# SUMMARY

<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestinal Anatomy and Wound Healing for Surgeons</td>
<td>p. 2</td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Physiology of Intestinal Obstruction and Patient Stabilization:</td>
<td>p. 7</td>
</tr>
<tr>
<td>Hydro-Electrolyte Derangements</td>
<td></td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Diagnostic and Therapeutic Considerations for Septic Peritonitis</td>
<td>p. 17</td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Enterotomy or Enterectomy? Viability Assessment</td>
<td>p. 22</td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Intestinal Suture: Materials, Technique and Patching Options</td>
<td>p. 30</td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Feeding Tubes for Enteral Nutrition</td>
<td>p. 34</td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Colectomy in the Dog and Cat: Technique and Indications</td>
<td>p. 39</td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
<tr>
<td>Shunts to and from the Intestine: Biliary Diversion, Colectomy and</td>
<td>p. 42</td>
</tr>
<tr>
<td>Uretero-Colonic Anastomosis</td>
<td></td>
</tr>
<tr>
<td>Ellison Gary W.</td>
<td></td>
</tr>
</tbody>
</table>
Wound dehiscence of an intestinal anastomosis often leads to generalized bacterial peritonitis and subsequent death. Therefore factors which negatively affect visceral healing are potentially of great clinical significance to the surgeon. Factors that cause intestinal anastomoses to leak include etiology of obstruction, failure to adequately identify ischemic tissue, improper suturing or stapling technique and factors that negatively effect wound healing such as sepsis, malnutrition and anti-neoplastic therapy.

Anatomy

The small intestine measures about 3.5 times the length of the body. The duodenum begins at the pylorus and runs caudally in a dorsolateral direction to the right of the midline as the descending duodenum. The mesoduodenum becomes shorter and at the level of the 5th lumbar vertebra the duodenum turns medially to the left of the midline in close proximity to the root of the mesentery. The ascending duodenum is closely adhered to the mesocolon by the duodenocolic fold. Immediately cranial and to the left of the mesenteric root the transition to jejunum begins. Unlike the relatively fixed duodenum with its short mesoduodenum and duodenocolic fold the jejunum and ileum are loosely coiled and freely moveable because they are suspended from a long mesentery. The jejunum comprises the bulk of the small intestine. A gross division between the jejunum and ileum is difficult to determine but the terminal contracted portion of the ileum is characterized by prominent antimesenteric vessels. The ileum is attached to the cecum by the ileocecal fold and lies principally to the right of the midline.

The duodenum is supplied by the cranial pancreaticoduodenal off the celiac artery and the caudal pancreaticoduodenal off the cranial mesenteric artery. The jejunum is supplied by 12-15 jejunal arteries which arise from the cranial mesenteric. The ileum is supplied by the ileocolic artery which arises from the cranial mesenteric both on its mesenteric and antimesenteric surface. The four tissue layers of the intestine include serosa, muscularis, submucosa and mucosa. The muscular coat consists of a relatively thick outer longitudinal layer and a thinner inner circular layer. At the junction of the small and large intestine this circular muscular layer is grossly thickened becoming the ileocolic sphincter. The submucosa contains the main vascular supply to the bowel wall known as the submucosal plexus. It is also rich in collagen and is the layer of greatest suture holding capacity.

Microanatomy of the Gut

The small intestine has rich arterial blood supply which originates with the cranial mesenteric artery and branches into many mesenteric arteries. These vessels branch prior to giving rise to multiple arcuate vessels which run within the fat of the mesenteric surface of the intestine. The arteries perforate the muscular wall of the gut and develop into a rich plexus within the submucosa. The submucosal vessels give off branches to the mucosa and then run centrifugally outward where they supply the inner circular and outer longitudinal muscular branches of the intestine as well as the relatively avascular serosa. The venous drainage
mirrors the arterial supply but the cranial mesenteric vein dumps into the portal vein so that absorbed nutrients from the gut can be metabolized by the liver.

**Normal Wound Healing of the Viscera**

The gastrointestinal tract follows the same basic healing curve as the skin but has accelerated healing properties. The lag or inflammatory phase of healing lasts three to four days. Immediately after wounding, contraction of blood vessels occurs, platelets aggregate, the coagulation mechanism is activated, and fibrin clots are deposited to control hemorrhage. The fibrin clot offers some minimal wound strength on the first postoperative day, but the main wound support during the lag phase of healing comes from the sutures. Enterocyte and regeneration begins almost immediately after wounding; however, the epithelium offers little biomechanical support. The lag phase is the most critical period during visceral wound healing, since most dehiscences take place within 72 to 96 hours after the wound has been created.

The proliferative or logarithmic phase of visceral wound healing lasts from day three or four through day 14. Rapid proliferation of fibroblasts occurs logarithmically during this period. The fibroblasts produce large amounts of immature collagen, resulting in rapid gains in wound strength. The proliferative phase of wound healing is a dynamic process in which collagen synthesis takes place in the presence of collagenolysis. In the stomach, small intestine, and urinary tract, collagenase activity at the wound edge is minimal and rapid gains in tensile and bursting strength occur. At the end of 14 days, gastric and small intestinal wound bursting strength is approximately 75 percent that of normal tissue. The urinary bladder heals even faster, regaining 100 percent of its normal tensile strength 14 to 21 days. Conversely, the colon heals much more slowly due to marked collagenase activity at the wound edge and regains only about 50% of its normal tensile strength 14 days after wounding. Factors such as traumatic suturing, fecal material, bacterial contamination, and infection all increase the amount of local collagenase produced at the wound edge.

The maturation or phase of wound healing is characterized by reorganization and cross-linking of collagen fibers. This phase extends from day 14 through day 180 in the gastrointestinal tract of the dog and from day 14 through day 70 in the dog bladder. As with skin wounds, the size and thickness of the scar decreases during this time without weakening the wound. The maturation phase is relatively unimportant clinically in visceral wound healing, since acceptable tensile and bursting strength have been established by the end of the proliferative phase of wound healing and leakage is virtually nonexistent at this point.

**Importance of Tissue Apposition**

Direct approximation of the wound edge allows for optimum rapid healing characterized by primary intestinal wound healing. With good apposition rapid mucosal re-epithelialization and early formation of young well-vascularized collagen between the submucosa, muscularis and serosa occurs. Other advantages of approximating patterns for intestinal anastomosis are: 1) lumen diameter is not compromised, 2) wound strength meets or exceeds evert ing or inverting wound strengths, and 3) adhesions are minimal. The crushing suture has been shown to cause more tissue ischemia directly at the suture line and its use is discouraged.

Mucosal eversion or tissue overlap retards healing and should be avoided. Delayed fibrin seal formation, delayed mucosal re-epithelialization, increased mucocele formation, prolonged inflammatory response, and marked adhesion formation all characterize everted
healing. Eversion may initially widen the lumen diameter, but the prolonged inflammatory response usually narrows the lumen sometimes resulting in stenosis. Everting anastomoses also have an increased tendency for leakage especially in the face of a septic abdomen and should never be used in the colon.

Inversion of the wound edge creates an internal cuff of tissue that reduces lumen diameter. Hemodynamic compromise of the inverted submucosa occurs resulting in mucosal edema and necrosis. After five days the internal cuff usually sloughs. Inverting anastomoses are characterized by a rapid serosa to serosa seal and minimal adhesion formation. Because of their safety against leakage, inverting patterns may be the preferred technique for the colon.

The GIA and TA auto staplers lay a double row of staples for security and when used in combination create a functional “end to end anastomosis”. The GIA portion of the anastomosis is inverted whereas the TA portion of the anastomosis is everted. Recent studies have shown that leakage rates are similar to hand sewn techniques but auto stapler usage significantly reduces surgical time.

A rapid alternative to sutured anastomosis is the use of an AutoSuture 35 skin stapler with stainless skin staples (United States Surgical Corp., Norwalk, CT). After triangulating the intestine with three stay sutures, the skin stapler is used to place staples every 2-3 mm around the perimeter of the wound. These closures are more rapidly done than hand sutured anastomosis and have similar bursting strengths but mucosal eversion may occur between staples.

A taper-cut, narrow-taper, or small reverse-cutting needle with 3-0 or 4-0 swaged-on suture material is suitable for most anastomoses. Braided, nonabsorbable materials such as silk or braided polyesters should be avoided. Chromic surgical gut rapidly loses tensile strength due to collagenase and phagocytosis at the wound edge and is not recommended. Synthetic, braided, absorbable suture materials such as polyglactin 910 (Vicryl, Ethicon, Inc., Somerville, NJ) are acceptable, but they have significant tissue drag. I prefer poliglecaprone 25 (Monocryl, Ethicon Inc., Somerville, NJ), glycomer 631 (Biosyn, United States Surgical Corp, Norwalk, CT), polydioxanone (PDS, Ethicon, Inc., Somerville, NJ), and polyglyconate (Maxon, United States Surgical Corp., Norwalk, CT), which are monofilament absorbable sutures with little tissue drag and have all been used successfully for intestinal anastomoses. Nonabsorbable monofilament sutures such as nylon (Ethicon, Ethicon, Inc., Somerville, NJ) or polypropylene (Prolene, Ethicon, Inc., Somerville, NJ) also are acceptable for simple interrupted anastomoses, but they should not be used for simple continuous anastomoses because they do not allow luminal distension. Newer versions of triclosan impregnated polygalactin 910 (Vicryl plus, Ethicon Inc., Somerville, NJ) and poliglecaprone 25 (Monocryl plus, Ethicon Inc., Somerville, NJ) are undergoing investigation in hopes that this bacteriostatic compound will reduce wound infection.

Other Factors Effecting Dehiscence

Healing of visceral wounds is negatively affected by a number of factors. *Chronic weight loss of 15 to 20 percent* due to cancer cachexia or other reasons has a negative effect on visceral wound healing. Correction of cachexia as well as early postoperative enteral feeding appears to increase collagen deposition and bursting wound strength. *Glucocorticoids* have a negative effect on wound healing when given in large doses prior to the third day after wounding. *NSAIDs* appear to affect the early inflammatory phase of
wound healing, but do not appear to interfere with the proliferative phase of wound healing or have a significant negative effect on visceral healing strength. Radiation therapy interferes with fibroblast mobilization, replication, and collagen synthesis as well as causing sclerosis of microvasculature, thereby reducing oxygenation at the wound site. Whenever possible, radiation therapy should be initiated after visceral wound healing is complete. The negative effects of cancer on wound healing appear to be secondary to nutritional deficiencies rather than direct tumor impairment on wound healing. Visceral wound healing may actually be mildly augmented owing to release growth factors by the neoplasm. Effects of chemotherapeutic agents on visceral wound healing are variable. Drugs such as vincristine, vinblastine and azathioprine seem to be safe when used in therapeutic doses. Drugs such as cyclophosphamide, methotrexate, 5-FU, and doxorubicin have been shown to delay wound healing in both experimental and clinical studies. Cisplatin appears to significantly impair intestinal wound healing in rats and should be used with caution after intestinal surgery.

Nutritional Depletion

Tissue trauma, sepsis, burns, and major surgery induce major metabolic changes in small animal patients. With each of these stresses the animal’s basic metabolic rate is accelerated and protein metabolism occurs, leading to a potential state of negative nitrogen balance. Protein-calorie malnutrition (PCM) occurs because of starvation, when a metabolic response to injury becomes prolonged, or with hypermetabolism secondary to sepsis. It takes only five to 10 days of anorexia to compromise the immune system and deplete the bodies muscular and hepatic glycogen stores. When PCM is present cell mediated immunity is impaired, there is a high risk of infection, anemia and hypoproteinemia and impaired wound healing.

Caloric and protein depletion in animals has been shown to inhibit visceral healing, but only after a loss of 15 to 20 percent of body weight. Decreases in wound breaking strength are directly proportional to the carcass weight loss. It is estimated that 75 percent of animals with elective surgical wounds attain functional wound union during the period of negative nitrogen balance; however, extended PCM from muscle, visceral, or plasma tissue losses increases the risk for visceral wound disruption. Impaired visceral wound healing is due to both a prolonged lag phase of healing and diminished capacity for fibroplasia within the logarithmic phase.

Effect of Early Postoperative Enteral Feeding on Visceral Healing

Malnutrition induces intestinal mucosal atrophy, reduced motility, increased incidence of ileus and the potential for bacterial translocation through the bowel wall, with resultant sepsis. Impaired wound healing due to nutritional causes may be ameliorated by feeding an enteral or parenteral diet that supplies energy needs in the form of fatty acids and sugars and provides essential amino acids. Feedings of high protein meals after injury can optimize conditions for normal visceral wound healing. Amino acids provided through enteral nutrition are utilized for the synthesis of structural proteins such as actin, myosin, collagen, and elastin.

Early if not immediate postoperative enteral feeding has been shown to have a positive influence on the healing rate of intestinal anastomosis in dogs. Bursting pressures and collagen levels of ileal and colorectal anastomosis were compared in Beagles fed elemental
diets versus those fed only electrolyte and water for four days. The dogs fed elemental diets had nearly twice the bursting strengths of the control group and nearly double the amount of both immature and mature collagen at the wound site. Total parenteral nutrition (TPN) does not appear to ameliorate the mucosal atrophy or increase collagen deposition as does enteral nutrition. In human studies, the incidence of septic complications was significantly lower in people fed between eight to 24 hours after surgery versus those maintained on TPN. Additionally early fed patients had a reduced incidence of postoperative ileus and reduced hospital stay.

References

Intestinal Obstruction

By definition, intestinal obstruction implies the failure of ingesta or intestinal secretions to move in a normal aboral direction. Obstructions are typically classified by their duration, their severity and their location. Partial or incomplete obstruction is incomplete occlusion of the bowel lumen that allows limited passage of fluid or gas. Complete obstruction is total occlusion of the intestinal lumen, with failure of gas or fluid to pass the point of obstruction. Blockage in the duodenum or upper jejunum constitutes a high intestinal obstruction; blockage in the midjejunal area constitutes a midintestinal obstruction and blockage in the distal jejunum, ileum, or ileocecal junction constitutes a low intestinal obstruction.

In terms of pathophysiologic changes, obstructions are usually best described as either simple mechanical or strangulating. With simple mechanical obstruction there is partial or complete obstruction of the bowel lumen, but the blood supply to the intestinal wall is usually not impaired. Conversely, with strangulation obstruction, the circulation to the involved segment of intestine is impaired, and usually complete obstruction is present.

Etiology of Simple Mechanical Obstruction

The causes of mechanical obstruction can be subdivided into three general categories: intraluminal mechanical obstruction, intramural mechanical obstruction, and extramural mechanical obstruction.

Intraluminal mechanical obstruction is the most common type in small animals. The oropharyngeal opening is larger than any other orifice in the alimentary tract and foreign bodies such as bones, balls, or corncobs can traverse the esophagus and stomach and become lodged in the smaller-diameter intestine. Large intraluminal foreign bodies often cause signs consistent with complete luminal obstruction, although slow aboral passage of the foreign body may occur. Polypoid intestinal masses or linear foreign bodies such as string may cause partial or incomplete luminal obstruction. In cats, benign adenomatous polyps of the upper duodenum can cause intermittent hematotemesis. The trailing end of linear foreign bodies often becomes anchored over the base of the tongue or in the pyloric antrum. Normal intestinal peristalsis moves the foreign body distally, but since it is fixed proximally the bowel plicates itself along the length of the foreign body.

Intramural mechanical obstruction is most commonly caused by intestinal wall neoplasia or fungal granulomas. Intestinal neoplasms such as adenocarcinoma, leiomyoma, leiomyosarcoma, fibrosarcoma, and lymphosarcoma commonly invade the muscular layer of the intestinal wall. These tumors not only compromise lumen diameter, but they also reduce the pliability of the intestinal wall at that point, reducing its distensibility and likening the occurrence of intussusception. In the Southeastern United States intestinal granulomas
caused by the algae Pythium species is a common cause of intestinal obstruction. This obligate organism creates mural thickening and fibrosis that interferes with normal intestinal absorption also prevents normal intestinal distention. Both intestinal neoplasms and fungal granulomas tend to cause incomplete mechanical obstruction. Onset of clinical signs is often delayed or insidious.4

Extraluminal small intestinal obstruction due to adhesions is a potential sequela of elective abdominal surgery. Because of this, there is increasing emphasis on minimal invasive laparoscopy techniques in human and veterinary medicine. Most experimental and clinical studies in people found a reduction of adhesions with laparoscopy versus laparotomy.5 Although adhesions do occur in the dog and cat abdomen following laparotomy, functional obstruction is less common. In fact, studies of intestinal transit times after planned enteroloplication techniques for intussusception in dogs have shown that there is no delay in actual transit time when planned adhesions are created.5 Clinical signs relating to extraluminal obstruction in small animals are more often a result of compression due to pancreatic abscess or neoplasia or to translocation of the bowel through rents in the mesentery or through hernias in the diaphragm, umbilicus, or inguinal or femoral triangle region. These latter translocations usually lead to strangulation obstruction.

Pathophysiology of Mechanical Obstruction

Accumulation of Gas and Fluid

Complete intraluminal mechanical obstruction results in distention of the bowel proximal (oral) to the obstruction, owing to accumulation of gas and fluid. The gas accumulating proximal (oral) to the obstruction consists of swallowed air (72%) and gas formed in the body (28%).1 Of the gas formed in the body it is estimated that approximately 70% is gas that diffuses from the blood into the bowel lumen, and a smaller percentage (30%) results from intramural decomposition of food material by bacteria. The gas in the distended bowel is composed principally of nitrogen (70%), oxygen (10% to 12%) and hydrogen (1% to 3%) which mimic those percentages seen in atmospheric air. Additionally small amounts of carbon dioxide (6% to 9%) can be formed from neutralization of bicarbonate in the bowel lumen. Organic gases such as methane (1%) or hydrogen sulfide (1% to 10%) when present are the result of low-level bacterial fermentation.1

Fluid accumulation is due not only to retention of ingested fluids but also to the significant production of secretions in the upper gastrointestinal tract. It is estimated that a 40 kg dog actually produces in excess of 2100 ml of secretions per day. Most of these secretions are reabsorbed in the lower jejunum and ileum; only an estimated four percent to 10 percent of the water volume reaches the colon.7 Water transport in the gut is normally passively regulated, principally by hydrostatic pressure gradients that are created mainly by solute transfer. The intracellular solute pathway that allows passive diffusion between the pores and tight junctions of epithelial cells is controlled by electrochemical, osmotic, and hydrostatic pressure gradients.

During mechanical obstruction the absorption of water from the gut lumen is reduced by several mechanisms. Transport of solutes through the epithelial cells which normally occurs by active transport via sodium ion membrane pumps or brush border membrane carriers is impaired. Intraluminal osmolality is usually increased and additive factors such as lymphatic and venous congestion also reduce the absorption of solutes.8 In addition, intestinal
mucosal secretion is increased due to the cyclic AMP mechanism. Factors believed to contribute to increased secretion and decreased absorption include increased concentration of intraluminal bacterial enterotoxins, increased levels of bile and fatty acids, or products of tissue ischemia. The distended bowel may lose its ability to absorb fluids within 24 hours after onset of obstruction.

Normal intraluminal pressure in the canine small bowel is 2 to 4 mm Hg. It is estimated that normal peristalsis may produce pressures in the range of 15 to 25 mm Hg. Three days after creation of a total obstruction, intraluminal pressure in the small bowel of dogs can be as high as 44 mm Hg. During vomiting it can rise as high as 95 mm Hg. Rapid lymphatic and capillary stasis occurs when intraluminal pressure reaches 30 mm Hg, and total occlusion of venous drainage occurs at 50 mm Hg. Since the arterial supply is not affected capillary congestion can occur at the microcirculatory level. The increased hydrostatic pressure at the capillary bed level produces a net shift of fluid into the interstitium, resulting in bowel wall edema. Eventually, fluid can shift not only from the bowel wall into the lumen but also through the serosal surface into the peritoneal cavity.

Pressure increases also cause circulatory impairment of the submucosa and muscular layers of the bowel wall. Early impairment of the villous circulation within the mucosa is seen when the pressure reaches 20 mm Hg. Reductions in mesenteric and submucosal blood flow occur when the intraluminal pressure reach 30 mm Hg. Oxygenation of the intestinal mucosa decreases significantly when the intraluminal pressure exceeds 40 mm Hg. At 44 mm Hg arteriovenous shunting occurs at the mucosal villous base. Therefore, selective mucosal ischemia can follow simple mechanical obstruction if intraluminal pressure rises above 40 mm Hg. Since in naturally occurring mechanical obstruction physiologic pressure probably does not exceed 50 mm Hg, full-thickness devitalization of the wall usually does not occur in the dilated proximal segment.

Reduced Motility and Bacterial Overgrowth

The bowel responds to gaseous and fluid distention with periodic bursts of neuromuscular activity, resulting in peristaltic rushes. These wave-like movements begin in the proximal bowel and traverse the entire length of intestine above the point of obstruction. Periods of hyperactivity are then followed by quiescent periods of varying duration. Experimental studies in dogs have shown increased myoelectric activity above the point of obstruction. The intestine distal to the obstruction simultaneously exhibits reduced peristaltic activity. With increased distention from prolonged obstruction there are clusters of intense myoelectric activity that are felt by the patient as intermittent cramps (colic). It is believed that this phenomenon is largely due to stimulation of the proximal bowel through cholinergic pathways and inhibition of the bowel distal to the obstruction through noncholinergic nonadrenergic pathways.

Small intestinal stasis may lead to luminal bacterial overgrowth. In normal intestinal mucosa, bacteria and their enterotoxins are not able to cross the mucosal barrier. In the impaired mucosal barrier there is a potential for increased permeability and migration of bacteria and their toxic products into the systemic circulation or the peritoneal cavity. Decompression of the distention usually allows reversal of the circulatory changes and provides for rapid mucosal regeneration. However, if necrosis of the bowel wall occurs due to prolonged severe distention or direct pressure from the obstructing object the
mucosal barrier may break down, and transmural migration of bacteria and endotoxins beneath the obstructing foreign body may occur.

**Level of Obstruction and Electrolyte Loss**

The classic clinical signs associated with high (duodenal and proximal jejunal) obstruction are described as frequent vomiting that begins soon after the onset of obstruction. Yet experimental data support the observation that dogs and cats with high intestinal obstruction may not begin vomiting for 24 to 72 hours. Electrolyte loss is closely associated with the level of obstruction. With obstructions at the pylorus, gastric fluids that are rich in potassium, sodium, hydrogen, and chloride ions are vomited. Hypochloremic, hypokalemic, moderately hyponatremic metabolic alkalosis with dehydration may result in early stages. Animals that vomit profusely do not survive as long as those that do not vomit at all.

Severe vomiting associated with duodenal and proximal jejunal obstruction, causes loss of gastric hydrochloric acid and bicarbonate-rich alkaline pancreatic secretions. Dehydration with mild metabolic acidosis usually results. With continued fluid depletion progressive hypovolemic shock occurs. The major cause of mortality from upper small intestinal obstruction is associated with this severe and rapid hypovolemia. Dogs with complete upper intestinal occlusion usually die within three or four days or it may be greatly reduced with parenteral infusion of physiologic saline or lactated Ringer's solution. Experimental reinfusion of vomitus into the dog’s bowel below the obstruction was also life saving.

With low small intestinal obstruction the onset of vomiting may not occur until 2 or 3 days after onset of obstruction, and it is often intermittent. The distention usually is gaseous during the initial 24 hours but thereafter is accompanied by loss of varying quantities of fluid into the bowel lumen. Fluid sequestered in low intestinal obstructions is usually mildly hyperosmotic and similar in composition to plasma. Analysis of intraluminal fluid after experimental low obstruction in dogs reveals a mean sodium level of 140 mEq/L; of potassium, 16.8 mEq/L, and of albumin, 3.6 mg%. The sequestration of these fluids from the upper gastrointestinal tract and increase in secretion of new fluid electrolytes and protein cause a net loss of these compounds. The intraluminal fluid volume increases as the obstruction persists, though some sequestered fluid may move orally and eventually reach a nondistended loop of bowel, where normal reabsorption occurs. Lethargy and anorexia are often apparent in dogs with low intestinal obstruction. These animals exhibit steady weight loss and drink but do not eat. Dogs with experimentally created complete low intestinal obstructions may survive three weeks or longer if adequate water is provided.

**Strangulation Obstruction**

**Causes and Classification**

By definition, strangulation obstruction implies an obstructive process with loss of vascular integrity to the bowel wall. Common causes include intussusception, traumatic avulsion of the mesentery, mesenteric arteriothrombosis, mesenteric (intestinal) volvulus, and strangulated diaphragmatic, inguinal, or abdominal hernia. Foreign bodies can also create local small discrete areas of "pressure" strangulation necrosis. Strangulation obstruction may occur secondary to venous obstruction or thrombosis, arterial obstruction
or thrombosis, or a combination of both. If the strangulating process incorporates the mesenteric vessels, devitalization of large segments of the intestinal tract may occur. Strangulation obstruction should be considered a medical and surgical emergency. Death is often rapid, the result of hypovolemia and septic shock secondary to devitalization of the intestinal wall.

**Pathophysiology**

Pathophysiologic changes as described under simple mechanical obstruction occur proximal to the strangulation obstruction in addition to the direct changes attributable to the strangulated bowel segment. Partial venous occlusion, such as that caused by partially strangulated hernia or intussusception, is a common type of intestinal strangulation in small animals. With venous occlusion alone the arterial supply remains intact, allowing bowel wall edema and sequestration of blood in the intestinal wall. Motility changes in the bowel wall are proportional to the duration of venous obstruction. Initially there may be increased spike activity and increased motility of the affected bowel segment. As tissue hypoxia progresses cyanosis becomes evident and motility gradually decreases until it ceases completely.

With complete venous occlusion or thrombosis, wall edema, hemorrhage, and mucosal epithelial sloughing can occur as early as one to three hours after the insult. The strangulated loop then gradually becomes more turgid, and whole blood begins accumulating in the bowel lumen and extravasating into the peritoneal cavity. The bowel wall becomes visibly thickened and dark red to blue. Eight to twelve hours after complete venous occlusion the bowel segment turns black and distends maximally. When complete arterial occlusion occurs, as with mesenteric volvulus, full-thickness ischemia of the bowel wall occurs and bacteria and red cells invade all layers of the wall within 20 hours of strangulation.

**Translocation of Bacteria**

The flora in the proximal small bowel consists primarily of gram-positive, facultative bacteria, whereas the distal small intestine contains primarily aerobic coliforms and anaerobic species. There is movement of organisms that are normally found in the terminal intestine to upper levels of the small intestine. Marked increases in aerobic coliform bacteria, and Streptococcus species occur, in addition to large increases in anaerobic Clostridia and Bacillus species. Small intestinal bacterial concentrations that normally range from $10^2$ to $10^3$ per milliliter liquid secretion may increase to $10^8$ to $10^{11}$ per milliliter within just six hours after the onset of strangulation. A massive proliferation of resident bacteria also occurs within the strangulated section of bowel. Bacteria particularly *Clostridium perfringens* play a key role in the mortality of strangulation obstruction since germ free animals live significantly longer than those with normal intestinal flora in experimental models.

Loss of mucosal function leads to passage of viable bacteria or endotoxins through the epithelial mucosa into the lamina propria and then to the intra-abdominal cavity and systemic circulation. Bacterial translocation does occur in simple mechanical obstruction of the small bowel or colon. In strangulation obstruction, the loss of gut barrier function occurs more severely, compared with simple obstruction because the ischemia promotes the rapid destruction of the intestinal epithelium. Three mechanisms that promote bacterial
translocation have been identified: 1) intestinal bacterial overgrowth; 2) increased permeability of the intestinal mucosal barrier; and 3) deficiencies in host immune defenses.\textsuperscript{14}

**Clinical Presentation**

Clinically free peritoneal fluid begins to accumulate shortly after the development of strangulation obstruction. Initially the fluid is a transudate resulting from effusion from serosal vessels secondary to venous congestion or obstruction. The initial fluid is pink, clear, and odorless, and relatively low in protein. As the length of time of strangulation obstruction increases there is a change in the appearance of the fluid: it becomes black and has a foul odor. This is thought to be due to filtration of lumen contents through the devitalized bowel wall.\textsuperscript{14} After prolonged strangulation obstruction, hypoxia in the intestinal wall results in complete breakdown of the mucosal barrier. With arterial thrombosis full thickness necrosis occurs and perforation and septic peritonitis with resultant inflammatory cells, ingesta and bacteria.

Many experimental and clinical studies have independently proposed the hypothesis that the loss of gut barrier function and the consequent bacterial translocation, and their products may play an important role in the development of multiple organ failure (MOF) in strangulation obstruction. There is growing evidence that loss of gut barrier function to bacteria and or "endotoxins" might induce a local intestinal inflammatory response and lead to the subsequent release of cytokines (TNF, IL-1, 1L-6, IL-8 etc.).\textsuperscript{15} Strangulation obstruction has been shown to cause increased release of IL-6 to intestinal venous blood in pigs.\textsuperscript{16}

Plasma lactate has recently been shown to be of value in the diagnosis of gastrointestinal ischemia. In dogs with GDV, serum lactate values of > 6 mm/l have been shown to be a positive predictor of gastric necrosis.\textsuperscript{17} Likewise peritoneal lactate levels in dogs with experimentally induced ischemia of the small intestine were found to be highly predictive for intestinal ischemia in these animals.\textsuperscript{18}

**Treatment**

Treatment for strangulated intestinal obstruction involves not only fluid and electrolyte support but also aggressive antibiotic therapy, and possibly nonsteroidal anti-inflammatory drugs. If massive blood loss has taken place, blood transfusions may also be warranted. Early surgical removal of the devitalized section of bowel wall is essential. In experimental models of strangulated obstruction in dogs, death is delayed by the administration of broad-spectrum systemic antibiotics, particularly the aminoglycoside derivatives in combination with penicillins and metronidazole or third generation cephalosporins.

**Intestinal Pseudo-obstruction**

Chronic intestinal pseudo-obstruction is a syndrome, originally described in humans, characterized by chronic or recurrent symptoms of intestinal obstruction. The obstruction occurs in the absence of both organic luminal obstruction and a recognized underlying disease. Although previously limited to the human literature, the disease syndrome has recently been described in two dogs and a cat.\textsuperscript{19-21} Clinical manifestations result from delayed intestinal transit caused by disordered motility. Although all portions of the alimentary tract can be affected, the small bowel is most often involved. In some cases of pseudo-obstruction no pathologic abnormality can be found. In other cases histologic examination has shown two distinct pathologic abnormalities. In humans, one pathologic subtype includes direct
degeneration of intra-mural neurons, which are specifically associated with arrangement of the myenteric plexus, the celiac ganglion, the spinal cord, and even the brain. This type of chronic pseudo-obstruction has been labeled "visceral neuropathy." The second type of idiopathic intestinal pseudo-obstruction is caused by degeneration of intestinal smooth muscle cells-so-called visceral myopathy. Histologic sections of this type have shown vacuolization of the smooth muscle and atrophy of muscle fibers in both the longitudinal and circular muscle layers of the bowel wall. All effected animals had chronic vomiting and weight loss with concurrent dilatation of the small intestine. In a mixed breed dog, a atrophy of cecal smooth muscle fibers as well as infiltration of plasma cell lymphocytes and macrophages and mild lymphoplasmacytic enteritis were present. In an English bulldog both atrophy and fibrosis with monolocellular infiltrate was noted from the duodenum to the colon. In the cat, a regional jejunal area also displayed marked atrophy of the outer longitudinal muscular layer along with fibroplasia and vacuolar degeneration consistent with visceral myopathy in humans. Clinical signs of idiopathic intestinal pseudo-obstruction in man include abdominal pain, constipation, diarrhea, and vomiting. In the acute stages abdominal distention may occur. Surgery is usually avoided if possible; it is rarely beneficial. Stimulation of the gastrointestinal smooth muscle by prokinetic agents such as metoclopramide or cisapride might be helpful, as might nutritional support, in the form of enteral hyperalimentation. Results of surgery in animals have been similarly unsuccessful.

Intestinal Volvulus

Incidence/Clinical Signs

Intestinal volvulus is defined as to the rotation of the bowel around its mesenteric axis. Because of its freely movable unattached mesentery the jejunum or proximal ileum are the most likely locations for occurrence. (Intestinal torsion or rotation of the bowel along its longitudinal axis is uncommonly seen in small animals.) Intestinal volvulus is seen primarily in large dogs greater than 20 kg in weight. There is an approximate 4-1 male to female ratio and the German shepherd breed is predisposed.

The cause of canine small intestinal volvulus is unknown. Most dogs present without prior history of illness. Some patients have diarrhea prior to the onset of signs and in at least one case, the volvulus was associated with an intestinal tumor. Also some cases have undergone either elective or exploratory laparotomy shortly prior to the episode suggesting that translocation of the viscera may enter into the etiology of the disease. As with gastric volvulus, intestinal volvulus patients undergo a peracute onset of signs and may progress from being normal to near death in less than 6 hours. The most common clinical sign is hematochezia and surprisingly vomiting is uncommon. Abdominal distention is present in some but not all cases. Intestinal obstruction occurs because of kinking of the bowel at each end of the twisted loop. Bowel distension occurs due to entrapped ingesta and buildup of fluid and/or gas occurs. The combination of obstruction and bowel ischemia results in rapid production of toxins. Once full thickness necrosis occurs and significant distention has taken place, the bowel may rupture. If the patient has survived to this point, bacterial peritonitis, endotoxic shock and death will rapidly ensue.
Diagnosis

Successful formulation of a diagnosis and early surgical management of intestinal volvulus is essential for patient survival. Mortality is high with 6 of 6 dogs dying in one study. Unfortunately, volvulus of the intestine does not reproduce consistent signs. Acute onset of bloody stools, weakness and collapse may occur within a period of 6 hours. Palpation of multiple turgid dilated loops of bowel in the midabdominal region may be suggestive that a volvulus is present. Due to the acute onset, PCV, WBC counts and electrolyte values are often near normal. Radiographic diagnosis is difficult because of the many radiographic patterns seen with the disease. Radiographic signs may include 1) gaseous distention of multiple loops of bowel, 2) gross distention of the small intestine with ingesta or fecal material, and/or 3) loss of abdominal detail or ground glass pattern indicative of peritoneal fluid. Definitive diagnosis of intestinal volvulus is made during exploratory laparotomy or necropsy. The decision to operate is based on acute onset of weakness, hematochezia, and cardiovascular collapse in conjunction with abdominal radiographs suggestive of a complete obstruction.

Treatment

Rapid surgical intervention is necessary for cure of intestinal volvulus. It is rare to find viable intestine within the volvulus. Intestinal viability cannot be fully evaluated unless the volvulus is reduced (untwisted) and the venous lymphatic compression relieved, but inherent dangers of releasing sequestered endotoxins are present when the volvulus is reduced. If tissue necrosis is already present, the involved segment of bowel should be resected without reduction being performed. Crushing clamps are placed at the corners of the volvulus and the mesenteric vasculature is identified, ligated and divided before removing the twisted volvulus in toto. End to end anastomosis is then completed as previously described. Euthanasia may be a consideration if the length of the intestine resected exceeds that sufficient to support life. Postoperative care is similar to that described under foreign bodies and intussusceptions.

Managing Animals with Massive Resection

The propensity for short-bowel syndrome after massive intestinal resection depends on the amount of tissue excised, the location of the resection, and the time allowed for adaptation. Resection of up to 80% of the small intestine in puppies may allow for normal weight gain, whereas resection of 90% produces morbidity and mortality. After resection of large portions of small intestine, malabsorption, diarrhea induced by fatty acids or bile salts, bacterial overgrowth, and gastric hypersecretion may occur. Location of the resection is important. High resection of the duodenum and upper jejunum may decrease pancreatic enzyme secretion because pancreatic-stimulating hormones such as secretin and cholecystokinin are produced in the mucosa of these sections. These reductions in release of pancreatic enzymes contribute to malabsorption. Malabsorption of protein, carbohydrate, and fat leads to catabolism, negative nitrogen balance, and steatorrhea. Unabsorbed sugars also may cause osmotic diarrhea. If the ileocecal valve is resected, bacteria may ascend, overgrow in the small bowel, and contribute to diarrhea.

After massive resection, the remaining small intestine adapts by increasing lumen diameter, enlarging microvilli size, and increasing mucosal cell number. These compensatory changes may take several weeks; during this period, parenteral fluids, electrolytes, and hyperalimentation may be necessary for the survival of the animal. During this time, the
animal ideally will be able to maintain weight even with diarrhea. Medical treatments for unresponsive diarrhea after massive resection include frequent small meals, low-fat diets such as intestinal diet (I/D Hills, Topeka, KS) elemental diet supplements, medium-chain triglyceride oils, pancreatic enzyme supplements, B vitamins, kaolin antidiarrheals, and poorly absorbed oral antibiotics such as neomycin.

References


DIAGNOSTIC AND THERAPEUTIC CONSIDERATIONS FOR SEPTIC PERITONITIS

Gary W. Ellison, DVM, MS, Diplomate ACVS
University of Florida, College of Veterinary Medicine, Gainesville, FL

Etiology

Bacterial or septic peritonitis is usually the result of bowel perforation distal to the duodenum, surgical contamination, or intraperitoneal extension of urinary or reproductive tract infection. A combined form of peritonitis is commonly seen when simultaneous chemical and septic processes are going on. Gastrointestinal perforations can be due to any type of sharp abdominal trauma. Nontraumatic gastrointestinal perforations may result from gastrointestinal ulcers, sharp penetrating foreign bodies, string foreign bodies, or secondary to necrosis from a mechanical obstruction or incarceration.

The principal sites of peptic ulceration in the dog are the nonacid-producing sites that include the proximal duodenum, pyloric antrum, and gastric fundus. Perforating gastric ulceration has been reported but is rare in the cat. Ulcers in the dog are almost always associated with an underlying systemic disease process or are drug induced. Bile duct obstruction, mast cell tumors, liver disease, or gastrin-like secreting tumors of the pancreas (Zollinger-Ellison syndrome) have been incriminated. Ulcers have also been associated with oral nonsteroidal anti-inflammatory drugs such as aspirin, phenylbutazone, Carprofen, and Deracoxib. Gastric and colonic ulcers are reported with greater frequency in dogs with intervertebral disc herniation that have been treated with injectable dexamethasone. Identification of the etiology in ulcer formation is paramount to successful management of the disease.

Pathophysiology

With localized peritonitis, the peritoneum initiates an inflammatory response which causes local vasodilation, afferent sensory stimulation, extravasation of blood and plasma, neutrophil migration and platelet aggregation. Fibrin clots cause adhesions between adjacent peritoneal surfaces and localize the irritant. If neutrophils and macrophages do not phagocytize the irritants completely, fibroplasia may occur causing the agent to be walled off within heavy fibrous adhesions.

With diffuse or generalized peritonitis, massive fluid and protein movement to the peritoneal cavity result in a shift of fluid away from the intravascular space, causing hemoconcentration and eventual hypovolemic shock. The presence of large numbers of free bacteria or endotoxins cause massive shifts of neutrophils to the abdomen, massive vasodilation of the visceral vasculature, high hepatic energy demand (hypoglycemia), metabolic acidosis and often fatal septic shock.

Common clinical signs include inappetence, intermittent vomiting, melena, regenerative anemia, and abdominal pain. With ulcer perforation, acute abdominal pain, pneumoperitoneum, sudden collapse, and death are common sequelae. A perforation from an ingested foreign body can create signs that range from a localized peritonitis to those associated with generalized peritonitis. Fever, a painful and distended abdomen, and progressive onset of endotoxic shock may be seen. Occasionally, omentum or adjacent mesentery will seal the perforation, and clinical signs will resolve spontaneously.
Diagnosis

The diagnosis of gastrointestinal perforation is usually made radiographically by the presence of free peritoneal air. A fine, radiolucent line may be seen separating the tissue densities of the liver from the diaphragm. If the diagnosis is in question, ultrasound guided peritoneal centesis may show free bacteria, degenerated WBCs or fecal material. Barium sulfate should be avoided for fear of causing barium-induced peritonitis. With gastric or proximal duodenal ulcers, the ulceration can often be delineated with a fiberoptic endoscope.

Diagnostic Paracentesis and Peritoneal Lavage

Diagnostic peritoneal lavage is a clinically reliable technique that has been of great benefit in the diagnosis of intra-abdominal emergencies in man and has been established as an invaluable technique in veterinary institutions. It is a technique that can be easily done in veterinary practice with the same beneficial results. Diagnostic lavage has been shown to be 95 percent accurate in picking up intra-abdominal injury and disease in 129 dogs and cats, whereas needle paracentesis alone was only 47 percent accurate.

A local lidocaine block is performed 1-2 cm caudal to the umbilicus after a 10 cm square clip and prep. A commercial peritoneal dialysis catheter (Trocaht, McCaw Laboratories, Div. of American Hospital Corp., Glendale, CA) or a 14 gauge indwelling intravenous catheter (Sovereign Indwelling Catheter, Monoject, Div. of Sherwood Medical, Burbswick Company, St. Louis, MO) is directed through the body wall and aspirated for fluid. If no fluid is aspirated, 20 ml/kg of warmed lactated Ringer's or 0.9 percent NaCl solution is infused via gravity flow through a sterile IV infusion set. The fluid is mixed with abdominal contents by gentle abdominal massage and then collected by gravity flow into a sterile receptacle. Ten ml of fluid is sufficient for most diagnostic tests; however, as much of the fluid should be removed as possible to minimize discomfort to the patient.

<table>
<thead>
<tr>
<th>Test</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC Count</td>
<td>1,000/mm³ is indicative of peritonitis</td>
</tr>
<tr>
<td></td>
<td>2,000/mm³ is indicative of severe peritonitis</td>
</tr>
<tr>
<td>PCV</td>
<td>1% PCV elevation is seen for every 10-20 ml of free blood in abdomen per 500 ml of lavage fluid</td>
</tr>
<tr>
<td>Cytology</td>
<td>Many toxic neutrophils indicate supportive peritonitis. Careful examination of neutrophils for intracellular bacteria should be made. The presence of vegetative fibers with free bacterial indicates hollow viscous leakage or disruption.</td>
</tr>
<tr>
<td>Amylase</td>
<td>&gt; 1,000 units indicates intestinal ischemia or pancreatic inflammation</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>&gt; serum concentration indicates intestinal ischemia or strangu-</td>
</tr>
<tr>
<td>Creatinine</td>
<td>lation</td>
</tr>
<tr>
<td>Ictotest</td>
<td>&gt; serum creatinine levels indicates the presence of uroperitone-</td>
</tr>
<tr>
<td></td>
<td>neum</td>
</tr>
<tr>
<td></td>
<td>Ictotest positive - leakage form the biliary tree</td>
</tr>
</tbody>
</table>

*a*Urine bilirubin test tablets, Ames Division of Miles Laboratories, Inc, Elkhart, IN.
Other Laboratory Tests

A markedly elevated white count due to neutrophilia and a left shift may be caused by a localized peritonitis, inflammation or bowel wall necrosis. The markedly painful animal may also demonstrate a mature neutrophilia and lymphopenia due to a stress leukogram. Leukopenia with degenerated or toxic neutrophils is often seen with acute septic peritonitis or bowel incarceration along with an elevated PCV and reduced serum glucose. Plasma lactate has recently been shown to be of value in the diagnosis of gastrointestinal ischemia. In dogs with GDV, serum lactate values of > 6 mm/l have been shown to be a positive predictor of gastric necrosis. Likewise peritoneal lactate levels in dogs with experimentally induced ischemia of the small intestine were found to be highly predictive for intestinal ischemia in these animals. Recently the value of peritoneal glucose values have been shown to be highly sensitive indicators of abdominal sepsis. With septic peritonitis the glucose of abdominal fluid typically runs 20 mg% less than the serum glucose (BFG difference) and is 100% sensitive and specific for the presence of sepsis whereas with lactate the abdominal lactate level typically runs 2 mmol/Lg higher than what the blood lactate runs.

Treatment of Peritonitis

Appropriate treatment of peritonitis is based on: 1) medical management of endotoxic shock and treatment of sepsis 2) stopping the source of contamination and 3) providing peritoneal drainage when necessary.

Medical Management

Patients in severe shock should be given a balanced electrolyte solution at a rate of 90 ml/kg over the first hour. Metabolic acidosis is corrected with Na bicarbonate calculated at a dose of: mEq = .3 x kg x Base Excess. Aerobic bacteria commonly associated with bacterial peritonitis include E. coli, Enterococcus, Proteus and coagulase positive Staphylococcus aureus. Definitive antibiotic therapy should be based on bacterial culture and sensitivity but commonly used regimens include beta-lactam derivatives in combination with aminoglycosides. The penicillins and cephalosporins also have good antibacterial activity against anaerobes. Metronidazole is also effective for use in this regard. Recently the quinoline (Baytril) derivatives have gained widespread use in veterinary medicine. These drugs have broad spectrum activity and specificity toward resistant gram negative organisms especially persistent pseudomonas organisms. However, they have poor activity against anaerobes.

Intravenous flunixin meglumine has been shown to decrease morbidity and mortality associated with septic peritonitis in dogs. As a potent NSAID flunixin helps block prostaglandin induced inflammation in the peritoneal cavity. Flunixin is contraindicated when GI perforation or bleeding is evident. The use of corticosteroids for septic shock is controversial however since they must be given early in the course of treatment to be effective.

Stop the Source of Contamination

Therapy of peritonitis is fruitless if the etiology, ie., intestinal perforation, leaking anastomosis or ruptured bile duct is not repaired. For this reason successful management of diffuse peritonitis usually requires an exploratory laparotomy. When performing an exploratory celiotomy, complete systematic evaluation of the entire intestinal tract is performed to locate
any and all sites of perforation. Small perforations less than 0.5 mm are debrided back to healthy bleeding tissue and closed with a simple interrupted appositional Gambee or inverting Lembert suture pattern. The surgical site is covered with an omental wrap to act as a seal. If the defect is greater than 5 mm, resection may be necessary or a serosal patch procedure can be performed. Serosal patching utilizes the antimesenteric surface of the small bowel to cover or buttress an adjacent area of questionable tissue viability or an area that cannot be reliably sutured. Jejunum is commonly used because its freely movable mesentery allows it to be mobile. The serosal patch provides mechanical stability and will help to induce and localize a fibrin seal over the questionable area.

Providing Peritoneal Drainage

Thorough peritoneal lavage of the abdominal cavity is performed with body temperature 0.9% NaCl or lactated Ringers solution. Lavaging a cat with 500-750 ml and a large dog with 3-5 liters of fluid will help remove the bacteria and foreign debris which are the initiators of the peritoneal inflammation. Although lavage of the peritoneum may spread bacterial contamination its value in reducing morbidity and mortality from peritonitis is established without question. All lavage fluid should be aspirated because when bacteria are suspended in residual lavage fluid phagocytosis is impaired.

The addition of povidone iodine solution to lavage fluid is not efficacious and may in fact increase the mortality associated with peritonitis. The povidone portion of the compound causes renal hypotension and adversely effect phagocytosis. The addition of antibiotics to lavage fluids does not increase survival rates of animals with bacterial peritonitis but may be helpful as a prophylactic measure in preventing its development in patients undergoing peritoneal dialysis.

Heparin has decreased adhesion formation and increased survival time in some types of experimental peritonitis. Heparin possibly works by allowing PMN's better access to bacteria through reduced fibrin deposition.

Diffuse peritonitis is not effectively treated by silicone or latex rubber tubes alone. These are rapidly surrounded by fibrin and omentum and peritoneal drainage ceases rapidly. Drainage tubes are only useful in selected types of localized peritonitis such as that caused by a prostatic abscess. With intermittent peritoneal lavage sterile fluids are administered via a peritoneal dialysis catheter or fenestrated tube. The fluids are then removed by gravity flow back through the inflow tube. Our clinical impressions are that this technique has been helpful in reducing mortality associated with diffuse peritonitis. However experimental results suggest that good lavage of the entire abdomen is not provided. The development of hypoproteinemia and hypokalemia is a common sequelae.

Open peritoneal drainage is a technique whereby instead of closing the abdominal incision interrupted nylon or polypropylene sutures are place loosely in the linea alba allowing it to gap 1-2 cm. The skin and subcutaneous tissues are not closed and are covered with antibiotic ointment and a non-adherent gauze dressing before applying a large cotton padded bandage. The animal is continuously observed and the bandage aseptically changed at 12-24 hour intervals using heavy sedation. The abdominal wound is closed primarily in 1-5 days depending on when the peritoneal inflammation has resolved. Common complications associated with open peritoneal drainage are patient dehydration and hypoproteinemia secondary to massive fluid and protein loss into the bandage. A recent study of the efficacy of open peritoneal drainage in 25 dogs and cats revealed there was a 48% mortality associated
with the procedure. Admittedly these were all cases with severe diffuse peritonitis in which a high mortality rate would normally be expected. A follow up to that study indicated a mortality rate of only 34% in 24 cases.

References

ENTEROTOMY OR ENTERECTOMY? VIABILITY ASSESSMENT

Gary W. Ellison, DVM, MS, Diplomate ACVS
University of Florida, College of Veterinary Medicine, Gainesville, FL

Additional Reading:

Indications
The most common indication for enterotomy in small animals is to remove intraluminal intestinal foreign bodies that cause obstruction. Foreign bodies can be present in animals of any age, but they are most common in puppies or kittens because of indiscriminate eating habits. Common intestinal foreign bodies in dogs include bones, balls, corncobs, and cellophane wrappers. Cats commonly ingest sharp foreign bodies (e.g., straight pins and needles) and linear foreign bodies (e.g., yarn, tinsel, fishing line, and string meat wrappings). Enterotomy also is performed to examine the intestinal lumen for evidence of mucosal ulceration, strictures, or neoplasia. Superficial ulcerations or intestinal polyps sometimes can be resected via enterotomy, but most intramural lesions require intestinal resection and anastomosis.

Determining Intestinal Viability
With a complete obstruction, intestinal distention is often severe and the distended loops of bowel take on a cyanotic appearance. Intestinal viability is best evaluated after: 1) decompression of dilated loops of intestine, and 2) removal of the foreign body. Decompression of fluid and gas from the proximal segment of the distended bowel is performed with a 20-gauge needle and suction apparatus or a 60 cc syringe with a three-way stop cock. If intestinal wall ischemia and necrosis is present, then resection and anastomosis is performed immediately. However, in most cases of simple non-strangulated obstruction, bowel viability is maintained and the visual appearance of dark distended loops of bowel improves rapidly after removal of the obstruction.

Usually non-viable intestine is distended, blue, black or grey in appearance and easily discernable from normal bowel. Sometimes determining viability in cyanotic appearing bowel is difficult. The intestine should be decompressed with a needle and suction apparatus to relieve venous congestion. Standard clinical criteria for establishing intestinal viability are color, arterial pulsations, and the presence of peristalsis. Of these three parameters, peristalsis is the most dependable criteria of viability. The "pinch test" should be performed on questionable areas of bowel to determine whether smooth muscle contraction and peristalsis can be initiated. If clinical criteria are inadequate to determine viability, intravenous fluorescein dye or surface oximetry can be used. A 10 percent fluorescein solution (Fundescin-10, Cooper Laboratories, San Germain, PR) is given at a dosage of 1 mL/5 kg intravenously through any peripheral vein. After 2 minutes, the tissues are subjected to long-wave ultraviolet light (Wood's lamp). Areas of bowel are considered viable if they have a bright green glow. Areas of bowel are not viable if they have a patchy density with areas of nonfluorescence exceeding 3 mm, have only perivascular fluorescence, or are completely nonfluorescent. O2 saturation is also shown to be a reliable
method of determining intestinal wall viability. A sterile probe is placed on the surface of the bowel and an oxygen saturation level reading will occur. According to published reports in people saturation levels above 81 percent or above typically mean that the bowel is viable whereas values below 76 percent were consistent with mucosal necrosis and those below 64 percent indicated transmural intestinal necrosis.

Intestinal Foreign Bodies

Incidence/Clinical Signs

Surgical management of gastrointestinal foreign bodies varies depending on the type and location of the foreign body. Sharp foreign bodies such as straight pins, safety pins, bones, nails, or glass will usually pass through the gastrointestinal tract without creating intestinal perforation. Rubber balls, cellophane, or corn cobs tend to pass slower or not at all and are more likely to cause complete mechanical obstruction requiring emergency laparotomy.

Enterotomy Surgical Technique

A ventral midline laparotomy incision is made. The entire intestinal tract should be evaluated to determine the number of foreign bodies and assess the viability of the bowel wall. The affected bowel segment is isolated from the remainder of the viscera with saline-soaked laparotomy sponges. In patients with a complete obstruction, intestinal distension proximal to the obstruction is often profound, and the distended loops of bowel usually take on congested or cyanotic appearance.

Intestinal contents are milked 10 cm to either side of the foreign body and the bowel is held between an assistant’s fingers or with Doyen intestinal forceps. With a No. 15 scalpel blade, a full-thickness longitudinal incision is made in the antimesenteric border of the intestine in the viable tissue immediately proximal or distal to the foreign body. The length of the enterotomy approximates the diameter of the foreign body. Continuous suction is used to reduce spillage, and the surgeon pushes the foreign body gently through the enterotomy, taking care not to tear the incision margins. The bowel lumen is examined for evidence of perforations or strictures before closure.

Linear foreign bodies such as string, fishing line, meat wrappers, and sewing yarn present a difficult surgical problem. The trailing end of a linear foreign body usually catches over the base of the tongue or in the pyloric antrum and acts as an anchor. Intestinal peristalsis attempts to move the foreign body distally, but because it remains fixed proximally, the bowel plicates itself along the length of the foreign body, which often cuts through the intestinal wall on the mesenteric surface, resulting in local peritonitis.

Linear foreign bodies should be managed by identifying the glossal anchor point initially and releasing it before laparotomy. Commonly, a gastrotomy is also necessary to free wadded string or fishing line from a gastropyloric anchor. The traditional way for linear foreign body removal requires multiple enterotomies to complete removal of the linear body. If too few enterotomies are made with too much traction placed on the linear body, the mesenteric border may be perforated in an area that is difficult to explore and suture. Occasionally, the intestinal foreign body perforates at several locations before surgery, and local peritonitis is evident. Sometimes, enough fibrosis has occurred around the foreign 

23
body so, even after its removal, the bowel retains its plicated conformation. In these patients, intestinal resection and anastomosis may be necessary.

Linear foreign body removal may often be facilitated using a urinary catheter technique. With this technique only one or two enterotomies are necessary. Once the foreign body is released from its proximal anchor point it is tied or sutured to the tip of an eight to 12 French vinyl urinary catheter. The catheter tip is then pushed distally along the pleated length of bowel. As the catheter is pushed distally, the imbedded linear foreign body disengages from the intestinal wall and the bowel unpleats itself. Once the foreign body is completely disengaged from the bowel wall a second short enterotomy is made distally over the distal tip of the catheter and the remainder of the foreign body is retrieved. Alternatively a longer catheter can be used and pushed down through the colon. The foreign body can then be retrieved from the anus (not shown). The author has found catheter facilitated removal to be a very useful method for linear foreign body retrieval.

**Surgical Technique for Enterectomy (Resection and Anastomosis)**

The area to be resected is packed away from the abdomen with moistened laparotomy sponges. Intestinal contents are milked proximally and distally, and the bowel is held between an assistant’s index fingers or with Doyen intestinal forceps 4 to 5 cm from the proposed resection site. A 1- to 2-cm margin of normal viable intestine is included in the proximal and distal boundaries of the area to be resected, which is clamped with Carmalt or Doyen forceps. If luminal disparity is present, the forceps are placed at a 75 to 90 degree angle on the dilated proximal segment and at a 45 to 60 degree angle on the contracted distal segment of bowel. Branches of the mesenteric artery and veins supplying the devitalized bowel are isolated with curved mosquito forceps and are double-ligated. The arcadial vessels located within the mesenteric fat are double-ligated at the area of proposed resection. A scalpel blade is used to excise the bowel along the outside of the intestinal forceps. With dissecting scissors, the vessels are divided, the mesentery is transected, and the excised bowel is removed from the surgical field. After resection, the small intestinal mucosa has a tendency to evert and can be trimmed back with Metzenbaum scissors.

If angling the intestinal incision does not adequately correct for luminal disparity, the smaller stoma can be enlarged by incising the bowel section for a distance of 1 to 2 cm along the antimesenteric surface and then trimming off two triangular flaps. This procedure creates an ovoid larger stoma, which can be anastomosed to the larger-diameter section of the bowel.

When the anastomosis is closed with a simple interrupted suture technique, the first suture is placed at the mesenteric border because the presence of fat in this area makes suture placement most difficult, and this is where leakage is most likely to occur. The second suture is placed on the antimesenteric border, and the third and fourth sutures are placed laterally at the 90 degree quadrants. Depending on bowel diameter, two to four more sutures are placed between each of the quadrant sutures. All sutures are placed 3 to 4 mm apart and 2 to 3 mm from the wound edge. Suture bites on the dilated side of the anastomosis are placed farther apart than on the contracted side of the anastomosis to correct for luminal disparity. Once one side of the anastomosis is sutured, the bowel is flipped over, and the opposite side is completed. From 12 to 20 sutures are used to complete the anastomosis. After the anastomosis has been completed, it is checked for leakage by infusing saline under low pressure into the bowel lumen and massaging the fluid past the anastomosis. The anastomosis can also be checked by gently probing the spaces between
sutures with mosquito hemostats for openings. The surgeon then closes the mesenteric defect with a simple continuous pattern, taking care not to include any mesenteric vessels within the suture line.

Occasionally, the small-diameter loop of bowel cannot be enlarged enough to be anastomosed to the larger one. In this case, the large-diameter stoma is reduced by initially angling the cut at 45 degrees. The anti-mesenteric portion of the incision is then apposed with simple interrupted sutures in side-to-side fashion until the remaining opening is an appropriate width to anastomose to the smaller-diameter loop of bowel.

Alternatively, a simple continuous approximating technique can be used to create the anastomosis. This is performed with two lengths of suture. The first knot is tied at the mesenteric border and the second at the antimesenteric border. The sutures are then advanced around the perimeter approximately 3 mm from the cut edge, with the wound edges gently approximated. The needles are advanced in opposite directions, so one knot is tied to the tag at the antimesenteric border. The final knot is tied to the tag on the mesenteric border. If the knot is tied too tightly, a pursestring effect will be produced, and stenosis of the anastomosis may occur. The completed anastomosis is tested for leakage, and the mesenteric defect is closed.

A rapid alternative to sutured anastomosis is the use of stainless steel skin staples. Three stay sutures are used to triangulate the bowel ends and an end-to-end anastomosis is performed with an AutoSuture 35 skin stapler with stainless skin staples (United States Surgical Corp., Norwalk, CT). After triangulating the intestine with three stay sutures, the skin stapler is used to place staples every 2-3 mm around the perimeter of the wound. These closures are more rapidly done than handsewn anastomosis and have similar bursting strengths, but some mucosal eversion is created.

Leakage of any intestinal anastomosis is most common in animals with pre-existing peritonitis, low serum albumin and in those animals where intestinal foreign bodies have created intestinal ischemia. To help prevent anastomotic leakage, a pedicle of greater omentum is wrapped around the suture line. The omentum is critical to the successful healing of intestinal wounds because it can seal small anastomotic leaks and can prevent peritonitis. Dogs with the greater omentum removed have significant morbidity and mortality associated with intestinal anastomosis, whereas most dogs survive and do well when the omentum is retained. The omentum is tacked to the serosa with two simple interrupted sutures of 3-0 suture material placed on each side of the bowel wall.

Postoperative Care

Fluid and electrolyte deficits are corrected and antibiotic therapy is continued in the postoperative period. The author uses metoclopramide 2.2 mg/kg IV every eight hours to reduce ileus and promote intestinal motility. Feeding a bland diet such as canned I/D gruel (Hills Pet Nutrition Inc., Topeka, KS) is initiated the day following surgery. In uncomplicated cases, reasonable appetite usually resumes within 48 hours. Anorexia or vomiting in the presence of fever, abdominal tenderness, and leukocytosis suggests that anastomotic leakage and peritonitis may have occurred. If degenerate neutrophils with engulfed bacteria or free peritoneal bacteria are present on abdominocentesis, early reexploration of the abdomen is warranted. Further resection and reanastomosis or use of one of the serosal patching techniques described later in this section may be required. Aggressive treatment of generalized peritonitis may be needed to salvage the patient.
Intussusception

Etiology and Clinical Signs

Intussusceptions are most often seen in immature animals. The exact biomechanical cause of the condition is unknown and has not been reproduced experimentally. Probably a local incongruency of the intestine caused by induration or spasticity (intestinal parasitism) or sudden diameter change (ileocecal area) occurs in which a proximal bowel segment invaginates (intussusceptum) into a distal section of bowel (intussuscipiens). Three layers of bowel wall are thus created if one were to make a cross section through the intussusception. Heavy intestinal parasitism with ascarids or coccidia as well as severe enteritis seen with canine distemper are reported as predisposing causes. Intussusceptions are also seen with increasing frequency after laparotomy on elective or non-elective intraabdominal procedures. Recently, we have seen a high number of cases associated with parvovirus enteritis in puppies. Although conclusive studies regarding morbidity and mortality are not available, early experience suggests that these cases carry a more guarded prognosis. Increased mortality of these cases may be due to 1) predisposing fluid and electrolyte imbalances, 2) agranulocytosis and secondary bacterial septicemia, 3) increased tendency for the intussusceptions to recur.

Clinical signs depend on the completeness and level of obstruction. The majority of intussusceptions occur at the ileoceocal junction but jejunojejunal or even higher pylorogastric and gastroesophageal intussusceptions are reported. Patients with high intussusceptions usually undergo profuse vomiting, rapid dehydration and early death. Ileocolic intussusceptions often present with a history of sporadic vomiting, inappetence, or bloody stools.

Pathophysiology

Acuteness or severity of signs also depends on the seriousness of the obstruction. With an acute massive intussusception, vomiting, rapid dehydration and electrolyte imbalance may be seen. In the early stages, venous congestion causes the walls of the intussusceptum to become edematous, turgid and engorged with blood. The intussusception rapidly becomes irreducible as a result of the congestion and outpouring of fibrinous exudate from the serosal surfaces. As vascular supply is more completely embarrassed, ischemia and necrosis of the invaginated bowel occurs.

In most cases, the obstruction is incomplete and a chronic course of inappetence or bloody tenesmus is seen for several weeks. Although the invaginated bowel may become devitalized, perforation is rare because the outer ensheathing layer retains its viability and fibrinous adhesions seal the proximal border of the intussusception. Occasionally spontaneous recovery occurs when the nonviable intussusceptum is sloughed and patency of the intestinal lumen is reestablished.

Diagnosis

Diagnosis of intussusceptions can usually be accomplished by simple abdominal palpation and radiography. A cylindrical sausage shaped mass located in the mid to caudal sublumbar abdomen is pathognomonic for the disease. An obstructive pattern is seen on plain radiographs. With an upper GI series or Barium enema, and "coiled spring" appearance of the intussusceptum may be seen.
Surgical Treatment

Surgical management of intussusceptions involves: 1) reduction and or 2) resection and anastomosis coupled with 3) prophylactic enteropexy. Upon identifying the intussusception, it is isolated and packed off from the peritoneal cavity. Reduction is facilitated by gentle milking of the intussusceptum from the intussuscipiens. The ensheathing layer is gently compressed over the apex of the intussusceptum while gentle traction is placed on the ileum. In relatively acute cases, reduction is usually accomplished and bowel viability is closely scrutinized. When mature adhesions have formed between the invaginated and ensheathing layers, reduction is usually not possible, and resection and anastomosis are performed.

Following reduction and/or resection and anastomosis of the intussusception, a bowel plication or enteropexy technique should be performed. Bowel plication involves laying the bowel side by side in a series of gentle loops. At least three loops of plicated bowel are used proximally and three distally to the origin of the intussusception. The loops are sutured together on their antimesenteric border using simple interrupted sutures of 3-0 to 4-0 chromic surgical gut, which penetrate the seromuscular layers of bowel but does not enter the lumen. An alternative to this is an enteropexy technique where the intestinal serosa is sutured to the adjacent areas of the bowel and to the peritoneal surface using surgical gut sutures, which are placed every eight to twelve cm.

Postoperative Management

Postoperative care of the patient involves fluid and electrolyte support (previous section) and treatment of the primary cause if identifiable. Appropriate anthelminthics should be prescribed for endoparasitism. Antidiarrheals (Kaolin and Pectin) or poorly absorbed (Neomycin) antibiotics may be used if bacterial enteritis is felt to be the cause. Recurrence of the intussusception is not uncommon and usually occurs aborally to the plicated segment of bowel within a few days of the initial surgical repair. Return of vomiting or inappetence usually signals a recurrence and necessitates further surgical repair. The use of anticholinergics post-operatively is not recommended, since this may merely compound the often grossly distended dilated loops of bowel which are present.

Intestinal Neoplasia

Lymphosarcoma and adenocarcinoma are the most common intestinal tumors in dogs and cats. Leiomyosarcomas or leiomyomas are seen less commonly.

Intestinal lymphosarcoma may occur as a diffuse infiltrative lesion or a distinct nodular mass. Extensive infiltration of the lamina propria and submucosa with neoplastic cells occurs. Malabsorption, steatorrhea, diarrhea and weight loss are usually seen. With mural masses varying degrees of intestinal obstruction may occur.

Intestinal adenocarcinoma is seen in older animals. In dogs, adenocarcinoma is seen more commonly in the colon and duodenum whereas in cats the ileum is a more common location. The Siamese cat is at increased risk. Intestinal adenocarcinoma usually causes a segmental intramural mass which expands toward the lumen. This constricting ring which is classically called a "napkin ring lesion" usually undergoes very little external enlargement and may be mistaken for a stricture. Local invasion or seeding of the mesentery omentum and regional lymph nodes is common. Sporadic vomiting, anorexia and weight loss are the most common clinical signs.

Diagnosis of intestinal neoplasms can sometimes be made on palpation but often requires contrast radiography. Distinguishing neoplasia from other conditions that cause
intramural obstruction may be difficult or impossible until exploratory laparotomy and biopsy is performed.

Surgical treatment involves wide resection of the neoplasm and anastomosis of adjacent segments of bowel, if metastasis is not apparent. Resection of regional lymph nodes is advisable but usually not possible. Prognosis for adenocarcinoma or LSA is poor in that margins are often infiltrated or regional lymph nodes involved. Often the animal is extremely cachectic and hypoproteinemic so that chances for leakage or dehiscence of the intestinal anastomosis is high. Average survival time is less than six months, however, survival times of better than two years are reported in selected animals. Effective chemotherapy is not available for adenocarcinoma. For LSA chemotherapy is effective in reducing tumor mass, but may "melt" the tumor so that intestinal perforation may result. For this reason we are now often resecting obstructive lymphosarcoma lesions prior to instituting chemotherapy.

References


Enterotomy Closure

Closure of the enterotomy incision usually is performed with a simple interrupted suture pattern in side-to-side longitudinal fashion. Single-layer closures are recommended because double-layer closures may cause excessive narrowing of the lumen diameter. Various suture patterns are acceptable, but with all techniques, the vascular and collagen-rich submucosa must be incorporated in the sutures. Single-layer appositional techniques such as the simple interrupted appositional is most commonly used. A simple interrupted approximating suture can be used. Sutures are placed 3 to 4 mm apart and 2 to 3 mm from the cut edge, taking care to incorporate all layers of the intestinal wall. Crushing sutures are tied tightly and cut through the muscularis and engage the submucosa. The author feels they should be avoided since they cause excessive hemorrhage and tissue ischemia. I prefer a modified Gambee suture, which incorporates the serosa, muscularis and submucosa but excludes the mucosa and is helpful in reducing mucosal eversion.

The enterotomy also can be closed using a simple continuous approximating pattern. Suture bites are taken perpendicular to the bowel wall 2 to 3 mm from the cut edge and 3 mm apart. The suture line is advanced outside the bowel lumen. Sutures are pulled snugly enough to appose the wound edges gently. Pulling the suture line too tightly may cause strangulation of the wound edge and may lead to dehiscence. Some surgeons tend to close the enterotomy with a Cushing pattern. A continuous inverting Cushing pattern gives good serosa-to-serosa apposition and luminal bursting strengths that exceed those of the interrupted approximating patterns for the first day post-operatively. However, lumen diameter is reduced. Suture bites are placed 2 to 3 mm from the wound edge to minimize the amount of inversion. The tough submucosal layer is secured with each pass of the needle.

A rapid method of closing multiple enterotomies involves the use of a regular dimension skin stapler (AutoSuture multifire premium, United States Surgical, Norwalk, CT). Traction sutures are placed on both ends of the enterotomy and full thickness skin sutures are placed every 2-3 mm. If the enterotomy is made in a small-diameter loop of bowel, longitudinal closure may cause luminal constriction. To prevent this constriction, the ends of the enterotomy can be closed in transverse fashion. A simple interrupted suture is used to approximate the proximal and distal ends of the longitudinal incision. Additional sutures are then placed 3 to 4 mm apart to appose the remaining bowel wall, resulting in a widened lumen diameter. For intestinal biopsies and for enterotomies in small animals I prefer to make a short transverse incision which goes not more than 30-40% around the diameter of the enterotomy and then close this wound transversely. I find that making the wound in this direction preserves lumen diameter better than either a longitudinal incision with side to side or transverse closure.

I prefer to close enterotomies with 3-0 to 4-0 synthetic monofilament suture material. Acceptable materials include polidioxanone (PDS, Ethicon, Inc.), poliglecaprone 25 (Monocryl, Ethicon, Inc.), polyglycomer 631 (Biosyn, United States Surgical) or polyglyconate (Maxon, United States Surgical) on a narrow-taper, taper-cut, or small reverse-cutting.
need1e. Due to their rapid absorption time poliglecaprone 25 (Monocryl, Ethicon, Inc.) and polyglycomer 631 (Biosyn, United States Surgical) should be avoided in colonic surgery. Chromic surgical gut has been used with clinical success, but it is not recommended for intestinal closure because it loses tensile strength rapidly in the presence of collagenase and is quickly phagocytized in an infected environment. Nonabsorbable monofilament materials such as nylon (Ethicon, Ethicon, Inc.) or polypropylene (Prolene, Ethicon, Inc.) also may be used but have foreign bodies that may become attached to their exposed intraluminal segments. Stainless steel skin staples are reported to migrate into the lumen of the bowel and may be extruded in the feces. After the enterotomy closure is complete, it is rinsed with saline and covered with omentum, as described in the next section.

**Anastomotic Pattern and Suture Material**

Although numerous suture techniques have been used for end-to-end intestinal anastomosis in small animals, approximating patterns are recommended at present. Properly performed approximating techniques create a lumen diameter comparable to normal, result in rapid and precise primary intestinal healing, and minimize the potential for postoperative adhesion formation. Everting techniques (e.g., horizontal mattress pattern) initially create a larger lumen diameter, but ultimately they cause narrowing and stenosis of the lumen. Everting anastomoses are not recommended because they have a greater tendency to leak and because of delayed mucosal healing, prolonged inflammatory response, and increased adhesion formation compared with approximating anastomoses. Inverting anastomoses using Cushing or Connell patterns provide a temporary leak-resistant serosa-to-serosa approximation but they create an internal cuff of tissue, which may cause luminal stenosis. Inflammation is more severe and healing time is slower than with approximating techniques. Despite these dangers, inverting techniques should be considered in patients with a high risk of leakage or for use in colonic resection and anastomosis; in the latter situation, the high bacterial content of feces makes leakage of the anastomosis extremely dangerous.

Approximating end-to-end intestinal anastomoses can be created with various simple interrupted suture patterns or with a simple continuous suture pattern. Interrupted patterns generally are easier to perform, but the simple continuous pattern minimizes mucosal eversion and therefore provides better serosal apposition and primary intestinal healing. Regardless of the suture technique used, proper incorporation of the tough submucosa and reduction of mucosal eversion are vital in performing consistently successful intestinal anastomosis.

A simple interrupted appositional suture incorporates all tissue layers and gently apposes the wound edges. A crushing suture is pulled tightly and cuts through the serosa, muscularis, and mucosa and engages only the tough submucosal layer of the bowel wall. Crushing sutures create more microhemorrhage and tissue necrosis directly at the anastomosis and the author feels they should be avoided. With both the appositional and crushing techniques, mucosal eversion tends to occur between sutures. I prefer a modified Gambee suture pattern because it reduces mucosal eversion. In this technique, the needle is passed through the serosa, muscularis, and submucosa, but the mucosal layer is not incorporated in the suture. The suture is tied snugly enough to approximate all layers of the intestinal wall gently. The mucosa tends to be pushed into the intestinal lumen and does not evert between sutures.
Selection of Suture Material, Needle Type and Number of Sutures

Both absorbable and nonabsorbable suture materials have been used successfully for anastomosis. The braided nonabsorbable suture materials such as silk or Dacron may harbor bacteria create granulomatous inflammatory reaction or draining suture sinus. Monofilament non-absorbable sutures such as Nylon and polypropylene are safe in contaminated environments. However, polypropylene has been associated with foreign body adherence in one case series. Absorbable suture materials are usually used since the GI tract heals very rapidly and suture tensile strength is only needed for 2-3 weeks. Absorbable suture materials reported in the literature include chromic gut, polyglycolic acid (Dexon), polygalactin 910 (Vicryl), polydioxanone (PDS) and polyglyconate (Maxon) and poliglecaprone (Monocryl). Of these, surgical gut is not recommended for anastomosis because it is rapidly broken down by collagenase. Polygalactin 910 and polyglycolic acid are multifilament derivations of glycolic acid which retain good tensile strength for up to 28 days. Both sutures have good knot tying and handling characteristics with the exception of significant tissue drag. Vicryl is commonly used for intestinal anastomosis in Europe with good published success. Polydioxanone (PDS) and polyglyconate (Maxon) are polyester monofilament suture materials which are also absorbed by hydrolysis and therefore are unaffected by contaminated environment. They maintain up to 40% of their original tensile strength after 3 weeks. Many surgeons are starting to use shorter acting monofilaments such as Monocryl or Biosyn for intestinal anastomosis. They have similar handling properties to PDS but its tensile strength are resorbed by within 10-21 days. The newer “Plus” sutures are impregnated with the antibacterial agent Triclosan. Their efficacy in reducing infection in contaminated dermal wounds may foster an increased use in intestinal anastomosis.

Suture size, needle type and number of sutures are also important factors to consider. For cats, I use 4-0 suture on an RB1 needle. Usually 12-16 sutures are needed to complete the anastomosis. For dogs I typically use 3-0 suture on an SH needle and 18-20 sutures are needed to complete the anastomosis.

Small intestinal resection is limited to 70% of its length in adult dogs and 80% in puppies. Beyond that short bowel syndrome with malabsorption, maldigestion and chronic diarrhea will result. After transection, the wound edges are trimmed to remove everted mucosa and suturing is begun at the mesenteric border. Sutures are then placed on the anti mesenteric border, then at the 3 and 9 o’clock position before filling in the gaps.

All anastomosis should be covered with a vascularised omental flap which is tacked in place. Omentum is useful in 1) restoring blood supply to a devascularized area, 2) facilitating lymphatic drainage, and 3) minimizing mucosal leakage and secondary peritonitis. The role of omentum is significant when one considers that in one study 90% mortality rates were seen with intestinal anastomoses after omental resection was performed in dogs. Free omental flaps are not as effective as pedicle omental flaps and may in fact lead to anastomosis failure.

A rapid alternative to sutured anastomosis is the use of stainless steel skin staples. Three stay sutures are used to triangulate the bowel ends and an end-to-end anastomosis is performed with an AutoSuture 35 skin stapler with stainless skin staples (United States Surgical Corp., Norwalk, CT). After triangulating the intestine with three stay sutures, the skin stapler is used to place staples every 2-3 mm around the perimeter of the wound. These closures are more rapidly done than handsewn anastomosis and have similar bursting strengths, but some mucosal eversion is created.
After the anastomosis has been completed, the mesenteric defect is closed with a simple continuous pattern taking care not to include the mesenteric vessels within the sutures. The anastomosis is then covered with a pedicle of greater omentum. The omentum is critical to the successful healing of the intestinal wounds especially in patients with peritonitis. In one study, 9 of 10 dogs with experimentally induced peritonitis died after intestinal anastomosis whereas 10 out of 10 dogs survived when the omentum remained.

**Serosal patching** utilizes the antimesenteric surface of the small bowel to cover or buttress an adjacent area of questionable tissue viability or an area that cannot be reliably sutured. Jejunum is commonly used because its freely movable mesentery allows it to be mobile. The serosal patch provides mechanical stability and will help to induce and localize a fibrin seal over the questionable area.

**Surgical Technique for Serosal Patch**

A section of jejunum free of mesenteric tension is transposed over the perforation or area to be buttressed. It is important not to stretch, kink, or twist its mesenteric root or the vascular supply may be disrupted. The bowel chosen for the patch is then gently looped to prevent luminal bowel obstruction. Multiple perforations sometimes require patching using a back and forth looping of the entire segment of bowel. The lateral aspects of the bowel wall or antimesenteric border is used for the patch. The patch is not sutured directly to the edges of the defect but rather 3-4 mm beyond its margins. Simple interrupted sutures of 4-0 nylon or polypropylene are placed 3-4 mm from the wound edges and 3-4 mm apart. The sutures grasp the submucosa of the patch and bowel wall but do not penetrate the lumen.

Peptic ulcerations in the gastric fundus may mandate a partial gastrectomy. Peptic ulcerations of the proximal duodenum are often difficult surgical tasks because of their close proximity to the pancreatic duct or common bile duct and major duodenal papilla. If the ulcers are located on the antimesenteric border they can be debrided and closed or buttressed with a serosal patch or jejunal onlay graft. More commonly, they are located on the mesenteric border. The margins of the ulcer may involve or be closely adjacent to the major duodenal papilla and common bile duct. Resection of the involved proximal duodenum and pylorus is sometimes necessary, and a gastroduodenostomy (Billroth I) or gastrojejunostomy (Billroth II) is performed. If the major duodenal papilla or bile duct is included in the resection, then transposition of the bile duct (choledochoduodenostomy) or gallbladder (cholecystoduodenostomy) must be performed.

**Suggested Readings**

Metabolism of the Traumatized Patient

Tissue trauma, sepsis, burns and major surgery all induce major metabolic changes within the small animal patient. With each of these stresses the animal’s basic metabolic rate is accelerated and protein catabolism occurs leading to a potential state of negative nitrogen balance. The metabolic response to injury is an adaptive mechanism whereby the body mobilizes and accelerates immune defense and wound repair mechanisms in the face of little or no nutritional intake. This metabolic response to injury is in contrast to starvation whereby the body lowers its metabolic rate and nitrogen loss to conserve body resources.

Protein-calorie malnutrition (PCM) occurs when the metabolic response to injury becomes prolonged due to the severity of the injury or because of a hypermetabolic state such as that caused by sepsis. When protein-calorie malnutrition is present cell mediated immunity is impaired, there is a higher risk of infection, wound healing may be impaired, anemia and hypoproteinemia may occur, generalized muscle weakness may be present and multiple organ dysfunction leading to death may occur. It may take only 5-10 days of anorexia to compromise the immune system and deplete the body’s muscular glycogen stores. Without adequate nutrition, during this period humoral and cellular immunodeficiency occurs and animals frequently die despite the use of aggressive antibiotic therapy.

Most anorexic animals that are too weak or sick to eat still have a GI tract which is capable of sufficient digestion and absorption of nutrients. This is especially true in the small intestine. Enteral alimentation offers the clinician an alternative to parenteral (intravenous) nutrition when normal ingestion of food is not possible. Enteral nutrition is both practical and easy to perform and not fraught with the expense and serious complications often seen with parenteral nutrition. The goal of enteral alimentation is to provide calories and protein sufficient to achieve a positive calorie and nitrogen balance in a safe and convenient manner.

Indications

Enteral feeding is indicated in those patients where anorexia is present and evidence of protein energy malnutrition is present prior to surgery. In animals where the stomach is functioning, feeding can be accomplished through the use of nasogastric, esophagostomy, pharyngostomy, or gastrostomy tubes. Nasogastric tubes are small bore (3.5-8 Fr) usually necessitating a liquid diet whereas esophagus or gastrostomy tubes are often larger (14-20 Fr) and a blended gruel of the animal’s regular food can often be fed through the tube. It is, however, important to strain the food prior to injecting it, since one small clump of food may plug the system. With the pharyngostomy or gastric tube the animal’s daily caloric needs can usually be met by dividing the feedings into 2-6 hour intervals. It is especially important to feed slowly through a pharyngostomy tube, since the possibility for regurgitation and aspiration does exist. In those patients where the stomach is not functioning or needs to be bypassed after surgery, the use of enteroostomy tubes or needle catheter jejunostomy is an effective way of feeding. Fluids, electrolytes and oral medications can also be administered through these tubes if necessary.
Placement of enterostomy catheters should be considered in those patients 1) that have an acute weight loss of 10% BW or chronic weight loss of 15-20% BW, 2) are hypoproteinemic, 3) are septic, 4) in gastric or pyloric resections, 5) in extra hepatic biliary diversions, 6) in multiple enterotomy or massive intestinal resections, 7) in any condition where chronic cachexia may adversely affect wound healing or 8) animals with unresponsive pancreatitis. Studies show that immunocompetence returns within as little as 48 hours after hyperalimentation. If the surgeon anticipates that oral feeding might be delayed or that hypermetabolic complications might be present, the tube should be placed at the time of surgery. These tubes are easily removed but difficult to place after surgery.

Surgical Techniques

**Nasoesophageal Tube** - This method of feeding is commonly used in cats and will also be tolerated by dogs. Lidocaine 2% is instilled down the nostril and a lubricated 3.5-5.0 Fr (cats) or 8 Fr (dogs) polyvinyl feeding tube is passed in a ventromedial direction through the ventral meatus of the nose. The tip of the tube is passed into the distal esophagus. Passage into the stomach may result in reflux esophagitis. Radiographs can be taken to determine the tube location. The end of the tube is then affixed to the cat's face with sutures. An Elizabethan collar is applied. The tube is capped. Feeding can be performed through intermittent bolus injections of 10/ml/kg or using a slow infusion of 2-4 mg/kg/hr.

**Pharyngostomy Tube** - The original technique suggested that the tube be placed rostral to the hyoid apparatus but aspiration pneumonia and airway obstruction resulted because of interference with the glottis, particularly in cats. Now it is recommended that the tube be introduced distal to the hyoid apparatus. A 10-16 Fr tube can be used in cats and a 16-24 Fr tube for dogs. Recently pharyngostomy tubes have fallen out of favor because of more complications in comparison to esophagostomy tubes.

**Esophagostomy Tube** - This method has been recently described and apparently eliminates much of the potential for airway obstruction and aspiration pneumonia. The tube is inserted using a curved Carmalt or Mixter right angle forceps. The forceps are advanced 3-4 cm past the larynx and then a stab incision is made over the intranasal. The tip of the catheter is then advanced into the lumen of the esophagus and either normograded directly down the esophagus or pulled back into the mouth, reversed and then inserted down the esophagus. The tube should be premeasured up to the level of the 10th rib to make sure it does not enter the stomach. A 14-16 Fr catheter is commonly used in cats and 18-20 Fr for dogs.

**Gastrostomy Tubes** can be placed through an open operative technique or via a percutaneous technique using either an endoscope (PEG tube) or nonendoscopic method using an Eld cannula (PNG tube). With the open technique a ventral midline approach is made and the stomach is exteriorized. A double pursestring suture is placed and a 16-22 Fr Foley catheter is introduced through the left flank and placed into the stomach lumen. The pursestring sutures are tightened and invert the stomach around the tube. Four simple interrupted preplaced sutures are used to draw the stomach to the oblique muscles and the balloon is inflated. With slight traction on the catheter, the sutures are tied. Omentum is wrapped around the gastrostomy site. The subcutis and skin are closed and the catheter is fixed to the skin using a Chinese finger snare suture.

With the percutaneous endoscopic gastrostomy (PEG) the patient is positioned with the left side up and the stomach is insufflated. A Bard or Pezar mushroom-tipped catheter is drawn "inside-out" by tying it to a piece of suture material which has been passed into the
stomach lumen percutaneously through an IV catheter and retrieved using an endoscope. Traction is placed on the catheter to keep the stomach wall against the peritoneum and prevent leakage. With the percutaneous nonendoscopic gastrostomy an Eld cannula is inserted through the oral cavity and into the stomach. The curved portion of the cannula is tipped up against the left body wall behind the 13th rib and the sharp stylet is pushed through the stomach and body wall. A piece of suture material is tied through the hole in the stylet and the cannula is retrieved out through the oral cavity. The mushroom catheter is then affixed to the suture and the remainder of the procedure is exactly like the PEG tube placement.

**Low Profile Gastrostomy Button**

Some patients require long-term or even permanent gastrostomy tube placement for conditions such as esophageal neoplasia, strictures, megaesophagus or as an aid to malnutrition caused by cancer or chronic renal failure. These patients may benefit greatly from the use of commercial low profile feeding devices such as the Passport® (Passport-Wilson-Cook Medical, Winston Salem, NC 27105 (800-245-4717)) or Surgitek One Step Button® (One Step®-Surgitek - Racine, WI 53404 (800-558-9494)). The Passport button has a one way valve that is convenient to use. It is made to be used as a replacement unit for a standard mushroom catheter. When the old mushroom catheter is pulled, the new unit is put in its place by merely collapsing the mushroom of the new unit with a stylet and gently inserting it into the old stoma. The one step button is placed exactly like a mushroom PEG tube, but once the unit is in place an outer wrapper comes off the guide tube and two side flanges, one with an attached stopper plug is left. The disadvantage of the Surgitek button is there is no valve and food spillage is more common.

**Jejunostomy Tube**

A loop of distal duodenum or proximal jejunum distal to the surgery site is isolated and a pursestring suture using 3-0 or 4-0 suture material is placed on the antimesenteric border. A commercially available jejunostomy tube, a 3-5 Fr vinyl feeding tube (introduced through a 12-14 gauge needle) or a 16 gauge through the needle polyvinyl catheter (Intra Cath, CR Bard Inc.) is passed through the abdominal wall 4-5 cm from the midline. The needle is then inserted through the intestinal pursestring suture and tunneled subserosally 1-2 cm before entering the intestinal lumen. The catheter is advanced aborally in the lumen for a distance of 20-25 cm, the needle is withdrawn, and the pursestring suture tied. The enterostomy site is then fixed to the abdominal wall using 4-6 preplaced sutures or a simple continuous pattern of 4-0 monofilament suture material. The catheter is then fixed to the skin using a finger trap suture or tape. An extension tube is attached and abdominal dressings are applied.

**Caloric Requirements**

The following chart describes the formula used by the author for estimating caloric needs. It must be remembered that basal caloric requirements increase by 50-150% depending on the degree of hypermetabolism.
Caloric Needs Estimation Chart

<table>
<thead>
<tr>
<th></th>
<th>Basal Needs</th>
<th>Major Surgery</th>
<th>Polytrauma/Burns-/Sepsis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cat</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Dog</strong></td>
<td>1-10 kg</td>
<td>110 kcal/kg</td>
<td>150-190 kcal/kg</td>
</tr>
<tr>
<td></td>
<td>75 kcal/kg</td>
<td>100-120 kcal/kg</td>
<td>160-200 kcal/kg</td>
</tr>
<tr>
<td></td>
<td>70-80 kcal/kg</td>
<td>90-110 kcal/kg</td>
<td>140-180 kcal/kg</td>
</tr>
<tr>
<td><strong>Dog</strong></td>
<td>10-20 kg</td>
<td>60-70 kcal/kg</td>
<td>120-160 kcal/kg</td>
</tr>
<tr>
<td></td>
<td>50-60 kcal/kg</td>
<td>75-90 kcal/kg</td>
<td></td>
</tr>
<tr>
<td><strong>Dog</strong></td>
<td>&gt; 20 kg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Protein requirements are estimated at 4.4-8.8 gm/kg BW for dogs and are probably slightly higher for cats. The protein content of most commercial liquid diets ranges from 16-20%. This is based on a current recommendation for people that 16% of available calories be provided by protein. This value is probably lower than ideal for use in dogs and cats.

**Diets**

There are two basic types of diets available for enteral tube feeding, polymeric and monomeric. Polymeric diets are composed primarily of polypeptides and polysaccharides (starches). They require digestion as well as absorption. The advantage of these diets is that they have low osmolality (350-450 mOsm/L) and therefore are less likely to promote osmotic diarrhea. Polymeric diets are cost effective and are good for use in most standard enteral hyperalimentation regimes. Polymeric diets used at the University of Florida include Isocal (Mead Johnson), Ensure (Ross Labs) and Pulmocare (Ross Labs). These diets have low osmolality and contain approximately 16% protein. Ensure and Isocal has a caloric density of 1 Kcal/ml while Pulmocare has a density of 1.5 Kcal/ml. Diet higher in protein are the Clinicare Diets produced by Ag Vet. These liquid diets are specifically formulated for dog (24%) and cat (28%) protein needs.

Monomeric are elemental diets composed of amino acids and monosaccharides with a minimal amount of fat content. The diets require minimal to no digestion and are absorbed over as little as 100 cm of bowel. They are hypoallergenic and have a low residue. Unfortunately, they are hyperosmolar (600-800 mOsm/L) and often cause diarrhea. Additionally they are relatively expensive. Monomeric diets are usually reserved for use in patients with significant intestinal tract disease. They are indicated for use in malabsorption syndromes or when short bowel disease is present. Examples of monomeric diets are Vital (Ross Labs) and Vivonex (Norwich Eaton). These diets are approximately 16% protein and have a caloric density of 1 Kcal/ml.

**Administration and Complications**

With esophagostomy or gastrostomy tubes feed no more than 30 ml/kg of body weight with each feeding. The liquid diets can be divided and injected in bolus form every 6 hours or they can be given at a slow continuous infusion of 5-10 ml/kg/hr. The main complications associated with enteral hyperalimentation are osmotic diarrhea and hyperglycemia. Osmotic diarrhea can usually be avoided by diluting the liquid diet with water by 50% on the first day...
and feeding only the calculated volume. The concentrations and volumes are then gradually
increased to that by day 4 or 5 the concentrated diet at full volume can be fed. Hyperglycemia
can often be controlled by slowing the diet administration rate.

An example of a starting regimen may be as follows:

<table>
<thead>
<tr>
<th>Day</th>
<th>Volume</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50%</td>
<td>50%</td>
</tr>
<tr>
<td>2</td>
<td>75%</td>
<td>50%</td>
</tr>
<tr>
<td>3</td>
<td>75%</td>
<td>75%</td>
</tr>
<tr>
<td>4</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Contraindications to the use of enteral hyperalimentation include adynamic ileus of the
intestinal tract and persistent diarrhea which is not controlled by administration rate or drugs.

References

2. Thatcher CD. Case presentations utilizing nutritional support. Eastern States Veterinary
Conference Proceedings, 1989;96.
3. Crow DT, Devey JJ. Nasal, nasoesophageal, nasotracheal, nasoesophageal, nasogastric and
and Wilkins, 1997;161-166.
1997;167-170.
1997;170-176.
7. Devitt CM, Seim HB. Use of jejunostomy and enterostomy tubes. In: Current Techniques in
Management of Megacolon in Cats

Etiology
Megacolon is defined as a dilated or elongated colon which often lacks smooth muscle motility. The condition can be seen in dogs or cats but is more common in the latter species. Megacolon can be congenital or acquired. Congenital aganglionic megacolon or Hirschsprung's disease results from a congenital absence of myenteric ganglion cells which produces an area of contraction and secondary dilatation. This condition has been described in one litter of Siamese cats. Congenital megacolon has also been reported secondary to atresia ani. Acquired megacolon is a more common clinical condition and is seen in adult cats and dogs. Acquired megacolon may be caused by obstructive lesions such as neoplasia, strictures or foreign bodies. Healed pelvic fractures may also obstruct the pelvic canal and lead to megacolon. Dietary irregularities such as ingestion of bones or sand may also create chronic constipation which results in an irreversible megacolon.

Idiopathic megacolon is a disease of middle-aged cats. It has been reported in cats as young as one year and as old as 15 years. The mean age however is about 5 years. In most cats the exact cause of megacolon remains obscure. Hence the term Idiopathic Megacolon. There are no characteristic histological changes, but it is believed there is a defect in the ability of the colonic smooth muscle to contract effectively.

Clinical Signs
Commonly, animals with megacolon won't pass stools for days or weeks. The large dilated colonic segment can be easily palpated through the abdomen. Plain film radiographs will disclose colonic dilatation and impaction. A barium enema may be performed after complete evacuation of the feces to try to locate a stricture. Proctoscopy if available is also very useful in the diagnosis of colorectal strictures.

Medical Management
Initial treatment involves complete evacuation of the colon. This usually requires general anesthesia, multiple soapy water or mineral oil enemas and fecal evacuation with sponge or clamshell forceps. Bran or cereal-based diets, stool softeners such as Metamucil or dioctyl sodium sulfosuccinate are then used in an attempt to manage the problem. Hypertonic sodium phosphate (Fleet) enemas should be avoided in cats because they may cause dehydration, hypernatremia, hyperphosphatemia, and tetany due to hypocalcemia.

The prokinetic agent cisapride (Propulsid - Jansen Pharmaceuticals) has been used in conjunction with oral lactulose to successfully treat some cats with idiopathic megacolon. Cisapride works by causing the release of acetylcholine from the enteric nervous system which stimulates colonic smooth muscle to contract. The treatment is effective in some cats but not in all yet it is worth trying prior to the recommendation for surgery. The dosage used at the University of Florida is 5 cc of lactulose and 5 mg of cisapride given BID to TID. Dosages of up to 7.5 mg of cisapride can be given safely. Once constipation is relieved some animals
can be maintained on alternate drug treatment. Although the treatment is successful in some cats many owners have to have subtotal colectomy performed because the cats develop an aversion to the lactulose therapy.

**Surgical Management**

Longitudinal resection or plication of the colon wall in an effort to reduce its diameter often has not worked because the primary cause was not removed. Megacolon secondary to strictures or neoplasia is sometimes reversed if the stenosis is resected in dogs. However, subtotal colectomy is a viable technique for treating idiopathic megacolon. In our hands the technique is reserved primarily for cats. We attempt to treat dogs medically because of their propensity for postoperative diarrhea. Some controversy exists as to whether the cecum should be preserved and a colon-to-colon anastomosis done or whether it can be sacrificed and an ileocolostomy performed. Colocolostomy is sometimes more technically difficult because the relatively immobile mesocolon places considerable tension on the suture line. Ligation of the ileocolic artery and removal of the cecum allows easy transposition of the mobile ileum down to the colonic stump. Preliminary studies indicate that removal of the cecum does not lead to ascending bacterial enteritis and cats with ileocolostomies do as well clinically as those with the cecum preserved. Colocolostomy can also be performed using a 1.8 mm end to end anastomosis autostapler (EEA, U.S. Surgical Inc.).

**Surgical Technique for Subtotal Colectomy**

When performing subtotal colectomy in cats the right colic and part of the left colic artery can be ligated but the cranial rectal branch off the left colic artery should be preserved. Impacted feces are milked into the enlarged colonic segment proximally and distally and intestinal forceps are applied. The proposed resection sites are at the ileocecal junction and in 1-2 cm cranial to the brim of the pubis. The ileum is anastomosed to the colon in end-to-end fashion using a simple interrupted pattern of 4-0 prolene. Luminal disparity is corrected by cutting the mesenteric surface of the colonic stump back at a 60° angle and opposing the cut edges with simple interrupted sutures until the stoma diameter approximates that of the ileal lumen. An alternative approach is to do an end-to-side anastomosis. The final anastomosis is wrapped with omentum. Preoperative bowel preparation is usually not possible and perioperative IV administration of Cefoxitin (40 mg/kg) 2 hours prior to and BID after surgery is recommended.

Copious intraperitoneal lavage is performed prior to abdominal closure. The use of intraperitoneal drains however, should be avoided. Studies have shown that they tend to increase risk of anastomotic dehiscence.

**Postoperative Course**

Cats are often somewhat depressed and anorectic for 48 hrs following surgery. They will sometimes have a moderate fever of 103-103.5°F in the absence of leukocytosis. Dark tarry liquid feces are usually noted for about 3-4 days. Feces remain liquid and poorly formed for 2-6 weeks. At which time they usually become soft and poorly formed (cow pie consistency) for the remainder of the cats life. Some excellent studies have recently been done looking at the physiologic after effects of this surgery. Most cats seem to maintain their normalbad, weight or even gain a little after the surgery. The cats generally use the litter box 2-3 times a day but the total amount of water loss in the feces equals that of normal cats. The
ileum increases its absorptive capacity by increasing villus height. Bacterial overgrowth is not a problem as evidenced by normal serum albumin levels in operated cats. Folic acid deficiency or anemia does not seem to be a problem either. The major complaint by some owners is chronic perineal soiling caused by the loose feces. If this becomes a problem, it can often be managed by clipping hair in the perineal area. Antibiotic therapy is dependent on presence or absence of peritoneal contamination. Onset of fever, abdominal tenderness, vomiting and positive peritoneal tap warrants early reexploration of the abdomen.

References

SHUNTS TO AND FROM THE INTESTINE: BILIARY DIVERSION, COLOSTOMY AND URETERO-COLONIC ANASTOMOSIS

Gary W. Ellison, DVM, MS, Diplomate ACVS
College of Veterinary Medicine University of Florida, Gainesville, FL 32610

Biliary Tract Obstruction

Incidence/Clinical Signs. Biliary obstruction may be caused by biliary concretions, stenosis of the common bile duct, neoplasia, choledolithiasis, or choledocholithiasis. Cholelithiasis occurs uncommonly in the dog and even less frequently in the cat. Proposed etiologies for cholelithiasis are bile stasis, infection, changes in bile composition, injury to the bile duct mucosa, and reflux of pancreatic juices. Cholelithiasis does not usually cause overt clinical signs in dogs or cats. Occasionally, choleliths formed in the gallbladder or bile ducts will pass into the common bile duct and cause permanent or temporary obstruction. Bile peritonitis may also result if the cholelith erodes the gallbladder wall. Clinical signs of biliary obstruction include anorexia, vomiting, abdominal pain, and icterus.

Diagnosis. Evidence of extrahepatic biliary obstruction is suspected with grossly elevated serum bilirubin levels of which 60-90 percent is in the conjugated form. Elevation of urine bilirubin to a 2-3+ level, and absence or reduction of urine urobilinogen levels further substantiates the obstruction. Clinical steatorrhea may also be seen.

Radiographic diagnosis of biliary obstruction is sometimes difficult because only 20-30 percent of the choleliths are radiodense, and oral or intravenous cholecystography is not always successful in identifying gallbladder pathology. The technique of percutaneous cholecystography has been recently described and will distinguish hepatic from posthepatic jaundice. The disadvantage of this technique is that it must be performed under fluoroscopy.

Fluid distention of the abdomen with the recovery of Ictotest positive coffee-colored fluid indicates that bile peritonitis is present and leakage from the hepatobiliary tree has occurred. Exploratory celiotomy is warranted.

Treatment. Biliary obstruction should initially be managed by cholecystotomy and exploration of the extrahepatic biliary tree with a probe or catheter. Obstructing calcium bilirubinate stones or inspissated bile may be removed from the gallbladder or dislodged from the bile ducts with saline under pressure. If severe cholangitis or erosion of the gallbladder wall is present, a cholecystectomy should be performed. A cholecystoenterostomy procedure is performed if the calculus can not be dislodged or secondary stricture of the common bile duct is present.

Cholecystotomy. The gallbladder of the dog and cat is a pear-shaped structure that lies between the quadrate liver lobe medially and the right medial lobe laterally. It is connected to the common bile duct by a single cystic duct. The common bile duct has three or four hepatic lobar ducts that enter at its proximal origin. It then enters the wall of the duodenum obliquely, expands into a well-defined ampulla, and opens into the lumen adjacent to the minor pancreatic duct at the major duodenal papilla.
Stay sutures are initially placed in the fundic region, following isolation and packing off of the gallbladder. Bile is aspirated with a 22-gauge needle and 20 cc syringe to reduce distention before the lumen is entered with a blade. Care is taken to minimize any bile spillage. The contents of the gallbladder are aspirated and all inspissated bile is removed using curettage, saline irrigation, and suction. A 3.5 French infant feeding tube is used to explore and flush the cystic duct, hepatic lobar ducts, and common bile duct to determine patency. If there is increased resistance to flow without apparent extra-hepatic biliary obstruction, potential obstruction at the papilla must be considered, and an enterotomy is performed. Once the obstruction is relieved, the gallbladder is ravaged and closed with a two-layer pattern. A simple continuous layer of 40 chromic catgut is covered by a continuous Connell suture pattern using the same suture. The gallbladder is distended with saline solution using a 22-gauge needle and inspected for leakage prior to abdominal closure.

**Cholecystectomy.** If chronic inflammation or erosion of the gallbladder wall is apparent, cholecystectomy is opted. After cholecystectomy, dilatation of the extrahepatic bile ducts occurs, but detrimental clinical signs have not been noted.

Cholecystectomy can be performed through a transdiaphragmatic incision after performing a right 8th intercostal thoracotomy or through a ventral midline/paracostal abdominal approach. After placing a stay suture in the apex of the gallbladder, it is gently dissected from its fossa. Saline may be injected suberosally to identify a better plane between the gallbladder and liver. The cystic artery is identified and ligated. The cystic duct is double-clamped and severed 5 mm from its junction with the common bile duct. The gallbladder is then removed. The abdomen is copiously irrigated before closure.

**Cholecystoenterostomy.** Bile stasis caused by cholelithiasis, fibrosis with stricture, and invasion of the common bile duct by neoplasia are all reasons why the common bile duct must be bypassed. The small diameter of the canine and feline common bile duct make resection and anastomosis or reimplantation difficult. Side-to-side anastomosis of the gallbladder lumen to the duodenum or jejunum allows retrograde flow of bile away from the obstruction and into the intestinal lumen.

The gallbladder fundus is partially dissected and freed to provide some mobility. The serosa of the jejunum or duodenum is approximated to the gallbladder serosa for a distance of 3 cm with 4-0 simple interrupted nylon sutures. Two longitudinal 2.5 cm incisions are made in the gallbladder and duodenum closely adjacent to the initial sutures. The mucosal surfaces of the gallbladder and duodenum are then approximated with a continuous Connell suture pattern of 4-0 chromic catgut, thereby creating a stoma. The remaining serosal surfaces are then approximated with simple interrupted 4-0 nylon sutures to prevent bile leakage.

**Postoperative Care/Complications.** If bile or bacterial peritonitis is present, multiple thorough irrigations of the peritoneal cavity are performed. A sump type of peritoneal catheter may be placed in the vicinity of the gallbladder fossa, and the area may be ravaged with sterile saline for 2-4 days postoperatively. Parenteral antibiotics with a broad spectrum of bactericidal activity are given for 7-10 days postoperatively. Adequate hydration is maintained with intravenous crystalloid solution. Postoperative hypoalbuminemia (less than 3 g/percent) may result from effusion of protein from the peritoneal surface. Whole blood or plasma transfusions may be needed to counteract this loss. Patients are kept NPO for 48
hours postoperatively and then begun on multiple feedings of a low fat ration to diminish cholecystokinin-induced bile secretion.

Ascending cholecystitis has been reported as a common sequela to a cholecystoenterostomy. The pathogenesis is thought to be due to a reflux of duodenal contents into the gallbladder. Clinical signs include fever, abdominal pain, vomiting, neutrophilia, and elevation of SGPT and SGOT. Patients usually respond to oral antibiotic therapy, but recurrent episodes are common. Creation of a stoma at least 2.5 cm in length may decrease gallbladder retention of ingesta and minimize the occurrence of postoperative cholecystitis.

**Colostomy.** Colostomy can be used to treat rectal perforation, stricture or colorectal neoplasia. Management of dogs with colostomy is difficult due to the ensuing fecal incontinence which occurs after the procedure and long term studies studying the efficacy of the technique in dogs are nonexistent. Two techniques have been described for colostomy in dogs both using the left flank for creation of the colostomy site. In the one technique a 3 cm circular incision is made in the left flank, the muscle layers are separated and the descending colon is exteriorized and supported with a straight plastic ostomy rod which is sutured to the underlying muscles. The serosa and muscularis are then sutured to the subcutaneous tissue, the colon incised longitudinally and the sub mucosa and mucosa is sutured to the skin. In the second technique a ventral midline approach is first made to transect and over sew the terminal colon and the abdomen closed. The patient is then turned and a left flank incision made similar to the first technique. The serosa and muscularis of the colon is sutured to the abdominal muscles and the mucosa and submucosa then sutured to the skin in an end to side fashion. Which both techniques the dog must be fitted with a colostomy bag. Irrigation of the colostomy between bag changes decreases the amount of feces collected in the colostomy bag.

**Ureterocolonic Anastomosis.** Ureterocolonic and trigonalcolonic anastomosis have been performed after sub total cystectomy for treatment of large bladder tumors in dogs. After cleaning enema's have been performed the implantation segment is isolated between Doyen forceps. The serosa and muscularis of the colon are incised to allow the submucosa to pouch out. A 3-5mm oval of submucosa is then incised and the end of the ureter is spatulated and anastomosed to the submucosa with 5or 6-0 suture. The seromuscular layer is then closed over the anastomosis site to create a tunnel. The right ureter is typically implanted 2-3 cm caudal to the left ureter. Diarrhea frequently follows this technique and metabolic abnormalities including hyperchloremic metabolic acidosis, hyperammonemia and elevated creatinine levels are commonly seen after the procedure. Development of pyelonephritis is also very common due to colonoureteral reflux of feces. Aggressive surgical techniques including cystectomy and ureterocolonic anastomosis have not resulted in prolonged survival times in dogs with transitional cell carcinoma.

**References**