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How and Why to Feed Canine Parvovirus Patients Right Away

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Ken practices at Adobe Animal Hospital as an ICU and Blood Bank Manager. He is an active educator, lecturing internationally, providing practical instruction, and authoring texts, chapters, and articles on transfusion medicine, respiratory care, and critical care nursing. He serves on the boards of the Veterinary Emergency and Critical Care Society and the Academy of Veterinary Emergency and Critical Care Technicians, on the Veterinary Innovation Council, and as the NAVTA State Representative Chairperson. He is a graduate student in veterinary medicine and surgery through the University of Missouri. Ken invites all veterinary technicians to ask "Why?" to understand the "What" and "How" of our field and to constantly pursue new goals as veterinary professionals.

Canine parvovirus is a longstanding nemesis of veterinary professionals. It is a very common disease leading to a mortality rate of approximately 90% in untreated animals.[1,2] Even with aggressive treatment involving hospitalization, intravenous fluid replacement, antibiotics, antiemetics, gastroprotectants, and antiviral agents, the observed mortality rate is as high as 10%[1] to 36%.[2]

OVERVIEW OF CANINE PARVOVIRUS

Parvoviruses are nonenveloped, single-stranded DNA viruses. Canine parvovirus commonly infects young dogs 6 weeks to 6 months of age and unvaccinated adults. It is transmitted through the fecal–oral or oronasal route by exposure to fecal material, vomitus, or fomites. In exposed animals, the virus has an incubation period (time between first exposure and appearance of clinical signs) of 3 to 14 days, and it is shed for as long as 3 to 4 weeks after onset of clinical disease.[2]

The hallmark clinical signs of parvoviral enteritis are vomiting and diarrhea resulting from enterocyte destruction, which is caused by the virus taking advantage of the high mitotic rate of intestinal crypt cells to replicate. The virus also localizes in the tongue, oral cavity, esophagus, bone marrow, thymus, and lymph nodes. Key signs of lethargy, inappetence, abdominal pain, and subsequent dehydration and hypovolemia are related to breakdown of the gastrointestinal mucosa and increased intestinal permeability; neutropenia can result from bone marrow involvement. Intestinal damage can lead to secondary bacterial infection, bacterial translocation, bacteremia, and sepsis. Sepsis can then lead to systemic inflammatory response syndrome and shock. Patients in shock may exhibit tachycardia or bradycardia, obtunded mentation, and poor pulse quality, and death may result.[1,2]

The mainstream treatment plan for canine parvoviral enteritis includes fluid therapy to replace deficits and keep up with ongoing losses, antimicrobial therapy against secondary bacterial infection, and isolation of the patient to prevent exposure of other animals. Electrolyte and dextrose supplementation through intravenous fluids should be adjusted according to regularly monitored blood glucose and electrolyte levels. Common electrolyte abnormalities include hyponatremia, hypochloremia, and hypokalemia. Antiemetics such as metoclopramide, ondansetron, dolasetron, or maropitant are provided to control vomiting and nausea.

Traditionally, synthetic colloids (e.g., hydroxyethyl starch) have been used to augment colloid osmotic pressure, which is decreased when the protein-losing nature of the enteritis leads to hypoalbuminemia. Their use is now debated because of the potential for acute kidney injury, which seems greatest when they are administered as

TECHPOINT

A fasted animal experiences reduced secretion of digestive enzymes. Lower levels of digestive enzymes impair an animal's ability to digest food, leading to less efficient use of nutrients when food is reintroduced.

a constant-rate infusion (CRI).[3] Recombinant feline interferon omega has shown some beneficial effects in reducing mortality from canine parvovirus.[4] Other, less commonly used treatment options, such as human recombinant factors, equine lipopolysaccharide antitoxin, oseltamivir, and antibody-rich plasma, have not shown beneficial effects.[2]

THE TRADITIONAL NUTRITIONAL APPROACH: NPO

Traditionally, interventions used in parvoviral enteritis patients include the practice of placing the patient on NPO, or nil per os ("nothing by mouth"), treatment for 24 to 72 hours, preventing any food from entering the gastrointestinal tract.[5] While the application of NPO treatment is common, growing evidence indicates that early implementation of enteral nutrition is beneficial for patients with a variety of gastrointestinal diseases, including canine parvoviral enteritis.[6–8]

A number of longstanding reasons advocate for an NPO strategy in a patient that is exhibiting gastroenteritis with signs of vomiting and diarrhea:

1. It has been thought that the presence of food in the gastrointestinal tract can delay recovery by stimulating intestinal contractions and increasing frequency of defecation, thereby increasing patient discomfort, and that fasting allows the bowel to rest.
2. The introduction of food is thought to stimulate further vomiting in animals with gastroenteritis, which can lead to higher chances of aspiration.
3. Undigested food in the gastrointestinal lumen is thought to serve as nutrition for bacteria, leading to further proliferation of detrimental microbes.
4. The presence of food in the gastrointestinal lumen can draw exudate into the lumen through osmosis and exacerbate diarrhea.
5. Offering food to a patient that is nauseated and feeling ill can lead to food aversion, delaying the return of appetite when the patient is ready to eat.

While these reasons seem sound, evidence points toward various benefits of feeding and supports arguments against an NPO strategy.[5–7,9,10]

REASONS TO FEED

Fasting Causes Contractions and Pain

While the common belief is that fasting allows the bowel to rest, it seems to increase the degree of intestinal contractions instead. The lack of nutrients in the gastrointestinal lumen leads to vigorous contractions from the pylorus to the ileum, causing sensations described as "hunger pains."[5] These contractions are observed to be inhibited by the presence of food.[9] The presence of luminal nutrients also seems to promote contractions of normal intensity sooner, preventing the persistence of ileus related to gastroenteritis. Introducing food early thus prevents pain, promotes normal contractions, and shortens recovery time from impaired gastrointestinal motility.[5]

Feeding Shortens Duration of Nausea

Feeding animals with gastroenteritis is thought to stimulate vomiting. One study that evaluated the effect of early enteral nutrition on patients with hemorrhagic gastroenteritis observed that the chances of vomiting did increase, although the frequency subsided starting on the second day.[6] Similarly, another study evaluating dogs with parvoviral enteritis observed a shorter time to vomiting cessation in the group that was fed starting 12 hours after admission compared with the group that was fasted.[7]

The presence of luminal nutrition is thought to help maintain the integrity of the gastrointestinal mucosa and promote healthier motility, leading to less vomiting. However, food containing high amounts of fat or poorly digestible starches causes maldigestion and gastrointestinal distention and promotes vomiting through afferent stimulation of the vomiting center in the medulla oblongata. When feeding is instituted, small, frequent meals of highly digestible foods are recommended to prevent excessive secretion of gastric acids and minimize gastric distention, which can stimulate emesis.[5]

Feeding Reduces Bacterial Proliferation and Translocation

While the presence of undigested food might lead to the proliferation of some species of microbes, the presence and production of volatile fatty acids such as proprionic acid and butyric acid acidify the environment and suppress pH-sensitive pathogens such as *Campylobacter* and *Clostridium* spp.[10] In addition, fasting seems to increase the chances of bacterial translocation and bacterial adherence, as seen in several experimental studies,[5] leading to worse consequences such as bacteremia and sepsis. Providing nutrition leads to a healthier mucosal barrier.[5]

Feeding Maintains Digestive Function and Structure

Changes are seen in the gastrointestinal system when an animal is fasted, even when the animal is healthy. First, fasting promotes negative changes in the intestinal mucosa, such as decreased villus height and crypt depth, decreased antioxidants within enterocytes, and increased enterocyte apoptosis. These changes lead to increased permeability of the mucosal barrier and higher chances of bacterial translocation. Negative changes such as small intestinal villous atrophy and infiltration of the lamina propria with white blood cells have been observed even in animals that were provided parenteral nutrition in the absence of oral nutrition, indicating that the presence of food in the gastrointestinal tract has beneficial effects beyond nutritional content in the blood.[5]

Second, a fasted animal experiences reduced secretion of digestive enzymes. Lower levels of digestive enzymes impair an animal's ability to digest food, leading to less efficient use of nutrients when food is reintroduced. Basal and histamine-stimulated gastric acid levels and secretion of pancreatic exocrine enzymes (lipase, trypsin, and amylase) decrease when an animal is fasted, potentially contributing to diarrhea.[5] The presence of food is therefore important to both structural and functional soundness of the gastrointestinal tract.

Feeding Reduces Inflammation

The number of neutrophils primed for activation through the expression of adhesion molecules increases when an animal is fasted. The adhesion molecules enable neutrophil sequestration in the microvasculature of the intestinal tract, where the neutrophils cause oxidative and enzymatic damage upon activation and degranulation. Fasting also leads to impairment in the interaction of T and B lymphocytes and, subsequently, reduced production of immunoglobulin A and cytokines, which are important in immunologic function and regulation of inflammation.[5] This dysfunction is seen even when total parenteral nutrition is provided,[5] further supporting the importance of enteral nutrition.

Enteral Nutrition Literally Feeds the Gut

Normally, enterocytes of the small intestine are passively exposed to nutrients in ingested material and use them to their benefit. Glutamine, an amino acid derived in the intestinal lumen, serves as an antioxidant as well as the carbon skeleton and amino acid for DNA synthesis during enterocyte turnover.[5] Mucosal cells normally expire every few days; therefore, healthy mucosal turnover is vital to maintaining a functional gastrointestinal barrier. Deficiency in other nutrients, such as essential fatty acids, folate, zinc, vitamin A, and vitamin B_{12}, decreases mucosal turnover. The presence of nutrients in the intestinal lumen allows enterocytes to directly acquire these nutrients.

Summary of Benefits

Providing enteral nutrition early in the course of parvovirus treatment instead of applying an NPO strategy has significant benefits, including the following:

- → Alleviating pain through promotion of normal peristaltic activities
- → Shortening the duration of the vomiting period and limiting aspiration
- → Thwarting microbial proliferation and reducing bacterial translocation
- → Promoting the return to healthy enterocyte and mucosal turnover
- → Limiting inflammation and improving immune function

For these reasons, providing enteral nutrition as soon as fluid deficits are replenished and adequate perfusion of the gastrointestinal tract is reestablished is recommended.[8] Adequate perfusion is indicated when mentation, heart rate, pulse quality, mucous membrane color, capillary refill time, and core-to-extremity temperature gradient return to normal. Many negative effects of feeding can be alleviated through providing smaller amounts (25% of resting energy requirement, or RER) of a highly digestible, low-fat diet, preferably consisting of a novel protein source.[5]

METHODS OF FEEDING

Methods of providing enteral nutrition include voluntary eating, hand or syringe feeding, and feeding through orogastric, nasoenteric, esophagostomy, gastrostomy, or jejunostomy tubes. In parvoviral enteritis and other gastroenteritis patients, less invasive methods of feeding

that are appropriate to short-term hospitalization are desirable. The best method is for the patient to eat voluntarily, indicating the limited presence of nausea. Hand or syringe feeding an inappetent patient can lead to food aversion caused by the patient's association of food with nausea and procedures performed during hospitalization. A reasonable in-hospital method of enteral feeding is through a nasoenteric tube, alleviating this effect.

Nasoesophageal Versus Nasogastric Tubes

Nasoenteric tubes are tubes inserted through the nasal passage into the oral cavity and then advanced through the oropharynx to end in the esophagus or stomach (FIGURE 1). Tubes that end in the esophagus are called *nasoesophageal tubes*; tubes that end in the stomach are called *nasogastric tubes*. Nasoenteric tubes are useful in hospitalized patients for short-term (days to a couple of weeks) enteral feeding with a liquid diet. Unlike esophagostomy, gastrostomy, or jejunostomy tubes, they often do not require sedation or anesthesia during placement.[11]

The determination of whether to terminate the tube at the esophagus or the stomach depends on the purpose of the tube and the potential complications. Epistaxis, rhinitis, clogging of the tube, and early removal can be associated with both nasogastric and nasoesophageal tubes. Nasogastric tubes, which are placed through the lower esophageal sphincter, are thought to increase the chance of gastroesophageal reflux leading to esophagitis or strictures; nasoesophageal tubes are not. Advantages of nasogastric tubes are the lower risk for malpositioning after placement and the ability to perform gastric decompression and monitor gastric motility. Gastric decompression is beneficial because it can decrease nausea by removing stagnant gastric fluid.[11]

FIGURE 1. A patient in isolation for parvovirus infection with a nasogastric tube in place.

A study evaluating the complication rates associated with nasoesophageal and nasogastric tubes saw no difference in the occurrence of vomiting, regurgitation, diarrhea, epistaxis, tube clogging, early tube displacement or removal, aspiration pneumonia, or hyperglycemia.[11] In this study, the presence of esophagitis was not evaluated, thus leaving open the possibility of gastroesophageal reflux, although its presence would be subclinical. Given the lack of difference in complication rates, nasogastric tubes are more commonly used because they allow for serial aspiration of gastric contents to monitor gastric motility (FIGURE 2).

Tube Placement

Nasoenteric tubes can be placed with ease by trained veterinary technicians. The process starts by gathering the supplies and tools needed for the procedure (BOX 1) and

FIGURE 2. Gastric contents can be aspirated through a nasogastric tube to quantify the gastric residual volume and assess gastric motility.

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The single most important step in the placement process is the confirmation of proper location; specifically, avoiding endotracheal intubation.

forming a plan for sedation, if necessary. Tubes made of polyurethane and silicone, with or without guide wires, are available. Polyurethane tubes have been observed to be more tension-rupture resistant than silicone, although no difference in bacterial adhesion was seen in one study.[12] Placement of the tube requires at least two individuals. One technique involves one person placing the tube while the second restrains and positions the head; how the steps are shared may vary. Practices should have an established protocol for tube placement to ensure consistency in training and placement procedures among team members (BOX 2).

The major difference between nasoesophageal and nasogastric tube placement lies in the landmark used to measure the length of the tube to be inserted. Measurement from the nares to the sixth to eighth intercostal space is appropriate for nasoesophageal tubes, while the distance from the nares to the 10th intercostal space to the last rib is appropriate for nasogastric tubes (FIGURE 3).

Confirming Tube Location

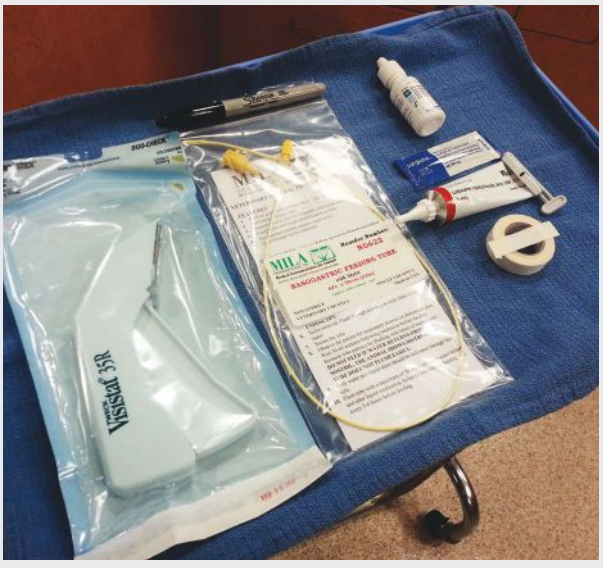
The single most important step in the placement process is the confirmation of proper location; specifically, avoiding endotracheal intubation. Accidental infusion of liquid diets into the airway leads to impaired gas exchange and inflammation of pulmonary tissues and promotes bacterial proliferation and infection of the pulmonary parenchyma (pneumonia). Diligence in confirming proper tube placement is of utmost importance to prevent causing additional harm to a patient.

Common Methods

Methods commonly used to determine tube placement include the presence or absence of coughing during intubation or upon saline infusion, aspiration of air or gastric contents, and radiography. Visual confirmation through the oropharynx is also possible in animals that are heavily sedated or placed under anesthesia. No one method is completely reliable, and a combination of methods, including radiography, should be used to confirm proper placement. Insufflation of air while auscultating the stomach, although sometimes used for nasogastric tubes, is not thought to be a reliable confirmation method in humans.[13]

Coughing. The absence of coughing upon placement of the tube or infusion of saline should not be considered

BOX 1 Supplies Required for Nasoenteric Tube Placement



- Nasoenteric tube
  - Cats and small dogs: 5–8 Fr, 40–60 cm
  - Larger dogs: 8–12 Fr, 60–90 cm
- Analgesic drops (proparacaine, lidocaine)
- Lidocaine gel
- Suture or skin stapler
- Tape
- Marker
- Elizabethan collar
- Mild sedative of choice, if required (maintain patient's ability to swallow)
- 3–6 mL syringe (for confirmation tests)
- 10–60 mL syringe (if performing gastric decompression)

BOX 2 Sample Protocol for Nasoenteric Tube Placement

Preparation

1. Select the appropriate-size tube.
2. Place 1 or 2 drops of proparacaine in each naris. This can be done before gathering the necessary equipment, but usually only takes 1 or 2 minutes to take effect.
3. Measure the tube according to intended placement:
   - **a.** Nasogastric: From the nares to between the 10th intercostal space and last rib
   - **b.** Nasoenteric: From the nares to the sixth to eighth intercostal space
   - **c.** Place tape or mark tube at appropriate length if no measurement marks are available.
4. Consult veterinarian for feeding plan.
5. If appropriate, obtain sedation protocol from veterinarian and ready needed items for swift administration.

Procedure

1. Wash hands.
2. Coat tubing with a thin layer of lidocaine gel/lubricant, at least up to ½ inch before measurement mark.
3. Elevate the nose without extending the neck. Slight flexion of the neck can facilitate intubation of the esophagus rather than the trachea.
4. Direct the tube ventromedially into the nostril, past the ventral meatus. **Do not force the tube.** It should slide in fairly easily, frequently with visible swallowing.
5. Swiftly advance the tube to the premeasured mark.
6. Place a skin staple over the tube as close to the nares as possible (not on the nose itself) and an additional staple midway on muzzle or cheek. These staples are to hold the tube in place until correct location has been confirmed with radiography.
7. Aspirate the tube with an empty 3- to 6-mL syringe, based on patient size. Negative pressure should be achieved if the tube is in the esophagus, and gastric contents if in the stomach.
8. Take placement/confirmation radiograph(s). Have the veterinarian confirm placement. The tube may need to be adjusted in or out for proper positioning. Additional views may need to be taken until placement is confirmed.
9. Once placement is confirmed, mark the tube at nares entrance. If a tube with a guide wire was used, remove the guide wire ***gently*** from the tube. If removing the guide wire is difficult, the initial staples may need to be removed and the tube straightened. **Be cautious of the tube moving from its original placement.**
10. Secure the tube at the side of the nares and one more location approximately caudal to the zygomatic arch. Skin staples and tape or a finger-trap suture pattern may be used.
11. Place and secure an Elizabethan collar on the patient. If a significant portion of the tube remains hanging, it may be anchored to the tie of the collar.
12. Document tube size and intended length of insertion on the patient record sheet.
13. Begin feeding based on nutritional plan.

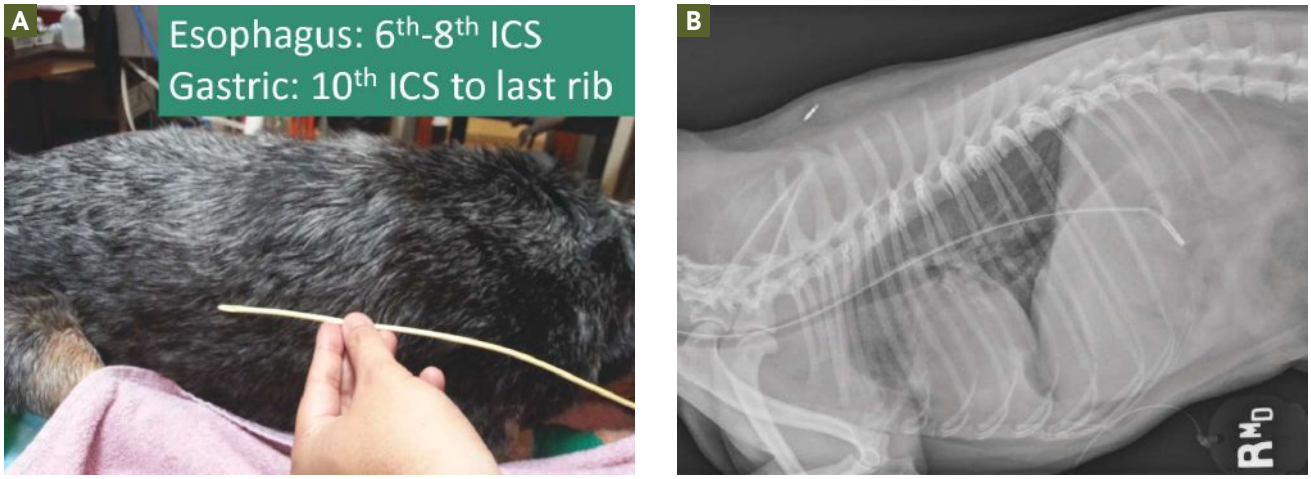


FIGURE 3. Measurement of a nasogastric tube. The tube can be measured (A) externally to between the 10th intercostal space (ICS) and the last rib, which (B) is used as a landmark for the tube to terminate at the stomach, as confirmed in this radiograph.

a certain sign of placement into the gastrointestinal tract, as debilitated patients often exhibit a lack of normal reflexes and saline infusion into the lower airway does not always lead to coughing.

Aspiration. If the tube is placed in the flaccid esophagus, aspiration results in negative pressure; if the tube is in the stomach, gastric contents will be aspirated. Placement of the tube in the trachea often results in a continuous supply of air because the cartilaginous rings prevent collapse of the tissues around the opening of the tube, although it can be possible to achieve negative pressure.

Carbon dioxide (CO_2) measurement. Use of a colorimetric CO_2 sensor has been evaluated to confirm tube placement with 86.7% sensitivity and 99.8% specificity in human patients.[14] Use of sidestream capnometry is reasonable for additional support for proper placement, with measurement of any CO_2 indicating improper placement of the tube in the airway.

Radiography. The 3 basic points evaluated on radiographs for confirmation of placement are the tube's entry point into the oropharynx, a lack of overlap with the trachea, and the terminating location. The oropharynx can be evaluated by including the cervical region in the radiograph. A properly placed tube should be seen to divert away from entering the trachea; this often serves as a reliable sign of its avoidance of the airway. A lack of overlap of the tube with the trachea as it travels distally toward the target area is also a good indication of proper placement. The correct terminating location of the tube depends on whether a nasoesophageal or nasogastric tube is intended, with proper termination in the stomach (i.e., abdominal cavity) being much more obvious than in the esophagus (FIGURE 4). A lateral view can be initially obtained for evaluation, with the option of obtaining a perpendicular view for further confirmation if any of the 3 points are uncertain. Alternatively, practice protocols may call for a standard of 2 views as a part of the due diligence in placement confirmation.

Tube Anchoring

Once placement is confirmed, the tube is anchored to the patient with skin staples, tape, or sutures in a finger-trap pattern placed along the side of the face, along the cheek, or up the forehead between the eyes. An Elizabethan collar should be used to prevent dislodgment by the patient. Once stabilized, the tube can be used immediately in nonsedated patients that do not have a risk for aspiration.

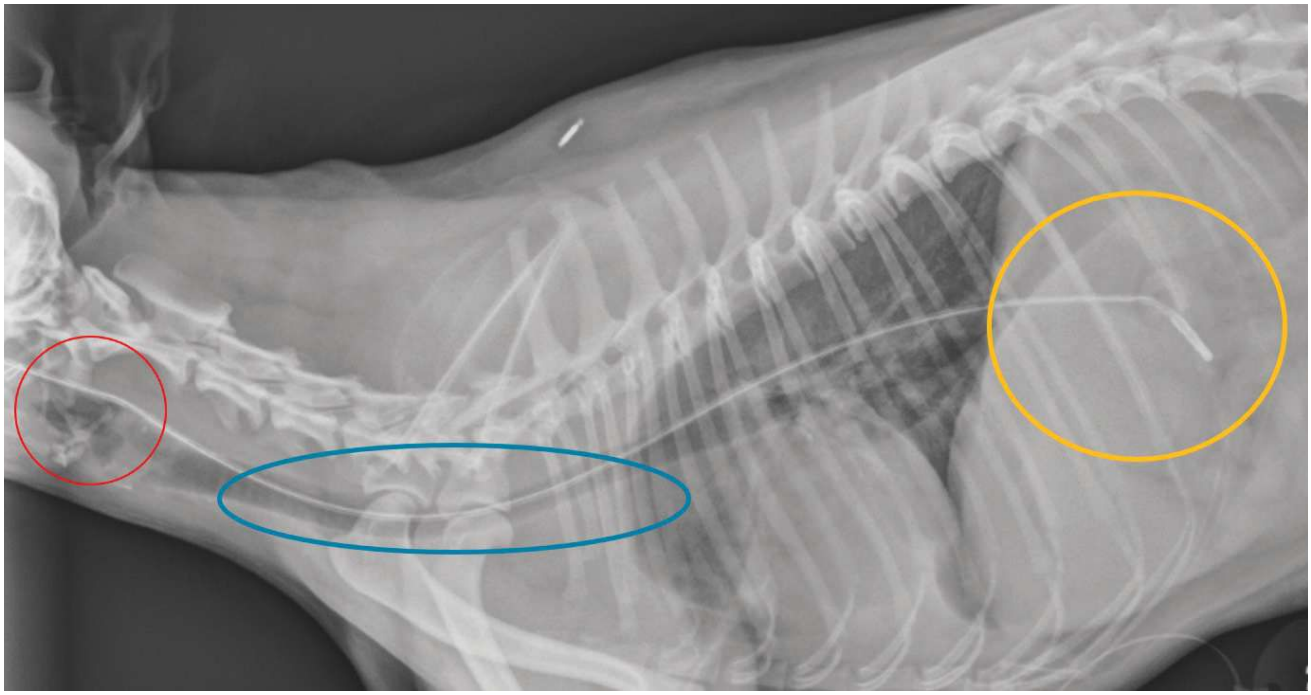


FIGURE 4. The 3 points to evaluate on a radiograph to confirm tube placement are the oropharynx (red circle), trachea (blue oval), and terminating location (yellow circle). This radiograph shows the tube avoiding the airway opening (red), overlapping with most of the trachea (blue), and terminating in the stomach (yellow), making 2 of 3 points favorable to proper placement. Other information that would support proper placement would include the ability to see the tube separately from the trachea in a perpendicular radiographic view or aspiration of gastric contents through the tube.

TECHPOINT

It is a veterinary technician's role to be a patient advocate for and to help reinforce the notion, "While patients do eat when they feel better, they also feel better when they eat."

INTRODUCTION OF FOOD

Food should be introduced cautiously in a critical care patient while the patient is monitored for tolerance. Feeding 25% of RER is a common initial approach, with the amount gradually increased over a course of 2 to 3 days. This approach is well aligned with the strategy of providing small amounts of highly digestible, low-fat food. Because patients with parvoviral enteritis are often young and generally have a higher energy requirement, consideration should be made to meet energy demands for the specific age sooner, as long as the increase is tolerated by the patient.

RER is estimated using an allometric formula intended to scale properly with changing body sizes. An extrapolated linear equation is often used for simpler calculation and yields similar results within the range of 2 to 20 kg body weight (BOX 3). Either formula can be used in parvovirus patients to provide enough food for a beneficial effect.

Typical canned food, even when blended, can clog the largest-diameter nasoenteric tubes, limiting delivery of nutrition provided this way to liquid diets. Commercial liquid diets differ in composition and caloric density. The caloric density of the diet being provided must be known because the delivery is based on the patient's caloric needs. The use of a liquid diet makes CRI of enteral nutrition easily possible through the use of syringe pumps (BOX 4). Bolus feeding is also possible, but to prevent stimulation of vomiting, the portions must be small and frequent, resulting in at least 3 feedings per day. In terms of achieving target caloric intake, there is no clear evidence of either CRI or bolus feeding having an advantage. Gastric residual volume, or the volume of food and gastric secretions remaining in the stomach, can be measured periodically to evaluate gastric motility. Aspiration of gastric contents can lead to electrolyte and metabolic disturbances, especially through the removal of hydrochloric acid. Hydrochloric acid helps limit bacterial overgrowth in the intestine, and discarding of residual volume has been linked to increased intestinal inflammation. A portion or the entire volume of the gastric contents can be refed to the patient, although evidence regarding the benefits of this practice is mixed.[15,16]

Liquid diets for enteral nutrition should be handled with caution to prevent bacterial contamination. Handwashing and donning clean examination gloves before handling the solution and lines are required. The length of time a liquid diet can be left at room temperature or refrigerated is variable, typically ranging from 2 to 6 hours and approximately 24 hours, respectively, and practices should follow manufacturer recommendations for storage. When a syringe or volumetric pump is used for CRI, the volume of liquid diet it contains should be no more than the amount that can be infused in the time the diet is allowed to stay at room temperature. The entire line setup is generally recommended to be changed every 24 hours, although protocols calling for shorter hang times exist.[17,18]

CONCLUSION

Early implementation of enteral nutrition is a key element in reducing morbidity and shortening recovery time in patients with parvoviral enteritis. The provision of a liquid diet helps

BOX 3 Comparison of Two Resting Energy Requirement Formulas

Allometric RER formula (orange line):

$$RER_{(kcal\ per\ day)} = 70 \times BW_{(kg)}^{0.75}$$

Linear RER formula (blue line):

$$RER_{(kcal\ per\ day)} = 30 \times BW_{(kg)} + 70$$

Note: RER calculations with both formulas are within small percentage differences in patients weighing between 2 to 20 kg. Larger differences are seen with much smaller and much larger patients. $BW_{(kg)}$ = body weight in kilograms.

directly replace fluid lost through the gastrointestinal tract and reduces the ongoing loss by reducing the chance of vomiting, reducing intestinal mucosal permeability, and alleviating diarrhea. The positive effects on the mucosal barrier reduce the rate of bacterial translocation, the chances of bacteremia and subsequent sepsis, and the further consequences of systemic inflammation that can lead to patient death. Implementation of enteral nutrition as soon as vomiting is no longer intractable and initial fluid deficits are replaced is a powerful form of intervention, leading to better outcomes.

"Patients eat when they feel better." It has been a longstanding notion that animals will regain an appetite and start eating when they feel well. While this is true, we must recognize the critical role nutrition plays in recovery, greatly aiding the patient in its path to feeling well enough to eat voluntarily. It is a veterinary technician's role to be a patient advocate for and to help overturn this archaic notion, amending it to "While patients do eat when they feel better, they also feel better when they eat." ■

BOX 4 Initial Nutritional Calculation in a Parvoviral Enteritis Patient

The patient: A 2-month-old, 2.3-kg, mixed-breed dog presenting with vomiting and diarrhea, with diagnosed canine parvovirus infection.

The plan: The veterinarian orders for a highly digestible liquid diet of 1.0 kcal/mL caloric density to be fed through a nasogastric tube at 25% RER for the first 24 hours.

Step 1: 25% RER Calculation

$$RER_{(kcal/day)} = 70 \times BW_{(kg)}^{0.75}$$
$$= 70 \times 2.3^{0.75}$$
$$= 130.7 \text{ kcal/day}$$

25% RER = 32.7 kcal/day

Step 2 (Option 1): Liquid Diet Constant Rate Infusion Calculation

25% RER = 32.7 kcal/day ÷ 24 h/day
= 1.4 kcal/h ÷ 1.0 kcal/mL
= 1.4 mL/h

If this option is used, the patient will be placed on a constant rate infusion via a syringe pump at 1.4 mL/h.

Step 2 (Option 2): Liquid Diet Bolus Feeding (4 times a day)

25% RER = 32.7 kcal/day ÷ 4 feedings/day
= 8.2 kcal/feeding ÷ 1.0 kcal/mL
= 8.2 mL/feeding

If this option is used, the patient will receive 8.2 mL per feeding every 6 hours (4 times a day).

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CE Test How and Why to Feed Canine Parvovirus Patients Right Away

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1. **Which of the following best describes the pathogenesis of canine parvovirus?**
   a. The virus crosses the blood–brain barrier and overstimulates the emetic center.
   b. The virus infiltrates the intestinal mucosa, paralyzing peristaltic actions.
   c. The virus causes intestinal enterocyte destruction, increasing mucosal permeability.
   d. The virus enters the bloodstream, causing systemic inflammation and sepsis.

2. **The most common therapies for parvoviral enteritis include _____.**
   a. feline interferon omega
   b. fluid therapy and antibiotics
   c. human recombinant factors
   d. plasma transfusions

3. **Which of the following nutritional approaches to treatment of parvoviral gastroenteritis promotes healthy turnover of enterocytes, leads to swifter cessation of vomiting, and helps maintain normal gastric function?**
   a. Place a central line to provide parenteral nutrition.
   b. Offer food in a bowl and wait for appetite to return.
   c. Provide early enteral nutrition through a nasoenteric tube.
   d. Withhold food for 24 to 72 hours, then reintroduce food.

4. **Fasting leads to which of the following gastrointestinal changes?**
   a. Pancreatic and digestive secretions are primed and ready to express adequate enzymes when food enters the gastrointestinal tract.
   b. Enterocyte villus height and crypt depth are reduced, and mucosal permeability is increased.
   c. Intestinal contractions become less vigorous.
   d. Bacterial proliferation increases.

5. **A key benefit of early implementation of enteral nutrition in gastroenteritis patients is**
   a. immediate reduction in vomiting frequency.
   b. support for healthier enterocyte turnover and stronger mucosal barrier.
   c. increased activation of neutrophils through stimulation of adhesion molecule expression.
   d. prevention of diarrhea by providing osmotic material in the intestinal lumen.

6. **A study comparing the complication rates of nasoesophageal and nasogastric tubes found that**
   a. the use of nasogastric tubes caused gastroesophageal reflux.
   b. there was no difference in the complication rates for the two types of tubes.
   c. the use of nasoesophageal tubes led to higher chances of aspiration.
   d. esophagostomy tubes were preferable to nasoenteric tubes.

7. **The length of tube required for a nasogastric tube is the distance from the nares to the**
   a. thoracic inlet.
   b. sixth to eighth intercostal space.
   c. 10th intercostal space to the last rib.
   d. point where resistance is felt during insertion.

8. **Proper placement of a nasoenteric tube is best ascertained by**
   a. aspiration of the tube lumen with an empty syringe.
   b. radiography to view the location of the end of the tube.
   c. experience.
   d. using a combination of methods.

9. **Which statement is true with regard to providing early nutrition to parvoviral enteritis patients?**
   a. The patient should be fed small amounts of highly digestible, low-fat food.
   b. The patient should be fed enough food to meet its full resting energy requirement.
   c. The patient should be fed a large amount of high caloric density food.
   d. The patient should not be fed until it is ready to eat on its own.

10. **To receive 25% of RER, a 5 kg patient on a liquid diet CRI should be fed approximately _____.**
    a. 2.44 kcal/h
    b. 9.75 kcal/h
    c. 15.8 kcal/h
    d. 81 kcal/h