

# Meconium

Meconium is formed from swallowed amniotic fluid, intestinal secretions (e.g. bile), cellular debris, and other debris. It first appears during the 1<sup>st</sup> trimester and accumulates throughout fetal development. Bile acids are excreted by the beginning of the second trimester. At full term fetal meconium consists of water, mucopolysaccharides, cholesterol, sterol precursors, proteins, lipids, bile acids, bile salts, enzymes, blood group substances, squamous cells, hair and vernix caseosa (sebum and desquamated cells from fetal skin).

*In utero* meconium passage is often associated with fetal distress. But it can occur as early as the second trimester and is not associated with fetal compromise at this early stage. Late term meconium passage appears to occur as the fetal GI nervous innervation matures and responds to stimuli. This is one reason that it is more often seen in post term gestations. Defecation is primarily under the control of parasympathetic pathway stimulation. It may be secondary to vagal stimulation with cord or head compression. Although it is commonly believed to be a response to stress, this has not been confirmed experimentally.

In human medicine the finding at birth of “thin meconium” in the fetal fluids is not associated with fetal asphyxia or low Apgar scores. Many believe it is a result of passage of meconium from earlier fetal distress (not at birth). Finding “thick meconium” at birth is thought to represent more recent passage, and is associated with meconium aspiration syndrome (MAS) and fetal asphyxia. In large animals thin meconium may represent fetal diarrhea. **\*\*Fetal diarrhea\*\*** usually begins just before birth and the neonate is born passing profuse, liquid meconium. This diarrhea usually resolves within 48 hours of birth. I have associated fetal diarrhea in the foal with intrauterine stress such as hypoxic insults or placental inflammation, but others have wondered if it is a manifestation of fetal enteritis as may be caused by *Neorickettsia risticii* and some feel it is most common with herpes virus infection.

## **Meconium Impaction/Retention**

Meconium retention occurs when meconium is not passed because of abnormal motility during the first 12 hours of life. Meconium may be retained in the large intestine for up to

2 weeks or on rare occasions for 4 weeks or more. **\*\*Foals with meconium retention are not aware of its presence and do not have colic, strain and may not respond to enemas. In fact, these foals often respond atypically to enemas by retaining the enema fluid. A meconium impaction is the painful retention of meconium associated with colic, straining and tenesmus. \*\***

Traditionally, it is thought that there is an increase incidence of meconium problems in colts. This is often blamed on their narrow pelvic canal. Meconium retention may be caused by excessive meconium formation *in utero* or impaired gastrointestinal motility associated with intrauterine distress (asphyxia, hypoxic-ischemic insults or associated with placentitis), sepsis, IUGR or prematurely. It may be secondary to prolonged recumbency, dehydration, or changes in motility caused by drugs such as dopamine.

In contrast to foals with meconium retention, foals with meconium impactions will act very painful. Foals with meconium impactions will strain to defecate with an arched back, nurse frequently as a source of comfort, and shows signs of persistent abdominal pain (colic) including rolling on their back, kicking at their abdomen and frantically swishing their tail. Although they nurse frequently they don't nurse effectively because of the distraction of pain. They often have dried milk on their head from standing under the mare's streaming udder. They develop significant abdominal distension if the meconium occludes the lumen sufficiently to prevent gas passage. During periods of tenesmus or colic the umbilicus may reopen and either bleed or drip urine. These foals are predisposed to umbilical infections. On rare occasions rectal prolapse occurs.

The diagnosis is usually made by reviewing the history and performing a physical examination. At times attendants place much emphasis on whether the foal "past his meconium". However, since there is a variable amount of meconium in the colon of each foal, seeing some being passed cannot rule out impaction. The absence of observation also should not be weighted too heavily. Despite careful observations, passage of meconium is easily missed. Searching the stall looking for the meconium often is futile unless it ends up on the bottom of your shoe. However the general observation that little or no meconium has been passed coupled with the onset of straining and colic should lead you to suspect meconium impaction. Usually performing a digital rectal examination leads to a definitive diagnosis. Often a meconium fecal ball can be palpated in the pelvic canal. An enema can also be diagnostic if it produces meconium and temporarily relieves signs.

When digital examination is negative, the value of deep abdominal palpation should not be overlooked. If the foal is quiet (sleeping or sedated) and is not overly distended with gas, deep abdominal palpation may reveal large amounts of retained meconium or an impaction. Meconium is readily identified because it is firm and distinct. There are no other structures in the neonate's abdomen which can be confused with it. Usually the most rewarding areas to palpate are in the caudal abdomen above the bladder, or in the anterior abdomen under the ribs (in the sternal and diaphragmatic flexures of the colon). At times the meconium can be palpated coursing from the anterior abdomen to the caudal abdomen. When abdominal palpation is unrewarding or impossible because of the resistance of the foal to manipulations, abdominal ultrasound can be helpful, especially when several differential diagnoses remain possible. Ultrasound examination can not differentiate between meconium impaction and meconium retention but combined with clinical signs will make the diagnosis. Ultrasound examination can also rule out the presence of several other possible causes of colic.

Differential diagnosis for meconium impaction include: colonic atresia (is ruled out if significant amounts of feces has been passed or the enema fluid comes back colored), rectal perforation (usually caused by over zealous enema administration), ruptured bladder, NEC, intestinal volvulus, intussusception, and unusual conditions such as a pendulous ovary wrapping around colon causing an obstruction (much like a lipoma does in aged horses).

Mild meconium impactions respond readily to soapy water gravity **\*\*enemas\*\***. A soft enema tube should be used with care, since it is possible to perforate the rectum. Three hundred to 500 ml of warm soapy water (a mild soap such as Ivory<sup>®</sup> is usually used) should be given gently. The soapy water solution can be replaced by a solution of lubricants (e.g. J-lube<sup>®</sup>). Soapy water enemas can be repeated frequently however even with mild soap a certain degree of rectal irritation occurs. This rectal irritation may result in persistent tenesmus interpreted as continued impaction, resulting in the perceived need for more enemas. Also, even before the first enema is given, often the rectum is very irritated and edematous secondary to the impaction. Great care should be taken not to exacerbate this condition. Rectal perforations are more likely with a damaged rectum.

When solutions such as dioctyl sodium sulfasuccinate (DSS) are used, great care should be taken. DSS is irritating and results in sloughing of epithelial cells. Even though this

solution is being given as an enema, care should be taken not to use too high a dose. Glycerin is another irritant that is sometimes used as an enema solution. The mild irritating properties of all of these solutions (including soapy water) enhance their action. Irritation results in active colonic secretion helping loosen the impaction. However, too much irritation not only increases the likelihood of traumatic perforation, it also can act as a portal for sepsis.

An alternative to a gravity enema in meconium impactions is the so-called **\*\*\*retention enema\*\*\***. To give a retention enema, a large bulb (50-75 mls) Foley catheter is placed in the rectum. The bulb is inflated so that a moderate seal is produced between the catheter's bulb and the rectum (it is vital that the catheter's bulb is in the pelvic inlet and not pushed further into the abdomen when it is inflated - this helps confine the bulb and prevent rectal perforation). The enema solution is introduced through the Foley catheter (a volume of 100 to 150 mls), the catheter is clamped and allowed retained for up to 20 minutes. Foals often strain against the catheter and may push it out prematurely. The most common solution to use in a retention enema for meconium impactions is 4% acetylcysteine (six grams acetylcysteine powder in 150 mls of water). Acetylcysteine is expected to dissolve disulfide bonds, which are commonly found in meconium. This solution will not dissolve the meconium but will make the outer surface slippery because of its action and may allow the meconium to pass. Much of the effect of a retention enema may be in the rectal distension resulting in secondary stimulus of motility. In man, often barium is mixed with the acetylcysteine to result in an osmotic effect. I have used milk of magnesia for an osmotic effect in foals, but have not seen a dramatic effect. Others occasionally add sodium bicarbonate, however the large amount of sodium introduced to the rectum could be dangerous as well as irritating. Retention enemas can be repeated several times without a problem. If they are not retained for the desired time period, they can be repeated.

If the impaction persists despite the use of enemas, oral laxatives may be considered. Mineral oil (2 to 4 ounces), milk of magnesia (2 to 4 ounces) or DSS (10 to 15 mls) can be used. DSS presents more of a risk of secondary sepsis resulting from translocation of bacteria due to damaged epithelium. Some practitioners like to use Castor oil. I feel this is a bad idea because, although it is effective, it is also extremely irritating and many foals become leukopenic and show of signs sepsis after its use. Since neonates are so susceptible to sepsis and the Castor oil mediated damage can result in bacterial translocation, I feel it is contraindicated.

If the impaction is present for a long period of time and if the impaction prevents adequate nursing on part of the foal, intravenous fluids are indicated and supplementing the patient with dextrose may be required. The foal can be fed although large quantities of luminal fluids may result in reflex motility and more colic. This problem occurs during the period when colostrum is absorbed. Close attention should be paid to adequate passive transfer. Plasma transfusions may be required. As noted above these foals are at a higher risk for generalized sepsis with translocation of bacteria through damaged colonic epithelium and through the umbilical structures.

Meconium impactions almost never require surgical intervention. Patience is the key to success in most cases. When surgery is required, a poor outcome is more likely. Meconium impactions are very rare in other farm animals, but do occasionally occur in small ruminants. A distinction should be made between meconium impaction (colic, straining and no feces) and meconium retention (no colic, no straining and no feces). The later is associated with neonatal gastroenteropathy and dysmotility. In such cases meconium can easily be retained completely for 7 – 10 days. Enemas are ineffective. In fact, the enema fluid is often retained, not resulting in the expected tenesmus. Giving repeated enemas which are retained can lead to hyponatremia secondary to water overload. The best treatment for meconium retention is patience on the part of the clinician as intervention will not speed resolution.

## Birth Trauma

The explosive nature of equine birth predisposes to the development of secondary traumatic lesions. The most common of these is **\*\*fractured ribs\*\*** which can occur even in what appears to be a normal birth. When the foal is mal-positioned other injuries can occur such as a rupture of the gastrocnemius muscle when the foal was born in a dog sitting position. Rib fractures occur a few centimeters above the costochondral junction with multiple fractures commonly occurring in a straight line. Single fractures are a rare. Most times there are anywhere from 4 to 12 fractured ribs on a side. The etiology of the fractures is not clear but I think that dorsal ventral compression of the body as it passes through the birth canal may result on stress above the costochondral junction resulting in the fractures. Although the ribs are attached throughout their length by intercostal

muscles which tend to stabilize the fractures, the ends may lacerate this girdle of muscles and cause trauma to the lungs, heart, major blood vessels, liver, diaphragm or muscles. When fractures occur they usually involve ribs 4 and caudal so almost inevitably fractures occur over the heart, putting the heart and great vessels at risk. This secondary trauma can easily result in extreme distress or sudden death of the foal.

Foals with fractured ribs show signs of pain associated with the fractures or signs caused by the secondary damage caused by the ribs. The most common sign is tachypnea which may vary depending on the foal's position, but unlike tachypnea secondary to contracture, will occur when the foal is lying down as well as standing. Since hematomas often only form under the ribs and displacement is minimal, careful examination is needed to identify fractures. A distinct click may be found on palpation. Palpation of each rib with particular attention to the usual area where fractures are found may result in slight displacement. Frequently a click associate with heartbeat can be heard on auscultation, although it may be thought to be an extraneous sound and ignored. Fractures are usually evident on physical examination. Confirmation is usually based on radiographs or ultrasound examination.

**\*\*Bleeding secondary to fractured ribs\*\*** frequently occur from the intercostal arteries. It is most often diffusely in the chest wall or subpleural space. Hemothorax may develop. Hemorrhage in any or all of these areas may be extensive but not evident externally. Lung contusions, lacerations of the myocardium or the great arties can also occur. Myocardial damage can result in serious arrhythmias which may also be devastating. Clinical signs of rib fractures are variable resulting from pain, anemia, cardiac arrhythmias and usually include tachycardia and tachypnea. Often the signs are positional and are exacerbated during examination or when the foal is down.

## Umbilical Problems

The umbilical structures can be a **\*\*major source of bleeding\*\***. The bleeding may be extracorporeal or into the body wall or internal with rupture of the umbilical vessels in the abdomen. Bleeding secondary to internal umbilical artery rupture is more common in calves than foals. Normally in calves the umbilical arteries rupture along the bladder so that at birth the external umbilical remnant consists primarily of the fascial covering which is empty of vessels. When things go as designed the arteries seal as the arterial

muscle curls back on itself. Sometimes the seal is not effective and major hemorrhage may occur usually confined by the fascia surrounding the arteries but the bleeding can break into the abdomen. In foals the umbilical arteries usually break external to the body at the end on the external umbilical remnant. They too should seal when broken. If they don't, bleeding can occur external to the body or if the artery breaks in the body wall or abdomen, the bleeding can occur into the body wall or in the abdomen as with calves. If the external umbilical remnant is either held by the attendant or clamped or the external route blocked in some other fashion but the artery is not effectively closed and continues to bleed, the blood will either travel along the fascial planes along the arteries or, because it is a path with little resistance, up the urachus. Large urachal hematomas can result as well as considerable bleeding into the lumen of the bladder. This can result in a large quantity of free blood in the bladder lumen or hematomas in the bladder as large as a softball. As a result the foal may posture to urinate and pass a large quantity of blood and blood clots. Blood clots may potentially obstruct the urethra but if so usually the urachus opens so the obstruction is not as serious as it could be. If the urachus opens, large clots may drain and the internal hematoma resolve more rapidly than if it has to be resorbed. The bleeding may recur during the first 24 to 36 hours of life and may be extensive enough to cause hemorrhagic shock. Internal umbilical remnant bleeding is a common cause of neonatal anemia and icterus. Most often the bleeding is extensive and the condition is clinically unapparent but may be discovered by careful abdominal palpation or ultrasound examination.

The internal umbilical remnants are a common location of infections in large animal neonates. Although some infections may be ascending and thus drain externally and easily identified by palpation of the external umbilical remnant, most appear to be hematogenous and only affect the internal umbilical remnants. Although some veterinarians still remove infected internal umbilical remnants surgically, most will respond to medical treatment. I only find myself recommending surgical removal in about 2% of the cases and then only when medical therapy has failed.

**\*\*Patent urachus\*\*** is most often first noticed when the foal is 3-5 days old when the external umbilical remnant "scab" falls off. Diagnosis is made by close observation of urination and noting urine flow from the external umbilical remnant. The area around the external remnant may always be damp and in some cases the "leak" is constant and at a rate that the foal doesn't urinate normally as the bladder never fills. The observer needs to be aware that colts who don't drop their penis when urinating may have urine run out

of their sheath, along the ventral body wall and off the external umbilical remnant simulating a patent urachus. Treatment is primarily benign neglect as the urachus, given time, will close on its own. Placing the foal on antimicrobials targeted toward common umbilical pathogens and excreted in the urine is rational. Topical therapy such as scrubbing, applying disinfectants (especial tincture of iodine), silver nitrate cautery or other manipulations should be avoided.

The most common urachal disease, occurring more commonly than patent urachus is a condition we refer to as **\*\*urachitis\*\***. This is often secondary internal umbilical remnant infection but also may be caused by the presence of a hematoma or delayed atrophy of the urachus as seen in foals spending the first days of their lives recumbent. The signs of the condition include straining to urinate after normal urination and repeat posturing to urinate after an initial urination either producing small amounts or no urine with each attempt. There are usually no consequences to this condition although at times the repeated straining may result in a patent urachus. Therapy is primarily benign neglect although if the straining is excessive, the urinary tract analgesic/anesthetic, phenazopyridine (pyridium) is sometimes used. This will give symptomatic relief but is not therapeutic.