GENERAL INTRODUCTION

Reproduction is all about maintenance of the species, and is taken further in production systems to amplify a product, usually for profit. The whole reproductive process is a continuum, but we divide it into manageable sections to better understand or manipulate the processes. The general segments are achieving pregnancy (becoming fertile), maintaining pregnancy, neonatal survival and the successful attaining of puberty and fertility. I call this the circle of life.

Reproductive pathology is then divided into:
- disorders of sexual development
- female genital pathology
- male genital pathology,
- failure of pregnancy (embryonic mortality, abortion and stillbirth),
- perinatal mortality,
- mammary pathology and

We will investigate failure of pregnancy here.

Pregnancy is subdivided, for practicality, into the embryonic period, the fetal period, and the preparturient period – with death being embryonic mortality, abortion and stillbirth, respectively.

There are 3 fundamental questions to be answered when investigating failure of pregnancy:
1. What are our expectations?
2. Is the failure of pregnancy due to maternal, fetal or placental disease?
3. Is the failure of pregnancy due to (a) an infectious disease (with or without a lesion) or (b) a non-infectious disease (with or without a lesion)

Many people are cynical about investigating failure of pregnancy because of a perceived lack of positive results. There are many factors involved with failure of pregnancy and no one test is 100% effective. Identifying the cause is more likely in outbreaks of abortion (abortion storms) whereas diagnoses in individual (sporadic) cases are less likely. Failure to find a lesion or an infectious cause is not failure, but rather a step that eliminates one major cause of failure of pregnancy and directs the veterinarian toward managerial or other areas for the answer. Because pregnancy and parturition are highly intricate and often poorly understood processes, there will always be cases for which there is no detectable cause and therefore the failure of pregnancy will be truly idiopathic (no infectious agent responsible and no lesion found).

In this section we will deal with a general approach to the diagnosis of abortion in all species, and then each species will then be dealt with individually.

REFERENCE MATERIAL

There are few good sources of information about failure of pregnancy.
1. My notes and website (www.vetrepropath.com) has information that is updated annually.
2. There is a section on failure of pregnancy in ‘Pathologic Basis of Veterinary Disease’ (eds MD McGavin and JF Zachary).
3. Detailed information about individual diseases is in Pathology of Domestic Animals. 5th edn. (ed MG Maxie).
4. Buergelt, CD Color Atlas of Reproductive Pathology of Domestic Animals, Mosby Toronto has information also.
6. Your diagnostic pathology laboratory will provide local information on infectious disease.

EMBRYONIC MORTALITY

The conceptus is the product of conception and the embryo as that part of the conceptus that gives rise to the adult. Embryos become fetuses at the time when they develop features that allow their species and sex to be determined phenotypically. This occurs at about 35 to 45 days of age in large animals.

Embryonic mortality occurs when the embryo dies during this early stage of pregnancy. They are seldom found and the conceptus is unavailable for examination.

Embryonic mortality in the early embryonic period is often attributed to infections and post-partum or postbreeding endometritis. This could be from a recognised uterotrophic infection or from non specific infection and inflammation acquired at mating or after parturition.

Embryonic mortality in the late embryonic period can also be infectious, but it appears that chromosomal abnormalities account for many of these. This is based on limited studies in animals and extensive studies in humans were about 61% of spontaneous ‘abortions’ or miscarriage have chromosomal abnormalities. Geneticists recognise many traits where the adults are heterozygous – thus monozygosity is lethal!

INVESTIGATION OF FETAL DEATH

Fetal death later in development will lead to abortion, mummification or maceration. Mummified fetuses may be retained indefinitely if there is the only one fetus. Maceration requires bacterial infection of the fetus. It is common with Campylobacter fetus and Tritrichomonas foetus in cattle and with nonspecific endometrial infections.

Stillbirth is the death of the fetus in the last part of gestation during the period where the fetus is independently viable.
Regardless of which species is involved, investigation of fetal death requires a keen sense of what can be achieved. Depending on the situation, the investigation may have an economic impact, a zoonotic impact or appeal to your scientific curiosity. The investigation should not be restricted to examination of the fetus or fetal membranes. One should consider maternal factors also.

It is important to identify lesions and to correlate them with known causes. The corollary is that if an infectious agent is found, its significance must be assessed in light of the whole situation. One should also be clear about what one will do with the information!

THE POSTMORTEM

Examination of the fetus and membranes, and sampling of tissues primarily answers the question ‘are there any infections or fetal lesions or abnormalities to explain the abortion or stillbirth?’ Depending on the species, there may be little chance that the postmortem examination can help. This does not mean that examination is useless, only that it is a component of the investigation, and its usefulness must be assessed.

Some of the lack of success is due to the failure to submit the appropriate specimens. **Where feasible, send the whole fetus and membranes to the laboratory.** When this is not feasible, success in ruling out fetal factors depends on submission of the correct specimens, and this, in part, depends on knowing what specimens to send. **The appropriate specimens should be selected to diagnose the common or most important diseases and conditions.** Few laboratories charge by the tissue. **Therefore send samples of all tissues including brain, thyroids, thymus, and all the major organs.**

**Common causes of abortion**

Before we begin any investigation, we should be aware of the commonly diagnosed diseases in our area. This allows us to take the appropriate samples. How would you find out what is the most common in your area?

**Postmortem approach and findings**

There is some basic information that can be gleaned from examination of the fetus and placenta from each species. For the moment, we will consider changes in the ‘generic’ fetus – changes that are meaningful regardless of the species. A keen observation of the fetus is necessary to identify some of the changes.

The external appearance of the fetus provides important information. Record:
- weight of fetus and placenta
- size, including crown rump length
- degree of development for gestation length
- presence of meconium staining
- presence of dehydration
- localized swellings, such as swollen tongue and face
- other obvious abnormalities

**Figure 1. Stillborn sheep with swollen head and meconium staining of body. Dystocia.**

These factors may lead us to identify a problem or problems with nutrition, placental sufficiency, dystocia, and time of death before expulsion.

Fetal autolysis was studied in sheep and gives a rough guide in timing when death occurred **en utero.** Some people designate the time of death as either dead for a long time, or dead for a short time. This is adequate for many cases, but a more precise guide is as follows:

<table>
<thead>
<tr>
<th>Time since death</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>12 hours</td>
<td>cornea cloudy, amnionic fluid blood tinged</td>
</tr>
</tbody>
</table>
It is generally believed that an autolysed fetus died too quickly for it to initiate its own parturition – as would occur with fetal sepsis or viremia. A fresh fetus initiated its own parturition.

Fetal mummification occurs periodically in animals and has a common cause for each species as follows:

<table>
<thead>
<tr>
<th>Species</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horse</td>
<td>Twinning</td>
</tr>
<tr>
<td>Cattle</td>
<td>BVDV infection</td>
</tr>
<tr>
<td>Dog</td>
<td>Canine herpes virus</td>
</tr>
<tr>
<td>Cat</td>
<td>Torsion of a uterine horn</td>
</tr>
<tr>
<td>Sow</td>
<td>Parvoviral infection</td>
</tr>
</tbody>
</table>

It is important to examine and submit the brain, especially in ruminants.

**ABORTOGENIC (UTEROTROPIC) AGENTS**

There are a group of abortogenic agents that occur in almost all species. They include:

- *Brucella* sp
- *Campylobacter* sp
- *Chlamydia abortus*
- *Coxiella burnetii*
- Herpesviruses
- *Leptospira interrogans*
- *Listeria monocytogenes*
- *Mycoplasma and Ureaplasma*
- *Neospora caninum*
- *Salmonella* sp
- *Toxoplasma gondii*

**SAMPLES**

The samples to be collected can be divided into two basic categories; those that would sample diseases that came via the amniotic fluid, and those that affected the fetus directly.

Amniotic fluid bathes the external surface of the fetus and is swallowed. In distress, the fluid is inhaled into the lungs. Samples of skin, eyelids, lung and glandular stomach are indirect samples of the amniotic fluid.

The fetus is similar to an adult in the general processes that operate in disease, and we will not deal with all the possible diseases and processes. The fetus is inherently different in several aspects. To our knowledge, the fetus lives in a sterile environment, and has no ‘flora or fauna’. Contamination is possible as soon as it contacts the external environment, including the vagina. Fetuses are susceptible to maternal deficiencies such as iodine deficiency because they depend on ‘parasitizing’ the mother. The Great Lakes basin is iodine deficient and neonatal goitre is common in sheep and goats especially.

**REFERENCES**


Veterinary Laboratory Services Annual Report, Ontario Ministry of Agriculture and Food 1995/96

**FAILURE OF PREGNANCY IN THE MARE**

Examination of the equine placenta is arguably the most important part of the examination of the fetus and placenta. The overall pregnancy rate in mares is about 50%, and you are already aware of the estimation of the success of pregnancy based on uterine biopsy. Part of the accuracy of this test is probably the apparent lack of functional reserve in the equine placenta. This is also why birth of live twins is so rare in horses.

**AVILLOUS REGIONS IN DISEASE.**

Regions may lack villi in certain specific diseases. These include

- Twinning, where the connecting surfaces of adjoining placentas meet.
- Ascending infection, which is one of the common ways that agents gain access to the fetus and membranes in horses.
- Premature separation of the placenta, that occurs, in part, with cervical laxity or with body pregnancies.

![Figure 2. Large avillous area due to twinning.](image)

![Figure 3. Region of the cervical star with placentitis.](image)

![Figure 4. Premature separation of the placenta due to body pregnancy.](image)

**THE UMBILICAL CORD**

The equine umbilical cord is prone to develop abnormalities. The factors that control the length of the cord are not known although increased fetal movement and paternal genetic factors are involved. The cord should be between 36 and 83 cm long. Longer cords are associated with strangulation of the cord around the fetus, torsion of the cord and necrosis of the cervical pole of the placenta. A cord that is less than 36 cm is excessively short and may be associated with premature rupture in labour or with premature separation of the placenta at the site of attachment. The cord should have no more than 4 twists.
Figure 5. Torsion of umbilical cord.

The common causes of failure of pregnancy in horses are listed below. One half of the cases have causes that can be determined by direct examination of the placenta and umbilical cord.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Kentucky (%)</th>
<th>MSU</th>
<th>VLS/ AHL</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis</td>
<td>16</td>
<td>33</td>
<td>42</td>
</tr>
<tr>
<td>Noninfectious</td>
<td>50</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Dystocia</td>
<td>19</td>
<td>19</td>
<td>4</td>
</tr>
<tr>
<td>Congenital defects</td>
<td>10</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Placental separation</td>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Twins</td>
<td>6</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Cord abnormality</td>
<td>3</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>Body pregnancy</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infectious</td>
<td>34</td>
<td>31</td>
<td>37</td>
</tr>
<tr>
<td>Placentitis</td>
<td>10</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>EHV</td>
<td>4</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Bacterial</td>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Leptospiral</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Fungal</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Nocardia</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Other important causes
- Insufficient serum progesterone
- Fescue toxicosis (*Neotyphodium coenophialum*)
- Thyroid hyperplasia/musculoskeletal syndrome
- Mare reproductive loss syndrome (processionary caterpillar)

Infectious causes of failure of pregnancy are conveniently divided into those caused by Equine Herpesvirus type 1 (EHV-1) and others! Most of these ‘others’ can be diagnosed by examination of the cervical star. Most of the cases are related to ascending infection from the vagina because of the cervical anatomy of the horse. Conformational problems in older mares can predispose to greater contamination of the cranial vagina. *Streptococcus zooepidemicus* is a common isolate from bacterial placentitis in horses. Infection and inflammation of the placenta releases proinflammatory cytokines that cause fetal ‘distress’ and abortion.

Equine herpesvirus is an important cause of abortion and there are prominent changes in the fetus. One or more of the following may be found
- fibrin cast in the trachea
- interstitial pneumonia
- focal necrosis in the liver
- prominent lymphoid follicles in the spleen.

Abortion is caused by EHV-1 and occasionally by EHV-4. Introduction of an infected mare to a group of naïve mares is a common way to begin an outbreak. Vaccination against equine herpesviral abortion is with Pneumabort, a killed vaccine that requires multiple injections. It probably reduces the prevalence of abortion, but vaccinated mares may still abort.

Routinely weighing the fetus and placenta (separately, normal ratio is 10:1) is advantageous to identify placental edema – an indication of fescue toxicosis.

Examination of the aborted equine fetus and placenta is a very rewarding procedure. Your likelihood of obtaining a diagnosis is excellent.

Review questions
1. What part of the chorioallantois would you routinely sample when investigating an abortion?
2. Mummification of a fetus in mares is associated with what situations
3. How would you determine if there were torsion of the umbilical cord and what would you expect to find in the fetus to confirm this?
4. What is the difference between an allantoic pouch and a chorioallantoic pouch?
5. Why does *Streptococcus zooepidemicus* cause abortion in mares when it only affects a small part of the placenta?
6. What is a hippomane?
7. When is placental mineralization abnormal?

References


FAILURE OF PREGNANCY IN CATTLE

The common causes of abortion and stillbirth that can be identified by examination of the fetus and placenta are as listed below. Caution in interpreting these results is warranted. Some agents cause failure of pregnancy but are not found in the fetus or placenta – BVDV is one notable example. Abortion occurs 10 to 27 days after infection, but there may be no macroscopic or histological lesions, and virus isolation is not always successful.

ABORTION

<table>
<thead>
<tr>
<th>Cause</th>
<th>CVDLS</th>
<th>PEI*</th>
<th>AHL</th>
<th>Dakota</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis</td>
<td>57</td>
<td>52</td>
<td>58</td>
<td>67</td>
</tr>
<tr>
<td>Noninfectious</td>
<td>6</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Infectious</td>
<td>37</td>
<td>46</td>
<td>42</td>
<td>30</td>
</tr>
<tr>
<td>Bacteria</td>
<td>18</td>
<td>24</td>
<td>17</td>
<td>16</td>
</tr>
<tr>
<td>Viral</td>
<td>3</td>
<td>6</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Protozoal</td>
<td>15</td>
<td>2</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Fungal</td>
<td>1</td>
<td>7</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Ureaplasma</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

* One study separated stillbirths from Abortions.

STILLBIRTH

<table>
<thead>
<tr>
<th>Cause</th>
<th>CVDLS</th>
<th>PEI</th>
<th>AHL</th>
<th>Dakota</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis</td>
<td>32</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noninfectious</td>
<td>62</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dystocia</td>
<td>23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anomalies</td>
<td>23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amnion fluid aspiration</td>
<td>16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nutritional myopathy</td>
<td>7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infectious Bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Viral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protozoal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fungal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ureaplasma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Other important causes

- Brucella abortus
- Foothills abortion
- Ponderosa pine abortion
- Campylobacter fetus
- Tritrichomonas foetus

Clearly, of the diagnosable diseases, infectious disease is more important in the bovine species. Of
late there has been a quantum shift in the diagnosis of these diseases, with the discovery of the protozoan *Neospora caninum* in most locales. There are regional differences in the most common causes of abortion, so one must be familiar with what to expect in the area that one practices.

The comforting news about diagnosing abortion in cattle is the similarity of findings in many of the cases and the reliance on microbiological examination. Despite this, pathogenic organisms must be differentiated from contaminants. Identifying lesions is relatively straightforward, because the lesions are stereotyped.

Examination of the placenta is primarily focused on identifying lesions that are caused by infectious agents.

Basic observations should include

- freshness
- placental and fetal weight
- distribution of lesions, both within each membrane and between membranes

Placental weight is usually about 14% of fetal weight. Placental insufficiency would cause this ratio to be lower.

The distribution can assist in determining the route of entry of the agent. The usual possibilities include ascending infection through the cervix, descending through the uterine tube, and random multifocal if hematogenous. *Ureaplasma diversum* typically causes amniotic lesions. Also, because the amnion and chorion are fused over a part of their surface area, infecting agents have a more direct access to amniotic fluid than is the case in the horse, cat and dog.

Abnormalities of the placenta seen in disease include

- necrosis and infarction
- fibrin exudation
- edema
- cupping of the cotyledon
- fibrosis and thickening
- exudation onto the surface

- mineralization
- vasculitis

Figure 6. Lesions of placentitis in cattle are stereotyped.

**LESIONS OF SPECIFIC DIAGNOSTIC SIGNIFICANCE**

Several lesions may be found in fetuses that may carry diagnostic significance. Heart failure as a cause of abortion is associated with BVD myocarditis, *Neospora* myocarditis, or nutritional myopathy (Vitamin E / Selenium deficiency). Dermatitis is often associated with mycotic abortion, and amnionitis is seen in *Ureaplasma* abortion. Focal necrosis in the liver or other tissues suggests herpesviral infection (IBP/IPV).

**SAMPLING FOR MAXIMUM EFFECTIVENESS**

Samples collected for investigation of infectious causes of abortion should include the tissues most often affected; bovine brain and heart especially. In addition, tissues that sample amniotic fluids are a must to collect.

**Review questions**

1. How does one find out the most common cause of abortion in the area?
2. What samples must be taken to ensure a diagnosis of neosporiosis?
3. How does one determine if an agent recovered from a fetus or membranes is pathogenic?
4. Can a caruncle be sampled without damaging the cow?
5. What does the distribution of lesions in fetuses infected with *Ureaplasma* or *Mycoplasma* sp. tell us about the route of infection?
6. How does one differentiate autolysis of a placenta from necrosis?
7. What can cause placental edema?
8. What lesions would you expect to see in fetal heart failure?

References

**PREGNANCY FAILURE IN SHEEP**

The first steps in investigating failure of pregnancy in sheep are to answer these questions:

1. What are our expectations?
2. Is the failure of pregnancy due to maternal, fetal or placental disease?
3. Is the failure of pregnancy due to infectious causes?

**WHAT ARE OUR EXPECTATIONS?**

The likelihood of finding the cause of an abortion outbreak or storm is much greater than in a sporadic abortion.

Data from diagnostic laboratories indicates that about 50% of submissions yield an infectious cause. The likelihood of finding a cause is much greater if the placenta is available for evaluation.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Dakota</th>
<th>AHL</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis</td>
<td>56</td>
<td>48</td>
</tr>
<tr>
<td>Noninfectious</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Infectious</td>
<td>39</td>
<td>50</td>
</tr>
<tr>
<td>Placentitis</td>
<td>25</td>
<td>6</td>
</tr>
<tr>
<td><em>Campylobacter</em></td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td><em>Chlamydia</em></td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td><em>Toxoplasma</em></td>
<td>11</td>
<td>19</td>
</tr>
<tr>
<td><em>Coxiella</em></td>
<td>0.1</td>
<td>7</td>
</tr>
</tbody>
</table>

Other important causes
- Cache Valley virus
- *Brucella ovis*
- Iodine deficiency
- *Listeria monocytogenes*
- Rift Valley fever
- *Salmonella enterica* subspecies *enteric* serovar Abortusovis
- Schmallenberg virus
- Wesselbron

**IS THE FAILURE OF PREGNANCY DUE TO MATERNAL, FETAL OR PLACENTAL DISEASE?**

Because in sheep the most common location for lesions is the placenta, the most recognised of causes is placental disease. It is very important that any investigation includes examination of the placenta.
Placentitis and multifocal placental cotyledonal necrosis are the main lesions.

Fetal lesions occur periodically. Iodine deficiency and the resultant goitre is a regional disease.

**IS THE FAILURE OF PREGNANCY DUE TO AN INFECTION**

The big four causes of infectious abortion in sheep are *Chlamydia abortus*, *Campylobacter* sp, *Toxoplasma gondii* and *Coxiella burnetii*, Each will be discussed in detail below.

**CHLAMYDIA ABORTUS ABORTION**

Of the infectious causes of abortion, *Chlamydia abortus* is very important, both as an agent and as a potential zoonosis. Chlamydial abortion is also called enzootic ovine abortion, and the lesion is chronic placentitis.

![Figure 7. Placentitis in a sheep.](image)

Ewes are infected through mucous membranes including those of the mouth, conjunctiva and reproductive tract. The source of the agent is aborted fetuses and uterine discharge, vaginal secretions of carrier ewes at estrus or there is spread through the prepuce and semen of rams (at least temporarily). After exposure to *Chlamydia abortus*, sheep develop antibody, but infection can be detected for a month or more as interstitial pneumonia or focal hepatitis. The organism can be found in the mononuclear cells of the uterus. From there they infect the epithelial cells of the placentomes, and control of infection is by neutrophilic infiltrates. Trophoblasts in the periplacentomal region become infected and there is a logarithmic increase in numbers, with subsequent necrosis and inflammation of the placenta. Maternal inflammation becomes restricted to lymphocytic inflammation around the endometrial cells of the uterine glands. It is assumed that this immune reaction prevents infection of the placenta in subsequent pregnancies.

The incubation period during gestation is very long -- from 50 to 90 days. If a ewe is infected early in gestation, she will abort in the same gestation (gestation is 145 days). If infection occurs in late gestation she will abort during the next pregnancy. In general, once a ewe aborts, she will not abort a second time. She may remain a carriers for several years however.

Introduction of infected replacements is a common way to introduce infection into a flock and to begin an abortion storm. In the first year following introduction there may be a few animals abort but most of the flock will become infected. Abortions normally occur the following year with up to 75% of pregnant ewes aborting. In subsequent years the disease becomes enzootic and only first time lambers abort.

Diagnosis of infection can be made on aborted tissues, and serology is possible but it is very expensive. There is a low level of false positives and there is some cross reaction with *Chlamydia pecorum*, a *Chlamydia* found in the faeces.

Treatment in the face of an outbreak of *Chlamydia* abortion includes the use of long acting oxytetracycline. This should be repeated every 10 to 14 days. Orally administered tetracycline is an alternative. Because of the long incubation period, there is usually a poor to moderate efficacy of this treatment.

Vaccination is possible with a killed vaccine but it is not readily available and has a variable efficacy.

**References**


**CAMPYLOBACTER ABDOTION**

Abortion due to *Campylobacter fetus* subsp *fetus* and *Campylobacter jejuni* causes an intercotyledonary placentitis with abortion and stillbirths, and the birth of weak lambs. Ewes are infected through the feco-oral route and carriers have the bacterium within the gall bladder. Aborted fetuses and placentae are a source of infection, but outbreaks are not as dramatic as with chlamydirosis. The incubation is 7 to 60 days, and abortion can 'cycle' within a lambing season.

Control of abortion in the face of an outbreak can be achieved with a good degree of success with long acting oxytetracycline or oral tetracycline. There is a vaccine for prevention (given prior to breeding) and control (in the face of an outbreak).

**TOXOPLASMA GONDII**

Toxoplasmosis can play a major role in ovine abortion. The lesions are grossly visible focal necrosis in the cotyledon and histologic changes in the brain of fetuses.

![Figure 8. Focal necrosis of cotyledons in Toxoplasma gondii infection.](image)

The source of infection is usually infected cats which pass oocysts in their faeces for approximately seven days. Oocysts can remain infective for six months. Naive ewes will develop a protective immunity but if they are pregnant, infection of the cotyledon will result in abortion.

Control of toxoplasmosis involves removing the source of oocysts. Control of rodents and cats and their access to feed and pasture is particularly important. Even this will not guarantee complete control as purchased feed may be contaminated. Feral cats are very difficult to control also!

Prevention of infection or abortion can be achieved by including monensin or decoquinate in the feed throughout gestation.

**OTHER INFECTIOUS DISEASE IN THE CANADIAN CONTEXT**

**Brucella ovis**

Ovine brucellosis is rare in the eastern United States and Canada. It is an important cause of disease in rams in a both Western US and Western Canada. The primary presenting condition is epididymitis of rams. In most flocks, abortion occurs only occasionally. Infection of ewes is usually by infected rams at mating and the infection is cleared spontaneously. It is possible for outbreaks to occur and to have an abortion storm of up to 70%.

Introduction of infection into a flock usually occurs with the introduction of an infected ram. Control of the disease is the best achieved by elimination of infected rams using scrotal palpation and serology outside of the breeding season. In flocks that are heavily infected, vaccination may be helpful but it is only available in the USA. It is important to prevent infection and this requires purchasing rams only from flocks known to be *Brucella* free.

**Border disease**

Border Disease Virus (BDV) and Bovine Viral Diarrhea Virus (BVDV) are very closely related, as are the diseases they cause. Infection of a pregnant ewe can result in fetal loss, birth of weak lambs or birth of lambs that are immune. Infection at less than 65 days of
gestation results in embryonic death. Infection at 65 to 85 days of gestation can result in the birth of a persistently infected lamb that may be weak, have a hairy coat and shake (thus the clinical manifestation of a "hairy shaker"). Ewes infected after 85 days of gestation will give birth to normal lambs with titres to BDV.

The control of border disease revolves around reducing exposure of naive individuals during pregnancy. Persistently infected sheep provide a source of infection so identifying and removing them from the flock is essential. Border disease is a self-limiting disease if persistently infected animals are identified and removed. They can be identified by virus isolation from the blood (buffy coat) of infected animals. Vaccination of ewes with a killed BVDV vaccine is not effective as there is no cross protection.

**PREGNANCY FAILURE IN GOATS**

Abortion in goats is very similar to sheep, except that *Coxiella* and trauma and stress are more common.

<table>
<thead>
<tr>
<th>Cause</th>
<th>AHL</th>
<th>CVDLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis</td>
<td>52</td>
<td>53</td>
</tr>
<tr>
<td>Noninfectious</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Infectious</td>
<td>40</td>
<td>37</td>
</tr>
<tr>
<td><em>Coxiella</em></td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td><em>Chlamydia</em></td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td><em>Toxoplasma</em></td>
<td>17</td>
<td>4</td>
</tr>
<tr>
<td>Bacteria</td>
<td>3</td>
<td>7</td>
</tr>
</tbody>
</table>

Other important causes

*Brucella melitensis*

Iodine deficiency

**COXIELLA ABORTION**

*Coxiella burnetii* (Q fever) is a very important disease of sheep and goats, and especially of people. It is less common in sheep. The pathogenesis of Q fever begins with the organism. It is an obligate intracellular gram-negative bacterium that is closely related to *Legionella*, *Francisella* and rickettsias. It is highly resistant to physical and chemical degradation, and is infective in very low doses – as low as one infectious particle – making it the most infectious agent known. Aerosolisation as ‘dust’ is significant. Naïve animals are infected by aerosol or from infected unpasteurised milk. After primary replication in local lymph nodes, it becomes bacteraemic and localizes in the mammary gland and pregnant uterus. It can be found in large numbers in amniotic fluid and in the placental membranes, especially trophoblasts. It will cause placentitis and abortion, but some placentas have no lesions. Some animals become carriers and shed the organism in milk and in uterine fluids. Shedding in the periparturient period is especially common.

A naive goat herd may have up to 50% of pregnant does abort. There is a very significant zoonotic risk. Goats can be persistently infected and shed the organism at subsequent kidding. Because the organism can be highly infective in a dried state, it is dust born, and barns and sheds become
contaminated and can be potentially infective for many years. Infection of the premises can also occur with carrier animals including sheep, cattle, cats, birds and other wildlife.

Treatment in the face of an abortion storm can be achieved through the use of oxytetracycline as with chlamydiosis. Special precautions are required for any infective environment and when assisting kidding. The use of gloves, appropriate masks and eye protection while assisting in kidding or cleaning barns is strongly advised. Children, elderly and pregnant individuals should stay out of the barn.

References.

Miscellaneous causes of failure of pregnancy

Does maintain their corpus luteum for the entire pregnancy and are more prone to luteolysis following stress and or trauma such as dog or coyote attacks.

Pregnant goats in close proximity to cattle persistently infected with BVDV may abort and BVDV is recoverable from some. Abnormalities are similar to those seen in BVDV infection of fetuses in cattle and BDV in sheep.

Pregnancy failure in dogs and cats

The placenta of the dog and cat is zonary and therefore is circumferential. Sampling of the zonary portion is best done to include the non-zonary chorion. Likewise, when sampling the uterus after ovariohysterectomy include the placenta and adjacent uterus.

Table 1. Maternal database for the investigation of FOP in dogs and cats

<table>
<thead>
<tr>
<th>Test bitch for</th>
<th>Test queen for</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brucella canis, Toxoplasma spp. Neospora spp.</td>
<td>Feline herpesvirus Feline calicivirus Feline immunodeficiency virus Feline leukemia virus</td>
</tr>
</tbody>
</table>

There is little published information about the major causes of abortion in dogs and cats. The rate of stillbirth in cats about 7%. The identification of the cause of abortion in a dog or cat, or a cattery or kennel of dogs, requires considerable and often expensive investigation. Most veterinarians concentrate on the infectious diseases because they are often easier to confirm or reject than some of the other causes. Of the infectious diseases in dogs, infection with Streptococcus canis, an opportunistic pathogen, is probably the most common. Salmonella spp is often overlooked.
Investigation of the cause of abortion includes considering the history, physical examination and results of ancillary tests outlined in the table.

Submission of maternal serum and whole fetuses and placentae is preferred. If this is not feasible, a complete set of tissues for histopathology, bacteriology, mycoplasmology and virology should be collected. Specific requirements can be obtained from each laboratory.

Table 2. Causes of FOP in dogs

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Brucella canis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Campylobacter</td>
</tr>
<tr>
<td></td>
<td>Mycoplasma/Ureaplasma</td>
</tr>
<tr>
<td></td>
<td>Salmonella</td>
</tr>
<tr>
<td></td>
<td>Streptococcus canis</td>
</tr>
<tr>
<td></td>
<td>Other</td>
</tr>
<tr>
<td>Viruses</td>
<td>Canine distemper/hepatitis</td>
</tr>
<tr>
<td></td>
<td>Canine herpesvirus</td>
</tr>
<tr>
<td>Protozoa</td>
<td>Toxoplasma</td>
</tr>
<tr>
<td></td>
<td>Neospora</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Progesterone deficiency</td>
</tr>
<tr>
<td></td>
<td>Hypothyroidism</td>
</tr>
</tbody>
</table>

Table 3. Causes of FOP in cats

| Viruses                  | Feline calicivirus |
|                         | Feline herpesvirus |
|                         | Feline immunodeficiency virus |
|                         | Feline leukemia virus |
| Bacteria                | Salmonella        |

Reference
