

INTRODUCTION:

Colic, defined as any abdominal pain, has resulted in the death of horses throughout recorded history. Risk factors identified for colic include breed, feeding grain, changing feed, changing housing, previous colic, and treatment for parasites. Identified factors are associated with simple colic or impactions, while risk factors for the deadliest strangulating lesions are just beginning to be elucidated.

CAUSES OF COLIC:

The signalment and history may guide the clinician to a presumptive diagnosis and shape the diagnostic workup. The following are some examples of signalment leading to presumptive diagnosis:

- A young racehorse with a recent orthopedic injury should be evaluated for impactions of the large colon or cecum.
- A broodmare that recently foaled and is now presented with acute severe colic should be evaluated for a large colon volvulus.
- Older, obese horses with colic may have a lipoma strangulating the small intestine.
- Adult horses that have mild recurrent colic and are passing mineral oil and scant feces should be evaluated for enterolithiasis. Chronic intermittent low-grade colic is typical of partial intraluminal obstructions (enteroliths) or displacements.
- Cribbers should be evaluated for epiploic foramen entrapments.
- Foals with a recent history of deworming may have an ascarid impaction.

SPECIFIC CAUSES OF COLIC INCLUDE:

1. Sudden changing of food (from oats to corn), overeating, damaged feed, irregular work, fatigue, drinking excessively after work, stormy weather, increased temperatures, idiosyncrasy, and faulty mastication. Dietary factors, including the feeding of concentrates or poor quality roughage, were considered primary causes of colic.

2. Parasites were recognized as a major cause of colic, and *Strongylus vulgaris* blamed as the chief cause of simple and thromboembolic colic in horses. Recently, small strongyles, rather than *Strongylus vulgaris*, were determined to be the main cause of increased colic incidence on farms. Not until the widespread use of ivermectin was it clear that the decrease in thromboembolic colic paralleled the decrease in thrombotic lesions found in the cranial mesenteric artery (Fig. 1). Parasites tend to cause Spasmodic colic.

3. Small intestine can become strangulated in mesenteric ligamentous bands that cannot be exteriorized at surgery and must be cut blindly with scissors. Small intestine can also become strangulated by uterine torsion; through rents in the mesometrium, gastrohepatic ligament, small colon mesentery, lateral ligament of the urinary bladder, cecocolic fold and mesentery of the large colon; by components of the spermatic cord, particularly the mesoductus deferens; and by omental adhesions. Duodenal torsion has been reported but is rare. Evisceration through a lacerated vaginal

fornix, a defect in the bladder and urethra, or a castration wound may cause small intestinal strangulating obstruction. Entrapment of small intestine within the nephrosplenic space with the bowel passing from cranial to caudal. Mesenteric hematomas of unknown cause can cause colic and ischemic necrosis of affected intestine.

4. Other specific causes include gastric ulceration, gastric impaction, gastric rupture, small intestine obstruction, small intestine adhesion, small intestine lack of function, ceceal impaction, ileocecal intussusception, cecal perforation, enterolithiasis, sand enteropathy, and neoplasia.

5. Adhesions after small intestinal surgery or any intraabdominal procedure can form an axis around which attached small intestine can form a volvulus, or adhesions can form fibrous bands through which small intestinal loops can become strangulated.

6. Nonstrangulating infarction and necrotizing enterocolitis in the small intestine are rare and have a poor prognosis.

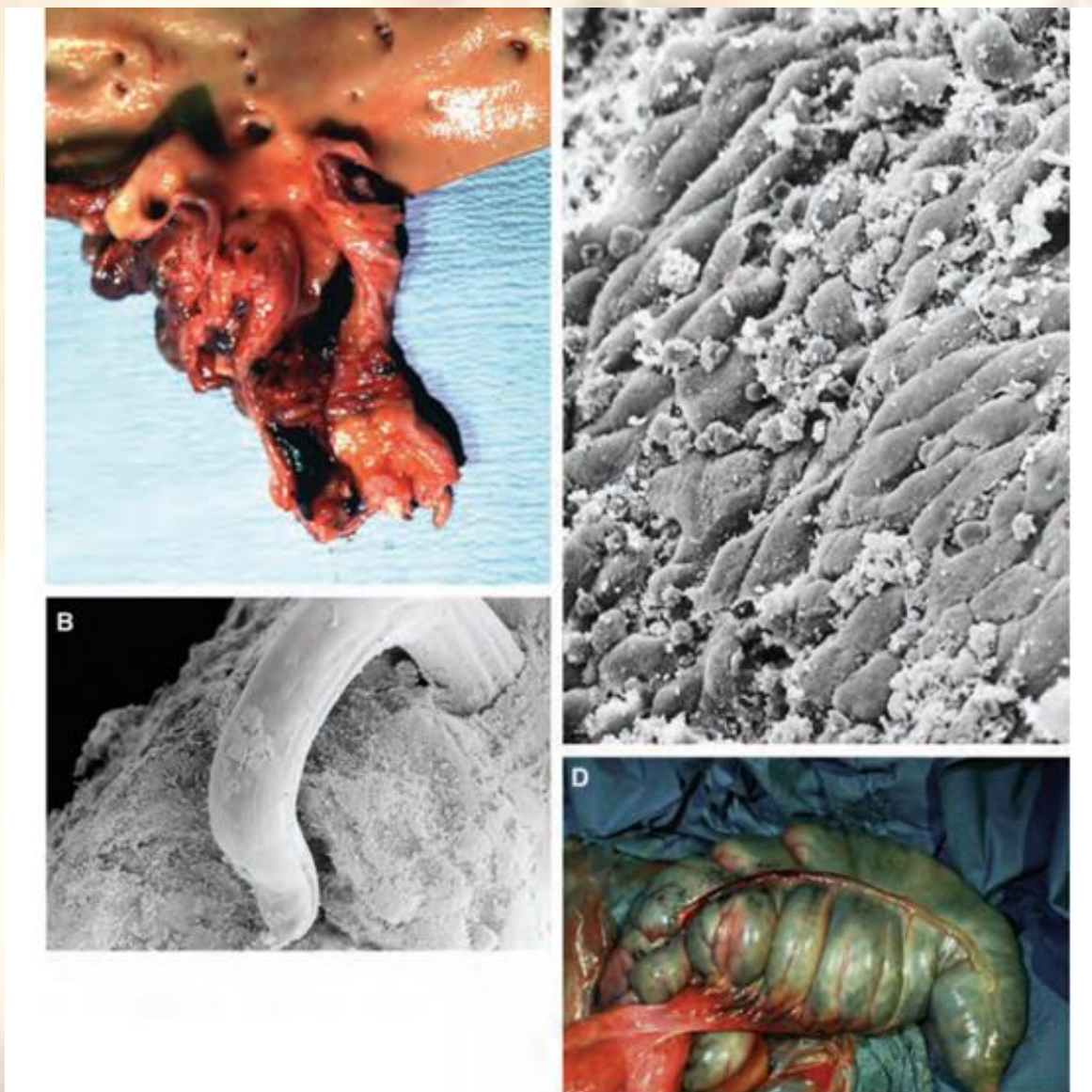


Fig. 1. (A) Verminous arteritis with thrombus formation in the cranial mesenteric artery was caused by 4th stage larva of *Strongylus vulgaris*. (B) Scanning photomicrograph of a perfused cranial mesenteric artery with a *Strongylus vulgaris* larva within a thrombus. (C) The endothelium surrounding the thrombus was disrupted with adherent platelets and WBC. These lesions were very prevalent in horses prior to the use of ivermectin and were associated with infarction predominately of the cecum (D) and colon. (Figure 1B reprinted from *Equine Vet J* 1983;15:349–353; figure 1D reprinted from *J Am Vet Med Assoc* 1981;178:259–262)