Abdominal disease is common in camelids and can present many diagnostic and treatment challenges. The presentation of mild colic with abdominal distension and decreased or abnormal fecal output in camelids may indicate either mechanical or functional (ileus) intestinal obstruction. Differentiation of the possible causes and their management is important for a successful outcome and often relies on additional diagnostic tests including a CBC, serum biochemistry profile, gastric fluid chloride analysis, abdominal ultrasound, and sometimes abdominal radiographs. Even with the additional diagnostics, differentiation between medical versus surgical conditions can be challenging, making the treatment approach decisions particularly difficult. This presentation will discuss diagnostic approaches to these conditions along with medical and surgical treatments.

There are numerous causes of abdominal discomfort or colic in camelids. Signs of abdominal pain or colic in camelids may be pronounced and include active kicking or rolling. However, more commonly it is more subtle and includes increase time in a crouched position, particularly with the hind legs extended to the side rather than directly underneath the animal. The animal will often switch from side to side and may lie in lateral recumbency but does not overtly roll. Camelids with colic may also have a hunched back posture when standing and may demonstrate discomfort upon deep abdominal palpation. Two of the primary causes of colic in camelids are enteritis and intestinal obstruction.

Physical Exam Findings
Enteritis and GI obstruction can be difficult to differentiate by physical examination because they both present with many common clinical signs including decreased appetite, abdominal distension, abnormal feces, abnormal GI sounds, and bruxism. When overt diarrhea (watery feces of increased frequency and/or volume) is present, differentiation of enteritis versus obstruction is often straightforward and enteritis should be considered the primary differential. However, both conditions can present with no feces, or decreased amount of feces that may be soft, watery, or contain mucous. The abdominal distension and decreased fecal output that occurs with enteritis is due to small or large intestine ileus and can be quite profound. Because of the decreased passage of ingesta, fecal output may also be decreased and may be mistaken as a GI obstruction. Absolute lack of feces or scant normal consistency of feces with mucous is more suggestive of GI obstruction, but can occur in the acute stages of enteritis as well. Serially monitoring abdominal girth can be helpful, particularly in early stages of the disease. With GI obstruction, abdominal girth will often increase until the obstruction is relieved. In cases of enteritis, abdominal girth tends to stay static and then decrease as the condition resolves.

Decreased GI sounds (borborygmi) may be identified with both enteritis and GI obstruction. Gastric compartment 1 (C1) sounds are often diminished with both conditions due to gastric distension, or negative feedback of C1 motility due to pain, inflammation, or sepsis. Intestinal borborygmi are usually decreased in frequency and intensity for both conditions. However, in some cases they are increased and this seems to be more common with hypermotility associated with enteritis. Even though intestinal contractions may be increased, normal peristalsis may not be present resulting in accumulation of ingesta in the intestinal tract.
Diagnostics
Additional diagnostics can be extremely helpful in differentiating between GI obstruction and enteritis. However, many cases can still be equivocal after a thorough diagnostic workup and may take time and close monitoring to differentiate. Typical diagnostic workup includes:

- **CBC:** Helpful in differentiating inflammatory from non-inflammatory conditions. Note that GI obstruction may result in a systemic inflammatory response if there is compromise and leakage of the intestine at the site of obstruction. Also, enteritis may present with variable indications of inflammation, particularly enteritis caused by coccidia.

- **Serum Chemistry Profile:** The serum chemistry profile is particularly useful in evaluating electrolytes, total protein, albumin, globulin, liver enzymes, creatinine, and triglycerides. These are all important in evaluating the secondary organs that may be involved and directing supportive care. Enteritis, particularly coccidiosis, is often associated with pan-hypoproteinemia in camels. TP less than 5.0 g/dl should be recognized as an important finding that may be due to either enteric protein loss or lack of absorption of nutrients due to GI inflammation. Hypochloremia is suggestive of proximal GI obstruction, but this could be due to functional obstruction from profound ileus caused by enteritis (particularly Clostridial enteritis) as well as mechanical obstruction. Unfortunately, serum chloride concentration is not as valuable in cases of distal GI obstruction and can be normal or low. Hyponatremia may be suggestive of a secretory enteritis. Triglycerides are helpful in assessing the animals overall metabolic state and potential of excessive fat mobilization. Liver and renal function should always be monitored in these patients.

- **Fecal Float:** Gastrointestinal parasites should always be evaluated by fecal floatation in cases of suspected enteritis. In the Rocky Mountain Front Range region, we tend to see more coccidiosis than nematode intestinal parasites in llamas and alpacas. Evaluation for coccidiosis should always be performed, especially in cases with a low serum TP concentration. However, clinical coccidiosis can occur before coccidia oocysts are present for both small (E. alpacae, llamae, and punoensis, prepatent period 14-21 days) and large (E. macusaniensis, prepatent period as long as 50 days) coccidia.

- **Fecal Cytology:** Direct smear fecal cytology can be very helpful in assessing both inflammatory component of the intestinal condition as well as overall bacterial population. The smear should be examined for the presence of fecal WBCs and RBCs that may indicate mucosal inflammation and necrosis. Normal feces should consist of a pleomorphic population off Gram negative and positive bacteria. An overabundance of any one type of bacteria, particularly large Gram + rods, suggests bacterial overgrowth and should be addressed with either specific antimicrobial treatment or reinoculation with normal flora (fresh rumen fluid, fecal slurry, or commercial probiotics).

- **Gastric C1 Fluid Analysis:** An evaluation of protozoal activity of C1 contents is helpful to assess microbial flora status. Increased C1 fluid chloride concentration indicates regurgitation of C3 contents into C1 and is most suggestive of a proximal small intestinal obstruction. The C1 fluid chloride is often normal (<30 mEq/L) in distal GI obstruction and enteritis. It can sometimes be mildly elevated in cases of Clostridial enteritis. C1 fluid chloride can be assessed by blood chemistry analyzers after the
sample has been centrifuged and then passed through a 0.2 um Millipore syringe filter in order to remove particulates prior to analysis.

- **Abdominal Ultrasound:** Some degree of expertise with camelid abdominal ultrasound evaluation can be achieved by any veterinarian. The left side is evaluated for evidence of C1 motility and size. Generally, the C1 compartment will encompass the entire left side of the abdomen. In cases of distal GI obstruction or ileus from enteritis, dilated small intestine (>1.5 cm diameter) may be seen in the caudal ventral left abdomen. Evaluation of the right abdomen will allow assessment of the 2nd and 3rd gastric compartments, the duodenum, distal small intestine, and spiral colon. The C1 gastric wall will normally extend at least to ventral midline or a little beyond to the right (8 to 12 cm). The 3rd gastric compartment normally lies ventrally and just to the right of C1. C3 diameter is reported to range from 5 to 11 cm and is slightly large in adult alpacas than llamas. However, in our experience, the C3 compartment diameter in alpacas is less than this and in juvenile alpacas it is generally 3-5 cm diameter. The duodenum maximum diameter is reported to range between 0.8 to 2.8 cm and the maximum small intestinal and spiral colon diameter ranges from 0.9 to 1.6 cm diameter. Small intestinal wall thickness ranges from 2 to 4 mm. Evaluating these areas of the GI tract for increased diameter and wall thickness is helpful in assessing for enteritis or GI obstruction.

- **Abdominal Fluid Analysis:** Abdominocectesis can be performed at a ventral paramedian location, or a right paracostal location. Camelids often have a thick fat pad between the ventral internal rectus sheath and the peritoneum that can result in unsuccessful abdominocectesis from the ventral paramedian location. The right paracostal location is at the level of the costochondral junction about 2-3 cm caudal to the last rib. Abdominocectesis at this location can be performed with either a teat canula or a needle. Abdominal fluid is assessed for total protein and cellularity. Normal total protein should be <2.5 g/dl with a total WBC count <3000/ul. Increased total protein without changes in WBC is consistent with a modified transudate and is more often seen in cases of enteritis. Mechanical GI obstruction generally has no changes in abdominal fluid analysis unless there is compromise to the intestinal wall. Suppurative inflammation is suggestive of compromise to the bowel wall and possible septic peritonitis.

**Differentiation of Medical and Surgical Conditions**

Many cases of enteritis are straight forward due to the presence of watery feces with increased volume or frequency. Additional diagnostic tests including fecal float, fecal culture, fecal cytology, and fecal virology tests can be pursued to help identify a cause. Fecal cytology can often give an indication of the degree of mucosal inflammation by the observation of fecal WBCs and RBCs. An abundance of large Gram positive bacterial rods is suggestive of Clostridial overgrowth as either a primary or secondary etiology. *Clostridium perfringens* enteritis is sometimes seen as a complicating condition with coccidiosis and should be treated accordingly. *Clostridium perfringens* enteritis (most commonly Type A) can also be a primary etiology and will often cause signs of colic, ileus, and distended abdomen. Because of the pronounced ileus, fecal production may be absent for several days and can be difficult to differentiate from mechanical GI obstruction. Coccidiosis is currently the most common cause of enteritis that we see at the CSU VTH. Both small and large coccidia may be identified as the primary cause. Salmonellosis and Campylobacteriosis are other important differentials, but are much less common in our area.
The hallmarks signs of mechanical GI obstruction include abdominal distension with scant or no fecal production. It can take up to 48-72 hours to see fecal production cease in cases of proximal GI obstruction. Serum and C1 fluid chloride concentrations can be very helpful in diagnosing proximal GI obstruction but may be equivocal in cases of distal GI obstruction including spiral colon impaction. Abdominal ultrasound can be extremely helpful in helping confirm GI obstruction. Distension of the proximal duodenum and C3 with empty distal small intestine is very suggestive of a proximal duodenal obstruction (phytotrichobezoar). Distal small intestinal obstruction or spiral colon impaction can be more difficult to differentiate from enteritis with generalized ileus.

**Medical Management**
Basic supportive care including oral or IV fluid therapy is important in cases of enteritis. Additional medical treatment can be determined based on confirmed or suspected cause of the enteritis. Medical treatment will be discussed in further detail at the meeting.

**Surgical Management**
Exploratory laparotomy is often indicated in cases of mechanical obstruction. In cases where it cannot be determined if the primary condition is enteritis or mechanical obstruction, an exploratory laparotomy may still be indicated. We have seen few adverse complications associated with laparotomy in patients with primary enteritis and in cases of Clostridial enteritis, there is the opportunity to provide direct intra-intestinal treatment. Both right paracostal and ventral midline approaches are acceptable. Further considerations in surgical treatment will be discussed in further detail at the meeting.

**Suggested References:**