Continuing Education Article #6

Ileal Impaction in Horses*

Auburn University
R. Reid Hanson, DVM
A. N. Baird, DVM, MS
D. G. Pugh, DVM, MS

KEY FACTS

- ❑ Ileal impaction is the most frequently reported small intestinal, nonstrangulating obstruction in adult horses.
- ❑ Feeding hay with a high-fiber content has been associated with ileal impaction in horses in the United States.
- ❑ The initial abdominal pain caused by ileal impaction is due to distention of the small intestine and spasm at the impaction site.
- ❑ Persistence of abdominal pain after nasogastric decompression and the presence of a very turgid small intestine on rectal examination are useful indicators in determining whether surgical intervention or medical therapy is required.
- ❑ Intestinal resection should be reserved for horses with small intestinal obstruction compounded by intestinal ischemia.

Acute abdominal pain in horses is often caused by abnormal conditions of the small intestine.[1] Simple obstruction, strangulating obstruction, and nonstrangulating obstruction are the general categories of small intestinal disorders that require surgical intervention.[1] Ileal impaction is the most frequently reported cause of small intestinal, nonstrangulating obstruction in adult horses.[2–4] The condition accounted for 0.5% to 9.3% of total colic cases reported in several surveys, retrospective studies, and individual case reports.[2–11] In a retrospective report involving surgical diseases of the ileum, ileal impaction accounted for more than 41% of the cases.[12]

ANATOMY

The ileum originates in an aboral direction to the jejunum in the left flank, crosses to the right abdominal quadrant at the level of the third to fourth lumbar vertebrae, and then passes upward to the lesser curvature of the base of the cecum, where it ends at the ileal orifice into the cecum[5] (Figure 1). The ileocecal junction is situated in the right dorsal quadrant of the abdomen, and it is relatively immobile because of the short mesenteric attachments to the cecum and right dorsal colon.[5]

The antimesenteric border of the ileum is attached to the dorsal taenia band of the cecum by the ileocecal fold.[13] In adult horses, this fold terminates approximately one meter from the cecum.[5] The ileal orifice is partially inverted into the cecum, which places the orifice in the center of a slight elevation formed by an annular fold of mucous membrane that contains a network of veins.[5,13] This network together with the muscle coat of the ileum serve as a functional ileal sphincter.[5]

The blood supply to the ileum is provided by the ileocolic artery.[13] This artery courses along the terminal portion of the ileum in a retrograde fashion and unites with the terminal jejunal artery. Because of the lack of distinguishing features between the jejunum and ileum, the point of transition from the typical arborization of the jejunal artery to the ileocolic artery is usually considered the start of the ileum.[5,13] When the ileum is relaxed, it is difficult to distinguish from the jejunum. In contrast, when the ileum is

*Publication Series No. 2486, College of Veterinary Medicine, Auburn University, Auburn, AL.

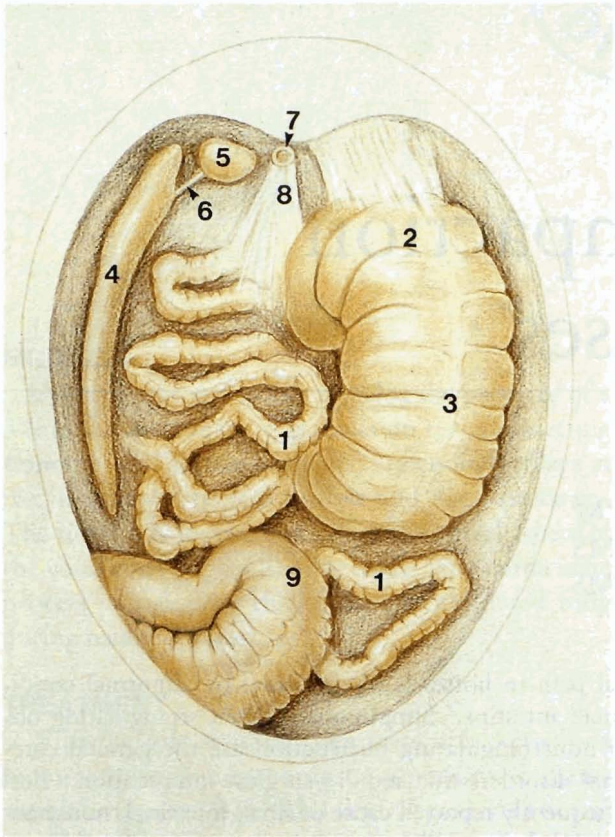

Figure 1—Normal rectal examination findings. *1* = small colon with distinct fecal balls, *2* = base of the cecum containing some gas, *3* = ventral cecal taenia band, *4* = spleen, *5* = kidney, *6* = renosplenic ligament, *7* = aorta, *8* = cranial mesenteric root, *9* = pelvic flexure and parts of the left large colon. The ileum cannot normally be palpated and was not included in this illustration.

contracted it is easily distinguished from the jejunum by its thicker muscular wall and narrower lumen.

MOTILITY AND DIGESTIVE FUNCTION

Experimental studies of cannulated isolated loops of the ileum in horses indicated that bidirectional movement of water occurred in the ileum when there was little or no intraluminal pressure.[14] An increase of 25 mm Hg in intraluminal pressure decreases ileal contractions and slows the absorption and secretion of water. Intraluminal pressure has the greatest effect on water absorption. Although the movement of sodium, potassium, bicarbonate, and chloride is minimal in the ileum, an increase in intraluminal pressure decreases the bidirectional movement of water and causes a net secretion of sodium and potassium.[14]

The ileum is highly resistant to changes in intraluminal osmolality, even when challenged with media having one-half or two times the osmolality of a buffered, isotonic solution.[14] Although absorption of water is prevented by increased intraluminal pressure, secretion of water continues. Horses recovering from ileal dysfunction should, therefore, be given water or a low-osmolar solution to drink to maintain intestinal motility, aid absorption, and discourage sequestration of water.[14]

Liquid digesta is rapidly propelled through the ileum into the cecal base, conducted to the cecal apex (where it is mixed with cecal contents), and then transported into the right ventral colon.[15] The myoelectric activity of the ileum has been studied in weanling ponies, and all phases of the migrating myoelectric complex and the migration action potential complex (MAPC) were identified. The MAPC is a prominent myoelectric complex and is a normal event in the equine ileum.[16] The MAPC has not been identified in the jejunum of horses.[15] These motility patterns are stimulated by the presence of liquid digesta and are responsible for the aboral transport of the digesta.[16] Data suggest that the MAPC rather than other migrating myoelectric complexes of the ileum may be responsible for the transit of digesta through the ileum into the cecum and is the only ileal event related to cecal motility patterns.[15]

Although the cranial and caudal cecal bases are capable of generating independent retrograde (base-to-apex) spiking activity, this activity may also be initiated in part by the MAPC of the ileum.[15] Ileal and cecal filling may, therefore, be more important in the regulation of ileocecal motility events than the neural or endocrine stimuli associated with eating.[15] Because the progressive myoelectric activity from the cecum to the right ventral colon is initiated by an electrical pacemaker near the cecal apex, surgically removing or bypassing the ileum does not adversely effect the motility of the large intestine.[15] Bypassing the ileocecal valve, however, disrupts the normal MAPC progression from the ileum to the cecum and right ventral colon and may allow bacterial overgrowth in the small intestine. Bacterial overgrowth in the small intestine can result in mucosal cell **damage**[17,18]; therefore, the ileocecal orifice should be preserved if possible.

An overall decrease in ileal migrating myoelectric complex spike activity and an increase in the MAPC frequency occur when *Strongylus vulgaris* larvae penetrate the ileal mucosa and migrate in the submucosa.[16] This resultant increase in MAPC frequency indicates that a relationship may exist between *S. vulgaris* larval infection and spasmodic colic in horses.[19] Most or all of the ileal smooth muscle responses to live, third-stage (L3) larvae may not be due to larval penetration and migration into the wall of the ileum but may be due to elaboration of various larval antigens.[16] Questions concerning mucosal receptor re-

sponses to larval antigens and the interaction of the receptors with the enteric nervous system, which controls the migrating myoelectric complex pattern and periodicity, remain to be investigated.[16]

The tapeworm *Anoplocephala perfoliata* is pathogenic for horses. Heavy burdens of these parasites can be associated with severe histologic changes at the ileocecal junction. Several clinical reports have linked tapeworm infections with intestinal diseases in horses,[19] including ileal thickening, obstruction and intussusception, and colonic torsion.[20–22] In addition to macroscopic thickening of the ileocecal valve, morphometric analysis of the ileal mucosa in horses with more than 100 tapeworms revealed that the mucosa is significantly thicker than the mucosa of normal horses. Eosinophil infiltration of the mucosa and submucosa was also present.[20] The severity of these histopathologic changes at the ileocecal junction supports the belief that appropriate anthelmintic treatment for tapeworms is beneficial in minimizing the associated pathology of the ileum.

PATHOGENESIS

The ileum has a limited blood supply that is provided solely by the ileocolic artery. In addition, the ileal artery is the branch of the cranial mesenteric artery most frequently involved in verminous arteritis caused by *S. vulgaris* larvae.[23] Lesions caused by these larvae can further predispose the ileum to episodes of hypoperfusion and segmental atony. The blood supply of the ileum and its fixed position in the intestinal tract may be important factors in determining whether the ileum is more frequently affected by obstructive disease than are other sections of the small intestine.[5]

Ileal impactions can exist several hours before signs of colic are exhibited.[2,24] Necropsy of several affected horses revealed the composition of the impaction to be dry, fibrous ingesta.[10] Violent peristalsis is the initial response to intestinal obstruction.[25–27] Presumably, these abnormal intestinal contractions extrude water from the accumulated mass of ingesta, thereby creating a drier, firmer, obstructing mass.[28]

Absorption of water is impaired and secretion of fluid is increased proximal to the obstruction in the intestine, which results in loss of fluid into the intestinal lumen. Intestinal distention, ileus, pain, and poor cardiovascular function are associated with these fluid shifts.[2,29] A decrease in circulatory function develops secondary to dehydration, which is caused by the sequestration of fluid in the intestine, insensitive metabolic fluid loss, and a reduced fluid intake.[29] A decrease in survivability is associated with progressive deterioration in circulatory function and concurrent intestinal distention.[2]

Ileal impactions are most common in the southeastern United States[2,9,10] and Europe.[3] Although the cause of ileal impactions is unknown, feeding horses hay with a high-fiber content has been associated with ileal impaction in the United States.[2,10,11,30] Coastal Bermuda grass hay, which is commonly fed in the southeastern United States, is often dry, fine, and stemmy. The lignin and crude fiber content of coastal Bermuda grass increases markedly as pastures mature (as seen in tall stands or late-summer cuttings).[30,31] When the mature grass is cut and fed as hay, the increase fiber content of such hay can predispose horses to impaction colic when associated with weather changes that limit water consumption.[11]

This condition for impaction is further aggravated by a combination of heat and stress, limited consumption of digestible roughage, ingestion of pelleted feeds, and limited twice-daily feeding schedules.[32] Ileal impaction was documented in four horses within four weeks of changing the hay ration to coastal Bermuda grass.[2] Incidence of ileal impaction is low in areas of the United States where legume or other hay combinations are the primary sources of roughage. In contrast, ileal impactions reported in Europe were not associated with high-fiber roughage but were primarily idiopathic in nature and had been associated with vascular thrombotic disease.[5]

Other causes of ileal obstruction include mesenteric vascular thrombotic disease,[2,4,10,12] ileocecal intussusception associated with *A. perfoliata* infection,[19] ileal hypertrophy of the mucosa and muscularis,[2,5,9,33–35] hernias involving the body wall, internal hernias involving mesenteric rents or the epiploic foramen, incarcerated scrotal–inguinal hernias,[12] and intraabdominal adhesions.[9] Although rare and difficult to document, impactions associated with mesenteric vascular thrombotic disease are believed to be caused by intestinal dysfunction due to ischemia.[4] In affected horses, the arterial lesions most often occur in the ileocecocolic branch of the cranial mesenteric artery.[4,36]

Tapeworms cause edema, ulceration, and formation of granulation tissue at the site of scolex attachment to the mucosa.[21,37] Tapeworms normally attach to the ileocecal orifice where the diameter of the bowel wall abruptly changes.[21] Heavy tapeworm infections[19] may obstruct the ileocecal orifice and cause fatal colic.[22]

Hypertrophy of the muscular layer of the ileum results in luminal narrowing and partial obstruction. Muscular hypertrophy occurs in two forms: idiopathic (primary) and compensatory (secondary). With idiopathic muscular hypertrophy, there is no detectable stenosis of the distal intestine to cause hypertrophy of the proximal intestinal muscularis. With compensatory muscular hypertrophy, the muscular layer

of the small intestine hypertrophies in response to chronic distal intestinal stenosis.

The hypertrophied muscle narrows the intestinal lumen, causing partial obstruction and distension of the intestine proximal to the obstruction, all of which result in abdominal pain.[33] Partial anorexia and chronic weight loss of one to six months in duration are common findings in patient histories.[34] Exploratory celiotomy is the only definitive method for diagnosing ileal muscular hypertrophy as a cause of colic.[33] Full-thickness rupture of the ileum with subsequent diffuse septic peritonitis has been reported in horses that have idiopathic muscular hypertrophy.[33,34]

Trauma to the body wall can result in abdominal-wall hernias in which an ileal impaction can develop subcutaneously.[7] Ileal impactions associated with internal hernias, scrotal–inguinal hernias, and adhesions are usually complicated by the incarceration of small intestine, which requires resection.[12]

CLINICAL FINDINGS AND DIAGNOSIS

The initial abdominal pain caused by ileal impaction is due to distention of the small intestine and spasm at the site of impaction.[9,28] Because fluid loss is minimal, there are few systemic effects during this stage of impaction development. The pain becomes more severe as the intestine proximal to the impaction distends with gas and fluid. On rectal examination, distention of the small intestine is a consistent finding, and the impacted ileum can occasionally be identified if the examination is performed early in the course of the disease.[3,10,38]

An increased heart rate (> 60 beats/min), nasogastric reflux, and decreased intestinal sounds are usually present.[3] The packed cell volume, plasma protein, serum anion gap, and protein concentration in the peritoneal fluid are usually increased.[39] In contrast, the white blood cell count; serum urea nitrogen; and peritoneal fluid white blood cell count as well as sodium, potassium, and chloride levels, are normal. Mild metabolic acidosis is usually present.[39] Significant differences in serum anion gap and plasma protein concentration have been reported between survivors and nonsurvivors of ileal impaction; values for nonsurvivors are higher.[2] Sequestration of fluids in the intestinal tract can eventually result in hypovolemic shock.[28]

These clinical findings, although they can vary with individual cases, are indicative of a nonstrangulating obstruction of the small intestine. Gastric reflux on nasogastric intubation and the presence of small intestinal distention on rectal examination are consistent with small intestine obstruction or proximal enteritis, although other diseases, such as impaction or torsion of the large colon, can infrequently cause these findings.[3,5,10,32,37] Peritoneal fluid analysis can usually differentiate simple obstruction from strangulating obstruction of the small intestine. Abnormal findings in the peritoneal fluid appear earlier with strangulating obstruction than with simple obstruction.[25,26,28,40,41]

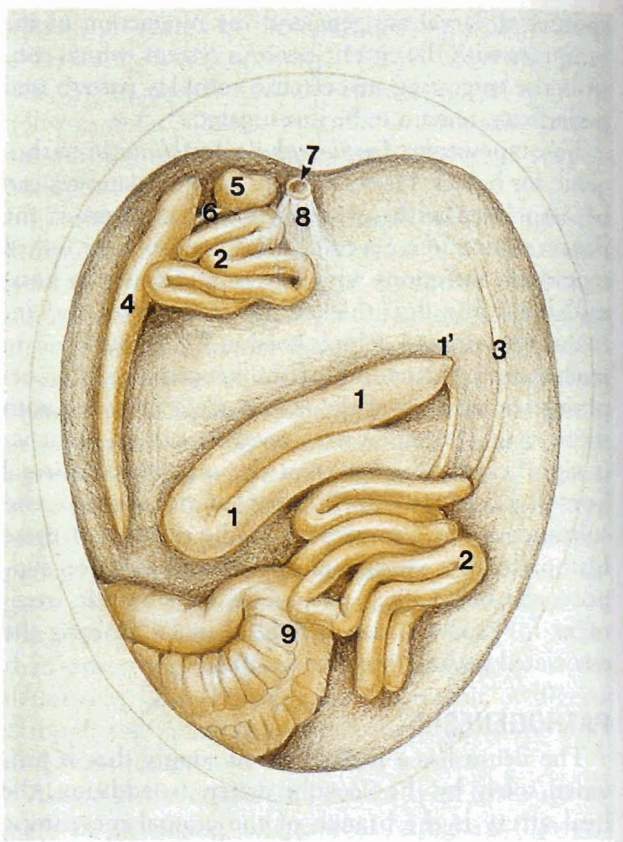

Figure 2—Rectal examination findings in patients with impaction of the ileum. *1* = enlarged ileum containing doughy ingesta; *1'* = ileocecal orifice; *2* = distended loops of small intestine, either fluid-filled or tympanic, without thickening of the bowel wall; *3* = ventral cecal taenia band; *4* = spleen; *5* = kidney; *6* = renosplenic ligament; *7* = aorta; *8* = cranial mesenteric root; *9* = pelvic flexure and parts of left large colon.

Useful indicators to distinguish horses that require surgical intervention for ileal impaction from those that do not are the persistence of abdominal pain after nasogastric decompression and the presence of a very turgid small intestine on rectal examination. The impaction may be palpated medial to the cecum on rectal examination early in the course of the disease (Figure 2); however, this finding was identified in only 18 of 75 and in 0 of 12 cases in two retrospective studies.[2,39] Small intestinal distention continues as the disease progresses, and the ileal impaction usually is obscured.

TREATMENT

Accurate, early diagnosis can facilitate successful medical treatment of horses with ileal impactions.[28,43] Medical treatments include intravenous fluid therapy, analgesics, intestinal lubricants, and intestinal stimulants.[28,43–45] Although intestinal stimulants have been suggested as possible treatments, they should be used with caution in prolonged ileal impactions because mucosal necrosis, perforation, and secondary gastric rupture can occur.[10,34,38] Early recognition of the failure to respond to medical management and of the need for surgical intervention is paramount to successful treatment of ileal impaction.[2,9,10]

Surgical intervention is generally indicated if rectal palpation over time reveals persistent impaction of the ileum and distention of the small intestine rather than a softening of the impaction.[10,36,43] Retrospective studies have demonstrated that the mean duration of clinical signs before surgery ranges from 13 to 17 hours for survivors and 18 to 25 hours for nonsurvivors.[9,10] Delays in surgical intervention result in a decrease in survival rate because of continuing deterioration of circulatory function and progressive intestinal distention.[2]

Manual reduction, enterotomy, intestinal resection, jejunocecostomy, and other anastomoses have been performed in the treatment of horses with ileal impaction.[2,3,5,9,10,12,38] Although some researchers recommend an intestinal bypass procedure,[9,12] more recent retrospective studies support extraluminal massage of the impaction and manual emptying of the ileal contents into the cecum.[2,10,38] If necessary, the mass can be softened by intraluminal injection of saline.[2,5,9,10,38] An increase in postoperative morbidity and mortality for ileal impactions has been associated with intestinal resection or jejunocecostomy.[2,9]

Intestinal resection should be reserved for horses with small intestinal obstruction compounded by intestinal ischemia. In these cases, intestinal resection and anastomosis are mandatory for survival.[2,10] Although there was no significant difference in the incidence of intraabdominal adhesions between horses that had intestinal resection or bypass and those that did not, fewer than 20% of horses with clinically significant postoperative adhesions survived.[8] Horses that remained asymptomatic for more than 60 days after small intestine surgery were less likely to develop complications associated with intraabdominal adhesions.

Ileal impactions have been previously associated with muscular hypertrophy of the ileum and ileal dysfunction.[12,33,35] As a result, jejunocecostomies were routinely performed to prevent reimpaction.[2,9,12] Jejunocecostomy has been abandoned except in cases in which ileal ischemia or hypertrophy of the muscular layers is suspected.[2,10]

If muscular hypertrophy of the ileum is present with associated ileal dysfunction, a bypass procedure should be considered to prevent recurrence of the impaction.[2,9,10,12,21,33,35] If necrosis or evidence of infarction is not present and is unlikely to develop, however, a bypass between the distal jejunum and cecum (without ileal resection) should be created.[12] This bypass ensures passage of ingesta and preserves the original anatomic conformation. Although feed material may still attempt to pass through the ileum and potentially cause abdominal pain, clinical case report surveys suggest that postoperative morbidity and mortality are lower after this procedure than if resection and anastomosis are performed.[12,46,47]

Studies have reported that 55%, 39%, and 64% of horses in which ileal impaction was diagnosed by exploratory celiotomy survived long-term (i.e., five months to six years).[2,9,10] Reasons for death or euthanasia include ileus,[2,9,10] shock,[2] gastric rupture,[2,9] laminitis,[2,9] intestinal adhesions,[9,10] jejunal incarceration,[2,10] impaction, and/or peritonitis.[2,9,10] Most of these conditions (and corresponding increases in mortality) were due to complications associated with the intestinal resection or bypass procedure.[2,9,10] Reimpaction of the ileum following manual reduction has not been reported. Although ileus is a common complication of abdominal surgery, there was no difference in the rate of occurrence of postoperative ileus between survivors and nonsurvivors.[2]

SUMMARY

Successful medical therapy of horses with ileal impaction can be facilitated by an accurate, early diagnosis of the disease.[28,43] Early surgical intervention is generally indicated, however, if rectal palpation reveals persistent distention of the small intestine rather than progressively smaller impaction of the ileum.[10,28,43] As the time from disease onset to surgical intervention increases, the continuing deterioration of circulatory status and progressive intestinal distention become the primary reasons for the decrease in survival rate.[2] Manual reduction of ileal impaction produces fewer postoperative complications and greater long-term survival than enterotomies or intestinal bypass procedures.[2] In horses with inadequate patency of the ileal lumen and/or intestinal ischemia or necrosis, the surgeon should not hesitate to perform an intestinal resection and/or jejunocecostomy. These procedures are, however, associated with increased morbidity and mortality.

ACKNOWLEDGMENT

The authors thank Mrs. Debi Burelle for her assistance in the preparation of this article.

About the Authors

Drs. Hanson, Baird, and Pugh are affiliated with the Department of Large Animal Surgery and Medicine, College of Veterinary Medicine, Auburn University, Auburn, Alabama. Drs. Hanson and Baird are Diplomates of the American College of Veterinary Surgeons. Dr. Pugh is a Diplomate of the American College of Theriogenology and the American College of Veterinary Nutrition.

REFERENCES

1. Snyder JR: Pathophysiology, diagnosis and treatment of equine small intestinal disorders. *Proc AAEP* 35:79–89, 1989.
2. Parks AH, Doran RE, White NA, et al: Ileal impaction in the horse: 75 cases. *Cornell Vet* 79:83–91, 1989.
3. Huskamp B: The diagnosis and treatment of acute abdominal conditions in the horse: The various types and frequency as seen at the animal hospital in Hochmoor. *Proc Equine Colic Res Symp* 1:261–272, 1982.
4. White NA: Intestinal infarction associated with mesenteric vascular thrombotic disease in the horse. *JAVMA* 178(3): 259–262, 1981.
5. Edwards GB: Obstruction of the ileum in the horse: A report of 27 clinical cases. *Equine Vet J* 13(3):158–166, 1981.
6. Parry BW: Survey of 79 referral colic cases. *Equine Vet J* 15(4):345–348, 1983.
7. Ibrahim IM, Gohar HM, Ahmed AS, et al: Small intestinal herniation associated with an ileal impaction in a foal. *Equine Pract* 9(5):15–16, 1987.
8. Baxter GM, Broome TE, Moore JN: Abdominal adhesions after small intestinal surgery in the horse. *Vet Surg* 18(6): 409–414, 1989.
9. Doran RE, White NA, Allen D: Clinical aspects of ileal impaction in the horse. *Proc Equine Colic Res Symp* 2:182–185, 1986.
10. Embertson RM, Colahan PT, Brown MP, et al: Ileal impaction in the horse. *JAVMA* 186(6):570–572, 1985.
11. Pugh DG, Thompson JT: Impaction colics attributed to decreased water intake and feeding coastal Bermuda grass hay in a boarding stable. *Equine Pract* 14(1):9–14, 1992.
12. Kersjes AW, Bras GE, Nemeth F, et al: Results of operative treatment of equine colic with special reference to surgery of the ileum. *Vet Q* 10(1): 17–25, 1988.
13. Kotze SH: Arterial blood supply to the ileocecal junction in the horse. *J S Afr Vet Assoc* 61(1):2–4, 1990.
14. Donawick WJ, Ramberg CF, Topkis GA: Absorption and secretion of water and electrolytes by normal and obstructed ileum of ponies. *Proc Equine Colic Res Symp* 1:95–103, 1982.
15. Ross MW, Cullen KK, Rutkowski JA: Myoelectric activity of the ileum, cecum, and right ventral colon in ponies during interdigestive, nonfeeding and digestive periods. *Am J Vet Res* 51(4):561–566, 1990.
16. Berry CR, Merritt AM, Burrows CF: Evaluation of the myoelectrical activity of the equine ileum infected with *Strongylus vulgaris* larvae. *Am J Vet Res* 47(1):27–30, 1992.
17. Schumacher J, Moll HD, Spano J, et al: Effect of intestinal resection on two juvenile horses with granulomatous enteritis. *J Vet Intern Med* 4(3):153–156, 1990.
18. Tate LP, Ralston SL, Koch CM, et al: Effects of extensive resection of the small intestine in the pony. *Am J Vet Res* 44:1187–1191, 1983.
19. Levine ND: *Protozoan Parasites of Domestic Animals and of Man*, ed 2. Minneapolis, MN, Burgess Publishing Co, 1973, pp 1–21.
20. Pearson GR, Davies LW, White AL, et al: Pathological lesions associated with *Anoplocephala perfoliata* at the ileocaecal junction of horses. *Vet Rec* 132:179–182, 1993.
21. Barclay WP, Phillips TN, Foerner JJ: Intussusception associated with *Anoplocephala perfoliata* infection in five horses. *JAVMA* 180(7):752–753, 1982.
22. Bello TR: Perspectives on current equine anthelmintic therapy: Misunderstandings and clarification, *Proc AAEP* 25: 261–269, 1979.
23. Rous RC: Mesenteric thrombosis. *J S Afr Vet Assoc* 46(1): 79–80, 1975.
24. MacHarg MA, Adams SB, Lamar CH, et al: Electromyographic, myomechanical, and intraluminal pressure changes associated with acute extraluminal obstruction of the jejunum in conscious ponies. *Am J Vet Res* 47(1):7–11, 1986.
25. Moore JN, White NA: Acute abdominal disease: Pathophysiology and preoperative management. *Vet Clin North Am Large Anim Pract* 4(1):61–78, 1982.
26. Robertson JT: Conditions of the stomach and small intestine: Differential diagnosis and surgical management. *Vet Clin North Am Large Anim Pract* 4(1):105–127, 1982.
27. Tulleners EP: Small bowel obstruction, in Robinson NE (ed): *Current Therapy in Equine Medicine*. Philadelphia, WB Saunders Co, 1983, pp 224–231.
28. Huskamp B, Daniels H, Kopf N: Diseases of the digestive system and peritoneum, in Dietz O, Wiesner E (eds): *Diseases of the Horse: Part 2*. New York, Karger, 1984, pp 184–191.
29. Lantz GC: The pathophysiology of acute mechanical small bowel obstruction. *Compend Contin Educ Pract Vet* 3(10): 910–921, 1981.
30. Cunha TJ: *Horse Feeding and Nutrition*, ed 2. San Diego, CA, Academic Press Inc, 1991, pp 274–293.
31. Johnson JT, Seqors WI, Murphy TR: *Bermuda Grass in Georgia*. University of Georgia Cooperative Extension Service Bulletin 911, 1990.
32. Morris DD, Moore JN, Ward S: Comparison of age, sex, breed, history and management in 229 horses with colic. *Equine Vet J (Suppl)* 7:129–132, 1989.
33. Chaffin MK, Fuenteabla IC, Schumacher J, et al: Idiopathic muscular hypertrophy of the equine small intestine: 11 cases (1980–1991). *Equine Vet J* 24(5):372–378, 1992.
34. Lindsay WA, Confer AW, Ochoa R: Ileal smooth muscle hypertrophy and rupture in a horse. *Equine Vet J* 13(1): 66–67, 1981.
35. Robertson JT: Muscular hypertrophy of the ileum, in White NA (ed): *The Equine Acute Abdomen*. Philadelphia, Lea & Febiger, 1990, pp 352–354.
36. Wright AI: Verminous arteritis as a cause of colic in the horse. *Equine Vet J* 4:169–174, 1972.
37. Drudge JH: Metazoal diseases, in Catcott EJ, Smithcors JF (eds): *Equine Medicine and Surgery*, ed 2. Wheaton, IL, American Veterinary Publications, 1972, pp 175–177.
38. Robertson JT: Ileal impaction, in White NA (ed): *The Equine Acute Abdomen*. Philadelphia, Lea & Febiger, 1990, pp 351–352.
39. Adams SB, McIlwraith CW: Abdominal crisis in the horse: A comparison of presurgical evaluation with surgical findings and results. *Vet Surg* 7(3):63–69, 1978.
40. Bramlage LR: Examination in acute abdominal crisis, in Mansmann RA, McAllister ES (eds): *Equine Medicine and Surgery*, ed 3. Santa Barbara, CA, American Veterinary Publications, 1982, pp 548–559.
41. Foerner JJ: Diseases of the large intestine: Differential diag-

nosis and surgical management. *Vet Clin North Am Large Anim Pract* 4(1):129–146, 1982.
42. Kohn CW: Pathophysiology of obstructive GI disease, in Mansmann RA, McAllister ES (eds): *Equine Medicine and Surgery*, ed 3. Santa Barbara, CA, American Veterinary Publications, 1982, pp 524–526.
43. Hofmyer CF: The digestive system, in Oehme FW, Prier JE (eds): *Textbook of Large Animal Surgery*. Baltimore, Williams & Wilkins, 1974, pp 437–446.
44. Blood DC, Henderson JA, Radostit OM, et al: Diseases of the alimentary tract, in *Veterinary Medicine, A Textbook of the Diseases of Cattle, Sheep, Pigs, and Horses*, ed 5. Philadelphia, Lea & Febiger, 1979, pp 144–145.
45. Page EH, Amstutz HE: Gastrointestinal disorders and peritonitis, in Catcott EJ, Smithcors JF (eds): *Equine Medicine and Surgery*, ed 2. Wheaton, IL, American Veterinary Publications, 1972, pp 268–269.
46. Edwards GB: Surgical management of intussusception in the horse. *Equine Vet J* 18(4):313–321, 1986.
47. Edwards GB: Resection and anastomosis of small intestine: Current methods applicable to the horse. *Equine Vet J* 18(4):322–330, 1986.

ARTICLE #6 REVIEW QUESTIONS

The article you have read qualifies for ½ hour of Continuing Education Credit from the Louisiana State University School of Veterinary Medicine. *Choose only the one best answer* to each of the following questions; then mark your answers on the registration form inserted in *The Compendium.*

1. Impaction accounts for what percentage of cases that involve surgery of the ileum in horses?
   a. more than 5
   b. 20 to 25
   c. 40 to 45
   d. 60 to 65

2. The ileocecal fold attaches the antimesenteric border of the ileum to which taenia band of the cecum?
   a. dorsal
   b. medial
   c. lateral
   d. ventral

3. The ileum has a thicker muscular wall and narrower lumen than the jejunum. This difference is most conspicuous when the ileum is
   a. impacted.
   b. contracted.
   c. relaxed.
   d. distended with gas.

4. Which of the following parasites has been reported to be associated with ileal impactions and is found near the ileocecal junction?
   a. *Strongylus vulgaris*
   b. *Anoplocephala perfoliata*
   c. *Oxyuris equi*
   d. *Dictyocaulus arnfieldi*

5. Ileal impaction is most commonly seen in which region of the United States?
   a. Northwest
   b. Southwest
   c. Northeast
   d. Southeast

6. What type of hay diet is most commonly associated with ileal impaction?
   a. alfalfa
   b. coastal Bermuda grass
   c. timothy
   d. none of the above

7. The surgical treatment of choice for ileal impaction when no hypertrophy or ischemia of the intestine is evident is
   a. enterotomy.
   b. ileal resection.
   c. side-to-side jejunocecostomy.
   d. manual reduction only.