HAEMATEMESIS AND MELAENA. WHEN BLOOD IS ADDING SOME EXTRA COLOUR

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Introduction

Haematemesis is defined as vomiting blood, and represents a small, but significant, proportion of cases that present with vomiting. Melaena, defined as darkened faeces due to the presence of blood pigments, often accompanies haematemesis. The differential diagnoses for both signs are similar (Table 1) although, if haematemesis is present, the site of haemorrhage will be cranial to the proximal duodenum. Haematemesis is a clinical sign rather than a diagnosis, and can be caused by a range of systemic or primary gastrointestinal diseases. Severity can vary from mild (often simply associated with persistent vomiting and of no concern) to severe life-threatening haemorrhage. A logical approach to investigation and diagnosis is required so that appropriate diagnosis can quickly be made and treatment be provided. This presentation will review the investigation, diagnosis and treatment of haematemesis in dogs.

Where do I start?

Cases of haematemesis can range markedly in clinical presentation; from acute to chronic; from mild and inconsequential to severe and life threatening. Added to this, haematemesis is a sign that often induces panic with owners; ‘a little bit of blood goes a long way’. Therefore, in the early stages of investigation, it is important to get your priorities right; you should quickly obtain an accurate assessment of the severity of the problem by integrating historical information and preliminary physical examination findings. The urgency of subsequent investigations depends upon the individual case. You should also take steps to reassure the client at an early stage, because this will improve the amount, detail and accuracy of information provided.

The signalment, history and physical examination form the basis of the preliminary data from which an initial problem list and set of differential diagnoses can be established. Although, in most cases, further diagnostic investigations are required never underestimate the importance of this background information. The priority given to various differential diagnoses depends upon the signalment e.g. neoplastic disorders are more likely in middle-aged to older animals of certain breeds
(e.g. rough collies, Staffordshire bull terriers); portovascular anomalies (causing secondary ulceration and haematemesis) are more likely in younger patients. Historical information will suggest the existence of haematemesis, but the problem can be verified by asking the owners to bring in a representative samples. The severity, frequency and progression of clinical signs can also be determined. Other important information includes data on previous problems (which may be related) and current drug therapy. The owner should also be asked about home medications (e.g. aspirin, ibuprofen), since such information may not be volunteered until asked. Possible exposure to toxins should also be noted. Clinical signs pertaining to other body systems must be accurately recorded given the possibility of associated disease.

A thorough physical examination is also essential; little direct information is usually obtained on the gastrointestinal system, with the exception of abdominal palpation, which may reveal mass lesions or abdominal pain. More information can be gained about systemic disorders causing haematemesis. Oral cavity examination confirms the existence and severity of anaemia (mucous membrane colour, CRT), hypovolaemia (CRT, pale mucous membranes, tacky etc), and jaundice (if hepatopathy is present). The presence and site of oral cavity or nasal bleeding may also be documented (a cause of indirect haematemesis if swallowed). Physical examination will also reveal whether bleeding is localised to the GI tract, or instead part of a systemic haemostatic disorder. Finally, never ignore the skin since mast cell tumours are an occasional cause of secondary GI ulceration.

How do I plan my diagnostic approach?

It is best to follow a ‘problem-orientated’ approach by first developing a problem list, and then determining and prioritising differential diagnoses (Table 1). Although the list is extensive, preliminary information (history, physical examination etc) will enable refinement and reordering of this list. Given that systemic disorders usually cause haematemesis secondary to gastroduodenal ulceration, it is important to eliminate these causes ahead of invasive measures to exam the GI tract. Preliminary investigations (routine laboratory investigations and diagnostic imaging) are employed first, enabling the clinician to target the most valuable additional diagnostic test. If systemic disorders are excluded the investigation can concentrate on the proximal alimentary tract. In most cases, a diagnosis is made with a combination of laboratory testing, diagnostic imaging, direct or indirect visualisation of the GIT (endoscopy and exploratory coeliotomy, respectively) and biopsy for histopathological assessment (Table 2).
How should I approach treatment?

Again, there are no universal rules, and therapy employed will depend upon cause and severity. For emergency cases it may be necessary to stabilise the patient prior to or concurrent with diagnostic investigations. Blood product therapy or colloids may be required in some cases, whilst an emergency coeliotomy may be necessary if bleeding is severe and persistent. If a systemic cause is found (e.g. causing GI ulceration), specific treatment should be provided or considered. Specific therapy for gastroduodenal ulceration may also be required, and examples include acid-blocking agents (H2-antagonists, proton pump inhibitors), mucosal protectants (sucralfate) and/or prostaglandin analogues (misoprostol). Haematemesis secondary to severe mucosal (lymphoplasmacytic) inflammation may require glucocorticoids or other immunosuppressive therapy. Iron supplementation may be required if haematemesis has lead to iron deficiency.
<table>
<thead>
<tr>
<th>Major causes of haematemesis / melaena</th>
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### Ingestion of blood
- Oral, nasal, pharyngeal, haemoptysis

### Metabolic
- Uraemia, hepatic disease, pancreatitis?

### Inflammatory
- Gastritis, enteritis (often eosinophilic), Haemorrhagic gastroenteritis, ulceration, infectious (Parvovirus etc)

### Neoplastic
- Smooth muscle, lymphoma, epithelial cell

### Paraneoplastic
- Mast cell tumours, hypergastrinaemia (gastrinomas/APUDomas)

### Vascular / Ischaemia
- A-V fistula, aneurysms, hypovolaemia, hypoadrenocorticism, thrombosis, infarction, reperfusion injury

### Foreign bodies
- NSAIDs, thrombocytopenia, thrombocytopenia, vWD, factor deficiencies, coumarin toxicity, DIC

### Drug induced
- NSAIDs, thrombocytopenia, thrombocytopenia, vWD, factor deficiencies, coumarin toxicity, DIC

### Haemostatic disorders
- Primary
- Secondary
- Mixed
Table 2. Investigation of haematemesis

<table>
<thead>
<tr>
<th>Emergency database (if required)</th>
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<tbody>
<tr>
<td>PCV/TP [refractometer?]</td>
</tr>
<tr>
<td>Quick blood smear assessment</td>
</tr>
<tr>
<td>BUN</td>
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<tr>
<td>Glucose</td>
</tr>
<tr>
<td>Electrolytes</td>
</tr>
<tr>
<td>Urinalysis [dipstick, USG by refractometer, ?sediment]</td>
</tr>
</tbody>
</table>

**Preliminary tests** (consider in most cases, especially with chronic signs)

<table>
<thead>
<tr>
<th>Haematology</th>
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<tbody>
<tr>
<td>Serum Biochemistry</td>
</tr>
<tr>
<td>Urinalysis</td>
</tr>
<tr>
<td>Faecal bacteriology</td>
</tr>
<tr>
<td>Faecal parasitology</td>
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<tr>
<td>Diagnostic imaging (radiography +/- ultrasound etc)</td>
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</tbody>
</table>

**Tests to classify significance of GI blood loss**

<table>
<thead>
<tr>
<th>Serum iron, total iron binding capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone marrow aspirate/biopsy</td>
</tr>
<tr>
<td>(Ferritin – availability?)</td>
</tr>
<tr>
<td>(Faecal occult blood not usually necessary if obvious haematemesis!!!)</td>
</tr>
</tbody>
</table>

**Tests to assess proximal gastrointestinal tract**

<table>
<thead>
<tr>
<th>Upper GI endoscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exploratory coeliotomy</td>
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</tbody>
</table>

**Additional tests to investigate specific diseases**

<table>
<thead>
<tr>
<th>Suspected endocrinopathy, consider:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoadrenocorticism – ACTH stimulation test</td>
</tr>
<tr>
<td>Gastrinoma - Gastrin measurement +/- coeliotomy (visualisation/biopsy)</td>
</tr>
<tr>
<td>Suspected intestinal disease consider:</td>
</tr>
<tr>
<td>TLI (all GI cases, although haematemesis would be rare)</td>
</tr>
<tr>
<td>Folate and cobalamin</td>
</tr>
<tr>
<td>Histopathological assessment stomach / small intestine (endoscopy or coeliotomy)</td>
</tr>
</tbody>
</table>
**Suspected mast cell tumour**

Cytology

Histopathology

Buffy coat smear?

Staging (diagnostic imaging)

**Suspected pancreatitis (occasional indication)**

Pancreatic enzyme measurement (cPLI, TLI, [Amylase], [Lipase])

Biopsy? [coeliotomy, laparoscopy]

**Additional tests with limited indication**

Tests for gastric spiral organisms

Histopathological assessment

Other

**Suggested approach**

1. *If the case is critical, consider the emergency database to guide initial stabilisation and treatment.*

2. *Preliminary tests should be considered in most cases, especially if signs are chronic.*

3. *Assessment of iron status may be required to guide iron supplementation in some chronic cases.*

4. *Additional tests for systemic disease can be run, when history, physical examination and preliminary diagnostics suggest particular disorders.*

5. *An ACTH stimulation test should be considered (in both species), especially if supportive clinical signs and electrolyte changes are present.*

6. *Gastrin measurement should be considered if a pancreatic mass is detected +/- if hypertrophic gastropathy is detected on gastric biopsies. Assuming preliminary diagnostic eliminate systemic disease, more detailed examination of the proximal GIT may be necessary (unless an obvious cause for haematemesis is evident (e.g. recent NSAID use).*

7. *Exploratory coeliotomy should be considered subsequent to endoscopic biopsy, if results do not fit or response to chosen therapy is poor.*

8. *Additional tests listed have rare indications, but may be worth performing in occasional cases.*
**WEIGHT GAIN – CAN IT BE A GASTROINTESTINAL ISSUE?**

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**Introduction**

For many gastrointestinal disorders, we are concerned with malabsorption and weight loss, as a result of a negative energy balance. Weight gain also arises from energy imbalance, whereby intake exceeds utilisation. Although gastrointestinal disorders are an extremely common reason for dogs and cats being presented to veterinarians, weight gain and excess weight (‘overweight’ and obesity) is arguably more common. Given that obesity predisposes to numerous secondary diseases, it is actually the most important medical disease of our time, and veterinarians must be familiar with causes and consequences.

**Definition**

Obesity is defined as an accumulation of excessive amounts of adipose tissue in the body. In humans, strict definitions of the degree of adiposity exist, and these are based upon epidemiological data. Overweight cats and dogs are greater than 15% above their ideal weight, whilst the term obese’ is used when current weight is 30% above ideal. Disease associations have been proven for companion animals that are both overweight and obese.

**Causes of obesity**

The main reason for development of obesity is an imbalance in the ‘energy balance equation’, in that either excessive dietary intake or inadequate energy utilisation cause positive energy balance leading to increased body fat stores. Numerous factors may influence the relative ease with which weight is gained, and these include genetics, age, neuter status, amount of physical activity, and the caloric content of the diet.

It is known that a number of canine breeds are predisposed to develop obesity e.g. Labrador retriever, Cairn terrier, cavalier King Charles spaniel, Scottish terrier, cocker spaniel for dogs. Neutering is also an important risk factor in both cats and dogs, by causing behavioural changes, which lead both to an increased food intake and decreased activity. Gender itself is also a predisposing factor in some canine studies, with females over-represented. Other recognised associations in dogs include indoor lifestyle, inactivity, and middle age. In cats, middle age and apartment dwelling are possible risk factors.
Dietary factors can also predispose to obesity in both species, with both the number of meals and snacks fed and the feeding of table scraps being key. Further, obese cats more commonly have free choice of food intake. Behavioural factors also play a part in the development of obesity. For cats, possible factors involved in development of obesity include anxiety, depression, failure to establish a normal feeding behaviour, and failure to develop control of satiety. The human-animal relationship is also of importance, and has been shown to be more intense in owners of obese cats. Further, misinterpretation of feline behaviour on the part of the owner; in this regard, many owners misread signals about the behaviour of their cat with regard to eating. In contrast to humans and dogs where eating is a social function, cats do not have any inherent need for social interaction during feeding times. When the cat initiates contact, owners often assume that they are hungry and are asking for food when they are not. Nevertheless, if food is provided at such times, the cat soon learns that initiating contact results in a food reward.

Disease risks associated with obesity
In humans, the medical importance of obesity lies in its effects on lifespan and on predisposing to other diseases. Obese humans, on average, do not live as long, and are more likely to suffer from conditions such as diabetes, high blood pressure, coronary heart disease, certain types of cancer (e.g. breast, ovarian, prostate), arthritis, and respiratory disease. Similarly, obesity has detrimental effects on health and wellbeing of dogs and cats.

Clinical evaluation, physiology and anaesthesia
Overall, clinical evaluation is more difficult in an obese patient compared with a patient in ideal body condition. Techniques that are complicated by obesity include physical examination, thoracic auscultation, palpation and aspiration of peripheral lymph nodes, abdominal palpation, blood sampling, cystocentesis, and diagnostic imaging (especially ultrasonography). Anaesthetic risk is also reportedly increased in obese companion animals and problems include estimation of anaesthetic dose, catheter placement, and operating time. Finally, decreased heat tolerance and stamina have also been reported in obese animals.

Longevity
A recent prospective study has demonstrated an effect of obesity on lifespan in dogs. 24 pairs of Labrador retrievers (48 total) were used, and one dog in each pair was randomly assigned to one of two groups. The dogs in one group were fed ad libitum, whilst the dogs in the other group were fed 75% of the amount
consumed by the respective pair. In the energy-restricted group, body condition score was closer to ‘optimal’ (e.g. group mean 4.5/9) than in the *ad libitum* feeding group (e.g. group mean 6.8/9); lifespan was also increased (e.g. 13 years with energy restriction c.f. 11.2 years when fed *ad lib*). Other beneficial effects included reduced risk of orthopaedic disorders such as osteoarthritis and hip dysplasia, and improved glucose tolerance was also seen.

*Endocrine and metabolic diseases*

Hormonal diseases with a reported association with obesity include diabetes mellitus, hypothyroidism, and insulinoma.

**Insulin resistance, diabetes mellitus and the metabolic syndrome.** Insulin, secreted by cells in the islets of Langerhans of the pancreas, controls uptake and use of glucose in peripheral tissues. In humans, tissues become ‘insulin resistant’ with obesity, and this can lead to type II diabetes and a condition called the metabolic syndrome. The latter is an associated group of disorders including diabetes, cardiovascular disease, high blood pressure and thrombosis (which can lead to heart attacks and stroke). Since cats most often suffer from a similar type of diabetes mellitus (DM) to humans, obesity is similarly a major cause of this condition in the feline species. Interestingly, although dogs tend to suffer from diabetes that resembles human type I DM, obesity is still known to increase the risk of dogs developing the condition. Like with other species, overweight dogs develop insulin resistance and this improves weight loss.

**Hypothyroidism and thyroid function.** Although hypothyroidism is commonly cited as an underlying cause for obesity, such cases are the exception rather than the rule. The prevalence of hypothyroidism is reportedly <1%, with under half of such cases presenting with obesity; in contrast, the prevalence of obesity is far greater (see above). Hypothyroidism is extremely rare in cats. Thus, whilst hypothyroidism should always be considered, it is rarely the reason for the obesity in most cases. Obese dogs have marginally higher (but within the reference range) concentrations of both total T4 and total T3 concentrations than non-obese dogs, but other parameters (e.g. free T4, thyroid stimulating hormone [cTSH], TSH stimulation tests) are not significantly different. Thus, although obesity may have some effects on thyroid homeostasis, such changes are unlikely to affect the interpretation of thyroid function tests.
Orthopaedic disorders

At the author’s clinic, orthopaedic diseases are the most-commonly recognised obesity-associated problems in dogs. The types of problem to which dogs are known to be predisposed include osteoarthritis, hip dysplasia, disc disease, and some types of fracture. Again, weight loss is an important part of management of these problems.

Cardiorespiratory disease and high blood pressure

Obesity can have a profound effect on the functioning of the respiratory system. Most notably, obesity is an important risk factor for development of tracheal collapse in small dogs; other respiratory diseases that can be exacerbated by obesity include laryngeal paralysis and brachycephalic airway obstruction syndrome. Obesity can also affect cardiac function; increased body weight can result in effects on cardiac rhythm, increase left ventricular volume, blood pressure and plasma volume. Finally, obesity has been shown to have a significant, but minor, effect on the development of hypertension. Obesity may also be associated with portal vein thrombosis and myocardial hypoxia.

Urinary tract and reproductive disorders

An association between obesity and some cases of urethral sphincter mechanism incompetence (USMI) has been reported. Weight reduction in overweight dogs with USMI can often be all that is required to allow continence to be restored. The reason for the association is not clear, although the effect may be purely mechanical e.g. increased retroperitoneal fat leading to caudal displacement of the bladder. Although the risk of developing calcium oxalate urolithiasis has been reported to be higher in obese dogs, this may be related to dietary factors. Obese animals are reported to suffer from increased risk of dystocia, likely related to excess adipose tissue in and around the birth canal.

Neoplasia

In humans, obesity is known to predispose to various types of cancer, including breast (postmenopausal), colonic/rectal, renal cell and oesophageal cancer. It is estimated that if this link is entirely causal, being overweight or obese may account for one in seven cancer deaths in both men and women in the USA alone. Epidemiological studies have shown that obese dogs and cats are more likely to suffer from neoplasia than animals in ideal weight. An association between mammary carcinoma and obesity has also been reported in some but not all canine reports. Further, overweight dogs reportedly have an increased risk of developing transitional cell carcinoma of the bladder.
Skin problems

Obese animals have been reported to be at increased risk of certain skin disorders. Seborrhoea is commonly observed (especially in cats), most likely due to reduce ability to groom efficiently, and this can also cause perineal soiling. Animals that are severely obese can develop pressure sores.

Why does obesity lead to other diseases?

There are two main mechanisms by which excessive body fat in obesity can predispose to the various associated disorders. First, deposition of excessive fat can exert ‘mechanical’ or ‘physical’ effects e.g. excessive weight bearing exacerbating orthopaedic diseases, constriction of upper airways exacerbating respiratory disorders, inability to groom in dermatological complaints, and reduced heat dissipation due to the insulating effect of fat for heat stroke. Second, alterations can occur in normal endocrine functions of adipose tissue. In this respect, human fat cells have recently been shown to be able to produce a number of factors, termed ‘adipokines’, which can have a regulatory effect on many body systems. Examples include hormones (e.g. leptin and adiponectin), cytokines (e.g. tumour necrosis factor alpha and interleukin 6), ‘chemokines’ (which attract inflammatory cells into tissues; e.g. macrophage-chemotactic protein 1), and other inflammatory factors (e.g. haptoglobin, and C-reactive protein). In obesity, adipose tissue not only expands (increased numbers of fat cells and increased size of cell) but also becomes unhealthy. In short, it is in a state of subclinical inflammation, and this then leads to disturbances in production of adipokines. It is now known that alterations in the levels of many of these factors plays a key role in the development of many disorders linked to the obese state.

References

References are available on request from the author
Introduction

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. Therefore, in order to fulfil the need of each patient, it is important to understand the types of diet available, and their key ingredients. It is also important to understand, the key issues that impact on nutrition for all of the major gastrointestinal disorders. This should enable the clinician to select the correct diet for the correct circumstances. The current lecture will first discuss the main dietary strategies available to the clinician, and then provide nutritional recommendations for the major alimentary tract diseases.

Dietary strategies

Exclusion diets

There are two forms of exclusion diet that can be used:

- **Single source protein/carbohydrate diets** e.g. chicken/rice, lamb/rice, lamb/barley, duck/rice, fish/potato, white fish/tapioca. Fish-based diets may be best for dogs, but perhaps less so for cats?

- **Hydrolysed protein diets** e.g. Royal Canin Hypoallergenic diet. Recently, diets formulated with protein hydrolysates have become available for the management of dogs and cats exhibiting adverse reactions to food. By enzymatically breaking (either soy or chicken) protein down into smaller peptide fragments, it becomes intrinsically less allergenic. In addition, this greatly improves digestibility, and it may be this that is most important for treatment! A recent randomised controlled trial has demonstrated
that diets containing a hydrolysed protein, are superior to highly digestible diets for the management of chronic enteropathies in dogs (Mandigers etc al., in press).

Highly digestible diets

Highly digestible diets are so-called because they contain ingredients of high biological availability, and are deigned to assist any patient with evidence of malabsorption. In addition, they often contain a range of functional ingredients (i.e. fructose-oligosaccharides (FOS), mannan-oligosaccharides, and fish oils etc) that are designed to improved gastrointestinal health and stabilise the microflora.

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 also slow gastric emptying and prolong digestion, which can be beneficial for some forms of intestinal disease. Furthermore, fat is the most highly digestible of all the nutrients, with digestibility values exceeding 90%. The benefits of feeding a high fat diet have been seen in a variety of gastrointestinal disorders including chronic enteropathy and exocrine pancreatic insufficiency. The author believes that thigh fat diets are preferred over low fat diets in the majority of cases. They are particularly useful where severe malabsorption and weight loss has occurred, since it is easier to feed the desired amount of food. In fact, the only disorders in which fat restriction is recommended are delayed gastric emptying, lymphangiectasia, and pancreatitis (dogs but not cats).

Fibre-supplemented diets

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. However, they are not recommended for gastric disease (since they may delay gastric emptying) or small intestinal disease where there is malabsorption (because they can adversely affect digestibility).

Fermentable fibres such as beet pulp, pectin, guar gum, gum arabic, and FOS may have a positive effect on the mucosal barrier by stimulating the growth of intestinal bacteria such as *Lactobacillus* and *Bifidobacterium*. 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.

There are two methods of fibre supplementation. First, a proprietary high fibre diet can be used (i.e. Royal Canin Fibre Response diet); alternatively a fibre supplement can be added to an existing diet. The choice very much depends upon the preferences of the clinician.

**Management of specific gastrointestinal disorders**

*Chronic enteropathy (inflammatory bowel disease)*

Chronic enteropathy is a common gastrointestinal condition in both dogs and cats, although the pathogenesis is not clearly established. It has been suggested that immune dysregulation may be involved, whereby normal immunological tolerance to endogenous bacterial flora is lost. This leads to uncontrolled mucosal inflammation and disruption of architecture and function. A range of clinical signs can be seen, including vomiting, small intestinal diarrhoea, larger intestinal diarrhoea, and weight loss.
There are a number of dietary considerations:

1. The disease is often characterised by varying degrees of malabsorption.
2. Some cases may develop a secondary dietary allergy
3. There may be varying degrees of weight loss
4. Large intestinal disease may be present

For chronic enteropathy involving the small intestinal predominantly, a highly digestible diet is preferred, that is not fat restricted. Given the propensity for developing food allergy, an exclusion diet is also recommended. In order to fulfil all of these goals, a hydrolysed protein diet is usually preferred. If signs involve the large intestine exclusively, fibre supplementation may help.

*Lymphangiectasia*

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. In addition, the condition is usually associated with leakage of lymph into the intestinal lumen, thus causing a protein-losing enteropathy. However, in contrast to chronic enteropathy, it is not common for dietary allergies to develop secondary to the condition.

Thus, the best diet to choose in patients with lymphangiectasia, is highly digestible diet that is low in fat. It is not usually necessary to feed an exclusion diet, and fibre should be avoided since it may interfere with digestibility.
Adverse reactions to food

Dietary sensitivity can be divided into non-immunologically mediated 'food intolerance' and immunologically mediated 'food allergy' (or hypersensitivity). Clinical signs can either affect the skin or gastrointestinal tract; in the case of the latter, diarrhoea, vomiting, abdominal discomfort and/or weight loss can be seen.

Food intolerance. The clinical signs may result from:

- contamination by chemicals (preservatives), microbes and/or toxins
- idiosyncratic responses due to enzyme deficiencies (e.g. lactase)
- pharmacological effect (e.g. caffeine, tyramine, chocolate)
- direct histamine release (e.g. strawberries, shellfish)
- fermentation of unabsorbed solute (e.g. sorbitol)

True food allergy is often suspected but rarely proven. This suggests a true immunologically-mediated response to a food component (usually a protein). However, in reality, clinical signs (and response to therapy) are identical to food intolerance, and no reliable tests exist to differentiate them.

The gold standard to diagnosis remains the response to exclusion diet & challenge, judged by clinical signs and/or biopsy. Some clinicians believe that home-cooked diets are best, as you can guarantee exactly what is fed. Further, some cases that improve on a home cooked diet relapse when fed the commercial equivalent. However, home-cooked diets are rarely properly balanced and are often inconvenient. Therefore, commercial diets may be preferable.

The length required for feeding of an exclusion diet has not been determined. Some recommend up to 3 months. However, GI signs will often resolve quicker (than dermatological signs?), and owners of (especially large!) dogs with diarrhoea rarely have that degree of patience! Therefore a trial length of ~2 weeks is appropriate for GI cases. If a response is documented, food provocation trials should be performed to identify the exact
casual protein(s) e.g. single proteins are added in sequentially for 7 days at a time. However, this is laborious and many owners elect not to pursue it (if the diet works!).

Other diagnostic tests include serum allergen tests (e.g. IgE antibodies to food allergens), skin sensitivity testing, gastroscopic and colonoscopic food sensitivity testing. Skin sensitivity testing is not useful, whilst gastroscopic food sensitivity testing has not been perfected. Many laboratories now offer serological tests but beware!!! These tests have not been standardised and the presence of serum antibodies does not necessarily correlate with the actual food allergy (determined by exclusion diet trial).

Once a diagnosis is made, long-term remission can usually be achieved by feeding an appropriate exclusion diet long-term. The clinician must, of course, insure that it is properly balanced.

Exocrine pancreatic insufficiency
This is a condition characterised by absence of ancreatic enzyme secretion leading to severe malabsorption. Although a range of gastrointestinal signs can be present, the most common signs are large voluminous ('cowpat') faeces and poor body condition (+/- cachexia). The major dietary consideration is the severe malabsorption, whilst secondary dietary allergy is unusual. However, since these patients are also deficient in intrinsic factor, they can also be severely cobalamin deficient and require parenteral supplementation.

The mainstay of therapy involves pancreatic enzyme replacement. However, dietary management is a useful adjunct, and highly digestible diets are the best choice. Fibre should be avoided, given that it may affect digestibility, and exclusion dies are not usually required. Traditionally, people recommended low fat diets, on the basis that many patients have fat malabsorption. However, the wisdom of this strategy has been questioned recently, since it can prove to be very difficult to correct weight loss with such a diet. Nowadays, in agreement with the approach I human medicine, fat is not restricted.
Pancreatitis

Acute pancreatitis is a common disease, and remains a problematic disorder to manage. 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. There are typically three phases to nutritional management of cases: initial therapy, interim therapy and long-term therapy.

Initial therapy. 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. Assuming that the patient will not eat, then assisted feeding is required and this may involve tube-feeding (naso-oesophageal, oesophagostomy, jejunostomy etc) or parenteral nutrition.

Interim therapy. Once the patient begins to recover and regains its appetite, feeding frequent small meals of a high-carbohydrate, low-fat diet should be gradually introduced as appetite returns. The use of a low fat diet seems to be less critical in cats, given that pathogenesis may differ between the species (less likely to be triggered by a high fat diet). Pancreatic enzyme supplements may diminish the pain associated with pancreatic enzyme secretion during recovery.

Long-term therapy. Long-term dietary control with a low-fat diet is usually recommended, although this may be less essential in cats than in dogs. In addition, since frequent variations in the diet (feeding table scraps etc) can predispose to pancreatitis, it is essential to stress the need to maintain a consistent ration. If the dog or cat is obese, then weight management is recommended once the cat is in clinical remission.
*Feline idiopathic megacolon*

This is a condition usually seen in older cats, and characterised by a massive dilatation of the colon. It is usually idiopathic, and it has been suggested that an underlying myopathy (or neuromuscular disorder?) may be present. Other cases may rise secondary to chronic intractable constipation (e.g. fractured pelvis) or neurological diseases such as feline dysautonomia.

In many cases medical therapy is required i.e. laxatives and prokinetics, whilst the most severe cases may require surgery (sub-total colectomy). However, dietary management is a useful adjunct and the best solution is a fibre-supplemented diet.
Vomiting is defined as retrograde ejection of food or fluid from stomach or small intestine (duodenum). It is a complex reflex act in 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 neurones 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 trough 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 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 of 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 immunoreactivity, 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 anti-emetic.

**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-emetics should be used in most cases to control 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 licensed for treatment of vomiting and emesis in a number of European countries. It 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.