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MEDICAL TREATMENT OF HORSES WITH COLIC

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The cornerstones of medical treatment of horses with abdominal pain include withholding food and possibly water, administration of analgesic drugs, antispasmodics, administration of IV or oral fluid therapy, administration of laxatives via nasogastric tube, and controlled exercise. In some cases, it is necessary to administer medications to enhance or modify intestinal motility. This discussion will focus on laxatives and methods and drugs to modify intestinal motility.

ANALGESIC THERAPY

Analgesic therapy is an important component of medical treatment of horses with colic. Horses may not only cause self trauma or injure people when experiencing abdominal pain, but pain also causes release of catecholamines which can have inhibitory effects on intestinal motility.

The most common analgesic medications used in horses with abdominal pain are the nonsteroidal anti-inflammatory drugs (NSAIDs), • "$_2$ agonists, and opioids. NSAIDs are the principle analgesics drugs used to control abdominal pain; flunixin meglumine appears to have the superior inhibitory effect on visceral pain; however, ketoprofen (2.2 mg/kg, IV once daily) also apparently is useful to control abdominal pain. Phenylbutazone has the least analgesic effect for pain associated with colic. The recommended dose of flunixin meglumine is 1.1 mg/kg IV or IM once or twice daily. It is important to remember if a horse does not respond to the first dose of flunixin meglumine it is not likely to respond to multiple doses. However, it will increase the likelihood of toxicity. There is the potential concern of masking abdominal pain associated with a surgical colic when flunixin meglumine is administered. If flunixin meglumine is administered at the recommended dose and frequency and the horse is closely examined (including complete physical examination, rectal examination, passage of nasogastric tube) and monitored, then it is unlikely that a surgical disease (or at least one that is an emergency) will be masked. Repeated administration of flunixin meglumine without re-examining the horse can certainly mask appreciable pain associated with a surgical disease. Lower doses of flunixin meglumine can be used for their anti-inflammatory effects and to ameliorate the effects of endotoxin. Dosages of 0.25 to 0.5 mg/kg are administered IV every 6-8 hours. Phenylbutazone has also been shown to be more effective than flunixin meglumine in ameliorating the effects of endotoxin on intestinal motility, and may be added to the treatment regimen at a dose of 0.5-2.2 mg/kg twice daily. Caution should be used in administering as

combination of NSAIDs, and particular attention should be given to hydration status to prevent deleterious toxic effects on the gastrointestinal tract and kidneys.

The • "$_2$ agonists commonly used in horses with abdominal pain include xylazine and detomidine. Xylazine is commonly used in horses to control moderate to marked abdominal pain for relatively short periods (20-30 minutes). This may be helpful when passing a nasogastric tube or performing a rectal examination. The heart rate should be evaluated prior to administering these drugs because of their bradycardic effect. Xylazine is often administered at a dose of 0.2 to 0.5 mg/kg IV, which will provide potent analgesia for 20-30 minutes or 0.6 to 1.0 mg/kg IM, which will prolong the analgesic effects for approximately 1-2 hours. Detomidine is an "• $_2$ agonist with more potent analgesic effects than xylazine; it has the potential to mask abdominal pain associated with a surgical disease for extended period, particularly if administered repeatedly. It should probably be reserved for horses with marked abdominal pain in which surgical exploratory is not an option or those which must be transported long distances to a referral hospital for surgical intervention. The dose of detomidine is approximately 0.01-0.02 mg/kg IV or IM. The "• $_2$ agonists can have inhibitory effects on gastrointestinal motility, which can be deleterious in horses being treated for intestinal impactions.

Butorphanol tartrate is an opiate agonist-antagonist that is useful in providing analgesia for horses with moderate to marked abdominal pain. It is most often administered (0.01-0.02 mg/kg, IV) in combination with xylazine (as it can result in excitement IV if administered alone) or by itself (0.02 to 0.1 mg/kg, IM). A continuous rate infusion dose of 13 • g/kg/hour has been reported to decrease cortisol concentration and improved recovery characteristics when administered to post-operative colic patients compared to horses not receiving butorphanol. In the study however, butorphanol was found to significantly delay the time to first passage of feces. Therefore judicious use of butorphanol is warranted as deleterious effects on intestinal motility may occur if administered at high doses or repeatedly.

ANTISPASMODIC MEDICATION

Treatment of spasmodic colic can be aided by the use of anti-spasmodic. N-butylscopolammonium bromide (Buscopan) has anticholinergic and antispasmodic properties. Although commercially available in Europe for many years (in combination with hyoscine) it has been fairly recently approved for use in the United States (without hyoscine). When administered to ponies in a cecal balloon distention study, a brief analgesic effect and a transient decrease in cecal contractions were observed. A typical dose is 0.3 mg/kg IV once. It is important to evaluate the heart rate prior to administration as due the drug's parasympatholytic response increase after administration has been noted. The administration of 3 ml IV to 1000 lb horse can also be useful for relaxation during the rectal examination. Buscopan's effects are relatively brief, lasting for approximately 30 minutes, and therefore should not have a significant masking effect in horses with surgical lesions.

INTRAVENOUS FLUID THERAPY

Many horses with abdominal pain (colic) require IV fluid therapy to correct dehydration, provide maintenance requirements in those that cannot be allowed to drink (gastric reflux), prevent dehydration in horses with excessive water losses (enterocolitis or gastric reflux), and to increase intestinal luminal water content in horses with impactions.

The volume and content of the IV fluids depends upon the magnitude of dehydration, the expected, measured, or estimated volume of water loss (quantify volume of reflux), and whether or not the goal is to slightly "overhydrate" the horse in an attempt to increase intestinal luminal fluid content. The estimated percentage dehydration can be based upon clinical signs

(heart rate, mucous membrane color, capillary refill time, skin turgor) and clinicopathologic abnormalities (packed cell volume, total plasma protein, creatinine). Fluid volume deficits are calculated in liters by multiplying the estimated percentage dehydration by the body weight in kilograms. For example, a 450 kg horse that is 6% dehydrated would require approximately 27 liters to correct the fluid deficit. Maintenance fluid requirements for the adult horse are approximately 40-60 ml/kg/day or approximately 24 liters per day. Overhydration achieved by administering approximately twice maintenance levels can be useful for softening impactions of the cecum and large colon, but may be detrimental in horses with small intestinal ileus because it promotes sequestration of gastric fluid.

The composition of the fluids to be administered should be selected based upon the most likely fluid and electrolyte needs and upon results of a chemistry profile. Frequently, a balanced polyionic fluid, such as lactated or acetated Ringers, is appropriate. However, sometimes it is necessary to administer other types of fluids. The most common electrolyte abnormalities that develop are hypokalemia and hypocalcemia; these are often exacerbated by administration of high volumes of IV fluids, particularly in horses that are not allowed to eat. Horses with gastric reflux often develop hypochloremic metabolic alkalosis; these horses should probably be administered 0.9% NaCl with KCl. The IV fluids may need to be supplemented with various electrolytes and bicarbonate. Because potassium is predominantly an intracellular ion, it is difficult to estimate a potassium deficit from plasma concentrations. However, horses that are not eating should be routinely supplemented with 10-20 mEq/L of potassium (KCl) per liter of IV fluids.

Calcium is important for mediating vascular and intestinal smooth muscle contractility, which affects intestinal blood flow and motility. Measurement of ionized calcium can be used to determine if supplementation is needed. Endotoxemia, sepsis and diarrhea will result in low serum calcium concentrations, and one report found 88% of all colic patients to have ionized calcium levels below normal at admission. This study also found that hypocalcaemia was prognostic for survival and the development of ileus, with correction improving clinical outcome. Horses should be supplemented with 5 to 20 ml of calcium gluconate (23%) per liter of fluids. Greater quantities can be administered, but should be done so cautiously and with close monitoring. Magnesium is also believed to be important for the maintenance of normal intestinal motility and function. Ionized magnesium can be used to measure serum magnesium concentrations; however, the majority of magnesium is intracellular and therefore may not be an accurate assessment of total body magnesium. Several reports have found between 44-54% of horses with gastrointestinal disease were hypomagnesemic. Furthermore, hypomagnesaemia can result in hypocalcaemia secondary to inhibition of parathyroid hormone release. This will result in the inability to correct hypocalcaemia without correcting hypomagnesemia. Hypomagnesemia should be corrected in horses that are symptomatic (intestinal ileus, cardiac arrhythmias) by administering 100-200 mg/kg/day (50-100 grams to 450 kg horse) of $MgCl_2$ or $MgSO_4$ IV or PO. This should be administered throughout the day rather than as a bolus treatment. Horses with hypomagnesaemia that are asymptomatic may be supplemented with Mg by administering 60-70 mg/kg/day (30 grams to 450 kg horse) of $MgCl_2$ or $MgSO_4$ IV or PO.

In some instances, sodium bicarbonate may be required to help correct metabolic acidosis. This treatment is usually reserved for horses with a base deficit greater than 10 mEq/L. The base deficit can be calculated from the blood gas analysis or can be estimated by subtracting the plasma bicarbonate or total carbon dioxide from the normal value of approximately 25. The bicarbonate deficit is calculated using the following formula:

Base deficit x 0.3 x body weight (kg) = mEq HCO_3^- deficit

Half the deficit should be replaced rapidly, and the remaining deficit replaced over 12-24 hours. It is also important not to supplement sodium bicarbonate in calcium containing fluids.

It is critical to closely monitor horses that are receiving IV fluids to make sure they do not develop problems with the catheter and that they receive an adequate volume to keep up with water losses. A written record should be kept on the “colic flow sheet” of the volume of net reflux that is obtained to help assure that total water losses are replaced throughout the course of the day. Hydration can be monitored by clinical signs, PCV and total plasma protein. Development of specific electrolyte abnormalities should be monitored with chemistry profile analyses.

ENTERIC TREATMENT

The least expensive enteric treatment for horses with impaction colic is water. It can be administered via nasogastric tube (NGT) at approximately 6 to 8 liters every 2 hours in an adult horse. Since water is a hypotonic solution with very low concentrations of electrolytes, monitoring of plasma electrolyte concentrations and/or supplementation may be indicated particularly if administered for greater than 24 hours or if the horse has renal insufficiency. NGT can be secured and appropriate personnel can be instructed on how to administer the water. This can help maintain hydration and assist in increasing intestinal luminal water content in horses that have no gastric reflux.

The most commonly used laxatives include magnesium sulfate, dioctyl sodium succinate (DSS), mineral oil, and bulk laxatives such as psyllium hydrophilic muciloid. Magnesium sulfate is an osmotic cathartic that is usually administered to horses at a dose of 1 g/kg every 1 to 2 days; administration should probably be delayed until rehydrated to prevent exacerbation of dehydration. Excessive doses could cause signs of magnesium toxicity, enterocolitis, and dehydration. Aspiration of magnesium sulfate can also cause pulmonary edema. DSS is a surface acting agent that decreases surface tension and allows fluid to be imbibed into the impacted material; it also stimulates intestinal secretions. It is usually administered at a dose of 10 to 30 mg/kg as a 10% solution. Excessive doses can lead to intestinal irritation and dehydration. DSS should not be administered to horses with sand impaction because it may cause sand to become more solidified. Mineral oil is not absorbed from the intestinal tract and thus serves as an intestinal lubricant. It is usually administered at a dose of 2 to 4 liters per 450 kg horses every 1 to 2 days. Mineral oil should be administered to horses with feed impactions to lubricate the ingesta and facilitate its passage after magnesium sulfate has been given to help draw water into the intestinal lumen and soften the impaction. The bulk laxatives such as psyllium hydrophilic muciloid or bran are most useful to help prevent impaction colic. Although psyllium is frequently administered to horses with colic suspected to be associated with sand impactions, a controlled experimental study revealed that it did not facilitate removal of sand from the large intestine.

MOTILITY MODIFIERS

Motility modifiers are principally used in horses to prevent or treat postoperative ileus (POI); however, they are occasionally used to facilitate passage of feed impactions once they are softened with appropriate medical treatment. This discussion will primarily focus on drugs used in treatment and prevention of POI.

Postoperative ileus (POI) is a relatively common complication encountered in horses following surgery for gastrointestinal disease. It has been reported that POI occurs in approximately 16% of horses undergoing abdominal surgery for colic; it occurs in about 25% of horses with small intestinal and 12% with large intestinal disease, respectively. Horses with evidence of shock, dehydration, and small intestinal obstruction with or without ischemia are at risk for development of POI. Postoperative ileus can occur secondary to dynamic (spastic),

adynamic (paralytic), or mechanical (occlusive) obstruction of the intestinal tract; however, it typically occurs owing to a loss of coordinated progressive motility of the stomach and small intestine. This results in the accumulation of large volumes of fluid, which leads to abdominal pain owing to gastric and small intestinal distention. The abdominal pain can contribute to a vicious cycle of ileus. Postoperative ileus is more common in horses with ischemic or inflammatory disease of the small intestine, but can occur in horses with any type of gastrointestinal disease. The pathophysiology of POI is not completely understood, but is multifactorial. Visceral pain, dehydration, electrolyte imbalances, ischemia, intestinal inflammation, intestinal obstruction, peritonitis, endotoxemia, bowel trauma, and bowel distention are believed to be contributing factors to the development of POI. These factors may act through adrenergic (sympathetic) hyperactivity, cholinergic (parasympathetic) hypoactivity, and dopaminergic hyperactivity. This discussion will focus on gastric and small intestinal ileus.

Intestinal motility proximal to experimentally-induced jejunal obstruction in ponies is markedly increased. These intestinal spasms occur immediately after the onset of the obstruction indicating it is likely a response to localized intestinal irritation. This increase in intestinal motor activity proximal to an obstruction is mediated via cholinergic nerves. After a period of intestinal obstruction, borborygmi decrease; this is believed to be the result of luminal distention and interstitial edema, which together inhibit gastrointestinal smooth muscle contractile activity. Gastrointestinal distention associated with an obstruction leads to signs of abdominal pain. Abdominal pain associated with gastric/ intestinal distention and peritonitis can contribute to POI via stimulation of the sympathetic nervous system.

Intestinal ischemia alters gastrointestinal motility. Complete arterial occlusion of the equine small intestine causes an immediate decrease in contractile activity. It has been postulated that local intrinsic nerves mediate this response. It is more likely that the early changes in gastrointestinal motility during ischemia are associated with disturbances in electrolyte balance across the intestinal smooth muscle membranes. Duration of intestinal ischemia is a critical factor in the restoration of motility after the intestine is reperfused. Complete ischemia for up to 3 hours does not prevent the return of normal contractile activity after restoring blood flow in the dog small intestine. However, after 4 hours of total ischemia there is a loss of normal motility after reperfusion, as characterized by decreased frequency of the BER, which initiates the spike potentials. Prolonged ischemia may alter the permeability of the sarcolemma to electrolytes, which could disable enzyme-mediated membrane pumps required to maintain membrane potential. Loss of intestinal motility could be due to altered smooth muscle excitability, impulse conduction, and excitation-contraction coupling. Derangements in potassium, calcium, and chloride could contribute to POI. The myenteric plexus can become damaged with prolonged ischemia resulting in asynchronous smooth muscle contractions.

It has been shown that endotoxin produces profound inhibition of gastrointestinal motility. Prostaglandin synthesis can also affect gastrointestinal motility, which is probably mediated locally rather than systemically. Prostaglandins are released during inflammation and may contribute to POI; they cause stimulation of longitudinal muscle and either stimulation or inhibition of circular smooth muscle. Endotoxin-induced ileus can be reproduced by infusion of prostaglandin E_2. This effect of endotoxin on motility is completely blocked by NSAIDs. In general, prostaglandins of the E and I series have inhibitory effects on bowel motility, whereas F-series prostaglandins are stimulatory.

Stimulation of the visceral and parietal peritoneum by trauma, nonseptic inflammation, or septic inflammation can inhibit gastrointestinal motility. Stimulation of the peritoneum stimulates autonomic reflexes that inhibit motility. This inhibition occurs predominantly in the stomach and colon. It appears that spino-vagal pathways are involved in this reflex ileus. Because inhibition of gastric motility is not affected by adrenergic or cholinergic antagonists,

non-adrenergic vagal fibers that inhibit the release of acetylcholine are involved.

Because the pathophysiology of POI is multifactorial there is no one treatment or medication that reliably resolves it. Therefore, multimodal therapy is necessary. Gastric decompression is vital to decrease abdominal pain associated with gastric distention, prevent gastric rupture, and interrupt the pain-ileus cycle. Passage of an NGT is imperative in horses with POI. An indwelling NGT can be maintained or intubation can be repeated as necessary. Controlled exercise may help stimulate motility.

Hydration, electrolyte, and acid-base status should be evaluated and any abnormalities corrected. Particular attention should be given to potassium, ionized calcium, and chloride concentrations. Ionized calcium is responsible for evoking the action potential of visceral smooth muscle; hypocalcemia can result in decreased gastrointestinal motility. If serum ionized calcium is low or is suspected to be low, calcium gluconate can be given IV at a dose of 100 to 300 milliliters of a 23% solution. The solution can be administered as a slow drip or can be mixed with IV fluids (10 ml/L). Hypokalemia may predispose horses to POI and is often present in horses in the postoperative period because of the diuretic effect of IV fluids and the lack of food intake. Potassium chloride can be supplemented in IV fluids safely at a dose of 20 mEq/L. The maximum safe rate for IV potassium administration is 0.5 mEq/kg/hr. Postoperative ileus has been shown to be associated with hypochloremia, which may be secondary to sequestration of chloride in the stomach. Chloride is required for the release of calcium from the sarcoplasmic reticulum and, therefore, can affect gastrointestinal motility. Normal hydration status should be maintained, but excessive fluid should not be administered. Hypochloremic metabolic alkalosis often accompanies disease of the small intestine owing to loss of hydrogen and chloride ions in gastric reflux; these horses should probably be treated with 0.9% NaCl supplemented with KCl.

It is important to control pain to interrupt the pain-ileus cycle; however, the use of certain analgesic agents could adversely affect gastrointestinal motility. Xylazine and butorphanol are used commonly in horses with colic for their analgesic properties; however, they both can have deleterious effects on motility. Both xylazine and butorphanol prolong the migrating myoelectric complex in the small intestine and appear to have additive effects when used together. It is probably more indicated to provide visceral analgesia in the postoperative period with flunixin meglumine or another NSAID. Flunixin meglumine can be administered at a 1.1 mg/kg BID or 0.25 to 0.5 mg/kg TID-QID. The lower dose rate has been shown to be effective in ameliorating the effects of endotoxin on the cardiopulmonary system and has minimal deleterious effects on gastrointestinal mucosa and renal papilla. Phenylbutazone is not as commonly used as flunixin meglumine, but has been shown to be more effective than flunixin meglumine in ameliorating the deleterious effects of endotoxin on gastrointestinal motility. Administration of phenylbutazone (2.2 mg/kg IV BID) and flunixin meglumine (0.25 mg/kg IV TID) may be useful in horses with POI, but it is imperative to maintain adequate hydration to minimize risk of NSAID toxicity.

Cisapride is an indirect cholinergic prokinetic agent that acts by promoting the release of acetylcholine from intramural nerve terminals (myenteric plexus) through stimulation of 5-HT_4 receptors. It may also antagonize peripheral dopamine receptors. It augments gastric contractions, stimulates jejunal activity coordinated with gastric contractions, enhances contractile activity of large and small colon, and stimulates coordinated activity in the ileocecocolic junction. It has been shown to increase gastric contractions, increase phase 2 activity in the small intestine, and restore motility after experimentally-induced and naturally-acquired POI. It is often administered to horses in which there has been severe small intestinal injury. In a clinical trial in horses, cisapride (0.1 mg/kg IM every 8 hours) significantly decreased the incidence of POI; many of the horses that did not respond had peritonitis or bowel ischemia. Because only tablets are available, they are dissolved in a small volume (20

ml) of DMSO and administered at a dose of 0.1 to 0.25 mg/kg per rectum. In one hospital, it is administered at 0.4 mg/kg once the lesion has been corrected at surgery and then is repeated every 2 hours until a response is observed. The frequency of administration is then decreased by 2-hour intervals providing the horse continues to respond. It is unknown whether therapeutic levels are achieved with this dosage regimen; however, the clinicians have the impression that it is beneficial. There have been no apparent side effects observed with this treatment protocol. If motility disturbances have not been corrected within 48 hours then other drugs may be added to the therapeutic regimen.

Lidocaine has been used for treatment of POI in humans and horses. When administered to humans undergoing elective cholecystectomy IV at surgery and continued for 24 hours postoperatively, lidocaine improved propulsive motility and decreased the requirement for narcotic administration for pain control. Lidocaine has been postulated to work primarily by removing sympathetic inhibition to the bowel by blocking afferent sympathetic input (which is inhibitory to motility). It also has direct stimulatory effects on intestinal smooth muscle, relieves pain, and reversibly modulates the immune system by decreasing neutrophil activation and lysosomal enzyme release. Furthermore, the combination of lidocaine and flunixin meglumine administered post-operatively in an experimental model of jejunal ischemia found improved mucosal barrier function/recovery compared to flunixin alone. This may allow for less endotoxin to cross into systemic circulation along with improving pain control in clinical cases. A bolus (1.3 mg/kg) of lidocaine is administered and then a continuous IV infusion (0.05 mg/kg/min) is initiated immediately and continued for 24 to 36 hours. The rate of administration may need to be altered if signs of toxicity appear (muscle fasiculations, bradycardia, seizures, ataxia and collapse). The quantity of reflux usually decreases or stops within 24 to 36 hours if it is going to respond to lidocaine. Although no increase in intestinal motility was seen after lidocaine administration in a study with normal horses, other studies examining the efficacy in horses with POI have found up to 83% of horses stopped refluxing within 24 hours of starting lidocaine infusion. Furthermore, lidocaine is purportedly the most commonly administered prokinetic agent used for the treatment of POI according to a survey of the American College of Veterinary Surgeons. Lidocaine may also be administered intra-operatively to not only decrease the amount of inhalant needed to maintain anesthesia but also to decrease the development of POI, as administration may have resulted in a reduction of POI from 19% to 9% in one study.

Metaclopramide stimulates intestinal motility by stimulating the release of acetylcholine from postganglionic nerve terminals, blocking inhibitory dopaminergic-sensitive receptors and also by blocking adrenergic activity. There is also some evidence that metaclopramide may have a direct effect on the intestinal smooth muscle. Dosage rates are variable (0.1 to 0.25 mg/kg/hr as a continuous IV infusion). It has been shown to improve motility in an experimentally-induced model of POI in ponies when administered at a dose of 0.25 to 0.50 mg/kg/hr. In a clinical study, metaclopramide (0.04 mg/kg/hr) decreased the total volume, duration, and rate of gastric reflux when administered prophylactically after small intestinal resection and anastomosis. Because metaclopramide causes extrapyramidal side effects such as CNS stimulation (excitement, restlessness, and sweating) caution should be used when administering this drug to horses. The use of metaclopramide is variable depending on clinicians' experience and preference.

Erythromycin promotes intestinal motility by acting directly on motilin-sensitive receptors on gastrointestinal tract smooth muscles. Motilin is a hormone released from enterochromaffin cells, which stimulate contractile activity of smooth muscle of the stomach and small intestine. Erythromycin also acts on enteric cholinergic neurons through motilin and/or 5-HT_3 receptors to stimulate release of acetylcholine. The dose of erythromycin necessary to achieve the effect on gastrointestinal motility is much lower than the antimicrobial dose. Erythromycin appears to

induce the migrating myoelectric complex in the small intestine and promote gastric emptying. Erythromycin is usually administered at 0.1 to 1.0 mg/kg IV. A study in horses demonstrated that 1.0 mg/kg stimulates gastric and cecal emptying. Erythromycin lactobionate is often administered to horses (2.2 mg/kg diluted in 1 liter of 0.9% NaCl) IV over a 30-60 minute period every 6 hours; the dose in a 450 kg horse is 1 gram erythromycin per dose. Doses exceeding 10 mg/kg can disrupt propulsive motility. There has been some concern that the effect on motility can diminish with repeated dosing. Erythromycin can down-regulate motilin receptors, providing a possible explanation for the resistance of motility stimulation with repeated dosing. The most common side effect observed is abdominal pain; however, this is relatively infrequent.

Acepromazine and yohimbine are alpha adrenergic antagonists that are administered to horses with ileus based on the assumption that sympathetic hyperactivity contributes to motility dysfunction. Acepromazine causes a reduction in intestinal tone and facilitates transit of ingesta by increasing the cross-sectional area of the intestine. Caution should be used in administering acepromazine to horses with colic because it can cause hypotension owing to its vasodilatory effects (•"$_1$ blockade). If administered, it should be given either IM or slow IV at a dose of 0.05 to 0.10 mg/kg (10-20 mg to 450-kg horse). Some people have the clinical impression that administration of 0.01 mg/kg IM and repeated at 4-hour intervals after recovery from anesthesia reduces the severity or ileus in horses with small intestinal lesions. Yohimbine hydrochloride is a selective competitive antagonist of "•$_2$ receptors. It has been shown to reduce the severity of experimental POI when administered at 0.15 mg/kg IV at 1, 4, 7, and 10 hours after surgery. Acepromazine and yohimbine are not commonly used for treating horses with POI.

Bethanecol is a muscarinic cholinergic agonist causing contraction of intestinal smooth muscle via stimulation of acetylcholine receptors. It is used in horses with POI based on the assumption that decreased parasympathetic activity contributes to abnormal motility. There is conflicting data regarding its efficacy in restoring normal intestinal motility. It has a number of side effects owing to increased parasympathetic tone, including abdominal pain, diarrhea, salivation and increased gastric secretion. It is not commonly used in horses with POI, but it is occasionally given to foals with gastroduodenal ulcers to improve gastric emptying.

Neostigmine is a cholinesterase inhibitor that slows the breakdown of acetylcholine and thus prolongs its activity. Some clinicians have the impression that it will appreciably decrease the severity of POI in horses in which the large colon is involved. It is sometimes useful to help promote evacuation of ingesta from the large and small colons once impactions in these segments are softened with appropriate medical treatment. A common side effect is abdominal pain owing to intestinal contractions. The dose used clinically is 0.044 mg/kg (approximately 2 mg in 450 kg horse) SQ or IV. The dose can be repeated every 0.5 to 2 hours. If there is no response and no side effects are observed, the dose can be increased by 2 mg increments to a total of 10 mg per treatment.

REFERENCES

References are available from author upon request.