

Managing the Calf at Calving Time

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Abstract

Perinatal mortality rates are increasing internationally, particularly in Holstein-Friesian primiparae. The prevalence of perinatal mortality in US dairy herds is currently 8%. This article outlines our current knowledge of bovine perinatal pathophysiology and presents practical guidelines on management of the calf at calving time. Biophysical profiling of the newborn calf, without sophisticated equipment, is the first step in diagnosing at-risk perinates. Resuscitative techniques adaptable for both the producer and the veterinarian are detailed. In addition, management of prolonged recumbence, hypothermia, failure to suck, umbilical antisepsis and calf movement after calving are discussed. Knowledge gaps constraining future progress towards better newborn calf management are highlighted. Finally, current topics in perinatal mortality are presented.

Résumé

Le taux de mortalité en période périnatale s'accroît partout dans le monde particulièrement chez les vaches primipares de la race Holstein-Friesian. La prévalence de mortalité en période périnatale est de 8% présentement dans les troupeaux laitiers des États-Unis. Cet article ébauche nos connaissances actuelles de la pathophysiologie néonatale bovine et présente des directives pratiques pour la régie des veaux au moment du vêlage. L'établissement d'un profil biophysique du veau nouveau-né, sans équipement sophistiqué, est la première étape afin de diagnostiquer les veaux périnataux à risque. Des techniques de ressuscitation qui peuvent être adaptées à la fois pour le vétérinaire et pour le producteur sont détaillées. On discute aussi de la régie des longues périodes couchées, de l'hypothermie, de l'inaptitude à téter, de l'antisepsie du nombril et des mouvements du veau après le vêlage. On met aussi en évidence les zones grises dans nos connaissances qui ralentissent le progrès afin de mieux gérer les veaux en période périnatale. Finalement, les développements récents concernant la mortalité en période périnatale sont présentés.

Introduction

Perinatal mortality (PM) may be defined as calf death prior to, during or within 48 hours of calving, fol-

lowing a gestation period of at least 260 days, irrespective of cause of death or circumstances of the calving.²¹ The perinatal period is the most hazardous in the lives of all animals. More than 60% of US dairy producers reported that the majority of their calf mortality occurs at birth.²⁹ The main causes of perinatal morbidity and mortality are, in descending order of importance, combined respiratory and metabolic acidosis, parturient trauma, hypoglobulinemia, congenital infections and deficiencies and omphalophlebitis. Since 90% of calves which die in the perinatal period were alive at the start of calving, much of this loss is preventable. Perinatal mortality rates are increasing internationally, particularly in Holstein-Friesian primiparae.^{12,23,27} The prevalence of PM in dairy herds in the US has increased in recent years and is currently 8% (11.3% in primiparae and 5.3% in pluriparae).²⁷ Perinatal morbidity and mortality are growing welfare concerns, given their impact not just on losses around calving but also on subsequent productivity, health, reproduction and farm economics.³ It is estimated that the economic impact due to loss of replacement heifers from stillbirths in US dairies alone is \$125 million per year.³

Management of the newborn dairy calf to prevent perinatal morbidity and mortality is best achieved through implementation of simple protocols which document the correct strategies to be followed at the herd level, and the correct procedures to be carried out at the individual animal level. These protocols cover management of the prepartum cow, management of calving (monitoring of eutocia and detection and management of dystocia)²² and newborn calf care. Discussion with producers about best-practice newborn calf care or problems represents a contact moment which veterinarians should utilize to expand their role in veterinarian-led dairy herd reproductive management.

Perinatal pathophysiology

All calves suffer some degree of respiratory-metabolic acidosis at birth,³¹ but the duration of calving and the duration and force of assistance during calving affect fetal survival.^{16,31} Passage through the birth canal triggers fetal catecholamine release, which inhibits lung liquid secretion and stimulates its absorption, promotes surfactant secretion and increases lung compliance and fetal oxygenation. Calves delivered prematurely naturally or by pharmacological induction (<270 days) or cesarean section, or possibly following trace element deficiency at term, may suffer from re-

spiratory distress syndrome (RDS) due to a surfactant deficiency. Fetal acid-base status remains relatively stable during gestation until parturition. Changes in fetal acid-base status begin with the onset of abdominal and uterine contractions and placental separation, with resultant episodic reductions in umbilical blood flow in stage two of calving. Calves born following prolonged calving at term have increased respiratory and metabolic acidosis.^{16,31} Whether calves are assisted or not during calving, and the degree of assistance, also affects perinatal vigor. Thus, calves assisted compared to unassisted, or pulled out by strong compared to mild traction, have increased respiratory-metabolic acidosis and take longer to achieve sternal recumbence.^{16,25} One of the constraints currently hampering predictive perinatal management is the unavailability of inexpensive intrapartum fetal viability monitoring equipment suitable for use in large animal veterinary practice.

Assessment of newborn calf viability

The vigor of the newborn calf can be assessed before, during and immediately after calving by the producer or attending veterinarian without sophisticated point-of-care diagnostics. Particularly at-risk

calves are those delivered following malpresentation, malposture, prolonged dystocia or slow eutocia. Key indicators of the perinatal ethophysiological profile are correlated with newborn calf acid-base balance (Table 1).³⁰ Transient tachypnea is normal immediately after calving, but at-risk calves may experience prolonged tachypnea and bouts of primary or secondary apnea interspersed with dyspnea. Cyanosis of the buccal and lingual mucous membranes indicates prolonged dystocia. A weak response or no response to stimulation and poor muscle tone indicate prolonged, non-compensated acidosis. While the normocardiac range in calves is high immediately after calving (100-150 bpm), heart rate normally stabilises within minutes of calving, but in at-risk calves peripartur tachycardia (>150 bpm) is followed by a declining bradycardia.³¹ The rectal temperature of a normal newborn calf is higher than normal at calving 102-104°F (39-40°C) and gradually drops to normal within three to five hours. Calves that have had a protracted, difficult calving may experience hyperthermia (>103°F, >39.5°C) at birth, which drops in the hours following birth and is slow to return to normal.³⁵ Ability to head-right and to achieve sternal recumbence are key indicators of vitality, with delays

Table 1. How to assess newborn calf vitality.

Criterion	Good vitality	Poor vitality
Respiration	50-75 bpm and thoracic breathing	Gasping, primary apnea, irregular, abdominal breathing, bellowing and secondary apnea
Hair coat appearance	Placental fluid-covered	Meconium-stained
Peripheral edema	None	Capital, lingual or limb edema
Mucous membranes	Pink and normal capillary refill time	Cyanotic, pale and slow capillary refill time
Response to reflex stimulation	Vigorous head shake, strong corneal, suck or pedal reflex	Weak or no response
Muscle tone	Active with head-righting within minutes	Inactivity and flaccid musculature
Heart rate	100-150 bpm and regular	>150 bpm followed by bradycardia (<80 bpm) and an irregular, decreasing rate
Rectal temperature	102-103°F (39-39.5°C) after calving declining to 101-102°F (38.5-39°C) by 1 hour and stable	103-104°F (39.5-40°C) after calving declining to 101°F (<38.5°C) by 1 hour and decreasing
Sternal recumbence	Achieved within 5 minutes	Prolonged lateral recumbence
Attempts to rise	Attempting to stand within 15 minutes Standing within 1 hour	Delayed or no attempts to rise
Suckling	Commences within 2 hours	Delayed or no attempts to suckle

beyond three and 15 minutes, respectively, indicating a poor prognosis.^{16,31} Similarly, attempts to rise, time to stand and time to suckle unaided all take longer in compromised neonates,³¹ but are often less likely to be observed by veterinarians.

Calf resuscitation

Perinatal calf care attempts to reduce the impact of parturient respiratory and metabolic acidosis on perinatal mortality and on time to stand and suck, and the efficiency of colostrum consumption and immunoglobulin absorption, both of which critically impact subsequent health and productivity.

Most calves which require resuscitation are usually not attended by a veterinarian, as most dystocia and prolonged calvings are attended by herd staff only or are unattended. Hence, the role of the veterinarian in calf resuscitation is twofold: firstly, to draw up an SOP for at-risk calves for herd staff, and secondly to resuscitate calves after veterinary-assisted calvings. The SOP should document for herd staff a standard resuscitation equipment kit, to be located in the maternity pen area, and details of first-aid procedures to be followed with at-risk calves (Figure 1). At-risk calves are those likely to need resuscitation because of their calving outcomes. These calves can often be detected before birth (by the likelihood of dystocia, e.g. in small, obese heifers), during birth (large forelimbs, swollen tongue, cyanosed muzzle and gums or posteriorly presented or delivered by cesarean operation) or after birth (apnea or dyspnea, lateral recumbence, flaccid musculature, poor pedal and suck reflexes). A resuscitation kit for herd staff on large dairies should include a stethoscope, rectal thermometer, compressed air device (e.g. Kruuse Calf Resuscitator, McCulloch Medical Respirator/Aspirator, Ambu bag), needles, suction pump and oxygen delivery equipment.⁶ For successful resuscitation of at-risk calves, herd staff members need to practice good calving supervision (i.e. be present to assist the calf), prompt calf viability evaluation (during and immediately after birth) and early aggressive intervention (i.e. active management of calving and calf care).¹⁷

Physical resuscitation

The 'ABC' of resuscitation (airway patency, breathing stimulation, circulation support) indicates the sequence of priorities for herd staff members in dealing with at-risk calves. Resuscitation can commence while the calf is still in the birth canal and continues until the vital signs have normalised (e.g. posture, activity, respiratory function, rectal temperature) or until a heart-beat is undetectable with a stethoscope. Resuscitative first-aid procedures can be implemented by all herd

staff members using physical techniques requiring little equipment. Once the calf's thorax has emerged from the cow, the calf can breathe even if it remains *in situ* due to hip-lock. Thus, resuscitation can begin during a problem calving by stimulation of the calf's nasal receptors with straw or a finger (or an intranasal tube if oxygen therapy is available). Immediately after birth, the calf should be briefly suspended upside-down. This procedure facilitates postural drainage of pulmonary fluids and has a positive impact on pulmonary gas

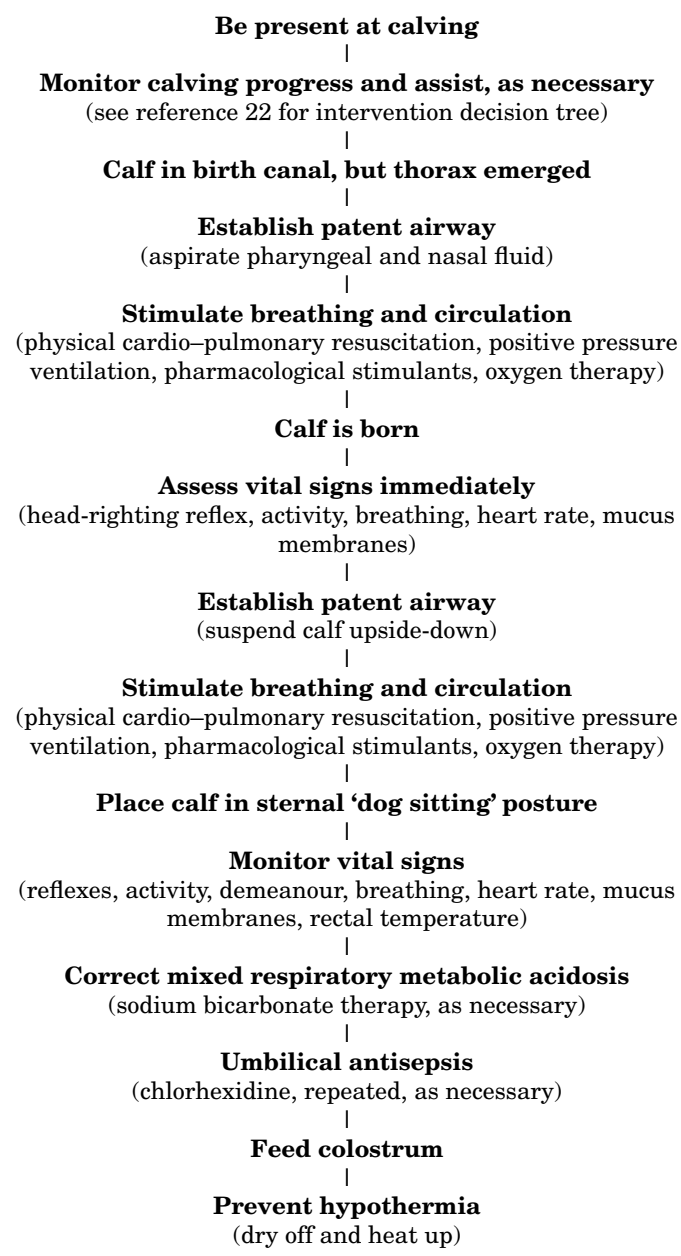


Figure 1. Standard Operating Procedure (SOP) for intensive care of at-risk newborn calves.

exchange, correction of mixed acidosis and subsequent absorption of colostral immunoglobulins.³³ These benefits accrue despite the fact that most of the fluid which drains from the calf is of abomasal origin. Clearance of the airways can begin with pharyngeal and nasal suctioning using an aspirator pump.¹⁷ Though only a small volume of fluid (<10ml) is generally removed, the procedure significantly benefits pulmonary gas exchange and acid-base balance.³⁴

Hypothermal stimulation has become the most common technique used to resuscitate calves by pouring cold water down the calf's ear or over the head or whole body to induce a gasp reflex. Recent research indicates that it has a beneficial effect on pulmonary gas exchange and acid-base balance in calves.³⁴ Squeezing the trachea gently may also illicit a cough reflex, which assists airway clearance. Once a patent airway has been established and breathing stimulation commenced, the calf should be placed in sternal recumbence in the 'dog sitting' posture, but with lateral support. This has a positive impact on physiological adaptation mechanisms, prevents hypostatic congestion in the dependent lung of lateral recumbence and facilitates attempts at positive pressure ventilation.³³ Acupuncture of the nasal philtrum or pinching of the nasal septum is also recommended, as it stimulates a specific inspiratory initiating reflex. Artificial respiration by expired air resuscitation techniques, such as mouth-to-mouth or use of emergency artificial respirators, has not been very successful in calves at this center.¹⁷ Compressed air devices (e.g. Ambu bag, Kruuse or H-K Calf Resuscitator), on the other hand, when used correctly (usually necessitating two people to operate), are clinically effective in newborn calves even without intubation.¹⁷

Oxygen therapy

Though widely used in human perinatology and for valuable calves (e.g. cloned calves), oxygen therapy for calf resuscitation is not widely practised on commercial dairy farms primarily due to the inconvenience of the equipment required. Studies in neonatal calves indicate oxygen therapy can improve perinatal survival.² For herd staff use, industrial oxygen can be administered with a face mask or intranasal tube, while experienced veterinary practitioners can administer oxygen via a cuffed endotracheal tube (7-9 mm).¹⁸ The latter route permits lower flow rates, and is more effective in lung expansion and ensures a patent airway.⁶ Oxygen can be used initially in primary apnea, where the calf has a heart beat but is not breathing immediately after birth, and in secondary apnea following dyspnea. A high flow rate (25L/min) is used along with sealing of the mouth, nostrils and esophagus to ensure immediate lung inflation in cases of partial atelecta-

sis. In cases where the calf is breathing but dyspneic, a lower flow rate (5L/min) can be used for insufflation until eupnea returns.¹⁸

Pharmacological stimulants

The clinical benefits of pharmacological respiratory stimulants, such as doxapram 0.45-1.36 mg/lb ([1-3 mg/kg] sublingual) and etamphylline (700mg sublingual or 9.1 mg/lb [20mg/kg] IM), in newborn calves are inconclusive,¹⁷ hence their use is often discouraged.⁶ However, they are widely used in practice and studies have shown positive effects on acid-base balance,³⁸ hence, they should be considered for use in dyspneic perinatal calves. In cases of bradycardia (<50 bpm) or cardiac asystole, external cardiac massage should be attempted and epinephrine can be administered intravenously (0.045 mg/lb; 0.1 mg/kg). Cases of sinus bradycardia may respond well to atropine (0.0045 mg/lb; 0.01 mg/kg).⁶ Given the limited use of i-STAT or Harleco portable blood acid-base analyzers under field conditions in veterinary practice, and our reliance on secondary signs of acidosis, all available combinations of resuscitation techniques should be attempted unless they are harmful to the calf or the operator. Further research studies are needed on best practice resuscitation protocols in compromised perinatal calves.

Fading calf syndrome

Following successful perinatal resuscitation, many calves that experience dystocia or prolonged calving and were resuscitated will still have a persistent mixed metabolic-respiratory acidosis in the first six hours of life. In addition, some calves develop secondary acidosis within 24 hours of birth with a poor suck reflex, poor colostrum immunoglobulin absorption, tachypnea, bradycardia, musculature flaccidity, depression and hypothermia. Such postnatal acidosis signs are significant risk factors for neonatal morbidity and mortality. In addition, calves suffering from full-term RDS show similar clinical signs which may be prevented by trace element supplementation. These calves deteriorate clinically and die within hours or days of birth, a condition described as the "fading calf syndrome" (personal communication 9 April, 2008: Reny Lothrop, Ontario, Canada). Correction of postnatal metabolic acidosis can be effectively achieved with drip or bolus intravenous infusion of sodium bicarbonate (250ml of 5% solution) instituted after resuscitation and repeated as necessary.⁴

Prompt feeding of colostrum, by esophageal feeder as necessary, provides both immunological and nutritional support for these weak hypoglycemic, often recumbent, calves that are at increased risk of omphalophlebitis, septicemia and diarrhea.

Recumbence in the newborn calf

Dairy calves born indoors should normally lift their head, attain sternal recumbency, attempt to stand and to stand spontaneously, on average, 3, 5, 20 and 60-90 minutes after birth, respectively.^{5,10,25} These intervals are primarily influenced by calving difficulty, metabolic acidosis, dam stimulation and condition of the floor. Occasionally, the calf remains recumbent after birth or is unable to stand, despite assistance, when the herdsman attempts to provide an early first suck of colostrum. The primary reason for this is trauma to the pelvic limbs' nerves, spinal cord, rib cage or occasionally the umbilical vessels, following excessive traction. Neurological trauma results in pelvic-limb paralysis but normal thoracic-limb function. Calves with unilateral paralysis may recover with intensive care over weeks, while the prognosis of those with bilateral paralysis is frequently hopeless.²⁶ Severe metabolic acidosis following prolonged calving or forced extraction is the next most likely cause of recumbence.²⁵ Affected calves have generalized, rather than localized, muscle flaccidity and superficial abdominal breathing. Recovery depends on the severity of the acidosis and the success of buffer therapy. Other uncommon causes of neonatal recumbence include congenital defects of the CNS (hydrocephalus or cerebellar hypoplasia, possibly due to genetic or infectious [bovine viral diarrhoea virus, *Neospora caninum*] causes); limbs (contracted tendons); heart (ventricular septal defects); or the umbilicus (omphalocoele). In all cases of neonatal recumbence, it is critical to ensure adequate colostrum feeding and provision of a clean, dry bed, as death is often due to secondary sequelae such as omphalophlebitis or infectious diarrhea.

Perinatal hypothermia

Though calves are born with precocial thermoregulation, primary hypothermia (rectal temperature 98.6°F, <37°C) may occur in up to a quarter of young calves in a temperate environment.²⁸ Perinatal hypothermia can be associated with increased time to stand, delayed absorption of colostrum immunoglobulins, frostbite and increased calf mortality. At birth, calf rectal temperature is higher than that of the dam and varies between 101-104°F (38.5 and 40.0°C).³⁵ While calves which suffer dystocia have a higher body temperature at birth than eutocical calves, they lose more body temperature after birth. This increased heat loss and reduced thermogenesis is due to acidosis and hypothyroxinemia in dystocical calves.³⁵ Other factors contributing to neonatal hypothermia include wind chill, wetness of the coat with placental fluids, mud or rain or wet bedding, and starvation. Thermoregulation can be achieved initially by non-shivering thermogenesis with catabolism of tissue substrates; activity, such as struggling to stand; licking

by the dam; and piloerection. If calf rectal temperature falls below 98.6°F (37°C) immediately after birth, warming procedures should be implemented such as placing the calf at the cow's head to encourage licking, drying off the calf with straw and ensuring an early intake of sufficient colostrum, which is essential to continued heat production after the first day. Recommended treatment of severely, prolonged cold-stressed calves is parenteral administration of a glucose solution (3-4ml/kg of a 10% solution IV)⁷ during rewarming with an infrared lamp, hair dryer, heat pad or warm water.

Umbilical care

Spontaneous rupture of the umbilical cord occurs during calving or shortly after, depending on whether the cow is standing or lying at calving and how lively the calf is immediately after birth. After rupture, the urachus and vessels normally retract into the abdomen, thus protecting them from environmental contamination. At assisted calvings there is a tendency to immediately rupture the cord in order to sit the calf up or to pull it around to the dam's head to allow her to lick the calf. Though research in calves is limited, one study found a long-term decrease in efficiency of pulmonary gas exchange in calves with assisted premature umbilical cord rupture compared to those with spontaneous rupture.⁹ Thus, there may be an advantage to leaving the cord to rupture spontaneously.

Omphalitis, or navel ill, occurs in 5 to 15% of newborn calves.³⁶ As it is generally untreated,³⁶ it can lead to reduced growth, joint ill and other sequelae. Omphalitis is a significantly bigger risk factor for reduced calf survival than respiratory, gastrointestinal or septicemic conditions and some authors have suggested it may warrant culling immediately upon diagnosis.

Prevention of navel ill is based on maintenance of maternity pen hygiene, reducing the residency time of calves in unhygienic maternity pens, ensuring adequate early intake of good quality colostrum and navel antisepsis. Despite widespread adoption and diverse, often conflicting, recommendations, there are very few research data on umbilical care in calves and no consensus of opinion. This is a knowledge gap requiring good applied research.

The benefits attributed to navel antisepsis include reduced calf morbidity and mortality, and reduction in respiratory disease. However, published studies showing a significant reduction of navel ill in calves were not found in literature searching for this article. Studies in babies have shown antiseptic cord care reduces bacterial colonization, exudate formation and foul odour compared to dry cord care.¹¹

In contrast to these positive outcomes, there are numerous reports of detrimental sequelae following navel antisepsis including increased calf morbidity

and mortality,³⁷ specifically prolonged cord drying time and detachment time, increased incidence of navel ill and treatment for pneumonia. In other studies, navel antiseptics failed to prevent omphalitis or was no more efficacious than no treatment. Studies in babies have linked topical iodine cord antiseptics to iodine overload, thyroid blockade and hypothyroidism, while *in vitro* studies have shown povidone-iodine (10%) to be markedly cytotoxic and to delay wound healing.

Choice of cord care procedure is also under-researched in calves. Topical antiseptics, particularly iodine and chlorhexidine (dip or spray), are more widely used in calves than topical antibiotic spray, cord clamping or ligation. Proposed state legislation to restrict supply of tincture of iodine may affect its future availability in the US. Chlorhexidine has a wide spectrum of antimicrobial activity, sustained residual activity and is efficacious in the presence of organic matter such as blood. With babies the cord is swabbed with triple dye, clamped and kept dry.

There are only a small number of studies comparing cord antiseptics in farm animals. In one epidemiological study, risk of calf mortality was significantly lower following use of chlorhexidine compared to iodine or no cord care; iodine tended to increase mortality risk.³⁷ A comparison of iodine (1, 2 and 7%) or chlorhexidine (0.5%) dipping or dry cord care in foals concluded that chlorhexidine and 7% iodine were the most effective in reducing cord bacterial growth, but 7% iodine occasionally sloughed the adjacent skin and may contribute to aseptic omphalitis.¹³ In one Irish study, the incidence of joint-ill did not differ significantly between lambs treated with an iodine dip or spray, or clamp, or lambs left untreated. The incidence of omphalitis did not differ between iodine dipping, cord clamping or cord emasculation in piglets.

It may be concluded from these widely varying reports that in herds without umbilical-associated problems, farmers should avoid possibly harmful cord application procedures and concentrate on maternity hygiene and calf immunity. In herds with serious navel ill problems, producers should consider how to improve maternity pen hygiene, institute immediate and repeated cord dipping with chlorhexidine (possibly in a paper cup to avoid container contamination), calf snatching, hand-feeding colostrum and regular checking for omphalitis (swollen and painful navel, pyrexia, disinclined to stand) with metaphylactic parenteral antimicrobial therapy based on veterinary advice. The need for controlled experiments on cord care in calves on commercial dairies is reiterated.

Umbilical haemorrhage

Occasionally newborn calves haemorrhage externally from the umbilical vessels, and this can be fatal.

Severe internal umbilical haemorrhage occurs in some 5% of stillborn calves,¹⁶ though this usually goes undiagnosed. Umbilical haemorrhage is often attributed by herd staff to a cow standing on the calf or sucking the navel, though it is more likely to be caused by incomplete contraction and closure of the umbilical vessels. Contributory factors may include inherited deficiency of Factors VIII or XI, maternal vaccinal-induced hemolytic disease, sweet clover poisoning or prematurity. While full-term newborn calves have the equivalent blood coagulation competency of adult cattle, premature calves have immature blood clotting mechanisms. Though vitamin K deficiency has been reported in cattle, it is not common. Prompt detection and cord ligation is successful in cases of external haemorrhage, but cases of internal haemorrhage are rarely diagnosed and so are not treated.

Failure to suck

Dairy calves born indoors should normally attempt to suck and to suck spontaneously, on average, 1-2 and 2-4 hours after birth, respectively.^{5,10} These intervals are primarily influenced by udder conformation; maternal behaviour, especially in heifers; and poor calf vigor, especially after dystocia. Occasionally calves will refuse to suck despite otherwise appearing healthy. The most likely cause is parturient metabolic acidosis leading to loss of the suck reflex. These calves, often described as 'stupid calves', are usually fed colostrum and later milk with an orogastric feeder, and while some cases resolve, others persist. Recommended treatment is to feed milk with an oral rehydration solution (e.g. Effydral; Solvay-Duphar) containing high levels of base precursor by orogastric feeder twice daily for one to two days.⁸

Calf movement after calving

In herds where paratuberculosis may be present based on a risk assessment, clinical history or laboratory results, newborn heifer calves in particular should not be allowed to suckle, but should be fed hand-milked colostrum from their dam and immediately removed ('calf snatch') and placed in a calf house or hutch (Figure 2). Where the risk of infectious disease is acceptable, calves may benefit from remaining with their dam to increase their opportunities to suckle naturally and to enhance the absorption of colostral immunoglobulin over colostrum fed in the absence of the dam. As cows tend to lick off antiseptics applied to the umbilicus, such antiseptics should be reapplied upon removing the calf from the maternity pen. Where maternity pen hygiene is poor, calf residency time should be reduced to a minimum to prevent common calthood infectious diseases. Moving the calf from the maternity pen to the calf house presents an opportunity to conduct a quick

Period	Do	Don't
Pre-calving	Move cows to the maternity unit before calving commences.	Move cows during stage one of calving.
Calving	1. Provide deep straw bedding in individual maternity pens. 2. Monitor cows every 3-6h after the onset of stage I of calving. 3. Intervene at least 2hrs after the onset of stage II of calving.	1. Overcrowd group maternity pens. 2. Unnecessarily disturb cows during stage 1 or 2 of calving. 3. Tether heifers at calving, unless for assistance.
Post-calving	1. Assess calf vital signs immediately after calving. 2. Acquire resuscitation aids and train staff members in use of resuscitative techniques. 3. Implement umbilical antiseptics.	1. Cut the umbilical cord or rupture it prematurely. 2. Assume a weak calf will suck adequately eventually. 3. Leave the calf with the dam in herds with paratuberculosis.

Figure 2. Dos and Don'ts of newborn calf management.

check on the calf's health status. Problems to look out for include persistent signs of acidosis, dyspnea, umbilical bleeding or organ eventration and hypothermia.

Perinatal mortality

Despite the best efforts of producers and their veterinarians to manage calvings and newborn calves successfully, perinatal mortality can be a perennial problem on some dairies, yet rarely occur on others. Currently, the reported stillbirth rate (born dead) in US dairies is 8% (singletons 7.2, twins 28.2%).²⁷ This average figure obscures the fact that PM follows a right-skewed distribution where most herds have none or minimal losses, but some herds have high (25%) mortality.²⁴ There is inadequate existing research on the causes of this wide variation in herd PM rates and well designed, transdisciplinary studies are warranted. Significant animal-level risk factors for PM include parity, twinning, calf gender, shorter or longer gestation length and sire predicted transmitting ability (PTA) for PM.^{24,27} Traditionally, the majority of PM has been attributed to dystocia.¹⁹ Recent research indicates that the proportion of PM attributable to dystocia may be decreasing.^{1,20} The main causes of PM are anoxia and trauma, following dystocia, and to a much lesser extent death *in utero* and premature placental expulsion.^{15,16} One author reported that up to 40% of veterinary-assisted deliveries may result in rib fractures, and up to 10% in vertebral fractures³² and 10-15% of calves delivered using a calving aid suffered traumatic injuries at calving.³⁹ Perinatal mortality following eutocia (often called 'weak calf syndrome') may be associated with intrauterine growth retardation (IUGR) or prematurity, congenital defects, infections, pre-calving nutrition,

dysmaturity, twins, placental dysfunction or sire-specific genetic weakness leading to poor perinatal viability. Other causes are prolonged stage-one labor with premature placental separation or prolonged stage-two with uterine atony, or nitrate toxicity and accidents. As in many cases of PM the etiology is undetermined, it has been suggested that veterinary pathologists may need to utilize the experience from the work-up of human stillbirths.¹⁴ As the incidence of idiopathic PM appears to be increasing, there is a need for renewed research focus on this cohort of calves to determine the modifiable risk factors and etiology of this syndrome.

Conclusions

Despite advances in dairy herd health and productivity, perinatal calf mortality rates are still unacceptably high on many dairy farms. While some of this loss has a genetic origin and may be outside the producer's control, management strategies at the herd level and management procedures at the animal level can be implemented to improve perinatal welfare. The key features of successful newborn dairy calf management are ensuring heifers and cows are moved in time to calve in suitable maternity housing, discreet calving supervision and appropriate timing of any necessary calving assistance, immediate parturient evaluation of at-risk newborn calves followed by aggressive resuscitation, strategic umbilical antiseptics, early detection (and treatment) of perinatal problems and prompt movement of the newborn calf to hygienic calf housing. Veterinarian-led producer implementation of active management of calving and newborn calf care can improve perinatal welfare and health.

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