

Clinical Management of Pregnancy Complications in the Cow

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Fetal Mummification

The reported incidence of fetal mummification is 0.43 to 1.8% of pregnancies. The condition can occur from the 3rd to 8th month of gestation (esp. 4 - 6). In feedlot heifers there may be an incidence of 3-4% due to incomplete regression of the corpus luteum following attempts to induce abortion. Fetal death without luteolysis and adequate cervical dilation is followed by autolysis and fluid resorption in a non-pyogenic environment. The fetus and membranes become dehydrated leaving a dark brown and leathery mass (shrunken dried skin covering calcified portions of fetal skeleton). There is no vaginal odor or discharge, and cows are usually presented with a history of failing to calve on time. On palpation the uterus feels tight and the fetal remnant is hard to the touch. Usually a persistent corpus luteum is palpable, consistent with the history of no observed heats.

Spontaneous expulsion seldom occurs. There may be partial cervical dilation but dryness of the reproductive tract often results in incomplete expulsion through the cervix and/or vagina. Manual removal is indicated in such cases. Glucocorticoids are not indicated in the management of fetal mummification cases since an intact fetoplacental unit is essential for their mode of action. The treatment of choice is exogenous prostaglandin, and in most cases the mummified fetus will be expelled in 2-4 days. In some instances infusing the uterus with saline may moisten the tract sufficiently to facilitate expulsion. The cow's breeding prognosis is good. Strategic use of prostaglandin may enhance fertility by inducing a couple of estrous periods prior to rebreeding.

There will be the occasional case in which the mummified fetus is not expelled from the uterus after repeated prostaglandin treatments. This is the typical case that the author sees in our referral hospital. I have had no luck using estrogen (ECP) to dilate the cervix. Since the referring DVM has already treated repeatedly with prostaglandin, I routinely remove the mummified fetus

through a flank laparotomy incision. Post-surgical fertility in these cases has been very good.

Fetal Maceration

Fetal maceration occurs when abortion fails to occur following fetal death and regression of the corpus luteum. Maceration with a closed cervix is rare. Gradual bacterial digestion of the autolyzing fetus occurs in the uterine fluid, and this continues until only a compact mass of bones remains. Prior to calcification there is merely fetal tissue debris in a purulent discharge. Usually there is a history of a chronic, fetid reddish-gray watery or mucopurulent discharge from the vulva over a period of several weeks to months. The cow may exhibit signs of toxic metritis early but systemic illness is usually absent in the later stages. In some cases there may be gradual weight loss and a decline in milk production. Sharp bone fragments are palpable within the uterine lumen, and a vaginal speculum exam may reveal some fetal bones protruding through the cervix.

There is no satisfactory treatment as some bone fragments typically remain embedded in the endometrium. The chronic endometrial damage means that the prognosis for breeding is poor. In most instances the animal should be culled. In those rare cases where the cow's genetic merit warrants a heroic approach, attempts at transvaginal follicular aspiration and in vitro fertilization may be possible. However, there may be an increased risk of peritonitis due to the chronic endometritis and bacterial contamination of the cranial vagina.

Dropsical Conditions

(a) *Hydramnios (Hydrops of the Amnion)*

The amniotic fluid is clear, colorless, and mucoid in nature. Towards the end of gestation the volume is 5-6 liters. The fluid is watery in early to mid-gestation

since the source is the amniotic epithelium and fetal urine. In the latter part of gestation the fetal bladder sphincter prevents urine outflow into the amniotic cavity, and the fluid becomes progressively more viscid. The mucoid nature of the fluid is due to saliva and secretions of the fetal nasopharynx. Normally the volume is regulated by fetal swallowing. The condition is rare, accounting for only 5-10% of uterine dropsy cases. It has been associated with a genetic (autosomal recessive) or congenitally defective fetus where swallowing is impaired, and the amount of amniotic fluid gradually increases (up to 25-150 liters) over several months. The slowly progressive accumulation of fluid during the latter half of gestation means that the abdominal muscles become stretched and the cow takes on the typical "pear-shaped" abdomen during the last 4-6 weeks of gestation. In fact, the abnormality may not be noticed until parturition when a large volume of syrupy, viscous, meconium stained fluid is released.

If called to evaluate such a case, the placentomes, and often the fetus, can be palpated. The history of gradual onset of abnormal abdominal enlargement and palpation of a grossly distended uterus with palpable placentomes does not eliminate the possibility of twins. A definitive diagnosis can be difficult, although trans-abdominal ultrasonography may be useful to detect twins.

When counselling the owner on the best approach, it is reasonable to expect that the breeding prognosis of the cow will be good provided that there aren't severe postpartum sequelae. Although the amnion, chorioallantois (cotyledons) and endometrium (caruncles) are normal, retention of the fetal membranes should be anticipated. Milk production in the subsequent lactation is generally poor due to problems with energy balance (anorexia) caused by the pathologically enlarged uterus. The fetus itself is invariably defective and will not be viable. If the owner elects to keep the cow, the genetic implications should be discussed. Both the cow and bull may carry an undesirable recessive gene, and a different sire should be used to breed this cow in the future.

Treatment options are discussed below.

(b) *Hydrallantois (Hydrops of the Allantois)*

Although *hydrops uteri* is only seen sporadically in cattle, the majority of cases (approx 90%) involve hydrops of the allantois. Allantoic fluid is clear, watery, and amber colored. There is only a small amount of fluid contributed by the allantoic epithelium prior to the development of functional fetal kidneys. The allantoic cavity then stores fetal urine that is carried down the amniotic portion of the umbilical cord via the urachus. In the normal late gestation cow the volume reaches about 9.5 liters, but pathologic accumulations of fluid may

reach 150-200 liters. The etiology of the condition is not clear but an abnormality of placentation appears to be involved. The hydropic fluid has the specific gravity and characteristics of a transudate due to a vascular disturbance in the allantois. Fetal abnormalities such as cystic kidneys, hydronephrosis and dysfunction of the fetal tubules with resultant polyuria are seldom involved in the pathogenesis of the condition. Affected cows are usually older than 3 years, unless there is a congenital caruncle deficit in a heifer. In older cows the caruncle deficit may be the result of prior uterine infection, or the injudicious removal of a fetus and/or retained membranes. The diseased uterus has many non-functional caruncles, with the remaining placentomes being greatly enlarged. Adventitial placentation is common, and portions of the placenta may appear necrotic and edematous.

Although the condition occasionally develops as early as the 5th month in severe cases, it typically develops rapidly over 1-3 weeks during late gestation. The rapid distension means that the abdomen becomes tense and barrel-shaped. Typically the farmer questions the breeding dates and may suspect a multiple pregnancy. As the condition progresses the cow develops digestive symptoms (anorexia, decreased ruminations, constipation) and may be misdiagnosed as having indigestion, bloat, or traumatic gastritis. The pulse will be elevated (90-140/min) and there may be an audible expiratory grunt. The distended uterus occupies much of the abdominal cavity to the extent that the cow may become dyspneic. In more advanced cases the cow may become weak and experience difficulty in rising. On palpation per rectum the uterus is grossly enlarged, pushing upwards and backwards into the pelvic cavity. Normal placentomes are not readily palpable through the tense uterine wall. The fetus is generally inaccessible. In mild cases, the condition may not be diagnosed until term when there is excessive clear, watery, amber fluid released.

The prognosis for the cow should be extremely guarded and immediate salvage is recommended. If the cow is to be managed the farmer should realize that potential complications include uterine rupture, rupture of the pre-pubic tendon, and ventral herniation. Affected cows may also develop musculoskeletal complications such as coxofemoral luxation or femoral fractures.

(c) *Treatment Options*

Factors to consider when *hydrops uteri* is suspected are the prospective calving date, the cow's ability to rise and eat, and the availability of facilities to manage the case (nursing care). The decreased appetite results in a negative energy balance, and many of these cases are literally "skin and bone" once the fluid is released. Thus, the farmer should be aware of the likely poor lactation

prospects. Unless the cow is extremely valuable the best option is to salvage the cow for slaughter. The farmer should realize that the fetus is likely to be very small and poorly developed, with possible anasarca or ascites.

If the owner elects to manage the condition, parturition can be induced with a combination of prostaglandin and dexamethasone. Dystocia is common due to uterine inertia and the presence of a defective fetus (*hydrops amnion*). The edematous fetal membranes may be tough and difficult to rupture. The stretched abdominal musculature may diminish the effectiveness of the cow's abdominal press. The amount of assistance required is usually minimal as the fetus is generally small, but may show edema and ascites. Supportive treatment may be necessary to compensate for the sudden loss of uterine fluid. An alternate approach is cesarean section. The grossly distended uterus will be thin-walled and atonic. Some clinicians advocate partial drainage of the hydropic fluid prior to incising the uterus. This may reduce the risk of hypovolemic shock. Administration of IV fluids is recommended. In some cases the uterus may continue to accumulate fluid for several days after the surgery. Retention of the fetal membranes should be anticipated and aggressive therapy may be necessary to prevent septic metritis.

Uterine Torsion

Uterine torsion has been reported in all domesticated species, but it is most prevalent as a cause of dystocia in cattle. Expulsion of the fetus is impossible unless the condition is corrected, and circulatory disturbances can result in death of both the fetus and cow if a prompt diagnosis is not made. Despite this, the etiopathogenesis of the condition in cows is poorly understood. A diverse list of contributing causes has been proposed, including the anatomy of the cow, hilly terrain, slipping, a butt in the flank from another cow, the manner in which a cow rises, and the energetic movements of the fetus during the first stage of labor. Essentially the condition can result from either a predisposing or a direct cause. The predisposing cause is without doubt the unstable anatomical arrangement of the gravid bovine uterus, just as the anatomy of the equine gastro-intestinal tract predisposes the horse to colic. During pregnancy, there is a relatively small increase in the length of the broad ligaments when compared to the massive elongation of the gravid uterine horn, causing the uterus to curve around the point of attachment. As pregnancy advances, the gravid horn in particular extends away from its area of attachment, coming to lie between the rumen, intestines and abdominal wall. Thus, this anatomic arrangement means that any time the prevailing conditions permit increased uterine mobility, a cow in late gestation is predisposed to develop-

ment of a uterine torsion. However, even in the last months of pregnancy, when horn asymmetry becomes maximal, uterine torsion is an exception rather than the rule. The direct cause is most certainly an active one.

The incidence of the condition appears to vary with geographical location. This probably reflects an influence of husbandry practices, most notably nutrition. Animals on a concentrate diet have a smaller rumen volume than those at pasture, and thus there is more space in the abdominal cavity for the unstable gravid uterus to occupy.

Season of the Year

Our data suggest that a cow is not at an increased risk for uterine torsion if she reaches term in a particular month. The higher number of uterine torsion cases seen in the early spring is merely a reflection of the higher number of cows that are due to calve during that period. However, the effect of season on the occurrence of uterine torsion is controversial, and may reflect climate dictated changes in husbandry procedures. It has been suggested that confinement housing for long periods may favor the development of uterine torsions, possibly due to weakness of the abdominal musculature brought on by lack of exercise.

Breed

The influence of breed is also controversial. It has been reported that uterine torsion is most commonly seen in dairy cattle, but other authors do not support this opinion, and in their studies the affected breeds merely reflected the composition of the surrounding cattle herds. In contrast to the previous studies, our data demonstrate that there is definitely a breed effect, and that a beef or dairy designation is not the deciding factor. A Brown Swiss cow is significantly more likely to experience uterine torsion than a Holstein, and relative to all cattle breeds, Brown Swiss, Charolais, and Holstein cows are at an increased risk for uterine torsion. The deep, capacious abdomen in the large framed Brown Swiss breed, together with large fetal birth weights, may predispose these cows to uterine torsion. The Charolais breed is renowned for big calves that have muscular hypertrophy at birth. Angus, Hereford and Jersey cows were found to be at a decreased risk for uterine torsion relative to Holsteins and to all cattle breeds. This may reflect the relatively small fetal birth weights in these breeds. We did not observe any cases in *Bos indicus* breeds of cattle, possibly due to anatomical variability between *Bos taurus* and *Bos indicus* cattle. Instability of the gravid bovine uterus in *Bos taurus* cows is due to the fact that the broad ligaments slope ventro-medially from their subilial attachments to insert on the ventrolateral margin of the uterine body and the ventral (con-

cave) surface of the uterine horns. The insertion of the broad ligaments is more dorsal in the *Bos indicus* breeds, possibly explaining why the condition is rare in these animals; moreover, the *Bos indicus* breeds have small calves.

Age of the Cow

The influence of parity number is controversial, with some authors claiming that the number of previous pregnancies does not appear to influence the incidence of uterine torsions. However, others contend that uterine torsion is more common in pluriparous cows. Suggested reasons include the larger abdominal cavity in mature cows together with decreased uterine tone and mesometrial stretching. I am not aware of any cases where a uterine torsion recurred in the same animal in subsequent pregnancies. This suggests that fetal size, fetal movement, and the amount of uterine asymmetry in a particular pregnancy are more important in the etiopathogenesis of the condition than any cow-specific factors.

Stage of Gestation

The most striking aspect of bovine uterine torsions is that they almost invariably occur at term, and although the exact cause remains controversial, there is general agreement that most of the bovine uterine torsions occur in association with first stage labor. This is based on the fact that in most cases a variable degree of cervical dilation will be noted prior to or immediately after detorsion. This supports the theory that there must be some initiating factor that is peculiar to first-stage labor; otherwise, the condition would be more common in preparturient cows. Occasionally the cervix may be found to be fully dilated, with the membranes already ruptured. Thus, these cows may have actually entered the initial phase of stage 2 labor before the torsion developed. Uterine torsion may occasionally be diagnosed at 5 to 8 mo of gestation. Our figure of only 8% preterm cases in the referral population supports the contention that this is an extremely rare occurrence in the general population. One author reported that torsions of less than 180° often arise during advanced pregnancy, and that they may be present for several weeks with little or no effect on gestation. Minor torsions (45 to 90°) may be detected during routine pregnancy diagnoses, and probably undergo spontaneous correction. More severe torsions may cause secondary complications due to uterine congestion and edema, possibly leading to fetal death and/or uterine rupture. There are several reports in the literature of a live extra-uterine fetus being found subsequent to uterine rupture in a preterm uterine torsion.

Clinical Signs

Uterine torsion almost invariably occurs at term

and the animal usually presents with a distended udder. Thus mammary development is unlikely to arouse suspicion unless the cow is some weeks from the due date. In the periparturient cow, the increasing strength of myometrial contractions and associated cervical dilation are the perceived source of the animal's abdominal discomfort. Thus, indications of restlessness and mild distress do not generally attract attention. Symptoms of pain such as colic and teeth grinding are seldom noticed. In torsion cases suspicion is more likely to be aroused if the animal remains restless for a prolonged period, or the restlessness wanes and the cow does not progress into second stage labor. A reduction in food intake may be noted in some cases. Abdominal straining is not reported to be a feature of the condition, but it was the most common presenting clinical sign in our referral hospital, being reported in 23% of cases. The higher than expected incidence of straining is probably due to the more severe torsions represented in this referral population. In 31% of cases the torsion was greater than 270°, and this degree of tension may stimulate stretch receptors in the vagina, thereby invoking reflex abdominal straining. The absence of straining in the majority of torsion cases is due to the failure of either the intact fetal membranes or a fetal limb to enter the anterior vagina. The remarkably high prevalence of intact fetal membranes accounts for the fact that vaginal discharge is seldom noted.

The rate of onset of life-threatening complications depends on the severity of the torsion and whether bacteria can invade the uterus. Typically the veins and lymphatics are compressed first as a torsion develops. This leads to congestion and edema of the uterine wall. In more severe cases arterial compression can result in hypoxic changes, thrombosis, and eventually gangrene. Even though most (76%) of our referral cases are bright and alert upon arrival at the hospital, some of these animals are found to have a severely compromised uterus at surgery. It is probable that in these cases the severe torsion was of acute onset. Tachycardia is a consistent feature, and detection of a markedly elevated heart rate in the clinical examination of a preparturient cow should arouse suspicion that a uterine torsion may be present. Our findings agree with previous reports that the heart and respiratory rates tend to be elevated, and that the rectal temperature may be slightly increased in some cases. Surprisingly there are many cases in which fetal death and autolysis are present but the cow only develops slight or mild signs of toxemia. This is most likely because often the fetal membranes remained intact even though the placenta had separated. The invasion of putrefactive bacteria is then delayed and thus emphysematous decomposition of the fetus, and the onset of toxemia takes several days longer than in other types of prolonged dystocia.

Displacement of the upper commissure of the vulvar inwards, or to the left or right has been reported in some cases of uterine torsion. Vulvar edema is occasionally observed. It seems likely that compression of the vaginal veins and lymphatic drainage may be involved in the development of vulvar edema. One case of uterine torsion was presented with a large edematous mass protruding between the vulvar lips. This was determined to be a partially everted urinary bladder with necrotic loops of small intestine contained within. A slight depression of the lumbo-sacral vertebrae has been reported to be a frequent symptom of uterine torsion but this has not been my experience.

Uterine Torsion

Approximate duration. The lack of definitive clinical signs means that it is not unusual for a uterine torsion to be present for 6, 12, 24 or even 48 h before the owner summons veterinary assistance. Vaginal involvement may not be obvious if the torsion is less than 180°. However, in most cases the torsion extends posterior to the cervix such that the vaginal wall is involved in the rotation. Although the cases in our study were statistically more likely to have vaginal involvement in the torsion, an important point about this referral population became apparent. In 34% of cases vaginal involvement was not evident on speculum examination, and thus the diagnosis of uterine torsion would have been missed if palpation per rectum had not been performed. Precervical torsions are more likely to occur during the last trimester. I therefore advocate that palpation per rectum be the definitive procedure before the presence of a uterine torsion is eliminated from a differential diagnosis list.

Direction. Determining the direction of the torsion by vaginal palpation alone can sometimes be difficult, and palpating the taut broad ligaments and uterine artery per rectum reduces the risk of misdiagnosing a precervical torsion. It is essential that the direction of the twist is accurately determined prior to attempts at correction as rotation in the wrong direction will exacerbate the problem. The broad ligament on the side of the torsion is pulled vertically downward under the uterus, whereas the other ligament is pulled tightly across the top of the uterine body. The amount of tension on the broad ligaments helps to determine the severity of the torsion. The gravid horn usually rotates over the non-pregnant horn, and thus counter-clockwise torsions would be expected to be mostly associated with right horn pregnancies, and vice versa. Unfortunately the horn of pregnancy was not recorded in our cases, but the direction of the torsion (63% counterclockwise) does fit with the expected 60% frequency of right horn pregnancies. Instability caused by the increasing asymmetry of the uterine horns in advanced pregnancy does

not explain this phenomenon as the condition is relatively rare in the general population, and tends to be associated with parturition. The rumen would be expected to prevent rotation of the right horn over to the left, yet our data conclusively demonstrates that counter-clockwise torsions are significantly more likely to occur. This preponderance of counter-clockwise torsions is similar to other reports in which the occurrence of counter-clockwise torsions ranged from 59 to 75%.

Severity. The degree of torsion is generally recorded to the nearest quadrant. In the general population 90 to 180° torsions are considered to be most common. It has been suggested that many dystocias with so-called dorso-iliac, and even dorso-pubic, positional abnormalities may actually be mild uterine torsions which are not diagnosed as such. If this observation is true then uterine torsion may be a more significant cause of bovine dystocia than is commonly recognized. A torsion of greater than 45° may result in dystocia. Torsions of less than 180° are usually managed in the field and account for only 6 to 15% of referral cases. Most (57%) cases in our hospital are in the 180° to 270° range. This would suggest that torsions of more than 270° are extremely rare in the general population. Torsions involving 2 or 3 complete revolutions of the uterus seldom occur.

Method of Correction

There are several methods available to untwist a bovine uterus and there is no standard method which is applicable to every torsion. Often one or more of these techniques have been attempted prior to referral, especially cases of greater than 180° torsion. The choice of method of correction varies with clinician, stage of gestation, severity of torsion and the condition of the cow, uterus and fetus.

Manual correction and rolling the cow. Manual correction is only feasible if the main site of the torsion is caudal to the cervix. The severity of the torsion and the amount of cervical relaxation are critical factors. Since approximately 90% of bovine uterine torsions occur during labor, and most of the uterine torsions are less than 180°, detorsion per vaginam is probably the most common method of correction. In the general population the technique is effective in 96% of cases, but in referral populations the success rate ranges from 23 to 62%. The technique only works if the cow is standing and an arm can be passed through the vagina and cervix to grasp the fetus. Correction *per vaginam* is much easier if the fetus is alive, because active fetal rotation is readily elicited in response to the manipulations. It is unusual for the fetal membranes to be ruptured in the more severe cases where the torsion is at least 180°. In these cases if the fetus is still alive and the cervix does not appear to be completely dilated, it may be prefer-

able to attempt detorsion without rupturing the fetal membranes (ie., roll cow). Once the fetal fluids have been released the duration of fetal viability is markedly reduced. However, even in cases with intact membranes there may already be some placental separation and fetal hypoxia. If the fetus is believed to be dead then release of the fetal fluids will reduce the weight of the uterus and facilitate manual detorsion. The alternate nonsurgical approach (rolling the cow) is indicated if the cow is recumbent, the fetus cannot be reached due to the location or severity of the torsion, or if the animal is pre-parturient. Ultimately, clinician preference and the availability of assistance are determining factors in the management of each case. It has been suggested that sometimes the mere act of dropping the cow suddenly onto the side of the torsion may correct the condition. This has not been our experience, and then I caution that in more severe torsions extreme care should be exercised when casting a cow prior to rolling due the possibility of a sudden fall causing the friable uterus to rupture. Rolling may be successful in correcting the torsion in 84% of cases, but if the torsion has not been relieved after 3 to 5 attempts then surgery is indicated. A modification of the above technique entails the use of a plank (Schaffer's method) to assist in holding the uterus stationary. The use of the plank improves the success rate, with up to 90% of attempted cases being corrected.

Fetal extraction. Once the torsion has been corrected the degree of cervical dilation determines whether fetal extraction can proceed. Fetal viability may be compromised unless it is promptly delivered. Oxytocin therapy is more likely to be successful if the fetal membranes are still intact. The cervix seldom dilates if the fetus is already dead, and often this is the case even if the fetus is alive. A delay of only 2 or 3 h may result in the death of the fetus. In some cases cesarean section becomes necessary because of failure to correct the torsion by conservative means, and in other cases surgery may be necessary because the cervix fails to dilate sufficiently after the torsion is corrected. In our hospital the calf can only be successfully delivered *per vaginam* in 59% of cases where nonsurgical correction was attempted. The severity and duration of the torsion appear to play a major role in the probability of further cervical dilation. An indurated cervix is unlikely to dilate and manual stretching of a partially dilated cervix is seldom successful. If the vaginal canal is relaxed and the fetus does not appear to be excessively large, one author has suggested sectioning the cervical rim as it is stretched into a thin sleeve over the fetus when traction is applied. This approach is contra-indicated if the cervix is thick and indurated. The uterine and cervical tissues in the vicinity of the torsion may be extremely friable and subject to rupture when traction is applied to the fetus. I am convinced that above average fetal

size plays a major role in the etiology of this condition and thus do not consider cervical sectioning to be a viable option.

Flank laparotomy and/or Cesarean Section. Flank laparotomy may be indicated in the rare instances when a preterm torsion is diagnosed and attempts at correction by rolling the cow are unsuccessful. If the uterus has not ruptured and the fetus is still alive, the pregnancy may be able to continue on to term. Some clinicians prefer intra-abdominal manipulation as their first choice approach for correction of a uterine torsion prior to hysterotomy. However, most uterine torsions do not warrant surgical intervention. Cesarean section is definitely indicated if a term torsion cannot be reduced by alternate methods, or if the cervix is insufficiently dilated to permit fetal extraction. Although there is an excellent prognosis for the cow, the surgery is more technically difficult if the torsion is not corrected first. Obviously individual clinician bias and the peculiarities of each case govern whether surgery is ultimately performed.

Fetus

Presentation. Abnormal presentation is not a factor in the development of the condition.

Viability. Prompt diagnosis and correction of a uterine torsion provide a favorable prognosis for both fetus and cow. Delay in diagnosis almost invariably results in the delivery of a dead fetus, since hypoxia can result from placental separation even in the presence of unruptured membranes. It has been suggested that the severity of the twist does not directly affect the survival of the fetus, but in my opinion the amount of uterine vascular compromise is definitely a factor. We frequently observe a cyanotic uterus, especially in the more severe torsions which have not been corrected prior to surgery. This may explain the extremely poor prognosis for fetal survival in our referral caseload, with only 24% of the calves being delivered alive. Factors such as duration of the condition and severity of the torsion are major determinants of the outcome.

Gender. My clinical impression has been that uterine torsions tend to be associated with oversized male fetuses. In our hospital there is a significantly greater proportion of male (67%) than female (37%) fetuses. I attribute this to the involvement of a heavier than normal fetus as male birth weights almost invariably exceed that of heifers.

Weight. This has been the observation of other authors as well. In our hospital the average birth weight across breeds for the 65 recorded singleton fetuses was 49.8 kg (35.5-68.2). Calf birth weight exceeded the respective breed mean in 89% of recorded cases. Both the Holstein and Brown Swiss fetuses are significantly heavier than the breed average. Although twinning

tends to cause a reduction in individual birth weights, the combined fetal weight inevitably exceeds that of a singleton. The type of twin pregnancy involved in the torsion is significant because uterine torsion involving a bicornual twin pregnancy is reported to be extremely rare. This suggests that bicornual twins may tend to stabilize the gravid uterus by forming a broader base.

Cow:

Uterine rupture. Uterine torsion is one of the most common causes of uterine rupture in the cow, and was diagnosed in 27% of rupture cases in one study. The gross appearance of the wound edges, and the extent and severity of peritonitis can be used to determine whether the uterus had ruptured spontaneously some time prior to surgical intervention. In my experience these cases involve torsions of 360° or greater. In some instances the condition of the wound edges is indicative of recent rupture, most likely occurring during manipulations either at the farm or upon arrival at the clinic. These cases are also primarily of 360° or greater, suggesting that vascular compromise and the resulting edematous changes may weaken the myometrial fibers. Although some hemorrhage from the uterine wound at the time of rupture is inevitable, death from hypovolemic shock is extremely rare. The fetal membranes rapidly become adherent to serosal surfaces, especially the mesentery. The severity of peritonitis depends on the condition of the fetus. Fibrinous peritonitis will develop even if the autolytic fetus is bacteriologically sterile. In cases where clostridial or coliform organisms have invaded the fetus through a partially dilated cervix the cow may rapidly succumb to acute peritonitis and endotoxemia. Out of our 15 cases with uterine rupture, 5 animals were euthanized because of the nonviable state of the uterus, and 3 died during surgery. Five of the 7 cases that had corrective surgery were subsequently discharged, a success rate that is very similar to 6 of 7 cases in a previous report.

Survival. Maternal recovery rates should remain high unless severe toxemia or necrosis of the uterus has developed. The duration of the condition and patency of the cervix dictate the severity of fetal putrefaction, and thus the likelihood of fatal maternal toxemia. The severity of the torsion and duration of vascular compromise influence the development of uterine edema and ischemic necrosis. In our hospital some cows are euthanized due to the compromised state of the uterus. The survival rate for the remaining cases is above 90% when those animals that died prior to or during correction are omitted from the calculations. These animals appear to die from endotoxic shock, and I believe that this is a potential complication once a severe torsion is corrected and perfusion returns. Thus, I advocate the use of nonsteroidal anti-inflammatory drugs prior to

surgical intervention in more severe cases. The low maternal death rates with uterine torsion surgery may be due to the decreased likelihood of intrauterine infection compared with other forms of dystocia. Sterile autolysis is more likely to be present than putrefactive emphysema because proliferation of bacteria is delayed in many cases by the effective closure of the cervix by the torsion and by the presence of intact fetal membranes. The condition of the fetus appears to be more influenced by the duration of the condition rather than the severity of the torsion. In our hospital most of the necropsy reports from cows that died or were euthanized mention thrombosed uterine vessels, intra-mural hemorrhage and necrosis, and fibrinous peritonitis.

Retention of fetal membranes. Uterine torsion in cattle is likely to be followed by retention of the fetal membranes, metritis, and maybe perimetritis. In our hospital over 50% of cows have retained fetal membranes following correction of the torsion. Many of the cows that don't retain the membranes actually have complete membrane detachment at the time of fetal extraction, and the membranes are delivered with the fetus. I attribute the higher incidence of membrane retention to vascular compromise induced noninflammatory edema of the fetal cotyledons. The edema extends to the tips of the chorionic villi and locks them into the caruncular crypts, thereby delaying placental separation.

Conclusions

Vandeplassche noted that even if uterine instability is accepted as a cause of torsions up to 180°, it does not explain those torsions of 360° or more. I concur with this statement and would argue that although uterine instability and all the other predisposing factors may be involved in the development of uterine torsion, there must be some active force which is present in the first stage of labor. It has been proposed that strong fetal movements during parturition may increase the chances of a uterine torsion occurring. First stage labor is associated with particularly vigorous fetal movements presumably invoked by the myometrial contractions and subsequent changes in intra-uterine pressure and fetal blood flow. I believe that it is not so much the violence of the fetal movements as it is the size of the fetus involved. Our data strongly suggests that fetal size is a major factor in the development of uterine torsion. In the normal parturient process the fetus is able to rotate and flex its limbs within the confines of the uterine walls. It is possible that in cases with oversized fetuses the fetal limbs actually catch on the uterine wall and the continued vigorous movements rotate the entire uterus. Although the incidence of left side torsions correlates well with the expected incidence of right horn pregnancies, it is interesting to postulate that the preponderance of

left side torsions may actually be due the limbs of a large fetus bracing against the rumen wall and “walking” the uterus down to the left. Another potential traction point is the strong prepubic tendon directly below the uterus. An alternate explanation is that right side torsions may often self-correct. However, if a left side torsion develops, the uterus may become trapped by the rumen. Uterine torsion is ultimately of fetal origin, and there is no evidence to suggest that an affected animal is predisposed to the condition in subsequent pregnancies.

Suggested further reading

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Abstract

Serum transferrin in calves infected with *Haemophilus somnus*

J. McNair, C. Elliott, D. G. Bryson, & D. P. Mackie
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A competitive immunoassay, based on time-resolved fluorimetry, was developed and used to measure the serum concentration of transferrin in 10 calves with acute pneumonia induced by an experimental infection with *Haemophilus suis*. The normal range for transferrin (1.37 to 3.72 mg/ml) was established by measuring serum transferrin in 160 normal cattle. The concentration of transferrin decreased after infection in all the

calves, although it remained within the normal range. It recovered to pre-infection levels within six days in the six calves which had either no lesions or mild lesions, but remained low in the four which had extensive lesions when they were examined postmortem. These four calves had had lower concentrations of transferrin than the other six before they were infected with *H somnus*.

Cattle plague in Shangri-La: observations on a severe outbreak of rinderpest in northern Pakistan 1994-1995

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Between April 1994 and November 1995 the most severe epidemic of rinderpest reported in the world for over a decade affected domestic livestock in the Northern areas of Pakistan. As many as 40,000 cattle and yaks died, more by some estimates, and mortality rates may have exceeded 80 per cent in these species in several villages. This report describes some of the

clinicopathological and epidemiological features peculiar to the outbreak, including laboratory-confirmed rinderpest in a goat, and the difficulties encountered before the disease was eradicated. It also describes the human costs and emphasises the need to accelerate the global eradication of this most eradicable disease.