VOMITING/REGURGITATION

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Vomiting occurs when a centrally-mediated reflex occurs which causes gastric and/or intestinal contents to be ejected from the alimentary tract.

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 the patient as it is to the client. Again, 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.

A less well appreciated problem is distinguishing expectoration from vomiting. Dogs that expectorate can gag so much that they eventually stimulate themselves to vomit. Therefore, a very careful history is necessary to determine if the spitting up only occurs after gaging and coughing.

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 ≤ 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.

ESOPHAGEAL FOREIGN BODIES

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.

CICATRIX

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.

IDIOPATHIC MEGAESOPHAGUS

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. The Bailey chair is a very useful device. You can find out more about it on the web (http://www.caninemegaesophagus.org/support.htm). 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

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, $\pm$ 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.

GI BLEEDING

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 inferior to proton pump inhibitors. Do not use them.

<u>Proton pump inhibitors</u> 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. <u>Major point to remember</u>: omeprazole and other proton-pump inhibitors seems to be most useful drugs for treating gastrinomas and gastroesophageal reflux.

<u>Misoprostol</u> (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.

LINEAR FOREIGN BODIES

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.