Complications of Gastrointestinal Surgery in Companion Animals

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KEYWORDS

- Gastric dilatation • Volvulus • Gastropexy
- Laparoscopic gastropexy • Intestinal anastomosis
- Intestinal dehiscence • Short bowel syndrome
- Subtotal colectomy

The small animal surgeon routinely creates wounds in the gastrointestinal (GI) tract for biopsy, for foreign body or neoplasm removal for correction of gastric dilatation volvulus, or to relieve intestinal and colonic obstruction. Unlike dehiscence of a skin wound, which is often easily remedied with appropriate local wound treatment, dehiscence of a wound of the GI tract often leads to generalized bacterial peritonitis and potentially death. Consequently, technical failures and factors that negatively affect GI healing are of great clinical significance to the surgeon. Surgery of the GI tract must be considered clean-contaminated at best, and as one progresses aborally down the GI tract, the bacterial population increases. Therefore, intraoperative spillage, wound dehiscence, or perforations that occur in the lower small intestine or colon tend to be associated with a higher mortality rate than those of the stomach or upper small intestine.

WOUND HEALING OF THE GI TRACT

Basic understanding of GI tract healing is essential to the surgeon since it dictates proper clinical approach in those cases in which GI complications develop. Immediately after wounding, platelets aggregate, the coagulation mechanism is activated, and fibrin clots are deposited to control hemorrhage.[1] The fibrin clot offers minimal wound strength on the first postoperative day, but the main wound support during the lag phase of healing comes from the sutures.[2] Fibrin also has adhesive properties and may increase the risk of secondary obstruction since these fibrinous adhesions may ultimately be converted to fibrous adhesions. Enterocyte regeneration begins almost immediately after wounding; however, the epithelium offers little biomechanical

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support.[2] This *lag* or *inflammatory* phase is the most critical period during GI wound healing, and most dehiscences take place within 72 to 96 hours.[2]

The *proliferative* or *logarithmic* phase of GI wound healing lasts from days 3 through 14.[1] Fibroplasia occurs logarithmically during this period. The fibroblasts produce large amounts of immature collagen, resulting in rapid gains in wound strength, but this is a dynamic process in which collagen synthesis takes place in the presence of collagenolysis. In the stomach and small intestine, 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% that of normal tissue.[1] 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 post wounding.[1] Factors such as traumatic suturing, fecal contamination, and infection all increase the amount of local collagenase produced at the wound and hence can increase the risk of infection.[1]

The *maturation* 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.[1] Similar to skin wounds, the size and thickness of the scar decrease during this time without weakening the wound. The maturation phase is relatively unimportant clinically in GI wound healing, except in those cases where significant adhesions are present or in cases of sclerosing encapsulating or fibrosing peritonitis.[3,4]

COMPLICATIONS ASSOCIATED WITH GASTRIC DILATATION VOLVULUS

Predictors of Mortality and Gastric Necrosis

Mortality continues to occur in all published reports of gastric dilatation volvulus (GDV). However, over the past four decades, survival rates have improved due to early recognition, rapid gastric decompression, earlier cardiovascular resuscitation, and availability of better medical and surgical care. In older studies from the 1970s and early 1980s, *GDV-related mortality rates* ranged from 43% to 60%.[5,6] However, in a large epidemiologic study of over 1900 dogs with GVD, the mortality rate had reduced to 33% in cases evaluated during the late 1980s and early 1990s[7] and was even reported to be as low as 15% in the mid 1990s.[8] In a recent study the postoperative mortality rate of 306 dogs with GDV was 10%. Those dogs receiving gastropexy alone had mortality of only 3%, while dogs receiving partial gastrectomy with gastropexy had a rate of 9% and those receiving partial gastrectomy, splenectomy, and gastropexy had a rate of 20%.[9] That study also showed a reduction of mortality in dogs if the clinical signs occurred less than 6 hours prior to presentation. While one study suggested that reduced duration of clinical signs and shorter time from presentation to the surgery table also decreased mortality rate in 166 dogs with GDV,[10] a second study contradicted these findings.[11] Severity of clinical presentation has also been related to outcome, with recumbency at presentation increasing risk of death by 4.4 times and dogs with varying degrees of obtundation having mortality that ranged between 3% and 36%.[7–9,12]

The presence of preoperative *cardiac ventricular dysrhythmias* has been evaluated as a predictive indicator for survival in dogs with GDV since they may act as a sentinel for gastric or splenic ischemia. In one study, dogs having intermittent ventricular arrhythmia on admission had a significantly higher mortality rate than did dogs with ventricular tachycardia. Additionally, 48% of those dogs presenting with preoperative cardiac arrhythmias underwent splenectomy or partial gastrectomy, whereas just 27% of the dogs without arrhythmias required those procedures.[9] However, in