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**KEY NUTRIENTS IN THE MANAGEMENT OF GASTRO-INTESTINAL DISEASES.**

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Dietary therapy is an essential aspect of the management of gastro-intestinal (GI) diseases. However, no single diet is likely to be effective for every patient. To best fulfil the need of each patient, it is thus important to understand the role of each key nutrient in the management of GI diseases. Care should also be taken to provide nutrients to optimise the intestinal microflora.

**Dietary rest.**

Traditionally, patients with vomiting and/or diarrhoea have been managed with dietary rest followed with small amounts of highly digestible “bland” diets. However, recent evidence suggests that enteral nutrition, early in the management of viral gastroenteritis, would be more effective than food deprivation.

**Protein.**

*Protein digestibility*

Patients with GI diseases should be fed diets with highly digestible (> 90 % digestibility) proteins. Undigested protein will over-stimulate the GI immune system and therefore increase the risk of inducing food allergy. Most patients with GI disease are even at higher risk as their mucosal barrier is damaged. Undigested proteins will also promote bacteria (i.e. Clostridium perfringens) that are believed to be harmful to the health of the colon and the host. Finally, putrefaction products (e.g. bioamines, mercaptans, indols, ...) resulting from the fermentation of undigested proteins in the colon will induce poor digestive tolerance, strong faecal odour and might result in inflammation, toxin production as well as hypersensitivity reaction.

Traditionally, animal protein sources have been considered as more digestible than vegetable protein sources. This is no longer true. Soy isolate, soy hydrolysate, corn gluten and wheat gluten are all purified sources of very highly digestible vegetable proteins.

**Protein hydrolysates**

Recently, diets formulated with protein hydrolysates have become available for the management of dogs and cats exhibiting adverse reactions to food. By enzymatically breaking the protein down into smaller peptide fragments it becomes intrinsically less allergenic. The very high digestibility of those peptides probably also contribute to reduce allergenicity. Indeed, if a dietary protein is properly digested prior to contact with the gastrointestinal mucosa, it will not activate the immune system. Most common protein allergens range in size from 14 to 40 kilo Daltons (kD), although smaller (10 kD) and larger (70kD) molecules can be immunogenic. Molecular weight is not a tool sensitive enough to compare the antigenic properties of protein hydrolysates. Therefore, only clinical trials can confirm the beneficial effects of a hydrolysate in the management of adverse food reactions.

**Fat**

The choice of a diet with a high or low fat content depends on the origin of the intestinal disorder and the patient’s clinical status. High fat diets are energy dense, reducing the volume of food consumed at each feeding. Fat will slow gastric emptying and prolong digestion, which can be beneficial for some forms of intestinal disease. Fat is the most highly digestible of all the nutrients, with digestibility values exceeding 90%. Forty-nine dogs with a confirmed diagnosis of chronic intestinal disease (exocrine pancreatic insufficiency, inflammatory bowel disease, bacterial overgrowth, acute or chronic gastritis) were fed a diet containing a high concentration of fat (> 20 % on a Dry matter basis). The benefits of the high fat diet were readily apparent with improvements in appetite, weight gain, and resolution of clinical signs of vomiting.
and diarrhoea noted at 15 and 30 days following institution of dietary therapy.

Bacteria in the intestinal tract can metabolise undigested fat to hydroxy-fatty acids, which leads to secretory diarrhoea in the large intestine. Bacteria also deconjugate bile acids further impairing fat digestion and absorption. For this reason, fat restriction might be beneficial for conditions where fat may become available for microbial metabolism, for example in malabsorption syndrome, small intestinal bacterial overgrowth, or bile acid deficiency [1]. Fat restriction is also indicated in pancreatitis and in patients at risk for pancreatitis as fat will stimulate pancreatic secretion and a fatty meal often appears to trigger episodes of acute pancreatitis [1].

Unlike amino acids and monosaccharides, which are absorbed directly into the blood stream, fat is discharged from enterocytes into lacteals and is transported to the systemic circulation via mesenteric lymph vessels and the thoracic duct. Lymphangiectasia, a disorder characterised by congestion and/or dilatation of lymphatic vessels, will impair fat transport. Therefore, restriction of dietary fat is clearly indicated for the management of this disease as well as other exudative enteropathies. [1, 14]

Fish oils
Long chain ω-3 fatty acids such as eicosapentanoic acid and docosahexaenoic acid, directly compete with arachidonic acid for lipoxygenase and cyclo-oxygenase enzymes. Subsequent metabolism of eicosapentanoic acid generates less inflammatory mediators compared to the metabolism of arachidonic acid. Short-chain ω-3 fatty acids such as α-linolenic acid are less efficiently metabolised by dogs and cats to reduce the production of inflammatory mediators [15].

Carbohydrates
Rice has long been considered the ideal carbohydrate of gastrointestinal disease [1,16]. Rice is very highly digestible because it has a limited branched starch structure (amylopectin) and very low dietary fibre content. Rice has rarely been implicated in adverse food reactions. Further, rice improves the digestibility of dry diets and contains soluble factors that inhibit secretory diarrhoea.

Dietary Fibres
Fibre can be classified according to solubility and fermentability. Soluble fibres, such as psyllium, form a gel in water which delays gastric emptying and slows absorption in the small intestine. Insoluble fibres such as cellulose, increase faecal bulk, absorb toxins and normalise both segmental and propulsive motility. Both insoluble and soluble dietary fibre may be beneficial in the symptomatic treatment of certain cases with large bowel diarrhoea, since fibre helps to normalise transit time and increase faecal water content. By normalising intestinal transit time, insoluble fibres are often recommended for patients with constipation.

Fermentable fibres such as beet pulp, pectin, guar gum, gum arabic, and fructo-oligosaccharides (FOS) may have a positive effective on the mucosal barrier by stimulating the growth of intestinal bacteria such as Lactobacilli and Bifidobacter [17,18]. These bacterial species have been shown to be beneficial to gastrointestinal health by decreasing the growth of pathogens such as Clostridia and E.coli. In addition, bacteria degrade fermentable fibres to produce the short chain fatty acids butyrate, acetate and propionate, which provide fuel for the colonocytes [20].

Zeolite
Zeolite, or sodium silico-aluminate a tetrahedral clay, is capable of absorbing bacterial toxins, bile acids, and gases. By forming a protective film on the intestinal mucosa, zeolite helps to enhance the intestinal mucosal barrier. Zeolite has been used in piglets and calves in the management of weaning diarrhoeas. Grandjean et al reported that the addition of clays to food decreased the duration and the severity of stress-induced diarrhoea in sled dogs [21]. Clays have also been shown by Fioramonti et al to reduce diarrhoea induced by cholera toxin in dogs [22].

Mannan-oligo-saccharides (MOS)
MOS are yeast wall extracts that contribute to intestinal health [23,24]. MOS has been shown to limit the development of potentially pathogenic bacteria by competitive inhibition of binding sites on the intestinal mucosa. Pathogenic bacteria cannot reach or attach themselves to the enterocytes and are evacuated with the faeces. In addition, MOS has been shown to stimulate local intestinal immunity by increasing the level of IgA.
**Conclusions**

Dietary therapy is a cornerstone in the management of intestinal disease. However, there is not one diet that is appropriate for every patient. Rather, the key nutrients of the diet such as protein, intact versus hydrolysed, and fat level must be selected according to the underlying pathophysiology of the disease process. The effectiveness of the dietary therapy can be reinforced by the addition of nutrients such as FOS, zeolite, MOS and long chain omega-3 fatty acids that nourish and protect the intestinal mucosa and promote a beneficial bacterial flora.

**Acknowledgement**

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References:

12. Royal Canin, Research Center, data on file (2005)
Companion Animal Programme

THE ANOREXIC CAT – WHAT SHOULD I DO?
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Introduction
Cats will often present to the veterinary surgeon with a diminished or absent appetite. Feeding is a focal interaction for owners and their cats, and anorexia can be the first sign of a number of disease processes. However, it is a non-specific sign and, as a consequence, the veterinary surgeon must consider a wide array of differential diagnoses. Significant and prolonged anorexia will lead to loss of weight and body condition. Occasionally, anorexia may be missed or underestimated by an owner, not least when the cats live with other cats, and where the cats feed communally. This may mean that the degree of weight loss evident is out of proportion to the apparent degree of anorexia.

Definitions
Anorexia describes a loss or lack of appetite, and may be partial or complete; inappetance or hyporexia describe a reduction in appetite.

There are two form of anorexia:

1. True anorexia. Due to decreased appetite; the animal has no interest in food.
   a. Primary anorexia.
      i. Neurological diseases reducing cerebral arousal; cranial trauma; anything affecting the appetite centre in the hypothalamus.
      ii. Anosmia. Cats are highly dependent on smell to stimulate appetite.
      iii. Behaviour, e.g. environmental (change in housing/family members/furniture); psychological (fear, anxiety)

2. Secondary anorexia. Due to disruption of endocrine, cytokine or neurological control of appetite.
   i. Systemic disease
   ii. Pain (abscess, fractures, osteoarthritis, periodontal disease, neoplasia, foreign bodies)
   iii. Neoplasia
   iv. Neurological diseases
   v. Chemotherapeutics causing nausea and opioids inhibiting the orexigenic (appetite-stimulating) network
   vi. Upper respiratory tract/nasal disease reducing the ability to smell
   vii. Lower respiratory tract disease causing difficulties in breathing

In addition to weight loss, prolonged anorexia causes systemic complications due to immunosuppression, reduced hepatic function (detoxification) and intestinal dysfunction.

Diagnosis
Anorexia is a syndrome and, as a result diagnosis and management depend upon the underlying cause. A thorough clinical examination of all organs, including the eyes, will help to refine the list of differential diagnoses and direct the diagnostic approach. Routine haematology and clinical biochemistry are usually required in most cases. Useful additional tests include feline pancreatic lipase immunoreactivity (to investigate pancreatic inflammation) and assessment of B vitamins (folate and cobalamin) to assess for deficiencies indicative of pancreatic, hepatobiliary or gastrointestinal disease. Other tests (e.g. endocrine tests, T4, fructosamine), virology and acute phase protein assessments may be required in some circumstances.

Intervention
Management of anorexia lies in identifying and managing the underlying disease whilst supporting the cat’s fluid and nutrient balance until appetite resumes. A complete discussion of therapy for underlying causes is outside the remit of this talk.

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However, one example is the need to control pain, for example sublingual buprenorphine 0.01-0.02mg/kg BID-QID, since pain/discomfort is a common reason for anorexia in cats. Feeding an anorexic sick patient can help to avoid or minimise the impact of malnutrition. This enhances recovery rates and reduces morbidity and mortality. Intervention should occur early once fluid and electrolyte imbalances are corrected.

Although the exact time of intervention may vary depending on the case in question, a good general rule is to consider placing a feeding tube in any cats that has been partially or completely anorexic for at least three days. Hepatic lipidosis is becoming more widely recognised in the UK and may complicate or exacerbate the underlying clinical condition. Additionally, a sick cat needs good nutrition to strengthen its immune system and recover from disease.

The main rules concerning when to intervene are as follows:

- When weight loss is greater than 10% (including obese patients)
- When there has been partial (<85% calculated energy requirements) or complete anorexia for more than three days.
- Earlier intervention may be required for patients in a catabolic state (burns, severe inflammation, major surgery or trauma).

**Monitoring response to therapy**
All cats should have their weight monitored daily and a regular assessment of body condition score. This is a method that frequently fails to provide sufficient daily food intake and, consequently, clinicians should be prepared to abandon it in favour of a more reliable means of nutritional support if nutrient requirements are not adequately met.

**Methods of nutritional support**

**Tempting a cat to eat** can be considered (partial anorexia only) and small food items should be used, which can be warmed to enhance smell. Again, the clinician should be prepared to reject the approach if it is not working (e.g. insufficient food intake), and move on to a more reliable approach (feeding tube placement). Syringe feeding cats can be stressful and encourage food aversion. Sometimes placing food in the mouth or on the lips can stimulate the cat to eat, but the method should only be used in cats that are stable and improving, as again this can encourage food aversion and heighten stress. Some cats respond well to food hides; other cats prefer company and encouragement to start to eat. Getting a cat to eat within a hospital environment is difficult due to the limitations of space and food placement.

**Appetite stimulants** (see table) can be considered (e.g. mirtazapine or cyproheptadine). However, they are usually only effective in cases of partial anorexia, where the underlying cause has been treated (and is resolving). If this approach is taken, the clinician should be ready to abandon it as soon as (e.g. within 24h) it is clear that food intake is not meeting requirements.

**Feeding by tube** is considered to be the most reliable approach to nutritional support. Feeding tubes enable enteral nutrition, and utilise either part or all of the gastrointestinal tract. Enteral nutrition is preferred to parenteral (“intravenous feeding”) as enterocytes derive 50% of their nutrients directly from the intestinal lumen. If the enterocytes are starved, the intestinal mucosa becomes hypoplastic and hypofunctional with increased permeability. Parenteral nutrition is technically difficult, requires good asepsis and should be reserved for malabsorption syndromes, acute severe pancreatitis and severe persistent vomiting. The choice of feeding tube will depend on length of support and the site of the disease (tubes should be placed distal to the problem area).

**Short-term support by feeding tube**
Here, the naso-oesophageal tube is preferred. For patients without oral, nasal, pharyngeal or oesophageal disease and who are not vomiting. Advantages: quick and easy to place, does not require GA. Disadvantages: short duration of use; liquid diet choice is limited; some cats do not like having the tube on their face.

**Longer-term support by feeding tube**
Three main options exist:

**Oesophagostomy tube.** These tubes are very useful and well tolerated in cats, being the authors preferred tube for nutritional support in this species. They are suitable for cats with oral and nasal disease. It is advisable not use them more cautiously in cases that are vomiting cats or those with oesophageal disease. A wider bore tube can be used, making feeding easier e.g. a range of diet formulation can be used rather than just liquid critical care diets. The tube is bandaged...
at the neck and does not affect whiskers or grooming. The main potential complication is infection at the stoma. A two-stage placement procedure is usual, where the tube is first inserted through the stoma in a retrograde direction and then subsequently redirected down the oesophagus.

**Gastrostomy tube.** The gastrostomy tube is also practical for long-term feeding in cats, but is contraindicated where there is significant gastric disease and/or persistent vomiting. Occasional complications include tube dislodgement (and possibly peritonitis) and infection at the stoma site. Placement techniques include:

- **Coeliotomy.** This method is invasive, but useful to place at the same time as an exploratory laparotomy.
- **Percutaneous endoscopically-placed gastrostomy (PEG) tube.** If equipment and expertise is available, this is the simplest and, perhaps safest method of placement.
- **Blind placement.** Using an applicator passed per os. There is less of a need for expensive equipment (e.g. endoscope), but there is greater potential for complications because the placement of the tube cannot be directly observed.

**Jejunostomy/enterostomy tube.** Entero-stomy tubes are usually placed surgically, although laparoscopic-assisted and endoscopic approaches are described. These tubes are rarely used, but occasional indications, included cases with gastric disorders or pancreatitis where small intestinal function remains normal (and an exploratory coeliotomy is being undertaken). Given that the stomach is bypassed, the patient is more prone to complications such as vomiting, diarrhoea and abdominal pain. Continuous food infusions are required to minimise complications.

**Feeding guidelines**

**How much should I feed?**

Food requirements are based on energy, the most important factor to support. For critical care patients, the daily requirements are best calculated from estimated resting energy requirement. The current controversy is over how much RER differs in critical illness. Correction factors used to be applied, but there is no validation for these factors. A better approach is to work with RER, monitor the patient’s weight and body condition and adjust the intake as necessary. For cats, a simple linear equation can be used to estimate RER:

\[ RER = 40 \times BW_{kg} \text{ (Kcal) per day} \]

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**What diet do I choose?**

In moist cases, it is best to use a commercial liquid critical care diet to satisfy energy, protein and micronutrient requirements. A high energy, high protein, easily digestible diet is recommended, though this may be manipulated depending on the underlying disease. Liquid enteral diets (e.g. Enteral Care, Fortol, RC Convalescence Support) are best for small diameter tubes. As enteral diets are low residue, there will be little faecal matter produced, though the cat should be monitored closely and hydration kept optimal to ensure against constipation.

Larger bore tubes (e.g. oesophagostomy and gastrostomy tubes) can carry a range of diets provided that they are first liquidised. This may be useful for patients with concurrent diseases requiring a modified diet (e.g. chronic kidney disease, chronic enteropathy).

**How long do I feed?**

Patients should be supported until their voluntary intake is >85% of maintenance requirements. Gastrostomy and enterostomy tubes must remain in place for at least 7-10 days to allow a seal to form with the abdominal wall. Oesophagostomy and gastrostomy tubes can be managed on an out-patient basis, allowing owners to feed their cat at home. This is particularly useful for hepatic lipidosis patients or those with severe rostral trauma.

**References:**

References are available upon request from the author.

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**ACUTE DIARRHOEA – CAUSES AND MANAGEMENT**

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**Causes of acute gastroenteritis**

Acute gastroenteritis is a general sign describing cases presenting with acute signs of vomiting and/or diarrhoea. It is very common, and is usually self-limiting. A number of categories exist:
Acute gastritis is very common in practice. It is characterised by acute onset of frequent vomiting, and is often associated with acute diarrhoea (i.e. acute gastroenteritis). Numerous aetiologies have been described, although the exact cause is often obscure. However, tracing the exact aetiology is rarely required because the process is usually self-limiting. Potential causes include:

- Dietary indiscretions, garbage intoxication
- Ingestion of foreign material esp. in young, e.g. poisonous plants, hairballs (bezoars)
- Drug therapy e.g. corticosteroids, digoxin, erythromycin, chemotherapy
- Acute systemic disease (uraemia, liver disease, sepsis)

Acute enteritis is again common in first-opinion practice. It is characterised by acute onset of profuse diarrhoea and is often associated with acute vomiting (i.e. acute gastroenteritis). Many causes have been described again the process is usually self-limiting and often remains obscure. Aetiologies include:

- Dietary indiscretions, garbage intoxication
- Enteric infections e.g. bacterial, viral, protozoal, parasitic
- Acute colitis

Acute colitis refers to acute onset of frequent, small volume diarrhoea. Associated signs include excessive straining (tenesmus), and mucoid faeces often containing fresh blood (haematochezia). The incidence of acute colitis is fairly common in dog, but rare in cats (except Tritrichomonas foetus). However, Possible aetiologies include:

- Dietary indiscretions, garbage ingestion
- Whipworm (Trichuris vulpis) infection
- Protozoal infections e.g. Giardia, Cryptosporidia
- Tritrichomonas foetus, an emerging cause of acute (progressing to chronic), severe large bowel diarrhoea in cats (especially young, pedigree, colony cats).

Diagnosis

History and physical examination are often all that is required to make a diagnosis of non-fulminating acute gastroenteritis.

**Clinical signs**

Signs of vomiting and diarrhoea frequently occur together, although in some instances there is sequential progression e.g. vomiting ± abdominal pain ± profuse diarrhoea ± blood ± mucoid diarrhoea ± tenesmus. However, the disease process may only affect one level of the GI tract, with just the relevant signs present (e.g. gastritis with vomiting alone; ‘enteritis’ with small intestinal diarrhoea etc).

**Classification of acute gastroenteritis**

In general acute gastroenteritis can be (rather arbitrarily) categorised as follows:

- **Non-fatal, self-limiting acute gastroenteritis** e.g.
  - Uncomplicated parasitism
  - Dietary indiscretion, dietary sensitivity, food poisoning or scavenging
- **Secondary to extra-intestinal / systemic disease** e.g.
  - Systemic infections e.g. canine distemper, leptospirosis
  - Metabolic disorders e.g. uraemia, hypoadrenocorticism
- **Severe, potentially, life-threatening acute gastroenteritis** e.g.
  - Enteric infections such as enteroviruses (i.e. canine enteric coronavirus), salmonellosis
  - Haemorrhagic gastroenteritis (HGE)
  - Intestinal obstruction; vomiting is usually the major sign, but diarrhoea may occur. Examples include foreign body, intussusception, volvulus.

**Decision making**

The first-opinion practitioner is often presented with a dog or cat with signs of acute gastroenteritis. Within the space of this initial (short!) consultation, a number of decisions must be made:

- **Is intensive emergency treatment needed?**
  Emergency treatment may be required if there is evidence of:
  - Dehydration
  - Electrolyte and/or acid/base disturbances
  - Shock
- **Is there an underlying non-enteric cause of gastroenteritis?**
- **Is surgical management needed?**
- **Is hospitalisation needed?**
- **Is an infectious cause likely and isolation needed?**
- **Are the clinical signs non-specific, and therefore will symptomatic treatment be sufficient?**
(toxins, plants, foreign bodies) or infectious disease. In addition, the nature of the signs should be clarified e.g. onset and severity, content of vomitus, stool characteristics, and presence of blood (haematemesis, melaena, haematochezia).

**Physical examination.** Relevant findings on physical examination include general body condition, hydration status (may also require PCV/TP), oral examination - mucous membranes etc, rectal examination, and abdominal palpation.

Based on this preliminary information, the decision-making process can commence. In some cases, a ‘minimum data-base’ of diagnostic tests will be required e.g.

- **Haematology, serum biochemistry and urinalysis**
- **Faecal parasitology**
- **Faecal bacteriology** (indicated if the patient is febrile, has an inflammatory leucogram, gastrointestinal bleeding (melaena, haematochezia), or is young)
- **Faecal virology.** This is indicated in some circumstances and includes:
  - **Faecal examination,** e.g. ELISA test for viral antigen (e.g. parvo) or electron microscopy (e.g. rotavirus, coronavirus)
  - **Serology.** This may be necessary in some circumstances, although paired samples required to demonstrate recent infection.
- **Imaging.** Plain radiographs are helpful to rule out gastrointestinal obstruction and other surgical diseases. Abdominal ultrasonography, in the hands of an experienced operator is also useful for this purpose.
- **Response to empirical treatment.** Diagnosis can be confirmed by response to any of the following therapies:
  - Dietary restriction
  - Discontinuation of drugs or toxins
  - Avoidance of plants or other environmental agents
  - Anti-emetics
  - Anti-diarrhoeals
  - Parasiticides

With some cases animals present as emergencies, with acute or peracute clinical signs. In these circumstances diagnostics should be performed in parallel to preliminary treatment to stabilise the patient’s condition. It is advisable to run an emergency database to allow decisions to be made about preliminary treatment (especially fluid therapy). A typical emergency database would include PCV, total protein estimate (refractometer or Vet-Test), blood smear examination (microscopy), blood glucose (glucometer), blood urea (Vet-Test or similar), urinalysis (dipstick), ± electrolytes (iSTAT or similar) ± blood gas analysis (iSTAT).

**Symptomatic treatment**

If a primary cause can be identified this should be treated. Many animals with mild, acute gastroenteritis improve spontaneously in 2-3 days, suggesting that treatment is not always necessary. Prognosis for complete recovery is usually good. However, the animal should be reassessed if there is persistence of clinical signs for ≥48 hours, despite symptomatic treatment, or before this time if the clinical signs are clearly deteriorating.

**Dietary manipulation**

“**Feeding through diarrhoea**”. An alternative approach is to continue to feed the animal despite the clinical signs. Such an approach has been adopted for diarrhoea in (human) infants, and may speed recovery. However, it is less practicable veterinary species if/when vomiting is persistent or diarrhoea is profuse (cosmetic problem).

**Anti-inflammatory medication**

Many practitioners treat acute gastroenteritis with glucocorticoids or NSAIDs. However, corticosteroids and NSAIDs are contra-indicated. There is little indication for either approach!
Anti-emetic medication
Anti-emetic drugs (e.g. maropitant, metoclopramide) are indicated, provided that pyloric/intestinal obstruction has been ruled out. Maropitant (Cerenia) is the licensed drug and, therefore, the primary choice for vomiting dogs. Anti-cholinergic anti-emetics, are rarely recommended, since they cause hypotonia and outflow obstruction. Examples of such drugs include atropine, methyscolopamine.

Gastric mucosal protectants and antacids
This group of drugs are frequently given for patients that are acutely vomiting, although it is important to stress that they are NOT anti-emetics. Recognised anti-emetic drugs should be used to control vomiting, and the use of gastric protectants reserved for those with evidence of haematemesis or gastric ulceration.

Antacids (e.g. aluminium or magnesium hydroxide; Aludrox, Gaviscon etc) are not very useful as they must be given frequently to be effective. Gastric mucosal protectants are only indicated if ulceration is present. Unless there is documented evidence (e.g. on endoscopy) of gastric ulceration, the proton-pump inhibitors cannot be justified in acute gastritis. Instead, it is preferable to use H2-receptor antagonists e.g. cimetidine, ranitidine, and famotidine. Cimetidine (Zitac) has recently been licensed for use in dogs and is technically, therefore, the drug of choice (based upon the cascade). Sucralfate can also be used, in combination, for such cases.

Anti-diarrhoeal agents
This term refers to a range of agents, which are designed to provide symptomatic control of clinical signs. Many different commercial products are available, often containing combinations of agents. Antibacterial products are sometimes combined, but the use of preparations containing anti-diarrhoeals alone is preferable. Other combination products include probiotics (see below - preferable to antibacterials), and occasional dietary fibre.

Types of anti-diarrhoeal preparation include:

- **Absorbents / protectants.** As their name suggests these preparations ‘protect the mucosa’, as well as binding toxins and excess water:
  - Kaolin, pectin, probiotic, prebiotic (Pro-Kolin)
  - Kaolin-pectin (Kaogel)
  - Montmorillonite, glucoamine, probiotic prebiotic (Promax)

- **Motility modifiers** e.g. opioids are used initially to control severe diarrhoea, although this may be contraindicated, if an infectious cause is suspected, since the purgative effect of diarrhoea may be desirable. They act by (i) increasing segmentation, (ii) decreasing peristalsis, and (iii) an anti-secretory effect. Examples include:
  - Diphenoxylate (Lomotil)
  - Loperamide (Imodium)

- **Anticholinergics/antispasmodics** are also motility modifiers, but are less effective since they decrease segmental contractions. This reduces resistance to flow, and may exacerbate diarrhoea. Examples include atropine and hyoscine. Antispasmodics (e.g. Buscopan) may be indicated if there if excessive straining.

Antibacterials
Antibiotics are the drugs MOST COMMONLY USED, to treat acute gastroenteritis, by veterinary surgeons in the UK. However, there is often no rational indication. Further, antibacterials are contraindicated for Salmonella infection (unless systemically ill), since this may cause a carrier state to develop. In addition, most cases of Campylobacter in dogs are actually C. upsaliensis (rather than C. jejuni or C. coli) and the pathogenetic potential of this species has not been established. Care should also be exercised with products containing neomycin. Although these would normal act locally in the intestine, absorption may occur if GI mucosa ulcerated, leading to ototoxicity. General overuse of antibacterials encourages the development of antibiotic resistance, and can have deleterious effects on the normal gastrointestinal flora (antibiotic-associated colitis etc).

Given the many contraindications, antibacterials should ONLY BE USED IN THE FOLLOWING CIRCUMSTANCES:

- If a specific bacterial infection is documented.
- However, care should still be exercised, since many infections are incidental!
- When severe mucosal damage (i.e. evidence of ulceration & haemorrhage – blood in the stool) is evident. This may predispose to bacterial invasion and systemic sepsis.
- If the patient is pyrexic
Companion Animal Programme

Treatment of gastric foreign material (foreign bodies, toxins etc)

Foreign bodies
When a foreign body is recognised, there is always a tendency to rush in and remove it. However, given that some gastric foreign bodies may be incidental, other causes of vomiting should first be ruled out before exploratory surgery! If bones reach the stomach, they will often be digested in this site without problem. Induction of vomiting for foreign bodies/toxins is only recommended if the object is smooth or is a non-corrosive poison recently ingested. Suitable emetics include apomorphine, xylazine, ipecacuanha and washing soda crystals. ‘Natural passage’ of a foreign body is recommended for small objects, if no signs of gastric disease. To facilitate this, a high fibre diet ± gastrointestinal lubricants should be administered, and the patient should be observed closely. Failure to pass in 48 hours necessitates endoscopic or surgical removal.

References:
References are available upon request from the author.

EXOCRINE PANCREATIC DISEASE IN CATS
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Acute pancreatitis in cats
Acute pancreatitis has been recognised as a common disease in dogs, with the highest incidence in obese, middle-aged, sedentary female dogs. Although acute pancreatitis was always thought to be much rarer in cats, it is now being recognised more often given greater awareness of the condition and improved diagnostic capabilities. Despite this, it remains a problematic disorder to manage.

Aetiology
Cases of pancreatitis are usually ‘idiopathic’ but various predisposing factors have been suggested in companion animals e.g. fat, (feeding a high-fat diet, obesity, and hyperlipidaemia), inflammation of other abdominal organs (e.g. the ‘triaditis’ syndrome), pancreatic ischaemia (e.g. hypovolaemia), trauma (e.g. the high-rise syndrome), drugs, (e.g. L-asparaginase, azathioprine, organophos-
Pathogenesis
Pancreatitis can have variable severity, ranging from mild disease (e.g. mild interstitial pancreatitis) to severe (e.g. haemorrhagic pancreatitis, acute pancreatic necrosis). It can be associated with both local effects (e.g. localised peritonitis and fat necrosis) and systemic effects (e.g. renal failure, cardiac arrhythmias, pleural effusion, shock, DIC, and death).

Clinical signs
As with the severity of pathology, clinical signs range from mild to fatal, and include depression, anorexia, vomiting, diarrhoea (± blood), icterus (jaundice) and abdominal pain. These signs are most consistent in dogs but, in cats, these signs are observed less consistently. In fact, the most frequently observed signs in feline pancreatitis cases are depression and anorexia! Thus, the clinician should maintain a high index of suspicion for this disease. Abdominal palpation may also confirm abdominal pain, a cranial abdominal mass lesion, and/or ascites (occasionally present). Pyrexia may also be noted.

Diagnosis
Haematology and serum biochemistry
Abnormalities may include leucocytosis (neutrophilia and left shift), hyperglycaemia, hyperlipaemia, hypocalcaemia (due to saponification of fat, glucagon release, and increased calcitonin) and increased liver enzymes ± icterus if bile duct obstructed. Nonetheless, the findings in pancreatitis are not pathognomonic and, changes tend to be far less consistent in cats.

Specific laboratory tests
Most diagnostic tests for pancreatitis are pancreas-specific proteins e.g. enzymes.

Totalling anylase and total lipase are the most widely available tests are completely unreliable in cats. Trypsin-like immunoreactivity (TLI) is pancreas-specific and is an excellent test for exocrine pancreatic insufficiency (EPI). It can also be elevated in the early stages of pancreatitis but, given its immensely short half-life, it is of limited use for pancreatitis. There are problems both with false negatives and false positives in cats.

Pancreatic lipase immunoreactivity (PLI) is a test that has recently become available for both dogs and cats. Early experience with this test suggests that it is the most sensitive and specific laboratory test available but further work will be required to determine its true value.

Diagnostic imaging
Radiography is of limited use in the diagnosis of pancreatitis, and ultrasonography is preferred. Typical changes in clued pancreatic enlargement, changes in echodensity and alterations in associated abdominal fat (hyperechoic changes). Ultrasound may also facilitate the collection of diagnostic specimens (e.g. fine needle aspiration cytology).

Pancreatic biopsy
Histopathological assessment of biopsy material (procured at coeliotomy or laparoscopy) is the best technique for the diagnosis of acute pancreatitis. However, it is invasive, and may be dangerous to perform in animals that are severely ill.

Treatment
The aims of treatment are to provide pancreatic rest and to enable spontaneous resolution of the disease. Nutritional support. Complete food withholding was the traditional approach to treatment of pancreatitis, but is no longer recommended since, in other species, early enteral nutrition is known to improve outcome. Therefore, many clinicians now attempt to recommence feeding as soon as vomiting subsides. Techniques depend upon the clinical case but may include tube-feeding (nasoesophageal, oesophagostomy, jejunostomy etc) and parenteral nutrition. Enteral nutrition is known to improve outcome. Therefore, no longer recommended since, in other species, early traditional approach to treatment of pancreatitis, but is critical. The choice of fluid depends upon the individual case. Analgesia is an essential part of treatment, and opioids/opiates are usually recommended e.g. buprenorphine. Since the majority of cases are sterile (if cultured) so use of antibacterials is controversial. However, there is some evidence that systemic translocation of bacteria can occur. Therefore, antibiotics are often used prophylactically, and intravenous bactericidal drugs are recommended e.g. amoxicillin-clavulanate. Further, given that Gram-negative bacteria commonly translocate,
fluoroquinolones may also be of use. Antibiotics are also indicated if a pancreatic abscess develops.

Corticosteroids
Traditionally, the use of corticosteroids was thought to be controversial because of concerns about inciting pancreatitis. However, given that many feline cases may be associated with the triaditis syndrome, and there may be an underlying immune-mediated aetiology, corticosteroids may have more use. The author often commences therapy without these drugs, but considers adding them to the treatment plan if the case is not responding.

Surgery
The use of surgery is also controversial. In the majority of cases, surgery is contraindicated, since hypotension may arise during anaesthesia. However, diagnosis of pancreatitis may only be possible through coeliotomy/laparoscopy. Surgery also provides the opportunity to debride necrotic tissue and lavage the abdomen. Surgery is also indicated if there is an abscess or cyst is present.

Prognosis
Prognosis for a full recovery is guarded; many acute cases die unless they are treated aggressively, but recurrence is common. Apparent treatment failure may also result from incorrect diagnosis!!! Possible sequelae to acute pancreatitis include chronic recurrent pancreatitis, exocrine pancreatic insufficiency (EPI) and diabetes mellitus (DM).

Exocrine pancreatic insufficiency in cats
Aetiopathogenesis
EPI is far less common in cats than in dogs, but occasional cases are seen. The most common aetiology in feline EPI is thought to be prior pancreatitis, which often may have been subclinical. EPI develops after repeated bouts of pancreatic inflammation, which eventually destroys enough (>90%) functional reserve, through atrophy and fibrosis. Lack of pancreatic enzymes then causes malabsorption and the resultant clinical signs.

Clinical signs
Weight loss and poor body are the most common signs of EPI in cats. Loose stools (but rarely watery diarrhoea) and polyphagia may be present. Poor coat condition is also seen and, sometimes, there may occasionally be greasy soiling of the perineal region.

Diagnosis
It is not possible to diagnose EPI on clinical signs alone. As with dogs, the recommended test for diagnosis is measurement of serum trypsin-like immunoreactivity (TLI), and concentrations are typically very low or undetectable. Like with dogs, the test is highly sensitive and specific for the diagnosis. Low serum cobalamin concentrations can occur in many gastrointestinal diseases, including EPI. Although such alterations are not pathognomonic, they may provide supportive evidence and highlight the need for therapeutic supplementation via the parenteral route.

Treatment and prognosis
The implications of treatment should be discussed with the owner prior to commencement of therapy. Treatment is lifelong and can prove expensive. Oral pancreatic extract is the mainstay of clinical management and both uncoated and enteric-coated products can be used. Clinical evidence is lacking as to whether one type is superior to the other. Therapy is usually started at 1 teaspoon per 10kg bodyweight, but the dose can often be tapered after the case has responded to therapy. There is no evidence that pre-mixing enzyme and food improves efficacy. An alternative to dried extract is to use fresh frozen pancreas. However, its availability is now limited. is excellent but very limited availability (relies on having an amenable local abattoir!).

Dietary therapy is also important and should be highly digestible, fed in small frequent meals. There is no need to use a fat restrict the diet.

Parenteral cobalamin supplementation is also required in many cases since hypocobalaminaemia commonly accompanies this disease and is a negative prognostic factor. Occasional cases of EPI, secondary to chronic pancreatitis, may have concurrent DM, and insulin therapy may be necessary in these individuals.

The prognosis of EPI is guarded to good. EPI is usually a lifelong condition and lifelong therapy is required. However, anecdotally, it may be possible to reduce the amount of enzyme supplementation with time. If cases respond well to initial therapy, long-term survival is likely.

References:
References are available upon request from the author.
**Effective Management of Vomiting and Nausea in Companion Animals**

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**Introduction**

Vomiting is defined as retrograde ejection of food or fluid from stomach or small intestine (duodenum). It is a complex reflex act in dogs and cats, requiring the co-ordination of the gastrointestinal and musculoskeletal systems in conjunction with the central, peripheral and autonomic nervous systems. Vomiting is triggered, by various stimuli, which activate the emetic centre within the reticular formation of the medulla oblongata. Some stimuli activate the so-called ‘humoral pathway’ whereby blood-borne toxins and (which the chemoreceptor trigger zone; CRTZ) located within the area postrema. Activation is induced by various emetogenic substances (e.g. uraemic toxins, apomorphine, cardiac glycosides, and cytotoxic agents). Alternatively, receptors in the abdominal viscera (GI tract, pancreas, liver, urogenital tract, and peritoneum) can activate either vagal or sympathetic neurons leading to vomiting via the so-called ‘neural pathway’. These receptors can be activated by inflammation, irritation, distension and hypertonicity. Finally, motion sickness can cause vomiting, due to impulses originating in the vestibular centre (inner ear), which then travel through the CRTZ to the vomiting centre.

**Approach to management of the vomiting patient**

The clinician has two main goals when presented with a vomiting animal: first, to establish the cause of vomiting, and second to stop the vomiting in a safe and effective manner. Early in the course of events, the clinician should determine whether or not the animal has a self-limiting or possible life-threatening problem. The cause is rarely apparent in animals with an acute, self-limiting problem; further, these cases rarely require detailed investigations, and symptomatic therapy is sufficient. In contrast, life-threatening acute vomiting requires both diagnostic evaluation, as well as specific and intense supportive therapy. Finally, cats and dogs with chronic vomiting always require detailed investigations to find the cause of the problem. In such circumstances, an organised approach is required, but anti-emetic therapy can be provided to control the clinical signs whilst the cause is established.

Initially, a history is taken, which should include information on diet, recent medication, vaccination status, and a complete description of the clinical signs shown. A description of the ‘vomiting’ act is necessary to confirm that the animal truly is vomiting rather than regurgitating. Information should be collected on the frequency and timing of vomiting (i.e. relationship to feeding), as well as on the nature of the vomitus (e.g. presence of food, bile, blood, coffee grounds). Physical examination is an important part of the minimum database, and particular attention should be paid to careful abdominal palpation. Oral cavity examination should include assessment of the base of the tongue for linear foreign bodies, whilst rectal examination is also recommended. The latter will enable gross examination of the stool (e.g. for identifying melaena if present).

The minimum database of signalment, history and physical examination enables an initial list differential diagnoses to be established, and allows the clinician to plan the next stages of the diagnostic investigations. General laboratory investigations (e.g. haematological examination, clinical chemistry, urinalysis, faecal bacteriology, and faecal parasitology) are usually considered at this stage. Additional laboratory tests that may be required in certain circumstances include faecal analyses, and ACTH stimulation test and bile acid stimulation. Further, specific alimentary tests are recommended in all cases (trypsin-like immuno-reactivity, pancreatic lipase, cobalamin, folate). Stage two of the work-up usually involves diagnostic imaging and the author prefers a combination of radiography (survey thoracic and abdominal views) and abdominal ultrasonography. The value of abdominal ultrasonography directly relates to the quality of the equipment and the expertise of the ultrasonographer. It has the potential to identify disease in other (non-alimentary) abdominal organs, as well as providing information on gastrointestinal lesions. If appropriate, targeted fine needle aspiration can also be performed if abnormalities are identified.

Assuming that the cause is not identified, the final stage of investigation usually involves collection of gastrointestinal tissue samples either by endoscopy or at exploratory coeliotomy. Which technique is used depends upon equipment available, expertise, prior imaging findings (focal lesions or lesions in other abdominal organs are better investigated with coeliotomy), and patient factors. A final diagnostic option is the therapeutic trial, and options include anti-parasitic medications, exclusion diet trial, antibacterial therapy and/or triple-combination therapy to eliminate gastric...
spiral organisms. These often provide an adjunct to the main work-up and help to confirm the exact nature and aetiology of the disease.

Where the vomiting is acute and severe, effective treatment should be provided quickly to control signs, prevent further deterioration in condition and to enhance recovery. In cases of chronic vomiting, it is usually most important to reach a diagnosis as quickly as possible, since this may provide more specific therapeutic options. Some chronic vomiting cases may not require any therapy if signs are only occasional and the animal is otherwise systemically well; for others however, treatment may be necessary to improve patient well-being, whilst the diagnosis is pursued.

Ancillary therapy

Gastric protectants and acid-blocking drugs can be useful where gastric ulceration is present or suspected. However, given that they have no anti-emetic effect, they should not be used as an alternative to using an a.

Starvation or feeding? For many simple, acute gastrointestinal disturbances, the current practice of food withdrawal (for 24 hours) is valid. Assuming vomiting has subsided, a bland diet can be offered, in small frequent meals, for the next 24-48 hours, before gradually reintroducing the normal diet. There is some evidence in dogs (but not yet cats) that early enteral nutrition improves outcome in severe acute gastrointestinal disturbances (e.g. pancreatitis, parvovirus).

Anti-emetic therapy – therapeutic options

Anti-nausea and improve patient wellbeing. However, it is essential first to ensure that there are no risks or contraindications. A number of anti-emetics exist, each designed to block different receptors, within the vomiting pathway.

Maropitant is a neurokinin-1 (NK-1) receptor antagonist, which has recently been licensed for use in dogs; it can be given both orally and by subcutaneous injection. It is highly effective against both peripherally- and centrally-induced vomiting and, in dogs, has been shown to perform favourably against many other anti-emetic compounds (e.g. metoclopramide, ondansetron, anti-histamines). It has a wide margin of safety and few side effects have been reported. The drug is licensed in dogs but not, yet, in cats. However, studies have been conducted, suggesting that it is effective in this species.

Metoclopramide is indicated for a number of disorders, which involve central or peripheral activation of the vomiting centre. The drug also has a prokinetic effect which can be useful where there is delayed gastric emptying, although this effect means it should be avoided if there is any possibility of pyloric or upper small intestinal obstruction. Finally, since the drug increases contraction of the lower oesophageal sphincter, it may be useful in disorders involving gastric reflux (e.g. hiatal hernia, oesophagitis). Side effects include mental changes such as hyperactivity, disorientation or frenzied behaviour; finally, the injectable form stings when given subcutaneously. Cats are more prone to the side effects of metoclopramide than dogs.

Phenothiazines can be highly effective for central or peripheral causes of vomiting, but are not licensed for this use in veterinary species and can have a number of adverse effects. These include hypotension, which can have adverse affects where the patient is dehydrated. Other adverse effects include sedation, rigidity, weakness or restlessness at high doses. Finally, these drugs are contraindicated in animals with a known seizure history.

Domperidone has a similar action to metoclopramide in that it primarily blocks D2 and secondarily blocks 5HT3 receptors. However, it is not thought to have prokinetic effects in dogs, and this may reduce its effectiveness against peripheral causes of vomiting. Less is known about this drug’s action in cats.

5HT3 antagonists are usually used to control drug-induced emesis such as chemotherapy. The main drug of this group is ondansetron. It has a high level of efficacy and has few side effects, but can mask the signs of ileus or gastrointestinal distension and is often prohibitively expensive.

Anticholinergics have potential indications for motion sickness as they block M1 and M2 receptors in the vestibular apparatus. However, adverse effects include delayed gastric emptying, which may prolong the retention of the agent that caused it, and ileus. It should not be used where gastrointestinal obstruction is suspected. The main agents in this group are atropine, propantheline and butylscopolamine but the author rarely, if ever, uses these drugs as anti-emetics.

References: References are available on request.
MANAGEMENT OF OBESITY IN DOGS AND CATS – MAXIMISING SUCCESS
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Introduction
Obesity is defined as an accumulation of excessive amounts of adipose tissue in the body, and predisposes to a variety of diseases including diabetes mellitus, and osteoarthritis. In most animals, obesity is the result of a simple imbalance between energy intake and energy expenditure. Therefore, at its simplest, successful management of obesity usually involves reversing this imbalance by reducing energy intake and/or increasing energy expenditure. This lecture will discuss current thoughts on both conventional and novel treatments for obesity in dogs and cats, and stimulate discussion on factors required for long-term success.

Causes of obesity
Obesity can arise secondary to a number of diseases including endocrinopathies (e.g. hypothyroidism and hyperadrenocorticism in dogs), drugs (e.g. polyphagia caused by glucocorticoids and anti-convulsant drugs), and rare genetic disorders (in humans), although most cases are primary i.e. the result of an imbalance in the ‘energy balance equation’. Thus, either excessive dietary intake or inadequate energy utilization can lead to a state of positive energy balance, leading to increased white adipose tissue deposition. Numerous factors may influence the relative ease with which weight is gained, and these include genetics, age, neuter status, concurrent diseases, amount of physical activity, and energy content of the diet. Many of these factors need to be considered when devising a weight program; for instance, the type and nature of physical activity will be greatly affected by the presence of concurrent disease.

Overview of treatment of obesity in dogs and cats
In humans, current therapeutic options for obesity include dietary management, exercise, psychological and behavioural modification, drug therapy, and surgery. Temporary weight loss, by liposuction, does not have an equivalent effect and does not affect metabolic risk. Liposuction removes only subcutaneous fat, which carries little metabolic risk, and energy intake is unaffected; therefore, body weight will rise again to achieve energy balance.

Bariatric surgery is the most successful method of weight loss in humans, and average weight loss is ~23%. Various approaches are described including gastric banding and the roux-en-y procedure. This success comes at a cost, since the complication rate is high (including perioperative mortality, short-term and long-term consequences). For companion animals, it is not considered ethically justifiable to manage obesity through surgical means. However, pharmaceuticals are available for treatment of obesity in dogs. For dirlotapide, considerable evidence exists, from randomized controlled trials, as to efficacy for weight loss. Nonetheless, for any therapy (pharmaceutical, dietary etc) to have long-term success, it is ESSENTIAL to modify owner and animal behaviour. Unless steps are taken to change feeding habits and exercise patterns, weight regain will occur. This rebound effect is a well-known phenomenon of any weight loss program. Therefore, to achieve long-term success weight loss is only the start rather than the end of therapy. Conventional options for weight management include dietary therapy and behavioural modifications; such strategies are likely to remain for dogs and, given that no pharmaceutical agents have yet been approved for cats, this approach will remain the mainstay of therapy in this species.

Dietary management
The weight loss protocol should always be tailored for the individual patient. Although complete starvation leads to rapid (~7%/week) weight loss, it has the disadvantages of causing excessive protein (and thus lean body mass) loss and requiring hospitalization to monitor. Further, adverse effects on body system function have been reported (e.g. cardiovascular function is compromised). Therefore, it is preferable to use purpose-formulated diets, and most formulated rations are restricted in fat and calories, whilst being supplemented in protein and micronutrients. Protein supplementation is important since, although weight loss is not more rapid, the amount of lean tissue lost is minimized. Micronutrients are also supplemented, relative to energy content, in order to reduce the chance of deficiency states arising during weight loss. Other diet components that have been employed in weight management include L-carnitine supplementation (to maintain lean mass), and altering macronutrient content (most notably fibre and protein) to maximize satiety (see below).
A major hurdle to conventional weight loss programs is the fact that energy restriction causes hunger, leading to increased begging and scavenging activity. This puts increased strain on the owner-animal bond, causing owner non-compliance or complete withdrawal from the program. Therefore, developing strategies to improve satiety would greatly assist in case management. The results of many human studies have shown that absorption of macronutrients is lower following consumption of high-protein foods than after consumption of foods with a high carbohydrate or fat content. Supplemeting dietary protein appears to improve satiety in dogs but not cats.

Under certain conditions in humans, dietary fibre has been shown to exert a satiety effect although some studies have failed to detect significant reduction in appetite. There are similar discrepancies in canine studies, although most recent reports do suggest improved satiety. The most recent work has demonstrated that supplementing diets with both protein and fibre has the greatest satiating effect in dogs, and such diets are known to improve outcome of weight loss regimes. However, given that protein content is a key determinant of voluntary food intake in cats, the best effect on satiety occurs with fibre supplementation, whilst only modestly increasing protein content.

**Lifestyle management**

Increasing physical activity is a useful adjunct to dietary therapy during weight management; studies in humans suggest that increasing activity promotes fat loss, whilst preserving lean tissue during weight loss. The exact program must be tailored to the individual, and take account of any concurrent medical concerns. Suitable exercise strategies in dogs include lead walking, swimming, hydrotherapy, and treadmills. Treadmill sessions are known to improve outcome in weight loss programs. Cats can be encouraged to exercise by increasing play activity, using cat toys (e.g. fishing rod toys), motorised units and feeding toys. Cats can also be encouraged to ‘work’ for their food by moving the food bowl between rooms prior to feeding, or by the use of feeding toys. Activity monitors (accelerometers, pedometers) have recently been validated for dogs, and may help to provide a more objective assessment of activity during weight loss programs in the future.

**Pharmaceutical agents**

In humans, diet and exercise are the main methods of achieving weight loss, but some patients require drugs to assist with and maintain the loss. The only drug currently licensed for weight loss in human is orlistat an intestinal lipase inhibitor. Other drugs previously licensed (i.e. sibutramine and rimonabant) have been withdrawn due to concerns over side effects. All drugs in this category typically have only a modest beneficial effect; they increase weight loss ~4-6 kg beyond what can be achieved by diet alone, maintain weight loss by ~2-15 kg below baseline, and they improve most cardiovascular risks in direct relation to weight loss.

Two drugs are currently approved to assist in the management of obesity in dogs. *These drugs are neither licensed nor safe in cats*. Both are microsomal triglyceride transfer protein inhibitors that block the assembly and release of lipoprotein particles into the bloodstream.

Mitratapide (licensed in Europe only) is a drug recently approved to aid in weight loss. It has both local (gastrointestinal) and systemic effects and is designed for short-term use in conjunction with dietary management and behavioural modification.

Dirlotapide (licensed in North America and Europe) can be used continuously as sole therapy for obesity for a maximum period of 12 months. It partly acts by preventing lipid absorption, but also by reducing appetite. The reason for the appetite effect is still debated, but only occurs when the drug is given orally, suggesting that its origin is a local one in the gastrointestinal tract. It is possible that this local effect stimulates the release of a gastrointestinal hormone (i.e. peptide YY), which then has a central effect on appetite.

This effect on satiety targets one of the major reasons for poor compliance with a weight loss regime, namely the development of negative behaviours such as begging and scavenging. It is also not necessary to change the current diet, provided that it is complete and balanced. In fact, the drug does not work as well when administered with a low fat diet and, from an empirical point of view, using it concurrently with a conventional weight loss diet does not appear to improve efficacy. The most common side effects are gastrointestinal, i.e. vomiting and diarrhoea, which can occur in up to 20% of dogs using the drug. Further, many clinicians have empirically reported variations in efficacy amongst individual dogs. Whilst on the drug, weight loss is highly successful, but a predictable rebound occurs when the drug is discontinued. It is necessary to monitor weight closely and to implement
other strategies (i.e. dietary and lifestyle changes) to minimize the likelihood of weight regain occurring.

**Monitoring weight loss**
In addition to the above strategies, it is essential that the whole weight reduction regime be closely supervised. This is labour-intensive, requires some degree of expertise and training in owner counselling, and often requires a dedicated member of staff. In the author’s opinion, correct monitoring is the single most important component to the weight loss strategy. A recent study has demonstrated that weight loss is more successful if an organised strategy is followed with regular weigh-in sessions. It is essential to continue to monitor body weight, after ideal weight has been achieved, to ensure that weight that was lost is not regained; as with humans, a rebound effect has been demonstrated after weight loss in dogs. This has been seen in ~50% of dogs that successfully lose weight.

**Review of conventional weight loss programs**
Conventional weight loss regimes, involving dietary caloric energy restriction, are highly successful in obese colony dogs. Rates of weight loss of 1.3-2.6%/week have been achieved with caloric allocations of 50-87%. However, weight loss in client own dogs is slower (average 0.85% body weight/week), and requires a greater degree of energy restriction i.e. mean 52% of mean energy requirement at target weight. Mean energy intake during a weight loss regime is 32 Kcal/kg TW has been reported in pet cats with naturally-occurring obesity. With this degree of restriction, the average rate of weight loss is 0.8% body weight/week.

The most important factors that influence response include breed, gender and neuter status. Previous work with colony dogs has demonstrated breed differences in the level of energy restriction required to achieve the same rate of weight loss, with Labrador retrievers requiring a greater level of restriction than beagles. Age, sex, neuter status and activity level have also been shown to be of importance. The main factor that has been shown to affect the rate of weight loss is the level of caloric energy restriction. Nevertheless, whilst the level of protein does not appear to affect rate of weight loss, the proportion of lean tissue loss is lower on a high-protein diet, compared with a diet of moderate protein content. As mentioned above, the use of formulated weight loss diets that are supplement in both protein and fibre improve outcomes of weight loss in dogs.

**Preventing weight regain**
In humans, long-term success of weight management strategies is disappointing, with some studies suggesting that some participants on diet-based weight loss strategies regaining more weight than they had originally lost. Whilst the reasons for this ‘regain’ are still unclear, the most likely explanation is that, when obese humans are returned to a lean state, their resting metabolic rate is lower. Experimental studies in dogs have demonstrated a similar tendency for weight regain, with maintenance energy requirements decreasing significantly after weight loss.

A recent study has examined long-term follow-up in obese pet dogs that had successfully reached target weight. 42% of dogs maintained weight, 9% further weight, and 48% regained weight. Dogs fed a purpose-formulated weight management diet, during the weight maintenance phase, regained less weight than those switched onto a standard maintenance diet.

**Summary**
Successful weight loss in dogs and cats requires dedication and commitment. Conventional weight loss strategies involving diet and exercise can be highly successful in both dogs and cats, whilst pharmaceutical agents provide another means to achieve target weight. Successful weight loss involves not only achieving an ideal body weight, but maintaining it subsequently. In the author’s opinion, preventing the known and predictable rebound effect seen with any weight loss strategy, requires changes in both owner and pet behaviour, and is the key factor in true success.

References:
References are available upon request from the author.