INVESTICE DO ROZVOJE VZDĚLÁVÁNÍ

Sborník k semináři
na téma:

Aktuální témata v gastroenterologii u malých zvířat

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CHRONIC VOMITING

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Once you have decided that an animal is vomiting gastric or intestinal contents as opposed to regurgitating esophageal contents, the next step is to determine if there is a substantial amount of blood in the vomitus. The key word here is "substantial"; any animal that is vomiting vigorously may have some flecks of blood in the vomitus due to trauma to the gastric mucosa during the act of vomiting. If there is substantial hematemesis, you can considerably limit your list of rule outs to initially consider.

The next major consideration is whether the vomiting is acute or chronic. Most dogs and cats that have acute vomiting but do not obviously have an acute abdomen (that is to say, they do not have abdominal pain or shock or sepsis) usually have some form of self-limiting gastroenteritis, although foreign objects and intussusception are possible (and parvovirus must always be considered in younger dogs). If you are unsure, modest diagnostics (e.g., chemistry panel and abdominal radiographs) are reasonable. If these diagnostics are non-revealing, then supportive and/or symptomatic therapy is usually sufficient. Vomiting that persists for more than a week, and especially that which occurs for over 2 weeks, is rarely self-limiting and indicates the need for a more aggressive diagnostic approach.

CHRONIC VOMITING

Differential diagnoses for animals that have chronic vomiting: Most patients with chronic vomiting that is not due to motion sickness have either a) alimentary tract obstruction, b) peritoneal or gastrointestinal inflammation, or c) any of several extra-alimentary tract (i.e., "systemic") diseases. Occasionally, there are other causes such as CNS dysfunction, but these causes are rare.

Gastric outlet obstruction may be caused by foreign objects, tissue proliferation, malpositioning of the stomach, or may be iatrogenic (i.e., due to poorly preformed surgery, especially pyloromyotomies). Contrary to what is suggested in some texts, you cannot diagnose or eliminate gastric outlet obstruction based upon the presence or absence of electrolyte changes. Even the so called "classic" hypokalemic, hypochloremic, metabolic alkalosis due to gastric vomiting is neither sensitive nor specific for gastric outflow obstruction. Finding such laboratory abnormalities simply means that a substantial amount of gastric fluid has been lost (or that furosemide has been administered). The specific diseases causing gastric outlet obstruction that are more difficult to diagnose or that are easy to misdiagnose include antral mucosal hypertrophy, gastric carcinoma, spontaneously resolving partial gastric dilatation-volvulus and iatrogenic obstruction due to poor surgical technique.

Gastric antral mucosal hypertrophy grossly resembles an adenocarcinoma. Gastric antral mucosal hypertrophy is usually found in older, small-breed dogs – the same signalment that is so suggestive of gastric tumor. However, mucosal hypertrophy has a much better prognosis because surgery (i.e., pyloroplasty, not pyloromyotomy) is curative. It is not

possible to differentiate gastric antral mucosal hypertrophy from cancer based upon gross appearance. Therefore, appropriate (i.e., deep enough and large enough) biopsy is always indicated before suggesting euthanasia, no matter how obvious the diagnosis appears.

Gastric malignancies are infiltrative lesions that may be proliferative and/or ulcerative. Chow chows seem to have an inappropriately high incidence of these tumors. These lesions usually have a very poor prognosis unless they are diagnosed very early. Unfortunately, these tumors are rarely diagnosed early because the most common clinical sign of gastric tumors (and for many patients with gastric disease, for that matter) is anorexia, not vomiting. The problem is that when an older animal does not want to eat as well as it used to eat, the change in appetite is often attributed to the pet's "getting along in years". Subsequently, nothing much is done until the problem is severe or the animal is losing significant amounts of weight or the animal starts vomiting. This means that these animals are often not given aggressive diagnostic work ups early in the course of the disease, which is when the clinician would have the best chance of curing the disease. However, if the tumor is at the pylorus, then obstruction may occur relatively early in the course of the disease, making early diagnosis and curative surgery more likely.

Abdominal ultrasound is typically the first step in such patients, and should generally be done before endoscopy, if possible. It is easy to miss infiltrative gastric lesions with an ultrasound, but if one if found, one may attempt to diagnose it with percutaneous fine needle aspiration. While it is common to not obtain diagnostic samples due to so many tumors being scirrhous, it is a quick, safe technique that occasionally is diagnostic (especially for lymphomas). Even when endoscopy will be done, ultrasonography may find submucosal lesions that can be easy to miss endoscopically. Clinicians are often reluctant to endoscope patients that have anorexia as their only sign of disease; however, that is wrong. While it is true that most of the time you will find little or nothing when you do scope patients with unexplained anorexia, you should still recommend endoscopy because this is your best chance of finding gastric diseases (especially malignancies) while they are more likely to be treatable. However, it is also possible to make mistakes during gastroscopy. In particular, tumors which are submucosal (i.e., are not invading the mucosa) may have normal appearing mucosa overlying them and be missed if a biopsy is too superficial (something very possible with flexible endoscopy).

Benign pyloric stenosis occurs in some of the brachycephalic breeds such as Boston Terriers; however, this is a tremendously overused diagnosis, often being more of an excuse than a diagnosis. It is rare except in the brachycephalic breeds, and even then it is very uncommon. You should be able to definitively diagnose pyloric obstruction at surgery or endoscopy. If a 7.9 mm outer diameter endoscope can pass into the duodenum of a cat or small dog, or if a 9.0 mm (or greater) outer diameter endoscope can pass into the duodenum of a larger dog, it almost certainly does not have benign pyloric stenosis. Even if a dog has a pyloric stenosis, it is usually not due to benign muscular hypertrophy. Carcinomas and infiltrative fungal infections (e.g., pythiosis) are the major causes of pyloric

stenosis in adult dogs. Finally, ulcers just inside the pylorus can be associated with so much mucosal and submucosal inflammation that the pylorus may appear to be constricted.

If there is not an obvious obstruction present, do not perform pyloric surgery. Animals may develop gastroduodenal reflux (and subsequent vomiting of bile, which was not occurring initially) and/or gastric outflow obstruction because of a pyloroplasty that did not need to be performed in the first place. If you decide to perform such surgery because you believe there is an obstruction, you should perform a pyloroplasty, not a pyloromyotomy because the former are much more effective than the latter. However, pyloroplasties are technically more difficult, and may have more complications when done by inexperienced surgeons. In particular, you need to be sure that you do not interrupt the blood supply to any parts of the gastric wall along your incision line, or you may have subsequent necrosis, perforation, and septic peritonitis. Major points to remember: pyloric spasm and stenosis (diseases that are classically treated by pyloroplasty) are rare in adult dogs and cats; therefore, we seldom need this surgical procedure.

Gastric foreign objects are common and are usually responsible for vomiting, anorexia, and/or abdominal discomfort when they are present. However, the mere presence of a foreign object in the stomach does not guarantee that it is causing that animal's clinical signs. Dogs have had foreign objects in their stomachs for months without any clinical signs. If there is any doubt about whether a particular foreign body is causing vomiting (e.g., some cloth that is not obstructing the pylorus or causing a liner foreign body effect) you should biopsy the gastric and duodenal mucosa while you are removing the foreign object so that you have tissue samples for histopathology in case the animal continues to vomit after you have removed the object. If you decide to remove the foreign object with endoscopy, be sure that you radiograph the patient immediately before the procedure because some objects will sit in the stomach (or for that matter, in the small intestines) for days and move down to the colon just before you do the endoscopy. Bones of incredible size can disappear within 24 hours of exposure to gastric acid; give them a chance to go on their own instead of removing them endoscopically or performing a laparotomy.

Linear foreign objects have some unique aspects that result in our approaching them differently than most other foreign objects. First and foremost, you must remember that a linear foreign object only causes problems when one end is "fixed" and the rest trails off into the intestines. The two principal places for a linear foreign object to fix are the base of the tongue and the pylorus. Other sites are possible, but these two clearly account for the vast majority of linear foreign objects which cause clinical signs. Therefore, everyone knows that you need to look under the tongue of every vomiting cat for such a linear foreign object. However, cats which have a painful lingual frenulum (due to the foreign object cutting into the area) and cats which simply resent having the underside of their tongue examined (which seems to be most cats where I work) cannot be adequately examined without chemical restraint. One mg ketamine/lb

given IV and the use of a mosquito hemostat will allow you to inspect this area adequately.

If you find a linear foreign object caught under the tongue in a patient that has only been sick for a day or two, you can try cutting the object at the base of the tongue and seeing if it will pass through the intestines without causing any further problems. Monitor the patient carefully; if it does not improve substantially within 18-24 hours, you will usually need to surgically remove the foreign object. If a linear foreign object is found endoscopically, it is often appropriate to try to remove it endoscopically if it has only been there for a few days. This is done by passing the tip of the endoscope between the pylorus and foreign object and hence into the duodenum, and then trying to grab the foreign object near its aborad end. In that manner, one may be able to pull the distal object into the stomach and then remove it. In general it is not a good idea to grab the object near the pylorus and pull hard. If the object has only been present for 2-3 days, grabbing the foreign object near the pylorus and pulling might be worth a try, depending upon the nature of the linear foreign object. If the foreign object is relatively broad and seems unlikely to readily cut into and perforate the intestine, you might try pulling; however, do not pull too hard; it's not worth the chance of perforating the intestines. On the other hand, if the foreign object has been there for several days or if the foreign object seems to be relatively thin and could easily cut through the intestine, do not try removing it endoscopically unless you can grab the foreign object near its aborad end.

Chronic linear foreign objects in dogs may present differently than most clinicians are aware. We have seen a few dogs that have had massive intestinal involvement with linear foreign objects that had minimal vomiting and even became normal for a few days to weeks before becoming symptomatic again.

Idiopathic gastric hypomotility is a diagnosis of exclusion. It seems to occur infrequently. There is no anatomic obstruction of the gastric outflow tract, but these patients typically vomit undigested food hours after being fed. Barium contrast radiographs document lack of gastric emptying. Surgery and/or endoscopy fails to reveal an anatomic gastric outflow obstruction. Remember that inflammation anywhere in the abdomen may produce gastroparesis; therefore, be careful when making this diagnosis lest an incorrect presumptive diagnosis of "idiopathic gastric hypomotility" result in failure to diagnose some other cause such as inflammation. Metoclopramide is often used to help empty the stomach and prevent vomiting, but some patients are refractory to this treatment. Cisapride (0.25-0.5 mg/kg bid to tid) may be effective in stimulating gastric motility when therapy with metoclopramide fails. Erythromycin is also an excellent prokinetic agent for the stomach. However, the prokinetic dose of erythromycin is 1 mg/kg, about 1/5th to 1/10th the antimicrobial dose. Ranitidine and nizatidine are H-2 receptor antagonists that have some gastric prokinetic activity.

Bilious vomiting syndrome is a situation in which otherwise normal animals vomit bile, usually in the morning, shortly after getting up. Typically, these patients are otherwise normal. This is also a diagnosis of exclusion. It

appears to be some sort of gastroduodenal reflux syndrome. Feeding the dog just before it goes to sleep, or sometimes giving a prokinetic (e.g., metoclopramide or cisapride or erythromycin) late at night before it goes to sleep usually solves the problem.

Alimentary tract inflammation can be the most difficult of the three main categories to diagnose. You cannot rely on CBC's or radiographs or ultrasonography; you need appropriate biopsies in order to make this diagnosis.

Gastric spirochetes were first discovered in the gastric mucosa of animals and people before the turn of the century. Today, there is strong evidence that *Helicobacter pylori* is responsible for most of the peptic ulcers diagnosed in people, as well as non-ulcer dyspepsia in many others. *Helicobacter pylori* is also a predisposing cause of human gastric carcinoma, and there are multiple reports of biopsy "proven" gastric lymphosarcoma completely regressing after eliminating resident *Helicobacter* with antibiotic therapy.

Diagnosis of gastric *Helicobacter* infection is usually obtained by cytology, urease testing of gastric mucosa, and/or histopathology of gastric mucosal biopsies. Histopathology is more than adequate for diagnosis in almost all dogs and cats. Cytology (which is actually a bit more sensitive than biopsy) is performed on gastric mucosal biopsy samples or on cytology samples obtained with endoscopic brushes. Slides can be stained with new methylene blue or Diff Quick or various other techniques. There is currently a serologic test for *Helicobacter* infection in people, but it is not valid for dogs or cats. Treatment in people has involved a variety of combinations of drugs. Because the bacteria live below the mucus layer, *in vitro* sensitivities do not always translate into *in vivo* efficacy. *Helicobacter pylori* tends to develop resistance quickly when only one drug is used (e.g., approximately 30% of strains are resistant to metronidazole). Certain other factors (i.e., short period of treatment, poor compliance, prior therapy with omeprazole) seem to be associated with an increased incidence of therapeutic failure. The classic triple therapy (i.e., metronidazole, bismuth subsalicylate, tetracycline for 2 weeks) had success rates of 90%. Macrolides may be the most effective antibiotics. There are anecdotal reports of erythromycin working well as a single agent in some cats and dogs. Clarithromycin and azithromycin have been used with apparently good success in infected people. The currently recommended dose of azithromycin appears to be 5 mg/kg qd for cats and 10-40 mg/kg qd to bid for dogs. Azithromycin has fewer side effects than erythromycin but is more effective. Use of omeprazole to eliminate gastric acidity seems to make antibiotics more effective. Omeprazole is, in people at least, distinctly more effective for helping to eliminate *Helicobacter* infections than either cimetidine or famotidine. Combinations of omeprazole, azithromycin, and metronidazole seemingly only need to be given for 7-12 days to effect a cure in people. However, because dogs and cats are infected with *Helicobacter* species other than *H. pylori*, much less aggressive therapy is typically satisfactory. Famotidine (0.5 mg/kg bid), amoxicillin (10 mg/lb bid), and metronidazole (10-15 mg/kg bid) for 12-14 days appears to be more than adequate; however, re-infection or

recrudescence seems common.

The fundamental question is, "When, if every, should we treat animals with upper gastrointestinal signs for *Helicobacter* infections?" One cannot rely upon the presence or absence of gastric inflammatory infiltrates to be able to determine the clinical significance of the presence of *Helicobacter.* One study found that cats that were not vomiting were just a likely to have these spirochetes and gastric inflammation as were cats that were not vomiting. Dr Guyer found that approximately 30% of vomiting cats and 30% of normal cats had both inflammatory gastric infiltrates and *Helicobacter*. Likewise, approximately 30% of vomiting cats and 30% of normal cats had neither inflammation nor *Helicobacter* in their stomachs. Because of our inability to look at a gastric biopsy and determine if the bacteria are responsible for the clinical signs, our current approach is to first determine if there are any other potential causes for the vomiting in the patient. If there is another potential cause that looks as or more likely to be causing the vomiting, we treat it first. If this treatment does not work or if there is no other identifiable cause of vomiting, then one may treat for gastric *Helicobacter* and see if the patient responds. It is also reasonable to treat vomiting dogs and cats for *Helicobacter* gastritis before proceeding with more aggressive diagnostics such as endoscopy. However, particularly ill animals should generally first have diagnostics. There is no evidence that dogs and cats are a zoonotic risk for *H. pylori* infection in people. *Helicobacter heilmannii* is distinctly less common as a human pathogen than is *H. pylori,* but dogs and cats may be a risk factor for human infection with this organism. At the time of this writing, it is still not clear how most people become infected with *Helicobacter pylor*, but dogs and cats do not appear to be involved.

Focal gastritis is a potentially confusing problem. Gastric lesions may be relatively focal and yet cause major vomiting. Lesions at the lower esophageal sphincter may be especially hard to diagnose since they are only diagnosed by the retroflexed view when doing gastroscopy.

Lesions of the ascending colon or ileo-colic area or cecum may also cause vomiting, especially in cats. The interesting point is the animal may have absolutely no other signs to indicate the lower intestinal involvement (e.g., diarrhea).

GASTROINTESTINAL BLEEDING

Michael D. Willard

Hematemesis necessitates a slightly different approach than we take with other vomiting cases because some rule-outs become more likely while others become much less likely. We will be including upper gastrointestinal bleeding of any cause in this discussion. For starters, we will not be discussing vomiting that produces "flecks" of blood because this can be seen in any dog (and perhaps cat) with vigorous vomiting in which the gastric mucosa is traumatized by the physical act of vomiting. It is easy to identify fresh blood in the vomited material as long as the patient is not eating something that is red or that produces a pink color to the vomited material simple secondary to pigment leaching out of the food material. Most of the time, hematemesis is denoted by a "coffee-grounds"-like material that most clients (and some veterinarians) do not recognize as blood. A common mistake is being concerned over "dark stools". Noting that a patient has dark stools is generally useless. Lots of dogs have dark stools and no problems or GI blood loss at all. The color of the stool is not an issue until the stool is pitch-tar-coal-asphalt black. Then it may be melena (if it is not due to Bismuth or a lot of green bile giving it a near-black appearance). If in doubt, just place some fresh feces on absorbent white paper and see if a reddish color diffuses out from the feces, confirming that there is blood present. Melena is only seen if there is acute loss of a lot of blood into the upper GI tract. Most dogs losing blood in the upper GI tract do not have any important changes in the color of the feces. Rather, you might see anemia and hypoalbuminemia. Also remember, you may or may not see hypoglobulinemia; it all depends upon what the serum globulin concentration was before you started losing blood. Sometimes the BUN is higher than expected based upon the serum creatinine, but again this is only expected if there is a lot of blood being lost in a short period of time. Fecal occult blood tests are seldom that helpful or necessary, but can occasionally be informative in confusing cases. However, you need to use a test for which the laboratory has substantial experience in dogs so that the results can be meaningfully interpreted. Some fecal blood tests will routinely give a positive reaction when used on canine feces.

When there is a substantial amount of blood being ejected from the mouth, there tend to be 3 major reasons: coagulopathy, swallowing blood from elsewhere and gastrointestinal ulceration/erosion (GUE).

Coagulopathies: Most coagulopathies cause concomitant bleeding from the nose or accumulation of blood in body cavities or petechia. However, there are many cases in which the only sign of a systemic coagulopathies is GI bleeding. Therefore, it is always appropriate to check the platelet count and some measure of clotting factor adequacy in animals with hematemesis or GI blood loss. While coagulopathies are a relatively uncommon cause of GI bleeding compared to ulceration/erosion, they can have devastating consequences if not diagnosed promptly. In particular, remember that some cats with intestinal

disease will malabsorb vitamin K to the point of having vitamin K-responsive coagulopathies. Also remember that just because the patient did well during a surgical procedure a few days before the current bleeding episodes does not mean that a coagulopathy is impossible; sometimes the prior surgical procedure apparently depletes the limited amounts of coagulation factors in patients with subclinical coagulation defects, causing them to become clinical after the procedure.

Ulcers and erosions: Gastritis due to any number of causes typically has some degree of GUE present. We will not discuss this cause too much since most of these cases quickly and spontaneously resolve. *Helicobacter* is important in people, but to date has not been shown to have a cause-and-effect relationship with GUE in dogs and cats. There are often incredible numbers of *Helicobacter* found in ulcer craters; however, *Helicobacter* can be found in ulcers caused by almost anything. Therefore, it seems likely that the *Helicobacter* are there because a) they are found in most dogs and cats and b) the ulcer crater is an opportune place for them to grow. Renal disease is often named as a cause of gastric ulceration, but this is actually very rare.

The most common causes of chronic, unresolving GUE that are also the easiest to check for are mast cell tumor, drug administration and "stress".

Drugs are still a very important cause of GUE in the dog, despite all the newer, "safe" NSAIDs. High doses of dexamethasone also have substantial potential for significant GUE. Prednisolone by itself is generally not ulcerogenic unless it is used in very high doses (e.g., > 2-3 mg/lb/day) or is administered to a patient with other "ulcerogenic" risk factors (e.g., hypoxia, poor perfusion), and even then it is not particularly bad. Combining steroids and non-steroidal drugs can be devastating. You can use ultra-low dose aspirin (0.5 mg/kg once daily) when treating IMHA dogs with steroids.

There continues to be a substantial problem with the use of nonsteroidal anti-inflammatory drugs (NSAIDs) in dogs. All NSAIDs have the potential to cause devastating GUE, and some of these non-steroidal drugs are renowned for their toxic effects (i.e., indomethacin, naproxen, flunixin). Ibuprofen is also particularly ulcerogenic in the dog because it undergoes an enterohepatic circulation. Flunixin is a particularly dangerous drug from the standpoint of causing GUE. It is extremely potent and can be devastating if combined with steroids like dexamethasone. While able to cause significant ulceration and bleeding all by themselves, the ulcerogenic potential of NSAIDs is particularly augmented by other factors, especially concurrent administration of another NSAID or a corticosteroid, and hypoperfusion of the alimentary tract. Even though many dogs seemingly tolerate such combination therapy, you need to realize that you are "walking on thin ice" (see comments above on use of ultra-low dose aspirin). Many to most of the dogs treated with NSAIDs have endoscopically visible erosions, hemorrhages and/or ulcers, depending upon the drug used and the dose administered. It is important to note that most dogs with GUE due to NSAIDs may be completely asymptomatic. Finally, there is tremendous between-dog variation regarding the

alimentary tract response to NSAID's; some dogs may almost bleed to death because of a small dose of aspirin while most dogs would tolerate a much larger dose with relative impunity.

While the newer Cox-2 NSAIDs (e.g., carprofen, etogesic, deracoxib, meloxicam, etc) have much less potential for causing GUE than the older NSAIDs, you can still see GUE (and even perforation) due to these drugs. Part of the problem is that these "safe" drugs are being used so extensively and casually. The problem often revolves around using inappropriately high doses (after all, the drug is so safe that ...), using the drug at the wrong time (e.g., when the patient is experiencing shock or has poor perfusion to the alimentary tract), and possible using the drug too soon after stopping some other NSAID. The concept of a "washout" period when changing from one NSAID to another is extremely controversial. There is published literature to the contrary, but the fact is that nobody really knows at this time.

Stress, when mentioned as a cause of ulcers, specifically refers to substantial decrease in visceral perfusion (e.g., hypovolemic shock, neurogenic shock, Systemic Inflammatory Response Syndrome) that is typically obvious from history and/or physical examination; or, it can refer to extreme exertion (e.g., sled dogs running in subzero weather for 100 miles).

Mast cell tumors may look like any skin lesion. In particular, they can perfectly mimic the appearance and feel of lipomas, such that the only way to distinguish them from lipomas is by aspirate cytology. When these tumors degranulate, they release histamine which if of sufficient magnitude can cause gastric acid hypersecretion. This can result in severe ulceration, especially just inside or just beyond the pylorus.

Hepatic failure seems to be another important cause of GUE in the dog. Anytime a dog with hepatic disease suddenly becomes clinically worse (especially if it becomes obviously encephalopathic), you should consider the possibility of GUE. Bleeding into the intestine counts as a high protein meal and predisposes to hepatic encephalopathy in these patients. In ill patients that cannot be scoped, aggressive therapy with famotidine and/or carafate is reasonable. Hepatic disease may cause disseminated intravascular coagulopathy which can cloud the picture when trying to determine the cause of hematemesis.

Gastric tumors may cause bleeding. The leiomyoma and leiomyosarcoma in particular may cause especially dramatic bleeding due to their propensity to ulcerate. This is especially important because this tumor is often curable with surgery, as opposed to lymphomas and carcinomas that are more common, have less dramatic signs, seldom cause GI blood loss and yet have a much worse prognosis. Unfortunately, it can be hard to adequately image the stomach with ultrasound, and these masses can be missed if there is blood, ingesta and/or air in the stomach.

Surgery can be responsible for GI bleeding. If the closure is done improperly and the mucosa does not cover the defect, then bleeding can easily result.

Hypoadrenocorticism may be responsible for severe hematemesis that can produce life-threatening shock. Such severe hematemesis

appears to be a rare complication of hypoadrenocorticism, but should be considered in cases with appropriate history, CBC and/or serum biochemistry changes, as well as patients that do are not readily diagnosed with other causes.

Inflammatory bowel disease has been reported to be associated with GUE, especially in the dog. Right now, the definition of IBD is under scrutiny, but at any rate it appears IBD is not commonly associated with GUE. However, when idiopathic GUE is found during endoscopy, the stomach and duodenum should be biopsied.

Heavy metal intoxication causes hematemesis, but this is fortunately uncommon at this time.

Gastrinomas are typically small pancreatic tumors which produce large quantities of gastrin, a hormone which causes gastric acid secretion. Multifocal duodenal ulceration/erosion is very suggestive of this tumor, as is a large ulcer just past the pylorus (as for mast cell tumors). Gastric erosions are sometimes seen, but gastric ulcers appear to be rare in this disorder. GI bleeding is not particularly common in these patients although it is possible. Since the gastric mucosa is stimulated to grow, ulceration typically occurs in the duodenum instead of the stomach. Esophageal ulceration may also occur if there is gastroesophageal reflux of the highly acidic gastric contents. Measurement of serum gastrin concentrations may be diagnostic. However, anything which causes gastric distention or renal failure can produce increased fasting serum gastrin concentrations. Treatment with H-2 receptor antagonists has been rewarding, although unexpectedly large doses or the more potent proton-pump inhibitors (e.g., omeprazole or lanosprazole) may be necessary.

Foreign objects get a lot of press as causes of GUE, but in fact they are relatively uncommon causes. However, they are particularly important in patients that have GUE because even the most innocuous of GI foreign objects (e.g., paper, small piece of soft cloth) can sometimes prevent a pre-existing ulcer from healing. They typically need to be removed in patients with GUE.

Ingesting blood: This is a possibility that is typically forgotten. However, it is surprisingly easy to have bleeding pulmonary lesions in which the blood is coughed up, swallowed, and later vomited. In most of these cases, the client does not report coughing (perhaps because the hematemesis "wipes" all else from their mind). In like manner, we have seen cases in which blood was trickling from the choana into the pharynx and being swallowed, and yet the patient had no history of sneezing or coughing or nasal discharge.

Cats are rare diagnosed with gastric ulceration/erosion. When a cat is found to have gastric ulceration/erosion, it is usually caused by lymphoma. However, we many times never determine the cause of GUE in cats.

Clinical approach to the patient with hematemesis or GI bleeding:

There is often something in the history that is suggestive of the cause of the bleeding (e.g., use of NSAIDs, shock, etc). If that is the case, then it is often reasonable to begin appropriate therapy after requesting basic laboratory testing (e.g., CBC, serum chemistry panel) to determine the

severity of the bleeding and if there are other diseases (e.g., hepatic disease, renal disease) present. Imaging (especially ultrasound) is typically appropriate but not necessarily imperative at this time. If the cause of the GI bleeding or hematemesis is not obvious, if the patient has not responded to 5-7 days of appropriate therapy, or if the bleeding is severe, then additional diagnostics are important and should be performed promptly.

Diagnostic approach: First, as stated above, it is wise to first eliminate coagulopathy with a platelet count and some measure of clotting factor adequacy. I typically request PT and PTT, but a mucosal bleeding time is a very useful screening test in these patients. Sometimes there is both a mucosal defect and a coagulopathy. In particular, if ehrlichiosis is possible, one must consider the possibility that the patient has what would normally be an insignificant mucosal defect but which is bleeding because of the effects of *Ehrlichia* spp. upon platelet numbers and their function. After coagulopathy has been eliminated, then imaging should be done if it has not already occurred, and ultrasound is especially important as it may reveal masses that can be aspirated percutaneously, thus avoiding the need for endoscopy/surgery.

If these tests have not revealed the diagnosis, then gastroduodenoscopy is generally performed next. The specific reasons to do gastroduodenoscopy in a patient with GI bleeding are to:

a) determine if this is a case in which surgery can remove a defined number of ulcers (this is for cases that are bleeding and have not responded to medical management or cases that are bleeding so badly that one cannot wait on medical management). In these cases, it is important to be sure that bleeding is not due to widespread erosions that cannot be cured surgically. There is no relationship between the size of the mucosal defect and the amount of bleeding; patients with lots of small erosions often bleed as bad or worse than patients with ulcers. It is also important to determine the number and location of such ulcers as they may be hard to find during a gastrotomy.

b) determine if there is a gastric tumor or some other infiltrate in a patient with GUE that is non-responsive to appropriate therapy.

c) determine the cause in a patient with GUE and no apparent cause on the history, physical examination, or routine blood work.

d) look for a cause of bleeding in a patient with GI blood loss of unknown cause.

It is important to note that endoscopy will not generally allow one to determine if an ulcer will or will not respond to medical management. In most cases, only by treating and observing the patient will you know.

It is important to not give carafate within 24-36 hr before endoscopy because it will cover the lesions and make evaluation more difficult. It is best if food has been withheld for at least 24 hours. Avoid prokinetics (e.g., metoclopramide). Endoscopy of these patients may be difficult is there is substantial blood present in the stomach. Patience is necessary to flush in water and then aspirate it and the blood over and over again. It is important to be able to view as much of the gastric mucosa as possible. In some cases, there are lots of hugh blood clots that cannot be removed endoscopically, in which case surgery might be necessary. It can be

especially easy to miss ulcers that are in the pylorus. The pylorus is typically infiltrated and inflamed, making it more difficult to pass the tip of the scope through that area. Therefore, the endoscopist often does not obtain a good view of this area. One may have to go in and out of the pylorus multiple times to be sure that there is or is not a lesion present. If a cause of upper GI bleeding cannot be found in the stomach or duodenum, strong consideration should be given to examining the trachea, bronchi, and choana while the patient is anesthetized. Patients with hematochezia may benefit from colonoileoscopy, but patients with hematemesis or melena rarely benefit from lower endoscopy.

If there is substantial upper GI blood loss and these tests do not allow diagnosis, then exploratory surgery is the next step. However, it is very easy to not be able to find the cause of the bleeding in these patients, so warn the owner before doing the procedure.

Medical management: If the patient is not exsanguinating, the cause is known or strongly suspected, and the patient has not had 5-7 days of appropriate medical therapy, then medical therapy is often reasonable as opposed to doing a major diagnostic work up. In distinction, if the patient is exsanguinating or if the patient has not shown any appreciable response to 5-7 days of <u>appropriate</u> medical therapy for the ulceration, then it is usually reasonable to surgically resect the ulcerated area. Note – when I say "response", I am not referring to the patient being cured; I am referring to clear evidence of improvement. If surgery will be considered, it is usually very wise to perform gastroduodenoscopy before the surgery to be sure that you find all of the sites of ulceration. It is very easy to fail to detect an ulcer at surgery, and endoscopy usually allows one to easily find all areas of ulceration. Sometimes intraoperative endoscopy is necessary to help the surgeon find the ulcer(s).

If medical management is elected, first be sure to remove the cause of the GUE. If the cause is not removed, medical management tends to be far less successful. Next, be sure that the patient is well hydrated; healing of the gut requires or is at least benefitted by adequate perfusion. If there is significant gastroduodenal reflux of bile, metoclopramide or cisapride may be helpful in preventing bile from entering and/or staying in the stomach and augmenting the ulcerogenic process.

<u>H-2 receptor antagonists</u> are commonly used. Cimetidine, ranitidine, and famotidine are good medications for decreasing the gastric hydrogen ion concentration. Cimetidine (5-10 mg/kg) needs to be given 3-4 times per day if you are really serious about decreasing gastric acid secretion. However, famotidine (0.5 mg/kg) only needs to be given once or twice daily. In most patients, cimetidine is more than adequate to allow healing of gastric ulcers. However, the over the counter (OTC) preparations are all oral and some patients are vomiting so vigorously that they must receive parenteral medication. Ranitidine (2.2 mg/kg) is usually effective if given twice daily, but may cause vomiting if given as an IV bolus. There is some evidence that ranitidine is not particularly effective in decreasing gastric acid secretion, but that is not certain at this time. Side-effects of the H-2 receptor antagonists are rare but may include diarrhea, drug eruption, hyperpyrexia, thrombocytopenia, granulocytopenia, and CNS problems

including seizures. I think I have seen a couple of dogs in which ranitidine was responsible for large bowel diarrhea or seizures. The primary value of the H-2 receptor antagonists is in treating existing ulcers and erosions. They can be helpful in preventing some types of ulcers, but this is not true with all types of ulcers (e.g., they are not effective in preventing ulcers due to NSAIDs or due to steroids).

Proton pump inhibitors are the most effective antacid drugs we have. Omeprazole, lanosprazole and pantoprazole are the most effective inhibitors of gastric acid secretion we currently have available. Omeprazole is available OTC as Prilosec®. The H-2 receptor antagonists seem quite adequate for GUE except in some animals with gastrinomas and those with esophagitis due to gastroesophageal reflux: these seem to be the main reason for using the PPI's. The dose of omeprazole is 0.7-1.5 mg/kg qd, although I have often used it at up to 2 mg/kg bid in patients with severe reflux esophagitis or gastrinomas. The dose of lanosprazole (Previcid), pantoprazole (Protonix), and esomeprazole (Nexium) is 1 mg/kg IV (not approved for use in dogs). It generally takes 2-5 days for a PPI to have maximal efficacy; but, the immediate effects on gastric acid secretion is often still better than that obtained by high dose H-2 receptor blockade. Very rarely, an H-2 receptor antagonist will work better than omeprazole; be prepared to experiment in your difficult cases.

Misoprostol (Cytotec®) is a prostaglandin E analog which was primarily designed to be a prophylactic drug used to prevent GUE due to NSAIDs. It is also useful in treating existing ulcers, but its higher cost and more plentiful side effects usually make it undesirable as a first line therapy for GUE. It is typically used at a dose of 2-5 ug/kg, 3-4 times daily. It can cause abdominal cramping and diarrhea, but the drug seems relatively safe in dogs. The main disadvantage is that it must be given orally, which is not possible in some vomiting animals. Because it is a prostaglandin analog, it should not be used in pregnant females for fear of causing abortion or miscarriage. It is the best drug available that can be used to prevent NSAID-induced ulceration, but it is not uniformly effective in dogs. The main indications to use it appear to be a) the patient that must have NSAID's to function, but which evidences side-effects from them (e.g., anorexia, vomiting) and b) the patient that seemingly needs to receive NSAID's that have substantial potential for such side-effects (e.g., piroxicam).

Sucralfate seems to be extremely effective in protecting those areas which are already ulcerated and helping them heal. The only common side-effect is constipation. There is minimal absorption from the intestines, but it does have the capacity for adsorbing other drugs (e.g., enrofloxacin). While carafate is effective in treating ulcers, it is not always effective in preventing ulceration. In patients with severe hematemesis and anemia, we sometimes us a large "loading" dose (e.g., 3-6 grams) initially and then decreasing the dose to 1 gram tid to qid. No body know if the loading dose is beneficial or not. My major problem with this drug is that it must be given orally, which does not always work in vomiting dogs. Sometimes you may dissolve the table in water or buy the suspension and have less problem with that being vomited.

ESOPHAGEAL DISORDERS

Michael D. Willard

Regurgitation occurs when there is either an anatomic obstruction or a physiologic weakness in the esophagus. In either case, food is retained in the esophagus and, if it passively migrates back into the oropharynx, can be regurgitated. The problem should be diagnosed quickly in an attempt to solve it before the esophagus becomes irreversibly-dilated or the patient experiences an aspiration pneumonia.

First, be sure to try to distinguish vomiting from regurgitation. We usually start doing this by considering the history and physical examination. This can be hard to do, and the following are guidelines only -- some animals that clearly appear to be vomiting are in fact regurgitating and vice-verse. In particular, it is very easy for a vomiting dog to appear to be regurgitating. However, these guidelines are still useful and usually point us in the correct direction.

● Prodromal nausea is commonly found with vomiting. Since vomiting is a centrally-mediated response, other signs such as salivating, discomfort and "gurgling" stomach are often seen beforehand. Many animals that are about to vomit will pace, whine or show some sort of anxiety or discomfort. With regurgitation the animal may be sitting and suddenly "gag" up some material. In general, animals know that they are going to vomit, but they are often unaware that they are going to regurgitate until they actually start doing it. Sometimes the regurgitation is as much a surprise to them as it is to the client. These are not absolutes - animals don't always read the book.

● Retching typically follows prodromal nausea and is characterized by forceful, abdominal contractions in animals that are vomiting. (You will see some abdominal contractions with regurgitation but they are not severe or forceful and they do not tend to be repetitive.) If you're not sure what retching is like, just think back to the last time you had to vomit. Don't just ask owners "Did the animal retch?" because they may consider any contractions of the abdomen to be retching. Clearly describe precisely what you mean so that they can give you an accurate answer.

● The material the animal expels sometimes help us distinguish what is going on. If possible, let the client describe the material first so they're not just agreeing with you to make you happy. So-called "undigested" material can be either vomited or regurgitated. If it is digested, then this would indicate that the material came from either the stomach or intestines; however, it can be very difficult or impossible to visibly differentiate undigested material that was chewed up, mixed with mucus and saliva and has been sitting in the esophagus for a long time from digested material.

● Mucus can come from either the salivary glands (i.e., the regurgitating animal) or the stomach (i.e., the vomiting animal).

● Red blood can be seen with either vomiting or regurgitation, but semi-digested blood that looks like coffee grounds is only seen with vomiting. However, finding digested blood does not ensure that the

bleeding originated in the stomach.

● Bile indicates that the material is from the stomach or intestines. Bile is a green, yellow or dark brown color. Don't just ask if the animal is vomiting bile; many clients assume that vomitus contains bile (i.e., my animal "vomited", therefore it must have vomited bile). Clearly describe what you mean by "bile".

● Occasionally (rarely), regurgitated material will take on the shape of the esophagus and come out as a tubular mass. However, the same can occasionally happen with material that is vomited. Therefore, this is not too helpful.

● The amount of material ejected from the mouth varies from large to small with both vomiting and regurgitation. Likewise, the timing of the episode relative to eating can vary from immediately after eating to 1 ½ days after the last meal, regardless of whether the animal is vomiting or regurgitating. Don't forget that you can regurgitate mucus even though you have not eaten for days.

If you are still confused as to whether the patient if vomiting or regurgitating, the most definite method of distinguishing vomiting from regurgitation usually consists of performing plain thoracic radiographs, possibly followed by a barium contrast esophagram. There are some causes of regurgitation that will be missed by such studies, but they are far and few between.

Physical examination may also help distinguish vomiting from regurgitation. Occasionally the esophagus is so dilated and flaccid that it can be seen expanding and collapsing near the thoracic inlet as the animal breathes (much like a bellows). A particularly nice trick is to test the expelled material with a urine dipstick. If the pH of the material that the animal spit out is $\leq$ 5 or if bile is present, then the material has been vomited. Otherwise, it has probably been regurgitated. Do not trust the reaction for blood. It is invariable positive and does not help distinguish vomiting from regurgitation.

Initial diagnosis: The first consideration in the animal that is regurgitating is to clarify whether the regurgitation is due to anatomic obstruction of the esophagus or due to esophageal weakness. A barium-contrast esophagram is the best way to determine which is occurring, and is often indicated in patients with suspected esophageal disease. Plain thoracic films should be done first because they will often reveal esophageal foreign objects, pneumothorax, and/or pleural effusion; signals that a contrast procedure is not needed and in fact is contraindicated. However, a contrast procedure is otherwise useful, even if plain films strongly suggest megaesophagus (i.e., an obvious air-filled, dilated esophagus). Some animals with aerophagia have plain radiographic findings suggesting esophageal weakness, but the contrast procedure will demonstrate normal esophageal function. I absolutely avoid using barium paste because it can seemingly cause worse problems than liquid barium if it is aspirated.

Esophageal obstruction, when present, must next be distinguished as either being congenital or acquired.

Congenital esophageal obstructions are usually vascular ring

anomalies, the most common probably being the persistent right fourth aortic arch (PRAA). The PRAA is typically seen as a dilated esophagus immediately cranial to the base of the heart, while there is no evidence of retention of contrast caudal to the heart. This radiographic pattern in a young patient or one which has had signs of regurgitation since it was young is almost pathognomonic. However, this congenital problem has been found in older animals. Furthermore, esophageal weakness may radiographically mimic a PRAA if the esophageal weakness is especially severe in front of the heart, causing a dilation near the thoracic inlet. Older animals may have a PRAA and only have intermittent signs. You cannot assume that a regurgitating patient has megaesophagus – it may have esophageal obstruction that requires a timely surgery.

Acquired esophageal obstructions may be due to foreign objects (especially bones and rawhide treats), esophageal parasites, esophageal or periesophageal tumors, cicatrix formation secondary to esophagitis, and rarely achalasia.

Esophageal foreign objects usually consist of bones but may be rawhide treats, food, dental chew toys, toys, balls, rocks, wood, etc. They usually lodge at the thoracic inlet, base of the heart, or lower esophageal sphincter. A history of a patient that begins to regurgitate (as opposed to vomit) acutely is very suggestive of acquired esophageal obstruction due to a foreign object. These patients may continue to drink water, but they typically refuse solid food because the food bolus cannot pass by a partial esophageal obstruction and causes pain whenever it tries to. A casual, careless history that fails to raise the suspicion of regurgitation will typically lead the clinician to suspect an acute gastritis. However, the realization that the patient is regurgitating (as opposed to vomiting) should be a "red flag". Too often, a pet which has ingested a foreign object is treated conservatively while we wait and see if the supposed gastritis spontaneously resolves. This is problematic because foreign bodies can erode and perforate the esophagus much quicker than they would stomach or intestines.

Plain radiographs should be performed first. Bones are a common cause of obstruction, and plain films that are made with proper technique and then carefully evaluated are diagnostic in most cases. Remember that poultry bones are not as radiodense as the patient's bones, which means that excellent radiographic technique is required to see them. Foreign bodies in the esophagus can perfectely mimic pulmonary or mediastinal masses; you often cannot tell the difference with plain radiographs. If poor contrast in the region of the esophagus, pleural effusion or pneumothorax are seen, one must seriously consider esophageal perforation and mediastinitis. If plain films are not diagnostic, then contrast films can be performed. Barium provides better contrast, but iodide is safer if there is an unsuspected perforation. Esophageal perforation may occur at variable times after ingestion of a foreign object. Even a blunt object, if tightly lodged in the esophagus, can cause ischemia and perforation in 2-3 days. The prognosis for animals with esophageal perforation and severe mediastinitis is guarded to poor, depending upon their condition at the time of diagnosis.

Endoscopy is almost always the preferred method of removing foreign objects, but fluoroscopic and surgical techniques can be effective if the operator is well trained. Rigid endoscopes allow much more control of the foreign object and are preferred to flexible scopes for removal of these foreign objects. It is especially useful to be able to pull the object into a rigid endoscope and then withdraw it and the scope as a unit, thus protecting the esophagus. The main disadvantage of rigid endoscopes is that they are often not long enough in larger dogs. Finesse is required; brut force can easily lacerate/perforate the esophagus. If a large object or a bone cannot be easily dislodged, do not force it lest you perforate a previously intact esophagus; instead, you can use rigid equipment to "chew" it up and hopefully dislodge it. If that fails, passing a large Foley catheter behind the foreign object and inflating the balloon often helps; it distends the esophagus (thus freeing the foreign object) and then is used to pull the object out. If you cannot pull a foreign object out of the esophagus, you can try to push it into the stomach. However, do not push bones or other foreign objects into the stomach unless you are sure that it is smooth on the aborad side and will not further damage the mucosa. Finally, be careful if you insufflate the esophagus lest you rupture a weakened area in the mucosa and/or cause a fatal tension pneumothorax.

Fish hooks terrify many clinicians, but they can often be successfully removed endoscopically. Fish hooks have usually penetrated the mucosa (and sometimes the muscular tunics); you will often have to use rigid equipment to carefully force the tip of the hook back out of the mucosa. A small hole is left, but there are very seldom any complications. After removing the foreign object, retake plain chest radiographs to be sure that a pneumothorax (which would indicate a perforation) is not present. Antibiotics are indicated if there is substantial esophageal mucosal ulceration (and especially if you remove a fish hook which had been used with various baits that can harbor anaerobic bacteria). Depending upon the amount of damage, corticosteroids may be used to try to prevent cicatrix formation; however, it is not clear that they are effective. Rarely there may be severe hemorrhage.

Primary esophageal carcinomas occur rarely although gastric carcinomas may spread into the lower esophagus. Most primary esophageal tumors are asymptomatic until they become very large. We have diagnosed a few primary esophageal tumors fortuitously when routine chest radiographs revealed a density in the diaphragmatic lung fields. Esophageal sarcomas are usually due to Spirocerca lupi, which is discussed below. Most esophageal tumors are secondary to mediastinal or thyroid tissues, and they cause esophageal obstruction by extramural pressure. Thyroid carcinomas may also invade the esophagus. The prognosis is usually poor.

Leiomyomas of the lower esophageal sphincter are occasionally seen. Endoscopy is the best tool to find them. They are best seen when one looks at the lower esophageal sphincter from the stomach using a retroflexed view. It is worth looking for these tumors as they are potentially curable with timely surgery.

Cicatrix (i.e., scarring) may occur after an episode of severe esophagitis from any cause (including foreign objects). It is particularly easy to miss this problem on a barium swallow if only liquid barium is used. If radiographs using liquid barium are nonrevealing, repeat the study using barium mixed with food, which is more likely to stop at a partial obstruction. Endoscopy is very good at finding these lesions; however, you must keep in mind the size of the patient as you evaluate the esophageal lumen. A partial stricture will be very obvious in a 10 lb dog or cat but may not be apparent in an 85 lb animal. Balloon-dilatation or bouginage is usually effective; it is also more likely to be successful than surgery and resection of the affected area. In general, surgical resection should be a last ditch resort and only used if esophageal ballooning or bouginage has failed despite repeated dilatations. However, you must use proper esophageal balloons because Foley catheters and endotracheal tubes with inflatable cuffs will often not allow you to dilate a dense or mature stricture. More difficult cases (i.e., those with extensive strictures or with concurrent severe esophagitis) may benefit from a couple of techniques. Endoscopic administration of intralesional steroids may help minimize reformation of the stricture. Typically we put 1-2 ml of Vetalog at the site of the stricture either before or after ballooning. Another technique is to make 3-4 equidistant cuts into the stricture using an electrocautery device (i.e., either a snare or a knife) prior to ballooning. This helps the stricture to "break" open at multiple spots with the idea that there will be 3 or 4 smaller, less deep lacerations at the stricture site instead of one major, deep laceration which is more likely to restricture. However, you should not attempt to use cautery through an endoscope unless you have some training less you cause too much trauma to the tissues or destroy your endoscopic equipment.

Another technique is to "paint" the site where the stricture was broken down with Mitomycin C (NOT mithromycin C, there is a difference). A 5 mg bottle is reconstituted and soaked up into a gauze sponge. Then this sponge is endoscopically placed on the site where the stricture was broken up for 5 min. Then it is flushed off with 60 ml of water.

Finally, for particularly difficult cases, stents may be placed in the esophagus. These must be sutured in place. The stents are made by Infiniti corporation (http://www.infinitimedical.com/p_stents.html) and the suture device is made by Pare Surgical Inc (http://www.infinitimedical.com/p_stents.html). The major point to remember is that if an animal starts to have problems days to weeks after anesthesia, consider strongly the possibility that an esophageal stricture has developed secondary to esophagitis. If you are treating an esophageal stricture, remember that you may need to do 1-15 dilatations. If esophagitis is diagnosed, you need to treat it aggressively in order to help prevent the stricture from recurring quickly.

Acquired esophageal weakness is usually (but not always) easy to distinguish from obstruction radiographically, especially when a barium contrast radiograph is performed. However, the severity of the radiographic lesion (i.e., the degree of dilatation) does not always correlate well with the clinical severity. Acquired esophageal weakness is typically difficult to resolve because it is hard to find the underlying cause.

Myopathy, neuropathy, myasthenia gravis, dermatomyositis, dysautonomia, esophagitis, Addison's disease, *Spirocerca lupi*, tick paralysis, central nervous system disease, or infiltrative non-obstructive esophageal tumors are possible causes. Generalized myopathies and neuropathies often affect the esophagus because it is composed of striated muscle in the dog. Signs of lower motor neuron disease in these patients are sometimes seen and can include loss of muscle mass, weakness, an inability to bark, or a change in the quality of the bark. Some clients report that their animal has laryngitis, which may seem likely because these pets typically have repeated respiratory infections due to aspiration pneumonia. Treatment of the myopathy or neuropathy should resolve the problem, but symptomatic therapy for the esophageal dilatation is indicated.

Generalized myasthenia gravis usually presents as weakness during exertion which resolves after resting; however, generalized myasthenia can present in a variety of ways, including apparent lameness or permanent weakness. Electromyography and assay for circulating antibodies to acetylcholine receptors are the most definitive tests. Localized myasthenia in the dog is a syndrome in which the esophagus is the only muscle which is obviously weak. Up to 25-30% of dogs with acquired esophageal weakness have this syndrome. Third degree heart block may also be seen in some patients with megaesophagus due to myasthenia. This is diagnosed in dogs with esophageal weakness by detecting serum antibodies to acetylcholine receptors. The antibodies are relatively stable and require little special handling other than refrigeration. If myasthenia is strongly suspected but the titer is negative, it can be valuable to repeat the titer as they sometimes seroconvert later. You cannot perform an edrophonium response test to diagnose localized myasthenia. Myasthenia gravis will sometimes spontaneously resolve. Treatment for myasthenia gravis that does not spontaneously resolve may include anti-acetylcholinesterase drugs, corticosteroids and/or cytotoxic agents. Azathioprine and mycophenolate seem to be effective drugs for this purpose. In general, we try to avoid steroids as they seem to be associated with more problems. In really severe cases, we can place a percutaneous gastrostomy tube to support the patient and lessen aspiration whle waiting for the drugs to have an effect. However, a gastrostomy tube will not prevent all aspiration as the dog is still swallowing saliva which can be regurgitated and aspirated.

Hypoadrenocorticism may be responsible for causing esophageal weakness, even when the serum electrolytes are normal. This is especially true in standard sized, black poodles, but it can occur in any breed. Treatment for hypoadrenocorticism is steroids, which can make the esophagus start functioning again. However, if your diagnosis is wrong and you give steroids because you suspect the dog may have hypoadrenocorticism, all you are doing is making aspiration pneumonia and subsequent death that much more likely.

True achalasia of the lower esophageal sphincter (i.e., failure of the sphincter to open), although distinctly rare, should be considered. At first everybody thought that megaesophagus in the dog was due to achalasia. Then we found out that dogs were different than people and we all forgot

about achalasia. Now we find out that it does occur, but very rarely. This is one reason why it is useful to perform a barium contrast esophagram in an animal with an obvious acquired megaesophagus. Failure of barium to pass from the esophagus to the stomach despite vertical positioning is suggestive of this rare, but potentially curable disease. Fluoroscopy and esophagoscopy are very useful in diagnosing this problem and eliminating other causes of esophageal retention. Even though achalasia is an obstructive disease, its distal location causes it to mimic generalized esophageal weakness on static image barium contrast esophagrams. This disease can be cured by lower esophageal cardiomyotomy. However, if this surgery is done incorrectly, it can cause gastroesophageal reflux with severe esophagitis and subsequent regurgitation. Therefore, this surgery should only be done by individuals well acquainted with it.

Idiopathic megaesophagus (i.e., either congenital megaesophagus or acquired megaesophagus for which a cause cannot be found) can only be treated with symptomatic therapy, which usually consists of feeding the animal 3-4 meals of gruel from an elevated platform and making the pet remain in the near vertical position from 5-10 minutes after eating. Near-vertical means just that. It is useless for the dog to just lift its head up while eating; it should be standing on its back legs. If necessary, use a portable ladder or put the dog in a large trash can to help it remain vertical during this time. This approach is a time-honored treatment, but it does not always work. Some animals with idiopathich esophageal weakness are controlled as well (or better) if they are fed free-choice dry food from an elevated platform. Some can even be fed from the floor. Free-choice feeding encourages the pet to eat small amounts of food throughout the day, thus avoiding intermittent large meals which are more likely to be retained and further dilate the esophagus. If there is any esophageal motility remaining, the dry food may be easier for the esophagus to propel then gruel. It is difficult to predict which feeding regime will work best for a particular patient, and both of these feeding regimes may need to be tried. While most dogs with idiopathic megaesophagus die from aspiration, there are enough of them that respond well that it is very much worth trying. A reasonable percentage of dogs with idiopathic, congenital megaesophagus will spontaneously improve and have normal or near normal function. You cannot predict response to therapy or spontaneous remission; all you can do is support the patient and see what happens.

Some individuals have tried using cisapride in selected patients with idiopathic esophageal weakness that do not respond well to nutritional modification. Theoretically, cisapride would not be expected to work in these animals because cisapride primarily works on smooth muscle and canine esophagus is striated muscle. Furthermore, cisapride is expected to tighten up the lower esophageal sphincter, thus making it harder for food to pass out of the esophagus and into the stomach. Perhaps cisapride helps patients when gastroesophageal reflux is part of the problem.

Some owners elect to have a permanent gastrostomy tube placed in the patient. This will not eliminate all regurgitation or aspiration, because the patient is still swallowing saliva which will remain in the esophagus until it

is regurgitated. However, gastrostomy tubes will help eliminate most of the regurgitation and can markedly prolong such a patient's quality, comfortable life.

Aspiration pneumonia is a major problem and cause of death in dogs and cats with esophageal weakness causing regurgitation. If the respiratory disease cannot be stopped by alleviating the regurgitation by dietary therapy, then it must be controlled by antibiotics. A transtracheal wash with cytology and culture will help identify optimal antibiotics. Until culture results are known, use of broad-spectrum, bactericidal drugs (i.e., amikacin plus either cephalothin or amoxicillin; enrofloxacin plus amoxicillin or clindamycin) are used. In severe cases of aspiration pneumonia, one may have to bypass the esophagus with a gastrostomy tube to prevent further aspiration. These tubes can be place with the aid of a flexible endoscope and be used for days to months.

Esophagitis is much more common than many clinicians are aware. The difficult partly arises from the fact that esophagitis can present with clinical signs that lead one to believe the dog is vomiting instead of regurgitating. Furthermore, mild esophagitis may only cause minor signs (mild regurgitation of mucus and phlegm) while severe esophagitis can cause so much pain that patients refuse to swallow water or even saliva. Because there can be so wide a range of clinical signs, it is easy to forget that esophagitis is a differential for a patient. It is critical to identify that esophagitis is present as delayed diagnosis can have serious clinical repercussions. Substantial inflammation of the esophageal mucosa causes muscular weakness by interrupting the reflex arcs within the esophagus and/or between the esophagus and the brain. However, this weakness is not always reflected by finding megaesophagus. Most patients have very minor esophageal distention and yet can have major signs. Likewise, barium esophagrams can have relatively minor changes and not reflect the severity of the esophagitis. Esophagoscopy typically shows an edematous, reddened, bleeding esophageal mucosa, ± structure formation, making it the diagnostic method of choice to find esophagitis. However, in rare cases, there may be more subtle changes with thickening and discoloration (especially at the lower esophageal sphincter of cats).

Adding to this problem is the fact that there is such a wide range of causes of esophagitis. Severe esophagitis may be caused by anesthetic procedures in which animals are placed in dorsal recumbency and then have gastric acid pool in their esophagus for relatively long periods of time. However, gastroesophageal reflux from any cause can be responsible. Hiatal hernias occasionally are responsible for such reflux. Rare animals ingest caustic substances (e.g., lye), and some cats will like caustic disinfectants off their fur. However, a surprisingly large number of animals are administered caustic substances by veterinarians. In particular, tetracyclines, NSAIDs, ciprofloxacin and clindamycin are recognized as having substantial potential to cause esophagitis. Pills and capsules are notorious for lodging in the esophagus of cats, and it is therefore not surprising that doxycycline is a recognized cause of esophageal stricture in cats. Esophagitis may also be secondary to any cause of protracted vomiting. In particular, parvovirus enteritis may cause such intense vomiting

that esophagitis results. If a vomiting animal has the character of its vomitus change, which seems to suggest regurgitation, consider the possibility that esophagitis has occurred secondary to the persistent vomiting. Gastrinoma (a tumor which secretes gastrin and results in massive gastric acid secretion) also causes esophagitis because of the vast and unending amounts of acid the esophagus is exposed to as the dog continually vomits. Gastroesophageal reflux may be potentiated by or even caused by esophagitis (which may be caused by reflux in the first place). Thus, there may be a positive feedback loop which can be hard to break (i.e., esophagitis causes more reflux which causes more esophagitis which causes more reflux which causes ...). Rarely there can be spontaneous inflammation, as seen with eosinophilic esophagitis in dogs. Brachycephalic dogs seem to have an increased incidence of gastroesophageal reflux, esophagitis and perhaps hiatal hernia. Finally, esophageal foreign bodies typically cause varying degrees of esophagitis. The esophagus is far more susceptible to pressure necrosis from a foreign body than are the stomach or intestines.

You should seek to prevent further gastroesophageal reflux by keeping the stomach as empty as possible by using prokinetics such as metoclopramide or, preferably, cisapride. Studies in people show that cisapride is clearly more effective than metoclopramide. The only real advantage of metoclopramide is that it can be given by injection; a useful fact in animals that are regurgitating profusely. In addition, gastric acid secretion should be minimized and preferably abolished. H-2 receptor antagonists (e.g., cimetidine, ranitidine, famotidine) suppress gastric acid secretion, but they do not eliminate it. This is because they are competitive inhibitors. That means that there is constantly some degree of competition between the H-2 receptor antagonists and the stimuli for acid secretion. Omeprazole, lanosprazole, pantoprazole and esomeprazole are non-competitive inhibitors of gastric acid secretion. Therefore, these drugs can be noticeably more effective and for much longer than the H-2 blockers. You can try to achieve greater efficacy with the H-2 receptor antagonists by doubling or tripling their dose, but the proton pump inhibitors are usually more effective.

Sucralfate is of uncertain value in patients with esophagitis. Unless there is some gastric acid reflux into the esophagus (which you are desperately trying to stop in the first place), it is doubtful that the sucralfate is of much use. If you use it, it should be administered as a slurry.

A combination of omeprazole and cisapride seems to be the most effective medical treatment regime. Antibiotics are used to treat secondary infections, but nobody really knows if they do anything in this regard. Glucocorticoids have been thought to help retard fibrous connective tissue proliferation and cicatrix, but their effectiveness is uncertain (and they might predispose to infection). Placing a PEG tube seems to have some real advantages in patients with very severe disease. First, we will then know that the cisapride and omeprazole tablets will reach the stomach. Second, we will also know that the animal will receive its caloric and protein needs, and hopefully with less irritation to the esophagus than would have occurred otherwise.

If there is severe esophagitis, cicatrix may form and obstruction develop subsequently. Diagnosis of stricture is best accomplished by esophagoscopy IF the operator is familiar with such obstructions. It is surprisingly easy to pass a slender endoscope through a stricture and never recognize the stricture. It is also surprisingly easy to miss a partial obstruction due to a stricture with a barium esophagram. If you suspect a stricture and must use a barium esophagram to make the diagnosis, use barium mixed with solid food. Balloon-dilatation or bouginage is recommended if a stricture has occurred. Many animals need to have 2-6 dilatation procedures (all the while being treated for esophagitis), although some only need one procedure and some need more than 15. Do not try to resect the stricture unless you have had prior dilatation procedures fail.

Hiatal hernias may be more common than suspected. Shar Pei's seem to have a relatively high incidence of hiatal hernias. They can be difficult to diagnose unless you know how to look for them. Sometimes seen on plain radiographs and simple barium contrast radiographs, the more occult cases sometimes need more aggressive diagnostics. Sometimes one must manually put pressure on the abdomen during film exposure to try to push the stomach through the hernia and into the chest so that it can be diagnosed radiographically. Endoscopic diagnosis is not always straightforward. You may need to put the endoscope into the stomach and retroflex it in order to see the abnormality. Even when found, the big question is whether the hiatal hernia is causing a problem or is an "innocent bystander". In particular, if you have an older dog or cat (i.e., > 1-2 years old) that just started having clinical signs, you should strongly consider that the hiatal hernia is a fortuitous finding that is not responsible for the clinical signs.

Gastrinoma is another cause of esophagitis. Dogs with this tumor usually vomit profusely and thus burn the esophagus by the large volumes of gastric acid which pass through it. Most affected dogs have vomiting, diarrhea, and weight loss. Increased serum gastrin concentrations are necessary for diagnosis. Aggressive H-2 receptor antagonist therapy can be used, but omeprazole is preferred.

Breed predispositions are important. English bulldogs and Shar Pei's commonly have a redundant esophagus in front of the heart. It is important that diagnosticians recognize this as a dramatic, but usually insignificant finding.

Esophageal parasites and non-obstructing tumors can cause megaesophagus by infiltrating the esophagus and disrupting motility. Thyroid carcinomas often do this because they are invasive and in close proximity to the esophagus. Interestingly, small Spirocerca lupi lesions may do likewise with the degree of dilatation appearing excessive in view of the relatively small parasitic granuloma(s) present.

PANCREATITIS

Michael D. Willard

Diagnosis

History and physical examination are helpful, but are not as useful as we'd like for diagnosing pancreatitis. Schnauzers and Yorkies are famous for pancreatitis, but these breeds get a lot of other diseases that cause vomiting, and pancreatitis can be found in any breed of dog. Canine pancreatitis is classically considered to present with acute vomiting and anorexia. Abdominal pain is frequently present, but it is easy to miss during physical examination, and fever is occasionally seen. However, we are recognizing more and more "atypical" cases to the point that many of us joking say that we are no longer sure what a "typical" case of canine pancreatitis is. We are now recognizing more and more cases of severe disease which present in shock due to systemic inflammatory response syndrome (what used to be called septic shock, until we found out that you can have the same thing occur with any cause of massive inflammation); such patients may die very suddenly. We are also recognizing more and more dogs with acute pancreatitis that present as though they had an acute, septic abdomen. Some have substantial amounts of abdominal fluid. If acute pancreatitis is associated with or due to pancreatic carcinoma (rare), you may also see a dog that has widespread subcutaneous fat necrosis causing sterile abscesses that are typically painful and cause cutaneous discoloration. Most cases of canine pancreatitis are related to either ingestion of fat or lipemia associated with diabetic ketoacidosis. Trauma and drugs can also cause canine pancreatitis. Drugs that are suspected of causing pancreatitis in people and animals include azathioprine, sulfonamides, tetracycline, and potassium bromide.

CBC's often show an inflammatory leukogram, but 1) this is a relatively nonspecific finding and may be due to any number of problems and 2) not all animals with acute pancreatitis have a notable leukocytosis. Degenerative left shifts and substantial toxicity of circulating WBCs can be seen if the patient is in systemic inflammatory response syndrome. Likewise, thrombocytopenia due to DIC is not infrequent in severely affected patients. However, some animals with clinically severe pancreatitis have absolutely normal leukograms.

Serum biochemical panels are not as helpful as we would like. At the time of this writing, there is no readily available biochemical test that has good positive or negative predictive values. Serum lipase and amylase activities are insensitive (each is about 50%, and that is optimistic) and nonspecific for pancreatitis and should probably never be requested. Dogs with acute pancreatitis and even pancreatic abscesses have had normal serum lipase activities. We have also identified dogs with drastically increased serum lipase activities that have intestinal foreign objects or gastritis, but no gross evidence of acute pancreatitis. Lipase is produced by the canine gastric mucosa which explains why inflammation or damage to the stomach can result in excessive serum lipase activity. Canine TLI is a little more specific than amylase and lipase, but it is still not a sensitive test

(approximately 35%). Therefore, it too has very poor negative predictive value. We have seen plenty of dogs with pancreatitis that had normal serum TLI's.

The immunoreactive canine pancreatic lipase assay (i.e., cPLI or Spec cPL) appears to be the most sensitive (approximately 80-85%) test for pancreatitis available. There are a few false negative results with this test, but it is clearly much more sensitive than any other blood test available. The real question is how specific it is for **clinically significant** disease (i.e., lesions of the pancreas that are causing clinical disease as opposed to microscopic lesions that are clinically silent). The biggest advantage is that if the cPLI test is negative, it is much less likely that pancreatitis is the real problem and you need to look very hard for extra-pancreatic disease in the dog.

Blockage of the main pancreatic duct due to swelling due to generalized pancreatitis, an intrapancreatic granuloma, or an abscess that subsequently blocks the pancreatic duct may cause extrahepatic biliary tract obstruction (EHBO) with a notable increase in serum alkaline phosphatase and serum bilirubin. Pancreatitis is probably the most common cause of EHBO in the dog. Thus, while EHBO is very suggestive of acute pancreatitis (assuming that the patient does not have a mucocoele, which is usually easy to detect with ultrasound), relatively few dogs with acute pancreatitis develop EHBO. Furthermore, there are reasons for this triad of signs besides acute pancreatitis and extrahepatic biliary tract obstruction (e.g., cholangitis-cholangiohepatitis). Ultrasonographic evaluation of the abdomen (discussed below) is particularly helpful in these patients.

Plain abdominal radiographs help eliminate other diseases which may mimic acute pancreatitis. Not finding evidence of other abdominal disease (e.g., foreign object) is helpful in eliminating obstruction and narrowing the list of differential diagnoses. Occasionally, one finds radiographic signs which specifically suggest acute pancreatitis: A sentinel loop (i.e., a dilated, air-filled segment) in the descending duodenum, lack of serosal detail in the upper right abdominal quadrant, lateral displacement of the descending duodenum on the ventro-dorsal projection, a mass medial to the descending duodenum (on the ventro-dorsal projection) and/or a mass just behind the liver and just below the pylorus (on the lateral projection) can be suggestive of pancreatitis. These findings are only meaningful if present; many dogs and cats with acute pancreatitis do not have these radiographic findings. Probably the most greatest value of abdominal radiographs is that they help eliminate other diseases that could be causing signs similar to those caused by pancreatitis.

Abdominal ultrasonography often finds abnormalities that suggest or are consistent with pancreatitis as well as eliminate other potential causes of vomiting and abdominal pain. Depending upon the ultrasonographer, it is about 40-70% sensitive in finding canine pancreatitis. One may sometimes detect hypoechogenicity surrounded by hyperechoic fat in the region of the pancreas that is due to pancreatitis. At other times, a markedly thickened pancreas may be found. Both findings are very

specific evidence of pancreatitis. Evidence of EHBO (i.e., dilated bile ducts, not just a big gall bladder) is very suggestive of pancreatitis. Rarely, you will find dilated bile ducts due to inflammatory biliary tract disease, but this is not nearly as common a cause as is biliary tract obstruction. Any dog with extra-hepatic biliary tract obstruction and any vomiting/anorexia should be assumed to have pancreatitis until proven otherwise. It is important to note that the ultrasonographic appearance of the pancreas can change dramatically within a few hours, so repeating abdominal ultrasound on the same day is not necessarily a bad idea if you strongly suspect pancreatitis but are finding nothing. Ultrasonography (assuming that it is performed by an accomplished operator – possibly no other diagnostic procedure is so operator dependent as ultrasonographer) currently seems to be the fastest test with good specificity for pancreatitis in the dog (but not necessarily the cat). However, ultrasonography is not perfect: we have seen several dogs with very severe pancreatitis that did not have ultrasonographic evidence of pancreatitis. At this time, a combination of abdominal ultrasonography and cPLI seems to be the best way to diagnose canine pancreatitis.

At this time, the combination of cPLI and abdominal ultrasound seems to be the best combination we have when looking for pancreatitis. The ultrasound can give you a quick answer, and failure to find pancreatitis on ultrasound is a good reason to submit a cPLI.

Diagnosing pancreatitis during laparotomy is the least desirable means of diagnosis. Some patients present exactly like acute septic peritonitis but are ultimately diagnosed as having non-septic pancreatitis. There is nothing wrong with doing an exploratory laparotomy in a patient in which septic abdomen is a major consideration, only to find out that the patient has non-septic pancreatitis. We very rarely have reason to biopsy a normal appearing canine pancreas. Furthermore, obvious pancreatitis in the dog seldom requires a biopsy unless carcinoma is a possibility. However, you should never simply look at what appears to be an obviously neoplastic mass in the pancreas and make a diagnosis of carcinoma without biopsying it -- no matter how extremely terrible it appears. Pancreatitis is much more common than pancreatic carcinoma, no matter how bad the pancreas looks or how many adhesions are present. If you biopsy the pancreas, it is important to obtain a biopsy that goes deeper than the superficial necrotic surface or adhesions. Cytology can be useful for making a presumptive diagnosis; however, I have seen at least one case in which cytology of a pancreatic mass was read out as carcinoma by two accomplished cytologists and yet multiple biopsies all came back as necrotic pancreatitis.

Chronic pancreatitis (i.e., chronic pancreatitis with intermittent, relatively mild recurrences) can be challenging to diagnose. Dogs with episodic vomiting due to recurrent bouts of pancreatitis may not have any other signs of disease, and they invariably are admitted to your clinic for a work up after the last bout has run its course or is on the mend. Episodes of vomiting and anorexia due to recurrent pancreatitis can be random and unpredictable. In such patients, the previously mentioned diagnostics may be attempted, especially when acute exacerbations occur. Very rarely,

upper gastrointestinal barium contrast radiographs may rarely reveal duodenal abnormalities (e.g., dilatation, stricture) which suggest that recurrent bouts of acute pancreatitis have caused scarring of the pancreas which in turn have compromised the maximum size of the duodenal lumen. Ultrasonographic changes are nice if they are present, but they can be minor making it difficult to accurately interpret them. Feeding an ultra-low fat diet for 3-4 times longer than what was previously the longest interval between episodes may be helpful in making a presumptive diagnosis. If episodic vomiting/anorexia does not recur while feeding such an ultra-low fat diet for an interval so long that you would have been sure to experience another episode, then we can often reasonably assume that the signs were due to pancreatitis (or perhaps some other dietary-responsive disease). The cPLI test may be useful for diagnosing this form of pancreatitis. In particular, anytime you find exocrine pancreatic insufficiency (diagnosed with TLI) in a breed that is not commonly affected with pancreatic acinar atrophy (e.g., German shepherd, rough-coated Collie), then chronic pancreatitis becomes a major concern.

Pancreatic abscesses in dogs (as opposed to cats) are invariably sterile. Affected dogs typically can have a much more chronic, smoldering course (e.g., vomiting for a month or more, mild loss of appetite) than most dogs with acute pancreatitis. We have even found a few dogs which had abscesses that were completely asymptomatic. Abdominal pain may be present or absent. CBC and serum biochemistry findings are unpredictable. Diagnosis requires ultrasound. Treatment may be surgical marsupalization, percutaneous ultrasonographic drainage or just observation.

Therapy

Nobody actually knows what is and what is not effective for canine pancreatitis. As of this writing, there is not a single, well designed, prospective, stratified study on the treatment of pancreatitis. Therefore, all any of us has is opinions, period.

Nothing per os (NPO) has been the classic therapy for pancreatitis for many years. While it is true that they feed people with pancreatitis earlier than we feed dogs, you must remember that human pancreatitis is unassociated with dietary fat. People get pancreatitis from alcohol, trauma, gall stones and MOF (multiple organ failure). Canine pancreatitis is associated with dietary fat (as well as surgical trauma when poor technique is used around the pancreas). Some preliminary work has been done in Australia that suggests that it is fine to feed dogs per os with pancreatitis, even if they are still vomiting. Time will tell if this is right or not. Based upon their work it appears reasonable to feed dogs with pancreatitis, but I recommend that a) you feed as low a fat content as possible, and b) if the feeding is associated with worsening of the vomiting, that you stop it and go to IV or jejunostomy feeding. Do not try to get full caloric intake into the patient; rather, start with small amounts to see if the patient will hold down the food. Obviously, if feeding is associated with worsening of the vomiting or general condition, stop the feeding. I

generally start feeding potato or rice (i.e., no fat) and gradually work my way up to commercial diets with low fat content.

Fluid therapy is critical, and subcutaneous administration of fluids is clearly inferior to IV fluids for all but the mildly affected animals. IV fluid administration plus NPO is often sufficient, even in dogs in which a pancreatic granuloma has temporarily blocked the main bile duct. Adequate pancreatic circulation is probably crucial for healing damaged pancreatic tissue; therefore, it is far better to provide a little too much fluid rather than a little too little fluid unless the patient has congestive heart failure or oliguric renal failure. The abdominal viscera is not "first in line" to receive circulation when the patient is dehydrated (which most dogs with pancreatitis are when they come to your office). Obese and fat dogs (which describes a lot of dogs with pancreatitis) do not necessarily have skin tenting when they are dehydrated. Likewise, although you might expect dry, tacky oral mucus membranes, a nauseated animal may be salivating enough to make the mucus membranes moist even though it is dehydrated. If the dog is not eating or drinking and is vomiting, it is dehydrated regardless of how well hydrated it appears on physical examination. All that being said, if you give far too much crystalloid and dilute the serum protein concentrations, this could be detrimental.

One should monitor the serum albumin concentration during fluid therapy in these patients. If the serum albumin concentration decreases significantly, then the plasma oncotic pressure likewise decreases which diminishes the effective perfusion at the capillary level. Since perfusion is so critical to treating dogs with pancreatitis, one should probably become concerned whenever the serum albumin concentration falls below 2.0 gm/dl. The most common error in administering plasma is to administer too little to significantly raise the plasma albumin concentration. Remember that half of the albumin that you administer will end up extravascular instead of being in the intravascular compartment. If you are going to spend the money to administer plasma, you need to monitor the serum albumin concentration after administering the plasma to see if you have meaningfully affected the values. Hetastarch is probably helpful since it will improve plasma oncotic pressure and help microcirculation in patients that are becoming hypoproteinemic.

Administration of plasma might be more effective than administering hetastarch because plasma also restores circulating protease inhibitors and replenishes AT III (which is a treatment for DIC). This is a very contentious point. One retrospective study has shown that plasma did not help treat dogs with pancreatitis; however, this study suffers from the problems inherent in all retrospective studies.

Total parenteral nutrition (TPN) seems like it can be useful in patients with severe acute pancreatitis. However, it is expensive, labor-intensive, and can only be done in facilities where there is 24 hour coverage by trained individuals. Partial parenteral nutrition (PPN) is something that almost anyone can use in practice. The goal is to provide approximately ½ of the caloric requirement by using a combination of D5W, 8.5% amino acids plus electrolytes, and 20% lipid emulsion. One typically provides approximately 1/3 of the desired calories with each of the three

ingredients, and then administers the solution through a peripheral catheter. Much less monitoring is required with PPN than with TPN. In general, PPN is used for 5-7 days to help the patient "get over the hump"; it is not usually intended to be used for more than a week. The best source of information on partial parenteral nutrition is: *Compendium of Continuing Education* 21: 512, 1999.

Enteral nutrition is another option that has advantages over parenteral nutrition: it is easier, less expensive, and less dangerous. In particular, it should be considered if an exploratory laparotomy was performed when the pancreatitis was diagnosed because a J-tube can be placed at that time. Alternatively, one can place a jejunostomy tube via laparoscopy, through a G-tube, and via the nose (naso-jejunostomy). Finally, we are playing with endoscopy-assisted placement of naso-jejunostomy tubes. Supplying adequate nutrition seems to be very valuable, especially in cases requiring longer therapy.

Antiemetics are useful in patients that are so nauseated that they feel terrible. I prefer to only use antiemetics for short periods of time because I want to see if the patient is improving enough so that it no longer needs the antiemetic to stop vomiting. However, if the patient is vomiting multiple times per day or obvious feels terrible due to the nausea, then maropitant (1 mg/kg SQ) appears to be very useful. Dolasetron (0.3-1.0 mg/kg qd) and ondansetron (0.25 mg/kg qd) can also be effective.

H-2 receptor antagonists can be used to treat dyspepsia; ulceration and erosion are very uncommon in most pancreatitis patients. I primarily use these drugs to help the antiemetics be more effective. I prefer famotidine (0.5 mg/kg qd) although some prefer ranitidine because it has prokinetic activity.

Antibiotics have been used to prevent infection of the inflamed pancreas which is supposed to be "fertile ground" for infection, but there is minimal evidence that infection is of any significance in routine canine pancreatitis. Antibiotics do not hurt these patients, but it is very questionable how helpful they are. However, dogs in SIRS due to pancreatitis are a different story. Any dog in SIRS from any reason is at increased risk of infection due to severely compromised mesenteric circulation.

Drugs designed to decrease pancreatic secretion have been disappointing, which is not surprising when one considers that acute pancreatitis may be associated with pancreatic hyposecretion instead of hypersecretion.

Corticosteroids are very controversial in the treatment of pancreatitis. First, while they increase serum amylase and lipase activities, they do not cause pancreatitis. It is possible that they may be useful in treating patients that are in Systemic Inflammatory Response Syndrome (i.e., SIRS, which used to be called "septic shock") due to the pancreatitis. At this time, there are good data showing that it is probably reasonable to give physiologic doses because dogs in SIRS typically are relatively hypoadrenal. This is a controversial statement. If steroid therapy for pancreatitis is contemplated, it should probably be reserved for the severely ill dog which is not responding to fluid resuscitation. If steroid therapy will be

administered, you need to warn the owners of the unknown nature of this therapy. A larger question is whether steroids may be useful in treating the inflammation found in severe pancreatitis. We have some minimal evidence at this time that anti-inflammatory doses of steroids might (?) be helpful in some patients. At least, the steroids did not overtly hurt the patient, and it got better (either because of or inspite of the steroids).

Although heparin therapy would seem to be helpful to treat early DIC (which can probably make acute pancreatitis worse), it has not been shown to be useful in treating acute pancreatitis. If DIC appears to be a major problem, aggressive administration of fresh frozen plasma to replace clotting factors and anti-thrombin III concentrations is probably more effective than heparin in treating DIC.

Analgesics can be very useful in animals with substantial abdominal pain. In very severe cases, a constant rate infusion of Fentanyl is very effective. For extreme cases, a constant rate infusion of fentanyl, lidocaine and ketamine appears to give the best analgesia. In less severe cases, buprenorphine given as needed may be used instead.

Taking a dog with pancreatitis to surgery is probably one of the hardest decisions in small animal internal medicine. Strict guidelines cannot be given, but some basic principals may be suggested. In general, one must realize that unless an abscess, pseudocyst, mass of necrotic tissue, and/or an obstructed gall bladder with a bacterial infection are found, surgery will probably not benefit the patient (and may even be detrimental -- anesthesia is usually associated with some decrease in visceral perfusion unless great care is taken to maintain circulation). The basic idea is to try to find reasons why you should NOT do surgery, only doing surgery if you cannot find any good reason to avoid it. Ultrasonography usually finds the type of lesions that would be benefitted by surgery (i.e., abscess or pseudocyst). If one is uncertain whether or not a cyst or abscess might be present and might be responsible for appropriate medical therapy being ineffective, one may reasonably decide to explore the abdomen. However, this decision should be based on the finding that 5-7 days of excellent, aggressive supportive medical management has not been of any appreciable benefit. The best way to tell if the therapy has been of any benefit is to look at the patient; if the patient looks better but the blood tests do not, go with the clinical appearance.

Again, do not be in a hurry to take dogs with pancreatitis to surgery; but, if you need to do surgery, then you should do it without any further delay. That is why it is important to provide the best possible medical management when first confronted with the patient with suspected acute pancreatitis. I will usually try aggressive medical therapy for 5-8 days, depending upon how quickly the patient is decompensating. If I have no indication that optimal medical therapy is helping after 7-9 days, then I will seriously consider surgery. If the patient has an EHBO, I generally will wait a longer time (i.e., a week or more, assuming that the patient is improving) before I consider surgery to relieve the obstruction. It seems as though just about all of these patients will eventually have the EHBO resolve if treated medically for long enough. If it appeared important to relieve EHBO, then percutaneous drainage using ultrasound guidance might be appropriate.

Rarely a stent can be placed in the bile duct. If a pseudocyst or abscess is not found, lavaging the area and resecting obviously necrotic tissue might help the patient, but this is uncertain.

Prognosis is difficult to predict. Hyperbilirubinemia is not necessarily a poor prognostic sign; pancreatic granulomas causing icterus due to obstruction of the bile duct typically resolve if the patient receives appropriate supportive therapy. Hypocalcemia, while used to prognosticate in people, is infrequently found in dogs and cannot be used to predict the outcome. Pancreatic abscesses are usually sterile. We cannot correlate the ultrasonographic appearance of the pancreas with prognosis. Finding a degenerative left shift and/or a marked thrombocytopenia (probably due to DIC) are not known to be prognosticators, but one intuitively fears that a poor outcome is more likely with such findings. Evidence of SIRS is probably the most important reason to give a poor prognosis.

When to send the patient home is the last question. The patient is sent home when it is doing well. Monitoring the ultrasonographic appearance or the cPLI or the lipase or whatever seems to be much less valuable than observing the patient. If the patient feels good and is eating and is not vomiting, then you can probably send it home regardless of what the ultrasound looks like or how high the cPLI is. When the dog is sent home, it should be on a very strict diet that is free of fat . If you tell them "fat free" and explain the hidden sources of fat in the diet, then maybe you will achieve "low fat". Also, if the dog ever gets sick again, they should come in and see you sooner rather than later, in case it is pancreatitis that is recurring.

Projekt OP VK (CZ.1.07/2.3.00/09.0193) "Endoskopie a miniinvazivní zákroky"

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AKTUÁLNÍ TÉMATA V GASTROENTEROLOGII U MALÝCH ZVÍŘAT

Tento sborník je vydán k semináři organizovaného FVL VFU Brno a LF MU v Brně v rámci realizace chirurgického projektu OP VK (CZ.1.07/2.3.00/09.0193) spolufinancovaného Evropským sociálním fondem a státním rozpočtem České republiky. Na tomto semináři budou presentovány aktuální témata v gastroenterologii malých zvířat celosvětově známým specialistou na gastroenterologii, hepatologii a pankreatologii u malých zvířat, kterým je profesor Michael D. Willard, DVM, MS, Dipl. ACVIM z Texas A&M University, USA.

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