In general, most patients undergoing surgery of the distal stomach have vomiting as their most important clinical sign. The vomiting may be sporadic and infrequent or chronic and frequent. The latter can result in serious metabolic changes. Hypochloremia, hypokalemia, metabolic alkalosis, and dehydration often accompany the more serious episodes of vomiting. Prior to any surgical intervention, it may be necessary to pay close attention to correcting fluid and electrolyte imbalances. If the animal is under 6 months of age, albumin and glucose deficits must be addressed as well.

NaCl solution with KCl added (40 mEq/liter) will help reverse the metabolic alkalosis and hypokalemia/hypochloremia. If indicated, dextrose can be included in the saline solution.

One very important factor to consider in patients with gastric retention is the risk of aspiration pneumonia. Delayed gastric emptying is a known risk and to counter that, injectable metoclopramide (0.2-0.4 mg/kg) is given subcutaneously preferably 2-4 hours prior to surgery. An injectable antacid is also recommended and the author prefers famotodine (0.5-1.0 mg/kg) intravenously just prior to surgery. Lastly, an orogastric tube is inserted into the stomach immediately after endotracheal intubation. This will allow a thorough evacuation of stomach contents thereby decreasing the chance of “silent reflux”. It will have an additional benefit of decreasing peritoneal contamination should the stomach be entered surgically.

Gastric outflow obstruction can be partial or complete and can be congenital in origin or acquired. Acquired disorders include foreign bodies, granulomatous eosinophilic gastritis, acquired antral musosal hypertrophy, chronic ulcer disease, neoplasia, or infiltrative fungal disease such as Pithium infection. Postprandial vomiting with intervals of 30 minutes to 24 hours often accompanies an obstruction. If the obstruction is nearly complete, as seen in the benign inflammatory condition known as acquired antral mucosal hypertrophy, then bile in the emesis is rare.

Acquired antral mucosal hypertrophy

This disorder is a benign inflammatory condition resulting in pyloric stenosis in the adult dog. It has been reported in cats, but is rare. It is seen most commonly in toy breed dogs that often are temperamental. In the author’s experience, there appears to be a slight predilection in the Lhaso Apso and Shih Tzu breeds. Other toy breeds commonly affected include poodles, terriers and the Pekinese. Most dogs are middle aged or older although animals under 2 have been reported.

The history includes sporadic vomiting which usually (but not always) is related to the ingestion of food. Over time, (weeks, month, years), the vomiting becomes more frequent. Weight loss, anemia, weakness and dehydration are seen in some dogs. In the dogs that are vomiting frequently just prior to presentation, (2-6 times daily), severe electrolyte and acid-base imbalances may be present as mentioned earlier.

In most cases, the physical examination will reveal few abnormalities and laboratory data will be within normal limits. Sometimes, a palpably enlarged, fluid-filled stomach is noted. In addition, some abdominal discomfort may be demonstrated.

The diagnosis of mucosal hypertrophy is based on the signalment, history, and clinical signs, the most important being the vomiting of food with NO bile up to 24 hours after eating. Plain film radiography will sometimes demonstrate an enlarged stomach and a rounded fluid-filled pyloric region (Figure 1).

A positive-contrast gastrogram will reveal delayed gastric emptying (sometimes up to 6 hours); irregular mucosal surfaces within the pyloric antrum or canal, and, on occasion, a filling defect that mimics a parrot’s beak (“parrot beak” sign, figure 2).

Endoscopy is helpful in demonstrating hyperemia of the gastric mucosa in the pyloric region, polyoid lesions, and on occasion, the presence of an ulcer. Endoscopic biopsies often reveal an inflammatory component, usually a plasmacytic-lymphocytic infiltrate.

Medical management may be attempted in these dogs but it usually fails to control the signs of vomiting. Feeding a low-fat soft diet, small in volume, and multiple feedings will sometimes help. Metoclopramide often fails to resolve the problem and, in some instances, seems to make the vomiting worse.

Surgical treatment is recommended for satisfactory resolution of this disease. However, as mentioned before, fluid, electrolyte, and acid-base abnormalities should be corrected first and orogastric suction employed immediately following endotracheal intubation. In addition, preoperative use of an injectable prokinetic agent
(metoclopramide) and an injectable antacid (ranitidine, famotidine) is highly recommended to prevent excessive gastroesophageal reflux.

**Surgical procedures**

Pyloroplasty procedures are intended to increase the diameter of the pylorus. It was originally designed to relieve obstruction and symptoms caused by duodenal ulcer in man. Today, these techniques are used in veterinary medicine for treatment of mucosal hypertrophy of the pylorus, ulcer disease (rarely in vet med), and, in some instances, for palliation of outflow obstruction caused by neoplasia of the distal stomach.

**Heineke-miculicz pyloroplasty**

This pyloroplasty procedure at one time was the procedure of choice in animals. However, more recently, it has been supplanted by the Y-U pyloroplasty. To perform the H-M technique, a full-thickness longitudinal incision is made through the pyloric sphincter extending 1 cm above and below the pyloric ring (figure 3). The incision is closed in a single layer transversely starting in the middle and moving laterally. A full-thickness simple interrupted approximating suture pattern is preferred. A synthetic absorbable or a monofilament nonabsorbable suture (polypropylene), 000 or 0000 size, is recommended. A 2-layer inverting closure is contraindicated, as it tends to invaginate too much tissue into the pyloric lumen. This pyloroplasty also affords limited exposure of the stomach lumen and proximal duodenum.

**Y-U Antral Flap Pyloroplasty**

This procedure has proven to be a very effective procedure to correct outflow problems of the stomach experimentally and clinically. It involves the plastic surgery technique of converting a “Y”-shaped incision into a “U”-shaped closure (Figures 4A, B, C). Each limb of the “Y” should be 3-5 cm in length depending on the size of the animal. The base of the “Y” should always extend a small distance into the proximal duodenum.

The Y-U pyloroplasty gives excellent exposure of the distal stomach. It easily accommodates resection of hypertrophied mucosa (Figure 4B). Once the submucosal resection of tissue is completed, the “V”-shaped antral flap is trimmed to a “U” shape. This full-thickness piece of stomach and the tissue removed during the submucosal resection of diseased tissue should be submitted for histopathological evaluation.

Closure of the pyloroplasty begins by moving the “U”-shaped flap of the antrum distally and suturing the center of the base of the “U” to the apex of the duodenal side of the incision (Figure 4B). The greater and lesser curvature sides of the flap are subsequently sutured. A simple interrupted or continuous suture pattern using 000 or 0000 sized suture is suggested.

The base of the “Y” incision should extend slightly onto the stomach side of the pylorus. Each limb of the “Y” is approximately 3 cm in length. Once the lumen is exposed and the extent of the lesions indentified, parallel incisions through the submucosa/mucosa above and below the pylorus are made extending from one edge of the exposed bowel lumen to the other side (Figure 4B). Following the submucosal dissection and removal of a strip of tissue incorporating the mass(es), the edges of the defect (submucosa/mucosa layers) are apposed with 3/0 or 4/0 suture using a continuous pattern.

At times, there may be hypertrophy of the pyloric musculature as well as the mucosa. The treatment is the same when both forms of pyloric pathology are present.

During the immediate postoperative period, gastroparesis and emesis may be a problem. Injectable metoclopramide (0.2-0.4 mg/kg subcutaneously) may be helpful for 24-48 hours postoperatively. Fluid therapy is continued for one day postoperatively. Feeding begins after surgery with the emphasis on a low-fat diet of spaghetti and other forms of pasta, rice and macaroni. Feeding small amounts 4-6 times daily for the first 72 hours is recommended. If emesis continues beyond 48 hours, metoclopramide may need to be continued at home. Ideally, the metoclopramide should be given every 8 hours 30 minutes before each meal. An alternative to metoclopramide is cisapride (if available), which is given at a dose of 0.1-0.2 mg/kg every 8 hours, also given 30 minutes before a meal. The owner is also advised to decrease the size of each meal as overfeeding may be contributing to vomiting in the early postoperative period.

**Billroth I (gastroduodenostomy) and Billroth II (gastrojejunostomy) with or without a Roux-en-Y diversion**

These techniques are reconstruction techniques following resection of various amounts of gastric, pyloric and duodenal tissue. Billroth I is probably the most common form of distal gastric reconstruction used in veterinary medicine. Both Billroth I and II are
amenable to stapling techniques. A Roux-en-Y is only necessary if bile and pancreatic enzymes need to be diverted away from the gastrojejunostomy site.