THE ROUTINE EXAMINATION OF THE EQUINE FETAL MEMBRANES

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Introduction

The examination of the equine afterbirth should be an integral part of the comprehensive post-foaling examination of any mare and her neonate. Signs of placental abnormalities or intra-uterine disease can be identified during the examination of the afterbirth and can serve as the earliest alert for impending neonatal disease in the days following the delivery of the foal. The villous (chorionic) surface of the fetal membranes constitutes a mirror image of the maternal endometrium and may thus provide important clues regarding the health of the endometrium and possibly the ability of the mare to conceive and carry another pregnancy to term.

The examination of the fetal membranes is also crucially important after a mare has aborted. Pathognomonic placental lesions, sometimes in the absence of any other significant fetal pathology, may have formed in the placenta, implying that the fetal membranes may provide the only means of diagnosing the cause of the abortion. It is thus important to examine all possible material, including the mare, the aborted foal and the afterbirth, in order to establish the cause of an abortion. The fetal membranes are an organ of the fetus and may provide information about systemic disease in the foal (fetus or neonate) that might be difficult to recognize during a clinical examination of the foal alone.

This presentation is neither a guide on equine neonatal disease nor is it a guide on equine abortions. Instead, it describes the normal gross anatomy of the equine fetal membranes and, where possible, signs of pathologic change and how these can help an attending clinician recognize diseases, events or defects that may in some meaningful way influence neonatal wellbeing or serve as clues in the clinician’s efforts at diagnosing the causes of equine peripartal diseases and abortions.

The Placenta In-utero and Post-partum

The equine placenta is of the epitheliochorial type and is diffuse. Its fetal parts (= fetal membranes or afterbirth) consist of three major components: the allantochorion (attached with its chorionic surface to the endometrium by means of diffuse villi), the allanto-amnion (often only referred to as the amnion; surrounding the fetus) and the umbilical cord (originating at the fetal navel and implanting onto the allantoic surface of the allantochorion) (Fig. 1 and 2).
During normal parturition, and in the case of most abortions, the increase in the mare’s abdominal pressure forces the allantochorion into the cervical canal. The tremendous pressure exerted on the pericervical part of the allantochorion (by the allantoic fluid) causes it to break, releasing its allantoic fluid and then allowing the passage of the fetus, still enclosed in its intact amniotic membrane. Once the fetus is delivered, the allantochorion remains attached to the endometrium while the amnion and umbilical cord hang from the vulva (Fig. 3). As the villous attachment of the chorion loosens, the weight of the amnion and umbilical cord gently pull the allantochorion out of the uterus. Since the umbilical cord is attached to the allantoic or inner surface of the allantochorion the latter is turned inside-out during its release (Fig. 4).

Placental separation occurs in a predictable sequence in most mares: The non-fetal horn is usually released first, then the fetal horn, the body, and lastly the pericervical portion of the allantochorion. As will be explained later it is this stepwise separation of the placenta that causes the differences in color between different regions of the chorionic membrane.
Most mares foal/abort in less than spotlessly clean surroundings. For this reason the spontaneously released afterbirth will be dirty on its outermost surfaces (normally the amnion, umbilical cord and allantoic parts), while the villous or chorionic surface remains relatively clean.

After foaling the amnion usually hangs so far out that it reaches the floor and it is thus exposed to mechanical tearing when the mare steps on it. Often, but not always, even the tip of the non-fetal horn of the allantochorion may be subject to the same damage. If the whole afterbirth has been voided, but is not removed from the stable the mare can cause damage to any part of the allantochorion.

If the examination of the fetal membranes is not performed immediately, they should be placed into a clean bucket and allowed to cool down as well as possible. Warm membranes should not be placed into a closed plastic bag - autolysis (decomposition of the tissues by their own enzymes) will progress much faster at body temperature than at any lower temperature. It is thus better to wait for the membranes to cool down and then place them inside the bag. The membranes should never be frozen if histological and viral diagnostic work needs to be done on them (which is almost always the case after abortion).

Examination Procedure

The procedure described here has proven most useful over many years and provides a systematic approach to the examination. The author enthusiastically acknowledges that other approaches to the examination may yield equally satisfying results, provided that they are systematic and consistent.

Before examining the afterbirth or just afterwards it should be weighed (all parts of it). The normal weight of a thoroughbred foal's fetal membranes is 4.5 – 6.7 kg (or 11% of the foal's birth mass). If the membranes are significantly lighter (< 4.5 kg), it implies that their surface area was smaller than normal, thus providing suboptimal nutritional support to the growing fetus. Fetal membranes weighing more than 9 kg are usually edematous or infected and almost certainly diseased.
Ideally, the membranes should be examined on a perforated surface (approximately 1.5 x 1.5 m) that allows fluid and debris to drain off the tissues when they are washed with running water. Keeping the allantochorion with its allantoic surface outermost, it is laid out in an F-shape (Fig. 4). The vertical leg of the F is open at the bottom (ruptured cervical star) and represents the uterine body component. Once the body has been laid out the two horns are pulled out to complete the F. Automatically the fetal (gravid) horn will form the upper arm of the F, while the non-fetal (non-gravid) horn forms the lower arm of the F (Fig.4).

With the allantoic surface positioned outermost the umbilical cord will be attached to the outer surface of the F. Attached to the umbilical cord will be the torn amniotic membranes.

If the surfaces of the allantochorion and amnion were badly soiled after falling to the ground, they can now be washed clean with tap water. The different parts of the afterbirth are then examined in the following order:

- Amnion
- Umbilical cord
- Allantoic surface of allantochorion
- Cut off the umbilicus and turn the allantochorion so that its villous (chorionic) surface lies outermost; reposition into F-shape.
- Chorionic surface of allantochorion

**Examination of the Amnion**

1. **Normal Features of the Amnion**

   a) The amnion (actually the allanto-amnion) envelops the fetus and contains the amniotic fluid. It usually only breaks once the foal has been born. During birth the smooth surface of the amnion provides easy passage of the foal through the birth canal (the amniotic fluid itself is not nearly as viscous as that of ruminants).
Usually the amnion is torn by head and leg movements of the foal immediately after the foal's thorax has passed through the vulva. It tears along its ante-umbilical portion.

b) The normal amnion is a thin, white membrane. It contains many blood vessels in the part closest to the umbilical cord. The blood vessels become sparser further away from the umbilical reflection. The latter fact probably explains why the amniotic membrane is often very edematous in the portions furthest removed from the umbilical reflection.

c) The blood vessels in the amnion are extremely tortuous, probably to provide elasticity to the membrane. Near its umbilical reflection the fetal (inner) surface contains the amniotic plaques which are small (1 mm) firm nodules that feel like grains of coarse sand.

Fig. 6: a. The amniotic membrane held up to demonstrate the relatively sparse distribution of vessels in its ante-umbilical region. b. The inner (fetal) surface of the amniotic membrane demonstrating its tortuous vessels and the amniotic plaques in the vicinity of the umbilical reflection.

2. Gross lesions of the Amnion

a) Bruising: This is easily recognized against the white background of the amniotic membrane. A minor degree of bruising is caused during normal parturition. If severe, the bruising may indicate excessive fetal movement during birth which, in turn, may be an indicator of excessive fetal hypoxia during birth. The latter is a consequence of protracted 2nd stage labor (dystocia, failure of the cervical star to rupture).

b) Intact membrane still covering a dead foal: This is a clear sign that the foal was dead, or at least comatose, at the time of its delivery. As the foal will not have breathed its lungs will not contain any air.
c) Vascular pathology:
This, again, is easily recognized when it is present. The blood vessels show perivascular edema, hyperemia and hemorrhage. The blood vessels are most severely affected nearest the umbilical reflection. Such pathology is very common in cases of abortions caused by viruses and babesiosis.

![Image of blood vessels in a herpes viral abortion case.](image1)

Fig. 7: The blood vessels of the amnion in a herpes viral abortion case. Also note the yellow discoloration of the membrane.

d) Color:
The most common discoloration of the amnion is icterus. True icterus will discolor the amnion throughout its thickness, making it appear yellowish on both surfaces. Such icterus is always an extension of fetal icterus (i.e. the foal will also be discolored) and is very common in cases of abortion caused by equine herpes virus and babesiosis.

False icterus results from muconium (fetal gut contents) staining of the fetal surface of the amnion. In these cases only the amnion’s inner surface will be discolored. Muconium staining indicates fairly severe intrapartal hypoxia and fetal distress.

![Image of muconium stained inner surface of the amnion membrane.](image2)

Fig. 8: Muconium stained inner surface of the amniotic membrane. Note that the outer surface (top half of the image) is not discolored.

e) Amniotic twisting:
A few cases of amniotic torsion have been seen. These cases are easily identified after delivery of the foal when a line of thrombi can be detected in the umbilical vessels – usually very near the umbilical reflection of the
amnion. In all cases some degree of twisting of the cord was evident as well.

Fig. 9: Amniotic torsion in an 8 month old fetus that was delivered by partial fetotomy. The mare was aborting her dead fetus that was affected by a massive hydrocephalus, causing dystocia. The intra-amniotic portion of this foal’s umbilical cord was excessively long and twisted, presumably causing the amniotic membrane to become twisted around its reflection on the umbilical cord. The cord of this fetus is shown in Fig. 21.

Fig. 10: This 6.5 month old fetus was found next to a mare that had died at pasture during the night. The mare’s uterus had prolapsed and the fetal membranes were completely intact. A necropsy on the mare revealed that she had died of a severe large bowel volvulus. When the allantochorion was incised to reveal the amnion and fetus we found the umbilical cord wrapped around the upper third of the amnion (arrow), causing mild compromise of the distal amniotic vessels. A cause-and-effect relationship between the placental lesion and the colic could not be established.

Examination of the Umbilical Cord

1. Features of the Normal Umbilical Cord

a) Length:
The umbilical cord consists of an intra- and an extra-amniotic portion and its total length should not exceed 80 cm in thoroughbreds. If it is longer it may twist around a fetal limb or the neck, leading to strangulation. One third of the normal umbilical cord is extra-amniotic while the other two thirds are intra-amniotic.

Fig. 11: The umbilical cord consists of intra-amniotic (left) and extra-amniotic (right) portions. The stumps of the umbilical arteries usually project beyond the severed end of the cord (white arrow). The indistinct opening of the urachus is located on the allantoic side of the amniotic membrane (black arrow).
b) Blood vessels:
Near its implantation onto the allantois the umbilical cord contains two veins and two arteries (Fig. 12). Approximately at the reflection of the amnion the two umbilical veins join so that only a single vein enters the fetal abdomen. The free end of the umbilical cord thus only contains 3 vessels. The free ends of the arteries of the spontaneously ruptured umbilical cord will usually protrude 1-5cm beyond the free end of the umbilical cord (Fig. 11). This gives the impression that the arteries have been torn out of the foal's abdomen, but even if that is so, it does not mean that the foal is suffering from intra-peritoneal hemorrhage. The arteries will have been stretched prior to breaking and their subsequent recoiling will have sealed them to prevent blood loss. Hemoperitoneum (bleeding into the abdominal cavity) as a result of the rupture of one or both umbilical arteries does occur, but in all these cases the umbilical arteries will have ruptured at the level of the foal's urinary bladder rather than near its abdominal wall).

The surface of the umbilical cord often looks extremely rough and irregular, but this is also normal. The opening of the urachus can be found on the allantoic side of the amniotic reflection.

c) Placental attachment:

Usually the umbilical cord implants onto the allantois near the base of the fetal horn. Although unusual, implantation of the umbilical cord onto the
non-fetal horn must not be interpreted as an abnormality. Until approximately the 6th month of gestation the fetus can lie in either horn or the body of the uterus, regardless of the specific horn where its umbilical cord had originally formed during the second month of gestation. After the 6th month the fetus will settle with its hind end inside either of the uterine horns and the torso and front end inside the uterine body. This means that the umbilical attachment to the allantoic membrane may be inside the non-fetal horn of the allantochorion (Fig. 13).

d) Twisting:
During gestation, particularly during the first two trimesters, the fetus can “roll” inside the uterus. During these movements the fetus can roll together with or independently of its surrounding amnion. These movements can then lead to twisting of the cord: Extra-amniotic twists result from movement of the fetus together with the amnion while intra-amniotic twisting results from fetal movements within the amnion. Twisting can occur in either, clockwise or anti-clockwise, direction and the intra- and extra-amniotic twisting may even occur in opposite directions. Any degree of twisting is considered normal, provided there are no signs of compromised blood flow (edema, perivascular hemorrhage, severe swelling).

![Twisting of the extra-amniotic portion of the umbilical cord without signs of compromised blood flow through the umbilical vessels.](image)

**Fig. 14:** Twisting of the extra-amniotic portion of the umbilical cord without signs of compromised blood flow through the umbilical vessels.


e) Amniotic plaques:
These are found on the intra-amniotic surface of the umbilical cord (Fig. 6 b.).

f) The yolk sac remnant:
The atrophied remnant of the yolk sac can be found in every fetal membrane. It consists of a 1 -3 mm thick strand that lies amongst the blood vessels of the extra-amnionic portion of the umbilical cord. Its proximal end always attaches to the inner surface of the allantochorion in the area originally delineated by the sinus terminalis during early pregnancy (Fig. 15).

Rarely, one will find an amorphous, usually spherical and hollow, structure attached to the extra-amnionic portion of the umbilical cord (Fig. 16). The size of the appendage varies from 1-15cm. The appendage is usually pedunculated with a set of blood vessels linking it to the main vessels of the umbilical cord. Except for nerve tissue, all other tissue components of horses have been identified inside these appendages. No distinct remnants
of any particular organ could be identified to date. It is thus highly unlikely that it is a retarded identical twin (acardiac monster). It is currently accepted that this is an ossified remnant of the yolk sac.

Unless the stalk of such an appendage loops around the umbilical cord, thus causing the occlusion of the umbilical vessels they are considered insignificant, incidental findings. We identified a globular ossified yolk sac remnant on the umbilical cord of a healthy fetus during a routine ultrasound examination of a mare in her 4th month of pregnancy (Fig. 17).
2. Gross lesions of the Umbilical Cord

a) Excessive twisting:
If the umbilical cord becomes so severely twisted that the flow of blood through its vessels is compromised severe swelling, edema and hemorrhage will be evident in the portion of the umbilical cord distal to the twist. The mere twisting of the cord without significant associated lesions has been diagnosed as the cause of abortion with reasonable frequency in the past, but it is suspected that, in the absence of other identifiable causes of abortion, the normal twisting of the cord may have been blamed incorrectly for a significant number of abortions.

![Excessive twisting of the umbilical cord. a. Excessive twisting of the intra-amniotic portion of this foal's cord was the only evident cause of abortion. b. and c. Hemorrhage and edema around the vessels of the excessively twisted cord.](image)

It remains an open question whether or not partial or temporary blockage of the umbilical vessels may be one of several causes of growth retardation seen from time to time in newborn foals.

Excessive twisting of the umbilical cord has also been seen in cases of placentitis and even twin abortions. In such cases it is reasonable to assume that the fetus was moving excessively (i.e. it was hyperactive) due to hypoxia caused by the placental compromise. Excessive movements lead to twisting of the cord and may have been the ultimate, but probably not the primary cause of the abortion.

![Aborted twins. The fetus on the right had died some days prior to abortion, while the fetus on the left was fresh. Inadequate placental support was presumed to have caused the live fetus to become hyperactive, thus twisting its cord until its blood flow through the umbilical vessels was finally occluded, leading to its death and the abortion of the pregnancy.](image)
Another sign of excessive twisting of the intra-amniotic cord seen from time to time is the saccular dilatation of a portion of the urachus. As the latter is the most thin-walled duct in the intra-amniotic portion of the cord it is likely to become occluded before any of the blood vessels do. Diverticula of the urachus have also been seen in normally delivered, healthy term foals.

Fig. 20: Twisting in the cord of this fetus caused occlusion of the urachus prior to obstruction of blood flow, leading to the formation of diverticula of the urachus and distension of the fetal bladder.

b) Inappropriate length:
As described above, if the umbilical cord is longer than 80 cm it predisposes to strangulation. Evidence of strangulation is easy to find when examining the fetus/foal. Excessively long cords are frequently twisted, suggesting that chronic twisting may lead to stretching of the cord. If the cord is very short (< 50 cm) it may break during parturition before the foal is delivered. Obviously, a foal without intact umbilical cord and unable to breathe freely, will suffocate. Foals like this can thus present as "still born".

Fig. 21: Excessively long cord associated with excessive twisting and a urachal diverticulum.

c) Hemorrhage, Edema and Swelling:
These signs can be of mechanical origin (twisting) or due to inflammation. In the latter case, similar lesions will be seen around the blood vessels of the placental membranes. As mentioned before, abortions caused by fetal babesiosis and viruses are often associated with such lesions.

Inflammation of the umbilical cord (funicitis or funiculitis) is a classic lesion in fetuses aborted as result of the Mare Reproductive Loss Syndrome.

Ulcerations on the surface of the umbilical cord have been seen in some cases of equine herpes virus abortions. Fungal plaques may occur on the umbilical cord in cases of fungal abortions.
d) The author has seen two cases of segmental aplasia of the urachus. In both cases, massive distension of the urachus was evident and both fetuses had huge urinary bladders. One of the foals was aborted, while the other was carried close to term at which time the mare suffered a terminal rupture of the abdominal wall.

![Segmental aplasia of the urachus in a near-term foal.](image)

**Examination of the Allantois**

1. **Features of the Normal Allantois**

a) The surface of the allantois is smooth and shiny with the blood vessels lying on its adluminal surface. Once the membranes have been voided the blood will have drained from the blood vessels, resulting in their pale color. Immediately after the foal's trunk has been born the normal foal will lie relatively still with its hind legs remaining inside the birth canal. The foal will only move its head and neck to shake off the amnion. While the foal lies in this position a strong pulse can be palpated in the umbilical artery (from the fetus towards the allantochorion) while strong flow is maintained in the umbilical vein (returning to the fetus). These observations suggest that placental blood gas exchange can continue even after delivery of the foal and may help the latter overcome the respiratory acidosis brought about by the birth process when blood flow through the uterus and fetal membranes will be reduced by the pressure of the abdominal contractions of the mare.

![Typical appearance of the surface of the allantoic membrane with a pale (empty) blood vessel.](image)

The umbilical arterial pulse usually disappears within one minute after the birth of the foal. After 3 -10 minutes the foal will start moving more
vigorously or the mare will stand up, thus severing the umbilical cord. This sequence of events results in the empty blood vessels seen in the normal afterbirth.

b) Thickness and Smoothness:
When the allantochorion is laid out in an F-position it is obvious that the fetal horn is smooth, while the non-fetal horn is thinner in diameter and much more wrinkled. The tip of the fetal horn is much more edematous (thick-walled) than the tip of the non-fetal horn.

c) Tears in the Allantochorion:
Ideally, there is only one opening (tear) in the allantochorionic sac. It is located at the bottom end of the vertical leg of the F, corresponding to the internal cervical opening. This area is called the cervical star. Other "normal" tears result from physical damage to the allantochorion when the mare steps on it after its expulsion. Whenever such tears are encountered the membrane edges surrounding the tear should be pieced together very carefully so as to establish whether any part of the afterbirth is missing (presumably retained) or not.

d) Hippomanes:
During advanced gestation fetal waste products suspended in the allantoic fluid may coalesce, forming lumps which can vary in size from 5 to 200mm. These are called hippomanes. Sometimes the hippomanes may be attached to the allantois by thin fibrin strands, but the larger ones usually remain free. Their color varies from khaki to brown and they have the consistency of hepatic tissue.
e) Remnants of the Endometrial Cups:
At about 90-120 days of gestation the endometrial cups are rejected by the maternal endometrium and are sloughed in the direction of the fetal membranes. The tissue remnants then invaginate into the allantochorion to eventually be enclosed in a pouch of this membrane. These pouches (±1 x 0.5 cm) can often be found protruding like polyps from the allantoic membrane of the full-term fetus. They are always found near the site where the umbilical vessels join the allantochorion, irrespective of whether the vessels are attached to the fetal or non-fetal horn.

Both, the endometrial cups as well as the umbilical cord are formed between Days 35 and 50 during pregnancy, while the conceptus is still entirely contained in one uterine horn (the same horn in which it "fixed" on Day 16). This means that when the umbilical attachment and the remnants of the endometrial cups are found in the non-fetal horn of an older abortus or a full-term foal the fetus must have fixed and implanted on one side, but later moved to the opposite side of the uterus where it did most of its growing. It also means that the fetal horn of a mid-to-late pregnant uterus need not necessarily have been the horn in which fixation had taken place much earlier during the pregnancy. These observations on the afterbirths of normal, full term foals support the rather well established observation of "transcornual" migration of equine pregnancies. It is also the reason why the terms "fetal" and "non-fetal" horns, rather than "pregnant" and "non-pregnant" are more appropriate when describing the fetal membranes.

![Fig. 26: Allantoic pouches (remnants of the endometrial cups) are always found in an arch around the insertion site of the umbilical cord. While they are not present in every membrane, there can be as many as 7 in a single afterbirth.](image)

f) Allantoic pouches:
These structures are occasionally found on fetal membranes of normal or aborted foals. Their origin is not known. They consist of pedunculated, fluid-filled structures that can reach a length of 25cm in extreme cases. Just like the remnants of the endometrial cups, allantoic pouches always occur on the allantoic side of the fetal membrane, but in contrast to the remnants of the cups, their position bears no fixed relationship with the site of the umbilical insertion onto the allantoic membrane.
2. Gross Lesions of the Allantois

a) Tears:
The absence of an opening in the allantochorion in the area of the cervical star is cause for serious concern. This is more frequently seen in mares that were induced to foal or that aborted. In either case the author postulates that this condition is caused by the insufficient "maturation" of the tissue of the cervical star during the prepartal period. Unruptured cervical stars are usually thicker and tougher than those that rupture normally at the onset of stage 2 of labor. If the star doesn't rupture, but the mare continues to strain, the allantochorion will eventually bulge through the cervix or even through the vulvar opening ("red bag"). In order to bulge so far caudally the villous attachment of at least a significant portion of the chorion to the endometrium is broken by mechanical force, resulting in the cessation of placental exchanges (oxygen, nutrients and waste products). This syndrome is commonly referred to as premature placental separation which, in the author's opinion is the result, not the cause, of the failure of the cervical star to rupture at the onset of stage 2 of labor. Continued straining will eventually result in the tearing of the allantochorion in a place other than the cervical star (usually through the uterine body component just caudal to the base of the non-fetal horn). The foal is then born with the caudal part of the allantochorion covering its head like a hood and can obviously not breathe normally. Even if the foal should survive the birth process the placental abnormality described above is a tell tale sign of a protracted birth process. The latter is associated with a number of neonatal problems (incl. the neonatal maladjustment syndrome).
Sometimes, after not rupturing initially, the cervical star ruptures much later and allows the foal to be born. The allantochorion of such a foal often has two tears: one at the cervical star and another across the uterine body just caudal to the non-fetal horn (Fig. 29 b.). If the above pattern is observed during the examination of a foal's afterbirth, the foal should be considered to have suffered from excessive intrapartal hypoxia resulting from its prolonged delivery. Obviously, where premature placental separation has occurred, the foal would also be deprived of its postnatal gas exchange across the placenta which adds to its already compromised metabolic state after a prolonged birth process.

Other tears of diagnostic significance involve the tips of the uterine horns (Fig. 7). One of the more common forms of partial placental retention is the retention of the tip of either uterine horn. Should there be tears in this area, the edges must be pieced together very carefully in order to differentiate between simple tears without any piece missing or tears resulting in the retention of a piece of the allantochorion. It is usually helpful to follow blood vessels in such cases. If the full length of a vessel is still present (even if it is torn) it is unlikely that any of the surrounding membrane was retained.

In order to differentiate between intrapartal and post-partal tears it may be useful to look for signs of hemorrhage from the edges along a membrane tear. If present, hemorrhage indicates that the foal must have been alive and its cord intact at the time when the tear occurred.

If there is no sign of hemorrhage three different situations are possible:

i. The foal was alive, but hemorrhage did not occur, because the blood flow to the particular area of the membrane was cut off by the high intrapartal pressure on the placenta.

ii. The foal died in-utero and the tear occurred during an abortion.

iii. Everything was normal, but the tear was a result of post-partal trauma to the already expelled allantochorion.
b) **Bruising:**
As with bruises on the amnion, excessive bruising of the allantochorion may serve as an indication of excessive fetal activity during delivery. Such hyperactivity is associated with fetal hypoxia that is the most common consequence of protracted labor. For this reason, allantoic bruises are often accompanied by a muconium stained amniotic membrane. It is also seen in fetal membranes of which the cervical star failed to rupture or only ruptured after some delay.

![Fig. 30](image)

**Fig. 30:**
- a. Hemorrhage at the edge of a tear in the allantochorion, suggesting that the tear occurred while fetal circulation through the membranes was still intact (intra-partal tear during a red bag delivery).
- b. Stumps of two ruptured blood vessels at the edge of a defect in the allantochorion, suggesting that a piece of the membrane was retained inside the mare’s uterus.
- c. The tip of this non-fetal horn had torn off and was retained after the remainder of the membrane was passed.

c) **Blood Stasis:**
It has been said that if the umbilical cord ruptures too soon after birth (e.g. a very short umbilical cord or when the mare foaled in the standing position or rose to her feet too soon) a certain amount of fetal blood will be trapped in the placental vasculature and that this blood will then be visible in the allantoic vessels during the examination of the afterbirth. Firstly, the amount of blood thus taken from the neonatal foal does not appear to affect the foal significantly. Secondly, the author has seen several cases of premature umbilical rupture and there was absolutely no blood present in the placental vessels afterwards. Two reasons are proposed for this observation:

i. During parturition the enormous pressure exerted on the fetal envelope probably forces most of the blood into the fetus so that little blood remains in the membranes during labor.
ii. If there is a significant amount of blood in the membranes, most of it would probably drain through the now open-ended umbilical cord before coagulation takes place.

However, should there be many blood clots in the vessels of the allantois it implies that the delivery was in some way abnormal.

d) Placentitis:
Inflammation as a result of viral infections of the feto-placental unit usually spreads along the allantoic blood vessels, starting at the umbilical attachment site (Fig. 32). It is postulated that the virus first infects the fetus (after reaching the latter via the hematogenous route from the mare, crossing the placenta and settling in the fetus), and then spreads along the umbilical vessels to the fetal membranes. Certain bacteria (Leptospira, *Salmonella abortus equi*) also reach the fetus and its membranes via this route.

![Fig. 32: a. Diagrammatic illustration of the typical distribution of placental lesions seen in cases of fetal viremia or bacteremia (V) and ascending bacterial or fungal (B) placentitis, respectively. b. Typical distribution of lesions along the major allantoic blood vessels after a herpes viral abortion.](image)

Most opportunistic bacterial and fungal pathogens that cause placentitis and abortion reach the uterus via an incompetent cervix. The resulting ascending infection will thus cause placentitis that spreads from the cervical star cranially (Fig. 32 and 33), sometimes causing an abortion before the fetus is infected. The distribution of lesions in the afterbirth may thus help in
differentiating between opportunistic, ascending (mostly sporadic) bacterial infections and viral infections (that are usually the cause of abortion storms).

In contrast to this general pattern, a different form of placentitis, caused by a Nocardia like bacteria, has been diagnosed in several hundred Kentucky brood mares, with only isolated cases being reported from other areas and countries. In these cases a very exudative placentitis occurs in a well demarcated area of the uterine body, well removed from the cervix (Fig. 34 a.). Neither the origin of the pathogen, nor the pathogenesis of the resulting placentitis is known.

Occasionally, bacteria and/or fungi that usually cause ascending infections through the cervix, appear to reach the uterus via a systemic route, causing lesions in areas other than the cervical star of the allantochorion (Fig. 34 b. and c.).

Fig. 34: Three examples of placentitis that did not involve the cervical star and were thus not caused by ascending infection through the cervix. a. Nocardioform placentitis; b. *E. coli* placentitis; c. *Aspergillus* spp. (note the thick, pseudomembranous exudate) on the chorionic villi.

e) Body Pregnancy:
When examining the allantochorion of an aborted fetus it may become apparent that both horns look "non-fetal" (both short, thin, wrinkled) (Fig. 35).

Fig. 35: Diagrammatic presentation of the shape of the allantochorion in a case of a body pregnancy. CS=cervical star. UH=uterine body

This placental shape is commonly reported to result from embryonic fixation in the uterine body instead of the base of either horn. Such pregnancies are said to then develop normally, but placental insufficiency (due to reduced placental surface area) during the latter stages of gestation presumably
results in fetal starvation, death and abortion. In the author's opinion the aforementioned theory is challenged by the fact that fetuses that have originally fixed in one horn can "migrate" into the contralateral horn where they develop into normal foals. The same should be possible for a fetus that initially develops inside the uterine body. The author postulates that body pregnancies result when a fetus, regardless of its original site of fixation, is trapped with flexed legs inside the uterine body at an advanced stage of gestation (the normal fetus will lie with its hind legs extending into either horn and its front legs and head extending into the uterine body towards the cervix). As fetal growth then accelerates during the last two or three months of gestation the fetus runs out of intra-uterine space and is aborted.

The author has also seen full-term dystocia cases that were associated with body pregnancies. The typical body pregnancy appearance of the allantochorion has also been seen in some cases of abortion in which the cause of fetal death was clearly unrelated to the intra-uterine positioning of

Fig. 36: Two typical body pregnancy membranes. a. Intact fetal membranes (fetal fluids and fetus still inside) of a mare that aborted at 6 months of gestation. At the time of abortion the mare was suffering from severe, clinical babesiosis and extensive lacerations sustained when she jumped several wire fences in an attempt at finding her recently weaned foal. The umbilical cord insertion was clearly located in the right uterine horn, proving that the "body pregnancy" had not developed as a consequence of embryo fixation in the uterine body. b. Fetal membrane of a foal that was delivered by fetotomy when the mare was presented for dystocia at full term. The entire foal was carried inside the uterine body (the hind legs did not extend into either of the uterine horns). Note the central insertion of the umbilical cord between the uterine horns, suggesting that this pregnancy may have developed after embryo fixation in the uterine body. Also note the unusually wide and long uterine body component of this fetal membrane.

Fig. 37: Fetus and its membranes after abortion at 8.5 months of gestation. The mare presented as an abortion case in dystocia when a severe fetal hydrocephalus prevented the delivery of the fetus. After delivery by partial fetotomy the fetus was placed on top of its membranes to illustrate its intra-uterine position and posture. The insertion of the umbilical cord is clearly located in the left horn of the allantochorion, demonstrating that this body pregnancy did not result from embryo fixation inside the uterine body. In this case it is possible that the hydrocephalic foal lacked some neurological functions that would have aided in its appropriate orientation inside the uterus.
the fetus. This may be explained by the observation that nearly all equine fetuses spend the 6th and 7th months of gestation inside the uterine body and if they should die and be aborted during this stage of gestation their membranes will have the shape that is typically associated with that of a body pregnancy.

f) Bilateral horn pregnancy:
Just as the horns in a body pregnancy will look small and “non-fetal”, they may both have typical features of fetal horns. This has been seen in two cases, both of which presented as dystocia cases at full term. One foal was in dorsal transverse presentation (spine to the cervix), while the other was in ventral transverse presentation (feet to the cervix). Fig. 37 shows one of the fetal membranes, illustrating that both horns of the allantochorion looked typical of fetal horns and that the uterine body component of the fetal membrane was much shorter than normal.

![Cervical star](image)

Fig. 37: Fetal membranes of a full-term foal that was delivered by fetotomy, because the mare presented in dystocia with the foal in dorsal transverse presentation. The foal's front legs and head were inside the right uterine horn, while the hind legs were inside the left horn

**Examination of the Chorion**

Once the allantoic surface of the allantochorion has been examined, the umbilical cord (with the amnion attached to it) can be cut off at its placental implantation site. This greatly facilitates turning the allantochorion so that the chorionic (villous) surface comes to lie outermost. In fact, this is how the membrane was lying inside the uterus during pregnancy.

**Outermost Surface:**
If the membrane is first found with its villous surface turned to the outside it implies that the allantochorion was expelled virtually at the same time as the fetus, suggesting that one is dealing with another possible manifestation of premature placental separation – and all its potential consequences for neonatal well-being. Rarely, however, the membrane is passed at the
normal time without turning inside out. This happens more commonly in mares that remain recumbent during the period between delivery of the foal and passage of the afterbirth, presumably because there is then no traction effect of the amnion and umbilical cord that tends to turn the membranes inside-out in the standing mare.

1. Features of the Normal Chorion

a) Length and Density of Villi:
This can best be evaluated by submerging pieces of the allantochorion in water. It can then be seen that the villi and microcotyledons on the non-fetal horn are best developed, intermediate over the body and sparsest and shortest over the base of the fetal horn. During examination the non-fetal horn of the chorion will thus appear extremely velvety while the base of the fetal horn often looks rather pale and "flat". The reason for the differences in the development of chorionic villi is probably a physical one: The gland density (that determines villous development in the placenta) of the endometrium is uniform during early gestation while placentation takes place, but the uterine wall and placenta become stretched in portions of the uterus that need to expand to allow for maximal fetal growth during late gestation.

![Fig. 38: Chorionic surface of a typical afterbirth: Villi are least dense along the greater curvature of the base of the fetal horn (b) and most dense in the non-fetal horn (c). Images b and c were generated by submerging small pieces of membrane in water.](image)

b) Color:
Once the placenta has separated after parturition, the chorionic villi autolyze very rapidly, provided the fetal membranes remain at body temperature inside the uterus. Their color will change from bright red to tan in the process. Despite the fact that the fetal placental circulation no longer exists at this stage, it appears as though villi that remain attached to the endometrium still receive some oxygen (presumably by diffusion from the maternal endometrium), thus delaying their autolysis and change in color.
Those parts of the membrane that detach last are thus the reddest in color at the time when the fetal membranes are examined. In the normal chorion the areas near the base of the fetal horn and around the cervical star usually show the brightest red color (detached last), while the non-fetal horn is least red (detached first) and the remainder of the chorion is intermediate in color. The most autolyzed parts are usually also covered by a layer of autolytic fluid of pea soup consistency.

c) Allantochorionic Folds:
Prior to the onset of implantation at about Day 35 of gestation and until implantation is complete by about Day 70 all or parts of the allantochorion are free and may develop folds. Where such folds result in chorion overlying chorion no villi will develop. After parturition these folds can be recognized as slender, fusiform, sometimes stellate, areas devoid of any villi. In many cases one will find a major blood vessel running in the center of such a chorionic fold, suggesting that some tension on the major vessels (presumably from the weight of the fetus suspended from the umbilical cord) may have pulled the chorion away from the endometrium at the time of initial placental development between 35 and 70 days of gestation.

d) Cervical star, endometrial cup sites and utero-tubal junction sites:
Those areas of the chorion that lie against the folds of the uterine portion of the cervix, the uterine papillae (uterotubal junctions) or the sites of the endometrial cups are usually devoid of villi. In the normal afterbirth the cervical star is torn to allow for the passage of the foal.
Rubbing lesions:
During the last three months of normal gestation the fetal hind feet will be positioned very near the tip of the fetal horn. Fetal movement causes the feet to repeatedly rub against a specific point in this area and this may cause trauma and necrosis to the villi and disruption of the villous interface of the placenta in this area. The resulting “rubbing lesion” is usually most obvious in membranes of large foals.

2. Gross Lesions of the Chorion

a) Twin pregnancy:
When a large, clearly demarcated portion of the chorion is completely devoid of any villi a twin pregnancy should be diagnosed without hesitation. It is sometimes difficult, or even impossible, to find the co-twin or its afterbirth, because it may have mumified. Such mumies can be as small as a mouse and may have been overlooked when the products of an abortion or even parturition were removed from the site where the mare aborted/foaled. In other cases the mummy may be contained in a pouch formed by the invagination of the tip of the allantochorion of the surviving fetus.

Where the two chorionic surfaces of the membranes of twin fetuses are in immediate contact with each other tissue and vascular anastomoses may form, resulting in the delivery of fused membranes. In the rare event that such fetuses are carried to term, they are usually chimeras, i.e. they contain...
cells of both genotypes. Twinning is an important cause of reproductive wastage, but mares that abort a twin pregnancy usually have no trouble falling pregnant when bred back after the abortion.

Accurately identifying the cause of such an abortion is important when deciding on the mare’s reproductive future and the management of future pregnancies (mares that have conceived twins once are likely to do so again when bred again).

b) Hypoplastic or Too Few Villi:
It has been said that the chorionic surface constitutes a mirror image of the endometrium. Detection of sparse and hypoplastic chorionic villi during the examination of the afterbirth may thus be of significant prognostic value for the future breeding soundness of any mare. It is thus assumed that underdeveloped villi result from fibrosis and glandular atrophy of the endometrium (endometrosis) lying opposite the affected portions of the chorion. In order to attach any significance to the detection of seemingly hypoplastic villi one would have to see associated signs of starvation in the newborn foal or aborted fetus.

Note: Endometrial cysts, such as are regularly detected during the
ultrasound examination of the empty uteri of mares, are only very rarely associated with avillous or hypoplastic villi in the opposing area of the chorion, because most cysts are covered by reasonably healthy endometrium.

Retention of fetal membranes:
When the fetal membranes are retained for longer than the usual 5 – 60 min after delivery the autolysis of the chorionic villi will also continue for longer, resulting in an even duller appearance of the detached portions of the membrane. In areas that remain attached, however, diffusion of oxygen from the maternal endometrium will slow down autolysis of these portions. When the membrane is eventually released (spontaneously or after therapeutic intervention) the very stark contrast between areas of advanced autolysis and areas of very recent detachment will be easy to recognize. Many of these membranes appear mottled in color: most of the chorion is dull and grey with only patches of bright red villi where the membrane was still attached to the underlying endometrium.
Placentitis (see also previous discussion of placentitis under allantoic features): Bacterial or fungal placentitis results in necrosis of the villi in the affected area. Frequently a purulent, pseudomembranous exudate is present, too. In most cases the infection enters the pregnant uterus through a defective cervix, resulting in a localized lesion near the cervical star. When placentitis is diagnosed during the examination of the fetal membranes it is prudent to schedule a thorough evaluation of the mare’s cervix after uterine involution is complete. If the foal was born alive it may be born septicemic or develop septicemia during the neonatal period.

Some mares require treatment for endometritis after aborting or delivering a full-term foal while suffering from placentitis. As has been stated in the chapter on allantoic lesions, some cases of bacterial or fungal placentitis may involve areas other than the pericervical area surrounding the cervical star.

Placentitis is diagnosed with increasing frequency and accuracy in mares that have not yet aborted, but are threatening to do so. A variety of therapeutic interventions are applied in such cases, some of which are obviously effective in controlling or even curing the disease, while some mares cannot be prevented from aborting. If treatment is either effective or the mare cures the condition on her own, evidence of the partial destruction of the chorionic villi is likely to remain until the foal is born, because the regenerative ability of placental tissue is extremely limited (Fig. 48).
The Red Bag Membrane:
This subject has already been discussed under the heading of abnormal features of the allantois. The typical red bag delivery is associated with a uniformly bright red color of the entire surface of the chorion, because detachment of the membrane is usually followed immediately by its delivery, leaving little time for autolysis to set in. If the foal was stillborn its umbilical cord is likely to be unbroken as the membranes are shed soon after passage of the foal. The folds of the cervical star are often very thick and tenacious in red bag membranes, presumably because this tissue failed to undergo some ill-defined maturation process that normally occurs during the very last days of gestation and aids in the rupture of the cervical star at the end of the first stage of labor. A third feature of red bag membranes is that they are usually heavier than normal membranes. Their increased weight is caused by mild to severe edema of the allantochorion.

Some believe that the thick, tough star tissue and the edema of especially the pericervical portion of the allantochorion may be causes, rather than effects of the failure of the cervical star to rupture, but the author has seen all of the above changes in numerous membranes of perfectly normal mares that were induced to foal for teaching and/or experimental purposes. In the author’s opinion a red bag delivery near the time of a mare’s due date must always be considered to have possibly been caused by an abnormal event that lead to a late abortion or premature labor. Alternatively, one should try to identify possible causes for the disruption of the orderly endocrine and enzymatic cascade that prepares the mare, her birth canal, the fetus and its membranes for normal delivery. Conditions that have regularly been associated with most or all of the
features described as those associated with red bag deliveries include induction of labor with oxytocin in mares that showed no signs of first stage labor, the Mare Reproductive Loss Syndrome (in which cases the red bag delivery probably constitutes a late abortion, rather than normal labor at the normal time) and the toxicosis resulting from the ingestion of endophyte infested tall fescue grass.

Fig. 49: The Red Bag Delivery and associated fetal membranes:

a. Typical intrapartal presentation of an outspoken red bag birth in a mare that was induced to foal for teaching purposes.

b. Red bag delivery: Note the unusual amount of blood surrounding the foal and its membranes (bleeding was presumed to have resulted from the forceful, mechanical separation of the fetal and maternal villi). The umbilical cord of the foal did not break, because its membranes were passed immediately after it was born. The allantochorion did not turn inside out during its delivery. Note also the uniformly bright red color of the chorion.

c. The thick folds of an unbroken cervical star after a red bag delivery associated with an acute abortion.

d. Fetal membranes of a foal that suffered from severe neonatal maladjustment. The foal was born unexpectedly in the absence of an attendant. Note the extensive tears in the pericervical area of the allantochorion, the bright red color of the membrane and the fact that its chorionic surface was soiled by bedding material (evidence that the membrane failed to turn inside-out).

e. Massive edema in a red bag membrane that weighed 12.5 kg (25% of the foal’s birth mass).

f. Membrane from a red bag delivery that was resolved by incising the allantochorion to allow the foal to be born. Note the thick folds of the cervical star.