Megacolon in the cat
Robert W. Bertoy, DVM, MS
Veterinary Surgical Service, El Dorado Hills, CA 95762, USA

Megacolon has been defined as a condition of persistent increased bowel diameter always associated with chronic constipation [1]; however; megacolon is not a specific disease entity. It is merely a subjective evaluation of the diameter of the colon, usually based on radiographic assessment.

Megacolon is the end result of an obstructive or pseudo-obstructive condition that causes constipation, which is progressive, often resulting in incapacitating and debilitating obstipation. Megacolon may be thought of as the most advanced stage in the spectrum of chronic constipation.

Because of the severity of the clinical signs associated with megacolon, surgical therapy may be indicated. The procedure used depends on the cause. In some cases, surgical intervention before the development of severe megacolon may alleviate the clinical problem before physical debilitation develops.

Classification

There are many reported causes of megacolon in human beings; fewer have been documented in the cat. As our awareness of this disorder and our diagnostic abilities increase, more specific causes may be uncovered. This is important, because a successful treatment plan requires accurate diagnosis of the primary problem. Megacolon has been categorized as congenital or acquired, primary or secondary, intrinsic or extrinsic, functional or mechanical, and dilated or hypertrophic. The terms dilated and hypertrophic recently appeared in the literature and are confusing [2]; the colonic musculature is histologically hypertrophied in idiopathic megacolon, yet, grossly, the colon is severely dilated [3,4]. The surgeon should conceptualize the causes of megacolon as either colonic inertia or outlet obstruction (Table 1), because these categories may dictate the surgical treatment. In the cat, idiopathic megacolon is the most common diagnosis, occurring in approximately 60% to 70% of the cases reported in the literature (this percentage may be much higher in
Table 1
Possible causes of megacolon in the cat

I. Colonic inertia
   A. Idiopathic megacolon
   B. Secondary to neurologic disease
      1. Trauma to colonic innervation
      2. Associated with congenital abnormalities of the caudal spine
      3. Chagas disease
      4. Dysautonomia
   C. Secondary to a variety of medical conditions
   D. Secondary to prolonged colonic distention (eg, outlet obstruction)

II. Outlet obstruction
   A. Pelvic fracture malunion
   B. Colonic, rectal, or anal stricture or tumor
   C. Intrapelvic extraluminal mass
   D. Foreign body or improper diet
   E. Anal or rectal atresia

clinical practice). Neurologic disorders constitute approximately 11% of the cases, and those under the heading of outlet obstruction occur in 24% of the cases, with most obstruction cases (96%) occurring as the result of pelvic fracture malunion [2].

Anatomy

The colon begins at the ileocolic sphincter. The cecum is a diverticulum of the proximal colon in the dog and cat. These species have a cecocolic orifice that lies just aboral to the ileocolic junction. The colon is divided into ascending, transverse, and descending portions. The ascending colon begins at the ileocolic sphincter and runs cranially, ending at the right colic flexure. The colon continues at this point as the transverse colon, which runs from right to left to its termination at the left colic flexure. The descending colon, the longest portion, begins at this point and ends near the level of the pelvic inlet, where the rectum begins [5]. The colorectal junction is difficult to identify morphologically; landmarks that have been used include the pubic brim, pelvic inlet, and seventh lumbar vertebrae. It may be more accurate to define it as the point where the cranial rectal artery penetrates the seromuscular layer of the large intestine, which it does just cranial to the pubic brim. This landmark is consistent, readily identifiable during celiotomy, and related to the vascular supply and not only to a nearby osseous structure. This should eliminate confusion between total and subtotal colectomy in which differentiation is often the result of one’s definition of the colorectal junction. This definition is used in this article.

The proximal portion of the colon receives its vascular supply from anastomosing arcades via branches of the ileocolic artery, a branch of the cranial mesenteric artery. The colic and right colic branches of the ileocolic artery
supply the ascending colon, and the middle colic branch supplies the transverse colon. The descending colon is supplied primarily by the left colic artery, the cranial branch of the caudal mesenteric artery. The left colic artery forms an anastomosis with the middle colic artery and supplies the descending colon by numerous vasa recti rather than by anastomosing arcades as in the small intestine and proximal portion of the colon. The cranial rectal artery is the caudal branch of the caudal mesenteric artery and primarily supplies the cranial rectum. It also supplies a short segment of the terminal colon via several vasa recti. The venous drainage mirrors that of the arterial supply, except that the cranial rectal vein continues cranially as the left colic vein, joining the ileocolic vein to form the caudal mesenteric vein in the area of the left colic flexure. The caudal mesenteric vein is short and empties directly into the portal vein [5].

Innervation of the colon is supplied by an intrinsic component and an extrinsic component. The intrinsic portion consists of groups of neurons that form the submucosal (Meissner’s) plexus and the myenteric (Auerbach’s) plexus located between the outer longitudinal and inner circular smooth muscle layers. The sensory component of the plexuses receives information on the composition of the intestinal contents and the state of the muscular wall from nerve endings near the epithelial cell layer and smooth muscle layers. Effector nerve endings innervate secretory cells and smooth muscle motor units. The intrinsic innervation is responsible for intestinal contractions, which occur even in the total absence of extrinsic innervation. The extrinsic innervation is provided by preganglionic parasympathetic (cholinergic) fibers that are responsible for stimulating smooth muscle cell activity and by postganglionic sympathetic (adrenergic) fibers that suppress smooth muscle activity [6].

**Physiology**

**Motility**

Muscle contractions facilitate mixing of ingesta and propulsion of fecal matter toward the anus. Slow-wave contractions, which originate in the middle of the colon, spread orally and aborally and account for antiperistaltic and peristaltic movement of ingesta. Segmentation contractions ensure adequate mixing of ingesta. Mass contractile propulsive movements that evacuate the colon and propel feces into the rectum and anal canal originate in the distal segment of the colon [6].

Movements in the proximal portion of the colon can occur independent of the extrinsic nerve supply. Distention of the colon usually initiates these intrinsic contractions. Entry of food into the stomach or duodenum is responsible for a reflex contraction of the colon, the gastrocolic and duodenocolic reflex [6]. The effects of extrinsic parasympathetic and sympathetic stimulation on the colon were discussed previously.
The distal colon, rectum, and anus are more dependent on the extrinsic nerve supply for contractile and reflex activity. Disruption of this innervation can result in decreased motility or incontinence. Distention of the distal colon and proximal rectum initiates general visceral afferent impulses that pass to the sacral spinal cord. General visceral efferent fibers in the sacral spinal cord complete a reflex arc that results in evacuation of the large bowel. Enhanced coordinated reflex contraction of the smooth muscle of the colon and rectum, reflex relaxation of the internal anal sphincter, reflex relaxation of the paraspinal muscles, voluntary relaxation of the external anal sphincter, and an increase in intra-abdominal pressure during the Valsalva maneuver result in defecation. Disruption of any of the steps of this process can result in constipation. Voluntary contraction of the external anal sphincter muscle can interrupt the defecation reflex, causing fecal material to move back into the distal colon and proximal rectum (accommodation) to be stored until the next defecation reflex is initiated [6]. An abnormally large rectal capacity or increased rectal compliance may result in retention of stool at this location. If this occurs, the rectoinhibitory and contraction reflexes may not be initiated, resulting in difficulty with evacuation as with megarectum or perineal hernia. Normal continence requires not only an intact defecatory reflex and sphincter mechanism but an adequate reservoir for storage of stool during accommodation. If stool cannot be adequately stored after conscious contraction of the external anal sphincter, incontinence results [6].

Absorption/secretion

Most large intestinal fluid absorption occurs in the proximal half of the colon. The mucosa of the colon has a high capacity for active sodium absorption. Chloride is absorbed passively by an electrochemical gradient created by this active sodium absorption. The colonic mucosa also actively absorbs chloride ions while simultaneously secreting bicarbonate ions in the same transport process. The absorption of sodium and chloride creates a large osmotic gradient, which is responsible for water absorption. Although only 20% of gastrointestinal water absorption occurs in the colon, this amount is critical for normal homeostasis. The bicarbonate ions help to neutralize the acidic byproducts of bacterial metabolism [6,7].

Potassium is lost in the feces by the addition of potassium-rich mucus and desquamated epithelial cells and by active transport into the colonic lumen by mucosal cells [8]. Mucus, the primary secretory product of the colon, lubricates and facilitates passage of fecal material and protects the mucosa from mechanical and chemical injury [6].

Synthesis of numerous nutrients occurs secondary to the metabolic processes of colonic bacteria. This is of little significance in the dog and cat, except for vitamin K synthesis, but plays a much greater role in herbivorous species [7].
Pathophysiology

General

Normal dogs and cats can retain feces in the colon for several days without suffering any demonstrable adverse effects. With prolonged retention of feces, the efficient absorptive processes of the colon further dehydrate and solidify the feces to the point that fecal concretions are produced. These are difficult and painful to eliminate and can become so large that passage through the pelvic canal is physically impossible [8]. This intractable constipation, or obstipation, can produce severe distention of the colon if not treated. If this distention is prolonged, irreversible changes can occur that affect normal colonic motility, and secondary colonic inertia can result. The degree of distention and duration of time needed to produce these irreversible changes are unknown; however, it seems that both are important factors in the development of secondary colonic inertia. Based on empiric observations, some have suggested that 6 months is a dependable period [9,10]; however, there are no hard data available to back up these assertions.

Severely constipated animals may exhibit central nervous system depression, anorexia, and weakness, which are signs that have been attributed to the absorption of unidentified toxins produced by bacterial metabolism in the stagnant colonic lumen. Vomiting can occur secondary to prolonged intestinal obstruction, the effect of the absorbed toxins of colonic bacterial metabolism on the chemoreceptor trigger zone, or vagal afferent stimulation of the vomiting center triggered by bowel distention. Paradoxically, diarrhea may be present. Liquid feces may pass around the fecal concretions. Additionally, these concretions can irritate the colonic mucosa and cause secretion of mucus and fluid as well as exudation of blood, resulting in watery and sometimes blood-tinged diarrhea. Severely constipated patients may present with many complaints, including tenesmus, depression, lethargy, anorexia, weight loss, and vomiting as well as occasional watery, mucoid, or bloody diarrhea solely related to severe fecal impaction [8,11].

Idiopathic megacolon

Idiopathic megacolon, a condition of unknown cause and the primary condition recognized in association with progressive intractable constipation in the cat, is seen almost exclusively in middle-aged and older cats. To date, there is no recognized specific histologic abnormality as there is for Hirschsprung’s disease in human beings. Despite this, it seems that an abnormality associated with either the intrinsic or extrinsic innervation to the lower large intestine, the myoneural junction, or the smooth muscle itself is the likely cause for the progressive colonic inertia. In human beings, idiopathic constipation of colonic origin seems to be caused by abnormal colonic motility, resulting in slow colonic transport [11–13]. Two causes of this altered motility are atonicity of the colonic musculature and excessive muscle activity or
spasticity of the distal large intestine or associated structures, resulting in failure of the defecation reflex [11,12,14]. Previous studies have shown that smooth muscle from megacolonic cats develops less isometric stress than normal feline colonic smooth muscle. It is entirely possible, however, that colonic distention from chronic obstipation may disrupt smooth muscle myofilaments, resulting in these functional abnormalities (ie, what is cause and what is effect is not known) [15]. Other studies in human beings have shown altered release of the inhibitory neurotransmitter nitric oxide in megacolonic smooth muscle. It is postulated that the loss of this inhibitory nerve input induces constipation by increasing nonpropagating contractions in the rectum (similar to that seen in opiate-induced constipation in human beings) with slowed transit in the right colon [16]. Investigation of this problem is necessary to elucidate further the mechanism of this disorder.

Megacolon secondary to neurologic or medical disease

Although trauma to the sacrocaudal spinal cord reportedly can cause secondary megacolon, there are few reports of this in the veterinary literature. Disruption of the extrinsic nerve supply to the distal large intestine can interrupt distal colonic motility and interfere with the complicated interaction between the colon, rectum, and anus during the defecation reflex, resulting in chronic constipation and megacolon. Theoretically, removal of the nonfunctional dilated colon would be therapeutic only as long as the rectoanal reflexes remain intact.

Experimental breeding studies in Manx cats produced several cats with congenital megacolon. These cats showed partial or complete absence of the sacral and caudal spinal cord in conjunction with sacral agenesis or dysgenesis. This results in interference with normal defecation and obstipation that shows up early in life. Most also had problems with urinary and fecal incontinence. Treatment was not attempted in any of these cats because of the presence of multiple defects [17]. Some Manx cats can exhibit clinical signs of megacolon later in life. It is unclear whether a milder form of neurologic dysfunction is responsible for this “late-onset” megacolon or if these cats are neurologically normal and suffer from idiopathic megacolon. I have performed surgery (colectomy) in several of these cats with excellent results.

Aganglionic megacolon (Hirschsprung’s disease), the most common cause of megacolon in human infants, has not been documented in cats or dogs. The aganglionic segment lacks the submucosal and myenteric plexuses and typically is located in the rectum and sigmoid colon. This segment remains tonically contracted, effectively producing an obstruction [11,12]. Classic surgical therapy involves removal of the aganglionic segment and any nonfunctional dilated colon.

Several other medical and neurologic diseases have been reported to cause megacolon in human beings [1], but they have not been proven to exist in the cat.
Outlet obstruction

There are many reported causes of megacolon secondary to outlet obstruction (see Table 1). Pelvic fracture malunion is the most common. Colonic or rectoanal tumors and strictures and intrapelvic extraluminal tumors or masses (eg, enlarged prostate or perineal hernia) are reported causes of megacolon in animals. Foreign bodies, improper diet, and anal or rectal atresia have been reported only in isolated cases.

As stated previously, prolonged retention of feces in the colon as a result of any cause can produce such severe distention that irreversible changes occur, resulting in significantly reduced colonic smooth muscle function. Reduced motility or severe colonic inertia can result, further complicating treatment.

There are human patients who present because of chronic constipation with a condition of paradoxic puborectalis contraction. During defecation, the puborectalis (and other paraspinal muscles) normally relax. Paradoxic contraction of any of these muscles during defecation results in an outlet obstruction causing constipation; if severe, this can result in megarectum or megacolon. Most patients are first treated conservatively, with surgical treatment (partial division or resection of the puborectalis muscle) reserved for those with persistent problems [18]. It is unknown if this condition occurs in the cat, but the observation of a dilated rectum in some cats with idiopathic megacolon suggests that a distal obstructive or pseudo-obstructive condition is possible.

Diagnosis

Because most owners are unaware of their animal’s bowel habits, it is difficult to judge what is normal for an individual animal. Consequently, diagnosis and treatment are often initiated only when signs are severe and the disease is long standing. Ruling out all other causes of intractable constipation results in a diagnosis of idiopathic megacolon. A complete physical examination should be performed on all cats suspected of having megacolon. A digital rectal examination should be performed to assess any evidence of distal colonic or rectal stricture or tumor. The presence of perineal hernia should also be noted, although this is likely secondary to the chronic tenesmus associated with chronic obstipation. A neurologic examination should be performed with specific emphasis on the function of the sacrocaudal spinal cord. Although laboratory data are usually normal in cases of megacolon, these tests (complete blood cell count, serum chemistry, and urinalysis) should be performed to rule out other causes of constipation and to help identify any complicating conditions before pursuing other more invasive diagnostic procedures. Abdominal radiography should be performed and demonstrates a distended colon impacted with fecal material in all cases of megacolon regardless of the primary cause. Radiography is used primarily to rule out obstructive diseases, such as pelvic fracture malunions, sacrocaudal spinal trauma or deformities, and intramural or mural colonic or
rectoanal obstructive lesions. Contrast radiography or endoscopy may also be necessary to rule out an obstructive disease. Only when all known causes of megacolon have been eliminated as a possibility can a diagnosis of idiopathic megacolon be made.

**Treatment**

**Medical**

Initial management involves correction of dehydration and acid-base and electrolyte abnormalities if obstipation has been prolonged. This is followed by evacuation of the colon using stool softeners and enemas. Manual removal of softened fecaliths often is necessary and should be performed with caution to minimize damage to the mucosal barrier and to prevent the absorption of luminal bacteria and toxins into the systemic circulation. Prophylactic antibiotics should be administered before manual removal, because mucosal trauma is inevitable. Long-term management involves high-fiber diets, stool softeners, bulk laxatives, and periodic enemas in the hope that constipation can be controlled. Long-term medical management has been reviewed elsewhere and is not discussed in this article. If recurrent obstipation or debilitation occurs, surgery should be considered as a therapeutic option.

**Surgical**

**General**

Surgical treatment involves identifying the underlying cause and surgically removing the obstruction when necessary. Partial pelvectomy or pelvic reconstructive procedures can be used for pelvic fracture malunion [10]. These procedures have been described; however, the criteria for selection of a specific orthopedic procedure are not well defined [19]. Mass excision, segmental proctectomy or colectomy, or bougienage can be performed to excise obstructive mass lesions or strictures. Foreign bodies should be removed and diet changes made when indicated. Anal or rectal atresia can be treated with pull-through procedures. The major dilemma in the treatment of outlet obstruction is whether the dilated colon can function properly after removal of the obstruction. In cases of pelvic fracture malunion, it has been recommended that removal of the obstruction (pelvic resection) be performed in cats that have had clinical signs of constipation of less than 6 months' duration, with colectomy being reserved for those cats with a longer history of clinical signs [9,10]. It seems reasonable to extend these guidelines to all forms of outlet obstruction causing megacolon. Again, these guidelines have been made empirically based on a small number of cases.

**Colectomy for megacolon**

This technique is the only surgical procedure presented in depth in this article, because most cats with megacolon are candidates for this procedure.
In cats with concurrent perineal hernia, colectomy is performed before perineal herniorrhaphy is considered. Colectomy alone often resolves the clinical signs [2]. Preoperative preparation using enemas to evacuate the colon is difficult to perform and not recommended. It is much easier to prevent hard feces from contaminating the peritoneal cavity than the liquid feces left in the colon after the administration of enemas. Antimicrobial prophylaxis is warranted, and cefazolin at the rate of 20 mg/kg of body weight given at the time of anesthetic induction and continued for 24 hours is one rational choice. The goal of surgery is to remove all the dilated colon, which means that the resection sites are the most distal aspect of the ileum (total colectomy) or 2 to 4 cm distal to the cecum (subtotal colectomy) and at a point as far distally as possible that permits suturing of the anastomosis, generally 2 cm cranial to the pubic brim. I find that a total colectomy is easier to perform and allows a more tension-free anastomosis than a subtotal colectomy, and it is my procedure of choice.

Any hard stool in the rectum and anal canal is removed digitally before surgery so that it does not cause excessive straining or difficult defecation after colectomy. The cat is placed in dorsal recumbency, and a ventral midline celiotomy is performed from a few centimeters cranial to the umbilicus to the pubic brim. The only abnormality in cats with idiopathic megacolon is a colon greatly distended with fecaliths. In cats with pelvic fracture malunion, there may be scar tissue at the pelvic inlet, which may complicate the distal colonic resection. The distal ileum, cecum, and colon are exteriorized and packed off with moistened laparotomy sponges. Solid feces can be displaced from the resection sites by squeezing the wall of the colon. Hard fecaliths may have to be broken down manually so as to facilitate this maneuver. Any impacted feces in the rectum not removed digitally before surgery should be moved proximal to the resection site at this time. The terminal arcade of the jejunal vessels supplying the most distal portion of the ileum is ligated if a total colectomy is performed. If the decision has been made to perform a subtotal colectomy, this step is eliminated and a site 2 to 4 cm distal to the cecum that easily allows vessel ligation is chosen as the proximal resection site. The ileocolic (total colectomy), right colic, and middle colic vessels (total colectomy and subtotal colectomy) that supply the ascending (total colectomy) and transverse colon (total and subtotal colectomy) are then doubly ligated and transected (Fig. 1). Care must be taken when ligating the colic and ileocolic vessels so that damage is not done to the cranial mesenteric artery. If this occurs, the blood supply to the small intestine can be interrupted with catastrophic results. The caudal mesenteric vessels are then doubly ligated and transected (Fig. 2), and the cranial rectal artery is ligated 2 cm cranial to the pubic brim approximately 1 cm caudal to the point where the artery penetrates the seromuscular layer of the rectum (Fig. 3). Atraumatic intestinal forceps are placed proximal and distal to the planned resection sites. The distal ileum, cecum, and colon (total colectomy) (Fig. 4) or
the transverse and descending colon (subtotal colectomy) are then resected. The bowel ends are anastomosed using a single layer of simple interrupted 3-0 or 4-0 polydioxanone suture. With a total colectomy, there is great difference in bowel diameters, and a simple end-to-end anastomosis is impossible. The ileal diameter is increased by making a diagonal cut, and the rectal diameter is decreased by oversewing the antimesenteric side as needed to match the

Fig. 1. Ligation site of the right colic vessels (isolated over the hemostat). The blue suture to the right of the hemostat is the ligation site for the terminal arcade of the jejunal vessels supplying the distal ileum (when performing a total colectomy).

Fig. 2. Ligation site of the middle colic vessels (isolated over the hemostat).
ileal and rectal diameters (Figs. 5 and 6). The ileal mesentery is sutured with 3-0 or 4-0 absorbable suture in a simple continuous pattern. The abdominal cavity is then lavaged with warm sterile saline, and the abdomen is closed routinely. To speed closure, I use a simple continuous pattern of 3-0 polydioxanone suture in the linea alba.

Although the exact pathophysiologic mechanism responsible for idiopathic megacolon is unknown, surgical therapy has been extremely rewarding
in cases refractory to medical management. Total colectomy with ileo-
rectal anastomosis or subtotal colectomy with preservation of the cecum and
ascending colon with colorectal anastomosis has been used successfully
without significant alteration in either reservoir or sphincter continence
[20,21]. Although stool storage and water absorption are altered after total
colecotomy, the distal small intestine apparently adapts by increasing its stool
capacity (by an increase in diameter) and its ability to absorb water [20]. In a
small study of four colectomized cats, there seemed to be no alteration in enteric function or general health [22]. When reviewing the available veterinary literature, it appears that greater than 90% of cats have an uncomplicated recovery and function “normally” and without long-term complications. There seems to be an overall complication rate of less than 5%. This includes stricture at the anastomotic site (1 of 150 cases), death caused by peritonitis from leakage at the anastomosis (1 of 150 cases), death from unknown cause (1 of 150 cases), persistent diarrhea (2 of 150 cases), and recurrent constipation (3 of 38 cases) [19].

Most cats begin eating within 24 to 48 hours after colectomy. Small-volume watery diarrhea develops for approximately 5 days. Most cats have semisolid nonformed stools at 1 week and soft formed stools by 6 to 12 weeks after colectomy [13,20,21]. Cats can be expected to have a softer than normal stool and an increase in stool frequency (2–6 times per day). This mirrors what is seen in human patients [1,11–13]. The occasional cat may have an episode of constipation, but this seems to be relatively minor and is easily managed by digital evacuation, diet changes, stool softeners, and possibly enemas [13,21]. It is possible that these cats represent those with a greatly dilated rectum before surgery. This enlarged rectum, or megarectum, may be caused by chronic distention from large fecaliths or the result of a distal obstructive or pseudo-obstructive condition that interrupts the defecation reflex, such as paradoxic puborectalis contraction in human beings.

There have been cats that are presented for chronic diarrhea or perineal soiling (sometimes with perianal dermatitis if chronic) after colectomy. It has been suggested (but not proven) that this may be the result of small intestinal bacterial overgrowth [20]. Continence requires not only normal anal sphincter function but an adequate reservoir for storage of feces. Because reservoir capacity is reduced after colectomy, malabsorption diarrhea (steatorrhea) caused by bacterial overgrowth may appear as perineal soiling. I have empirically treated some of these cats with antibiotics (typically metronidazole), which has resulted in elimination of clinical signs. In a few of these cases, the cats required long-term antibiotic therapy (up to 6 weeks) before returning to a more normal postoperative course. If small intestinal bacterial overgrowth is suspected, the diagnosis should be proven by proper testing. A low serum cobalamin concentration, high serum folate concentration, and elevated concentrations of breath hydrogen all help to support this diagnosis.

In human patients with megacolon, total colectomy with ileorectal anastomosis seems to be superior to subtotal colectomy, because recurrence of constipation has been seen with less extensive resections. In the cat, it has been reported that subtotal colectomy with preservation of the ileocolic valve is a successful surgical treatment for idiopathic megacolon. It is recommended that the ileocolic valve be spared if at all possible so as to reduce the risk of small intestinal bacterial overgrowth [21]. Whether preservation
of the ileocolic valve after removal of 80% to 90% of the colon actually prevents colonic microorganisms from colonizing the ileum is unknown but seems unlikely. It is my opinion that total colectomy is the technique of choice; it is easier to perform, allows a tension-free anastomosis, and is more likely to prevent recurrent constipation because all the colon is removed.

Summary

Megacolon is a condition that is not uncommon in the cat. Most cases are idiopathic (a cause cannot be determined), and these seem to be a result of colonic inertia. Pelvic fracture malunions are the next most common cause and result in a pelvic outlet obstruction. Total or subtotal colectomy offers good long-term results in cases of idiopathic megacolon and chronic cases of pelvic fracture malunion, and the technique is described in detail.

References