Uterine defense mechanism:

Uterine defense mechanisms against contaminant micro-organisms were maintained in several ways:

Anatomically : by the simple or pseudostratified columnar epithelium covering the endometrium, uterine muscles, natural barrier (cervix, vestibule and valve)

Chemically :by mucus secretions from the endometrial glands and hormones (estrogen).

Immunologically: through the action of polymorphonuclear inflammatory cells and humoral antibodies, but the degree of interaction is not clear.

Disruptions of these mechanisms allow opportunist pathogens, mostly microorganisms found in the posterior gastro-intestinal tract and around the perineal area, to colonise the endometrium and cause an endometritis.

Under normal circumstances, there are several mechanisms, which prevent pathogens from colonizing the genital tract.

The major **anatomical barriers** between the contaminated world and the relatively sterile environment of the uterus, include the vulva, the vestibule (guarded by a muscular sphincter), and the cervix.

It should be noted that, although the vulva may appear of little consequences as a barrier, it is, in fact, remarkably efficient at preventing faecal contamination of the tubular genitalia. In cattle, the cervix is formidable barrier composed of series of mucosal lined collagenous rings.

In addition, the cervical-vaginal mucus (especially the scant, tenacious mucus of the luteal phase) can function as a physical barrier for organisms that would otherwise ascend the reproductive tract.

The **circular and longitudinal** layers of the uterine musculature provide physical propulsion of particular material, including microbes.

Epithelial cells are the first to make contact with potential pathogens that enter the uterus. Epithelial and stromal cells interactions are critically important for endometrial function, with stromal cells affecting epithelial cells through both the release of soluble factors and turns over of extra cellular matrix. Conversely, epithelial cells affect stromal cells function through the release of soluble factors and cell to cell contact PGE2 regulate epithelial cells proliferation and is mediated indirectly by uterine stroma.

Estradiol and progesterone have both opposing and complementary effects on the female genital tract with estradiol stimulating epithelization (especially of the vaginal lining and endometrial gland), and vascularization of the endometrium, and increased production of cervical mucus and oviductal secretions, enhancement of uterine contractility, initiation of sexual receptivity.

Cattle are resistant to uterine infections when progesterone concentrations are basal and they are susceptible when progesterone concentrations are increased.

The high estradiol concentrations that occur at estrus and parturition cause changes in number and proportions of circulating white blood cells.

Moreover, at estrus, the blood supply to the uterus is increased under the influence of estradiol, whilst at parturition there is a massive blood supply to the gravid uterus. This increased blood supply, coupled with the migration of white cells from the circulation to the uterine lumen, enables vigorous and active Phagocytosis of bacteria to occur. Estradiol also causes an increase in the quantity and nature of vaginal mucus, which also plays an important role in defense of the uterus against bacteria by providing a protective physical barrier and by flushing and diluting the bacterial contaminants.

Puerperal Metritis (Acute septic metritis)

Puerperal metritis is inflammation of the uterus which involved all layers of the uterus (endometeriom,myometeriom and serosa) and it is affect the general health of the cow and occurs within a few days of parturition.

It usually follows an abnormal first or second stage of labour, especially when there has been a severe dystocia.

Causes:

- The disease associated with parturition problems like uterine inertia, twin births, RFM, prolonged traction and damage to the vulva and/or birth canal.
- Bacteria colonize the non-involuted uterus, producing toxins which are absorbed and cause severe symptoms.
- The most important infecting organisms are *Arcanobacterium pyogenes*, group C streptococci, haemolytic staphylococci, coliforms, and Gram-negative anaerobes, particularly *Bacteroides* spp.
- In rare cases, clostridia are present which rapidly produce disease that is serious and often fatal.

Clinical signs:

- Affected animals show both local and general symptoms.
- It is very common for toxemia, septicemia and pyrexia to occur.
- The temperature of affected cows may be elevated to 40–41C, but is more often subnormal.
- There is a rapid pulse rate (in the region of 100/minute) and the respirations may be sufficiently frequent to suggest a respiratory disease.
- Animals are anorexic and dehydrated; they often have a toxemia-induced diarrhea and exhibit signs of shock.

- It is common for the infection to extend through the uterine wall into the peritoneum, causing a localized or generalized peritonitis.
- The uterus contains large volume of toxic, fetid, reddish, serous exudates, containing pieces of degenerating fetal membranes; the exudates is discharged from the vagina by frequent expulsive straining efforts.
- Vaginal and uterine exploration of an affected case causes acute discomfort and is accompanied and followed by the most severe and persistent expulsive efforts.
- The cotyledons are swollen and the fetal membranes often remain firmly attached.
- The vulva and vagina are swollen and deeply congested.

D.D.:

Puerperal metritis must be differentiated from (primary) pneumonia, traumatic reticulitis and pericarditis, and from milk fever and acute mastitis.

Many animals with puerperal metritis also develop mastitis, particularly if they are recumbent, and many also have concurrent hypocalcaemia.

Treatment:

- The treatment of puerperal metritis requires both good nursing care and vigorous medication.
- The cow should first be kept warm and made as comfortable as possible by, for example, transferring it to a well-bedded and warm loose-box.
- An attempt should be made to remove the fetal membranes by very gentle external traction, but no attempt should be made to enter the vagina and uterus with the hand.
- It should be appreciated that the uterus is particularly friable and that it contains a voluminous mass of septic material, therefore Rough attempts at removal of the fetal membranes or even careful exploration of the vagina and uterus can cause severe damage and predispose to the absorption of toxins and entry of bacteria.

- If the case is seen within 2–3 days of parturition, 50 i.u. of oxytocin by intravenous injection may cause contraction of the uterus and expulsion of fluid and debris.
- The disease is best treated by systemic administration of broad-spectrum antibiotics and supportive therapy. The choice of antibiotic and the route of its administration have been the subject of much debate.
- Intrauterine infusions of tetracyclines may be effective against mild cases of endometritis, but they do not penetrate far enough into the uterine wall to be effective against full-thickness metritis.
- Systemic broad spectrum antimicrobials, fluid therapy and nonsteroidal anti-inflammatory drugs are widely recommended.
- The use of estrogens is contraindicated in cases of acute puerperal metritis since(why) , although they potentially increase the resistance of the genital system, oestrogens also increase the blood flow to the uterus and, thereby, increase the absorption of bacterial toxins.
- Once the temperature approaches normal and the cow shows some signs of improvement, some benefit can be obtained by uterine lavage and drainage. This can be done with a wide-diameter, soft rubber tube. The perforated end is carefully inserted through the cervix into the uterine lumen and several liters of warm (49 C) sterile saline are poured down the tube through the funnel. The funnel end is quickly lowered before the tube empties, thus establishing a siphon, until the uterus is as empty as possible.
- The warm saline solution is believed to exert both a soothing and a stimulating effect on the uterus, and this, together with the evacuation of exudates, promotes involution.
- Ideally, the patient should be given daily treatment as outlined above.

Retained fetal membranes (RFM)

Usually the placenta is passed in 3-8 hours after calving. If it has not passed after 12 hours the placenta is retained and the animal should be treated.

It is a common complication of bovine parturition and, although of little consequence per se, its role in predisposition to infections of the uterus means that retention of the fetal membranes is an important contributor to bovine infertility.

Aetiology

Detachment of placenta in the cow involves separation of the finger-like cotyledon villi from the caruncle crypts without significant tearing of either fetal or maternal epithelia.

Detachment of the fetal membranes indicates that uterine involution is progressing normally. Involution of the uterus is accompanied by a massive breakdown of collagen and other proteins. Lack of cotyledon proteolysis (collagenolysis) appears to be the underlying cause of RFM.

Retention of the fetal membranes occurs when the normal processes of dehiscence and expulsion fail to take place.

There appear to be three main factors involved in the separation and expulsion of the fetal membranes, namely:

- maturation of the placenta
- Collapse and shrinkage of the cotyledons which lead to physical separation from the maternal crypts.
- Uterine contractions.

Main causes:

1- ***Premature birth*** is very commonly associated with RFM. Cattle twins are usually slightly premature; hence, their birth is often followed by retention. heat stress can reduce gestation length and increase the incidence of RFM in dairy cattle.

2- ***Placentitis.*** Both placentitis and RFM occur in cases of abortion due to *Brucella abortus,Campylobacter fetus* and moulds such as *Aspergillus* or *Mucor* spp. Inflammatory swelling could affect the physical union between the maternal caruncle and fetal cotyledon.

3- ***Uterine inertia.*** Uterine inertia is frequently suggested as a predisposing factor for RFM.

4- ***The immune system.***

RFM may be related to failure of the release of inflammatory mediators. Deficient neutrophil phagocytic activity, decreased migration, and decreased superoxide anion production have been proposed as factors in the pathogenesis of RFM in cattle. Moreover, leukocytes are a mobile source of collagenases and may be involved in uterine regression and release of placenta.

5- Other factors.

- There is some evidence of a hereditary predisposition to RFM. Cows of the beef breeds are much less often affected than those of dairy breeds, and in the latter the incidence is higher in Ayrshires than Friesians.
- Old cows are more affected than young ones.
- Springtime calving exerts a predisposing influence.
- vitamin A , E or selenium deficiency.
- There is evidence of a high incidence of RFM in areas deficient in selenium.
- Older cows showed no benefit from the exercise.

Clinical features

- It should be noted that cows which fail to expel the fetal membranes within 36 hours or so are likely to retain it for 7–10 days.
- Myometrial contractions largely cease from 36 hours after the birth of the calf, so, if the membranes have not been expelled by this time, freeing of the fetal villi from the maternal crypts eventually occurs as a result of autolysis and bacterial putrefaction. This process starts within 24 hours of birth but takes several days to complete.
- The toxic products of putrefaction accumulate within the uterus causing a fetid odour which pervades the atmosphere and, more importantly, taints the milk.
- Delayed involution of the uterus and a variable degree of metritis commonly accompany retention.
- RFM increased the calving to conception interval, the number of services per conception and the culling rate, whilst reducing milk yield (probably because of reduced appetite).
- When retention is accompanied by metritis, the symptoms depend upon the severity of the uterine disease. As described earlier, severe disease is accompanied by increased pulse and respiratory rates, raised temperature, anorexia, diarrhoea, depression, reduced milk secretion, straining, fetid vaginal discharge and, occasionally, laminitis.

Treatment

The treatment of animals with retained fetal membranes has long been a contentious subject.

- manual removal
- administration of ecbolic agents (oxytocin, PGF2α)
- no treatment

Manual removal. The techniques used for manual removal of RFM range from externally applied gentle traction, through to forced extraction and separation of each cotyledon and caruncle.

In this method, the post-cervical portions of the placenta were twisted together into a 'rope', then a hand was inserted into the uterus and each cotyledon was squeezed out of the base of the maternal caruncle. Continuous steady traction and rotational force were applied with the other hand to withdraw the detached membranes.

Even when this procedure is undertaken with careful cleansing of the perineum and as high a standard of asepsis as possible, it causes considerable damage to the uterus.

Most evidence shows that manual removal of fetal membranes has a detrimental effect upon fertility.(https://youtu.be/ACLxATxexhs)

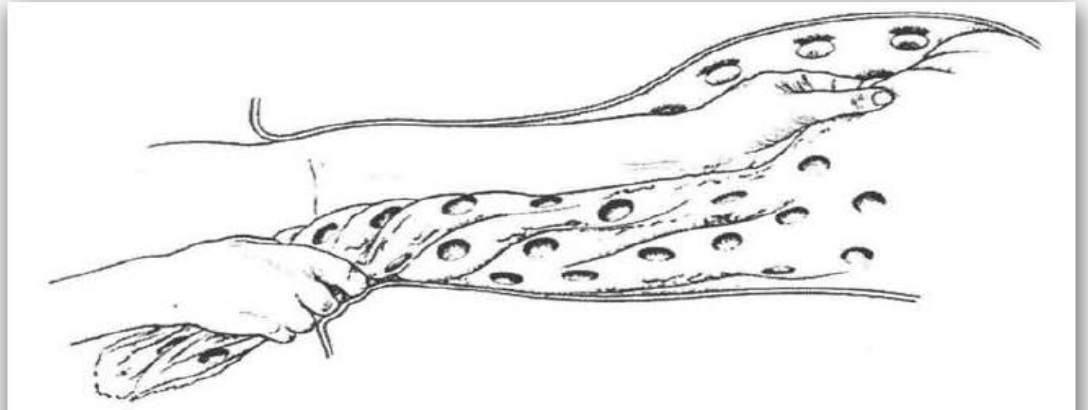

Fig. 37.1 : Manual removal of the placenta in a cow. The obstetrician grasps any protruding strand of placenta in one hand twists it into a 'rope' so that the placenta can be more easily managed. The other lubricated hands is introduced into the uterus. The hand should be inside the uterus but outside the placenta and the nearest attached caruncles and cotyledons should be searched for. (Courtesy of Jackson, P.G.G. 1995. Handbook of Veterinary Obstetrics. W.B. Saunders Company Limited).

Ecbolic agents.

The most rational measure for both the prevention and treatment of RFM would be to stimulate adequate myometrial contractions so that a 'natural' dehiscence and expulsion could occur.

1- the administration of 10 ml (100 i.u.) of oxytocin.
2- In order to attempt to achieve a more reliable response to oxytocin, oestrogenic substances have also been given, in the hope of both increasing the sensitivity of the myometrium to oxytocin and enhancing the natural uterine defense mechanisms. For these reasons, the synthetic oestrogens, stilboestrol dipropionate and oestradiol monobenzoate, have been widely applied to cows with RFM in the form of parenteral injection, or uterine infusion and pessary, and their use has sometimes been followed by injections of oxytocin .
3- Prostaglandin F2α and its derivatives have been used as ecbolic agents.
Prostaglandins may assist in detachment of the membranes through direct actions upon the placentomes rather than just by an ecbolic action.

No treatment.

Some authors were convinced, by the poor response to manual removal and the dubious effects of ecbolic agents, that uncomplicated cases of RFM require no treatment. The manual removal of the placenta can create uterine trauma and delay the return to normal reproductive status (Bolinder et al., 1988). It appears better to allow the placenta to separate of its own accord or to withdraw it gently from the uterus 7-10 days post calving.

Use of oxytocin is of questionable after 24 hours of calving because by this time, the response to oxytocin becomes poor. Unfortunately in cattle practice, generally veterinarian is not consulted until after 24 hours of retention of placenta because until then the farmer has hoped for a spontaneous expulsion.

If the cow is ill with metritis, antibiotic treatment and/or uterine drainage are probably indicated.

PROLAPSE OF THE UTERUS

Incidence Occurs in all the large animal species. It is most common in the cow and ewe, less common in the sow and doe goat, and rare in the mare. Normally the uterus prolapses only after fetal delivery but occasionally in the sow one uterine horn may prolapse while the other – still containing a number of fetuses – remains within the abdomen. In cattle the condition seems to be more common in fat animals with excessive slackening of the pelvic ligaments and perineal tissues. 'Outbreaks' occur on some farms during one calving season and may be associated with diet, possibly with a high estrogen content.

Etiology Uterine prolapse is essentially an eversion of the organ, which turns inside out as it passes through the vagina as a prolapse. Many factors may be involved in the etiology, including:

- Poor uterine tone: uterine inertia – in cattle hypocalcemia (a cause of primary uterine inertia) may predispose. Lack of tone may allow the uterus to fold in and permit part of the wall to move towards the pelvic inlet. Straining then pushes the flaccid organ through the vagina.
- Increased straining, which may be caused by pain or discomfort after parturition.
- Other causes of increased intra-abdominal pressure, including tympany and recumbency.
- **Excessive traction at assisted parturition and the weight of retained fetal membranes have been suggested as other predisposing factors.**

Clinical signs and treatment **These vary somewhat with the species involved and will be considered separately.**

The cow

Clinical signs **The patient is usually found with her uterus already prolapsed. One or both uterine horns may** be visible. The mucosal surface of the uterus – with its cotyledons – is visible and part of the chorioallantois may still be attached. The cow may be standing and apparently unconcerned or she may be shocked and recumbent. The uterus may be grossly contaminated with bedding and feces. It may also be lacerated, engorged, and edematous. If recently prolapsed it is warm to the touch but later becomes cold and discolored. Occasionally the cow is found dead. Death is often due to hemorrhage from the ovarian arteries, which may rupture as a result of the excessive tension placed on them by the prolapse.

Prognosis This depends on: (1) the duration of the problem; (2) the degree of damage and contamination sustained by the uterus; (3) the degree of shock in the cow; (4) the position and accessibility of the patient.

Treatment On receiving a call, the obstetrician should give advice on first aid care. The uterus should be protected from further damage, wrapped in a clean moist sheet, and, if possible, held above the level of the vulva.

On arrival the following treatment sequence should be followed:

1. Assess the cow's general condition: if she is moribund and severely shocked treatment may not be practical or economical. If there is evidence of hypocalcemia this should be treated.
2. Assess the cow's position: she may be in a most unsuitable position for treatment but it may also be impossible to move her. If her hindquarters are pointing downhill it would be advisable to move her so that her head is lower than her hindquarters. Gravity would thus help rather than hinder replacement.
3. Administer an epidural anesthetic.
4. Position the cow: this is best done by the 'New Zealand method'. The cow is placed in sternal recumbency with her hindlegs pulled out behind her. Two or three assistants are required for this. If the cow is standing she must be cast on her side and the uppermost hindlimb pulled out behind her. She is then rolled on to her other side so that the second hindlimb can be secured and extended caudally. An assistant sits astride her facing backwards and lifting the cow's tail out of the way (Fig. 13.3). See below * for an alternative method if sufficient help is not available.
5. Remove gross debris from the prolapsed organ by washing with saline or a very mild antiseptic.
6. Remove the placenta or its remnants from the cotyledons – if it separates easily. If not, leave it attached.

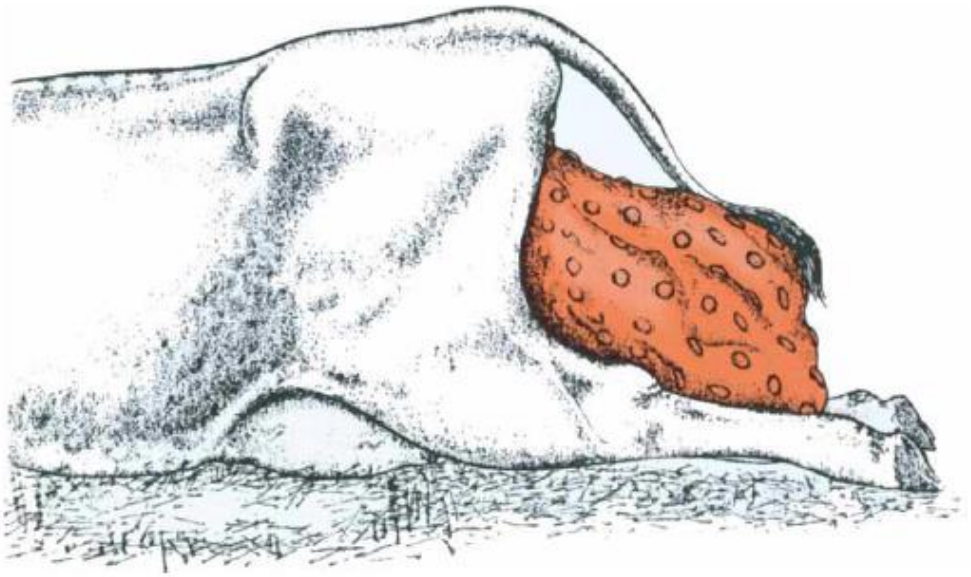

Figure 13.3 Cow – uterine prolapse, patient positioned for replacement.

*On occasion there may be insufficient assistance on the farm to place the cow in position with her hindlegs extended. In such circumstances the obstetrician must use an alternative method of replacement. The cow is given an epidural anesthetic and the uterus is prepared for replacement as in the New Zealand method. The obstetrician, wearing a parturition overall, kneels behind the cow and takes the prolapsed organ on his or her lap. The body of the uterus is first pushed back into the vagina while an assistant, if available, helps by holding the uterine horns above the level of the vulva. Pressure is now directed onto the horns, which are pushed back into their correct position. Replacement is greatly helped if the cow's hindquarters are higher than her forequarters. If raising her hindquarters or lowering her front end is possible this should be done, but only if there is no risk to the prolapsed organ. Postprocedure treatment and care is as before.

7. Repair any gross damage such as tearing using an absorbable suture.
8. Reducing the size of the prolapse – this is not really necessary. Some obstetricians recommend applying sugar or salt to 'draw out the edema'. The use of oxytocin *before* replacement is not advisable. The tightly contracted uterus can be very difficult to replace.
9. The prolapsed uterus is raised above the level of the vulva and eased back through the vagina. The body of the uterus is first pushed into the vagina followed by the horns. Handling the uterus may be aided by wrapping it in clean plastic and also by applying obstetric lubricant. The uterus is often very bulky and the help of an assistant in holding and replacing it is often very useful.
10. The cow is released from the sternal position and, if able, is encouraged to rise. The horns of the uterus are pushed fully back into position aided if necessary by a clean bottle used to fully invert
11. As soon as the uterus is replaced an injection of oxytocin (20–30 IU) is given by intramuscular injection. This will cause the uterus to involute and reduce the risk of recurrence of the prolapse.
12. Suturing the vulval lips should not be necessary or indeed helpful. Vulval sutures are, however, used by many obstetricians and are expected by many farmers. The suture pattern is as for vaginal prolapse (see Chapter 2).
13. Aftercare: good nursing, a light diet, and moderate exercise are required. Antibiotic cover is recommended. The vulval sutures are removed after 10 days.

If it is impossible to replace the prolapse, amputation may be attempted, although the prognosis for survival must be guarded. The prolapse is opened close to the vulva to reveal the uterine vessels lying within the tense mesometrium. These are each ligated in two places and the mesometrium severed between the ligatures. The vagina is ligated – taking care to avoid the external ure-