SURGICAL MANAGEMENT OF THE ACUTE ABDOMEN and REDUCING MORTALITY in PATIENTS WITH GASTROINTESTINAL DISEASE

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ACUTE ABDOMEN

Animals that present with an acute abdomen may have a variety of underlying causes. Prompt diagnosis and therapy are key to reducing mortality and morbidity. This lecture used a cased based approach to detail management of animals presenting with an acute abdomen.

UROABDOMEN

Bladder rupture is the most common cause of uroabdomen in dogs and cats. It may occur spontaneously (associated with tumor, severe cystitis, or urethral obstruction), be due to blunt or penetrating abdominal trauma, or be iatrogenic following cystocentesis or bladder catheterization or manual expression of the bladder. Urinary tract leakage may also be a complication of surgery. Any animal presenting after vehicular trauma should be assessed for possible urinary tract trauma. The impact of the collision may cause the bladder, urethra, or ureter to rupture or necrose. The sharp ends of pelvic fractures may sever or lacerate the urethra. Diagnosis is usually delayed because clinical signs are rarely present at initial examination (see below).

Immediate surgery is contraindicated in animals with uroabdomen that are hyperkalemic or uremic. They should first be treated medically to normalize electrolytes and acid base, as well as decrease circulating nitrogenous waste products. Intravenous fluids should be given and abdominal drainage performed. Penrose drains or a peritoneal dialysis catheter (preferred because it can be made into a closed system) can be placed in the ventral abdomen under local anesthesia (sedate if necessary) to allow drainage for 6 to 12 hours. This will stabilize most animals with previously normal renal function.

When urine leaks into the abdominal cavity, some nitrogenous waste products and electrolytes are reabsorbed across the peritoneal membrane and reenter the circulation. Whether molecules are reabsorbed depends on their size. Urea rapidly equilibrates across the peritoneal surface while some larger molecules (e.g., creatinine) cannot pass back into the bloodstream and remain concentrated in the abdominal fluid. Abdominal fluid creatinine

concentrations must substantially exceed serum concentrations to diagnose uroabdomen. Because urea rapidly equilibrates across the peritoneum, BUN may be approximately the same in both abdominal fluid and serum, regardless of the cause of the abdominal effusion. Potassium may alsohelp diagnose uroabdomen. A potassium abdominal fluid to blood ratio of greater than 1.4 to 1 is definitive for uroabdomen.

DIAGNOSIS

Clinical Presentation

Signalment. Urinary bladder rupture has been suggested as being more frequent in male versus female dogs because their long, narrow urethras cannot dilate rapidly; however, ruptured bladders are common in females that have sustained vehicular trauma. Traumatic urethral rupture in female dogs is uncommon. Male dogs and cats with obstruction due to calculi or sterile cystitis (FUS) have a high risk of bladder rupture if the obstruction is not alleviated promptly.

History. Clinical signs of urinary tract trauma are often vague and may be masked by other signs of trauma. In one study of dogs with pelvic trauma and concurrent urinary tract trauma, urinary trauma went clinically undetected in one third of the dogs. The animal may present for azotemia (i.e., vomiting, anorexia, depression, lethargy), or hematuria, dysuria, abdominal pain, and/or abdominal swelling or herniation may be noted. Abdominal and perineal bruising are common with vehicular trauma, particularly if there are pelvic fractures. Bruising in this region, however, may also indicate subcutaneous urine leakage. Further evaluation of the urinary tract is therefore warranted in such patients. In female dogs, there may be a history of previous catheterization using a rigid catheter. Rupture of the urethra is most frequently associated with pelvic fractures in male dogs. Urinary tract rupture is often overlooked in the initial workup of traumatized patients, and the diagnosis is not made until the animal shows signs of azotemia. It is important to remember that animals with ruptured bladders or unilateral ureteral trauma may urinate normal volumes, without evidence of hematuria. If the rupture is located dorsally or is small, leakage may only occur when the bladder becomes distended. Similarly the ability to retrieve fluid while performing bladder catheterization does not preclude a diagnosis of ruptured bladder.

Physical Examination Findings

Abdominal palpation should be performed to determine the size and shape of the bladder. The animal should be closely examined for abdominal swelling or fluid accumulation. Urine quantity and character (i.e., hematuria, dysuria) and bruising on the ventral abdomen or perineum should be monitored.

Diagnostic Imaging

Survey radiographs may show reduced size or absence of the urinary bladder, decreased visceral detail and increased size of the retroperitoneal space. If a ruptured bladder is suspected, a positive contrast cystogram should be performed; however, leakage of contrast medium into the peritoneal space during cystography does not necessarily mean that the animal needs exploratory surgery. If there is no clinical evidence of uroabdomen, conservative management of the patient may be appropriate. To perform cystography a balloon-tipped catheter is placed into the urinary bladder or to perform a cystourethrogram in a male dog, the catheter is placed into the distal urethra (just past the os penis), and the

balloon is inflated. While palpating the bladder for distention, approximately 2.2 ml/kg of diluted (1 part contrast medium to 2 parts sterile saline) aqueous organic iodide contrast medium is injected into the catheter. A radiograph is obtained while the last few milliliters of contrast are being injected. Fluoroscopy is advantageous to determine when the bladder is distended. It is critical to adequately distend the urinary bladder before determining that the study is normal as small lesions may not leak when the bladder wall is flaccid. Also, care should be taken not to completely occlude the neck of the urinary bladder with the bulb of the catheter as this may prevent leakage from a rupture in this area. Obtaining a radiograph while the contrast agent is being injected may show a "jet" lesion of contrast agent from the bladder. Free contrast agent in the abdominal cavity will coat and highlight abdominal organs. If a lesion is not identified in the bladder after adequate distension or urethra and the animal is well-hydrated, an excretory urogram can be performed. Contrast leakage into the retroperitoneal space (for proximal lesions) or abdomen (for distal lesions) occurs with ureteral rupture or laceration. If periureteral fibrosis has occurred, obstruction rather than leakage may be noted. Leakage of contrast from the renal capsule may be noted with renal parenchymal trauma. Parenchymal trauma of the right kidney should be suspected in dogs with uroabdomen and fractures of the thirteenth right rib.

Laboratory Findings

A CBC and serum biochemical profile with electrolytes should be performed. Hyperkalemia and azotemia may be noted. Analysis of abdominal fluid should be performed if urinary tract rupture is suspected. With uroabdomen, creatinine levels of the abdominal fluid will be greater than those in the blood (see above). Renal failure may be present if obstruction preceded the rupture. Bladder rupture secondary to urinary tract infection causes septic peritonitis.

MEDICAL MANAGEMENT

If the animal is not hyperkalemic or azotemic (e.g., uroabdomen is diagnosed within 12 to 18 hours after rupture), it should be rehydrated with 0.9% saline and immediate surgical repair should be considered. Occasionally, concurrent trauma (e.g., traumatic myocarditis, pulmonary contusions) will delay surgery. In such patients, abdominal drainage and/or urinary diversion (i.e., urethral catheter and/or tube cystostomy) may be necessary until the animal is stable. With delayed diagnosis, correction of electrolytes, hydration, and acid-base balance should be performed before surgery. Antibiotics may be administered based on culture results or upon bacterial morphology if a urinary tract infection is present, or prophylactically if abdominal drains are placed.

SURGICAL TREATMENT

Urethral trauma may be repaired by primary anastomosis (immediate or delayed) or the urethra may be allowed to heal over a urinary catheter if it is not completely transected. Ureteral rupture may be repaired by anastomosis or reimplantation into the bladder, depending on location of the damage. Bladder rupture generally occurs near the apex. Although small ruptures may heal if the bladder is kept decompressed, surgical exploration and repair are indicated in most patients. The entire abdomen should be explored to determine the reason for rupture and/or identify concurrent trauma. If bladder rupture is secondary to severe cystitis, tumor, or obstruction, the bladder may be extremely friable or large areas may be necrotic, making excision and primary closure of the rent difficult. In such

cases, prolonged urinary diversion may be beneficial. If cystitis or tumor is present, a biopsy of the bladder mucosa should be submitted for culture and histologic examination. In animals with rupture due to obstruction from calculi, the urethra should be carefully checked for calculi and its patency verified before repairing the bladder defect.

POSTOPERATIVE CARE AND ASSESSMENT

Intravenous fluids should be given until the animal is able to drink adequate fluids to maintain hydration. The patient should be observed closely after surgery for signs of urinary obstruction or peritonitis. If bladder atony is present, the bladder should be kept decompressed by intermittent urinary catheterization or by manual expression once the bladder incision has healed. Urinary tract infection is common with indwelling or repeated catheterization. An α-blocker (e.g., phenoxybenzamine) and/or a somatic muscle relaxant (e.g., diazepam) can help decrease urethral sphincter tone. Bethanechol is a cholinergic that increases detrusor contractility and may aid voiding. Manual expression of the bladder should be done with care following surgery (particularly in patients with friable bladders secondary to infection or obstruction) to avoid disrupting the suture line.

SPLENIC NEOPLASIA

The spleen is composed of a variety of tissues, and splenic neoplasia may arise from blood vessels, lymphoid tissues, smooth muscle, or the connective tissue that makes up the fibrous stroma. The most common tumor in dogs is HSA. Other malignant and benign neoplasms may also occur. The most frequently recognized nonneoplastic lesions of the spleen are nodular hyperplasia, hemangioma, and hematoma.

Canine splenic HSA is more common than all other types of malignant splenic tumors; it accounts for approximately half of all splenic malignancies identified. Because HSA arise from blood vessels, they may form in several different sites in the body (e.g., spleen, right atrium, subcutaneous tissues, and liver). As many as 25% of dogs with splenic HSA may have concurrent right atrial HSA. Splenic HSAs are aggressive tumors that frequently metastasize to the liver, omentum, mesentery, and brain. A majority of dogs with HSA have gross evidence of metastatic disease on initial presentation.

Splenic hematomas vary in size and are encapsulated, blood- and fibrin-filled masses that often are grossly indistinguishable from HSA. Histologically, the cavities are surrounded by congestion, fibrosis, and areas of necrosis. They may result from trauma, may occur spontaneously, or may develop secondary to other diseases (e.g., nodular hyperplasia). Hemangiomas and HSA may be difficult to distinguish histologically, but because the prognosis for these lesions is very different (see below), it is important that they be accurately differentiated. Splenic masses with evidence of malignant neoplastic endothelial cell proliferation can be easily identified as HSA. However, multiple sections of a malignant mass may be studied without obvious malignancy being seen. More important, proliferation of plump endothelial cells that resemble neoplastic endothelium but do not have evidence of mitotic activity may be misdiagnosed as HSA. Splenic hematoma and hemangioma account for 20% to 34% of splenic masses, whereas HSA accounts for 10% to 20% of all splenic samples submitted to veterinary pathology laboratories. However, this 10 to 20% underestimates the

true incidence of HSA in dogs with large splenic masses because many such masses are not submitted for pathologic examination, especially if apparent metastasis is seen at surgery. Hyperplastic nodules are an even more common finding at necropsy than HSA..

Diagnosis

Clinical Presentation

Signalment. Splenic tumors (including hematomas) usually occur in medium-to-large sized dogs. German shepherd dogs are at increased risk for hemangiosarcoma and hemangioma. Some authors have reported that spayed female dogs have increased risk, although others have reported this tumor to occur more commonly in male dogs. No obvious breed or sex predilection has been observed in dogs with nonangiogenic and nonlymphomatous splenic sarcomas.

History. Dogs with hemangiosarcoma may present for abdominal enlargement, anorexia, lethargy, depression, and/or vomiting or may have acute signs of weakness, depression, anorexia, and hypovolemic shock caused by splenic rupture and hemorrhage. Clinical signs with splenic hematoma are similar, except that rupture leading to collapse and anorexia are less common because large masses frequently become apparent before rupture occurs. The most common clinical signs of disease with other types of sarcomas are decreased appetite, abdominal distention (as a result of peritoneal effusion and/or tumor mass), polydipsia, vomiting, and/or lethargy. In contrast to dogs with hemangiosarcoma, splenic rupture and hemorrhage are uncommon in dogs with nonangiogenic and nonlymphomatous splenic tumors.

Diagnostic Imaging

Abdominal masses usually are detected radiographically in dogs with HSA and nonangiogenic and nonlymphomatous sarcomas; however, peritoneal fluid may make locating the lesion in the spleen difficult. Masses involving the tail of the spleen are typically identified in the cranial ventral abdomen on the lateral radiographic projection. Thoracic radiographs should be taken in animals with splenic masses to detect pulmonary or thoracic neoplasia.

Ultrasonography is more definitive in locating lesions in the spleen and detecting abdominal metastases than radiography; however, differentiation of hematomas from neoplastic lesions is unreliable. Finding internal septation and encapsulation or apparent metastasis may help differentiate hematomas from HSA. Magnetic resonance imaging may be a useful tool for differentiating benign and malignant splenic lesions in dogs.

Laboratory Findings

Neutrophilic leukocytosis may be present in some dogs. Mild or moderate anemia associated with chronic disease or hemoperitoneum also is common. Other hematologic abnormalities caused by HSA may include numerous nucleated red blood cells (inappropriate numbers for the degree of anemia), Howell-Jolly bodies, poikilocytosis, acanthocytosis, schistocytosis, and/or thrombocytopenia. Hemostatic disorders, particularly thrombocytopenia caused by DIC, are common in dogs with splenic tumors. Abdominal effusion generally is serosanguineous or hemorrhagic. Cytologic analysis of abdominal fluid rarely reveals tumor cells.

Physical Examination Findings

The physical examination findings include lethargy, weakness, abdominal distention, and possibly splenomegaly or a splenic mass. If a splenic mass is palpated, it should be palpated to prevent iatrogenic rupture. If abdominal effusion is present, it is not always possible to palpate the enlarged spleen. If rupture occurs, the animal may have signs of hypovolemic shock (tachycardia, pale mucous membranes, and weak peripheral pulses). Hemoabdomen is more commonly associated with HSA than hemangioma or hematoma. Some dogs with HSA will have cutaneous metastasis (dark reddish-purple mass). Sometimes a murmur is heard in patients with HSA in the right atrium; or, pericardial effusion due to such a HSA may cause muffled heart sounds or a jugular pulse.

Medical management

Surgical resection is the mainstay of therapy in dogs with splenic hemangiosarcoma, and there are few reports of postoperative chemotherapy or immunotherapy significantly prolonging survival. Readers are referred to an oncology text for discussion of protocols and treatment regimens used in dogs with hemangiosarcoma.

Surgical treatment

Splenectomy is the treatment of choice for animals with splenic hematoma and hemangioma. It is also the treatment of choice for animals with hemangiosarcoma, in whom evidence of extensive metastasis or other organ failure does not preclude the short-term benefits of removing the enlarged and/or ruptured spleen. The median survival time of dogs with splenic hemangiosarcoma is between 10 and 23 weeks after splenectomy, depending on the stage of the disease. With nonangiogenic and nonlymphomatous sarcomas, the median survival times after splenectomy were 2.5 months for all dogs that survived the early postoperative period, and 9 months for the subset of dogs that did not have evidence of metastasis at surgery. Splenectomy may not be warranted in dogs with concurrent right atrial tumors. Thus careful preoperative examination of patients is warranted. Dogs with splenic lymphoma and clinical signs associated with massive splenomegaly, splenic rupture, and hemoperitoneum may also benefit from splenectomy.

Preoperative Management

Anemic animals may require blood transfusions before surgery and should be preoxygenated. An ECG should be performed to determine if ventricular arrhythmias requiring preoperative or intraoperative therapy are present. Hydration, electrolyte, and acid-base abnormalities should be corrected before anesthesia. Perioperative antibiotics may be indicated in some animals undergoing splenectomy.

Postoperative care and assessment

Animals with splenic hemangiosarcoma should be closely observed for disseminated intravascular coagulation following splenectomy. Fluid therapy should be continued until the animal is able to maintain its own hydration. The hematocrit should be monitored and blood transfusions provided if the PCV is less than 20%. Septic complications following splenectomy appear to be rare, and antibiotic therapy

Total splenectomy is most commonly performed in animals with splenic neoplasia, torsion (stomach or spleen), or severe trauma. Splenectomy has previously been advocated for

immune-mediated hematologic disorders refractory to medical therapy (e.g., thrombocytopenia or hemolytic anemia); however, proper use of immunosuppressive drugs and corticosteroids has decreased the need for splenectomy. However, splenectomy may be used if drug therapy is unsuccessful or unacceptable. Although life-threatening sepsis has been associated with total splenectomy in humans, this has not been recognized in dogs.

GASTRONINTESTINAL DISEASE SURGERY

Intestinal Resection and Anastomosis

Intestinal resection and anastomosis are recommended for removing ischemic, necrotic, neoplastic, or fungal-infected segments of intestine. Irreducible intussusceptions are also managed by resection and anastomosis. End-to-end anastomoses are recommended.

Sutured anastomoses. *Make an abdominal incision long enough to allow exploration of the abdomen. Thoroughly explore the abdomen and collect any non-intestinal specimens, then exteriorize and isolate the diseased intestine from the abdomen by packing with towels or laparotomy sponges. Assess intestinal viability and determine the amount of intestine needing resection. Occlude (double ligate, staple or heat seal) and transect the arcadial mesenteric vessels from the cranial mesenteric artery that supplies this segment of intestine. Occlude (double ligate, staple or heat heal) the terminal arcade vessels and vasa recta vessels within the mesenteric fat at the points of proposed intestinal transection. Gently milk chyme (intestinal contents) from the lumen of the identified intestinal segment. Use fingers or intestinal forceps to occlude the lumen at both ends of the segment to minimize spillage of chime (Fig. 1). Place forceps across each end of the diseased bowel segment (these forceps may be either crushing or noncrushing because this segment of the intestine will be excised). Transect the intestine with either a scalpel blade or Metzenbaum scissors along the outside of the forceps. Make the incision either perpendicular or oblique to the long axis. Use a perpendicular incision (75- to 90 degree angle) at each end if the luminal diameters are the same. When the luminal sizes of the intestinal ends are expected to be unequal, use a perpendicular incision across the intestine with the larger luminal diameter and an oblique incision (45- to 60- degree angle) across the intestine with the smaller luminal diameter to help correct size disparity. Make the oblique incision such that the antimesenteric border is shorter than the mesenteric border. If further correction for size disparity is needed, space sutures around the larger lumen slightly farther apart than around the smaller lumen or remove a wedge from the antimesenteric border of the smaller segment. Suction the intestinal ends and remove any debris clinging to the cut edges with a moistened gauze sponge. Trim everting mucosa with Metzenbaum scissors just before beginning the end-to-end anastomosis.*

Use 3-0 or 4-0 monofilament, absorbable suture (polydioxanone, polyglyconate or poliglecaprone 25) with a swaged-on taper or tapercut point needle. In peritonitis cases monofilament, nonabsorbable suture (3-0 or 4-0 polypropylene, polybutester, or nylon) is sometimes used. Place simple interrupted sutures through all layers of the intestinal wall. Angle the needle so that the serosa is engaged slightly farther from the edge than the mucosa (Fig. 1). This helps reposition everting mucosa within the lumen. Tie each suture

carefully to gently appose the edges of the intestine with the knots positioned extraluminally.

Tying sutures roughly or with too much tension causes the suture to cut through the serosa, muscularis and mucosa, creating a crushing suture. Some surgeons prefer this suture, but most prefer a simple interrupted or simple continuous pattern. Pulling continuous sutures too tight has a purse-string effect, and significant stenosis may occur. A continuous pattern around the intestine may limit dilation at the anastomotic site and cause a partial obstruction. Therefore a divided, modified simple continuous suture pattern is used to avoid these effects. Two stay sutures are placed at the mesenteric and antimesenteric borders, then one simple continuous suture is place between the sutures on each side. Experimentally, skin staplers have been used successfully in lieu of interrupted sutures.

Appose the intestinal ends by first placing a simple interrupted suture at the mesenteric border and then placing a second suture at the antimesenteric border approximately 180 degrees from the first (this divides the suture line into equal halves and allows determination of whether the ends are of approximately equal diameter).

The mesenteric suture is the most difficult suture to place in the anastomosis because of mesenteric fat. It is also the most common site of leakage. *If the ends are of equal diameter, space additional sutures between the first two sutures approximately 2 mm from the edge and 2 to 3 mm apart. If minor disparity still exists between lumen sizes, space the sutures around the larger lumen slightly farther apart than the sutures in the intestine with the smaller lumen. To correct luminal disparity that cannot be accommodated by the angle of the incisions or by suture spacing, resect a small wedge (1 to 2 cm long and 1 to 3 mm wide) from the antimesenteric border of the intestine with the smaller lumen. This enlarges the perimeter of the stoma, giving it an oval shape. Do not suture together the edges of the intestine with the larger lumen in an attempt to reduce luminal size to that of the smaller intestine.* Narrowing the larger lumen is not recommended because there is greater tendency for stricture at the anastomotic site when the dilated intestine contracts to a normal size. *After suture placement, inspect the*

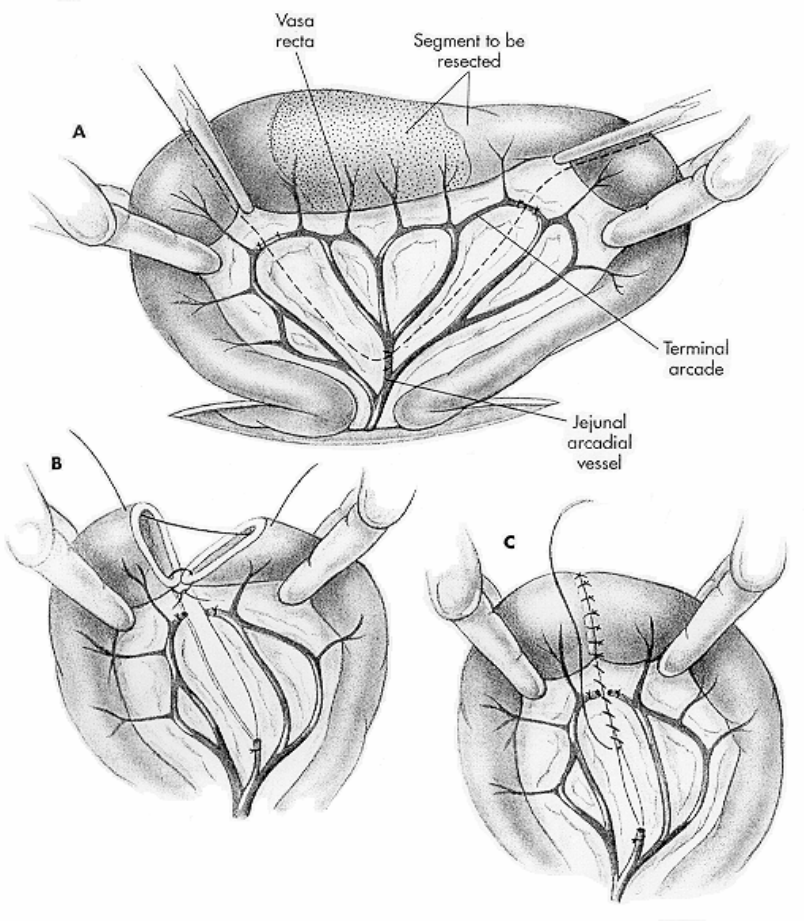

Figure 1 From: Fossum, TW: *Small Animal Surgery*, Mosby Publishing Co., St. Louis, Mo, 2002.

anastomosis and check for leakage. While maintaining luminal occlusion adjacent to the anastomotic site, moderately distend the lumen with sterile saline, apply gentle digital

pressure, and observe for leakage between sutures or through needle holes. This is a subjective test, because all anastomoses can be made to leak if enough pressure is applied.

Place additional sutures if leakage occurs between sutures. Close the mesenteric defect in a simple continuous or interrupted pattern using 4-0 monofilament absorbable suture, taking care not to penetrate or traumatize arcadial vessels near the defect. Lavage the isolated intestine and the entire abdomen if abdominal contamination has occurred. Wrap the anastomotic site with omentum before closing the abdomen or use a serosal patch if intestinal integrity is questionable and leakage is likely.

SEROSAL PATCHING

Serosal patching consists of putting the antimesenteric border of a loop of small intestine over a suture line or organ defect and securing it with sutures. Serosal patching provides support, a fibrin seal, increased resistance to leakage, and blood supply to the damaged area plus it may prevent intussusception. Patches are commonly used after intestinal surgery when closure integrity is questioned or when dehiscence is repaired. Patches that span visceral defects are covered with mucosal epithelium within 8 weeks. Most commonly, jejunum adjacent to the defect or area of questionable viability is used for the serosal patch. Other sources could include the stomach, other intestinal segments, or the urinary bladder.

Use one or more loops of intestine to form the patch. Use gentle loops to avoid stretching, twisting, or kinking the intestine and mesenteric vessels. If using more than one loop of intestine, suture these loops together before securing the patch to the damaged area. All sutures used to create or secure the patch engage the submucosa, muscularis, and serosa; they should not penetrate the intestinal lumen. Place interrupted or continuous sutures in healthy tissue to secure the patch and isolate the damaged area. Alternatively, to patch over an anastomosis, use a piece of normal intestine and loop it perpendicular to the area to be patched. Be sure that the loop is gentle so as not to cause obstruction. Using a simple continuous suture pattern, suture between the looped piece of normal intestine starting at the mesenteric border and continuing up to the antimesenteric border. Then suture across the anastomosis and back down to the mesenteric border on the same. Repeat the process on the opposite side of the anastomosis. Be careful not to compromise the vasculature at the mesenteric border with your sutures

BOWEL PLICATION

Enteroenteropexy, or bowel plication, is performed to prevent recurrence of intussusception. Suturing together adjacent loops of intestine forms serosa-to-serosa adhesions. The small intestine from the duodenocolic ligament to the ileocolic junction is sutured to decrease the potential for intestinal strangulation. The bends in the intestine are gentle to prevent obstruction and plication sutures are placed at intervals that will prevent entrapment and strangulation of other intestinal segments.

Place small intestinal loops side by side to form a series of gentle loops from the distal duodenum to the distal ileum. Secure the loops by placing sutures that engage the submucosa, muscularis, and serosa 6 to 10 cm apart. Use 3-0 or 4-0 monofilament, absorbable or nonabsorbable sutures with a swaged-on taper point needle. Avoid positioning the intestinal loops at acute angles, or intestinal obstruction may occur. Entering the lumen with pexy sutures may increase the risk of leakage and abdominal contamination.

COLOPEXY

Colopexy is done to prevent caudal movement of the colon and rectum and is especially useful in animals with recurrent rectal prolapse. The procedure creates permanent adhesions between the serosal surfaces of the colon and abdominal wall. Incisional and non-incisional techniques have been described and both are equally effective. A potential complication (but rare if the technique is performed properly) is infection as a result of suture penetration into the colonic lumen.

Expose and explore the abdomen. Locate and isolate the descending colon from the remainder of the abdomen. Pull the descending colon cranially to reduce the prolapse. Verify prolapse reduction by having a non-sterile assistant inspect the anus visually and perform a rectal examination. Make a 3 to 5 cm longitudinal incision along the antimesenteric border of the distal descending colon through only the serosal and muscularis layers. Create a similar incision on the left abdominal wall several centimeters lateral ($\geq$ 2.5 cm) to the linea alba through the peritoneum and underlying muscle. Appose each edge of the colonic and abdominal wall incisions with 2 simple continuous or simple interrupted rows of sutures using 2-0 or 3-0 monofilament absorbable (e.g., polydioxanone or polyglyconate) or nonabsorbable (nylon, polypropylene) suture material. Engage the submucosa as each suture is placed. Lavage the surgical site and surround it with omentum prior to abdominal closure. Alternatively, scarify an 8 to 10 cm antimesenteric segment of the descending colon by scraping the serosa with a scalpel blade or rubbing it with a gauze sponge. On the left abdominal wall opposite the prepared colon, scarify the peritoneum in the same manner. Preplace, then tie, 6 to 8 horizontal mattress sutures between the two scarified surfaces. Roll the colon toward the midline and place a second row of 6 to 8 sutures. Use 2-0 to 3-0 monofilament absorbable or nonabsorbable sutures which engage the submucosa, but do not penetrate the colonic mucosa. Tie the sutures apposing the scarified surfaces (see Figure 2).

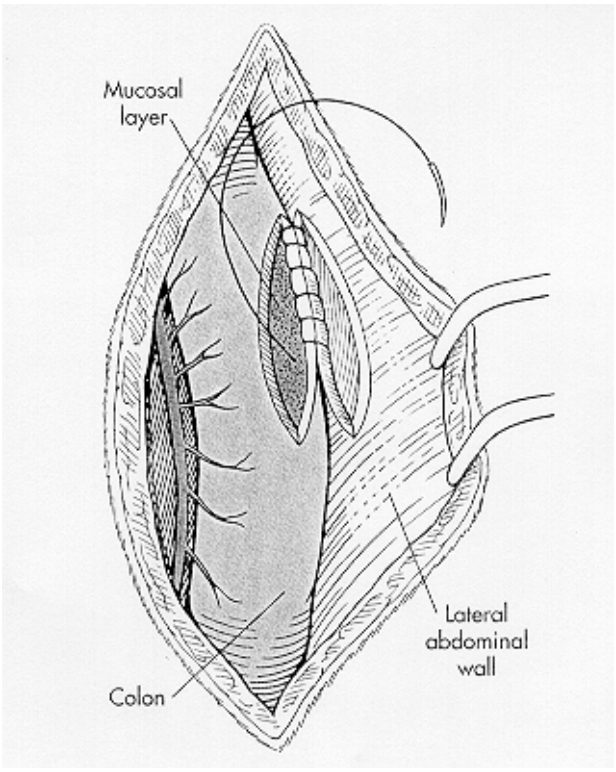

Figure 2 From: Fossum, TW: *Small Animal Surgery*, Mosby Publishing Co., St. Louis, Mo, 2002.