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Jejunojunostomy in horses: a comprehensive review of pathophysiology, surgical technique, and clinical management

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Abstract. Despite ongoing efforts to reduce post-operative complications in horses undergoing colic surgery, a deep understanding of the pathophysiological mechanisms remains essential to maximize survival rates and minimize occurrences such as post-operative ileus (POI/POR) and recurrent post-operative colic (POC). This paper provides a systematic clinical and literature review regarding the primary complications following small intestine anastomosis (jejunojejunostomy) in equines, alongside therapeutic approaches aimed at prevention and management.

Keywords: Equine colic, intestinal healing, POI, post-operative complications, small intestine anastomosis.

Jejunojunostomia em equinos: uma revisão abrangente sobre fisiopatologia, técnica cirúrgica e manejo clínico

Resumo. Apesar dos esforços contínuos para reduzir as complicações pós-operatórias em equinos submetidos à cirurgia de cólica, uma compreensão aprofundada dos mecanismos fisiopatológicos permanece essencial para maximizar as taxas de sobrevivência e minimizar ocorrências como o íleo pós-operatório (POI/POR) e a cólica pós-operatória recorrente (POC). Este trabalho apresenta uma revisão clínica e sistemática da literatura sobre as principais complicações após anastomose do intestino delgado (jejunojejunostomia) em equinos, juntamente com abordagens terapêuticas voltadas à prevenção e ao manejo.

Palavras-chave: Cólica equina, cicatrização intestinal, POI, complicações pós-operatórias, anastomose do intestino delgado.

Introduction

The management of equine small intestinal disorders requiring resection and anastomosis remains challenging in equine emergency surgery (Haddad et al., 2022). Historically, success was measured solely by immediate perioperative survival. However, contemporary veterinary medicine defines therapeutic success by the horse's full return to its functional and athletic activities. To achieve this goal, requires mastery of not only manual dexterity but also the complex pathophysiology of intestinal healing and the critical factors that predispose to postoperative complications (Santos, 2014). In this context, this study proposes a comprehensive bibliographic review, grounded in the author's clinical and surgical experience, on jejunojunostomy. It addresses aspects ranging from fluid management and endothelial stabilization to the technical refinements necessary to minimize the risks of stenosis and dehiscence. The primary objective is to discuss the factors contributing to complications following small intestinal anastomosis, as well as evidence-based preventive measures and therapeutic strategies.

Delayed surgical intervention: The time factor and pathophysiology

In equine acute abdomen, particularly when small intestinal (SI) lesions are involved, the interval before initiating appropriate treatment, whether medical or surgical, is the determining factor for the resolution of the case and therapeutic success ([Alves et al., 2005](#); [Aranzalez & Alves, 2014](#)). Time loss occurs not only due to delays by the owner in contacting a veterinarian but also as a result of diagnostic errors, hesitation in hospital referral, “false” clinical improvements, and the accumulation of “parasitic time” during initial care and surgical treatment. Examples of the latter include delays in surgeon scrubbing and gowning, surgical clipping, team disorganization, and the absence of surgical checklists ([Mair et al., 2002](#)).

In cases of SI distension without direct mesenteric vascular compromise, an initial microcirculatory alteration occurs in the mucosa, extending to the other layers of the intestinal wall as the clinical condition progresses. An intraluminal hydrostatic pressure of 18 cmH₂O can reduce blood flow in the jejunal arteries to half of the basal value, maintaining hypoperfusion even after segment decompression. Should the pressure reach 25 cmH₂O for 120 minutes, villous shortening, loss of mesothelial cells, neutrophilic infiltration, and edema of the seromuscular layer are observed. This process results in a pro-inflammatory environment with tissue injuries that may lead to local or systemic postoperative complications ([Freeman, 2025](#)).

The SI, unlike the large colon, possesses villi characterized by a “countercurrent exchange” vascular mechanism, composed of a central arteriole and two peripheral venules ([Blikslager & White, 2017](#)). This anatomy renders the villous tip hypoxic even under physiological conditions ([Blikslager, 2008](#); [Blikslager & White, 2017](#)). Such characteristics make the SI a highly sensitive segment, deteriorating approximately 25% faster than the large colon under similar insults ([Blikslager & White, 2017](#)). However, the large intestinal lesions should not be underestimated.

Criteria favorable to medical management include a satisfactory response to analgesia, normalization of heart rate (HR), passage of feces, and progressive return of intestinal motility. On the other hand, criteria unfavorable to medical management (indicators for referral or surgery) include pain refractory to analgesia; continuous and progressive elevation of heart rate (HR); alterations in mucosal color associated with a significant increase in capillary refill time (CRT); persistent hypomotility or intestinal atony; abdominal distension and/or nasogastric reflux; and inability to establish an accurate field diagnosis, requiring continuous intensive support and/or surgical intervention ([Blikslager, 2008](#)).

Tissue perfusion

Blood supply is the cornerstone of intestinal homeostasis, adequate healing, and the maintenance of tissue vitality. Blood flow distribution across the intestinal wall layers is non-uniform and correlates with the functional significance of each layer ([Matheson et al., 2000](#); [Rudorf et al., 2005](#)). Approximately 70% of the blood supply is directed to the mucosa, while the serosal and muscular layers receive about 25%, and a smaller proportion is directed to the submucosa. Within the mucosa, 60% of the flow is distributed to the villi and 40% to the crypts ([Matheson et al., 2000](#)).

Compromised blood supply at the anastomotic site may result from technical errors or systemic conditions such as hyperinflammation, hypotension, sepsis, and shock, which promote both micro- and macrovascular alterations ([Rosendorf, 2022](#)). Microcirculation is critical for healing, as it comprises the network of arterioles, venules, and capillaries responsible for oxygen and nutrient exchange ([Tavy et al., 2020](#)); insufficient perfusion drastically increases the risk of healing failure and anastomotic dehiscence (leakage) ([Paramasivam, 2026](#)).

A reduction in mucosal blood flow to levels critical for cellular viability triggers the production of oxygen free radicals (OFRs) and pro-inflammatory cytokines, predisposing to loss of epithelial barrier integrity ([Matheson et al., 2000](#)). At the surgical site, serosal perfusion is generally more compromised than mucosal perfusion because the ischemic segment has been removed. Thus, serosal injury may be associated with intraluminal distension, surgical manipulation, and inflammation, particularly within a 15 cm radius proximal to the anastomosis ([Tavy et al., 2020](#)).

Endotoxemic or septic shock and intraluminal distension profoundly alter intestinal microcirculation. Even after correction of perfusion through decompression, fluid therapy, electrolyte balance, and pharmacological support (e.g., vasoactive agents), serosal microcirculation may improve while mucosal microcirculation remains impaired for a prolonged period ([Freeman, 2025](#); [Tavy et al., 2020](#)). In cases of septic shock, microcirculatory changes occur both at the anastomotic site and in non-manipulated segments, with histological findings of pronounced hyperemia and vascular stasis, consistent with the initial vascular phase of the acute inflammatory response ([Paramasivam, 2026](#)). In summary, restoration of tissue perfusion and microcirculatory flow is the fundamental pillar for the return of full intestinal function following anastomosis.

Fluid therapy (quantitative vs. qualitative)

Fluid therapy is one of the most crucial interventions in the treatment of the equine acute abdomen, being essential for maintaining electrolyte and acid–base balance ([Mair et al., 2002](#)). However, it must be emphasized that animals affected by gastrointestinal (GI) disorders, especially those involving the small intestine and requiring ventral midline celiotomy, often present with nasogastric reflux (NGR) and prolonged fasting, leading to reduced maintenance fluid requirements. Evidence indicates that food deprivation in horses reduces water requirements by approximately 16% compared to animals on full feed ([Freeman, 2025](#)).

Consequently, the traditionally calculated maintenance rate (40-60 mL/kg/day) is excessive for horses undergoing postoperative fasting. Administration of this volume may induce iatrogenic hyperhydration, a condition for which the equine organism lacks homeostatic mechanisms as efficient as those developed to manage dehydration ([Freeman, 2025](#); [Alves, 2020](#)). The repercussions of circulatory volume overload are severe and include glycocalyx degradation, as increased cardiac filling pressure stimulates the release of natriuretic peptides by myocytes. These peptides cleave proteoglycans and glycoproteins of the endothelial glycocalyx, resulting in interstitial edema and impaired lymphatic drainage ([Freeman, 2025](#); [Uchimido et al., 2019](#)).

Subsequently, intestinal barrier dysfunction (leaky gut) may develop. When associated with systemic inflammatory response syndrome (SIRS), endothelial damage and increased membrane permeability are exacerbated. In this inflammatory milieu, iatrogenic fluid overload promotes the translocation of pathogen-associated molecular patterns (PAMPs), such as endotoxins, and damage-associated molecular patterns (DAMPs). The breakdown of the mucosal “gatekeeper” function allows these mediators to enter systemic circulation, potentially fueling a self-perpetuating cycle of multi-organ dysfunction and worsening the prognosis of the surgical patient. Another consequence is hypomotility and abdominal hypertension, as disruption of contractility signaling pathways leads to hypomotility, which may progress to abdominal compartment syndrome with intra-abdominal hypertension ([Freeman, 2025](#)).

Concomitantly, hyperhydration may reduce colloid osmotic pressure (COP). This condition is exacerbated by hypoalbuminemia, frequently observed in horses with small intestinal lesions due to protein loss into the intestinal lumen or third spacing into the peritoneal cavity as a result of transmural inflammation. Reduced serum albumin levels compromise anastomotic healing through two primary mechanisms:

- (1) decreased COP promotes fluid extravasation into the intestinal wall interstitial, causing edema at the surgical margins, increasing oxygen diffusion distance and delaying fibroplasia;
- (2) albumin is essential for transporting zinc and other cofactors required for collagen synthesis.

Thus, loop edema induced by hypoalbuminemia and fluid overload acts as both a mechanical and metabolic factor predisposing to dehiscence and anastomotic leakage ([Giusto et al., 2021](#); [Goulder, 2012](#); [Marjanovic et al., 2009](#)). To mitigate these risks, goal-directed fluid therapy (GDFT) and blood gas analysis have been proposed as individualized approaches tailored to the needs of the surgical patient ([Giusto et al., 2021](#); [Marjanovic et al., 2009](#)).

Non-steroidal (NSAIDs) and steroidal (sAIDs) anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs are widely used in equine medicine, particularly in acute abdomen cases, to modulate inflammation and provide analgesia ([Moyseos et al., 2024](#); [Santana et al., 2024](#)). However, understanding their postoperative adverse effects is essential.

The gastrointestinal tract depends on endogenous prostanoids to maintain mucosal blood flow, epithelial integrity, and fermentation in the large intestine, including the production of short-chain volatile fatty acids (SCVFAs). In pathological conditions, prostaglandins are crucial for restoration of the mucosal barrier ([Marshall & Blikslager, 2011](#)).

The potent analgesic effect of certain NSAIDs, such as flunixin meglumine, may delay referral when used indiscriminately, due to masking of clinical signs. In cases involving small intestinal lesions, inappropriate use of non-selective NSAIDs can be detrimental. Flunixin meglumine has been shown to delay mucosal barrier recovery following jejunal ischemia by increasing permeability to lipopolysaccharides (LPS), likely through inhibition of cyclooxygenase (COX) enzymes and prostaglandins E1 and E2, nevertheless, concomitant use of IV lidocaine may minimize this risk ([Marshall & Blikslager, 2011](#)). Furthermore, inappropriate dosing may predispose to compensatory anti-inflammatory response syndrome (CARS), leading to immunosuppression, delayed healing, and increased risk of dehiscence and sepsis ([Taylor, 2011](#)).

In such cases, the use of drugs with lower potency and shorter duration, such as dipyrrone and scopolamine, should be encouraged when no definitive diagnosis has been established or when managed by less experienced clinicians. These drugs are less likely to mask severe pain and therefore do not interfere with clinical judgment or delay referral ([Mair et al., 2002](#)). Dipyrrone acts on COX-3 and stimulates the endogenous opioid system ([Dreyfuss, 2020](#)).

In the postoperative period, although NSAIDs reduce prostanoids involved in SIRS and septic shock ([Gibbs, 2021](#)), prolonged use carries risks such as nephrotoxicity, gastric ulceration, and right dorsal colitis. Multimodal analgesia is recommended, combining synergistic drugs at safe doses, such as xylazine (0.3–1.1 mg/kg IV), butorphanol (0.01–0.02 mg/kg IV), and ketamine (0.3–0.6 mg/kg IM QID) ([Blikslager, 2008](#)).

Steroidal anti-inflammatory drugs (SAIDs)

The use of corticosteroids may be necessary in critically ill patients (e.g., sepsis, shock, SIRS) or those with severe strangulating lesions. Hydrocortisone (4.0 mg/kg) has potent anti-inflammatory effects and may reduce apoptosis in small intestinal loops ([Faleiros, 2011](#); [Gunn et al., 2016](#); [Stuhlmeier & Pollaschek, 2004](#)). A single preoperative dose has not been shown to significantly impair anastomotic healing ([Rushfeldt, 2016](#)). In cases of severe inflammation, characterized by marked edema and inflammatory infiltration, there is an increased risk of stenosis, postoperative ileus (POI), and mechanical obstruction ([Freeman, 2025](#); [Tai et al., 2021](#)).

In such scenarios, especially with ultrasonographic evidence of thickened, hypomotile loops and intraluminal sedimentation, judicious corticosteroid use may be beneficial due to its anti-edematous effects and restoration of luminal patency. However, excessive use must be avoided due to the risks of immunosuppression and impaired healing ([Tai et al., 2021](#)).

Antimicrobials

Colic surgery is generally classified as clean-contaminated. The objective of antimicrobial therapy is to prevent incisional and peritoneal infections and to manage systemic infections in immunocompromised or critically ill patients. However, therapy should be as short as possible to minimize bacterial resistance and dysbiosis ([Dunkel & Johns, 2015](#); [Rockow et al., 2023](#); [Southwood, 2013](#); [Teschner et al., 2015](#)).

While preoperative administration is recommended, caution is required with intraoperative redosing of penicillin. Intravenous potassium penicillin G may induce hypotension in anesthetized horses if

administered rapidly, due to potassium load and histamine release ([Dunkel & Johns, 2015](#); [Rockow et al., 2023](#); [Teschner et al., 2015](#)).

The pathogenesis of anastomotic leakage is multifactorial, involving technical errors, poor tissue perfusion, and local dysbiosis. Dysbiosis favors proliferation of collagenase-producing bacteria or microorganisms that activate matrix metalloproteinase-9 (MMP-9), leading to extracellular matrix degradation.

Anastomotic dehiscence typically occurs between the 3rd and 5th postoperative days, coinciding with peak bacterial collagenase activity and the early phase of host collagen synthesis ([Rosendorf, 2022](#); [Schardey, 2017](#)).

Judicious antimicrobial use enhances anastomotic stability by inhibiting these degradative processes, thereby increasing tissue tensile strength. Thus, antimicrobial therapy plays not only an anti-infective role but also a mechanical protective role for the anastomosis.

Studies suggest that combining routes of administration (e.g., intravenous and oral) may improve tissue resistance at the surgical site ([Schardey, 2017](#)). Conversely, indiscriminate use increases the minimum inhibitory concentration (MIC) and predisposes to diarrhea and severe dysbiosis ([Dunkel & Johns, 2015](#); [Rockow et al., 2023](#); [Teschner et al., 2015](#)). Therefore, treatment should be individualized based on clinical indicators of infection and inflammation.

Indicators for initiating or maintaining antimicrobial therapy:

- Persistent fever or hyperthermia;
- Severe leukocytosis or leucopenia;
- Lethargy and depression;
- Altered mucous membrane color (toxic line);
- Persistent tachycardia;
- Positive culture (e.g., diarrhea or peritonitis);
- Persistent nasogastric reflux (NGR).

Primary technical errors (refining surgical technique)

There are numerous suture patterns for the small intestine, but all must be evaluated to ensure correct applicability (Table 1).

Table 1. Biomechanical comparison of suture patterns for equine jejunojejunostomy

Suture Pattern	Tissue Interface	Collagen Stability (Submucosa)	Impact on Flow (Poiseuille's Law)	Risk of Edge Ischemia	Clinical Recommendation
Simple Appositional	Mucosa-to-Mucosa	High: Preserves original architecture.	Minimal: Maintains maximum loop radius (r).	Low	Ideal for segments with reduced luminal diameter.
Modified Lembert (Interrupted)	Serosa-to-Submucosa	Maximum: Superior anchoring in the tensile layer.	Controlled: Slight inversion compensated by angulation.	Minimal	Gold Standard for equine jejunal anastomosis.
Cushing (Continuous)	Serosa-to-Serosa	Medium: Risk of serosal tearing under pressure.	High: "Ring constriction" effect ($\downarrow r$).	Moderate	Only useful as a second layer if stoma is >30% larger.

The execution of a flawless surgical technique in end-to-end small intestinal anastomosis ([Figure 1A](#)) is the cornerstone of minimizing postoperative complications. The anastomosis must be anatomically and functionally precise, causing minimal interference with intraluminal flow. Therefore, the surgeon must devote rigorous attention to detail during intestinal reconstruction ([Espada, 2025](#)).

Furthermore, determining the resection margins in strangulating obstructions is one of the most critical factors for the viability of the jejunojejunostomy. The phenomenon of “perianastomotic vulnerability” occurs when suturing is performed in tissues with marginal micro perfusion. Although serosal coloration may suggest immediate recovery following decompression, the persistence of

microthrombi and submucosal degradation in transition zones predisposes the site to late-onset dehiscence. A safety margin of at least 10 to 15 cm into macroscopically viable tissue is recommended, ensuring that the anastomosis is performed in loops with vigorous arterial pulsation and absence of severe interstitial edema, thereby mitigating the risk of failure due to secondary ischemia (Espada, 2025).

According to Espada (2025), the highest probability of technical failure lies in over-inversion of the margins or in performing the anastomosis in two suture layers. Frequently, in an attempt to ensure a more hermetic seal against leakage, the surgeon may inadvertently cause stenosis and mechanical obstruction. Freeman (2025) emphasizes that leakage is not the most common complication, precisely due to the heightened concern in preventing it; conversely, obstruction due to excessive tissue inversion is more frequently observed.

When performing a two-layer anastomosis, particularly when the second layer utilizes a Cushing pattern, a "ring constriction" effect occurs, drastically reducing luminal capacity (Freeman, 2025; Espada, 2025). While all continuous sutures can produce this effect, it is more pronounced in the inverting Cushing pattern. On the other hand, interrupted sutures offer greater compliance with the passage of food bolus. The use of a Modified Lembert pattern (sero-submucosal) is recommended, as it combines appositional characteristics with slight inversion, favoring healing (Blikslager & White, 2017; Espada, 2025; Freeman, 2025). It is fundamental that the suture anchors into the submucosal layer, which, being rich in collagen, provides the necessary tensile strength for the suture material (Espada, 2025).

The intestinal wall possesses viscoelastic properties. An anastomosis performed with continuous inverting patterns (such as the Cushing) creates a "rigid ring." When the peristaltic wave reaches this non-compliant ring, a retrograde pressure reflex occurs. Consequently, the oral loop must exert greater pressure to overcome this mechanical barrier (Impedance). If the resistance is too high, the resulting proximal distension reduces intramural perfusion (as discussed in the Perfusion section), triggering a vicious cycle of hypomotility and inflammation (Southwood, 2013). To better understand this, According to Poiseuille's law, and Flow resistance. The resistance to fluid (or ingesta) flow in a cylindrical conduit is inversely proportional to the fourth power of the radius (r). The simplified equation for resistance (R) is:

$$R = \pi r^4 8 \eta L$$

Where:

- η is the viscosity of the ingesta.
- L is the length of the segment.
- r is the luminal radius.

Anastomotic application: Minor reductions in the stomal radius (caused by over-inversion or edema) result in exponential increases in resistance. If the radius is reduced by half, the flow resistance increases approximately 16-fold. Therefore, the recommendation to create a stoma 15% larger (Figure 1B) is a critical safety margin to compensate for postoperative inflammatory edema (Espada, 2025; Freeman, 2025).

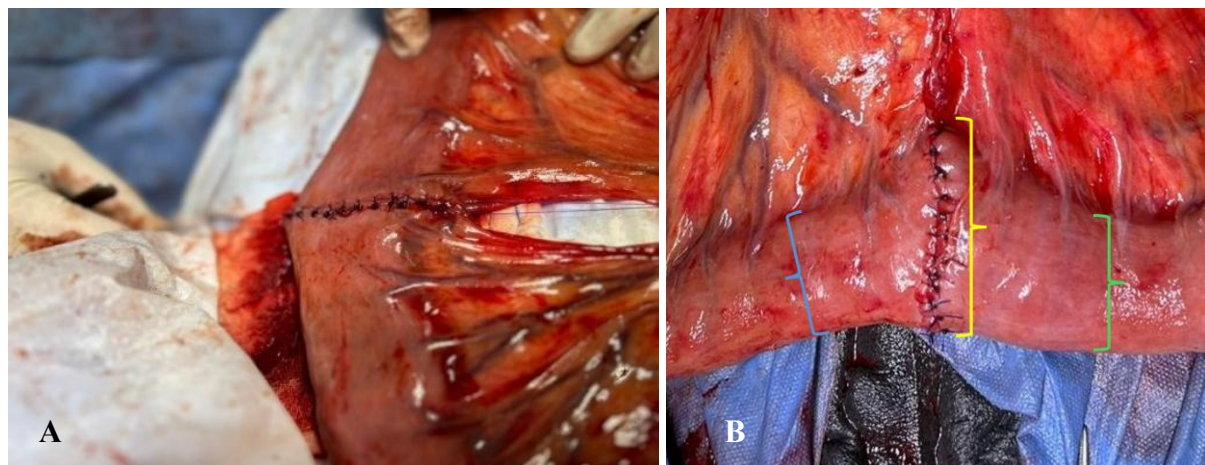


Figure 1. A: End-to-end small intestinal anastomosis. B: Stoma approximately 15% (yellow brace) larger than oral segment (blue brace) and aboral segment (green brace).

Stomal diameter is another critical factor. Considering that only the mucosa regenerates, while the remaining layers heal through fibrosis, the surgeon must anticipate the space occupied by edema and

the proliferative phase of healing (Blikslager & White, 2017; Espada, 2025). The ideal stoma should have a lumen approximately 15% larger than the adjacent segments. Thus, after scar contraction, the final diameter will approximate that of normal loops.

To achieve this dimension, the incision should be angled at 60° relative to the mesenteric border, and stay sutures should be placed at both the mesenteric and antimesenteric borders. These measures allow greater luminal expansion and help prevent suture line sinuosity caused by peristalsis (Espada, 2025). The use of crushing forceps should be minimized, as they increase tissue inflammation and restrict border extension.

In cases of luminal disparity between loops, the most prudent approach is to maintain the 60° resection angle and enlarge the smaller lumen via an antimesenteric border incision (spatulation), avoiding acute angles that predispose to leakage (Espada, 2025; Freeman, 2025).

Functionality also depends on proper organization and decompression of the small intestine prior to anastomosis, in order to prevent postoperative torsion and ischemia. Regarding the mesentery, three primary technical failures can lead to complications. The first is inappropriate vessel selection (“vascular nutrition”), which should be close to a primary jejunal arcuate artery (maximum distance of 8–10 cm), with similar calibers at both ends. The second involves errors in mesenteric closure that create traction on the intestinal loop (“mesenteric shortening”), resulting in extrinsic obstruction, pain, and reflux (Blikslager & White, 2017; Espada, 2025; Freeman, 2025). The third is incomplete closure or traumatic lacerations, which create defects (“mesenteric rents”) that predispose to loop incarceration (internal herniation), potentially causing immediate or delayed recurrence (Espada, 2025; Freeman, 2025).

Another common technical error is failure to identify lesions within the abdominal cavity due to incomplete exploration. Although not directly related to small intestinal anastomosis, this issue should be acknowledged. Therefore, thorough inspection of the entire abdominal cavity is strongly recommended during surgery.

Postoperative period: Differential diagnosis and refeeding

1. POI vs. Mechanical obstruction

In the postoperative period, it is essential to differentiate functional obstruction from mechanical obstruction (Table 2). Surgical manipulation of intestinal segments can induce transmural inflammation in both manipulated and non-manipulated segments (Freeman, 2018). However, proper decompression of the small intestine during surgery is critical to prevent postoperative reflux (POR) (Freeman, 2018; Freeman, 2025). Surgeons’ concern about inflammation as a cause of POR may lead to inadequate decompression of the small intestine during surgery. Consequently, distended loops may become kinked, resulting in POR (Freeman, 2018). Therefore, leaving small intestinal loops distended is more detrimental than performing appropriate and effective decompression.

Table 2. Differential criteria: POI vs. Mechanical obstruction

Criterion	Postoperative Ileus (POI)	Mechanical Obstruction
Response to Intubation	Stabilization of HR and pain following decompression.	Continuous cardiovascular deterioration (↑HR, ↑CRT).
Clinical Signs	Progressive improvement under medical therapy.	Persistence or worsening of abdominal pain.
Response Time	Resolution in approx. 36–48h PO with adequate therapy.	Absence of response within 36–48h.
Ultrasonography	Hypomotility with moderate distension.	Severe distension and ↑ free peritoneal fluid; may show collapsed SI (aboral to obstruction).

A period of dysmotility is physiologically expected following celiotomy for acute abdomen, resulting from tissue manipulation, decompression, and localized inflammation (Blikslager & White, 2017; Freeman, 2025). However, when this condition progresses to postoperative ileus (POI), severe functional paralysis may occur, accompanied by persistent postoperative reflux (POR), defined as a volume exceeding 2 L/h or more than 4 L in a single intubation (Blikslager & Merritt, 2008).

The clinical challenge lies in the fact that POR may be a manifestation of either functional failure (POI) or mechanical obstruction (e.g., stenosis, impaction, or anastomotic torsion). Early differentiation is essential: POI primarily responds to medical management, whereas mechanical obstruction typically requires repeat celiotomy (Freeman, 2025).

The decision to pursue surgical re-intervention must be based on critical assessment of the initial technique, including the number of suture layers, stomal diameter, and degree of tissue inversion. Early re-celiotomy (≤ 48 h) is associated with significantly higher success rates compared to delayed intervention (Freeman, 2025).

2. Impaction vs. Stenosis

Anastomotic impaction is more closely linked to the stoma's inability to permit flow than to the timing of refeeding. A fasting horse may suffer impaction if dehydrated gastric content migrates into a stenotic stoma (Freeman, 2025). Conversely, stenosis itself results from technical errors, such as over-inversion, excessive edema, hematomas, or mural lesions at the anastomotic "mouth" *Bernoulli's Principle* describes the relationship between fluid velocity and intraluminal pressure. In a stenotic (narrow) segment, the velocity of the ingesta increases while lateral pressure decreases. However, in horses, intestinal content is not an ideal fluid; it possesses high viscosity and fiber. When this content encounters a stenosis or an acute angle (caused by a failure in the 60° angulation), a loss of kinetic energy occurs, transforming laminar flow into turbulent flow. This favors the sedimentation of solid particles and the formation of impactions at the "step" created by the suture (Blikslager & White, 2017; Espada, 2025).

3. Prevention of postoperative adhesions

Adhesion formation is one of the most critical and frustrating complications following small intestinal resection and anastomosis, leading to recurrent obstruction and chronic pain (Blikslager & White, 2017). There are three main types of adhesions: they are epiploic/omental, fibrinotic and fibrotic. Surgical trauma and serosal exposure lead to fibrin deposition; if local fibrinolytic activity is insufficient, this matrix is invaded by fibroblasts, resulting in permanent fibrous adhesions. To mitigate this risk, the use of physical and chemical barriers has become standard in surgical routine. The intra-abdominal application of 1% Sodium Carboxymethylcellulose (CMC), acts as a hydrophilic "sliding layer" between intestinal loops, preventing direct contact between inflamed surfaces during the critical healing period (Freeman, 2025). In addition to CMC, the use of solutions containing sodium hyaluronate combined with carboxymethylcellulose has demonstrated superior efficacy in reducing adhesion severity in jejunal segments (Southwood, 2013). It is fundamental for the surgeon to understand that these substances do not replace atraumatic technique and rigorous hemostasis but serve as essential adjuvants in preserving intestinal mobility and reducing long-term morbidity (Blikslager & White, 2017; Freeman, 2025).

Literature suggests the use of low – molecular weight heparin (LMWH) via subcutaneous or intramuscular routes (40-100 IU/kg, BID or TID), starting pre- or intraoperatively. However, its use requires rigorous monitoring, as it may predispose to incisional hematomas and, at high doses, erythrocyte agglutination with a subsequent drop in packed cell volume (PCV) (Sullins, 2004). The combined use of physical barriers (CMC) and chemical modulation (heparin) offers synergistic protection against undesired fibroplasia, though it does not supersede the need for an atraumatic surgical technique (Blikslager & White, 2017). Other mechanical approaches to prevent adhesions are the performance of intraoperative peritoneal lavage with balanced crystalloid solutions to remove cellular debris and peritoneal clots and omentectomy to prevent epiploic adhesion (omental).

4. Motility modulation: Prokinetic agents

Postoperative prokinetic therapy aims to shorten the phase of intestinal adynamia and reduce the risks of impaction and luminal distension. First, the most effective and natural prokinetic is appropriate oral intake. Even in cases of POR, this can be cautiously stimulated while monitoring gastric distention via ultrasound; if necessary, gastric lavage and decompression should be performed. Lidocaine (continuous infusion with a loading dose of 1.3 mg/kg followed by 0.05 mg/kg/min) stands out not only for its analgesic and anti-inflammatory properties but also for its ability to reduce POI duration and nasogastric reflux (NGR) volume (Malone et al., 2006). Its mechanism involves sodium channel blockades and the suppression of inhibitory sympathetic activity, while also exerting a cytoprotective effect on the mucosa (Blikslager & White, 2017; Cook & Blikslager, 2008). Despite recent studies questioning its efficacy in postoperative colic (Sousa et al., 2024), it remains widely utilized in equine practice. Additionally, Metoclopramide acts as a 5-HT₄ receptor agonist and dopamine (D₂) antagonist, stimulating acetylcholine release in the myenteric plexus, with more pronounced effects on the proximal

GI tract (stomach and duodenum) ([Okamura et al., 2009](#)). Although its use requires caution due to potential extrapyramidal effects and central nervous system excitation, metoclopramide remains a valuable alternative in cases of severe gastroparesis and persistent reflux. The strategic combination of prokinetics with distinct mechanisms of action, integrated into a multimodal analgesia protocol, optimizes the return of motor function and favors mechanical stability of the SI anastomosis ([Cook & Blikslager, 2008](#)).

5. Endothelial glycocalyx (EG) integrity and anastomotic edema

The mechanical stability of the anastomosis intrinsically depends on the integrity of the endothelial glycocalyx (EG), a complex network of proteoglycans, glycosaminoglycans, glycoproteins and adherent plasma protein that serve as the primary determinant of the vascular permeability barrier ([Woodcock & Woodcock, 2012](#)). In cases of jejunal strangulation, ischemia-reperfusion injury and Systemic Inflammatory Response Syndrome (SIRS) trigger the degradation of this barrier, resulting in massive fluid extravasation into the intestinal wall interstitial ([Southwood, 2013](#)). This phenomenon is frequently exacerbated by purely quantitative fluid therapy protocols; volumetric overload increases hydrostatic pressure and dilutes serum albumin, promoting the mechanical destruction of the glycocalyx and culminating in severe edema at the anastomotic margins. The increased wall thickness resulting from this edema not only reduces the effective stomal diameter (according to Poiseuille's Law) but also compromises the anchoring strength of sutures in the submucosa, predisposing the site to dehiscence and POI. Therefore, EG protection through goal-directed fluid therapy and the maintenance of oncotic pressure are fundamental pillars to ensure microvascular perfusion and suture line viability ([Blikslager & White, 2017](#); [Freeman, 2025](#)).

The judicious administration of hydrocortisone has shown potential in stabilizing the EG by inhibiting mast cell activation and matrix metalloproteinase release, thereby reducing the shedding of components such as syndecan-1 ([Chappell et al., 2009, 2010](#)). Furthermore, restoring colloid osmotic pressure through albumin or plasma supplementation not only reduces mural edema but also exerts a direct protective effect on the glycocalyx structure, mitigating leukocyte activation and preserving endothelial barrier function ([Woodcock & Woodcock, 2012](#)). Additionally, LMWH can be utilized to protect glycocalyx from degradation during sepsis, as it acts as a heparanase inhibitor ([Uchimido et al., 2019](#)).

6. Early enteral refeeding

Historically, prolonged fasting was recommended to "rest" the anastomosis. However, current evidence demonstrates that early enteral nutrition is essential to preserve villous morphology, stimulate local immunity, and reduce negative nitrogen balance ([Thapa, 2011](#); [Watson, 2015](#)). Nutritional deprivation, conversely, delays healing and increases the risk of incisional complications.

Refeeding strategy:

- Initial Oral-Hydration (6-8h PO): Offering water to hydrate sedimented gastric content and activate intrinsic motility signals, reducing the risk of stomal impaction ([Freeman, 2025](#)).
- Mechanical Stimulation: Hand-walking assists in promoting motility.
- Controlled Grazing (~8h PO): Small amounts of fresh grass, under ultrasonographic monitoring to rule out gastric distension or NGR.
- Lipid Supplementation: The use of oils (e.g., fish or flaxseed) provides caloric density and may aid in inflammatory modulation ([Watson, 2015](#)).

7. Physiology and biomechanics: The three phases of anastomotic healing

Anastomotic integrity depends on a harmonious transition between biological stages of tissue repair, with the submucosa being the only component with sufficient collagen density to ensure suture anchorage ([Southwood, 2013](#)). First, the *Lag or Inflammatory Phase* (Days 0-4) Immediately post-suture, the site relies exclusively on the mechanical support of the suture material. During this phase, the fibrin clot forms and neutrophils and macrophages migrate. This is the highest risk period, as the lysis of old collagen by matrix metalloproteinases (MMPs) outpaces new collagen production, creating a "vulnerability window" where the tensile strength of the union decreases drastically ([Schardey, 2017](#)). The second is *Proliferative or Fibroplasia Phase* (Days 5-14), characterized by intense fibroblast

migration and angiogenesis. Type III (immature) collagen is deposited, and anastomotic strength begins to increase rapidly, reducing total reliance on the suture. Finally, the last phase is *Remodeling (Maturation) Phase*, where type III collagen is gradually replaced by Type I. Collagen fibers realign along mechanical stress lines, restoring the functionality and elasticity of the intestinal segment (Gershuni & Friedman, 2019).

Final considerations

The success of small intestinal anastomosis in horses transcends manual technical skill, residing in a profound understanding of intestinal pathophysiology and the rigorous mitigation of perioperative risk factors. "Parasitic time" and delayed surgical referral remain the primary determinants of failure, as prolonged ischemia triggers microcirculatory damage and mucosal degradation that no surgical technique can fully reverse. Modern clinical-surgical thought points toward the necessity of refined hemodynamic and pharmacological management. This includes transitioning from purely quantitative fluid therapy to a goal-directed approach, aiming to protect glycocalyx integrity and avoid hypoalbuminemia, factors which, if neglected, lead to mural edema and healing failure. Similarly, the strategic use of antimicrobials and anti-inflammatories must balance the need to control SIRS without compromising mechanical stability, especially regarding collagen preservation and the prevention of iatrogenic stenosis due to excessive tissue inversion. Ultimately, maximizing survival in jejunojejunostomy cases requires the surgeon to act not merely as a technical executor, but as a critical manager of patient homeostasis, grounding every decision in scientific evidence and continuous clinical vigilance.

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