

Management of the Patient with Canine Parvovirus Enteritis

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Canine Parvovirus is a highly contagious virus that affects dogs, resulting in severe gastrointestinal disease and occasionally cardiac disease. The virus itself is what is termed “non-enveloped”, and is made up of viral DNA. Canine parvovirus infection is a serious viral infection in dogs, and as mentioned above, is highly contagious, being readily transmitted from one dog to another through contact with infected faeces.

There are some important facts about the virus itself that underpin some important aspects of patient management...

- Canine parvovirus is a stable virus that can survive for up to five to seven months in the environment. This means, susceptible dogs can contract the virus simply by contacting and ingesting virus in a contaminated environment.
- One gram of faeces from an infected, virus-shedding dog is thought to contain enough viral material to infect over 10 million susceptible dogs by oral exposure!
- Canine parvovirus vaccination is very effective in preventing disease. However, young animals may still be at risk of contracting the disease despite vaccination, because antibodies in circulation from maternal colostrum inactivate early puppy vaccinations in a number of puppies. In fact, a 12 week vaccination with a modified live canine parvovirus vaccination will be inactivated by colostrum-derived antibody in the puppy in approximately 8% of puppies. **Vaccination protocols that stop at 12 weeks of age can result in susceptible puppies.**
- The virus initially infects the lymphoid tissue in the pharynx before it enters the bloodstream, where it contaminates cells around the body, including the intestines, bone marrow and dividing cardiac cells in the very young.
- Canine parvovirus replicates in dividing cells, and thus

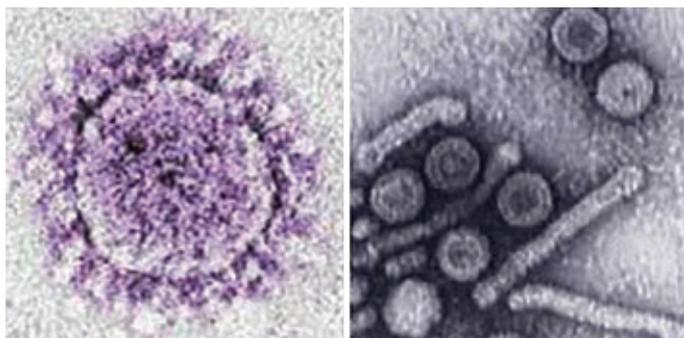
typically causes symptoms of disease in tissue with rapidly dividing cells such as the gastrointestinal tract and bone marrow:

- Cells of the intestines replicate rapidly on an ongoing basis, and are therefore affected by (and destroyed by) parvovirus infection, which results in diarrhoea and vomiting.
- Cells of the bone marrow of young dogs are replicating, producing red and white blood cells. Infection with parvovirus affects production of white blood cells, and can result in profound leukopaenia in infected patients.
- In young dogs that are born to unvaccinated parents, and that are infected within the first two weeks of life, the muscles of the heart may be affected as they replicate in the first two weeks of life, causing myocarditis. As most dogs have some history of vaccination, cardiac manifestation of parvovirus has become much less common – because maternal antibody usually provides protection against parvovirus infection in neonates during the first two weeks of life, during myocardial development.
- Symptoms appear when the virus infects cells within the body. Intestinal cells become infected by day four of infection – and gastrointestinal symptoms of disease usually appear over the subsequent two to three days.

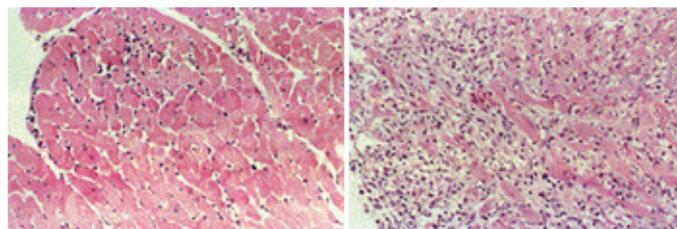
Clinical Signs

The clinical signs of canine parvovirus are usually the result of intestinal and bone marrow cell destruction by the virus, and therefore include the following:

- Anorexia
- Depression
- Vomiting
- Profuse haemorrhagic diarrhoea



Photographs of canine parvovirus. Being non-enveloped renders the virus extremely resistant to desiccation



Histopathology slides from a normal myocardium (left) and myocardium infected with canine parvovirus (right). Note the presence of large numbers of inflammatory cells and the loss of pink muscle tissue in the infected section.

- Abdominal discomfort
- Cardiovascular shock
- Dehydration
- Pyrexia
- Infection – resulting from leukopaenia – or decreased white blood cell count, and disruption to the intestinal mucosal barrier, which allows intestinal bacteria to gain access to systemic circulation
- Sudden death and congestive heart failure – may result if myocardial damage occurs in very young patients

Diagnosis

The diagnosis of canine parvovirus is made on the basis of clinical signs and a history of poor vaccination protocol, or lack of vaccination history, along with confirmation using a faecal antigen test (parvovirus “snap” tests). In addition, because there are many other diseases that may also result in similar clinical signs, a thorough evaluation of the patient must be performed as outlined below...

The diagnostic evaluation of the patient with acute gastroenteritis is critical to ensure that specific therapeutic modalities are not overlooked, and to ensure that inappropriate treatment is not administered to the patient. The diagnostic evaluation usually follows the following format:

1. **History** – obtaining a complete and thorough history can aid in directing an appropriate diagnostic approach to the patient. Pertinent questions to raise during history-taking include:
 - a. Diet – current diet, any recent change, previously noted intolerances
 - b. Behaviour of the patient - scavenging behaviour
 - c. Duration and progression of presenting signs
 - d. Environment – does the patient have access off the owners’ property etc?
 - e. Parasite control
 - f. Toxin exposure – are there herbicides, pesticides, household products, human medications that the patient has access to etc.
 - g. Is the patient de-sexed?
 - h. Concurrent medical conditions
 - i. Previous medical history
2. **Physical examination** – physical examination should reveal the presence of the following:
 - a. Fluid deficit – hydration status, evidence of blood volume depletion
 - b. Mentation
 - c. Abdominal evaluation
 - i. Conformation
 - ii. Auscultation
 - d. Palpation – palpate the abdomen several times during the course of initial evaluation. Evaluate for the presence of pain; abnormal organ shape, size, or position; evidence of obstruction; palpation of unusual structures e.g. tube-like thickening of intestines in intussusception etc.
 - e. Pain (nature, severity, location)
 - f. Rectal evaluation – palpate prostate or uterus if

possible; evaluate stool characteristics. Collect faeces for evaluation

g. Complete full physical examination

3. **Collect a Patient Data Base** – for patients with diarrhoea as a presenting sign, the following samples should be collected at admission, or shortly following admission, to facilitate diagnostic evaluation of the patient:
 - a. PCV/TP
 - b. Glucose
 - c. Electrolyte evaluation
 - d. Blood gas analysis
 - e. Serum biochemistry and complete blood count
 - f. Urinalysis
 - g. Faecal evaluation – smear, faecal flotation, immunologic assessment e.g. parvovirus, giardia, clostridium perfringens; culture if required e.g. campylobacter spp., salmonella spp.
 - h. Abdominal radiography – preferably three views – right and left lateral and ventro-dorsal recumbency views. Radiographic contrast studies may be carried out based on results of physical examination, blood tests, and survey radiography.
 - i. Abdominal ultrasound

A Note on Faecal Analysis

The diagnosis of acute infectious gastroenteritis is made on the basis of history (including the health of in-contact animals and people); clinical examination, results of serum biochemistry, CBC and urinalysis, and faecal evaluation.

Faecal analysis should begin with faecal flotation – which will enable detection of organisms such as giardia, toxoplasma, and the whipworm *Trichuris vulpis* etc. Direct faecal smears are useful for detection of intestinal parasites. A stained smear of faeces and immunological techniques can allow detection of clostridium enterotoxin etc., and faecal culture can be useful in other infections.

Staining of a faecal smear may allow detection of many organisms associated with infectious enteritis in dogs and cats, as outlined below:

1. Wright’s Giemsa or Diff Quick stain – allows detection of
 - a. White blood cells – the presence of neutrophils suggests inflammation, commonly seen in *Salmonella* spp., *campylobacter* spp., or *clostridium* spp., and should prompt faecal culture to be performed
 - b. Bacterial morphology consistent with *campylobacter* spp., *clostridium perfringens*
 - c. Mononuclear cell examination – may reveal presence of *Histoplasma* spp.
2. Methylene Blue staining allows detection of
 - a. Trophozoites of enteric protozoa

A Note on Faecal Elisa Tests for Parvovirus Enteritis

The faecal ELISA tests are very accurate, but can give erroneous results on occasion – for example:

- False positive results – where a dog does not have parvovirus, but tests positive for it – can occur between



Faecal analysis may reveal Whipworm (*Trichuris*) egg (left), *Giardia* cyst (centre) or hookworm (*ancylostoma*) egg (right)

- five and fifteen days following a vaccination
- False negative results – where a dog has parvovirus, but does not test positive for it – can occur if a faecal sample is taken very early in the disease (within one to four days of becoming infected). False negative tests should be repeated one to days days later in individuals suspected of having parvovirus based on history and clinical signs.

A Note on Blood Test Results for Parvovirus Enteritis

Blood test results in parvovirus-infected patients usually show non-specific changes, with the exception of the following...

- Leukopenia – low white blood cell counts are common in parvovirus infection due to viral infection of rapidly dividing white blood cell precursors in the bone marrow.
 - Lymphopaenia – results from damage to lymphocytes during the initial infection period
 - Neutropaenia and monocytopenia – result from damage to precursor cells in the bone marrow
- Biochemistry changes – usually reflect loss of protein and electrolytes into the gastrointestinal tract, as well as changes due to dehydration, and tissue hypoxia.
 - Albumin +/- globulin concentrations may be low
 - Liver enzymes may be slightly elevated

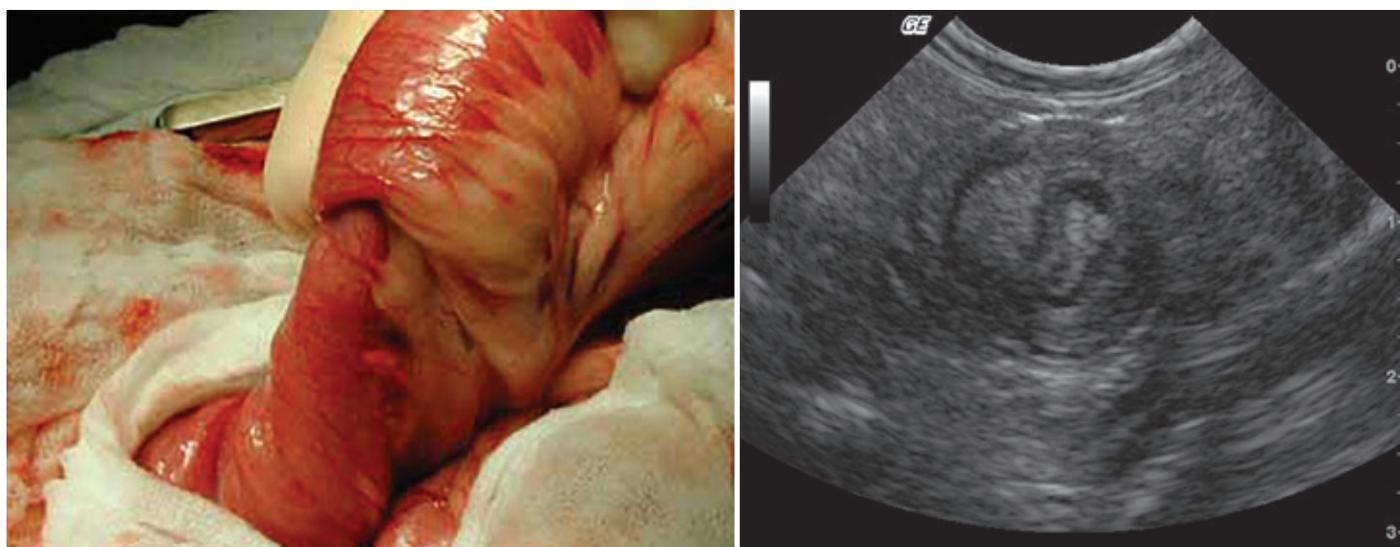
- Hypochloraemia may be present, along with hypokalaemia +/- hyponatraemia
- Urea and creatinine +/- phosphorus may be elevated also
- Platelets - numbers may be decreased in severe disease due to DIC, and reduced platelet production in the bone marrow
- Coagulation profile – the ACT may be prolonged or normal depending on the degree of illness, with severe illness commonly producing clotting abnormalities secondary to extensive tissue damage and DIC

Other Tests for the Parvovirus Patient

Diagnostic imaging – such as radiography and ultrasound can be useful in eliminating other causes of vomiting and diarrhoea, and to determine if intestinal obstruction or intussusception is present or not. This is particularly important when evaluating patients with persistent or recurrent vomiting and/or diarrhoea despite provision of intensive care over several days in hospital.

Treatment

The treatment for parvovirus enteritis revolves around good supportive care of the patient with fluid therapy, analgesia, transfusion therapy, colloid fluid therapy, anti-emetics,



Photograph showing intussusception (left) and ultrasound image of intussusception (right) – a common cause of patient deterioration despite therapy for parvovirus enteritis. Surgery is required to remedy this condition.

nasogastric suctioning and early nutritional support. In addition, supportive care of the intestinal tract is required. In order to be able to effectively care for the intestinal tract in such a severe illness, we must understand a little about what the intestine does in health. By knowing this, we will be able to more accurately assess, monitor and intervene appropriately for our patients when their intestines are infected or diseased.

The Physiology of Intestinal Functions

The normal physiological functions of the intestine include:

- Intestinal motility – the intestine broadly moves in two ways - segmental or ‘mixing’ contractions that facilitate exposure of ingesta with the intestinal mucosa, and propulsive contractions that facilitate movement of intestinal luminal contents distally within the gastrointestinal tract.
- Secretion – Normal intestinal secretion involves the secretion of an isotonic solution of water and electrolytes into the intestinal lumen. Secretion may be stimulated by bacteria, bacterial toxins, osmotically active non-absorbed solutes, drugs, and products of bacterial fermentation.
- Absorption – absorption of nutrients from the small intestine occurs through the cells of the villi. Most specific nutrients are absorbed by active means, using specific membrane-bound transport mechanisms in the cells of the intestinal villi. The movement or absorption of water occurs either by passive transport along trans-mucosal ionic or electrical gradients, by movement of water (solvent drag) or by active, cell-membrane mediated transport processes (ATPase pumps).
- Digestion – digestion of food is facilitated by motility, but primarily involves action of specific chemical reactions that facilitate degradation of food substances into small molecules that are able to be absorbed into the systemic circulation. Digestion begins in the oral cavity with secretion of the enzyme amylase, and continues in the stomach, with secretion of hydrochloric acid. Chemical reactions in the small bowel usually involve the action of cell membrane associated enzymes such as enterokinase; the action of pancreatic enzymes, and the action of bile acids.

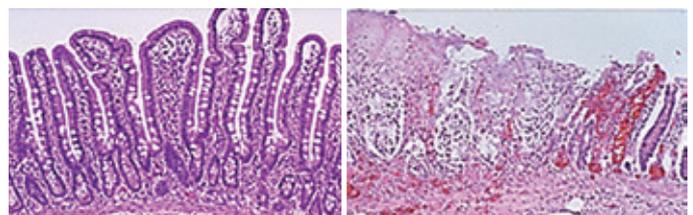
The structure of the intestine facilitates these key physiological functions. For example, very large quantities of water and electrolytes are cycled through the intestine each day. In fact, a volume equivalent to approximately half of the body's extracellular water is secreted and absorbed by the intestine in each 24-hour period; yet the intestine loses only 0.1% of this volume in faecal water. However, when normal intestinal secretory or absorptive capacity or function is disrupted, massive fluid and electrolyte losses may occur – as it does in diarrhoea or vomiting.

Treatment of Parvovirus Enteritis in Dogs

There is very little specific therapy that can be directed against the parvovirus virus itself by the time clinical signs of illness are evident. Treatment therefore remains supportive for the vast majority of patients clinically affected with signs of parvovirus gastroenteritis. In general, fluid therapy, analgesia, intestinal support and nutrition form the basis for successful patient management. A suggested treatment rationale follows...

1. **Intravenous Fluid Therapy** – in most patients presenting to the veterinary clinic with vomiting and diarrhoea, the presumption is that the patient will have lost sufficient fluids to have become dehydrated, even if this is not clinically apparent. Restoration of this fluid deficit is required to prevent further gastrointestinal injury, and patient hemodynamic compromise and renal injury. Fluid therapy is administered in the following manner:

- a. Correction of intravascular volume deficits – for patients displaying symptoms of shock or haemodynamic compromise, such as those with weak pulses, tachycardia, bradycardia, collapse, tacky mucous membranes etc., initial fluid resuscitation should begin with rapid intravenous administration of a balanced isotonic crystalloid solution such as Lactated Ringers Solution, or normosol-R. Initial fluid rates should be sufficient to correct intravascular volume deficits within one hour of patient presentation. The authors' preference is to administer an isotonic crystalloid in boluses of 7-12 ml/kg IV over 10 minutes, followed by patient reassessment, and for this procedure to be repeated until signs of haemodynamic compromise are no longer present – as evidenced by a return of normal pulse quality, improvement in capillary refill time, improved mentation, normal heart rate etc. If significant haemodynamic improvement is not noted within 30 minutes of fluid therapy, a bolus of synthetic colloid such as hydroxy-ethyl starch (Voluven) or Pentaspan is administered at a dose of 3-5 ml/kg, given intravenously over 10 minutes to prolong the effectiveness of isotonic crystalloid therapy within the intravascular space.
- b. Following correction of intravascular volume deficits, the patients hydration deficit is calculated (hydration deficit volume (ml) = % dehydration (e.g. 5% = 0.05) x bodyweight(kg) x 1000), and this volume of fluid is administered to the patient over 8-24 hours, in addition to the patients' normal daily fluid requirement (60 ml/kg/24hrs). Typically, fluid required to replace hydration deficits is similar in composition to extracellular fluid, but should be supplemented with potassium, to replace renal and gastrointestinal loss of potassium. An ideal fluid for replacement of hydration deficits is Lactated Ringers Solution, supplemented with 20-30 mEq/L potassium chloride.
- c. Following correction of hydration deficits, the patient



Photograph showing intussusception (left) and ultrasound image of intussusception (right) – a common cause of patient deterioration despite therapy for parvovirus enteritis. Surgery is required to remedy this condition.

requires fluid therapy for maintenance of normal body functions, PLUS replacement of ongoing fluid losses through the gastrointestinal tract, in patients that continue to have diarrhoea and/or vomiting. In most cases, a fluid type such as Lactated Ringers Solution, with 5% glucose, and 20-30 mEq/L potassium chloride is required. In patients without significant gastrointestinal fluid loss, 0.45% sodium chloride solution, 2.5% glucose, and 20-30 mEq/L potassium chloride is usually sufficient to meet ongoing fluid requirements. In patients with significant loss of gastrointestinal fluid, blood or protein, a combination of isotonic crystalloid and colloid therapy, using hydroxy-ethyl starch, Pentaspan, or blood or fresh frozen plasma transfusion is required, in order to maintain blood volume, hydration status, and colloid oncotic pressure necessary for tissue oxygen delivery to body tissues.

- d. Plasma and synthetic colloid use: Patients with parvovirus enteritis are high risk, critically ill patients that frequently suffer from significant losses of plasma proteins into the intestinal lumen. This can lead to hypoproteinaemia, hypoalbuminaemia, and, occasionally, coagulation abnormalities. The use of fresh frozen plasma and/or synthetic colloids in these patients is controversial, but it also has sound rationale. Hypoproteinaemia and hypoalbuminaemia are negative prognostic indicators in patients with severe illness. They are associated with decreased effective circulating blood volume, poor tissue perfusion, and worse outcome. Administration of fresh frozen plasma can reasonably be considered in any patient with parvovirus enteritis that is critically unwell. Optimum benefit from transfusion is obtained when transfusion is administered early in the course of disease, at dose rates of 10-30 ml/kg intravenously over four to six hours. Following plasma transfusion, synthetic colloids such as hydroxy-ethyl starch or Pentaspan may be administered at a rate of 10 ml/kg/day in addition to fluid therapy to provide for maintenance and ongoing losses.
2. **Dietary management** – Nil per Os (NPO) for an initial six to twelvehour period following admission to hospital may be beneficial in most cases of diarrhoea in dogs and cats. NPO decreases the number of osmotic particles available in the intestinal lumen contributing to osmotic diarrhoea, and allows more rapid re-growth of intestinal villi, and resumption of intestinal function. Following an initial short period of bowel rest, micro-ental nutrition should commence. Micro-ental nutrition, using glucose, hydrolyzed proteins and isotonic electrolyte solutions, such as Lectade or Vytrate, have been shown to maintain intestinal mucosal health, reduce atrophy of the intestinal lumen cells, and improve recovery rates from diarrhoea. The rate of administration is 1-2 ml/kg/hr. These solutions may be used in the presence of ongoing vomiting and diarrhoea, as they are readily absorbed by gastric mucosal cells, and enterocytes, even in the presence of disease. Following 12 hours of micro-ental nutrition, more

complex nutritional formulations should be fed to the patient – typically by nasogastric or oesophagostomy tube. On resumption of feeding, a low fat, high biological value protein, primarily carbohydrate diet should be fed. The carbohydrate present in rice is best because its starch is more completely assimilated than other carbohydrate sources. Select a lean protein source such as chicken. Veterinary prescriptions diets such as Hill's i/d diet are ideal for this purpose.

3. **Motility modification** – narcotic analgesics – loperamide (Imodium), Diphenoxylate (Lomotil) – increase segmental bowel contractions, and have been used for symptomatic management of acute diarrhoea. **They should not however be used in patients with infectious diarrhoea caused by bacteria or viruses, as they have been shown to prolong illness, and delay clearance of intestinal bacterial toxins.**
4. **Protectants and absorbents** – kaolin/pectin, charcoal, and barium sulphate – have questionable efficacy. Bismuth subsalicylate is the most useful, as it has anti-enterotoxin, antibacterial, anti-secretory and anti-inflammatory effect, and has been used as an adjunct to therapy, but is not considered essential in most cases.
5. **Antibiotics** – are usually not indicated except in cases of immunosuppression, fever, leukocytosis, leukopenia, melena, haematochezia, and shock. Initial antibiotic choices for diarrhoea include antibiotics effective against enteric bacteria – usually a combination of mixed aerobic and anaerobic, gram positive and gram negative bacteria. First generation cephalosporin antibiotics are commonly used at a dose of 22 mg/kg IV q 8 hrs. Alternatively, for aerobic infections, enrofloxacin at 5 mg/kg SC q 24hrs is effective. For anaerobic infections, metronidazole is the preferred agent, at 20 mg/kg IV q 24 hrs.
6. **Anti-emetic therapy** – anti-emetic therapy using metoclopramide, maropitant, Ondansetron or dolasetron may be used in patients that have persistent vomiting not caused by intestinal obstruction, in order to minimize fluid loss from the patient, and to improve patient comfort. Some clinicians insert naso-gastric tubes to remove gastric secretions as an adjunct to the use of anti-emetics, and to facilitate administration of micro-ental nutrition. The use of these tubes is associated with a dramatic reduction in nausea and vomiting – particularly in puppies with parvovirus enteritis and in patients with pancreatitis.
7. **Analgesia** – Opiate analgesics such as butorphanol or fentanyl are preferred in most cases of acute infectious gastroenteritis, and should be administered by continuous infusion for optimum benefit for the patient. Non-steroid anti-inflammatory drugs or steroid anti-inflammatory drugs are contraindicated in most cases of acute infectious diarrhoea because they decrease blood flow to the gastric mucosa, and can result in intestinal ulceration and acute renal failure – particularly in dehydrated patients.

For the most part, this supportive care will ensure optimum conditions or opportunity for gut function to return to

normal, once the underlying infectious cause resolves. Obviously, it is important in any patient to support airway, respiratory function, neurological function and renal perfusion with intravenous fluid therapy, oxygen therapy and other supportive measures.

Appendix 1: Nutritional Support of the Patient with Canine Parvovirus Enteritis

Step 1: Micro-Enteral Nutrition

The term micro-enteral nutrition was developed to define the delivery of small amounts of water, electrolytes, and readily absorbed nutrients (glucose, amino acids, and small peptides) directly to the gastrointestinal tract. These nutrients may be given as a bolus infusion every one to two hours or as a constant rate infusion directly into the oesophagus and stomach, duodenum, or jejunum via a feeding tube. Why do we give micro-enteral nutrition? – To improve blood flow to the gut, improve gut function, and to improve the tolerance to more complex diets when we begin feeding them.

Protocol for Micro-Enteral Nutrition

- Begin within 2-12 hours of admission to the hospital
- Solution - lectade, gastrolyte, OR 0.45% NaCl + 2.5% dextrose solution
- Additives:
 - Potassium chloride- 20 mEq/L
 - Amino acids - 3% amino-acid solution. Addition of amino acids promotes a protein-sparing effect in critically ill patients. Amino acids should not be supplemented in micro-enteral formulations for use in patients with pancreatitis
 - Glycine hastens the resolution of diarrhea, improves intestinal morphologic characteristics in patients with diarrhea, and enhances the uptake of glucose.
 - Glutamine is the preferred energy source of rapidly dividing cells (GUT cells, lymphocytes, and fibroblasts). Glutamine plays a role in the maintenance of the integrity of the GI mucosal barrier and preventing bacterial translocation
- Rate of administration:
 - CRI rate of 0.5 - 2.0 ml/kg/hr OR
 - Bolus infusion of 1.0-4.0 ml/kg every 4 hours
 - Increase volume administered by 0.2-0.5 ml/kg/hr every 8-12 hours if patient is tolerating the solution
- Begin feeding a commercial liquid diet if micro-enteral nutrition solution is being tolerated after 6-18 hours

Step 2: The Nutrition Formula – Beyond Micro-Enteral Nutrition

When formulating a diet for the patient, the following are taken into consideration

- Fluid Requirements - normal patient's fluid requirements are between 60 and 80 ml/kg/day
- Energy Requirements - the energy requirements of critically ill humans rarely approach normal maintenance requirements, due to a reduction in physical activity associated with illness. There are numerous methods and recommendations for estimating patient caloric needs

during illness - none of which has been validated. Current practice is to feed critically ill patients resting energy requirements (RER) only, and to only multiply RER in cases of severe malnutrition, and only then once the patient is tolerating feeding

$$\text{RER} = \text{Bodyweight (kg)} \times 30 + 70 \text{ (dog } > 2\text{kg)}$$

$$\text{RER} = \text{Bodyweight (kg)} \times 70 \text{ (dog } < 2\text{kg)}$$

$$\text{RER} = \text{Bodyweight (kg)} \times 40 \text{ (cat)}$$

$$\text{RER} = \text{Bodyweight (kg)} \times 0.75$$

Step 3: Feeding the Patient

Enteral feeding should be initiated within 12-24hrs of admission to the ICU. If oral feeding is not possible or feasible, a gastric feeding tube (nasogastric, oesophagostomy, or gastrotomy tube) should be placed. Feeding may be by bolus feeding, or by constant rate infusion. The residual gastric volume should be monitored, and the feeding regime adjusted as outlined below.

In all cases, the target energy and protein requirements for the patient should be met by the third day of feeding. If the protein content cannot be obtained by this time, a diet containing higher protein content should be fed.

Bolus Feeding Protocol for Gastrotomy, Oesophagostomy, or Nasogastric Tubes

If patient has had prolonged anorexia (2-3 days) use the following feeding protocol:

- Day 1 = feed 1/4 - 1/3 of daily feed requirement in several small feeds
- Day 2 = feed 1/2 - 2/3 of daily feed requirements in several small feeds
- Day 3-4 = feed entire feed requirements in several small feeds

NB - if feeding via G – tube, oesophagostomy tube, or nasogastric tube, suction the tube first - if you suction a volume greater than 1/3 of the previous volume of feed given, delay the next feed for 2-3 hours, and feed 1/3 - 1/2 the volume of the last feed. Consider the presence of intestinal ileus, and manage with metoclopramide and/or cisapride.

Appendix 2: Feeding Tube Placement Guide

Nasogastric Feeding

- Use silicone or polyurethane tubes (polyvinyl tubes can harden and be difficult to remove)
- Following placement of local anaesthetic in the nasal cavity, pass tube in ventral nasal meatus, and pass to the level of the distal esophagus – this reduces the chance of gastric contents ascending about the tube and causing a reflux esophagitis. Note that when feeding patients with nasal-esophageal tubes must be at a slow rate to avoid a rapid influx of food into the distal oesophagus, and subsequent regurgitation
- Following placement, apply suction to the tube – if air is freely aspirated, or the animal is coughing, the tube is probably in the lungs – relocation of the tube is necessary
- Radiography is desirable if doubt exists over tube location

and depth

- Resistance is normally felt on passing tube through caudal nasal meatus due to narrowing caused by ventral expansion of the maxilla-turbinate
- Following use, capping tube prevents gastric accumulation of air
- Following use, flush the tube with water
- Complications:
 - Gastric reflux
 - Inhalation of food
 - Patient intolerance of the tube

Oesophagostomy Tube Feeding

- Select the appropriate tube:
 - Polyurethane or red rubber tube
 - Size 10-12 Fr (cats and small dogs); 16-22 Fr (dogs)
- Decide on location of distal tube – oesophagus vs. stomach. Placement of the oesophagostomy tube in the distal esophagus reduces the risk of gastric reflux into the distal esophagus. However, placement of the tube into the gastric lumen allows assessment of residual gastric volumes, gastric suctioning, and hence reduced nausea. Distal tube placement into the gastric lumen is considered superior to distal esophageal placement
- Clip the skin over the LEFT side of the neck
- Place a mark on the tube (measured from its tip) equivalent

to the distance from the mid-cervical esophagus (insertion point) to the last rib (stomach)

- A curved Carmalt forceps (or similar instrument) is placed through the mouth down to a midpoint in the cervical esophagus on the left side of the neck, and pushed outward to tent the skin over the esophagus
- The skin is incised over the tip of the forceps; the skin incision is extended through the subcutaneous connective tissues, and through the wall of the esophagus. The esophageal incision should be as small as possible – just large enough to allow the tip of the forceps to be pushed through
- Push the forceps through the skin incision. Grasp the tip of the oesophagostomy tube with the forceps, and pull the tube through the mouth with the forceps. The tip of the tube is then re-directed down the esophagus to the pre-determined length – using the Carmalt forceps to direct the tube
- Confirm proper tube placement by suctioning the tube - if gastric contents are obtained on suction, the tube is in the stomach
- The tube is secured by placing an overlapping, interlocking suture pattern (Chinese Finger Trap), and bandaging to the neck

References available on request