

BOVINE NEONATOLOGY

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Summary

The term “neonate” defines calves under 28 days old. During the neonatal period, the calf is at high risk of suffering different diseases, and neonatal morbidity and mortality cause large economic losses. In fact, 75% of the mortality of dairy heifers occurs during the first month of life. In beef cattle, the mortality rate until weaning is lower, around 10%, but it also peaks during the neonatal period.

The first problem we find is prematurity (not-to-term calves) and several malformations. The most common congenital defects of the calves are described throughout the text. Additionally, dystocia (difficult delivery) is the first cause of mortality in the neonatal period, and can also predispose the animal to other infectious diseases. We have explained several problems induced directly by dystocia, such as obstetrics fractures, umbilical cord entrapment and birth edema. These circumstances lead to neonatal acidosis, hypoglycemia, hypothermia and to what is designated as “weak calf syndrome”. An appropriate resuscitation protocol, as described and recommended by the authors, is essential in order to ensure healthy, strong, neonatal calves.

During the neonatal period, the first infectious pathological disease we observe is neonatal septicemia, affecting calves between 2 and 6 days old. Neonatal diarrhea is by far the main infectious problem, followed by pneumonia in older neonatal calves aged 2-28 days old or even older.

By applying good practices, these diseases can be almost completely controlled, thereby reducing the associated losses. Indeed, by implementing good management procedures and a correct prophylaxis protocol as recommended by the authors, we can dramatically reduce the morbidity and mortality of rearing calves in both dairy and beef bovine

farms. There are several risk factors we should take into account, but the cornerstone of calves' health is intake of good-quality colostrum at the appropriate time. This topic will be explored in depth in the text.

Neonatal morbidity and mortality cause large economic losses in both beef and dairy cattle (Nagy, 2009). During the neonatal period, the calf is at high risk of suffering different diseases. In fact, 75% of the mortality of dairy heifers during rearing occurs during the first month of life (Radostits, 2007). In beef cattle, the mortality rate until weaning is lower, around 10%, but is maximum during the neonatal period, when it varies between 4 and 6% (Wikse *et al.*, 1994). Throughout this period, diarrhea is by far the main infectious problem, followed by pneumonia. Nevertheless, by applying good husbandry and management practices these infectious diseases can be almost completely controlled, thereby reducing the associated losses. Thus, depending on farm management, we can find herds where diarrhea or pneumonia is a significant cause of mortality, while in other farms both diseases are uncommon.

In addition, dystocia is the first cause of mortality in the neonatal period, and can also predispose the animal to other infectious diseases. This explains the differences between mortality in heifers and cows: mortality in the first 24 hours of life is around 8% in heifers and 3% in cows, while mortality from 24 h until weaning is 3% in heifers and 2% in cows (Toombs *et al.*, 1994).

1. Definition of Neonate

Due to the importance of this period, this chapter will cover the most critical points related with the neonatal calf. Thus, we should first define what we mean by neonatal period. Starting with the beginning of pregnancy until the calf stage we can consider different periods in the life of the calf (Table 1).

Period	Definition
Fetal	Between 42-270 days of pregnancy
Perinatal	For most authors, from 270 days of pregnancy to exactly 24 hours of life. However, the term "perinatal" sometimes includes the first 28 days, and even up to six months
Neonatal	From delivery of the calf until 28 days of life. This period can be subclassified into "early neonatal period", which includes the first 24 hours of life, and "late neonatal period" from 2-28 days of life
Calf	From 28 to 90 days of age. Some authors extend this period to 6 months postpartum

Table 1. Different stages of a calf's life, including the fetal period.

2. Physiological Characteristics of the Neonatal Calf

The event that can be considered the key to the transition from an intrauterine life, in which gas exchange is performed through the placenta by the umbilical cord, to an extrauterine life, in which lungs are responsible for blood oxygenation, is the initiation

of respiratory movements, which involve lung inflation and subsequent oxygenation of the blood. Cardiopulmonary adaptations to extrauterine life are one of the most important physiological changes that must occur for calf survival (Nagy, 2009).

These events begin with the rupture of the umbilical cord, which causes hypoxia and an increase in partial CO₂ pressure (PCO₂). This stimulates gasping reflexes in the calf and subsequent inflation of the lungs. Until the first inflation, the calf suffers a slight respiratory acidosis, which causes minor metabolic acidosis; thus, acidosis is a normal characteristic in recently delivered calves (Grove-White, 2000). Indeed, venous pH studies have shown that normal pH in healthy neonatal calves is 7.2-7.3, while in cases of dystocia at parturition the pH value can fall to 7 or even less (Schujt and Taverne, 1994; Grove-White, 2000). Metabolic acidosis is corrected a few hours after birth, while respiratory acidosis can persist for up to 48 h. Nevertheless, a study by Schujt and Taverne (1994) showed that under normal situations, the venous pH range reaches physiological values within 12 hours of life. A sign that we can use to evaluate acidosis is the position taken by the calf. A healthy calf puts his head up after just a few minutes, and adopts the position of sternal recumbence in 4 ± 2 minutes.

As a result of lung inflation, the pulmonary vascular bed opens up and pulmonary blood flow increases considerably. This causes a change in the pressure relationship within the cardiovascular system, producing the functional closure of the *foramen oval*, *ductus arteriosus* and *ductus venosus* (Grove-White, 2000).

3. Most Common Congenital Malformations

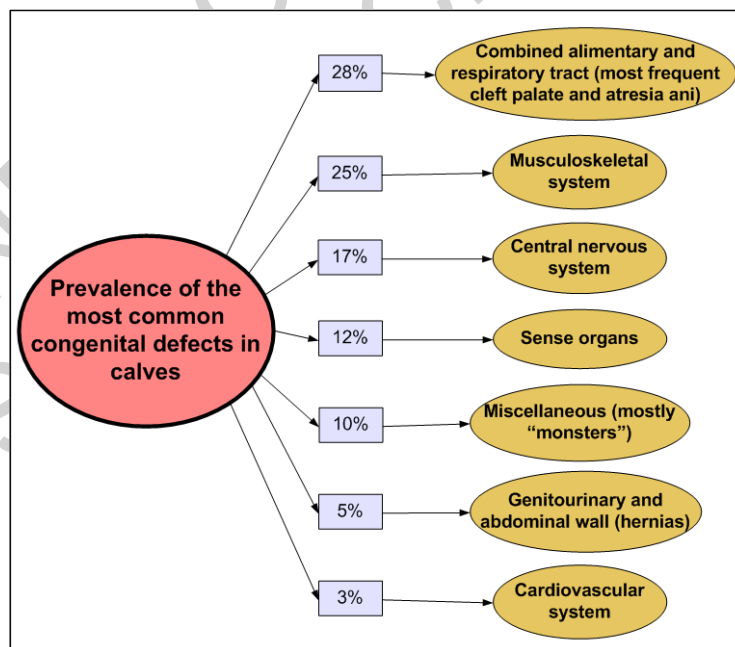


Figure 1. Prevalence of the most common congenital malformations in calves (Radostits *et al.*, 2007).

Figure 1 depicts the prevalence of the most common congenital problems of calves (Radostits *et al.*, 2007).

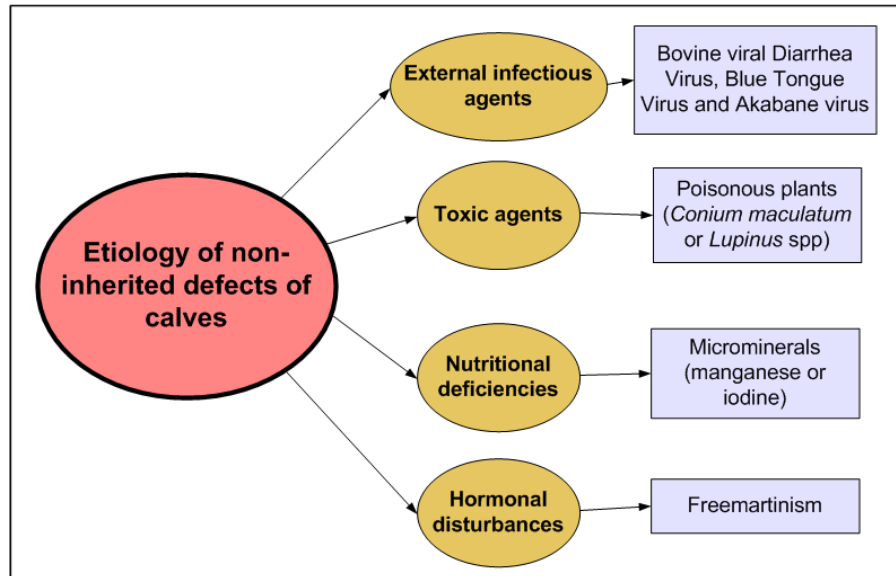


Figure 2. Etiology of the most common non-inherited problems of calves.

Although many malformations show similar clinical signs or patterns, most of them may have one or more etiologies. For this reason, we have classified in Table 2 the most frequent malformations according to the system affected and signs, and we explain the different possible etiologies. Congenital malformations may have a genetic origin (“inherited defects”) or may be “non-inherited defects”. In Figure 2 the etiologies of the most frequent non-inherited congenital defects in calves are shown.

Affected system or process	Designation and lesional and clinical pattern	Etiology	Type of malformation
Musculo-skeletal system	Contracted tendons (the least severe pattern) and arthrogryposis (the most severe one). Frequently associated with cleft palate	The most frequent musculoskeletal disorder. The mild cases of contracted tendons usually heal by themselves with time, while severe cases need surgery. Etiology <ul style="list-style-type: none"> • Lack of space in the uterus (heifers or twins) • Poisonous 	Inherited and non-inherited

		<p>plants such as <i>Lupinus spp</i>, <i>Astragalus spp.</i>, <i>Lathyrus spp.</i>, and <i>Oxytropis spp</i>. chemically reduce fetal movements</p> <ul style="list-style-type: none"> • Damage in the fetal central nervous system that reduces fetal movement. <p>The frequent association with cleft palate is due to a constant reflexed position of the neck that presses the tongue on the hard palate, preventing its closure.</p>	
	<p><i>Chondrodysplasia</i>. Diminished endochondral osteogenesis. “Bulldog calf”, shortened legs</p>	<p>Some chondrodysplasia phenotypes are lethal (produce abortion), while others produce viable but short-legged calves.</p> <p>Etiology: some forms are caused by genetic factors; the most common one, designated "bulldog calf", is caused by a mutation in the gene for the connective tissue protein, while other forms are multifactorial and are triggered by nutritional deficiencies.</p>	<p>Inherited and non-inherited</p>
	<p>Complex vertebral malformation syndrome (CVM). Mild bilateral flexion of the carpal and</p>	<p>Autosomal recessive congenital lethal malformation due to a single-nucleotide</p>	<p>Inherited</p>

	<p>metacarpophalangeal joints with rotation of the digits. Vertebral malformation, malformed ribs, arthrogryposis of the tarsal and metatarsophalangeal joints. Malformation of cervical and thoracic vertebrae, with shortening of the neck. The extent of vertebral malformation varies.</p>	<p>mutation in the <i>SLC35A3</i> gene, which results in insufficient function of a nucleotide–sugar transporter needed for the normal development of vertebrae. (This is a classic example of a malformation spread by insemination with semen from a carrier of the genetic defect). It causes abortions, stillbirths and viable calves. Viable calves show growth retardation.</p>	
	<p>Syndactylism or ‘mulefoot’. Fusion of digits III and IV, which are enclosed by a common claw capsule. Secondary adaptive changes are found in the muscles, tendons, nerves and vascular supply of the distal limb</p>	<p>Autosomal recessive congenital malformation. The inheritance of syndactylism in Holstein-Friesian cattle is slight because the number of affected limbs and degree of digital fusion varies, with some homozygous animals appearing grossly normal.</p>	Inherited
	<p>Muscular hypertrophy or “double-muscled cattle”</p>	<p>Mutation that represses the myostatin protein, thereby augmenting muscle growth. Although this characteristic has been selected in some breeds for beef cattle production, this malformation is associated with dystocia and is therefore an undesirable trait in</p>	Inherited

		most breeds.	
Central nervous system	Hydranencephaly, cerebellar hypoplasia, internal hydrocephalus	Genetic origin or infectious etiology: AKAV and BTV in the case of hydranencephaly and/or BVDV in the case of cerebellar hypoplasia and internal hydrocephalus.	Inherited or non-inherited
Cardio-vascular system	Ventricular septal defect. Poor prognosis but in rare cases, animals remain asymptomatic for a long time. Frequently associated with other congenital cardiac or vascular defects and other malformation such as microphthalmia and lack of tail	No known inherited etiology. The most frequent cardiovascular abnormality in bovine neonates.	Inherited
Digestive system	Cleft palate or palatoschisis. Frequently associated with arthrogryposis due to a constant reflexed position of the neck, causing the tongue to press against the hard palate, thereby inhibiting closure of the palate. The calves suckle with difficulties and develop aspiration pneumonia, causing death.	A common inherited defect in calves.	Inherited or non-inherited
	<i>Atresia coli</i> and <i>ani</i> . Complete closure of a segment of the intestinal tract: it is called <i>atresia coli</i> when the ascending colon is affected, and <i>atresia ani</i> when the anus is affected.	The origin can be genetic. The possibility of causing this malformation in early pregnancy by transrectal palpation of the amniotic sac before 40 days of gestation was suggested, but this has been called into question. Affected calves do not	Doubtful

		defecate, and present a progressive abdominal distention, anorexia and depression, and usually die in the first week of life, if the defect is not surgically corrected.	
Abdominal wall closure defects	From small umbilical hernia (the least severe pattern), omphalocele, gastroschisis or <i>schistosoma reflexus</i> (the most severe pattern). <i>Schistosoma reflexus</i> involves complete exposure of the thoracic and abdominal viscera. The calf is always stillborn, as they cannot adapt to extrauterine life. Acute angulation of the vertebral column and cardiac malformations are also frequent.	Etiology of umbilical hernia: <ul style="list-style-type: none"> • Non-genetic: traumatic or infectious, such as umbilical abscesses or suckling by other calves • Genetic 	Inherited or non-inherited
Reproductive tract	Freemartin (sterile female born with a male in a twin pregnancy). Freemartin heifers can be diagnosed. They present an enlarged clitoris, excessive hair growing at the ventral vulvar commisure and hypoplasia of female internal genitalia beyond the cervix	The most frequent reproductive malformation. Etiology: over 90% of twin calves have a fused placenta with a common blood supply. Thus, a heifer calf starts its development as a female, but undergoes exchange of embryonic cells and hormones before the development of sexual dimorphism, resulting in varying degrees of genital hypoplasia and masculinization.	Non-inherited
Metabolic malformations	<i>Bovine leukocyte adhesion deficiency</i> (BLAD). No observable lesions at birth. First signs are seen	Autosomal recessive inherited malformation. The mutation in the <i>ITGB2</i>	Inherited

	<p>between 2 weeks and 8 months of life and consist of recurrent infections. BLAD homozygotes are not viable and most calves die within the first year of life. BLAD should initially be suspected if there are signs of immunodeficiency combined with high numbers of circulating neutrophils. The diagnosis is confirmed by genotyping. Heterozygotes are not affected but show poor feed utilization and growth rates. Clinical signs consist of recurrent and prolonged mucosal and epithelial infections caused in most cases by opportunistic bacteria and fungi.</p>	<p>gene encoding CD18 leads to accumulation of neutrophils in circulating blood but not in the tissues.</p>	
	<p><i>Citrulinemia.</i> Lethal neurological disease of neonatal Holstein-Friesian calves. No observable lesions at birth. Usually clinical signs begin after 24 h of life. Clinical signs are the result of hyperammonemia: poor feeding ability and depression, aimless head pressing with apparent blindness and odontoprisis. Within 4-5 days of symptom onset, recumbence, convulsions, collapse and death occur.</p>	<p>Genetic etiology: a point mutation in the gene coding for argininosuccinate synthetase, an enzyme responsible for converting potentially toxic ammonia to urea through the urea cycle. The mutation results in an impaired urea cycle and subsequent extreme elevation of citrulline in plasma. Citrulline and ammonia concentrations in blood and cerebrospinal fluid are markedly elevated. As a result, the diagnosis can be confirmed through biochemical analysis or</p>	<p>Inherited</p>

		molecular identification of the mutation.	
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Table 2. Most common congenital defects classified by both affected organic system and clinical signs (adapted from Blowey and Weaver 2003; Radostits *et al.*, 2007; Agerholm, 2007; Windsor and Agerholm, 2009; White *et al.* 2010).

Inherited defects may result in a wide spectrum of disorders, not only severe malformations easily detected by exploration at birth, but also metabolic diseases, which are usually diagnosed days after birth. Some breeds and families show extraordinarily high prevalence of specific genetic disorders because of excessive inbreeding. It is certain that the extensive use of artificial insemination has resulted in a significant increase in the occurrence and nature of particular congenital defects in cases when the semen donors were carriers of genetic disease.

When environmental agents are present, the appearance of non-inherited malformations will depend on both the agent and the fetal age at exposure. Usually this kind of malformation appears in an epizootic way. Due to exposure early in the fetus' life, diagnosis is difficult at the time the calf is born.

In the face of an epizootic congenital defect we should look for:

- A genetic problem in which congenital defects are associated with a particular sire.
- Environmental agents such as poisonous plants or viral exposure, usually early in the pregnancy of affected cows. When viruses are involved, the outcome depends on the phase of pregnancy. Usual outcomes are abortions, stillbirths, birth of non-viable neonates or viable neonates with growth retardation or dysfunction (e.g. tremors, blindness).

The main viruses involved in non-inherited congenital defects are the following.

Bovine viral diarrhea virus (BVDV) of the genus Pestivirus and family Flaviviridae occurs in two genotypes (I and II), and two biotypes: non-cytopathic (NCP) and cytopathic (CP). Type I virus infection usually remains subacute in immunocompetent adult cattle, whereas type II infection has been associated with severe and highly fatal thrombocytopenia and a hemorrhagic syndrome that causes outbreaks in dairy herds. Infection of a non-immune pregnant cow causes *in utero* infection and produces different results depending on the pregnancy phase at the time of infection (Figure 3).

Infection during the first 45 days of pregnancy results in embryonic death and resorption, with irregular return to estrus and decreased conception rate in subsequent inseminations. Intrauterine infection between 45 and 125 days of pregnancy triggers embryonic mortality with abortion/mummification or congenital defects of the central nervous system and ocular abnormalities. Congenital morphological defects include: cerebellar hypoplasia (Image 1), in which calves are unable to stand and walk normally,

and eye defects, including retinal atrophy, optic neuritis, cataract and/or microphthalmia with retinal dysplasia, which results in varying degrees of blindness.



Image 1. Calf cerebellar hypoplasia is characteristic of intrauterine infection by BVD.

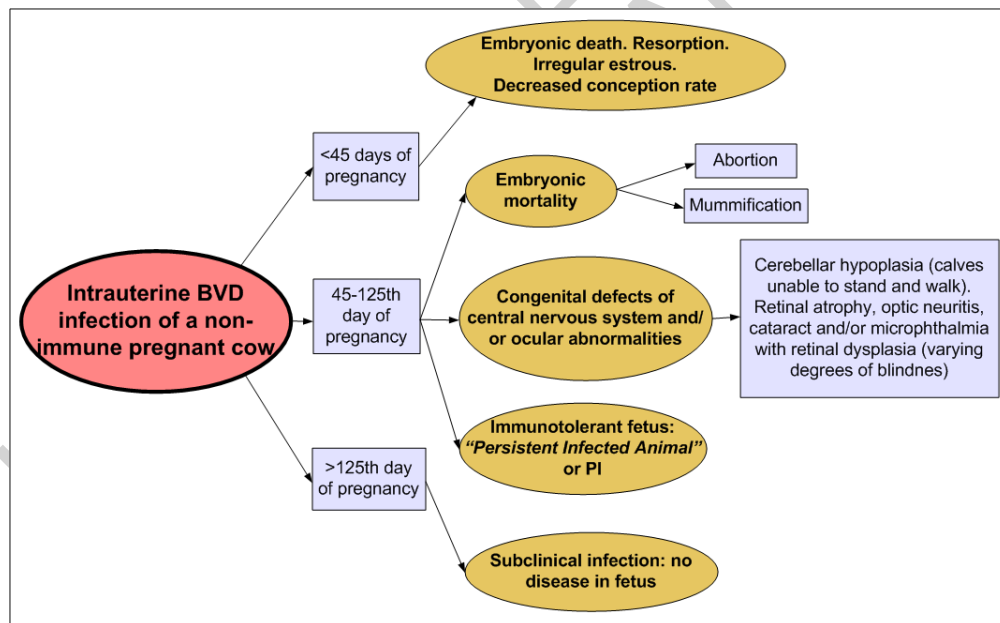


Figure 3. Effects of intrauterine infection of a non-immune pregnant cow by bovine viral diarrhea virus, based on when infection occurs during pregnancy.

In addition, when infection occurs before the 125th day of pregnancy, the surviving fetus suffers persistent infection (PI) and remains immunotolerant, meaning that its immune system does not recognize the virus as a foreign organism and does not fight against it. Such a calf subsequently becomes the main source of infection for other animals in the herd, because it sheds large amounts of virus into the environment. Finally, infections

after the 125th day of pregnancy are overcome subclinically by the fetus, with no long-term consequences.

Akabane virus (AKAV) of the genus *Orthobunyavirus* and family *Bunyaviridae* does not produce disease in adults. However, depending on the fetal period of infection, *in utero* infections can result in abortion, premature birth, stillbirth, and congenital defects such as microencephaly, arthrogryposis (when infection occurs between 104 and 173 days of pregnancy), hydranencephaly (infection between 76 and 104 days), and poliomyelitis (infection after the 173rd day of pregnancy).

Some strains of the Blue tongue virus (BTV) (genus *Orbivirus* and family *Reoviridae*) cause *in utero* infection that can result in early fetal death and resorption, stillbirth, and birth of weak animals. When infection occurs between 60-120 days of pregnancy, it can lead to congenital defects such as hydranencephaly, porencephaly, blindness, ataxia and arthrogryposis.

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