

Dystocia

Dystocia means difficult birth; the corresponding Greek word for normal birth is eutocia. The diagnosis and treatment of dystocia constitute a large and important part of the science of obstetrics, and require a good understanding of normal parturition, sensitivity to the welfare of both dam and offspring, and good and sensitive practical competences. In addition, veterinarians must always try to prevent dystocia where possible, by the application of sensible sire and dam selection, and good husbandry and health care.

CAUSES OF DYSTOCIA

Obstetricians have usually regarded dystocia as being either maternal or fetal in origin.

Each case of dystocia is a clinical problem that may be solved if a correct procedure is followed. A correct diagnosis is the basis of sound obstetric practice. **HISTORY**

OF THE CASE :

Therefore, before proceeding to examine the animal, a brief history of the case should, whenever possible, be obtained. Much of it will be the outcome of questioning the owner or attendant, but many points will also be elicited from personal observation of the animal.

- Has full term arrived or is delivery premature?
- Is the animal a primigravida or multigravida?
- What is her previous breeding history?
- What has been the general management during pregnancy?
- When did straining begin? What was its nature – slight and intermittent or frequent and forceful?
- Has straining ceased?
- Has a water-bag appeared and, if so, when was it first seen?
- Has there been any escape of fluid?
- Have any parts of the fetus appeared at the vulva?
- Has an examination been made and has assistance been attempted? If so, what was its nature?
- In the case of the multiparous species, have any young been born, naturally or otherwise, and if so, when? Were they alive at birth?
- Is the animal still taking food?
- In the case of the bitch and cat, has there been vomiting?

By a consideration of the answers to these and similar questions, it is possible to form a fairly accurate idea of the case to be dealt with.

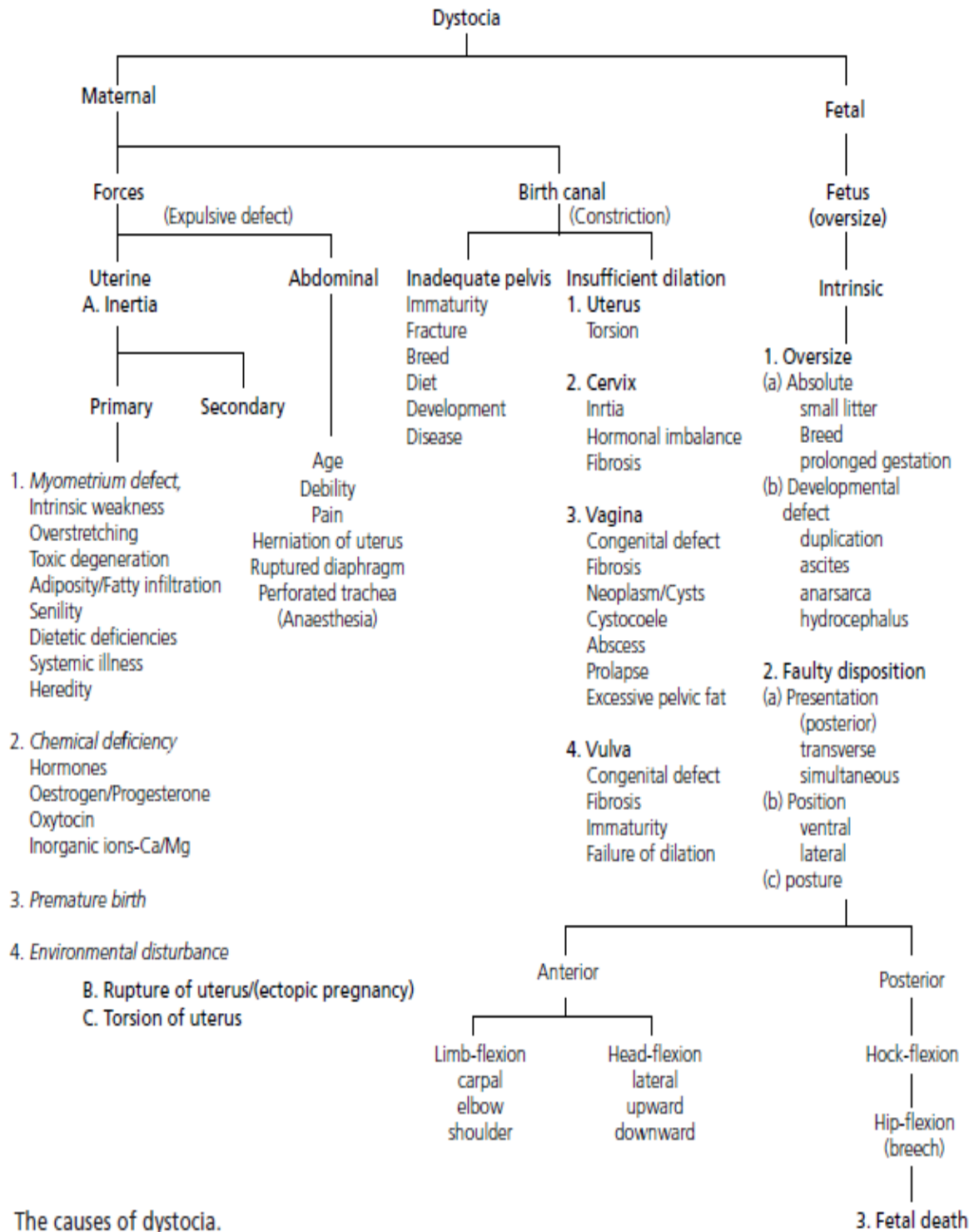


Fig. 8.1 The causes of dystocia.

Maternal causes of dystocia

Dystocias due to maternal factors are caused either by inadequacies of the birth canal or by a deficiency of expulsive force;

FAILURE OF THE EXPULSIVE FORCES

Uterine inertia

Primary uterine inertia

Etiology The most common cause is hypocalcemia, with the cow showing signs of milk fever as calving is about to begin. Other causes include distension of the uterus caused by hydrops uteri, general debility with reduced tone and responsiveness in the myometrium, and environmental disturbance. The presence of twins may cause such stretching of the myometrium that effective contractions cannot occur. Primary uterine inertia has also been seen in overweight beef cows that fail to go into labor. Some such animals may be mildly ketotic and possibly on the verge of pregnancy toxemia.

Clinical signs Preparations for birth begin but do not continue into second-stage labor. The fetus is normally in the correct presentation, position, and posture. The cervix is dilated or easily dilatable with manual pressure but there is no evidence of uterine contractions. The fetal membranes, especially the amnion, are often still intact. In cases of hypocalcemia the patient will be dull, reluctant or unable to rise, and have a low temperature, dilated pupils, and reduced rumenal activity. The head is turned back to the flank and, if untreated, the cow may become comatose with death ensuing. In cases of hydrops uteri there will probably have been a history during pregnancy of increasing abdominal size and debility. If accessible the uterine wall is found to lack muscle tone when palpated.

Secondary uterine inertia

Etiology The consequence of another cause of dystocia, for example fetal maldisposition, with resultant tiring of the myometrium.

Clinical signs The uterine wall is felt to be flabby and lacking in tone often after the fetus has been delivered.

Treatment The primary cause of dystocia is treated and the fetus delivered. Uterine involution is encouraged after delivery by injection of oxytocin as in primary uterine inertia.

Uterine rupture

Etiology Tearing of the uterus may occur as a result of traumatic injury to the cow, for example following collision with a vehicle. It may also occasionally occur spontaneously through an unsuspected weak point in the uterine wall. The fate of the fetus in such cases depends on whether it passes into the peritoneal cavity and the degree of compromise sustained by the fetal membranes. Small tears may be symptomless and the fetus remains in the uterus, where it develops normally and is born without difficulty. Larger tears may allow passage of the fetus into the peritoneal cavity. Maternal death may follow rupture with severe uterine hemorrhage. In cases where the placenta is compressed and its circulation is compromised, fetal death can occur. Sterile peritonitis may result in the fetus becoming adherent to the mesentery or other abdominal organs. Signs of pregnancy fail to develop and the intra-abdominal abnormalities may be detected by investigative clinical examination and possibly by exploratory laparotomy. If the placenta is unaffected by uterine rupture the fetus may survive until the end of gestation but its extrauterine location means that normal vaginal delivery is impossible.

Clinical signs These will depend on the degree of damage sustained and the fate of the fetus. External signs of hemorrhage following a road accident may suggest uterine damage, among other problems. Fetal death following uterine rupture may produce few signs other than failure of an established pregnancy to progress. If the fetus survives and develops within the peritoneal cavity, pregnancy may progress normally to term. Signs of imminent birth and even cervical relaxation may occur but birth does not follow. Transient colic may occasionally be seen. Vaginal examination may reveal a small empty uterus and the placenta disappearing through a defect in the uterine wall. The site of rupture – often on the dorsal curvature of the uterus or ventrally just beyond the pelvic brim – may be palpable or may be beyond reach. Occasionally, loops of maternal small intestine may be palpable. These must not be confused with the exposed intestinal loops often palpable with a schistosomus reflexus fetus (see p. 48). The uterus feels smaller than normal on rectal examination and occasionally the intraperitoneal fetus may be palpable. It may be possible to locate the fetus in an abnormal position – such as underneath the rumen – by external ballottement or a transabdominal ultrasonographic scan.

Torsion of the uterus

Uterine torsion has been found to be the cause of up to 7% of all bovine dystocia cases in some surveys. The pregnant uterus rotates about its long axis with the point of torsion being the anterior vagina just caudal to the cervix. Less commonly, the point of torsion is cranial to the cervix. In the majority of cases torsion is in an anticlockwise direction as the obstetrician stands behind the cow. The degree of torsion varies from 45 to 360°. A few cases of uterine torsion during pregnancy have been reported.

Etiology The bovine uterus has been said to be basically unstable for a number of reasons. These include: (1) the caudal parts of the uterus are attached to the lateral walls of the pelvis by the broad ligaments; (2) as pregnancy advances the cranial parts of the uterine horns lie on the abdominal floor with no stabilizing ligamentous attachment; (3) a single-calf pregnancy chiefly occupies one horn of the uterus, making the organ heavier and more bulky on one side than the other; (4) the instability may be increased by the cow lowering her front end first when lying down. Torsion occurs when the cow – or the fetus – makes a sudden movement causing the unstable uterus to rotate about its long axis. The bovine amnion is fused in places to the surrounding allantois, which is attached through the chorion to the uterine wall. If the fetus rotates about its long axis in late gestation the uterus may be rotated with it. Reduced exercise may increase the incidence of torsion.

Clinical signs The first signs may be noted towards the end of first-stage labor, which is prolonged, and the cow may show signs of mild discomfort. The patient may adopt a 'rocking-horse' stance so that the dorsal surface of her spine is concave and the fore- and hindlimbs are held respectively further forward and backwards than normal. Torsion of the birth canal may cause one or both lips of the vulva to be pulled in. Vaginal examination reveals an abnormal disposition of the birth canal (Fig. 4.2). The hand cannot immediately be passed anteriorly towards the cervix. The vagina narrows conically and folds of vaginal mucosa may be felt going into an oblique spiral. The direction of the vaginal folds may indicate the direction of the torsion – either clockwise or anticlockwise.

If the torsion is less than 180° the obstetrician's hand may be passed through the constriction to palpate the fetus. In such cases care must be taken to avoid mistakenly thinking a dead fetus is alive. When palpated through the twisted anterior vagina the fetus may appear to float away from the obstetrician's hand and then spontaneously return as if alive. The cervix is normally dilated.

If the torsion is greater than 180° the birth canal may be totally occluded, with the vagina coming to a conical end with no recognizable cervix being palpable. Rectal examination will confirm the displacement, with the broad ligaments being abnormally palpable as taught bands in the caudal abdomen.

Treatment A number of methods are available:

Rotation of the fetus and uterus per vaginam back into their correct position This is possible if the obstetrician's hand can pass into the uterus and touch the fetus and if fetal fluids remain within the uterus. The fetus is grasped by a convenient prominence such as the elbow, sternum, or thigh and is rocked from side to side before being pushed right over in the opposite direction to the torsion (Fig. 4.3). If the maneuver has been successful the torsion will have disappeared and the vagina regains its normal morphology.

Rolling the cow The principle of this method is to roll the cow around its uterus while that organ remains still. Three assistants are required. The cow is cast on the side to which the torsion is directed (Fig. 4.4). Thus in an anticlockwise torsion she is cast on her left side. The two forelegs and the two hindlegs are tied together and the head is restrained with a halter or head collar. The cow is rolled sharply over onto her other (right) side. The patency of the vagina is checked and if the torsion persists the cow is gently rolled back onto her other (left) side and the process is repeated. The cow may have to be rolled two or three times before the torsion is corrected.

The efficiency of rolling can be improved by putting external pressure on the cow's abdomen in an attempt to 'hold the uterus still' while the cow's body is rolled. Manual pressure over the uterus can be used or a board rested against the caudal abdomen and downward pressure exerted by a person standing on it.

The calf should always be delivered by the obstetrician as soon as the torsion has been corrected. The cervix may close within 30 minutes of resolution of the torsion preventing fetal delivery by the vaginal route and necessitating cesarean section.

Surgical correction This may be necessary if fetal rotation is impossible and rolling the cow is unsuccessful. A left flank laparotomy is performed on the standing cow under local anesthetic (see also Chapter 11). The uterus is located and the direction of the torsion confirmed by palpating and examining the cervical region. The uterine wall or a fetal limb within the uterus is grasped firmly and an attempt made to rotate the uterus back into its correct position.

Once the uterus is correctly in place the calf may be delivered per vaginam or by cesarean section. If the uterus cannot be rotated cesarean section must be performed with the uterus in its abnormal position. Once the fetus has been delivered the uterus can normally be readily rotated into its correct position after repair of the uterine wall. The condition of the uterine wall should be carefully checked before abdominal closure. If the uterus is discolored, its blood supply may have been compromised. If normal color is not restored after correction of the torsion the prospects for survival are not good. Antibiotic cover and the administration of a non-steroidal anti-inflammatory drug such as flunixin may aid recovery and provide analgesia.

Failure of abdominal expulsive forces

Etiology The abdominal musculature – so important during the second stage of labor – is either incapable of contracting or it is too painful for the animal to strain. In very old cows, or those suffering from hydrops, the abdominal muscles may have been stretched beyond the capacity of their natural elasticity. Tears in the muscles occur in cases of ventral hernia and as a result attempts to strain are compromised, as they are in cases of rupture of the prepubic tendon (see Chapter 2). Painful conditions involving the abdomen, diaphragm, or chest such as traumatic reticulitis/pericarditis may cause voluntary inhibition of attempts to strain. Laryngeal and diaphragmatic damage are rare in adult cattle but anything that compromises closure of glottis to enable straining to occur such as a tracheostomy wound could also compromise birth.

Clinical signs Birth fails to occur despite the presence of normal preparatory signs and first-stage labor. Difficulty should be anticipated following recognition of the primary problem. Vaginal examination reveals a dilated cervix with the fetus in normal presentation at the pelvic inlet. In cases of ventral hernia the fetus may be only just palpable or even beyond reach. Its position can be ascertained by external ballotement.

OBSTRUCTION OF THE BIRTH CANAL

The bony pelvis

The dimensions of the bony pelvis are too small to allow passage of the fetus.

Etiology *Maternal immaturity* is the most common cause and often occurs as a result of heifers being served at too young an age. This is a particular problem when the stock bull is allowed to run with the herd after the heifer calves have reached puberty at 6 or 7 months of age and misalliance takes place. Small pelvic size is seen in poorly grown heifers and may very occasionally occur as a result of pelvic fracture. A small pelvis is a component in dystocia due to fetopelvic disproportion and is exacerbated in cases where the fetus is larger than normal. In most heifers who suffer misalliance, fetal size is within the normal range for the breed.

A less common cause of a small bony pelvis is *sacral displacement*, in which the fused sacral bones and the first few coccygeal vertebrae are set at an abnormal angle to the lumbar vertebrae. As a result of the problem – which may have a hereditary etiology – the dorsoventral diameter of the maternal pelvis is severely reduced, allowing less room for passage of the fetus.

Lumbosacral subluxation may be caused by a cow or heifer being mounted by a very heavy bull. The downward displacement of the vertebral column that results at the lumbosacral junction may also reduce the size of the pelvic inlet.

Clinical signs There is lack of progress in the second stage of labor. If the fetus is able to partially enter the pelvis, severe unproductive straining may occur. A single large fetal foot may be seen at the vulva. If the fetus is too large to enter the pelvis, no progress is made after the completion of first-stage labor and the heifer looks uncomfortable, strains occasionally, and may stand with her back arched and tail raised. Vaginal examination reveals the presence of a small bony pelvis of insufficient size for the fetus to pass through.

The soft tissues

The vulva

Etiology Relaxation of the vulva is part of the normal preparations for birth but occasionally – especially in heifers – full relaxation of this part of the birth canal does not occur. In older animals damage sustained at an earlier calving or a horning injury with the formation of scar tissue and fibrosis may occur preventing normal relaxation.

Clinical signs Although relaxation of the pelvic ligaments has occurred, the vulva may not appear to be relaxed or there may be evidence on close examination of an earlier injury. Some difficulty may be experienced in inserting the lubricated hand, and the lips of the vulva may have a hard and fibrous consistency. Beyond the confines of the vulva, vaginal dimensions are normal.

The vagina

Etiology The vagina also relaxes in preparation for birth but the presence of scar tissue from a previous calving injury may cause a loss of elasticity. Less commonly, congenital stenosis of the vagina may be present. The lumen of the vagina may be obstructed by embryonic remnants, a hymen, perivaginal abscesses, and also by tumor or cyst formation. Vaginal prolapse seldom causes obstruction to birth but may require careful protection during fetal delivery.

Clinical signs Lack of progress during early second-stage labor may be observed but vaginal abnormality might not be detected until the obstetrician performs an internal examination. Scar tissue and perivaginal hematoma formation may have been detected at the routine postnatal check following a previous calving. In normal parturient cattle the vaginal wall is soft, gently elastic to manual pressure, and capable of expanding to accommodate fetal passage. The vagina is occasionally partially obstructed by a vertical pillar of mucosa-covered tissue just caudal to the cervix.

Hymenal remnants are unusual in cattle but if present are located immediately anterior to the external urethral orifice. Usually thin and easily broken down, they extend laterally from the vaginal walls to occlude part or almost all of the lumen. Perivaginal abscesses or hematomas may press on the vaginal walls, through which they may be felt as smooth fluctuating or firm masses. The contents of masses in the vaginal wall can be investigated by needle aspiration or a direct ultrasonographic scan. Vaginal tumors are uncommon in cattle but occasionally one or more leiomyomas may be palpable attached to the vaginal wall and, more seriously, a large invasive squamous cell carcinoma may obstruct the caudal vagina.

The cervix

Failure of cervical dilation is the third most common cause of bovine dystocia and its management requires careful clinical judgment.

Etiology The mechanism of cervical dilation in cattle is poorly understood. Hormonal factors together with physical dilation caused by the approaching calf and its fetal sacs are involved. Failure of these and other factors to exert their influence may result in the cervix remaining closed or only partially dilated. Cervical obstruction can also result from the presence of scar tissue arising from previous injury, possibly at an earlier calving.

Clinical signs Signs of first-stage labor are prolonged and do not proceed to the second stage. Cervical obstruction is detected on vaginal examination when the case is investigated. When fully dilated the cervix is flattened into the vaginal wall and is not palpable. When fully closed, the obstetrician's finger may be inserted into but not passed through the external os. During pregnancy the os is sealed with a thick mucous plug, which is passed out through the vagina a few hours before fetal delivery. The partially dilated cervix is palpable as a circular rim extending into the lumen of the vagina at the junction of the vaginal and uterine walls. Partial dilation may allow passage of parts of the fetus such as a foot or the nose but not wider parts such as the thorax, to which the edges of the cervical rim are tightly applied. If only slightly dilated the obstetrician may insert a finger through the external os and touch part of the fetus within the uterus. It may be possible to detect signs of fetal life such as spontaneous movement, and also to determine whether the fetal membranes are intact or have ruptured. In the latter case the fetus can be directly palpated and there may also be evidence of loss of fetal fluids into the vagina. Some assessment of fetal health may also be made by rectal palpation.

FETOPELVIC DISPROPORTION

Etiology This important cause of dystocia occurs when the fetus is larger than normal, the pelvis is smaller than normal, or there is a disproportion between them. Factors affecting pelvic and fetal size are discussed above (see p. 40). In summary, pelvic size is influenced by the age, breed, weight, and pelvic dimensions of the dam. Fetal size is influenced by many factors including breed, parental and grandparental factors, gestation length, sex of the calf, litter size, parity of the dam, double-muscling of the fetus, and the nutritional state of the dam.

Clinical signs The cow is unable to complete, or has great difficulty in completing, the second stage of labor. Straining of varying intensity is seen and the fetal feet and possibly the nose (in cases of anterior presentation) may be visible at the vulva. In a normal birth the calf is usually delivered within 2 hours of the fetal nose being seen at the vulva – in cases of fetopelvic disproportion this does not occur. If the case is untreated the calf will die, with serious consequences for the cow.

DYSTOCIA CAUSED BY FETAL MONSTERS

Etiology Fetal monsters arise from adverse factors affecting the fetus in the early stages of its development. The adverse factors are mostly of genetic origin but may also include physical, chemical, and viral factors. These adverse factors are particularly likely to affect the fetus before day 42, when organogenesis is complete in cattle. Fetal monsters are relatively uncommon and mostly occur sporadically but the incidence in cattle is higher than in other species. Occasionally a series of monsters may be encountered on one farm or a series of farms, which may have been sired by one bull.

Conjoined twins

Sometimes known as 'double monsters', these are the most common group of monsters and arise from incomplete division of a fertilized ovum and show great

variation from partial duplication to almost complete separation of the two individuals. Their presence, although rare, should always be suspected when an apparently normal birth cannot be delivered as anticipated.

Great variation on the degree of separation is seen but the following are amongst the most common conjoined twins:

- *Diprosopus*: the monster has two faces, including mouths with cleft palates, but not two complete heads. The wide double face will normally prevent the fetus from entering the maternal pelvis and cesarean section or fetotomy is required to permit delivery.
- *Dicephalus*: two heads and necks that join at the shoulder (Fig. 4.5). The divergence of the necks again prevents normal entry into the pelvis. Treatment is by removal of one head by fetotomy followed by vaginal delivery of the rest of the fetus or by cesarean section.
- *Dipygus*: duplication of the trunk and some of the limbs. Delivery is normally by cesarean section.
- 'Siamese twins' (the somatodidymi): separation of the twins is almost complete with points of attachment being along the sternum or elsewhere. One of the more common of these rare monsters is *pygodidymus*, in which the two calves are joined at the rump and are facing in opposite directions. It is seldom possible to detect the point of attachment on vaginal examination. The first calf is often in normal presentation, seems quite small, but cannot be moved by traction. Delivery is by cesarean section

Schistosomus reflexus

The monster most frequently described in the literature, this is also known as a 'celosomian monster' or 'moon calf'. In this monster the spinal column has undergone dorsiflexion and the head and tail approximate. The limbs are ankylosed and deformed (Fig. 4.6). The vertebrae and ribs form a discoid plate of bony tissue. The grossly abnormal shape makes unaided passage through the birth canal unlikely. The deformed calf may be a singleton fetus or co-twin to a normal calf.

Perosomus elumbis

This monster has a deceptively normal anterior end but rudimentary lumbar vertebrae and spinal cord, and hindlimbs that are contorted and ankylosed, possibly as a result of lack of movement by the developing fetus.

Hydrocephalus

Affected animals have a gross enlargement of the cranium that can prevent the fetus from entering and passing through the maternal pelvis. In some cases

Fetal ascites

This may be seen in calves at term or in cases of premature fetal death. The head, neck, and thorax of the calf will readily enter and pass through the maternal pelvis but the distended abdomen will not. If there is

Fetal anasarca

Generalized subcutaneous edema is present in this abnormality. Affected calves often have no hair and uterine fluids appear to be deficient leaving little natural lubrication.

FETAL MALDISPOSITION

The term 'maldisposition' includes abnormalities of presentation, position, and posture that render it difficult or impossible for the fetus to enter or pass through the birth canal.

Etiology It is not clear why the fetus adopts its normal birth posture but it has been suggested that the mammalian fetus may 'practice' assuming this posture during the later stages of pregnancy. Mild or severe fetal ill health and fetal death may predispose to fetal maldisposition, as may maternal ill health or abnormal hormone levels. A mild fetal maldisposition may be made worse as it fails to engage correctly at the pelvic inlet and expulsive forces compound the difficulty. Self-correction of a maldisposition is extremely unlikely.

Malpresentation

Posterior presentation

Although calves 'coming backwards' may be delivered spontaneously, posterior presentation is not normal in cattle. Assistance during delivery may be required, especially when fetopelvic disproportion is also present. Despite anecdotal accounts by farmers it is not normally possible to convert a posterior presentation into an anterior one. In an uncomplicated posterior presentation the fetal hindlimbs may be seen at the vulva with the soles of the hooves showing dorsally. This orientation of the hooves can also occur in very rare cases with the fetus in anterior presentation, ventral position, and with a deviation of the head, and care must be taken to ensure that the exact nature of the maldisposition is known.

Transverse presentation

This may be dorsotransverse, ventrotransverse, or laterotransverse, depending on whether the dorsal, ventral, or lateral surface of the fetal body is facing the pelvic inlet. In some cases the fetus may lie obliquely across the pelvic inlet. Careful palpation per vaginam will confirm the orientation of the fetus. An attempt is made to place the fetus in a longitudinal presentation by obstetrical version – repelling one end of the body and applying gentle traction to the other. Ideally the caudal end of the body is brought towards the pelvic inlet because the two hindlimbs may be more easily manipulated into the pelvis than the forelimbs and head. Once in a longitudinal presentation the fetus must be rotated from its lateral position into a dorsal position. Delivery by traction follows correction of the malpresentation. If the malpresentation cannot be corrected the fetus must be delivered by cesarean section or fetotomy.

Vertical presentation

An extremely unusual malpresentation in which the fetal body is found lying vertically across the pelvic inlet. The fetus may be in dorsovertical, ventrovertical, or laterovertical presentation, depending on which body surface is facing the pelvic inlet. An attempt is made to place the fetus in a longitudinal presentation by repelling one end of the fetus and applying gentle traction to the other. If manipulative delivery is impossible the fetus must be delivered by cesarean section or fetotomy.

Malposition

In normal delivery the calf is in dorsal position with its spinal column beneath that of the dam. Abnormalities of position include *ventral position*, in which the calf is 'upside down', or *lateral position*, when the calf is 'lying on its side'. These abnormalities of position may also be seen when the fetus is in posterior presentation. Although very small calves can occasionally be delivered in an abnormal position it is unwise to attempt to do so and the fetus should be manipulated into a dorsal position by the technique of obstetric rotation. An attempt is made to rotate the calf around its long axis by applying lateral pressure to the shoulders (or the hindquarters in a posterior presentation). This may be done by direct pressure or by rocking the calf from side to side around its long axis before pushing it firmly back into dorsal position.

Malposture

Abnormality of posture may involve the head, forelimbs, hindlimbs, or a combination of these. In some surveys lateral deviation of the head and carpal flexion have been recorded as the most common malpostures in cattle.

Lateral deviation of the head

Downward deviation of the head

Retention of a forelimb

This may involve carpal flexion or shoulder flexion affecting one or both forelimbs. Incomplete extension of the elbow, which causes dystocia in mares, is seldom encountered in the cow.

1. Carpal flexion

Clinical signs If only one leg is involved, the normal leg and the head are found within or protruding from the vagina. The flexed carpus of the other foreleg is found at the pelvic inlet or impacted in the vagina.

2. Shoulder flexion

Clinical signs If both legs are involved the fetal head alone may be found in the vagina or protrude from the vulva, where it may become swollen and edematous. If only one limb is affected the other limb often protrudes from the vulva with the head.

Retention of a hindlimb

This occurs in some cases of posterior presentation and may involve hock flexion or hip flexion of one or both hind limbs.

1. Hock flexion

Clinical signs The tip of the fetal tail may protrude from the vulva and the flexed hocks are palpable either at the pelvic inlet or impacted within the pelvis. If only

one limb is flexed at the hock the other may extend through the vulva.

2. Hip flexion (breech presentation)

Clinical signs The tail may protrude from the vulva or be held against the fetal flank. The fetal hindquarters are palpable on vaginal examination (see Fig. 12.8). The hindquarters may be level with the pelvic inlet or lying in front of and below the level of the pelvic floor. Occasionally, only one limb is retained and the other is in the normal extended position.

DYSTOCIA CAUSED BY FETAL DEATH

Death of the fetus in late pregnancy or in the early stages of parturition may result in dystocia, which may arise in a number of ways:

- The fetus may have suffered from chronic hypoxia during pregnancy, possibly as a result of an ineffective placenta. This situation may arise especially in first-calf heifers, which show little preparation for calving and in which the fetus is found to be dead when signs of impending delivery eventually occur.
- The fetus may fail to release sufficient quantities of those hormones, including ACTH and cortisol, that initiate parturition.
- The fetus is unable to adopt the normal birth posture and thus maldisposition may occur, preventing birth.
- The cervix may fail to dilate fully – thus not allowing the fetus to pass.
- Uterine fluids may be lost and fetal delivery may be impeded by absence of natural lubrication.

Clinical signs The first sign of fetal death may be a foul-smelling vaginal discharge at the time that birth is anticipated. Investigation will reveal a partially or fully dilated cervix through which may protrude necrotic fetal membranes and parts of the fetus. If infection has gained access to the fetus through the cervix the fetus may be bloated and emphysematous. Pockets of gas are palpable beneath the fetal skin and the hair is readily pulled out. The loss of fetal fluids makes it difficult for the obstetrician's hand to move around in the uterus, the walls of which are tightly applied to the dead fetus. There is no sign of fetal life. If the fetus has been ill before death it may have developed fetal ascites with gross abdominal enlargement. In the early stages the

cow may be quite unaffected but in a proportion of cases severe life-threatening metritis with toxemia ensues. Fetal death earlier in pregnancy may result in abortion, fetal mummification, or fetal maceration