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Medical Nursing Case Challenges Mini Series

Session Two: Infectious Challenges

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Chronic Diarrhoea.

Faecal Analysis.

A faecal examination is the microscopic evaluation of faeces. The test is indicated for pets with diarrhoea, straining, inability to maintain an ideal body condition score, lack of appetite or vomiting. In many cases samples are sent to external laboratories for analysis, but microscopic examination can be performed within the veterinary practice.

Faecal smears can be easily performed and microscopically examined. Their purpose is to provide a quick and simple but relatively insensitive method for demonstrating helminth infection and identifying the eggs and larvae present. This technique is non-quantitiative, (it doesn't provide a specific number of eggs, larvae) method and is a very insensitive method unless there is a very high helminth burden. Interpretations should be made carefully as debris can overlay eggs or larvae.

Materials required:

- Microscope slides
- Coverslips
- Saline solution (0.85%) or water
- Microscope
- Spatula to spread out the faecal matter.

Worm egg counts are the most commonly performed, this can be achieved with the modified McMasters egg count technique, other methods of confirming parasite presence can be achieved by floatation methods, larval culture, Baermann techniques or by faecal sedimentation techniques.

A full faecal analysis would need to be able to identify whether there are any undigested materials such as fats and proteins in the faeces, as these can show whether there is a possible maldigestion or absorption disorder.

Pooled samples.

In many cases shedding can be intermittent and therefore pooled samples are required in order to make a positive identification of a causal agent. Normally faecal samples are collected over a three day period and all samples are sent to the laboratory for analysis.

Pharmaceuticals.

Many different pharmaceuticals can be utilised in the treatment of the many different gastrointestinal problems that can be seen in veterinary practice. It is important when conducting nursing clinics to read the clinical history and to make note of the medication regimes that the veterinary surgeon has prescribed for the animal. In some chronic cases drug dosages are tapered to effect and therefore support from the veterinary nurse may be required in order to help clients achieve this.

Vitamin B12 (cobalamin) injections are commonly used in animals with low blood cobalamin, which is commonly seen in animals with gastrointestinal problems. Weekly injections in some cases may be required in some patients for a number of weeks, and this is a role for the veterinary nurse to undertake.

Adverse reactions to food – GI.

Food allergies (food hypersensitivity) have an immunological basis, and cause an adverse reaction to food or food additive. Food intolerances are non-immunological, where abnormal physiological responses to food or a food additive occur.[1] Specific food additives, which are known to cause problems include onions and propylene glycol, which can cause haematological abnormalities in cats. Lactose intolerance is a relatively common metabolic adverse reaction in dogs and cats. Diarrhoea can develop when given cows or even goat's milk, due to the lactose content being higher than that of

bitch's or queen's milk. Gluten sensitive enteropathy has been well documented in Irish Setters.

The role of food allergies in canine and feline IBD in unknown. It is thought that hypersensitivity to food is involved in the pathogenesis of this syndrome. The role of elimination food trials often alleviate the signs of IBD, which dies seem to imply that food allergies or food intolerances play a role in this syndrome.

Feeding Through.

In human medicine there is a continuing bank of evidence indicating that "feeding through" diarrhoea with an appropriate diet can be beneficial. Comparisons can be difficult, as many cases of diarrhoea in man tend to be secretory, whereas osmotic diarrhoea is more common in dogs and cats. The advantages of feeding through include maintaining mucosal health, reducing the risk of bacterial translocation, and aiding in the "flushing out" of the causal factor. Feeding through should not be used in animals that are vomiting or severely dehydrated. With animals that are vomiting or the GIT is not functioning, the animal's nutritional requirements should be met through parenteral nutrition. The obvious disadvantage is the risk of "accidents" in the house, and the increase in faecal volume. Whether food is withheld or not, unlimited access to water must be maintained at all times. The use of oral rehydration solutions containing carbohydrates, peptides and electrolytes should be advocated in these cases. When using this method feeding a diet, which won't cause an inflammatory reaction in the future is important. Dietary antigens can cross the compromised gastrointestinal mucosa and set up a hypersensitivity reaction. Use of a novel protein and/or carbohydrate source should be recommended. Once the animal is then well, and transferred to its original diet, it is unlikely to have any reactions to the original protein source.

Adsorbents are frequently used in these cases, and can be provided with the addition of pro- and prebiotics, and glutamine. The purpose of adsorbents is to bind bacteria and toxins, to protect the intestinal mucosa, and potentially for an antisecretory effect. Kaolin has been traditionally used, but montmorillonite has been reported to be twenty times more effective than kaolin at adsorbing pathogens.

Chronic diarrhoea can be a clinical signs of a vast number of different problems. It is essential to determine the underlying cause so that specific treatments for each individual case can be initiated. Dietary management plays an important role in long-term management of these cases, as relapses can be frequent. As each case is different there is no one specific diet aimed at animals suffering from chronic diarrhoea. Recommendations of low fat, single (novel) protein diets are given. Yet in some cases diets that have high fibre content are the diet of choice in resolving the clinical symptoms. The chronic diarrhoea disorders such as Inflammatory Bowel Disease (IBD), Protein-losing enteropathy (PLE), Antibiotic-responsive diarrhoea (ARD) and Irritable bowel syndrome (IBS) will be discussed separately due to their significance in veterinary medicine.

Inflammatory Bowel Disease (IBD).

The management of IBD involves the combined use of anti-inflammatory medication alongside the use of a single novel protein diet. A full dietary history is required in order to ascertain what protein sources the animal has been exposed to in the past. Diets, which contain novel protein sources such as venison or duck, have proved to be beneficial, as initially lamb was used as a novel protein source. Subsequently, many lifestage diets now include lamb and marketed at animals with sensitive stomachs. When using an elimination diet it is important to challenge the system to see if the original diet was the initial cause. See Feeding an Elimination diet for further details.

The use of fermentable fibre in these cases should not be under estimated. Colonic bacteria easily digest fermentable fibres, which in turn produce butyrate, a short chained fatty acid. Butyrate is the primary energy source for colonocytes, and thus helps to maintain gut health. Other SCFA are also produced, which reduce the colonic pH, and thus reduce the risk of pathogenic bacteria from colonising the area. As the body absorbs the SCFA there is an increase in the absorption of electrolytes and water from the colon.

When feeding an animal with IBD the nutritional goal is to provide adequate nutrient intake in order to meet the requirements of the individual, but also to compensate for ongoing losses through the

gastrointestinal tract.

Protein-losing enteropathy (PLE) and Lymphangiectasia.

Lymphangiectasia is characterised by abnormalities of the intestinal lymphatic system. The condition can be as a primary defect of the lymphatic system, or secondary as a consequence of severe intestinal infiltrative disease (e.g. IBD, lymphosarcoma).[4] Lymphangiectasia is the most common cause of PLE in dogs and cats. Not all animals present with the clinical symptom of diarrhoea, but often present with progressive weight loss even with a good calorific consumption. The leaky intestinal lymphatics result in hypoalbuminemia and loss of colloidal oncotic pressure.

When feeding animals with PLE the key is to control dietary fat levels. Long chain triglycerides provide a major stimulus for intestinal lymph flow; the protein content of the lymph tends to increase with the dietary fat content. Limiting dietary fat content will reduce the lymphatic flow, reduces lacteal and lymphatic distension and thus minimise protein losses. Dietary fat levels of <10% DMB for dogs and <15% DMB for cats is recommended. The protein levels of the diet is important, feeding a high-protein diet has shown to unsuccessful. The cause of the PLE is important, as in cases where severe IBD is the underlying cause; care should be given when selecting a protein source. Protein levels should be of a high biological value, and levels in excess of 25% DMB for dogs and in excess of 35% DMB in cats have been recommended.

Antibiotic responsive diarrhoea (ARD).

ARD should be best managed with a combination of antibiotics and diet. Dependant on the response to antibiotics will dictate the length of the course required. The antibiotic of choice in cases of ARD are tylosin or oxytetracycline. Little research has been conducted on the casual factors of ARD, yet speculation can be made that gastrointestinal factors must promote bacterial colonies that are causal agents for the diarrhoea. The use of prebiotics has been recommended in these cases in order to promote gastrointestinal health. Dietary manipulation should be considered if the animal is on an inappropriate diet that does encourage growth of pathogenic bacteria.

Irritable Bowel Syndrome (IBS).

IBS is a difficult disorder to diagnose, and thus only on limitation of all other causes of chronic diarrhoea can IBS be made. Clinical signs of IBS include bouts of abdominal pain and chronic large intestinal diarrhoea. The clinical signs can be intermittent, with stress also being a probably trigger factor. The addition of fibre to the diet has proven to be of a slight benefit in some cases.

Colitis.

Colitis is the most common causes of diarrhoea in the dog and cat. Colitis (inflammation of the colon) is characterised by the presence of mucus covering the faeces, and/or fresh blood. There are many causes of colitis, including stress. Many dogs have colitis diarrhoea when being hospitalised at veterinary practices, due to stress levels. Dietary management of mild cases is recommended, though in chronic or severe cases medical treatment may be required alongside dietary management. Modifications to the diet include:

- High-fibre diets that normalise the transit time and bind faecal water. Fibre also acts as a prebiotics aiding the strains and populations of gut bacteria.
- Low-fibre, highly digestible diets will aid in reducing the quantity of undigested food entering the colon.
- Hypoallergenic diets can also be used when an intolerance or hypersensitivity is present.

When the initiating cause of the colitis is unknown, dietary modifications can be very much trial and error, as the first two (high-fibre and low-fibre) do contraindicate each other. This should be relayed to owners so that they have a better understanding of the problem, and do not get frustrated with dietary managements. It is also recommended to advice owners that colitis is normally seen spasmodically, with intervals of normality between bouts.

Importance of Fibre for Chronic Diarrhoea cases.

Fibre plays an important role in the gastrointestinal system acting as a prebiotic, and in influencing absorption and motility rates. Manipulation of the fibre type and content within the diet can be utilised in the treatment and/or management of gastrointestinal disorders. There are numerous gastrointestinal disorders and with each individual reacting differently to different diets (and the different fibre types and content) it is important to obtain a full nutritional / diet history from owners about their pets. Other factors such as stress, water consumption, activity levels and genetics all play a part in many gastrointestinal disorders, hence a full detailed history incorporating this factors is required.

Fibre refers to a range of compounds classed as complex carbohydrates that are resistant to the action of digestive enzymes, (Figure1). The primary function and benefit of adequate dietary fibre is to increase bulk and water in the intestinal contents, it also helps to promote and regulate normal bowel function and transit times. Fibres include cellulose, hemicellulose, pectin gums and resistant starches. Fibre is classified by its chemical structure but also by their rate of fermentation by intestinal bacteria, digestibility and indigestible fractions, solubility in water, water-holding capacity and viscosity, (Figure 2), (Gross *et al.,* 2000).

Complex Carbohydrate Type	**Function**	**Digestion Site**	**Digestion Products**
Starch, glycogen	Storage polysaccharide in plants and animals	Small Intestine (enzymatic)	Mono- and disaccharides (glucose, maltose)
Hemicellulose, Cellulose	Structural parts of plant cell walls	Large intestine (microbial fermentation)	Volatile fatty acids (acetate, propionate, butyrate)
Lignins, Cutins, Waxes	Associated cell wall substances	Not digested or fermented	Excreted in faeces
Gums, Pectins, Mucilages	Naturally occurring polysaccharides in plants	Large intestine (microbial fermentation)	Carbon dioxide, methane, hydrogen, volatile fatty acids

Figure 1: Classification and digestion of complex carbohydrates, (Gross *et al.,* 2000).

<table>
<tr><th>Carbohydrate and fibre fractions</th><th>Method</th><th>Fibre solubility</th><th>Total dietary fibre analysis</th><th>Crude fibre analysis</th></tr>
<tr><td>Fructans, galactans, mannans, mucilages</td><td rowspan="2">Rapidly fermentable</td><td rowspan="2">Soluble Fibre</td><td rowspan="4">Total dietary fibre</td><td rowspan="3"></td></tr>
<tr><td>Pectin</td></tr>
<tr><td rowspan="2">Hemicellulose</td><td>Moderately fermentable</td></tr>
<tr><td>Slowly fermentable</td><td rowspan="2">Insoluble fibre</td></tr>
<tr><td>Cellulose</td><td rowspan="2"></td><td rowspan="2">Crude Fibre</td></tr>
<tr><td>Lignin</td><td>Not digested or fermented</td><td rowspan="4"></td><td rowspan="4"></td></tr>
<tr><td>Resistant Starch</td><td>Moderately fermentable</td><td rowspan="3"></td></tr>
<tr><td>Starch</td><td>Enzymatically digested</td></tr>
<tr><td>Mono- and disaccharides</td><td>Absorbed</td></tr>
</table>

Figure 2: Physiochemical and analytical properties of dietary fibre components, (Gross *et al.,* 2000).

Those fibres that are rapidly fermented (e.g. pectins), by the gastrointestinal bacteria, produce more short-chained fatty acids (SCFAs) and gases in a shorter period of time compared to fibre sources that are fermented more slowly, this can lead to borborygmus and flatulence. Pectins are commonly found in apple and citrus. The most commonly utilised fibre sources in pet foods contain a mixture of pectins, hemicellulose and cellulose and this mix is classed as moderately fermentable. These fibre mixtures used include rice bran, oat bran, wheat bran soy fibres, soy hulls and beet pulp. As the fermentation rate of the fibre used in the diet decreases this will have the effect of increasing gastrointestinal transit time, and increasing the faecal bulk. This can help to have the effect of increasing satiety in the animal, and is often included in weight loss diets. These slowly fermentable fibres (e.g. cellulose) are really effective in stool bulking agents because they retain their structure for longer and are thus able to bind water into the stool. This increase in faecal bulk / volume is advantageous for the treatment and prevention of irritable bowel syndrome and constipation.

The important end products of fibre fermentation are the SCFAs, and include acetic, butyric and propionic acids and are the preferred energy source of the coloncytes, which derive more than 70% of their energy requirement from the luminally derived SCFAs, (Bergman, 1990). There is a rapid turnover of the epithelial cells within the gastrointestinal tract, and therefore high energy needs. Dogs fed diets containing fermentable fibres have an increased colon weight, mucosal surface area and mucosal hypertrophy when compared to dogs fed diets containing non-fermentable fibres, (Hallman *et al.,* 1995). These changes indicate an increased absorptive potential, which is of benefit to the animal, as it will aid in the prevention of diarrhoea by enhancing the absorption of sodium. This in turn maintains the normal intestinal electrolyte and fluid balance.

The other beneficial effects of the production of SCFA include promotion of the growth of the indigenous microflora and inhibiting the proliferation of pathogenic microbes, (Kerley and Sunvold,

1997), acting as a prebiotic. Hence if probiotics are being administered to an animal, probiotics are also required in order to promote their proliferation within the gastrointestinal system.

Constipation.

Constipation is a clinical sign that is characterised by the absence, infrequent or difficult defeacation associated with retention of faeces within the colon and rectum, (Buffington *et al.,* 2004). There are numerous causes of constipation, and these can be drug-induced, inadequate water consumption, limited activity levels, and pain causing the pet not wanting to defeacate.

Maintenance of normal hydration is vital in these cases, and use of a tinned formulation of diet can aid, and even addition of water to the wet diet can help. In all cases of constipation, resolution of the presenting episode needs to occur prior to the initiation of any dietary changes. Increasing the fibre content of the diet with slowly fermentable fibres is indicated, as the animal needs an increase in gastrointestinal transit time and in stool volume. Dietary transitions to these diets should be performed gradually by increasing the fibre content in increments. If side effects occur (abdominal cramping, flatulence) it is recommended that the fibre content is decreased by 5% DMB and the animal reassessed, (Ackerman, 2012). Titration of the dietary fibre content of the diet can be achieved by the use of food combinations with diets of varying crude fibre content.

Cats will occasionally suffer from chronic recurrent constipation, and may lead to where the colon becomes severely and irreversible dilated and flaccid – megacolon. The cause of megacolon is not known but can be linked to factors such as inadequate fibre intake, ingestion of excessive hair, environmental and psychologic factors such as painful defeacation, obstruction of the colon or anorectum, neuromuscular diseases, dehydration, hypokalaemia and drug related constipation, (Buffington *et al.,*2004). Recommendations need to be made to the owner regarding diet, water intake, activity levels and environmental enrichment.

In situations of severe constipation or megacolon, where colonic motility is not present, the use of a high-fibre diet is not recommended. A highly digestible diet (DM digestibility >90%) is the diet of choice. Colonic motility modifiers such as cisapride can be useful, treatment is very much case specific, and can be dependent on the initiating cause, (Ackerman, 2012). Inn early cases of megacolon an increased fibre content can be beneficial, (Chandler, 2011).

Exocrine Pancreatic Insufficiency (EPI).

In cases of EPI it is important that a highly digestible diet is used, that is low in fat (≤ 2g/100kcal) and fibre (<2% dry matter base). The addition of exocrine pancreatic enzymes is required and diets high in fibre can impair enzyme activity.

Pancreatitis.

Dietary long-term control of pancreatitis is vital, but initial confirmation of the presence of hyperlipidaemia needs to be obtained. The presence (or not) of hyperlipidaemia will determine which diet to utilise. If triglyceride levels are within normal levels, then a highly digestible, controlled fat level diet is indicated. If hyperlipidaemia is present, fat levels need to be tightly controlled, but these types of diets can have a corresponding high-fibre content thus reducing digestibility, (Ackerman, 2008).

Small Intestinal Bacterial Overgrowth / Antibiotic Responsive Diarrhoea.

The use of a fermentable fibre source is vital in cases of SIBO/ARD, as a prebiotic for the indigenous bacterial populations. In order to help prevent the overgrowth of the pathogenic bacteria, a healthy maintained and balanced microbiota is required. Bacteria exert this effect by their patterns of SCFA production and through direct inhibition of the growth of other microbial species.

Chronic Idiopathic Large Bowel Diarrhoea (CILBD).

CILBD is presumed to be a stress-related disorder that may be concomitantly influenced by other factors such as inflammatory disease, dietary indiscretions, pathogen overgrowth, parasitic infection and neoplasia. Some suffers of CILBD can be fibre-responsive, with fibre aiding in promoting colonic health. Dietary management is recommended in all mild cases, though in chronic or severe cases

medical management may be required alongside dietary management. Modifications to the diet include:

- High-fibre diets that normalise the transit time and bind faecal water, (fibre also acts as a prebiotic aiding the strains and populations of gut bacteria).
- Low-fibre, highly digestible diets which aid in reducing the quantity of undigested nutrients entering the colon.
- Hypoallergenic diets, which can also be used when an intolerance or hypersensitivity is present.

When the initiating cause of the colitis is unknown, dietary modifications can be very much a case of trial and error, as the first two dietary modification options contradict each other, (Ackerman, 2012).

Diabetes Mellitus.

High fibre diets have been traditionally used for dogs suffering from diabetes mellitus. The increased fibre reduces the rate of absorption of simple sugars present in the diet. Thus reducing the post-prandial hyperglycaemia spike. The production of SCFAs also modify the secretion of some of the digestive hormones and sensitivity of tissues to insulin. Complex carbohydrates should provide 50-60% of the calories in diets for dogs, (Michel, 2005).

When feeding diabetic cats a high protein, low carbohydrate diet has shown to enhance insulin sensitivity.

Renal Disease.

The types and content of fibres in the diet designed for animals with renal disease can be of benefit in its management. As already discussed SCFA are an important energy source for the intestinal cells and can increase blood flow to the intestine. Nitrogenous waste products in the blood are presented to the intestinal lumen where urease, an enzyme produced by intestinal bacteria, hydrolyses the urea into ammonia and carbon dioxide. The ammonia is then utilised by the intestinal bacteria. This process means that nitrogenous waste products are excreted in faecal matter, rather than in urine by the kidneys, (Ackerman, 2012). Dietary fibre may also be beneficial for improving gastrointestinal motility in dogs with renal failure. Colonic transit times can be decreased in moderate renal disease as it alters duodenojejunal motility.

The decrease of protein levels in renal diets results in a relative increase of the fat and carbohydrate levels. This results in an increase in calorific value and probable palatability due to the fat content. The increased fat content can also cause digestive upsets such as diarrhoea. If the dietary transition has been made of a period of time then this is not a common side effect. However, if the diarrhoea persists a combination of a higher fibre diet, that has also a low phosphate levels alongside the renal diet can be utilised. The balance will depend on the pet’s response to the levels of fibre required.

Conclusion.

Diets formulated to manage intestinal disease should ideally contain between 3 and 7%, but no more than 10% fibre on a dry matter base, (Sunvold and Reinhart, 1997). A balance of fermentable and non-fermentable fibre sources should be utilised within the diet in order to gain a balance of promotion of motility and supply of SCFAs, whilst maintaining digestibility and palatability of the diet for the animal.

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Advanced Nutrition

Improving the Nutrition of Inpatients.

With all sick companion animals the nutritional goals is for the patient to eat the designated diet in its own environment. Unfortunately in critically sick animals they will be in a hospital environment, where the added stress of this different environment can effect food consumption. Most animals will require or benefit from a veterinary therapeutic diet, but the initial goal is to ensure that the patient is receiving its daily calorific requirement. Analgesia should not be forgotten, as pain can reduce food intake in some animals. The calorific intake required by the patient depends on several factors:

- The rate of energy use for basal metabolism (resting).
- Nutrient assimilation
- Body temperature maintenance
- Activity levels.

Energy requirements during sickness are based on RER values. This is due to the assumption that the patient is inactive and is often confined to a small area. Due to this calculated energy requirements, using an illness factor, are only a guideline. Daily weighing of the patient, accessing healing rates and accessing lean body mass is a good indicator that the patient is receiving sufficient calories. In herbivores (horses and rabbits) vital parameters should also include gastrointestinal mobility and faecal volume, appearance and frequency should be monitored.

Basal Energy Requirements (BER) = the amount of energy required by a healthy animal in a thermoneutral environment, whilst awake but not exercising, 12 hours after eating.

Resting Energy Requirement (RER) = The RER differs from the BER as it includes energy expended for recovery from physical activity and feeding, and is calculated from these equations dependant on the weight of the animal. The RER for dogs and cats can be calculated with these two formulas:

RER (kcal/day) = $70(BW_{kg})^{0.75}$ (if body weight (BW) is less than 2kg).

Or, $30(BW_{kg}) + 70$ (if bodyweight (BW) is between 2 and 45kg).

It has been calculated that RER maybe 1.25 BER, but in many texts often regard the two as interchangeable values.

As with all hospitalised patients, human and animal, malnutrition has been associated with increased in infectious morbidity, prolonged hospital stay and an increase in mortality.

The volume of liquid diets administered at each bolus feeding in dogs and cats should not exceed 50mls/kg. This is only an estimate and each animal should be judged on an individual basis. Tolerance to liquid diets is best when small feedings are delivered frequently.

Protein energy malnutrition (PEM) can occur during periods of illness, injury or even after routine operations. During these periods the body shuts down systems in order to conserve the limited resources. Physiological changes such as a drop in blood pressure, reduction in cardiac output and a drop in oxygen consumption occur. This is clinical shock and is known as the Ebb phase. Clinical shock is treatable but the length of time that shock is suffered from is variable, and can ultimately be lethal. Following the Ebb phase is the Flow or Hypermetabolic phase. During this second phase the patient's body defence and repair mechanisms are initiated. This process is where anorexia and starvation differ.

Nutritional support with adequate protein levels is vital, patients in a catabolic state will utilise the skeletal muscle proteins. Sufficient calories need to be supplied to the patient from fats and carbohydrates. This will ensure that proteins are not used as a source of energy. The quality of the proteins provided is of importance as is the digestibility. Specific amino acids are supplemented to critical care diets. Glutamine is an important substrate for the increased levels of gluconeogenesis, used in rapidly dividing cells. Any deficiency in this amino acid has shown to lead to gut mucosal atrophy, and an increase in bacterial translocation, due to a compromise of the mucosal barrier. This can lead to the suggestion that glutamine may behave as a 'conditionally essential' amino acid during severe illness. The essential amino acid arginine has a positive effect on the immune system and can subsequently improve survival times of septic patients. Many enteral diets are supplemented with both glutamine and arginine. High dose of glutamine have a trophic effect on the gut mucosa.

The use of a novel protein source in these cases has been advocated. Due to atrophy of the gastrointestinal tract, protein antigens can cross through to the bloodstream and set up hypersensitivity processes.

The supplementation of vitamins and minerals for hospitalised patients will depend on the disease and the severity of that disease that is present. Sodium, chloride, potassium, phosphate, calcium and magnesium should be used for short-term nutritional support. All animals that receive intravenous fluid therapy with or without parenteral support should have daily electrolyte levels monitored. If any polydipsia or polyuria is present, supplementation of the water-soluble vitamins is required. Zinc does aid in promoting wound healing and plays a role in protein and nucleic acid metabolism. Supplementation of nutritional support diets with zinc has been recommended.

Carbohydrates within any critical care diet, needs to be of a very high digestibility. The quantities of fibre need to be kept to a minimum, as this will decrease digestibility and bind up important nutrients. The level of carbohydrate in the diet needs to be adequate enough to supply the required calories for recovery.

The quantity of fat in a critical care diet does need to be increased. A higher level of calories can be obtained from fat rather than via carbohydrates. The inclusion of omega –3 fatty acids can help decrease any inflammatory response.

	Protein	Fat	Carbohydrate
Dog	20-25	50-55	25-26
Cat	25-37	41-50	22-25

Table 1: Macronutrient levels in critical care diets (as % energy content of diet).

Supportative Feeding Methods.

Supportative feeding methods should be considered when there is a history of greater than 10% weight loss, decreased food intake, increased nutrient demands due to trauma or surgery, increased nutrient losses resulting from vomiting, diarrhoea, burns or scolds, and acute exacerbation of chronic disease. The ideal diet to use in these cases should be highly palatable, digestible and have a high energy density. A relatively high percentage of energy should be achieved as protein and fat rather than carbohydrates.

Naso-oesphageal Tubes.

Naso-oesphageal tubes are generally well tolerated by cats and dogs and are suitable for short term nutritional support, usually 3-7days, though longer periods have been documented. Contraindications for the use of these tubes include unconsciousness, vomiting, disease or dysfunction of the pharynx, larynx, and nares, swallowing reflex, oesophagus and stomach. The preferred placement of the naso-oesphageal tube is in the caudal oesophagus, rather than the stomach as it reduces the risk of reflux oesphagitis.

Once the tube has been placed introduction of a small amount of water injected slowly into the tube to see if a cough reflex is induced, indicating aspiration. Lateral radiographs can also identify that the tube has been correctly placed. Due to the narrow bore of the tube blockages do occur. Injection of 5-10mls of water into the tube post introduction of the liquid diet, should aid in prevention of any blockages. If blockages do occur small amounts of carbonates drinks, cranberry juice or solutions of pancreatic enzymes have shown to aid in their removal. Pre feeding administration of water is required to ensure that the tube is still positioned correctly.

Pharyngostomy Tubes.

Pharyngeostomy tubes are commonly used in cats, which have suffered facial trauma, usually after a road traffic accident. Pharyngostomy tubes are of use when bypassing the nose and mouth is required in order to administer nutritional support. Animals that don't tolerate naso-oesphageal tubes can have pharyngostomy tubes placed. These types of tubes have been largely replaced by oesphgastomy tubes or gastrostomy tubes that are placed percutaneously. Aseptic placement of the tube under general anaesthetic is required. Frequent cleaning and inspection of the tube is necessary under aseptic conditions.

Percutaneous Endoscopic Gastrostomy (PEG) Tubes.

Placement of these tubes is required under general anaesthetic, and needs to be in place for at least five days before removal. PEG tubes are utilised when long-term nutritional support is required, and when oesophageal problems are present. In many cases of gastric dilation/volvulus (GDV), PEG tubes are placed at time of gastroplexy. Adhesions between the gastric serosa and the peritoneum can form within 48-72 hours. It should be noted that in malnourished patients these adhesions might take longer to form. Once the tube is placed, only a third of the calculated daily energy requirements should be administered, on day two, two thirds and by the third day the full amount.

Removal of the gastrostomy tube in animals over 10kgs is to cut the catheter off flush with the skin after pulling it taught. The catheter tip is then passed in the faeces. The resulting gastrocutaneous fistula will rapidly heal if kept clean.

In all animals receiving TPN hyperglycaemia and glucosuria can develop in some cases. This hyperglycaemic state can possibly reflect the decrease in peripheral glucose uptake resulting from mild insulin resistance. As a general rule, intravenous glucose solution should be used as the sole source of nutrition for no more than 2-3 days.

Calculations of enteral feeding amounts.

1. Calculate the RER of the animal

RER = 70 x (bwt kg)$^{0.75}$ for animals <2kg or >45kg, or 30 x (bwt kg) + 70

2. Add in the illnesses factor.

RER x Illness factor = kcal/day

3. Choose the specific diet, which is most beneficial for the patient and the method of feeding.

4. Divide the energy content of the diet (kcal/ml or gram) by the energy requirement of the animal (kcal/day) to achieve the daily amount of food required.

5. Divide the total amount to be given in a day by the total amount of feeding wished to be given, or by maximum volume of each feed.

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Food and Feeding.

Labelling of Diets.

In order to correctly identify the diet of choice for an individual, evaluation of the diet needs to be made. This is achieved by analysis of the ingredients. The labelling of pet foods, whether life stage diets or prescriptions diets must contain several pieces of information.

Diets, which are fed for a clinical application, are required to show further information relating to the particular purpose it is designed for and the species being fed. The type and levels of nutrients, additives and characteristics of the diet that have been modified to suit this purpose should be stated. Also stated is the length of time in which the animal should be fed the diet, this is normally six months. It is recommended that all patients on clinical diets, on a long-term basis, have a medical examination at least every six months. For example cats on diets designed to prevent FLUTD should have a urine sample analysed every six months or for renal patients to have repeated blood biochemistry and haematology analysis.

Food Comparisons.

The quality of the food cannot be assessed from the food label, especially the true digestibility of individual nutrients and the overall digestibility of the diet. The bioavailability of nutrients is not disclosed on the label, and this does need to be conveyed to owners when they compare foods. The typical analysis states the percentage of protein, oil (fat), fibre, ash and moisture (when over 14%). In the USA a guaranteed analysis is used, where percentages again are used, but a minimum and maximal value of each nutrient is given.

The moisture content of the diet has a direct effect on the remaining ingredients. Moist diets being more dilute than dry diets. Thus direct comparison of the typical analysis is inaccurate when moisture contents differ. Comparisons should only be made when using dry matter basis (DMB) percentages, or when based on energy content (e.g. per 100kcal).

Proximal Analysis of Food.

The most common method of determining the nutrient content of the diet is through proximal analysis. This method calculates the percentage of water (moisture), protein, fat, ash and crude fibre in the diet. The percentage that is left (i.e., 100% - % moisture - % crude protein - % fat - % crude fibre - % ash) is carbohydrates or nitrogen free extract, NFE. Crude protein levels are calculated on the basis the protein contains 16 +/- 2% nitrogen. The crude protein therefore equals nitrogen x 6.25, or nitrogen divide by 0.16. Errors can occur when non-protein nitrogen such as urea or ammonia are used with the product. Figure 3.2 demonstrates proximate analysis of foods.

Types of Proprietary Diets.

Proprietary diets are those, which are commercial made, i.e. processed, and fall into two basic categories – complementary or complete.

Complete Diets.

Complete diets are those, which provide a nutritionally balanced and adequate diet when fed as the sole source of food. All of the nutrient components are provided in the correct ratio and do not require addition of any source. In fact adding in large quantities to the diet can make the overall daily intake of food nutritionally unbalanced.

Complementary Diets.

Complementary diets are those, which do not provide a nutritionally balanced diet when fed alone. These diets are designed to be fed in combination with another diet, in order to form a balanced and adequate diet. All treats and snacks are labelled as complementary foods, and thus should only make up a small portion of the daily ration.

Homemade Diets.

It is still relatively common for cats and dogs to be fed homemade diets. If prepared well, and careful consideration has been made to ensure that it is balanced homemade diets can serve some purpose in certain cases, e.g. in food trails. Many homemade diets contain excessive quantities of protein and carbohydrates, and are limited in vitamins and minerals, especially calcium. If owners do wish to pursue the use of a homemade diet, examples of a diet should be obtained from the veterinary practice. It is also advisable that the animal has its BCS, weight and clinical health examined regularly, to ensure that the diet is balanced and no deficiencies are present. Aiding the owner in designing the diet is important, and there are six simple guidelines to follow when designing a diet.[1] They are:

1.) Do the five main food groups appear in the diet?

- A multivitamin and trace mineral source.
- A source of minerals, especially calcium.
- A fat source.
- A protein source, it is vital in feline diets, that an animal sourced protein is used.
- A carbohydrate source, this also includes fibre. Sources used should be from cook cereals, grain or potato. (Potatoes are an excellent carbohydrate source when performing food trials, as it is not commonly used in commercial diets).

2.) What is the quality and source of the protein?

- The protein levels and the protein quality within the diet are two very different elements. A high protein level does not necessarily mean that the animal is receiving all of the necessary essential amino acids it requires. Skeletal muscle protein from different sources does contain similar amounts of amino acids, and there is therefore no great advantage of feeding one type of meat over another. Unless it is for the purpose of food trials. Novel protein sources used include duck, venison, salmon and egg. Egg is an excellent protein source (BV 100%), and in cases where restricted protein levels are required the inclusion of egg as a protein source into the diet, is highly recommended.

3.) What is the fat content of the protein source?

- Cuts of meat vary greatly in their content of fat. Where the fat content is high other fat levels should be reduced in order to compensate for this.

4.) What is the carbohydrate to protein ratio within the diet?

- The carbohydrate/protein ratio should be approximately 1:1 to 2:1 for cat diets and 2:1 to 3:1 in canine diets.[1]

5.) Is there a source of vitamin and minerals?

- A homemade diet will be unbalanced in view of vitamins and minerals unless there are supplemented. When using homemade diets for food trial cases the use of supplements is not recommended, as this will effect the trial. As the trial is not long term, short-term deficiencies can be tolerated.

6.) Is there a source of other essential nutrients?

- Other essential nutrients such as EFAs, taurine and other EAA do need to be supplemented into the diet. This can be achieved through the addition of oils and other supplements.

Homemade diets should be cooked, as this aids in increasing the overall digestibility of the diet, and reduces the risk of food poisoning. Overcooking will result in a loss of nutrients from the diet, especially vitamins and the denaturing of proteins. There are a vast number of different homemade diets available on the internet, and can even be designed for a specific animal and/or clinical disease.

Palatability

Palatability of any diet is essential if the animal is to eat it. Palatability of a food is its degree of acceptance to an animal. There are three essential components to palatability: the pet (species and individual), the environment (owner, home, lifestyle), and the food itself (smell, shape, texture, taste, nutritional composition). The first component, the pet, cannot be altered in order to enhance the palatability of the diet. The second component, the environment, can be changed in some ways. The habits of the owner around feeding time, the designated areas in which the animal is normally fed, the types of bowls used etc., can all be changed to benefit the animal. Guidance from the veterinary practice should be able to be sourced by the client in order to aid the animal. The third component the food itself, plays the largest role in palatability, and will be discussed further.

Food aroma and Temperature.

The temperature of the food does play a huge role in the acceptance of the diet. Cats prefer food that is near body temperature. This is a direct reflection of their natural diet in the wild. Food which is taken straight from the fridge can be less appealing, as with foods above $40^{o}C$ /$104^{o}F$.[2] The aroma of the diet plays a significant role in the animal's acceptance to consume the food. Those animals, which have a reduced olfactory capacity, such as older animals, sick animals and those on medications, which reduce olfactory senses, can have a marked decrease in the acceptation of the diet. Cleaning any mucus from the animal's nose will aid in this. The use of moist diets can be advantageous; this is due to these diets giving off stronger aromas.

Prehension.

The way in which the animal picks up the food in its mouth, and the way in which it eats it have a role in the palatability of the diet. Cats exhibit three different methods of dry food prehension. The most common method is labial prehension. In this method the cat grasps the kibble between the incisors, without the use of the tongue. The second method, supralingual prehension, involves the cat using the dorsal side of the tongue to lap-up their kibbles. The third method, sublingual prehension, occurs when the cat applies the ventral side of the tongue to the kibble, turning the kibble backwards into the mouth. Sublingual prehension is commonly used in brachycephalic breeds, such as Persians. Different kibble shapes have shown to suit certain types of prehension. For example almond shaped kibbles are best suited for cats, which use sublingual prehension.

Prehension and kibble shape and size do not seem to be linked in the dog. The kibble shape and size does affect other feeding parameters. Including, the time taken to eat the diet, and encouraging the dog to chew the kibble rather than swallowing it whole.

Taste.

Once the animal has smelt the aroma of the food, and picked it up into its mouth, taste is the third stage in food selection and factors that affect the palatability of the diet. Taste perception can be modulated by four factors:

1. Sex: It has been found that female dogs are more receptive to sweet tastes than males.
2. Age: Taste sensitivity does decline with age.
3. State of health: Some diseases do affect taste. These include chronic renal failure, diabetes mellitus, thyroid dysfunction and cranial trauma. Have a blocked nose will also affect the ability to taste.
4. Drugs: Tetracycline alters taste perception.

The taste of the diet can be directly influenced by many factors, including the ingredients used, manufacturing practices, storage, pet food preservation systems, packaging, and palatability enhancers.

Texture.

The consistency of the diet does have large effects on the quantity fed, the palatability (in some cases), the way in which the diet is stored, and packaged, and the methods in which the animal is fed. Cats and dogs prefer meat based canned products rather than dry expanded diets.[3] This has been

attributed to the higher moisture content, and due to blood and fluids containing positive palatability factors.[3]

Moist Diets.

Moist diets contain 70-85% moisture, and are extremely popular. Packaging for moist diets can range from cans to foil trays to pouches. These diets are very palatable, and can lead to over eating and obesity. The moisture content of these diets can prove to be invaluable when trying to increase water content of the animal's diet, especially in cases of FLUTD.

Semi-Moist Diets.

Semi-moist diets contain around 30% moisture. The diet pieces are formed by the ingredients being cooked, and formed into a paste. The paste is then passed through an extruder and shaped. Acid preservatives are then added in order to inhibit bacterial and fungal growth. Corn syrup is used to coat the pieces to prevent drying out, and removes the availability of the moisture for bacterial and fungal growth. The coating of the syrup makes the pieces very palatable, but totally unsuitable for diabetic patients. Semi-moist diets should not be fed to cats as they can contain propyl glycol.

Dry Diets.

Dry diets contain 10-14% moisture, and can be made by a number of different methods. Using a mixture of dry flaked and crushed cereals and vegetables create meal type diets. These diets can be beneficial when catering for large numbers of animals, as each individual animal's requirements can be met. By forming the ingredients into a paste and being cooked creates extruded diets. This process involves steam and pressure-cooking. The cooking process can improve the digestibility of the diet, as some nutrients are broken down in the process. The extruded kibbles can then be coated in flavourings and packaged once dried.

Extruded diets tend to be complete and balanced for the life stage that they are designed for. In some species extruded and pelleted diets are preferred as they prevent selective feeding, a common problem in meal type diets, especially in rabbits and small rodents. Extruded diets do increase chewing compared to pelleted diets. Thus increasing palatability, and increasing positive eating behaviour, no gulping etc. This eating behaviour also causes an increase in the production of saliva. Extruded diets are ideal for working/performance diets, for any species, as higher fat levels can be utilised.

Post Ingestion Effects.

Once consumed the gastrointestinal tract provides many signals related to food ingestion. The distension of the stomach with food stimulates stretch and chemoreceptors. These receptors send vagal signals to the satiety centre. Both positive and negative feedback mechanisms are responsible for regulating food intake. Negative feedback mechanisms such as insulin, serotonin, leptin, neurotensin, and glucagons appear to attenuate food intake. Daily administrations of leptin have shown to decrease food consumption, and thus induce weight loss.[4] Positive feedbacks include the activation of the autonomic system during eating. Opioid and dopaminergic neurons involved in the stimulation of food intake, and aid in the positive reinforcement of food intake.

Feeding Behaviours.

Finicky Feeding Behaviour.

Fastidious or finicky eating behaviours can make a transition to a new therapeutic veterinary diet almost impossible. This commonly occurring eating behaviour is a human-caused problem, resulting from the animal's conditioned expectations for frequent changes in the food variety or flavour. Owners will often describe their pets as a finicky eater if they are viewed to be intermittent or slow eaters. This can however be due to the animal being overfed, or the animals own autoregulation of food consumption. Assessment of the animal's body condition score will conclude whether or not if the animal is consuming sufficient nutrients.

Behavioural modification of the animal to counteract this feeding behaviour is difficult, as it is reliant on the owner. Often the owner is the cause of the initial problem. Removal of the excessive rotation of different brands and flavours to less frequent changes may help resolve the problem. A ritualising feeding routine will need to be implicated, set meals at a certain time, place and brand of food. Some animals which do self regulate may benefit from ad libitum feeding.

Those animals, which have been fed a high-quality, very palatable diet on an *ad libitum* basis (mainly cats), will expect this unlimited food availability. In order to change the animal's diet, it must be made to become dependent on the owner for food. In order to accomplish this the animal should be offered *ad libitum* feeding for two set hours per day. Once this routine has been established, the old diet should be restricted to 75% of the previous food intake, and the rest of the daily requirement made up with the new diet, placed in a separate bowl next to the old diet.

Food Addictions.

Food addictions are fairly common, e.g. cats becoming addicted to tinned tuna. Addictions can lead to nutritional deficiencies, or even toxic syndromes. Counter-conditioning behavioural modifications is required over a period of time. This modification can occur by adding a distasteful substance to the food, which the animal is addicted to, whilst providing a balanced complete diet of the same flavour as the addicted food.

Food Aversions.
The implications of food aversions are dramatically underestimated in veterinary practice. In some regards it is not advisable to institute dietary changes whilst clinically ill patients are hospitalised. Diets should be introduced in the home environment once the pet is more stable.

Prevention of malnutrition by ensuring adequate nutrient intake is crucial in the management of all medical and surgical cases. The veterinary nurse or technician is crucial in practical measures to improve adequate intake. These practical measures include:

History Taking.

Talking to the owner can prove to be invaluable. Obtaining information on preferred types and consistency of food, and the animal's eating habits will contribute towards getting the animal to eat. Cats can be exceptionally fastidious concerning the size, shape and type of bowl or saucer that they eat out of. Wide rimmed bowls tend to be preferred, so that they have room for their whiskers.

Removal of the clinical problem.

Many medical problems e.g. renal dysfunction can result in changes of appetite and olfaction. Pain associated with dental disease or neoplasia in the mouth will also decrease appetite. Removal of pain or the causal agent will greatly improve the well-being of the patient.

Odorous foods.

Warming of the diet to body temperature can prove to be more appetising to the animal. Increasing the temperature will also increase the odour of the food. Use of highly odorous foods such as pilchards can aid to stimulate eating. None of these measures will work however if the animal is unable to smell the food. Any nasal discharges need to be cleaned away before offering the food.

Positive reinforcements.

Positive reinforcements such as hand feeding can be used to promote eating. Improving the animal's general well-being, e.g. grooming, can also aid in this process. Trying to encourage eating outside the practice environment can prove to be useful. Taking small quantities of food when exercising the animal outside, or feeding in a room away from all other animals might encourage the animal to eat. Timid animals can benefit from having somewhere to hide away in.

Food aversions can be reinforced by repeatedly offering a diet that the animal keeps refusing. Force-feeding can be used to aid encouragement to eat, but if the animal becomes distressed this can cause a negative reinforcement to eat that particular food. Any offered food that is not consumed

should be removed from the animal's environment after 15mins. Littering the animal's cage with a carpet of different foods will not encourage eating behaviours.

Feeding Methods.

The way, in which an animal consumes its diet, does have an impact on many different elements of its life. There are three basic methods of feeding cats and dogs: free choice (*ad libitum*), time limited or food limited. All have their advantages and disadvantages, and will suit different animals on an individual basis. Significant breed differences have been noted in digs in their feeding behaviours. Beagles have similar feeding patterns to cats. Whereas Poodles eat only during the daylight hours. There are however, always individuals that are exceptions.

Free-Choice Feeding.

This method of feeding does tend to suit those animals that will eat only what is required to meet their energy requirements. Over consumption can lead to obesity, and in growing large and giant breed dogs can predispose to developmental orthopaedic disorders (DOD). Other disadvantages include food wastage, especially if feeding a moist diet, and competition from other animals in the environment can lead to over eating and consequently under eating in other animals. Advantages include a more constant blood level of nutrients and hormones, and a decrease in coprophagy. Those animals, which are timid, are more likely to eat, as there is a longer period of access to the food in which the other animal is not present to eat in front of. This method of feeding is advised with animals with or predisposed to FLUTD. This is due to the post-prandial alkaline tide; see section on FLUTD for more details.

Time-Restricted Meal Feeding.

In this method the animal is allowed free access to the food for a set period of time each day. This is usually 10-15 minutes, once or twice daily. This can be a disadvantage in small dogs, puppies and kittens, due to their limited stomach size, insufficient food is consumed in order to meet their nutritional requirements. Over consumption can easily occur when using this feeding method, if the animal is greedy. In these cases reducing the amount of time that the animal has to the diet, or feeding a diet of a lower energy density is required. Advantages include aiding in house-training in puppies. A routine of feeding a puppy and then taking it outdoors can enforce housetraining by taking advantage of the gastrocolic reflex.

Food-Restricted Meal Feeding.

This method requires either calculating the DER of the animal, and thus the quantity of diet that needs to be fed, or following the manufacturer's recommendations on the food packaging. This method of feeding is recommended for animals predisposed to DODs. The calculated DER is then divided by the energy density of the diet in order to obtain the quantity of food that should be fed. The advantage of this diet means that when using a complete balanced diet, the animal is receiving the correct amount of nutrients. The disadvantage being that all animals are individuals, and when initiating this feeding method reassessment of the quantities fed should be made. Due to some animal having a higher metabolism or work load and requiring higher amounts of energy. In these cases a performance diet could be beneficial. Likewise, some animals may gain weight using this method, and in these cases a light diet could be recommended.

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Food A: 75% moisture, 10% protein Amount of dry matter (100 - 75) = 25% dry matter Protein content = 10/25 x 100 = 40% protein on DBM
Food B: 20% moisture, 10% protein Amount of dry matter (100 – 20) = 80% dry matter Protein content = 10/80 x 100 = 12.5% protein on DMB
Thus Food A contains over three times as much protein as Food B on a DMB

Table 3.1: Comparison between dry and moist diets when calculated on a DMB. (DMB = Dry Matter Basis).

Cancer.

Nutrition can play an important role in the 'prevention' and treatment of cancers. This does however vary with the type of tumour and stage of progression. The potential risk reduction of cancer when following dietary recommendations has not been well documented in veterinary medicine. In human studies "eating right", staying active and maintaining a healthy weight can reduce the risk of cancer by 30-40%.[1] It has been speculated that this reduction may be even more achievable in pets as their dietary intake can be more controlled. Correct feeding of the cancer patient can enhance quality and length of life. This is due to nutritional support reducing or preventing toxicoses associated with cancer therapy and ameliorate cancer induced metabolic alterations. Anorexia is a common clinical symptom in cancer patients, which can lead to weight loss and cachexia in conjunction with the metabolic alterations. Use of body condition scores can be an excellent monitoring tool to assess the overall nutritional effect of cancer and treatments. Cancer affects the nutritional status of the animal in three ways:

1.) Therapy (e.g. chemotherapy induced anorexia, nausea or vomiting.) Human chemotherapy patients have reported changes in taste and smell. These side effects experienced during therapy make it difficult for some patients to consume optimal quantity of calories and nutrients.
2.) Alterations of the metabolic pathways.
3.) Tumours can also have a primary effect such as compression or infiltration of the alimentary canal.

Cancer Cachexia.

Cachexia is defined as a profound and marked state of general ill health and malnutrition. It is a complex paraneoplastic syndrome that includes progressive weight loss that occurs despite adequate nutritional intake.[1] Loss of both fat and muscle can occur with the depletion of muscle mass often exceeding that of viscera in cancer patients. Cachexia is not common in veterinary cancer patients, but weight loss usually occurs early in the course of the disease. Cachexia can cause a decreased quality of life, a decreased response to therapies and shortened survival time, when compared to those with similar diseases without cachexia. Cancer cachexia may be partly due to a negative energy balance. It is therefore important for the owner to have the animal weighed and body condition scored on a regular basis.

The aim of nutritional management is to:

- Provide an energy source for the patient that the tumour cannot readily utilise.
- Meet the increased nutritional requirements of proteins.
- Limit lactate production by the tumour.
- Improve immune function, remission and survival times.
- Aid in limiting tumour growth.

Clinical Nutrition.

Carbohydrates.

The metabolism of carbohydrates is greatly altered in dogs with cancer. The alteration in metabolism is due to tumours metabolising glucose via anaerobic glycolysis to lactic acid. A net energy gain by the tumour, and net loss by the host results as the host expends energy converting the lactate back to glucose by the Cori cycle. The alterations in metabolism are suspected, but not yet proven in cats. Hyperlactatemia can result and this should be taken into consideration if intravenous fluid therapy is required. Hartmanns (Lactated Ringer's) solution can exacerbate any hyperlactatemia and energy demands.

Carbohydrates can be poorly utilised due to peripheral insulin resistance, excess dietary soluble carbohydrates needs to be avoided. NFE needs to be $\subseteq$25% of dry matter or $\subseteq$20% of metabolised energy. Providing metabolisable energy from fat is the most beneficial for the animal. Increased dietary fibre may aid in preventing abnormal stool quality (e.g. soft stools, or diarrhoea), which is commonly encountered when changing from a high carbohydrate commercial dry food to a high fat commercial or homemade diet. Recommendations of crude fibre levels greater than 2.5% DMB are documented.[1]

Protein.

Tumours use amino acids as a preferred source of energy. This results in an altered metabolism, with the patient having to increased skeletal protein breakdown, liver protein synthesis and whole body synthesis for tumour growth. This can become clinically significant when protein degradation exceeds intake. The immune response, wound healing rates and gastrointestinal functioning are also affected by the imbalances in protein levels. Specific amino acids have shown to have different effects in cancer patients.

Arginine and glutamine are commonly supplemented into diets specifically designed for cancer patients. The addition of arginine into TPN solutions can decrease tumour growth and metabolic rate in rodent models. In canine models quality of life and survival time have been increased when fed arginine in conjunction with n-3 fatty acids. Glutamine is also added to most human and veterinary enteral formulas. Glutamine has shown to be beneficial in improving intestinal morphometry, reducing bacterial translocation, enhancing local immunity and improving survival times. Glutamine is a conditionally essential amino acid. Supplementation is recommended when protein metabolism is altered, as in cancer patients.

The amino acids methionine and asparagines are both used in tumour cell growth. Methionine's precursor, homocysteine, when supplemented to the diet can stop tumour cell growth progression into different growth phases. This enables cell-cycle specific therapeutic agents to target more tumour

cells. Use of L-asparaginase in dogs and cats with lymphoma has high remission rates of 80%, due to asparagines being an essential factor in cell growth. The supplementation of glycine can also have important implications depending on the type of chemotherapy being utilised. Glycine will reduce cisplatin-induced nephrotoxicity.

Fats.

A larger proportion of the metablisable energy should be from dietary fat and oils. This is due to tumour cells having difficultly utilising fats and oils. If cachexia is present an increase in dietary fat can improve the body condition scores. 25-40% on a dry matter basis is an ideal dietary fat content, or 50-65% of the calories as an 'as fed basis'. Lipid metabolism also alters in cancer patients. Increased blood concentration of free fatty acids, very low density lipoproteins, triglycerides, plasma lipoproteins and hormone dependant lipoproteins lipase activity can occur due to an overall decrease in lipogenesis and increased lipolysis. These changes in lipid metabolism can also cause immunosuppression, which relates with a decrease in survival time.

Omega-3 (n-3) fatty acids, like EPA and DHA, generally have an inhibiting effect on tumour growth. EPA can decrease protein degradation without altering protein synthesis, thus aiding in the symptoms associated with cachexia. The ratio of n-3 to n-6 fatty acids is important, as omega-6 (n-6) fatty acids such as linoleic and γ-linolenic acid enhance metastases.

Vitamins and Minerals.

The levels of vitamins and minerals within the diet need to be sufficient o meet daily nutrient intakes. Higher levels of the antioxidants vitamins and minerals should be advocated in order to counteract damage caused by free radical action, and improve immune response. There are two hypotheses surrounding the use of antioxidants in cancer patients. The first supports the role of antioxidants within the diet. As they may improve the efficacy of cancer therapy by improving immune function, increasing tumour response to radiation or chemotherapy, decreasing toxicity to normal cells, and help to reverse metabolic changes contributing towards cachexia.[2] The second hypothesis states that antioxidants may have a deleterious effect by protecting cancer calls against damage by chemotherapy or radiation therapy.[2]

Serum levels of the trace minerals zinc, chromium and iron are lower in dogs with lymphoma and osteosarcoma than in healthy dogs.[3] Diets that are complete and balanced should not need supplementation of these trace minerals. Although in homemade diets it should be considered.

Feeding a Cancer Diet.

Initial assessment of the patient needs to be performed. Regular reassessments and calculations of DER will be required, especially if weight loss or cachexia starts to occur. If hospitalised, patients need to ingest at least their RER. At home, the calculated DER (RER x appropriate factor) is required. An initial illness factor of 1.25 in early stages of cancer, increasing to 1.75 in later stages has been recommended.

Due to the discussed altered metabolism processes, modifying nutrient intake is required. Several veterinary therapeutic diets are available, and with an increased fat content are very palatable. A suitable period of transition onto the new diet is required. Some animals, however, can suffer diarrhoea due to the increase in fat content, and may need a longer period of transition.

With all cancer patients care should be taken with monitoring weight loss, especially with those that are initially overweight. Rapid weight loss, due to anorexia and alterations in metabolism, can result in a loss of lean muscle mass. Fat stores may still remain and may mask the detrimental effect of protein catabolism. Careful muscle condition scores and body condition scores will need to be performed, as 'overcoat syndrome' can quiet easily be present.

The use of appetite stimulants can be useful in some cancer patients. If appetite stimulants fail and long-term nutritional support is required, enteral feeding techniques do need to be considered. Active encouragement to eat still needs to be initiated by the owner, or practice staff if hospitalised.

Support for the owner is greatly required with cancer patients. The word cancer will make any owner anxious, especially if the animal is anorexic, which can be very distressing. Many animals will be susceptible to this in their owners and can be another factor associated with anorexia.

Key Points.
Aim to prevent cancer cachexia.
If the animal is unwilling to consume the cancer diet, inadequate calorific intake can result. It is best to feed anything, to ensure correct calorific intake.
Quality of life for the animal is the most important factor.

Case Study.
Case Study Number 1.

A three-year-old Rottweiler was presented for examination by the owner who was concerned that the dog was suffering from a sudden onset lameness of its left forelimb. On radiography a tumour (osteosarcoma) was discovered in the distal left radius. The dog was obese, and weighed 60kgs (ideal weight 50kgs). A weight reduction programme (with prescription diets) with this dog at this stage is not currently advisable, as the leg was to be amputated and chemotherapy to be initiated. The dog was instead transferred to a light maintenance diet, and weight was lost. The dog was transferred to a prescription diet as target weight was reached. The diet did however induce diarrhoea in the dog. This can sometimes occur in animals that are used to consuming a diet, which has higher fibre content. Close monitoring of weight was required in this case due to the higher at content in the new diet.

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Cardiac Disease.

The nutritional status of the cardiac patient is exceptionally important to ascertain as this can have several effects on the animal. This can include the choice and dose rate of the drugs used in the medical treatment, interpretation of any laboratory results, interpretation of ECG data, prognosis of both surgical and medical intervention and the choice of diet for the patient. As part of the initial clinical assessment a full history of the animal's diet should be taken. Nutrition can be a causative factor in cardiac disease. If the animal receives unusual supplements, is not fed a complete diet, if more than one animal in the household is affected or if it is being fed a homemade or fad diet, cardiac disease can be induced. Micro and macro nutrient deficiencies (calcium, potassium) can cause cardiac problems and thus a complete blood work up needs to be performed.

Assessment of body condition and muscle scores of each individual animal is important with cardiac patients. A reduction in skeletal muscle mass might indicate energy malnutrition, and possibly a negative nitrogen balance. Animals with catabolic disease such as hyperthyroidism in cats, and cardiac failure lose body mass very rapidly, as with anorexic cats. The progression of cardiac disease can be exacerbated in obese animals. This can result in cardiacmegaly, circulatory congestion, oedema, ascites and hypocalcaemia. An overweight animal must be subjectively assessed; obesity must be differentiated from abdominal distension due to hepatomegaly or ascites. Obesity can also mask an underlying lean muscle body mass. Radiography can be useful in determining lack of lean body mass, this is especially notable on the proximal limbs. Obese animals need to lose weight in a controlled monitored way, the same as in a normal healthy animal. Obesity not only produces clinical

signs that mimic those of early heart failure, but can also cause cardiovascular changes that can exacerbate any underlying cardiovascular disease.

The aims of clinical nutrition are to:

- Help control signs associated with sodium and fluid retention, by avoiding nutritional deficiencies and excesses.
- Aid in maintaining normal heart muscle function.
- Slow the progression of concurrent renal disease.
- Support patients receiving diuretics or ACE inhibitors.
- Maintain optimal weight, aid in preventing cardiac cachexia.

Cardiac Cachexia.

34-75% of dogs suffering with heart disease suffer from anorexia, and is one of the multifactorial processes associated with the loss of lean body mass in cardiac cachexia.[1] Other factors include increased energy requirements and metabolic alterations. Cardiac cachexia is more commonly seen in dogs than cats, and in DCM or right-sided heart failure. The primary energy source for animals with acute or chronic disease is amino acids from muscle, thus causing a reduction in lean body mass. Cachexia is a slow progressive process of the loss of lean body mass/muscle. Careful examinations of obese animals should occur, as this lean body mass reduction can occur, creating overcoat syndrome, and be easily missed. Any clinical nutrition of these animals includes management of any anorexia is present.

Clinical Nutrition.

<u>L-Carnitine.</u>

L-carnitine is critical for fatty acid metabolism and energy production, and cardiac myocytes depend upon the oxidation of fatty acids for their energy. L-carnitine deficiencies and a causative link with DCM have not been established. L-Carnitine deficiencies within the cardiac myocytes occur in 50% of dogs suffering with DCM. Some affected dogs with DCM do respond to L-carnitine supplementation. Carnitine supplementation of 50-100mg/kg bwt orally every 8 hours has been recommended to dogs with DCM,[1] though most cardiac diets are already supplemented with L-carnitine.

<u>Fats.</u>

The role of fatty acids in the diet of cardiac patients has been widely reported. Supplementation of the diet with EPA and DHA has shown to improve cachexia scores, but had no effect on survival time, thus improving quality of life. Animals suffering from cardiac cachexia do have an increased production of inflammatory cytokines. These cytokines are directly linked to causing anorexia, to increase energy requirements and to increase catabolism of lean body mass.[1] Supplementation of the diet with omega-3 fatty acids (especially EPA and DHA) does decrease the production and effects of cytokines.

Recommended dosage of 40mg/kg bwt of EPA and 25mg/kg bwt of DHA for both dogs and cats with anorexia or cachexia have been noted.[1] With most fish oil supplements containing 180mg EPA and 120mg of DHA per capsule, dosages can be easily calculated.[1]

<u>Carbohydrates.</u>

The level of carbohydrates in the diet has to support adequate calories for the specific lifestage. In the majority of cases this is for senior animals. The calorific value of the diet does therefore; tend to be of a lower level. It should also be remembered that these animals do tend to have a more sedentary lifestyle do to the cardiac disease, and the risk for weight gain, or already being obese is greater.

The recommended fibre levels in the diet, is very much dependant on the individual. If the animal is obese it is initially recommended that the animal reduces in weigh, and reaches it's optimal body weight and BCS. This may involve the use of a high fibre diet. In animals, which are already at an ideal BCS and optimal weight, fibre should be present but not excessive. The presence of fibre in the

diet does reduce the bioavailability of many of the nutrients. As concurrent disease or impairment of the gastrointestinal tract, pancreas and liver an occur, a high fibre diet is not ideal.[4]

Vitamins and Minerals.

The nutrients of main concern in patients with heart failure include sodium, potassium, and magnesium. Treats and snacks, which are often fed to dogs, do have a high salt content, as does cheese and processed meats, which are often used to administer medications. The restriction of sodium in the diet is useful in the mechanism to reduce fluid retention that accompanies many forms of heart disease. Both sodium and water can be retained when the rennin-angiotensin-aldosterone (RAA) cascade is stimulated, or when the patient's blood pressure falls.[3] One of the current methods of treatment for fluid retention is the use of diuretics. The diuretics block sodium retention but also promote urinary loss of magnesium and potassium. The prolonged use of diuretics can also lead to deficiencies of the water-soluble vitamins.

As a result of the cascade being stimulated extracellular and vascular fluid volume increase, thus increasing preload. The plasma protein concentration therefore becomes more dilute which in turn decreases plasma oncotic pressure. Water moves from the vascular to the interstitial compartment resulting in oedema, ascites and congestion.

The use of angiotensin-converting enzyme (ACE) – inhibitors have led to modification of the recommendation for sodium restriction, from senior diets and early cardiac disease diets. ACE inhibitors are designed to block the production of angiotensin II and its subsequent stimulation of the secretion of aldosterone. Both of these chemicals promote retention of water and sodium by the kidney. Thus ACE inhibitors result in impaired sodium and water excretion. Sodium intake needs to be limited in proportion to the severity of the disease (See Table 6.2), in an attempt to avoid excesses. ACE inhibitors can also cause the retention of potassium, thus periodic serum levels should be monitored.[2] Spironolactone has similar potassium sparing effects, along with being an aldosterone antagonist. CRF is often a concoitant disease of patients with cardiovascular disorders. Diets for these animals also need restricted phosphorous levels. Levels of potassium and magnesium should also optimally controlled as it supports the patient receiving diuretics and/or ACE inhibitor therapy.

Magnesium levels can have a deleterious effect on a range of cardiovascular conditions including hypertension, congestive heart failure, coronary artery disease and cardiac arrhythmias.[2] Hypomagnesaemia can be induced through the use of digoxin and loop diuretics.

The B vitamin complexes are often supplemented into cardiac diets. At present there has been little investigation into the role of vitamin B deficiency as a cause of heart disease in dogs and cats. Polyuria and anorexia can both contribute to low vitamin B concentrations, and thus higher B vitamin requirements are needed.

Recommendations of higher levels of antioxidants in animals suffering from CHF are commonplace, due to the by-products ROS. Coenzyme Q_{10}, another antioxidant, has also been anecdotally recommended. Coenzyme Q_{10}, like carnitine, is a cofactor in a number of energy producing reactions. Benefits of supplementation include improved myocardial metabolic efficiency and increases in antioxidant production.[1] Although no direct evidence has been achieved in establishing Coenzyme Q_{10}'s direct benefits recommended dosages of 30mg orally bid, and in large breeds dogs up to 90mg bid PO have been rported.[1]

Proteins.

Taurine is an essential amino acid in cats, due to taurine being the only amino acid able to combine with cholesterol during bile salt synthesis. Whereas in other species another amino acid, glycine can be substituted. Cats do have a limited ability to synthesis taurine from cysteine and methionine, but its use out strips production. The mechanism of heart failure in cats and dogs with taurine deficiency is poorly understood. Prior to 1987 supplementation of commercial foods with taurine was not commonplace and the number of cases of feline DCM was large. Clinical studies have also shown that inadequate potassium intake may be sufficient to induce a significant taurine depletion and cardiovascular disease in cats. Most dogs presenting with DCM do not tend to have a concurrent taurine deficiency. Although in some breeds where DCM is not a common disease, a taurine

deficiency has been noted. Dog breeds reported to be associated with taurine deficiency include, the American cocker spaniel, golden retriever, Labrador, Newfoundland, Dalmatian and English bulldog.[1]

The overall protein level in the diet is reduced. This is due to the progression of renal disease, which is associated with cardiac disease. The protein levels are therefore restricted but of a high biological value, typical values of 17% (DMB) for dogs and 29% (DMB) for cats are often recommended.

It should be noted that is protein restriction is too restricted this can be detrimental to the animal. Protein malnutrition can rapidly occur in the presence of catabolic disease and inadequate food intake. Hypoproteinaemia can be present in cardiac disease with reduced liver function. In animals with cardiac cachexia, protein levels should be increased. Supplementing a cardiac diet with scramble eggs, or cottage cheese is an excellent method of increasing the protein levels without increasing the salt content of the diet.

Feeding the Cardiac Patient.

Each diet must be based around each individual's specific requirements. Overweight or obese animal will require diets aimed at calorie-restricted diets. Underweight patients a calorie dense diet. Concurrent disease also needs to be taken into consideration. Laboratory parameters should be obtained to identify any electrolyte excesses or deficiencies. Anorexia is a common side effect of CHF, and great effort on behalf of the owner may be required in order to entice the animal to eat.

Feeding a Low Salt Diet.

Low salt diets are used for both cardiac and renal patients. It is often a misconception that low salt diets have a reduced palatability. Studies have demonstrated that, diets with reduced sodium chloride levels used in patients with cardiovascular disease have comparable or better palatability than supermarket brands, (See Table 6.3).[5] Difficulties in transition from the patient's original diet to a low salt prescription diet can be attributed to:

1.) Advanced illness associated with renal and heart failure.
2.) Established eating and feeding habits of both the animal and the owner.
3.) Anorexia associated with disease process and/or medications.
4.) Too quick a transition and/or established food aversions.

Transition to a low salt diet is always easier if the animal has already been fed a diet in a lower salt category. Animals are often encouraged to consume a new diet by adding flavour enhancers. Low sodium additives can include low-sodium soups and sweeteners, such as honey or syrups. Table 6.4 demonstrates how adding certain foodstuffs to the diet can significantly increase the sodium intake. As with all clinical prescription diets the therapeutic agent of the diet should not be imposed if it is detrimental of the overall nutrient intake. Owners should be aware that both trial and error might be required in these patients. Some animals can "go off" certain diets and a cyclical approach with two or three commercial diets may be required. Use of different feeding methods along with different foods should be utilised. The success of a transition of a new diet does hugely depend on the dedication of the owner and the support received by the veterinary practice. Supporting the owner can be achieved by providing a list of foodstuffs and their sodium content. If the animal is overweight or underweight then a weight management programme must be initiated.

Administering medications can be difficult, and many of the foods used to "hide" medications can be high in salt. Advising owners on different methods can aid in an increase in compliance. Alternatives can include:

- Teaching the owner to administer medications without the use of foods. This can include the use of pill givers.
- The use of commercial treats designed to hold medications. Always double-check the salt content.

- Use of appropriate foods such as bananas (good potassium source), no-added salt peanut butter (no ideal in obese animals), and home-cooked meats (without added salt), not sandwich/processed meats.

Key Points.
Ensure that the daily calorie and nutrient intake is met.

The optimal BCS needs to be achieved in both obese and underweight animals.

Restrict sodium intake.

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- Increased perfusion requirements of expanding adipose tissue.
- Elevated cardiac output.
- Abnormal left ventricular function.
- Variable blood pressure response (normotensive to hypertensive).
- Increased retention of sodium and water by the kidney.
- Increased plasma aldosterone and norepinephine concentration.
- Increase left arterial pressure.
- Increased heart rate.
- Exercise intolerance.

Table 6.1: Cardiovascular adaptations that occur during the transition from ideal body score to obese.

Class	Description[a]	Recommended upper limits of sodium Intake (mg/kg bwt/day).
I	Normal. Physical activity. Symptoms not induced under normal exercise levels.	Unrestricted.
II	Slightly limited physical capacity: original physical activity leads to clinical signs.	6.8
III	Markedly limited physical capacity: limited physical activity leads to clinical signs.	4.5
IV	Unable to carry on any activity without signs: clinical signs present at rest.	2.8

Table 6.2: Functional classification of heart failure and corresponding sodium intake. [a] clinical signs include weight loss, exercise intolerance, coughing, respiratory distress and occasionally ascites.

Food A	Food B	Preferring Food A (%)	Preferring Food B (%)	Number of Animals
Canine h/d[a], dry	Purina Dog Chow	95	5	60
Canine h/d, dry	Iams Chunks	60	40	60
Canine h/d, dry	Pedigree Chum original	95	5	60
Canine h/d, canned	Ken-L-Ration Original	100	0	60

Table 6.3: Comparison of feeding restricted sodium diets vs. commercial with a high comparison sodium level. [a]Hill's Prescription Diet Canine h/d.

Food	Sodium intake (mg/day)
DOG	
Grocery moist diet	2,845
Grocery dry diet	1,144
Senior dry diet	390
Renal moist diet	400
Cardiac dry diet	111
30g cheese	262
1 slice of bread	218
CAT	
Grocery moist diet	952
Grocery dry food	371
Senior dry diet	186
Renal/cardiac moist diet	175
Renal/cardiac dry diet	156
½ tin tuna	160

Table 6.4: Daily sodium intake for a dog and cat eating various foods. Based on a 15kg dog consuming 935 kcal/day, and a 4kg cat consuming 270 kcal/day.

Critical Care Nutrition.

With all sick companion animals the nutritional goal is for the patient to eat the designated diet in its own environment. Unfortunately in critically sick animals they will be in a hospital environment, where the added stress of this different environment can effect food consumption. Most animals will require, or benefit from a veterinary therapeutic diet, but the initial goal is to ensure that the patient is receiving its daily calorific requirement. Analgesia should not be forgotten, as pain can reduce food intake in some animals. Hydration status must be maintained and corrected before any nutritional support can be initiated. The aim of critical care nutrition is dependent on the disease process and/or the individual's specific requirements. Each case must be considered on its own specific requirements once a full clinical examination and history has been achieved. This includes nutritional assessment and dietary history. The sole aim can be defined as to prevent and/or treat malnutrition when present. In order to define any nutritional aims in more depth, it is more beneficial to split the aims in to short-term and long-term goals. The short-term aims are to:

- Provide for any ongoing nutritional requirements (both in terms of energy and nutrients).
- Prevent or correct any nutritional deficiencies or imbalances.
- Minimise metabolic derangements.
- Prevention of further catabolism of lean body mass.

Long-term nutritional aims should include:

- Restoration of optimal body condition.
- Provide required nutrients to the animal within its own environment.

As disease processes change, and the animal's physiological and metabolic responses alter the nature of the nutritional support, and both the short-term and long-term nutritional aims may alter.

When assessing animals for the preferred method of critical care feeding the nutritional status of the animal needs to be evaluated. This should include BCS, MCS, hydration status, weight, hair coat quality, signs of inadequate wound healing, hypoalbuminemia, lymphopenia and coagulopathy.[1] Though should be given to "fluid shifts" in these animals as they can severely affect haematological values and the animal's weight. Factors that should be identified include specific electrolyte imbalances, hyperglycaemia, hypertriglyceridemia or hyperammoneia, as they will have large consequences on the nutritional critical care plan. Adequate adjustments will be required to the feeding plan and possibly the formulation of any parenteral nutrition to be utilised.

The calorific intake required by the patient depends on several factors:

- The rate of energy use for basal metabolism (resting).
- Nutrient assimilation
- Body temperature maintenance
- Activity levels.

Energy requirements during sickness are based on RER values. This is due to the assumption that the patient is inactive and is often confined to a small area. Due to this calculated energy requirements, using an illness factor, are only a guideline, (Table 6.5), many nutritionists no longer use illness factors. Daily weighing of the patient, accessing healing rates and accessing lean body mass is a good indicator that the patient is receiving sufficient calories is now the method of choice for deciding on calorific requirement. In herbivores (horses and rabbits) vital parameters should also include gastrointestinal mobility and faecal volume, appearance and frequency should be monitored. In the early phases of supportative feeding digital pulses in equines should be monitored. This is due to the potential to induce carbohydrate-induced laminitis.

As with all hospitalised patients, human and animal, malnutrition has been associated with increased in infectious morbidity, prolonged hospital stay and an increase in mortality. The recommendations of nutritional management of sick horses have been constructed from extrapolated data from other species. There have been no controlled studies on the relationship between nutritional support and clinically important endpoints, such as surgical complication rates, duration of hospitalised period and mortality rates in this species.

The volume of liquid diets administered at each bolus feeding in dogs and cats should not exceed 50mls/kg. This is only an estimate and each animal should be judged on an individual basis. Tolerance to liquid diets is best when small feeds are delivered frequently. In horses a reasonable target is to feed 2-4l every 2-4 hours. Once these volumes are well tolerated, the volume of food each meal can be increased with the frequency decreased. A 500kg horse has an average stomach capacity of 7-9l, but no single feed should exceed 6l.[2]

Starvation and Anorexia.

Starvation can leave the animal severely emaciated, but also the gastrointestinal tract will become atrophied, due to the inadequate nutrient supply. Intestinal villi become atrophied and the epithelial layers become thin and fragile. Bacterial translocation will often occur in these cases. The gastrointestinal capacity to digest and absorb nutrients will become severely limited. The loss of lean body mass occurs from skeletal muscle and internal tissues. Nutritional support of these animals is vital and required immediately. Initially hydration, electrolytes and acid-base status of the patient needs to be rectified. The diet chosen needs to be of a high digestibility, and primarily consisting of proteins and fats. This is due to the patient utilising these nutrients over carbohydrates. The patient will be suffering from protein energy malnutrition (PEM) and the quality of the protein feed is

important. On initiating nutritional support to the patient small frequent meals are required, slowly building up over a period of time to the full daily nutrient and energy requirements. PEM has a potential to occur during times of illness and when increased demands of protein and energy are required.[3]

Transient diarrhoea is a common side effect in these cases due to maldigestion, and should resolve as the patient recovers. Inclusion of dietary fibre to the diet should be avoided as it will reduce the digestibility of the diet and bind nutrients up that are required.

Clinical Nutrition.

Water (Hydration Levels).

The initial stage of nutritional support is correcting any dehydration, electrolyte replacement and normalisation of the acid-base status before starting assisted feeding. Initiating assisted feeding before the patient is haemodynamically stable can further compromise the patient. If oedema occurs or dehydration persists recalculation of flow rates is required. Monitoring of hydration level indicators is required during intravenous fluid therapy, (Table 2.1 and 2.2 on page 000). Daily maintenance fluid requirements are approximately 50-60mls/kg bwt/day, or 2ml/kg bwt/hr. If persistent vomiting or diarrhoea is present these additional losses need to be factored in.

Where dogs and cats are able to consume fluids without vomiting, the use of an oral rehydration fluid should be advocated. Unless contraindicated placement of a bowl or bucket of water in the animal's kennel or stable should occur. If required additional fluids can always be administered intravenously, or via feeding tubes.

Protein.

Protein energy malnutrition (PEM) can occur during periods of illness, injury or even after routine operations. During these periods the body shuts down systems in order to conserve the limited resources. Physiological changes such as a drop in blood pressure, reduction in cardiac output and a drop in oxygen consumption occur. This is clinical shock and is known as the Ebb phase. Clinical shock is treatable but the length of time that shock is suffered from is variable, and can ultimately be lethal. Following the Ebb phase is the Flow or Hypermetabolic phase. During this second phase the patient's body defence and repair mechanisms are initiated. This process is where anorexia and starvation differ.[4]

Nutritional support with adequate protein levels is vital, patients in a catabolic state will utilise the skeletal muscle proteins. Sufficient calories need to be supplied to the patient from fats and carbohydrates. This will ensure that proteins are not used as a source of energy. The quality of the proteins provided is of importance as is the digestibility. Specific amino acids are supplemented to critical care diets. Glutamine is an important substrate for the increased levels of gluconeogenesis, used in rapidly dividing cells. Any deficiency in this amino acid has shown to lead to gut mucosal atrophy, and an increase in bacterial translocation, due to a compromise of the mucosal barrier. This can lead to the suggestion that glutamine may behave as a 'conditionally essential' amino acid during severe illness. The essential amino acid arginine has a positive effect on the immune system and can subsequently improve survival times of septic patients. Many enteral diets are supplemented with both glutamine and arginine. High dose of glutamine have a trophic effect on the gut mucosa.

The use of a novel protein source in these cases has been advocated. Due to atrophy of the gastrointestinal tract, protein antigens can cross through to the bloodstream and set up hypersensitivity processes.

Vitamins and Minerals.

The supplementation of vitamins and minerals for hospitalised patients will depend on the disease and the severity of that disease that is present. Sodium, chloride, potassium, phosphate, calcium and magnesium should be used for short-term nutritional support. All animals that receive intravenous fluid therapy with or without parenteral support should have daily electrolyte levels monitored. If any polydipsia or polyuria is present, supplementation of the water-soluble vitamins is required. Zinc does aid in promoting wound healing and plays a role in protein and nucleic acid metabolism. Supplementation of nutritional support diets with zinc has been recommended.

Carbohydrates.

Carbohydrates within any critical care diet, needs to be of a very high digestibility. The quantities of fibre need to be kept to a minimum, as this will decrease digestibility and bind up important nutrients. The level of carbohydrate in the diet needs to be adequate enough to supply the required calories for recovery.

Fats.

The quantity of fat in a critical care diet does need to be increased. A higher level of calories can be obtained from fat rather then via carbohydrates. The inclusion of omega –3 fatty acids can help decrease any inflammatory response.

Rabbit Critical Care Nutrition.

Rabbits need prompt treatment when anorexia presents, this is especially important in obese rabbits and pregnant or lactating does. This is due to the greater risk of developing hepatic lipidosis, and/or other clinical disorders.[5] Table 6.6 sets out to describe some of the more common clinical signs associated with anorexia, and possible causes.

Syringe feeding must be initiated in rabbits that have not eaten for more than 24 hours. The use of nasogastric tube is rarely utilised, but is ideal. Rabbits do get stressed in unfamiliar environments; illness, anorexia and syringe feeding exacerbate this. The use off nasogastric tube should be used more as it reduced stress levels, whilst maintaining GIT health and providing the required nutrients for the rabbit. Several preparations are available on the market for critical care nutrition for rabbits, and it is important that they contain both soluble and insoluble fibre sources. Fresh grass and other foods should be made available to the rabbit at all times, even when not eating. The use of alfalfa to rabbits recovering from major surgery or severe illness is recommended. Alfalfa tends to be very appealing to the rabbit's taste bud and will promote weight gain.

Analgesia should be routinely used, pain will prevent the rabbit from eating and as a consequence reduced nutrient intake can occur. GIT motility stimulants such as cisapride (0.5mg/kg bwt) and metoclopramide (0.5mg/kg bwt) can be used in order to aid in the prevention of GI hypomobility in high-risk stimulations (e.g. after surgery).[5]

Supportative Feeding Methods.
Supportative feeding methods should be considered when there is a history of greater than 10% weight loss, decreased food intake, anorexia, increased nutrient demands due to trauma or surgery, increased nutrient losses resulting from vomiting, diarrhoea, burns or scolds, and acute exacerbation of chronic disease. Also, if specific areas of the alimentary canal need to be by-passed. The ideal diet to use in these cases should be highly palatable, digestible and have a high energy density. A relatively high percentage of energy should be achieved as protein and fat rather than carbohydrates. Table 6.8 gives suggestive levels of critical care diets of nutrients, in cats and dogs. Critical care diets for cats and dogs are available in three different forms; powdered, liquid and moist diets. Moist diets can have thixotrophic consistencies, when mixed their viscosity decreases becoming thinner.

The use of assisted feeding methods does have great advantages, but care of the feeding tube is vital. The artificial opening through the abdomen into the gastrointestinal tract through which the tube is inserted is referred to as a stoma. The stoma must be treated as a surgical wound, and cleaned daily with normal saline, or cooled boiled water for the first seven to 14 days, or until it is healed. Dressings around tubes are not always necessary, unless indicated, e.g. if the stoma site is infected, or the animal is interfering with the insertion site. Table 6.9 lists some examples problems that can arise with tube feeding. Deciding on which methods of tube feeding to be utilised does depend on a number of different factors. Figure 6.5 shows a simple flow chart with deciding factors.[7]

Encouraging Animals to Eat.

The process of encouraging animals to eat should never be forgotten. Voluntary intake can be established in a number of cases by taking time out to personally encourage the animal to eat. Grooming can actively encourage the animals (especially dogs and cats) to eat, removing an nasal discharge that is blocking their sense of small, as can TLC, providing competition, hand feeding, providing a selection of different diets, taking the animal into a different environment and in some

cases offering the animal some of the food that you are eating if it is suitable. This last method works especially well in dogs.

Syringe Feeding.

Many patients will tolerate syringe feeding well, as long as stress is limited during the process. Aversions can be initiated if the animal resents the process, and the food is forcibly fed. 50ml catheter-tipped syringes or Pasteur pipettes (cut down) are exceptionally useful in administering critical care diets.

Naso-oesphageal Tubes.

Naso-oesphageal tubes (Figure 6.6) are generally well tolerated by cats and dogs and are suitable for short term nutritional support, usually 3-7days, though longer periods have been documented. Contraindications for the use of these tubes include unconsciousness, vomiting, disease or dysfunction of the pharynx, larynx, and nares, swallowing reflex, oesophagus and stomach. The preferred placement of the naso-oesphageal tube is in the caudal oesophagus, rather than the stomach as it reduces the risk of reflux oesphagitis.

Once the tube has been placed introduction of a small amount of water injected slowly into the tube to see if a cough reflex is induced, indicating aspiration. Lateral radiographs can also identify that the tube has been correctly placed. Due to the narrow bore of the tube blockages do occur. Injection of 5-10mls of water into the tube post introduction of the liquid diet, should aid in prevention of any blockages. If blockages do occur small amounts of carbonates drinks, cranberry juice or solutions of pancreatic enzymes have shown to aid in their removal. Pre feeding administration of water is required to ensure that the tube is still positioned correctly. In human medicine preparations of “Clog Zapper” are commonly used, and their use can be utilised in veterinary situations.[8]

Pharyngostomy Tubes.

Pharyngeostomy tubes (Figure 6.7) are commonly used in cats, which have suffered facial trauma, usually after a road traffic accident. Pharyngostomy tubes are of use when bypassing of the nose and mouth is required in order to administer nutritional support. Animals that don’t tolerate naso-oesphageal tubes well can have pharyngostomy tubes placed. These types of tubes have been largely replaced by oesphgastomy tubes or gastrostomy tubes that are placed percutaneously.[9] Aseptic placement of the tube under general anaesthetic is required. Frequent cleaning and inspection of the tube is necessary under aseptic conditions. Complications can include airway obstruction, damage to the cervical nerves and blood vessels and infections.

Percutaneous Endoscopic Gastrostomy (PEG) Tubes.

Placement of these tubes (Figure 6.8) is required under general anaesthetic, and needs to be in place for at least five days prior to removal. PEG tubes are utilised when long-term nutritional support is required, and when oesophageal problems are present. In many cases of gastric dilation/volvulus (GDV), PEG tubes are placed at time of gastroplexy.[10] Adhesions between the gastric serosa and the peritoneum can form within 48-72 hours.[9] It should be noted that in malnourished patients these adhesions might take longer to form. Once the tube is placed, only a third of the calculated daily energy requirements should be administered, on day two, two thirds and by the third day the full amount. Feeding through the PEG tube can commence four hours after its insertion. If patients are unable to take fluids orally, mouth care should be encouraged at least every four hours. This involves ensuring that the mucous membranes remain moist, and that bacterial infections are prevented. This can be managed by the use of oral hygiene gels that contain chlorohexidine designed for dental care.

The procedure of removal of the gastrostomy tube in animals over 10kgs is to cut the catheter off flush with the skin after pulling it taught. The catheter tip is then passed in the faeces. The resulting gastrocutaneous fistula will rapidly heal if kept clean.

Microenteral Nutrtion.

Microenteral nutrition is the delivery of very small amounts of water, electrolytes and easily absorbable nutrients directly into the GIT. This method is often under used in veterinary practices, but allows the nutritional requirements of the intestinal mucosal. This helps to preserve the intestinal blood flow, the mucosal barrier and its immune function.[2] Initial volumes of 0.05 to 0.2 ml/kg bwt/hour are recommended, and will add exceptionally little to the volume of fluids normally produced by the stomach. Gradual increases can occur to 1-2ml/kg bwt/hour, over a 24 to 48 hour period. Enteral solutions that can be used include oral rehydrating solutions, and those containing glutamine.

Parenteral Nutrition.

PN is often fought with complications, but should be considered in animals that are unable to tolerate enteral supportative feeding methods. This includes animals that are vomiting or regurgitating, or those unable to protect their airway. Complications such as hyperalimentation or overfeeding can be common, and lead to metabolic complications.[1] Though these can be resolved easily by discontinuing, and do respond fairly rapidly. Other complications can be more severe, and include localised infections, which can lead to septicaemia. The use of TPN in equines can be limited due to the expense of maintaining fluids over a period of time. The use of a central line (in TPN cases) for administering supportative nutritional methods requires careful attention to aseptic techniques. The PN solution can also act as the perfect reservoir for bacterial growth. In many cases of post-operative colics, the animal is usually toxic, and very prone to jugular thrombosis, TPN can exacerbate this problem. Hence the use of thoracic, cephalics and saphenous veins, in order to preserve the jugular.

Parenteral nutrition can be divided into two different categories:

1.) TPN – where parenteral nutrition is formulated to meet 100% of the animal's energy requirements. In all animals receiving TPN hyperglycaemia and glucosuria can develop in some cases. This hyperglycaemic state can possibly reflect the decrease in peripheral glucose uptake resulting from mild insulin resistance. This can precipitate to laminitis in horses. As a general rule, intravenous dextrose solution should be used as the sole source of nutrition for no more than 2-3 days. An amino acid solution should be added to the dextrose solution, and then lipids. On day one of the administration ~12% of the animal's total fluid requirement should be given, on day two ~24%. The remainder of the fluids should be administered via IV fluids. An example of a TPN solution will contain 32% lipid energy, 9% protein energy and 60% carbohydrate energy.

2.) Partial parenteral nutrition (PPN) – formulated to meet 40-70% of the animal's total energy requirements. These solutions are diluted, which decreases the protein and caloric density. But by doing these allows the solution to be administered via peripheral veins. Peripheral PN can also be used to describe PPN.[1] A lower osmolaritry of solution is required, and this is achieved by using 5% dextrose preparations rather than 50%. PPN is only intended for short-term use (less than five days), and should be limited to animals that are not debilitated. It should be noted that purely dextrose solutions are not appropriate for the long-term treatment of hypoglycaemia. In critical care situations insulin resistance and glucose intolerance is exceptionally likely, and hence a fat source must be available. When using 5% dextrose infusions this will only provide <25% of the RER when administered at maintenance fluid rates.[1]

Parenteral Nutrition Administration.

Aseptic placement of a dedicated catheter is required in order to administer PN. When placing a catheter for TPN, a central venous (jugular) catheter is required. The use of multi-lumen catheters is advocated, as it can also be used for blood sampling, and the administration of fluids and intravenous medications, as separate lumen are required for each purpose. As with enteral assisted feeding methods, TPN should be instituted gradually over a three-day period. Flow rates of other fluids being concurrently administered should be adjusted accordingly.

Once the animal is consuming adequate nutrient intake voluntarily, the PN can be discontinued. TPN needs to be discontinued gradually over a 6 to 12 hour period. PPN can be discontinued abruptly. All animals receiving TPN and PPN should be closely monitored. Daily weighing of the animal must occur, alongside TPR throughout the day. The catheter site should also be monitored, and handled aseptically.

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Problem.	Common Causes.	Signs and clinical symptoms.	Treatment.
Infection	Poor hygiene, contamination of the tube site by oral flora at the time of tube insertion.	Inflammation, malodour, pain, increased exudates.	Swab the site for culture and sensitivity, administer appropriate antibiotics. Apply dressings if indicated.
Leakage	Poor designed tube, infection, and over-granulation.	Excessive movement of the tube or the tube cannot be moved, inflammation of the skin and/or excoriation.	Identify the cause of the problem. Use a barrier cream around the tube insertion site.
Over-granulation	Infection, incorrect positioning of the tube, excessive movement of the tube.	Inflamed, red raised tissue, bleeding, pain.	Treatment of any infection, correct any positioning problems.
Blocked tube	Inadequate flushing regime, damaged tube, medication interaction.	Difficulty flushing tube, unable to administer water, feeds or medications.	Review flushing regime, possibly medications. Flush with soda water, or enzymatic solution. Preparations used in human medicine use commercially prepared enzymes e.g. Clog Zapper to remove blockages.
Feed-associated problems such as poor tolerance	Feed rate, technique, method, timing of feeds or medications, or type of nutrition used.	Bloating, nausea, vomiting, diarrhoea, constipation.	Eliminate other causes. Review all feeding and medication regimes. Use a diet with a slightly higher fibre content.
Aspiration	Incorrect positioning of the tube when feeding, feed rate/volume too high, poor gastric emptying.	Chest infection, aspiration pneumonia, coughing, regurgitation of the feed.	Review feed rate and administration method. Confirm that tube has been placed correctly, e.g. with radiography.

Table 6.9: Problems that can arise when utilising tube feeding.

Pancreatitis.

The exocrine pancreas is highly responsive to changes in nutritional substrates present within the diet. When the pancreas becomes inflamed, symptoms such as depression, anorexia, vomiting, diarrhoea and displays of abdominal pain can present. Pancreatitis whether in an acute or chronic form is a common occurrence seen in veterinary practice. Pancreatitis is exceptionally painful and requires analgesia as its primary treatment. This is due to the proteolytic enzymes are activated *in situ* resulting in auto digestion. There are many factors, which can predispose to pancreatitis including breed, age, gender, neuter status and body condition.

Treatment of pancreatitis involves consideration of the following factors:

- Treatment/Removal of the initial cause.
- Fluid therapy, in order to maintain hydration and electrolyte levels. Plasma may be required in severe cases.
- Analgesia. Non-steroidal anti-inflammatory drugs (NSAIDs) are best avoided in these cases due to the gastric and renal side effects. Non-ulcerogenic NSAID could be considered in chronic cases.
- The nutritional status of the animal needs to be considered. On a short-term basis should the animal be starved, tube fed or use of total parenteral nutrition (TPN)? There is current conflicting evidence to support each. Thoughts should also be given to long-term dietary management.
- Symptomatic treatment with anti-emetics, antibiotics and anti-ulcer medication as necessary to prevent complications. Use of broad-spectrum antibiotics is two fold. Firstly to combat any infection that might be present, and secondly to protect against septicaemia caused by bacterial translocation.
- In cats hyperglycaemia is often noted, but can be mild and transient. Although in some cases diabetes mellitus can develop and may require insulin therapy.

The aims of clinical nutrition are:

- To provide sufficient calories and nutrients to the body without over loading the pancreas.
- Preventing the possibility of bacterial translocation from the gastrointestinal tract and intestinal atrophy.

Clinical Nutrition.

<u>Fat.</u>

Fat in the diet delays gastric emptying, which can in turn promote vomiting in the dog and cat. The delay in gastric emptying is a significant cause of upper gastrointestinal signs in the dog, including abdominal discomfort, nausea and vomiting. Preventing or inhibiting the release of pancreatic enzymes that aid in digestion of fats is an essential component of treatment. For long-term management feeding a low-fat diet with or without the use of pancreatic enzymes can reduce post-prandial pain. The restriction of fat content of the diet in cats is not as vital as with dogs, but should still be of a highly digestible nature.

<u>Carbohydrates.</u>

As the fat content in the diet needs to be reduced yet highly digestible, the carbohydrate content must provide a greater contribution to the ME content of the diet. The carbohydrates present in the diet must be easily digestible, as digestion and absorption may be adversely affected in all cases of gastrointestinal upset.

Vitamins and Minerals.

Guidelines for vitamin and mineral levels are the same as for that particular life-stage. If large volumes of vomiting or diarrhoea are present then care should be taken, as excessive amounts of the water-soluble vitamins can be lost. When feeding an exceptionally low fat diet, the quantity of fat-soluble vitamins can also be lost. As some of this vitamins are required in fat metabolism it is important that these losses are replaced.

Hypokalaemia is particularly common in animals suffering from pancreatitis, and can be severe and life threatening. If vomiting or diarrhoea is present then monitoring of electrolyte balance needs to occur, as it should be with any animal receiving intravenous fluid therapy. Oral supplementation may be required.

Supplements.

The use of exocrine pancreatic enzymes in cases of acute pancreatitis is starting to become commonplace in veterinary practice. Even if the animal is not displaying clinical symptoms of exocrine pancreatic insufficiency (EPI), the role of these enzymes has an important part to play. Addition of the enzymes to the meal at least five minutes prior to feeding has proven to reduce post-prandial pain, and does help to ease the pancreas back into work after a period of 'rest'.

Feeding a pancreatic patient.

Current dietary therapy for dog's suffering from acute pancreatitis is nil-by-mouth, until the clinical symptoms stop, and in some practices this can be up to five to seven days. Recent evidence has shown that nil-by-mouth in these cases has little benefit to the animal. Very short-term rest (48hours) is still indicated to allow pancreatic rest in acute pancreatic cases, but for no longer. For any cases that require starving over the 48hours total parenteral nutrition must be provided, along side very small amounts of nutrition being given by mouth (microenteral nutrition). The oral nutritional support is required to prevent gastroduodenal ulceration, bacterial translocation from the gut and septicaemia. Small amounts of watered down baby rice or cottage cheese can be used before moving onto a commercial low-fat, highly digestible diet. Baby rice is food of choice in these cases, and Table 6.17 demonstrates a typical nutrient analysis.

In cats, nutrition is generally supplied by enteral means (gastrostomy feeding tube), in order to avoid hepatic lipidosis. There is no clinical evidence that this type of nutrition exacerbates the course of acute pancreatitis. There is also evidence that enteral support is superior to parenteral support as described as above. Oral intake in cats should only be restricted if persistent vomiting is occurring, and then for as short a time as possible.

Dietary long-term control of pancreatitis is vital, but initially confirmation of the presence of hyperlipidaemia needs to be obtained. Those dogs suffering from pancreatitis with associated hyperlipidaemia will need to be maintained on a different diet than those without. If the lipid levels in the blood stream are within normal levels then a highly digestible low fat diet can be used. If hyperlipidaemia is concurrent then a low fat diet is required again, but these types of diets can have a corresponding high fibre content thus reducing digestibility. This can be advantageous as the majority of dogs, which suffer from hyperlipidaemia, as they do tend to be overweight and can benefit from this type of diet.

Weight control is important in these cases, as a major predisposing factor is obesity and being fed high fat treats. By altering the diet in order to reduce fat content, most animals will gradually lose weight but this does need to be monitored regularly.

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Classification of acute diarrhoea.	Pathophysiology	Commonly causes
Osmotic	Excess water-soluble molecules in the intestinal lumen result in osmotic retention of water. D+ occurs when the fluid volume overwhelms the absorptive capacity of the small intestine and colon.	Sudden dietary change, malabsorption.
Permeability (Exudative)	Inflammation in the intestine can stimulate increase secretion of fluid and electrolytes, and impair absorption.	Permeability can be affected by ulceration, especially in cases of neoplastic disease. Where severe damage is present serum protein and blood loss can also occur. Portal hypertension can also result in exudation of fluid into the intestinal lumen.
Secretory	When the absorptive capacities of the small intestine and colon are exceeded. Resulting D+ can be severe, and does not usually resolve with fasting.	Toxin release by enteric infectious agents (e.g. *Giardia* spp, *Escherichia coli*).
Dysmotility	D+ can result in secondary alterations in motility, usually reduced intestinal motility.	Ileus and abnormal dilation of the intestine can be physical, neuromuscular, metabolic or functional abnormalities, and can further promote D+ as stasis allows for bacterial fermentation.

Table 6.14: Classification and pathophysiology of diarrhoea.[2]

Clinical symptom	Small intestinal diarrhoea	Large intestinal diarrhoea
Blood in faeces	Melena	Heamatochezia
Frequency of defeacation	Mild increase (<3 x day)	Markedly increased (>3 x day)
Faecal volume	Large quantities	Small quantities
Faecal quality	Loose, watery, “cow-pie”	Loose to semi-formed, “jelly-like”
Urgency	Usually absent	Often present
Tenesmus	Rare	Common
Faecal mucus	Rare	Common
Dyschezia	Absent	May be present
Vomiting	May be present	May be present
Weight loss	Common	Rare
Flatus/borborygmi	May be present	May be present

Table 6.15: Differences between small and large intestinal diarrhoea in the cat and dog

Nutrient	Per 100g (as fed)
Fat	0.8g
Fibre	1.5g
Carbohydrates (as sugars)	85.6g
Protein	8g

Table 6.17: Typical analysis of baby rice.

Hepatobiliary Disease.

The role of the liver in the body is critical in maintaining homeostasis and the removal of waste products that have accumulated within the body. The liver is essential in the production of proteins and deamination of excessive unrequired proteins. Understanding the functioning of the organ is essential in designing a diet relating to the clinical symptoms that the animal is exhibiting. The liver can be damaged in a number of different ways, from infection and hepatic encephalopathy as an adjunct to medical therapies and from toxins. Congenital abnormalities such as portal systemic shunts will also affect the way in which the liver functions. Analysis of laboratory results and other diagnostic means will enable the practioner to identify where the damage is occurring within the liver. Delayed recovery from general anaesthesia may be the first indication of compromised liver function. Nutritional management of hepatobiliary disease is usually directed at clinical manifestations of the disease rather than the specific cause itself. The aims of nutritional management are:

- Maintaining the normal metabolic processes.
- Avoiding toxic by-product accumulation.
- Correcting any electrolyte disturbances.
- Providing substrates that support hepatocellular regeneration and repair.

There is no one ‘ideal’ liver disease treatment or diet. The dietary needs of the patient requires a good understanding of what you are trying to achieve and the individual animal’s needs. A palatable high quality protein diet, supplemented with zinc, B vitamins and antioxidants should be fed. Protein should not be restricted unless it is essential to control encephalopathy. Malnutrition is a very common feature of chronic hepatic disease, and correct nutrition for each individual patient is important to help regulate the hormonal milieu that occurs with hepatic injury.

Clinical Nutrition.

<u>Protein</u>
The maintenance of a positive nitrogen balance is important for the preservation of body condition and protein synthesis. Protein malnutrition is common in patients with chronic liver disease, where the clinical manifestations include weight loss, loss of muscle tissue and hypoalbuminaemia. Protein intake needs to be of a fine balance; in some dogs the protein requirements may exceed those of normal maintenance requirements due to the increased protein turnover and the demands of hepatocellular regeneration. A moderate restriction of protein levels is not always recommended, and the quality (Biological Valve) of the proteins should be increased. This is recommended as they fulfil the animal’s needs with minimal production of nitrogenous waste. Studies have shown that in human patients with hepatic failure that the nitrogen balance can be improved if the diet is divided into small, frequent meals.

Dairy products (cottage cheese, milk) or eggs are of excellent benefit, probably related to the factors such as the relatively high ratio of carbohydrates to protein, their influence on intestinal transit and colonic pH, as well as the differing amino acid composition. Fibre of soya origin has shown to provide an excellent source of dietary fibre that reduces ammonia production and absorption in the colon and assists ammonia elimination in the faeces.

One of the roles of the liver is a protein regulatory event, including degradation of the essential amino acids (including the aromatic amino acids [AAA], but not the branched-chain amino acids [BCAA]) and some of the nonessential amino acids. Dogs and other omnivores are able to down-regulate the activities of protein degradation when minimal dietary protein is consumed, cats are not able to do

this. Plasma amino acid concentrations differ depending on the type of hepatic failure. In the normal healthy animal AAA (i.e. tyrosine, phenylalanine and tryptophan) are effectively extracted from the portal circulation and metabolised by the liver. Reduced hepatic function is associated with an increase in circulating AAA. Conversely the plasma concentrations of BCAA (i.e. leucine, isoleucine and valine) and most other amino acids are reduced due to an increased rate of metabolising by muscle and adipose tissue. The ratio between AAA and BCAA can be used in order to evaluate the liver function. In healthy dogs a ratio between BCAA and AAA ranges between 3.0 to 4.0. This ratio can be reduced to 1.0 or less in dogs with portosystemic vascular anomalies and chronic hepatitis. Other factors such as increased levels of insulin, glucagons and catecholamines are thought to contribute to the altered amino acid metabolism seen in these patients. Alterations in this ratio have also been implicated in the pathogenesis of hepatic encephalopathy. Hence BCAA enriched solutions have been used in human nutritional support for many years in those patients suffering from chronic hepatic disease and hepatic encephalopathy.

Carbohydrates and Fats.

Non-protein calories should come from highly digestible carbohydrate and fat sources. The fat level in the diet should not be restricted unless there is clinical evidence of steatorrhoea.

Vitamins and Minerals.

The requirement of vitamins and minerals within the diet is dependent on the causal problem with the liver. Copper levels should be restricted due to accumulation within the liver, and is discussed further below. Vitamins, which are produced within the liver, should be supplemented, as deficiencies are common, as is deficiencies of zinc. Due to the oxidative cell damage, which is correlated with the severity of liver disease antioxidants should also be supplemented.

Copper-associated Hepatotoxicosis in Dogs.

Bedlington terriers can often develop copper storage disease and subsequently hepatitis and cirrhosis. Statistics state that roughly 25% of Bedlington terriers are affected and another 50% are carriers. Some other breeds have also been affected namely the West Highland White Terriers and Doberman Pinschers. Copper-associated hepatotoxicosis is caused by an inherited autosomal recessive trait that results is impaired biliary excretion of copper. Thus diets for these dogs do need to have a reduction in the copper levels. Supplements containing copper should also be avoided. Homemade diets should not contain liver, shellfish and organ meats, which are all high in copper content.

The role of copper in hepatic diseases in other breeds of dogs is less clear. There are theories that elevated hepatic copper concentrations precede liver damage, whereas others contend that the excess hepatic copper results from faulty copper excretion caused by chronic cholestasis. A third theory is that elevated levels are antecedent to the disease and are incidental to disease progression. Anti-inflammatory agents, such as prednisolone, can be beneficial in the management of chronic hepatitis in Bedlington Terriers and West Highland White Terriers.

The nutritional aims of managing patients with Copper-associated Hepatotoxicosis are:

- To decrease further absorption of copper from the gastrointestinal tract.
- To enhance copper excretion.

Portal Systemic Shunts (PSS) and Hepatic Encephalopathy (HE).

Portal systemic shunts (PSS) are vascular communications between the portal and systemic venous systems. This communication allows access of portal blood to the systemic circulation without passing through the liver. Congenital PSS are most common and are inherited. They may either be intrahepatic (large breeds such as Irish wolfhound and Burmese Mountain Dog) or extra hepatic (medium and toy breeds, such as Yorkshire terrier, Cairn terrier and Dachshund). Stunted growth or failure to gain weight can occur in these animals. Acquired PSS forms in response to portal hypertension caused by fibrosis and chronic cirrhosis. As a result of inadequate hepatic clearance of

toxins and altered liver function hepatic encephalopathy can result. Ammonium urate and other purine uroliths can also occur due to the high urinary excretion of ammonia and uric acid.

Shunting results in nutritional depletion of the liver as well as failed delivery of substrates to the liver for degradation and metabolism. Hepatic encephalopathy is a complex metabolic disorder, which is characterised by abnormal mental status. There are many factors that can precipitate HE, which are detailed in Table 6.18.

The nutritional aims for managing patients with hepatic encephalopathy include:

- Reducing dietary protein.
- Changing intestinal flora.
- Decreasing intestinal transit time.

Soluble or fermentable fibre in the diet such as cellulose and lactulose aid the animal's suffering from liver dysfunction as it helps reduce the side effects of deamination of proteins. Lactulose is a synthetic disaccharide that is hydrolysed into volatile / short chained fatty acids in the colon. The change in the lumen pH traps the ammonia as ammonium ions, which are removed in the faeces. There is also the beneficial effect that colonic bacteria use the increasing nitrogen in reproduction and growth, whilst also inhibiting ammonia generation by colonic bacteria through a process known as catabolite repression.

Insoluble or non-fermentable fibre is also an important constituent in the diet of animals suffering from hepatic disease. Constipation is a predisposing factor for the development of encephalopathy since it increases the contract time for colonic bacteria to act on the faeces and produce ammonia.

Vitamins and Minerals.

Vitamin E is a superb antioxidant, and may be cytoprotective especially in copper toxicity; due to the effects it has protecting against lipid peroxidation. Vitamin K supplementation may become a necessity if clotting times are prolonged, especially prior to hepatic biopsies being taken. The vitamin K stores within the liver are eliminated and do become rapidly depleted, although the function for synthesis of the prothrombin-complex clotting factors is always lost before the storage of vitamin K is depleted. The other fat soluble vitamins of A and D should not be supplemented, as vitamin A can cause hepatic damage and vitamin D can cause calcification within the tissues.

The level of calorific intake determines the requirements for the water-soluble vitamins. Thus if anorexia is present the requirement is low, though when nutrition intake increases, the water soluble vitamins are required in order to replenish coenzymes involved in metabolic processes in the liver and other tissues. As with all animals suffering from polydipsia and polyuria, supplementation with B vitamins is required. Animals suffering from hepatic disease have been recommended to receive a double dose of B vitamins. Vitamin C should not be supplemented as it can increase the tissue damage associated with copper and iron disease.

Choline is essential in the packaging of VLDL in the liver and therefore the exportation of triglycerides. A choline deficiency with concurrent lipolysis may slow down VLDL export and promote hepatic lipid accumulation. Hepatic diets should contain supplementation of choline and carnitine, but the efficacy levels are unknown.

There are large amounts of supportative data that zinc deficiency is common with hepatic injury. This is due to a reduction in absorption and an increase loss via the urine. Nutrition supplementation of the diet with zinc reduces encephalopathy; at an effective rate the same as lactulose.

Feeding a Hepatic Diet.

The aim of the dietary modifications is to meet the animal's daily requirement for nutrients, whilst reducing the metabolic demands placed upon the liver. The calorific needs of the liver should be met with non-protein calories and the nutritional deficiencies which can occur due to the loss of hepatic function, need to be met within the diet

Frequent feeding of small meals is preferred to ensure optimal assimilation and to limit the entry of dietary protein into the colon. Other complications of liver disease include gastrointestinal ulceration and ascites. If ulceration is known to be present the animal must not go without food.

Idiopathic Feline Hepatic Lipidosis.

The incidence rate of feline hepatic lipidosis is unfortunately increasing. Most of these cats are obese and usually present with a history of prolonged anorexia after a stressful event. Hepatic lipidosis is characterised by accumulation of excess triglycerides in the hepatocytes, which result in cholestasis and hepatic dysfunction. This disease may be idiopathic or may develop secondary to some underlying medical problem. Hepatic lipidosis is more commonly seen in cats due to the several special features, which make the cat less tolerant to periods of anorexia. Reasons for this include the cat's reduced ability to synthesise arginine being lower than that of other species. This specific amino acid is an intermediate of the urea cycle, in which ammonia is converted to urea. Its other role is in the production of apoproteins, which are incorporated in very low-density lipoproteins (VLDL). VLDL transport triglycerides from the liver to adipose tissue. Any changes in the availability of arginine can result in hyperammonaemia, even when there is minimal hepatic dysfunction, and disturbance to the transportation of triglycerides. Taurine is also required in VLDL production and the conjunction of bile acids. As taurine is also required in larger amounts in the cat, any deficiencies can result in VLDL disturbances. L-Carnitine is also essential in the transportation of long-chained fatty acids into the mitochondria. L-Carnitine is often supplemented into hepatic diets and obesity diets for this very reason, aiding in the mobilisation of circulating fats. If any of these supply chains become unbalanced then hepatic lipidosis can occur. Since any disruption in hepatic lipid metabolism can cause lipidosis, it is unlikely that there is a common cause for all cases of feline hepatic lipidosis. Hypokalaemia is present in about 30% of cats with severe hepatic lipidosis, hence electrolyte monitoring is important, as hypokalaemia can prolong anorexia and exacerbate expression of hepatic encephalopathy.

Insulin resistance with subsequent glucose intolerance represents a key metabolic abnormality in all patients with chronic hepatic injury. This can be due to reduced insulin activity from depletion of insulin receptors on the target cells. Hence, why many cats with hepatic lipidosis are hyperglycaemic. Reduced insulin activity results in activation of triacylglycerol lipase, with consequent hydrolysis of triglycerols. Resulting in the increased release of free fatty acids into the circulation from the adipose tissue.

<u>Treatment of Feline Hepatic Lipidosis.</u>

Nutrition plays a supportative role in the management of most hepatic diseases, but nutritional therapy is the primary treatment for feline idiopathic hepatic lipidosis. It is a condition that requires vigorous supportative care; this includes intravenous fluids and nutritional support. The provision of an adequate daily energy intake is the cornerstone of successful medical management. This is can only truly be achieved through early tube feeding via a nasogastric tube. Force-feeding of the cat or the use of appetite stimulants can work to a degree, but rarely meet the calorific needs of the cat. Food aversion is an exceptionally important component to be considered when tempting anorexic cats, hence why tube feeding is a more preferable method. Intravenous feeding (total parenteral feeding) is not recommended unless some enteral nutrition is also being concurrently provided. Parenteral feeding is associated with hepatic steatosis, villus atrophy, fluid and electrolyte imbalances, and an increased incidence of sepsis. This is due to enteral nutrition providing antigenic stimulation to the gut-associated lymphoid tissue (GALT) and stimulates secretion of IgA, which helps to maintain an intact gastrointestinal barrier, in order to prevent bacterial translocation. Other factors that need to be considered in these cases include:

- Correction of hypokalaemia. This can be achieved by potassium supplementation through intravenous fluids and oral, tube feeding methods.
- Correction of hyperglycaemia, and cell intake of calories. Carbohydrates should not replace the dietary fat due to this glucose intolerance.
- Dietary protein intake needs to be adapted to suit the individual patient. It is advised that the protein intake should not be reduced below 20% of calories from this source.
- When starting tube feeding a balanced formulation sufficient to meet the cat's RER at its current weight is ideal. In grossly obese cats carrying 35% or more fat, they should be fed at a lower estimated optimal weight rather than their current weight.

Supplements.

S-Adenosylmethione (SAMe).

Glutathione is a major antioxidant produced by the liver, its main function is to reduce oxidative damage to the hepatocytes by free radicals. When the liver is compromised there is an increased production of free radicals, and thus the extent of the oxidative stress suffered by heptaocytes is increased. Thus an increased supply of glutathione is required as production is also decreased during hepatic disease. The supply of glutathione is via S-Adenosylmethione (SAMe). SAMe initiates three major biochemical pathways: transmethylation, transulphuration and aminopropylation, which all help promote liver health and preserve functioning liver tissue.