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Faeces are not always uniform in consistency. Many owners believe that variations in faecal consistency are abnormal, but consistency may vary during one evacuation, i.e. initially firm but latterly soft, or over the day, e.g. the initial morning faeces are frequently firmer than those passed later in the day.

It is normal for a dog to defaecate more than twice daily. The accepted range of normality for the dog and cat is one to four times daily although this depends on the diet. The faecal output of a dog fed on a dry, complete diet may be twice that on a meat-based diet. The addition of dog biscuit to the meat has little effect upon faecal bulk as most biscuit is low in fibre.

**PATHOPHYSIOLOGY**

There are four basic reasons why an animal develops diarrhoea.

**Osmotic diarrhoea**

If, for any reason, an animal cannot digest or absorb a dietary component, then osmotically-active particles pass into the distal ileum/proximal colon with the following consequences:

a) Water is attracted into the lumen of the intestine.

b) These particles are fermented by the intestinal bacteria to produce organic acids, ammonia, hydrogen sulphide, amines and bacterial toxins.

   c) Poorly-absorbed fatty acids are hydroxylated by bacteria to produce compounds which reduce colonic absorption of water and stimulate secretion of fluids into the colonic lumen.

**Secretory diarrhoea**

Any agent, whether chemical or biological, which irritates or damages the intestinal mucosa induces an outpouring of fluid into the intestinal lumen. Certain bacteria do this without insulting the mucosa by secreting enterotoxins which mediate conversion of adenyl cyclase to cyclic adenosine monophosphate via prostaglandins. This mechanism in the dog and cat has not been well documented.

Most water resorption takes place in the distal ileum and proximal colon. Although the colon can absorb water and electrolytes, its capacity is finite and once exceeded diarrhoea occurs.
Increased permeability

Normally, the mucosa is protected by a mucous layer. In addition, microflora within the lumen adhere to this mucous layer forming a bacterial coating which resists colonization and penetration by other bacterial species. Certain bacteria can penetrate this mucosal barrier which in turn may allow the normal intestinal flora to enter the portal circulation resulting in systemic illness.

Abnormal intestinal permeability may result in leakage of fluids, electrolytes and large particles into the lumen. The most extreme example of this is seen in protein-losing enteropathies where massive loss of protein into the lumen may result in hypoproteinemia with generalized oedema.

Motility disorders

There are two distinct peristaltic patterns in the small intestine. Segmental peristaltic waves are associated with mixing and absorption from chyle, whereas propulsive peristaltic waves induce mass movements along the alimentary canal (Fig. 1).

It is now recognized that segmentation is the primary intestinal movement and this has resulted in revision of traditional views on motility. Contrary to popular belief, hypermotility is more likely to increase segmentation rather than propulsion resulting in a net increase in total transit time and increased water absorption. Intestinal hypomotility reduces segmentation giving rise to a reduction in water and particle absorption with retention of chyme in the terminal ileum. These are the ideal conditions for osmotic diarrhoea.

The commonest aetiology of diarrhoea in the dog and cat is a flaccid, hypomotile bowel with secondary peristaltic rushes.

The pathogenesis is summarised in Table I.

FAECAL APPEARANCE IN DIARRHOEA

The term diarrhoea can be used to describe an increase in the percentage of fluid in the faeces, an increase in the quantity of the faeces produced or an increase in the frequency with which an animal defaecates. It is important to remember that all three need not coexist.

Much can be learned by observation of the faeces (Table II) and by careful questioning of the owner.
DIARRHOEA IN THE DOG AND CAT

Table I
Pathogenesis of diarrhoea in the dog and cat

| Osmotic   | A. Nutritional intestinal overload |
|-----------|-----------------------------------|
| B. Malabsorption |
| 1. Impaired digestion |
| Exocrine pancreatic insufficiency |
| Bile salt deficiency |
| 2. Mucosal damage |
| Inflammation |
| Neoplasia |
| Gluten enteropathy |
| 3. Enzyme deficiencies |
| Lactose |
| 4. Intramural disease |
| Lymphosarcoma |
| Lymphangiectasia |
| Chronic cellular infiltrates e.g. eosinophils, lymphocytes, plasma cells |
| Amyloidosis |
| Regional enteritis |

| Secretory | 1. Secretory agent |
|------------|-------------------|
| Bacterial enterotoxins |
| Hydroxyalted bile salts |
| 2. Mucosal damage |
| Viruses |
| Bacteria |
| Parasites |

| Increased intestinal permeability |
|-----------------------------------|
| Lymphangiectasia |
| Mucosal damage |
| Protein-losing enteropathy |

| Motility disorders | 1. Increased transit rate |
|--------------------|--------------------------|
| Flaccid bowel with peristaltic rushes |
| (almost all the above problems) |
| 2. Reduced transit rate |
| Low fibre diet |
| Partial obstruction |

ACUTE DIARRHOEA

It is difficult to differentiate between the terms "acute" and "chronic" but in this article the term "acute" describes diarrhoea of sudden onset i.e. the previous day the animal was passing normal faeces. Most of the diarrhoeas classed as "acute" are transient, self-limiting and will respond to symptomatic treatment; conversely, included in this category are several life-threatening conditions e.g. canine parvovirus infection. (Chronic diarrhoea and the treatment of diarrhoea will be discussed in paper II).
Faecal appearance | Diagnosis
--- | ---
Watery, voluminous diarrhoea | Small intestinal problem
Bulky, soft faeces, normal in colour | Usually small intestinal problem
Pale, bulky, fatty, soft, evil-smelling faeces | Steatorrhoea
Watery, voluminous diarrhoea diluted with large amounts of fresh blood | Canine parvovirus
Frequent passage of small quantities of faeces | Haemorrhagic gastro-enteritis
Mixture of formed faeces with water and mucus | Colonic problem
Passing clumps of mucus and jelly | Colonic problem
Watery faeces mixed with fresh, red blood and mucus | Colonic problem
Pale, chalky faeces | Deficiency of bile
Black, tarry faeces | Melaena

NUTRITIONAL DIARRHOEA

Reduced digestion or absorption of nutrients

Diet changes. The dog tolerates a wide variety of dietary components but frequent changes may result in an increased incidence of diarrhoea. This is most commonly encountered when changing from a complete, dry, cereal-based diet to canned or fresh meat. Watery, dark, evil-smelling faeces may be noted for several days. This is also a problem when switching from a diet based on fresh meat to one of canned meat. This is responsible for the common misconception among owners that their dog cannot eat canned food as diarrhoea develops on this diet; in most cases, faecal consistency would return to normal within a couple of days of the diet change.

As a general rule, if a dog or cat has a history of recurrent gastro-intestinal upsets, frequent changes of diet should be avoided. Further, when instituting a radical change of diet, it is worthwhile “weaning” gradually from one diet to the other.

Problems with carbohydrates. Most dogs tolerate a high percentage of carbohydrate in the diet. If, however, a dog has frequent bouts of diarrhoea on a diet containing large quantities of carbohydrate e.g. cereal-based, biscuit, meal, bread, milk, potatoes etc., reducing the amount of carbohydrate in the diet may improve faecal consistency. This is especially important when the animal has a tendency to overeat, e.g. young pups or adults with a “non-selective appetite”, as intestinal overload will compound the situation.

It has been recognized for many years that feeding carbohydrates in the form of uncooked potatoes, grain or flour is poorly tolerated and associated with a high incidence of diarrhoea. A widely held belief among owners is that dogs should not be fed on white bread. This theory has some historical justification as white flour used to be bleached with agene, a compound which produced epileptiform convulsions in dogs. This agent is no longer used in the manufacture of flour.
Milk. Cat breeders have long maintained that feeding milk to oriental cats results in a high incidence of gastro-intestinal problems. It has been demonstrated that feeding a dog more than 1 g lactose/kg body-weight (one pint milk to a 20 kg dog) is potentially hazardous as adult animals may be deficient in the enzyme lactase. This is less of a problem when the pet has received milk every day but is important when milk is administered as a treatment for gastro-enteritis to an animal not used to a regular intake.

Problems with proteins. In the dog, proteins of animal origin are highly digestible if fed in sensible quantities and in the cat the metabolism is geared to a high intake of dietary protein. Dietary problems with proteins, however, do occur.

Overcooking meat reduces its digestibility and owners using fresh meat often ensure that the meat is well cooked to reduce bacterial contamination. Conversely, raw egg white contains a trypsin inhibitor which impairs normal digestion and this is especially significant when the owner feeds milk and raw eggs as a treatment for gastro-intestinal problems or to improve the animal's bodily condition. Cooking the eggs denatures this inhibitory protein.

There is no evidence to show that fresh meat is better for dogs and cats than a canned product. Indeed, in the author's experience, a higher incidence of recurrent diarrhoea is noted when feeding fresh meat compared to commercial diets. Feeding dogs on meat alone (usually because the pet selects meat as a diet) can result in frequent bouts of watery, evil-smelling faeces. The addition of bran to the diet is usually helpful.

There is anecdotal evidence that some dogs exhibit a poor tolerance of offal, especially liver, kidney and hearts, and these meats should be avoided if an animal has a history of gastro-intestinal problems.

Passage of substrates which encourage intestinal bacterial activity

The largest numbers of intestinal bacteria are located in the ileum, caecum and colon. The bacteria most commonly recorded from the intestine of normal dogs are *Escherichia coli*, lactobacilli, streptococci and *Clostridium perfringens*; the production of normal faeces depends upon a delicate balance between the commensals and the potential pathogens. Although the intestinal flora are basically established within the first few weeks of life, the ratio of the organisms present may be influenced by the diet e.g. the numbers of *Clostridium perfringens* rise when a dog is fed a diet rich in meat but fall when the diet is predominantly cereal.

Intestinal bacterial activity is increased when poorly-digested nutrients are presented to the distal intestinal tract (see earlier). Another common aetiology is the ingestion of damaged foodstuffs; indeed the odour of meat which is "off" is a potent stimulus to the canine appetite and accounts for the high incidence of diarrhoea in scavenging dogs.

If an animal consumes more food than it can efficiently digest before the next meal, undigested nutrients will be presented to the distal ileum resulting in diarrhoea. This intestinal "overload" is not uncommon, especially in puppies, and many pets will produce more consistently normal faeces when fed small meals frequently. If adopting this regimen, avoid feeding the last meal late at night as most dogs will defaecate within a few hours of consuming a meal.

Dietary Fibre. It has now been established that increasing the amount of dietary fibre is helpful in dogs and cats with recurrent gastro-intestinal problems. In the wild, dogs and cats ingest a mixture of highly digestible proteins (skeletal muscle, liver etc.) and
material of low digestibility (skin, hair, tendon etc.). There is little residue left in the distal ileum from the former while the latter stimulates peristalsis in the large intestine thus removing residues.

Pets, however, are usually fed diets of easily-digested food with a low fibre content despite the misconception that biscuit contains much “roughage”. The residues may be “held up” in the distal ileum with a resultant increase in bacterial activity. Adding wheat bran (5 to 10 % is usually sufficient) will reduce the incidence of recurrent bouts of diarrhoea. Although most cats dislike cereal products, the fine forms of bran are usually acceptable when mixed with meat.

**Dietary allergy.** The incidence of true dietary allergy (as opposed to dietary intolerance) as a cause of diarrhoea in the dog and cat is not known; cases implicating milk, beef, horsemeat and whalemeat have been described in the literature. Gluten, the protein found in wheat and many other cereals, has been anecdotally blamed for diarrhoea in both species although the only published scientific work suggests that it may be responsible for a malabsorption syndrome not necessarily accompanied by diarrhoea in the Irish setter (Batt, 1984). This will be discussed in paper II.

Where there is concern about a possible dietary allergy, a useful starter diet is a mixture of one type of meat and rice, e.g. cooked lamb, chicken or tripe, cooked rice, corn oil and a multivitamin/mineral supplement. This has the virtue of limiting intake of meat proteins and, assuming the diarrhoea clears up on this diet, individual meats can be added one at a time to assess the effects upon the faeces. A commercial diet made entirely from vegetable protein is also available.

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**BACTERIAL DIARRHOEA**

Experimental evidence exists that *Salmonella* spp., *Campylobacter* spp. and invasive *Escherichia coli* can penetrate the mucosal barrier, whereas other species, e.g. toxigenic *E. coli*, *Staphylococcus* spp. and *Klebsiella* spp., produce enterotoxins (Simon & Gorbach, 1982). Despite experimental data, there have been few well-documented descriptions of bacterial diarrhoea in the dog and cat.

The confusion surrounding this topic is exemplified by the role of *E. coli*; originally blamed for most diarrhoeas in small animals, recent work has cast doubt upon the pathogenicity of the organism (Sherding, 1983). Even if faecal cultures identify the strain as enterotoxic (not an easy laboratory procedure) this may not be significant unless this enterotoxic strain has the ability to adhere to the intestinal mucosa. A further complication is the absence of correlation between the faecal bacterial population and the ileal flora i.e. faecal cultures do not necessarily reflect the situation within the ileum (Thorne & Gorbach, 1978). Finally, even canine neonatal colibacillosis may be related to host resistance rather than bacterial pathogenicity (Dillon, 1984).

Other workers compared the flora from dogs exhibiting clinical diarrhoea with that from dogs with diarrhoea induced by purgatives. The species of bacteria isolated were similar both during the diarrhoea and after it had abated (Ishikawa, Baba & Matumoto, 1982). Thus, accurate interpretation of the results of faecal cultures is extremely difficult (Zimmer, 1983).
Campylobacter

Campylobacter, formerly grouped as a *Vibrio* spp., has been recognized as a veterinary problem since 1909 but it was not until 1972 that the problem in man was appreciated. Since then, isolations from man have increased dramatically (17,247 cases were recorded in England and Wales in 1983). In many countries now, isolations of campylobacter exceed those of salmonella and this increased incidence combined with the close association between pets and owners has stimulated interest in campylobacter in the dog and cat.

**Incidence in the dog and cat.** It has not been demonstrated conclusively that *C. jejuni* is a true enteric pathogen in the dog and cat. Most surveys agree that campylobacter can be isolated from the faeces of about 10% of clinically-normal dogs (Holt, 1980; Bruce & Fleming, 1983). This figure may rise to 50% when faeces from kennelled dogs are examined. There are two possible explanations: a) the dogs have become infected after reaching the kennels or b) dogs may be carriers and only excrete the organism under provocation, e.g. stress or concomitant intestinal infection (it is common to isolate campylobacter from dogs with canine parvovirus).

Similarly, campylobacter is frequently isolated from the faeces of normal cats.

**Identification of the organism.** *C. jejuni* is a curved, motile, Gram-negative rod which requires some oxygen for growth but cannot tolerate atmospheric concentrations of oxygen.

It is difficult to identify the organism by examination of faeces although it is possible to try using Gram’s stain. Culture of the organism can also be difficult as selective techniques are required to inhibit the growth of competing organisms. Further, because campylobacters are micro-aerophilic the oxygen tension over the culture plates must be reduced. Commercial packs are now available for culture of the organism.

When attempting to isolate *C. jejuni* from a clinical case, the following precautions should be observed:

a) Process as soon as possible after collection.

b) Specimens should be refrigerated.

c) Bacteriology swabs should be placed in an anaerobic transport medium as soon as possible after collection (NB anaerobic transport medium can also be used for aerobes).

**Treatment.** Erythromycin at 40 mg/kg for five days or tylosin at 45 mg/kg for five days (see paper II).

**Zoonotic implications.** Less than 5% of human cases have been associated with dogs and cats. Following a positive isolation, the possibility of a zoonosis should always be investigated. Faeces should be checked after treatment to ensure that excretion has ceased.

Salmonella

*Salmonella* spp. are frequently isolated from the faeces of normal cats and dogs and rarely cause clinical disease in adults. In young cats and dogs, however, salmonellosis can be fatal. Diagnosis is based on positive faecal cultures.

Cases of transfer from pets to man have been recorded and the clinician is faced with the problem of deciding what to do with the dog or cat excreting salmonella in its faeces. Most workers now agree that antibiotics should only be used in cases showing systemic signs as their use in simple enteric cases may prolong faecal excretion of the bacteria and encourage development of resistant strains.
ENTERIC VIRUSES

Canine parvovirus (CPV)

When the world-wide explosive outbreak of infection with CPV was noted between 1978 and 1981, two forms of the disease were recognized: the cardiac form which presented as sudden death in young pups due to a non-suppurative myocarditis, and the enteric form in older pups. As maternally-derived antibody protects pups against CPV and as most adults have now been vaccinated or previously infected, almost all pups are now immune to infection during the critical period in the first few days after birth and consequently the cardiac form is now extremely uncommon.

The initial clinical signs of CPV infection (anorexia and dullness) develop four days after contact with the virus; the alimentary signs develop one day later on the fifth day (McCartney et al., 1984b). Affected dogs can deteriorate rapidly and be recumbent within 8 h and dead within 24 h of the onset of clinical signs.

Faecal excretion begins on the third day after infection and is at its maximum between days 4 and 7, after which the amount of CPV particles in faeces drops rapidly (McCartney et al., 1984b).

Diagnosis. 1. Detection of virus in faeces samples. Virus can usually be detected in faeces between days 4 and 7. Demonstration of CPV particles in the faeces strongly supports a diagnosis of CPV enteritis. Conversely, failure to demonstrate CPV particles rules out CPV when the animal is acutely ill but is of no significance in the convalescent dog (Pollock & Carmichael, 1983).

2. Serology. Many different serological methods are in use. Even if the same method is being used, it is very difficult to compare results as the titres vary markedly between individuals. In most dogs, titres become detectable at the time clinical signs begin and rise rapidly during the course of the clinical illness (Pollock & Carmichael, 1983).

3. Haematology. McCartney et al. (1984a) reported that leucopenia and neutropenia occurred in dogs showing severe enteric signs. As neutropenia is thought to be a secondary effect due to loss of neutrophils through the damaged intestinal wall, leucopenia accompanied by neutropenia is considered a grave prognostic sign. American workers, however, claim that fewer than half the infected dogs showed lymphopenia at the time of admission (Kramer, Meunier & Pollock, 1980).

4. Histology. The histological appearance of the intestinal crypts and mucosa are pathognomonic for CPV enteritis. Samples should be taken from several sites and collected as soon as possible after death.

Prevention. Most workers now agree that a third vaccination at around 20 weeks of age is indicated as maternal antibody may persist until 16 weeks of age.

Other canine viral enteritis

Both canine rotavirus and coronavirus have been isolated from dogs' faeces using electron microscopy. The conditions are difficult to diagnose as the virus particles deteriorate rapidly after collection and are destroyed entirely by freezing (Hammond & Timoney, 1983). A dog may shed particles in response to stress, and virions may be noted as a secondary finding to another cause of diarrhoea.

Feline parvovirus (feline enteritis)

More widespread vaccination and the ability of kittens to acquire high levels of antibody from immune queens has meant that the in-utero form (stillbirths, resorption,
abortion etc.), neonatal (cerebellar hypoplasia) and the intestinal form (vomiting, abdominal pain and death) of feline enteritis are now uncommon. Like canine parvovirus, the feline strain is resistant to most chemicals and can remain viable and infectious for more than one year.

**Feline coronavirus**

Enteric coronavirus is widespread in catteries and multi-cat households in the United States but at present there are few figures available for the UK. The virus can cause typical signs of acute enteritis, usually non-fatal, in kittens between four and 12 weeks of age (Pederson, Boyle & Floyd, 1981).

The virus of feline infectious peritonitis (FIP) is antigenically related to the enteric coronaviruses. Serological tests for FIP are available but previous infection with enteric coronavirus may confuse the interpretation of the results obtained. Pederson et al. (1981) postulated that FIP may be a mutation of enteric coronavirus.

**Feline rotavirus**

Feline rotavirus has been isolated in the UK (McNulty, Allan & Thompson, 1978) but at present its importance as a primary enteric pathogen is not known.

**HAEMORRHAGIC GASTROENTERITIS**

Haemorrhagic gastroenteritis (HGE) is characterized by sudden onset vomiting and diarrhoea which rapidly progresses to the passage of frank blood per rectum. Although some cases are mild, most dogs become rapidly dehydrated and require fluid therapy. This fluid deficit may develop so quickly that affected dogs may not exhibit the skin turgor normally associated with dehydrated animals even though packed cell volume may exceed 60.

Presenting signs are similar to those seen with CPV although the age incidence differs (HGE is commoner over the age of one year). Surveys have suggested that the problem is commoner in toy and miniature breeds.

The aetiology is not known, although it has been postulated that the clinical signs are the result of an allergic or anaphylactic response to bacterial endotoxins.

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