Equine Colic

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I. Introduction

Colic, defined as any abdominal pain, has resulted in the death of horses throughout recorded history. The earliest treatments may have been acupuncture. The use of drugs for treatment of associated pain and ileus has only applied recently. A definition offered in a book entitled Diseases of Livestock (1880) by a human physician states that the spasmodic colic gripes is caused by “improper food, sudden changes in diet, exhaustion from overwork, drinking cold water when heated or exhausted, constipation, worms, and ulcers of the stomach.” Flatulant colic was “attributed to easily fermented food, such as raw potatoes, green clover, brewers’ slop and the like.” Recommended treatments included drenches such as chloroform, laudanum, sulphuric ether (1 oz each in 8 oz of linseed oil), and were reported to “never fail” in a genuine case of colic. Later texts separated simple colic such as spasmodic colic and tympany from more severe colic caused by intestinal strangulation, which was considered uniformly fatal. The causes of many types of colic were discussed and, in some cases, with equal accuracy, compared with what is known today.

Research on equine gastrointestinal disease was not mentioned in early veterinary texts. Initial research was mostly observational with conclusions about the cause of colic based only on lesions found at necropsy or a response to treatment. Some of the first observations about causes of colic by Udall are still professed today. Proposed etiologies included 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.

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. Verminous colic was reproduced by infecting foals with Strongylus vulgaris larva; however, vascular lesions were observed in ~90% of horse necropsies, and few of these horses suffered from colic. Therefore, the actual risk of colic associated with this parasite was questioned. 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).

Equine laparotomy was described in books in the early 1900s; however, the first journal articles did not appear in the veterinary literature until 1970. Surgery was considered risky, and even if a horse survived, a poor prognosis for return to use was the norm. The poor survival rate was predominantly due to attempts at completing surgery in horses already in shock. Tennant et al. published some
of the first epidemiologic work examining types of colic and survival of horses at a teaching hospital; overall survival rate was reported to be ~50%. This percentage included simple colic as well as strangulating lesions.

Written observations of horses with colic helped to establish characteristics of different diseases. The advent of inhalant anesthesia allowed surgeons to save more horses with severe obstruction or strangulation, and, subsequently, it stimulated clinicians to investigate the causes and pathophysiology of these severe diseases. Initial veterinary publications related to colic were largely descriptive reports of clinical cases, new surgical techniques, and treatment for shock.60,61 However, more recent data suggest that associated improvements in survival rates, reduction of ileus after surgery.57

In the 1970s veterinarians pioneered techniques for assessment and treatment of shock in horses. One was assessment of shock and use of fluid therapy to treat both medical and surgical diseases. Rapid assessment of the horse’s metabolic condition allowed the clinician to immediately replace serum electrolytes and total fluid deficits.55 Measurement of lactate concentrations helped to determine prognosis, but, more importantly, it highlighted the dramatic perfusion deficit created by the shock syndrome that many horses experienced during episodes of colic due to intestinal strangulation and infarction.56 Concurrent with improvements in metabolic assessment was the advent of peritoneal fluid analysis as a diagnostic aid, and this analysis helped to standardize the assessment for surgery.57

A second major advancement in colic treatment was the introduction of potent analgesic medications, especially flunixin meglumine (flunixin), which has been the standard for treatment of pain and shock in horses affected with colic for three decades. Flunixin provided longer analgesia and protected horses from some of the deleterious effects of endotoxin and, equally important, was found to be a potent anti-inflammatory agent.58,59 At first its use created diagnostic dilemmas, as improvement in clinical signs after treatment delayed referral of some horses that needed surgery. Currently, veterinarians in the United States use flunixin more than any other analgesic, and there have been associated improvements in survival rates, reduction of adhesions, and evidence of less ileus after surgery.60,61 However, more recent data suggest that flunixin also has potential deleterious effects, including decreases in wound strength of abdominal incisions, potentiation of gastric ulcers, and inhibition of mucosal barrier function after healing.62

As surgical skills and techniques developed, application of general anesthesia improved with new gas anesthetics, cardiovascular support, and monitoring.63–66 This provided safer general anesthesia, improved the physiological condition of the patient, and allowed more time for surgery. Decreasing pain and shock during anesthetic recovery has further improved the response to surgery.

Rapid recognition of surgical conditions, early surgical intervention, and enhanced critical care have all improved the prognosis for horses with severe colic. Strangulating lesions and associated endotoxemic shock and ileus are still cited as the chief treatment challenges after surgery.67–69 Survival after surgery has improved from 20–30% percent in the 1970s and 1980s to 80–90% in some practices where early recognition is combined with adequate post-operative care.8,16,20,70,71 With the proliferation of private veterinary practices that can provide surgical and critical care, the chance that a horse will survive a serious colic episode and return to normal function has markedly improved in the last 30 years.

Colic Research

Some of the first colic research using disease models began 40 years ago. Nelson et al.72 studied intestinal strangulation by creating a model of colon infarction that reproduced the classic signs of endotoxemia and death from shock. Chander73 examined duodenal obstruction, ileal volvulus, and small colon obstruction, as well as the associated changes in the peritoneal fluid and blood. Ruckebusch et al.74–76 was one of the first to investigate normal intestinal motility in horses. Sellers et al.77 and Roberts et al.78 reported on pulse-retroperitoneal activity of the large colon and showed that alpha agonists caused colon stasis. Methods of experimentally inducing abdominal pain included a cecal balloon model used to test analgesics.79 From this work, a system to measure potency of analgesics was created, and this classification is still used today. The effect of alpha adrenergic medications on colonic motility and their potential contribution to the development of colonic impactions was also investigated.80 Roberts and Argenzio80 determined that a specific arachnicide (aminotraz) causes impactions by its alpha-agonist effect in the colon, which suggests that alterations in motility are likely to cause naturally occurring impactions.

The physiologic changes that occur with intestinal obstruction and strangulation were reproduced in several models, most of which were based on previous work in laboratory animals. Distention of the small intestine was shown to create a secretory organ that ultimately became ischemic.81,82 Both partial and total ischemia created by arterial or arteriovenous occlusion caused intestinal damage and post-ischemic injury; this helped to explain post-operative syndromes of ileus and shock.83–87 Distention was found to create significant inflammation, which helped explain clinical signs that persisted after the apparent medical or surgical relief of a simple obstruction.81,82,83,89 This initial work has led to the
study of adhesions and the creation of models to study reperfusion injury and its sequela.106

As the understanding of the pathophysiology of ischemia and reperfusion increased, research to find treatments to nullify or prevent the untoward effects were initiated. Systemic drugs, such as flunixin and topical compounds like rinse solutions or films, were tested and are in use today to lower morbidity and increase survival.92,93 The usefulness of pro-kinetic therapies has been investigated, leading to the current realization that the inflammatory process resulting from ischemia or distension prevents stimulation of the inflamed intestine through the neuromuscular pathways.94–97 Although the ability to assess the degree of intestinal damage and to resuscitate ischemic bowel has improved over the last three decades, accurately determining whether a specific segment of bowel will ultimately live or die remains extremely difficult.98

Models of endotoxic shock in horses have paralleled human research and have identified the lung and intestine as specific shock organs in the horse. Improvements in clinical treatment of horses suffering from endotoxemia have included the use of flunixin, hypertonic saline, polymixin B, and hyperimmune serum.99–102 The quest for future treatment options now involves molecular research in an attempt to understand the mechanism of individual cell responses to cytokines that create the systemic response to endotoxin.99,103–106 Although research has improved treatment of endotoxemia, there is still no highly effective cure.

Understanding of the epidemiology of colic has been expanded by numerous case control and cohort studies. The incidence and mortality rates associated with specific diseases has been reported. Risk factors identified for colic include breed, feeding grain, changing feed, changing housing, previous colic, and treatment for parasites.105–110 Identified factors are associated with simple colic or impactions, while risk factors for the most deadly strangulating lesions are just beginning to be elucidated. Predictive models have been developed in hopes of being able to predict survival in horses presenting with severe colic.111–116 Although valuable in confirming the need for surgery or the chance for survival, these formulas are rarely used by clinicians, because many are not user friendly. Additionally, owners often elect treatment in hopes of a cure, even when the prognosis is guarded or poor.

Because there are few similar diseases in man, understanding intestinal displacements, such as large colon volvulus and small intestinal incarcerations, will require focused research. Epidemiologic studies and investigations of intestinal motility are needed to understand the causes and possible prevention of these disorders. Inheritance of particular traits that increase the risk of colic may become evident in susceptible animals. As for simple colic, much is now known about how to prevent this type of intestinal disturbance.110 Careful attention to diet, exercise, and environmental management can be efficacious in preventing many of these problems. Nevertheless, more studies on events and diet related to colic episodes are needed to define optimal strategies for the prevention of equine colic.

References

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