ mL = 379 mL/day of TPN solution}$
$379 \text{ mL/day} + 24 \text{ h} = 15.8 \text{ mL/h of TPN solution}$
| 6. Determine the daily vitamin requirements | Vitamin K: $0.5 \text{ mg/kg} \times 10 \text{ kg} = 5 \text{ mg SC once weekly, if needed.}$
Supplementation with vitamin B may be necessary. For example: $370 \text{ kcal/day} + 1 \text{ mL B complex/1000 kcal} = 0.37 \text{ mL/day}$
| 7. Administer TPN | Day 1: Administer one-third of the calculated requirement.
$\frac{1}{3} \times (379 \text{ mL/day} + 0.37 \text{ mL/day of B vitamins}) + 24 \text{ h} = 5.3 \text{ mL/h}$
Day 2: Administer two-thirds of the calculated requirement.
$\frac{2}{3} \times (379 \text{ mL/day} + 0.37 \text{ mL/day B vitamins}) + 24 \text{ h} = 10.5 \text{ mL/h}$
Day 3 and on: Administer the full calculated requirement plus 0.37 mL/day B vitamins.
$379 \text{ mL/day} + 0.37 \text{ mL/day B vitamins} + 24 \text{ h} = 15.8 \text{ mL/h}$

II. Drug–nutrient interactions

III. Precipitation of nutritional components such as separation or layering of the lipid emulsion

IV. Fat embolism.

C. Metabolic complications

I. Refeeding syndrome may occur (see description under Enteral nutrition).
II. Persistent hyperglycemia may occur and may require the administration of regular insulin 0.1 U/kg IV, IM, or SC, or CRI IV.

III. Signs of hepatic encephalopathy may develop in patients with hepatic insufficiency.

5. If total PN is not available or the cost is prohibitive, partial PN may be used to meet some of the energy requirements. There are several commercial nutritional combinations that are various combinations of amino acids and dextrose. A commonly used product is ProcalAmine®.

A. ProcalAmine has an osmolality of 735 mOsm/L and provides 246 kcal/L.
B. ProcalAmine may be administered through a peripheral venous catheter but the vein should be monitored for phlebitis.
C. A maintenance fluid rate of 66 mL/kg/day for dogs and 50 mL/kg/day for cats is recommended as a 24-hour CRI. At this dose, 30–40% of the patient’s energy requirements, 100% of a dog’s protein requirements, and most of a cat’s protein requirements are met.

ANALGESIA

Many of the patients encountered in emergency veterinary practice will be suffering from pain. In human medicine, pain is now considered the fifth vital sign. Stress and anxiety can also affect the emergency patient. There are many differences between dogs and cats, and between breeds of dogs, regarding their response to pain, stress and anxiety. When possible, anticipation of pain and the administration of pre-emptive analgesics should be provided. Analgesia improves the speed and quality of recovery. For dosages, see Table 1.9.

<table>
<thead>
<tr>
<th>Analgesic class</th>
<th>Analgesic</th>
<th>Canine dosage</th>
<th>Feline dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opioid</td>
<td>Buprenorphine</td>
<td>0.005–0.02 mg/kg q4–8 h IV, IM 5–20 μg/kg IV, IM q4–8 h 120 μg/kg OTM 0.12 mg/kg OTM</td>
<td>0.005–0.01 mg/kg q4–8 h IV, IM 5–10 μg/kg IV, IM q4–8 h 20 μg/kg OTM 0.02 mg/kg OTM q6–8 h</td>
</tr>
<tr>
<td></td>
<td>Butorphanol</td>
<td>0.1–0.4 mg/kg IV, IM, SC q1–2 h 0.05–0.2 mg/kg/h IV CRI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Codeine</td>
<td>0.5–2 mg/kg PO q6–8 h 2–10 μg/kg IV to effect</td>
<td>0.5–1 mg/kg PO q12h 1–5 μg/kg IV to effect</td>
</tr>
<tr>
<td></td>
<td>Fentanyl</td>
<td>1–10 μg/kg/h IV CRI 0.001–0.005 mg/kg/h IV CRI</td>
<td>1–5 μg/kg/h IV CRI 0.001–0.005 mg/kg/h IV CRI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Or transdermally as follows: Body weight Patch size</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;5 kg</td>
<td>Fold back the liner to expose ½–⅓ of a 25 μg/h patch</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt;5 kg</td>
<td>Fold back the liner to expose ½ of a 25 μg/h patch or use the full patch</td>
</tr>
<tr>
<td>Analgesic class</td>
<td>Analgesic</td>
<td>Canine dosage</td>
<td>Feline dosage</td>
</tr>
<tr>
<td>-----------------</td>
<td>----------</td>
<td>--------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Analgesic Canine dosage</td>
<td>Feline dosage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10 kg (20 lb)</td>
<td>25 μg/h</td>
<td>50 μg/h</td>
<td></td>
</tr>
<tr>
<td>10–25 kg (20–50 lb)</td>
<td>75 μg/h</td>
<td>100 μg/h</td>
<td></td>
</tr>
<tr>
<td>25–40 kg (50–88 lb)</td>
<td>0.05–0.2 mg/kg IV, IM or SC q2–6 h</td>
<td>0.0125–0.05 mg/kg/h IV CRI</td>
<td></td>
</tr>
<tr>
<td>&gt;40 kg (&gt;88 lb)</td>
<td>premixed 0.1 mg/kg with acepromazine, 0.02–0.05 mg/kg IM</td>
<td>0.02–0.1 mg/kg IV, IM or SC q2–6 h (C) and 0.0125–0.03 mg/kg/h IV CRI (C)</td>
<td></td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.05–0.2 mg/kg/h IV q4–12 h</td>
<td>0.05–0.2 mg/kg IM, SC</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.05–0.2 mg/kg IM, SC q2–6 h</td>
<td>0.05–0.2 mg/kg IM, SC q2–4 h</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.05–0.3 mg/kg/h epidural</td>
<td>0.05–0.1 mg/kg IV q2–4 h</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>2–8 mg/kg q8–12 h PO</td>
<td>2–5 mg/kg q12h PO</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10–20 mg/kg q8–12 h PO</td>
<td>1–25 mg/kg q72h PO</td>
<td></td>
</tr>
<tr>
<td>NSAIDs</td>
<td>4.4 mg/kg q24h</td>
<td>4 mg/kg SC or IV once</td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>2.2 mg/kg q12h PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carprofen</td>
<td>5 mg/kg q24h PO</td>
<td>0.75–3 mg/kg PO once</td>
<td></td>
</tr>
<tr>
<td>Deracoxib</td>
<td>10–15 mg/kg q24h PO</td>
<td>2 mg/kg IV, IM, SC once Or 1 mg/kg PO q24h for a maximum of 5 days</td>
<td></td>
</tr>
<tr>
<td>Etodolac</td>
<td>2 mg/kg IM, IV, SC q24h for maximum 3 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flunixin meglumine</td>
<td>1 mg/kg PO or IM once</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>2 mg/kg IM, IV, SC q24h</td>
<td>2 mg/kg IV, IM, SC once Or 1 mg/kg PO q24h for a maximum of 5 days</td>
<td></td>
</tr>
<tr>
<td></td>
<td>q4–8 h for 3–5 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Firocoxib</td>
<td>3 mg/kg q24h PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meloxicam</td>
<td>0.1 mg/kg initial dose then 0.2 mg/kg q24h PO</td>
<td>0.1 mg/kg SC or PO once</td>
<td></td>
</tr>
<tr>
<td>Naproxen</td>
<td>0.1 mg/kg initial dose then 0.2 mg/kg q24h PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piroxicam</td>
<td>2 mg/kg q48h PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Robenacoxib</td>
<td>0.3 mg/kg q24–48 h PO</td>
<td>1–2 mg/kg PO q24h up to 6 days or 2 mg/kg SC once</td>
<td></td>
</tr>
<tr>
<td>Tepoxalin</td>
<td>1–2 mg/kg or PO q24h or 2 mg/kg SC once</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tolfoxin</td>
<td>20 mg/kg initial dose then 10 mg/kg q24h PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tolnaftamic Acid</td>
<td>4 mg/kg IM, SC once then PO q24h</td>
<td>4 mg/kg IM, SC once, or PO q24h for 3 days</td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.2–0.6 mg/kg/h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amantadine</td>
<td>2–10 μg/kg/min CRI IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>α2-agonists</td>
<td>3–5 mg/kg PO q24h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexametomidine</td>
<td>0.1–1.5 μg/kg/h</td>
<td>0.1–1 μg/kg/h</td>
<td></td>
</tr>
<tr>
<td>Metadomimide</td>
<td>1–3 μg/kg/h</td>
<td>0.5–2 μg/kg/h</td>
<td></td>
</tr>
<tr>
<td>Gabapentin</td>
<td>2–4 mg/kg/h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>3–10 mg/kg PO q8–12 h</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*OTM = oral transmucosal.
1. Physiologic signs of pain
   A. Salivation
   B. Increased respiratory rate
   C. Dilated pupils
   D. Increased heart rate with or without arrhythmias
   E. Increased temperature

2. Behavioral signs of pain
   A. Increased aggression or timidity
   B. Restlessness or agitation to depression and inactivity
   C. Trembling
   D. Licking or chewing at the painful area and resisting handling of the painful area
   E. Alterations in gait
   F. Abnormal posturing or reluctance to lie down
   G. Fixed facial expression (staring or squinting)
   H. Vocalization
   I. Failure to groom or to use the litter box (cats)
   J. Increased or decreased urination
   K. Insomnia or inappetance

3. There are multiple classes of medications, administrative routes, and techniques for administration. It is recommended that the practitioners become familiar with the indications, contraindications and administration of medications of many different classes.

4. General information about each class
   A. Opioids
      I. Indications – include the treatment of acute and chronic pain and for sedation. They take effect quickly, and can be maintained for long periods. The pure agonists (morphine, hydromorphone, oxymorphone, fentanyl) can be reversed with an antagonist such as naloxone.
      II. Contraindications – they can cause gastroparesis and ileus, vomiting, stimulation of pancreatic secretions, respiratory depression and bradycardia. They should be used with caution in neonatal, geriatric or severely debilitated patients and those with adrenocortical insufficiency, hypothyroidism, severe renal insufficiency, head injuries, acute abdominal conditions, severe respiratory dysfunction or receiving monoamine oxidase inhibitors (MAOIs). Dose reduction should be considered in these patients. They should be avoided in those patients with hypersensitivity to opioid medications.
      III. Specific information – partial agonists and mixed agonist–antagonists have a ceiling effect, but the pure μ-receptor agonists do not, so they are beneficial in the treatment of severe pain. The potency, in order from weakest to strongest with morphine being the standard at 1, is:
          meperidine (0.1),
          morphine (1),
          oxymorphone (10),
          hydromorphone (10–15),
buprenorphine (25), fentanyl (100), and sufentanil (1000).

Opioids can be administered safely to cats to provide analgesia. They should be titrated slowly to effect. The onset of mydriasis indicates adequate analgesia in cats, additional opioid medication may result in agitation or hyperexcitability. Opioids can be combined with various other analgesics to lessen the side effects and provide analgesia.

B. Nonsteroidal anti-inflammatory drugs (NSAIDs)
   I. Indications – anti-inflammatory, antipyretic, analgesia, acute pain (surgically induced or traumatic), chronic pain.
   II. Contraindications – hepatic dysfunction, renal insufficiency, shock, dehydration, hypotension, coagulopathy, in the presence of gastrointestinal disease or pregnancy, trauma, pulmonary disease. They should not be used in combination with other NSAIDs or with corticosteroids. They should be used with caution in cats, geriatric patients and those with chronic illnesses. They should be used with caution during the perioperative period due to detrimental dysfunction of platelets.
   III. Specific information
      a. Meloxicam is labeled for one single dose for cats.
      b. Many NSAIDs are administered once daily owing to their extended duration of activity.
      c. NSAIDs can cause detrimental interactions with several other medications.
      d. Regular monitoring of physical exam, complete blood count (CBC), complete blood count (UA), and serum chemistry panel (including hepatic and renal function tests) are recommended.

C. NMDA (N-methyl-D-aspartate) antagonists (ketamine, amantadine)
   I. Indications – NMDA receptor antagonists are used in the treatment of acute and chronic pain. They are adjunctive analgesics, used in combination with other analgesics such as opioids. They are useful in the treatment of neuropathic pain, in the prevention of wind-up, to decreased opioid tolerance, allowing lower doses of opioids to be effective, and cause less dysphoria. Ketamine causes minimal cardiovascular depression and less respiratory depression than opioids. Tremors and sedation are side effects. Amantadine is used in the treatment of neuropathic pain. It is used to prevent wind-up pain, opioid tolerance and alldynia.
   II. Contraindications – ketamine should not be administered as a sole agent for sedation or analgesia. It is contraindicated in patients with head trauma and those in which increased CSF or intraocular pressure would be detrimental. Ketamine is also contraindicated in patients with heart failure, hepatic or renal insufficiency, severe hypertension, or seizures.
III. Specific information
   a. Loud noises and minimal handling help to decrease the incidence of emergence reactions.
   b. Cats keep their eyes open following ketamine administration and therefore require lubrication with an ophthalmic lubricant such as Puralube®.
   c. Amantadine appears to have a narrow margin of safety. It may cause agitation, diarrhea, and flatulence in dogs.

D. Alpha₂-adrenergic agonists – these medications bind to receptors in the CNS and cause sedation, analgesia, bradycardia, diuresis, peripheral vasoconstriction, muscle relaxation, and respiratory depression.
   I. Indications – they can be administered in conjunction with opioids to produce synergistic analgesia and increase the duration of analgesia. They can provide sedation for short procedures in the stable patient and are reversible.
   II. Contraindications include cardiac disease, shock, severe debilitation, hepatic or renal disease, and respiratory disease.
   III. Specific information
      a. Treatment of alpha₂-adrenergic-agonist-induced bradycardia with atropine or glycopyrrolate is not recommended.
      b. Atipamezole is the preferred reversal agent.
      c. Adverse effects include urination, vomiting, altered gastrointestinal (GI) muscle tone, hyperglycemia, hypothermia, bradycardia, A-V blocks, depressed respiration or apnea, paradoxical excitation, and death from circulatory failure.

E. Lidocaine
   I. Indications – local anesthetic and antiarrhythmic agent, useful as an adjunctive analgesic agent when combined with an opioid and also in opioid–ketamine combinations as a CRI IV.
   II. Contraindications – heart block or severe bradycardia, hypersensitivity to lidocaine, shock, hypovolemia, respiratory depression, hepatic disease, congestive heart failure, patients susceptible to malignant hyperthermia.
   III. Specific information
      a. Must be used cautiously in cats.
      b. Drowsiness, depression, nystagmus, ataxia, muscle tremors, and seizures are signs of overdose, which improve rapidly when lidocaine is discontinued.

F. Gabapentin
   I. Indications – treatment of chronic pain, allodynia, hyperalgesia, and refractive or complex partial seizures.
   II. Contraindications include renal insufficiency and patients with known hypersensitivity.
   III. Specific information
      a. The oral liquid usually contains xylitol so it should not be administered to dogs.
      b. Start at a low dose and gradually increase, and then gradually wean off when discontinuing to avoid seizures.
REFERENCES/FURTHER READING

ACID–BASE DISTURBANCES

OXYGEN THERAPY
Hackett, T.B., 2009. Tachypnea and hypoxemia. In: Silverstein, D.C.,


FLUID THERAPY


BLOOD PRESSURE ASSESSMENT


**INTRA-ABDOMINAL PRESSURE**


**NUTRITIONAL SUPPORT**

Emergency and Critical Care 16 (2) Suppl 1, S14-S20.

ANALGESIA


