

University of Nebraska
Great Plains Veterinary Educational Center

Calving Elective

INTRODUCTION

This manual is intended to serve as a review and study guide for students participating in the calving elective. The handbook deals with pertinent topics related to calving. The calving season has a variety of events that must be handled by the caretakers. As the calving season approaches, nearly all the pregnant cows are in the last third of the gestation period. This review will start with the gestation period and includes conditions associated with the pregnant cow, calving, and post calving care of the cow and calf.

KEY TERMS

GESTATION (PREGNANCY) - The period from the fertilization of the ovum, or conception, to the birth of the young animal.

ABORTION - The expulsion from the uterus of a living fetus before it reaches a viable age, or, more commonly, the expulsion of a dead fetus of recognizable size at any stage of pregnancy.

HORMONE - A chemical substance, produced in the body, which has a specific effect on the activity of a certain organ or organs.

PARTURITION (LABOR) - The series of physiological and physical steps which occur in the last days of the gestation period which results in the expulsion or attempted expulsion of a full term living fetus and fetal membranes from the uterus.

The normal gestation period for cattle varies from 273 to 296 days depending on the breed and species of the animal. Cows carrying twin fetuses usually have gestation periods 3 to 6 days shorter than similar cows carrying a single fetus. Diseases affecting the health of the uterus, placenta, or the health of the fetus can also cause shortened gestation periods. The gestation period is under hormonal control. Abortion, premature birth, and normal birth can be explained by changes in the balance of hormones maintaining pregnancy.

POINTS TO REMEMBER

1. Abortions can be caused by both infectious and noninfectious events.
2. A low level (2% or less) of gestational losses is probably average across all herds.
3. Diagnosis of causes of abortions is difficult. However, the animal's history, examination of the aborted placenta and fetus, and the collection of paired serum samples from the cow can be useful in establishing a diagnosis.

ABORTION can be caused by a variety of reasons and can be considered average in accounting for a 2% difference in the number of cows being diagnosed pregnant at 90 - 120 days of gestation and the number of cows giving birth to full term fetuses. Abortions can result from both infectious and noninfectious causes. Infectious causes could include, but not limited to, such bacterial causes as Brucellosis, Leptospirosis, and Vibriosis (Campylobacter); viruses like infectious bovine rhinotracheitis (IBR) and bovine virus diarrhea (BVD), as well as fungal and protozoal infections. Some noninfectious causes would include certain chemicals, drugs and poisonous plants; exogenous hormones, nutritional deficiencies, physical interruption of pregnancy, genetic or chromosomal defects, and such miscellaneous causes as multiple fetuses and tumors of the uterus.

If abortion rates closely approach or exceed the average 2% level, it is important to try to determine the cause of the abortions. The fetus and fetal membranes are useful sources of information both as to the number of fetuses present and for the potential to provide a laboratory diagnosis of infectious or toxic causes of abortion. Unfortunately, in extensive beef production systems, these tissues are frequently not found or are in too poor condition to provide a diagnosis. An alternate method is to collect paired blood samples (one at the time of abortion and a second 2-3 weeks later) from aborting cows to test for antibody titer changes for the more common infectious causes of abortion. This is the approach that is normally taken at MARC. Abortion is usually followed by a retained placenta in the cow. The treatment for this condition is described later under post partum care of the cow.

KEY TERMS

PLACENTA - The placenta is composed of two parts: the fetal placenta, or allantois chorion, and the maternal placenta, or endometrium. These membranes serve as protection to the fetus, a means of getting nutrients from the dam to the fetus, and storage of fetal waste products. The placenta also produces various hormones that are necessary for maintaining pregnancy and influencing parturition. These membranes are expelled following the expulsion of the fetus from the uterus.

FETUS - The fetus is the developing intrauterine individual following the embryonic formation of the major tissues, organs, and systems of the body. This stage begins at approximately 45 days of gestation in the bovine and ends at parturition. During this time the individual produces and utilizes the placenta for obtaining nutrients from the dam and undergoes minor differentiation of tissues, organs, and systems as well as the growth and maturation necessary to sustain life free of the uterus and placenta of the dam.

HYPOTHALAMUS - A portion of the brain located in close proximity to the pituitary gland that has considerable control over the physiological functions necessary to maintain the dynamic state that is life. It produces releasing factors which regulate the production of hormones that influence reproduction, pregnancy, and parturition.

PITUITARY GLAND - The endocrine gland which produces oxytocin, gonadotropic hormones (FSH - follicle stimulating hormone and LH - luteinizing hormone), and adrenocorticotrophic hormone (ACTH that regulates the production of cortisol by the adrenal gland).

ADRENAL GLAND - The endocrine gland closely associated with the kidney that is regulated by ACTH from the pituitary gland. The gland produces cortisol that plays an important role in the termination of pregnancy.

CORTISOL - A hormone produced by the adrenal gland in response to stress or stressors in the animals environment. Cortisol can be produced by both the fetus and the dam to cause adaptive changes that can cause the termination of pregnancy and the preparation for extrauterine life for the fetus.

ESTROGENS - A hormone produced by the ovary and the placenta that sensitizes the uterine musculature to become stimulated by oxytocin. It also causes relaxation of ligamentous structures of the pelvis and birth canal to aid the passage of the fetus from the uterus to the outside world.

OXYTOCIN - A hormone produced by the posterior portion of the pituitary gland under influence of neurotransmitters released by the hypothalamus. Oxytocin causes contraction of the estrogen sensitized uterine muscles, and milk letdown in the mammary gland.

PROTAGLANDINS - Hormones that are produced within the uterus that cause the destruction of the corpus luteum (produces progesterone) of the ovary. The removal of the calming action of progesterone on the uterus allows it to be influenced by other hormones. This allows the

events to occur that expel the fetus from the uterus. Though several types of prostaglandins are produced in the body, those produced locally in the uterus have the greatest effect on the reproductive tract.

MYOMETRIUM - The longitudinal and circular smooth muscle of the uterus. This muscle is capable of great changes in volume during the course of the gestation period, and when sensitized by estrogen and stimulated by oxytocin is capable of very strong productive contractions that help force the fetus and fetal membranes from the uterus.

PROGESTERONE - A hormone produced by the ovary within a few days after ovulation. It is essential for the maintenance of pregnancy. As the fetal membranes develop some progesterone is produced by the uterine glands. Progesterone has a calming effect on the myometrium and promotes development and maintenance of the fetoplacental unit.

AUTONOMIC NERVOUS SYSTEM - That portion of the nervous system that is responsible for the visceral or vegetative control of the body. Its overall action is to maintain the internal environment of the body within carefully controlled limits. This action incorporates many dynamic processes, the equilibrium of which is so highly controlled that under normal circumstances it may appear to be static.

CHORIOALLANTOIC SAC - That portion of the fetal membranes that allows the exchange of nutrients, gases, and wastes between the fetal and maternal circulation. This forms the fetal half of the placenta with the other half being the endometrium of the dam.

AMNIOTIC SAC - The inner most sac surrounding the fetus. It is the first sac to form. At term its mucoid fluid aids in the birth process by lubricating the fetus and the birth canal. It is often termed the "foot sac" when it appears at the vulva during the 2nd stage of labor.

POINTS TO REMEMBER

1. Parturition is under the control of hormones, the autonomic nervous system, and mechanical stimuli.
2. Temporary interruption of labor is possible if animals are not handled properly during the early stages of labor.
3. Labor is arbitrarily divided into three stages: Cervical dilatation, fetal expulsion, and the expulsion of the fetal membranes.
4. The signs of each stage are: Stage 1 - seeks out an isolated location, thick mucus type vaginal discharge, occasional signs of abdominal colic, and restlessness(takes from 6-10 hours); Stage 2 - the breaking of the "water sac", the appearance of the amniotic sac and feet at the vulva, an ever increasing frequency of abdominal press type contractions (3-5 minutes apart, early, and every 1 ½ minutes near the end), and the expulsion of the fetus. The completion of this stage should within 2-3 hours, depending on the parity of

the dam; and stage 3 the complete expulsion of the placenta within 8 hours after the end of stage 2.

PARTURITION (LABOR) is the period in which the multiple physiological and physical events occur that results in the birth of the calf. Parturition is a very complex event. Not all the hormonal and mechanical interactions are understood; however, the following summarizes the factors associated with control of labor:

Placental Morphologic Change may occur during the last part of gestation. The stimulus for these changes is not defined. *Result:* Decreased efficiency of the placental unit could decrease nutrients to the fetus or cause accumulation of toxic metabolites resulting in a stress situation.

Fetal Cortisol production is elevated by increased activity of the fetal hypothalamo-pituitary-adrenal axis. This may be due to fetal stress. *Result:* Cortisol stimulates production of estrogens and prostaglandins by the feto-placental unit. These sensitize the uterus to oxytocin, potentiate uterine contractions, and stimulate maternal release of oxytocin. Decreased progesterone production occurs, releasing the uterus from its influence.

Estrogens from the feto-placental unit are elevated in most species prior to and during parturition. Their production is associated with increased secretion of cortisol. *Result:* Estrogens stimulate increased release of maternal oxytocin and prostaglandins which work with estrogen to cause cervical dilation, increased myometrial sensitivity, and excitability, and stronger contractions.

Progesterone from the feto-placental unit and (CL in some species) prevents parturition until its production and effects are inhibited by glucocorticoids and prostaglandins. *Results:* Progesterone promotes a quiescent (tranquil) uterus by decreasing excitability of the myometrium. Decreased progesterone allows onset of labor by allowing increased myometrial sensitivity and excitability. Excess progesterone blocks parturition induced with steroids.

Oxytocin is secreted by the dam under the influence of estrogens and prostaglandins from the feto-placental unit and, especially, in response to mechanical stimulation of the cervix and vagina. *Results:* Oxytocin stimulates myometrial activity and speeds parturition. It also causes increased secretion of prostaglandins.

Production of Prostaglandins is stimulated by fetal corticosteroids and estrogens from the feto-placental unit. *Results:* Causes myometrial contractions. May also cause release of oxytocin, stimulate production of free estrogen, and inhibit production of progesterone.

The Autonomic Motor Innervation of the uterus can assist in regulation of parturition. The external environment affects both the time of onset and duration of labor, actions mediated through neural pathways. *Results:* Excitement can cause delay, temporary cessation or decreased intensity of labor. Quiet surroundings promote stronger contractions and hasten parturition. Oxytocin release can also be mediated through neural pathways.

Mechanical stimulation due to increased uterine size and from the presence of fetal parts in the birth canal. *Results:* Point pressure in the vagina reflexively stimulates the abdominal press

and oxytocin release. Contractility of uterine muscles is increased as the uterus enlarges. Uterine and abdominal muscle contractions are increased via direct myogenic and/or spinal reflex mechanisms.

These combinations of actions cause a number of outward signs of labor to appear which should be observed by those caring for the cows. Normal parturition (labor) is a continuous process, once it is started, but it is often divided into three steps called the stages of labor. They are: Stage 1, the stage of cervical dilation; Stage 2, fetal expulsion; and Stage 3, expulsion of the fetal membranes (placenta). Dystocia occurs when any stage is slow developing or fails to progress normally. Also, when dystocia occurs the stages may get out of sequence such as the fetal membranes being released from their uterine attachments prior to the delivery of the fetus.

Stage 1 (the stage of cervical dilatation) begins when the muscle fibers of the uterus start to contract and ends when the cervix is dilated and fetal parts enter the birth canal. Visible signs of labor are scanty or absent during this stage. The pastured cow will usually seek an isolated place and vaginal discharges increase with thinning and expulsion of the cervical plug. Occasionally, signs of colic (kicking at the flank and getting up and down) are evident, especially in heifers. Generally this stage lasts from 2-6 hours or longer in heifers.

Stage 2 (the stage of fetal expulsion) begins when fetal parts enter the birth canal and stimulate the abdominal press. The chorioallantoic sac ("water bag") is usually ruptured early in 2nd stage labor, and the unbroken amniotic sac is often forced through the vulva after the cow has been in labor for a short time. *Delivery should be completed in cattle within two hours after the amniotic sac appears at the vulva.* The feet of the fetus are forced through the amniotic sac either just before or after it comes to the vulva.

During stage 1, uterine contractions first occur every 15 minutes but by the beginning of the 2nd stage they occur about every three to five minutes. When point pressure is applied to the birth canal by fetal parts, the uterine contractions are accompanied by the abdominal press. The press is exerted more frequently as labor progresses until it occurs every one and one-half to two and one-half minutes. A series of frequent presses followed by a short period of rest is characteristic of this stage of labor. The greatest frequency and force occur when the fetal head is forced through the vulva. Following delivery of the head a short period of rest may ensue. The expulsion of the thorax also requires a strong effort on the part of the cow. The cow may again rest a short period before expelling the hips and hind legs. This is normally uneventful since the umbilical cord is still attached and is supplying oxygen to the fetus.

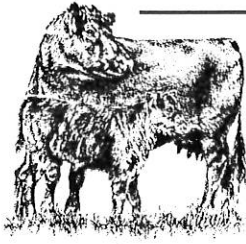
Functional Staging of Fetal Deaths

B E F O R E	Antepartum Death (APD) Classification	
	APD 1	Premature gestational age & mummification (Dead for a long time)
	APD 2	Premature gestational age & generalized hemoglobin staining (Dead for > 12 hrs)
	APD 3	Premature gestational age & yellow edematous fluid or no edema (Dead for < 12 hrs)

D U R I N G	Partum Death (PD) Classification					
	Class	Time Death	Duration Birth	Umbilical Art. Clot	Local Edema	Renal Autolysis
	PD 1	Early	Moderate	Absent	Little or None	Moderate
	PD 2	Early	Long	Absent	Little or None	Marked
	PD 3	Middle	Moderate	Absent	Moderate	Moderate
	PD 4	Middle	Long	Absent	Marked	Marked
	PD 5	End	Moderate	Absent	Moderate	None
	PD 6	End	Long	Absent	Marked	None
	PD 7	Early	Short	Absent	Little or None	None

A F T E R	Postpartum Death (PPD) Classification						
	Class	Umbilical Art. Clot	Breathed	Walked	Nursed	Food Passed GI	Fat Metabolized
	PPD 1	Yes	No	No	No	No	None
	PPD 2	Yes	Yes	No	No	No	None
	PPD 3	Yes	Yes	Yes	No	No	None
	PPD 4	Yes	Yes	Yes	Yes	No	None
	PPD 5	Yes	Yes	No	No	No	Moderate
	PPD 6	Yes	Yes	Yes	No	No	Moderate
	PPD 7	Yes	Yes	Yes	Yes	No	Moderate
	PPD 8	Yes	Yes	Yes	No	No	Complete
	PPD 9	Yes	Yes	Yes	Yes	No	Complete
	PPD 10	Yes	Yes	Yes	Yes	Yes	None
	PPD 11	Yes	Yes	Yes	Yes	Yes	Moderate
	PPD 12	Yes	Yes	Yes	Yes	Yes	Complete

Six Functional Classes of Perinatal Death		
Before	Antepartum Death (Abortion)	ADP 1-3
During	Early Parturient Death (Fetal Weakness)	PD 1-3 & 7
During	Later Parturient Death (Birth Trauma)	PD 4-6
After	Immediate Postpartum Death (Neonatal Weakness)	PPD 1-4
After	Delayed Postpartum Death (Neonatal Weakness)	PPD 5-7
After	Late Postpartum Death (Complicated & Uncomplicated Starvation)	PPD 8-12



Western Beef Resource Committee

Cow-Calf Management Guide

Reproduction Section

CL450

Designing a Cattle Obstetric Stall

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Every ranch needs to design a functional calving assistance area to increase profits by decreasing calf death losses, animal injuries, and increase subsequent conception rates. Proper and timely assisted births can increase cow and calf vitality, which in turn positively affects growth and reproduction resulting in higher dollar returns to the ranch. As with any other job it is much less difficult to assist in the delivery of a calf if proper equipment and facilities are available.

Proper facilities can affect the motivation to bring the cow or heifer in the barn and allow assisting birth without undue stress on the animal or the producer. The animal should move to the area easily, be constrained without fright, and then helped with the birthing process. With inadequate facilities the cattle producer often delays assistance and has difficulty in corralling and restraining the animal. Frequently, this results in problems with the "mothering up" or bonding process after birth is completed. A calm, unhurried manner promotes successful results.

The facilities should be designed for easy animal movement and located in an area familiar to the heifers. The OB stall can be outside although inside a barn is often a more pleasant environment on a cold snowy night. Feeding heifers in the general area will allow them to be familiar with the surroundings and move into the area with ease.

A concrete pad is helpful. After several births, the area tends to become muddy and slick. A pad of rough concrete provides sure footing, as well as a drier, cleaner environment. The pad can be swept clean or a floor drain provided to remove liquid and placenta. A floodlight above and behind the animal is also helpful. The obvious benefit is to be able to see what is needed. A light may not heat an area, however, it does "feel" warmer than working in the dark.

Hinged, swing away, or interchangeable panels (gates) allow flexibility in design and aid in cattle movement. These are attached on either side of the head catch to (1) facilitate moving the heifer into the catch and (2) aid in holding the heifer quiet as assistance is given. Once assistance is started the gates need to swing away from the animal so that it might lay down in the birth process. These panels can form a small pen to hold heifer and calf after birth.

The natural actions of cattle after an unassisted birth is to stand, pivot 180°, and begin to mother (lick, etc.) the calf. This action not only dries the calf but stimulates it to move, breath, and get up and bond with the mother. To simulate this action the heifer should be allowed to back out of the head catch and pivot with her head down. All she can smell at this point is the calf. Bonding (mothering) will generally occur quickly. If an animal is moved to a new location before bonding has taken place, this process is much slower. The design of the facilities should allow the heifer to mimic this natural instinct.

Head Catch

Several commercially available head gates are acceptable for a calving stall. It is essential they open all the way to the floor and have straight side bars that constrain the head. These design peculiarities allow the heifer to lay down during the process without the danger of "choking down." A curved head catch gate can be modified by welding a straight pipe into the curved section. A wooden head catch may be less expensive (Fig. 1) but should open to the floor.

The gate can be equipped with a rope to lock the head from the rear or side of the animal when desired. The area beyond the head gate should be open and lighted so the animal will readily enter. A dark hole

will discourage most cattle from putting their head through the opening to allow head catch closure.

A squeeze chute is not an acceptable alternative. In proper assistance the calf needs to be delivered in an arc with final pressure directed toward the heels of the mother. In many births the heifer will lie down on her side for delivery that she cannot do in a squeeze chute. Furthermore, an operator will not have room to maneuver the calf or any mechanical device to direct the appropriate "downward" pressure. Worse yet is the case when the heifer goes down on her belly. She cannot be rolled to her side in the squeeze chute.

Often a producer is tempted to use a board or belts to hold the animal up. This does not allow room for the arched delivery and eliminates the squeeze as an alternative to the simple self catch gates. Further, it is more difficult to release the heifer postpartum and mimic the natural instincts as described previously.

Design

The head gate is placed between two posts in a fence line. These posts are also used as hinge holders for 8- or 10-foot panels. The panels can be brought together to move the animal's head into the catch and then fastened with a chain at the back end. Often the gates need to be stabilized to prevent swinging from side to side. This can be as simple as an angled foot brace on both sides of the panels.

A second set of gates should be hinged on the opposite side of the pen so that both sets (4 gates) can be swung open as delivery occurs. This allows room to correctly work with assistance equipment. Also with these open there is sufficient area for the postpartum

bonding process. See Fig. 2 for one pen design example that can be modified to match gates and areas on most ranches.

One set of side gates can be closed, and the heifer brought to the small pen and manipulated into the head catch by using the second set of gates as leverage. A chain to hold the side panels together will confine the animal and reduce the possibilities of being kicked. Once assistance is started the gates can be swung back out of the way.

Once delivery is complete, place the calf in the back corner of the pen near the heifer's hind feet. Make certain the calf is breathing and apply iodine to the naval. Let the heifer back out of the head gate and leave her with the calf.

To accommodate the occasional cesarian section the left gate can be modified by being cut in half horizontally, thus allowing the top section to swing out of the way. If a calf needs assistance in nursing (a bad uddered cow, a weak calf, or a graft) the lower portion can be opened while the top restrains the heifer. These procedures should allow both animal and human relative safety.

Other items that will get used repeatedly are a few small 10- or 12-foot square pens. These can be used to continue and encourage the bonding process, help graft calves, or doctor sick cows and calves. The design of these pens should allow easy cleaning and sanitation.

Calving facilities should be user-friendly for both the cattle producer and the heifer. They should provide a safe, clean environment for the entire birthing process that should result in more live calves, healthier calves, easier rebreeding cows, and increased profits to the ranch.



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Second edition; January 2002 Reprint

Nutrition and Feeding of the Cow-Calf Herd: Production Cycle Nutrition and Nutrient Requirements of Cows, Pregnant Heifers and Bulls

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Developing diets and feeding strategies for the cow-herd is facilitated by a basic understanding of the production cycle of the cow and her changing nutrient requirements. By knowing and anticipating the changing nutritional needs of the cow, producers can plan their feeding programs and lower feed costs. Cows use the nutrients provided to them for bodily processes in the following order: 1) maintenance – keep alive and moving, 2) lactation – providing milk for the calf, 3) growth – including weight gain, and 4) reproduction.

Beef cow production cycle

For nutritional and most management purposes, the annual production cycle for the beef cow can be divided into 4 phases: Pre-calving, Postpartum, Lactating and Pregnant, and Gestation. Each one of these phases is physiologically unique and each has its own set of nutritional requirements (Figure 1). Calving is the event on which all of these periods are based, so that's where we will start.

Postpartum (after calving) is the 80 to 90 day period that begins at calving. It is the period of greatest nutritional demand (Table 1 & 2). Cows must lactate, repair their reproductive tracts, resume heat cycles, breed, increase activity and, if young, grow. All these processes put considerable strain on the cow. However, her voluntary feed intake, how much feed she will eat, is highest during the postpartum period. If she is not fed to meet her nutritional demands, she will fail or be delayed in rebreeding and lose weight.

Lactating and Pregnant is a period of 120 to 130 days. Nutritional requirements are still high. However,

energy requirements decrease about 13% and protein needs about 8% compared to the postpartum period. During the lactating and pregnant period, cows reach peak lactation and then decrease milk production. Cows are pregnant, but the limited fetal growth does not add much to requirements. However, activity is still high, and two and three year-olds must continue to grow. Cows usually lose some weight during this period.

Gestation is the 100-110 day period immediately after the calves are weaned. Nutritional requirements are at their lowest because lactation has ceased. Energy needs are 23% less than the previous period and protein requirements drop by 36%. This is the best time to put

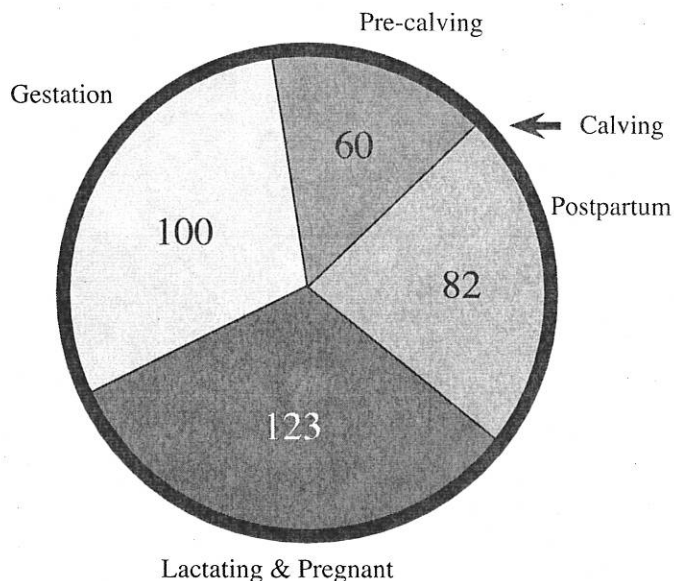


Figure 1. Nutritional & Management phases of the annual cow production cycle.

weight back on thin cows and increase body condition to BCS 5 or 6. Cows are pregnant, but growth of the developing calf is still slow and activity decreases; however, heifers still need to gain 1 to 1.5 lbs per day. The cow's voluntary feed intake is lowest during this period.

Pre-calving is the period 50 to 60 days immediately before calving. This is the most critical period of the year. Cows must reach or preferably maintain body condition score 5 or 6 during this period. Cows must calve in body condition score 5 or greater to have healthy calves and breed back quickly (Figure 2). Energy and protein needs increase by 20% or more compared to gestation (Table 1 and 2). Fetal growth is rapid. The calf may gain 60 lbs during pre-calving, and the placenta is also growing. Cows need to gain 1 to 1.25 lbs per day, while heifers and young cows need to gain 2 to 2.5 lbs per day. Along with fetal and placental growth, cows are preparing for lactation. Late in this period feed intake may decrease because the fetus and associated structures take up space normally occupied by the rumen.

Factors affecting nutrient requirements

The nutrient requirements of beef cows can be broken down into four principal components: Maintenance, Lactation, Growth, and Reproduction. From these components, requirements for energy, protein, minerals, and vitamins are calculated. By understanding the different factors that affect requirements, producers can make adjustments to changes

such as a month of cold weather, moving to a hilly pasture, or the last third of pregnancy.

Maintenance. The maintenance component includes all the nutrients required for the animal to breath, move, digest food, keep warm, repair tissues, and maintain body weight. Weight, age, breed, physiological status, activity, and environmental conditions are the primary variables impacting maintenance requirements. The larger the animal, the greater its maintenance requirement, especially energy and protein. Extremely heavy muscled breeds will have greater maintenance requirements than light muscled breeds. Pregnancy and lactation increase basal metabolism, so maintenance requirements are altered accordingly. Heavy milking breeds have an increased maintenance requirement. Increased activity or rough terrain will increase maintenance energy needs as will extremely cold, hot, wet, or muddy conditions.

Even though all nutrients are needed for maintenance, only energy requirements are divided into maintenance and non-maintenance portions. This division is made because energy is used more efficiently for maintenance than for other body processes such as growth. When net energy (NE) requirements are used instead of TDN, you will notice that there are separate requirements for NEm (maintenance), NEg (gain), and NEl (lactation). Furthermore, the NEm values for feeds are greater than those for NEg.

Lactation. Nutrient requirements for lactation are based on the amount of milk at peak lactation and the composition of the milk. Cows that produce more milk, and milk with more fat and protein, will have higher nutrient requirements.

Growth. Requirements for growth are determined by actual weight, average daily gain (growth rate), weight at maturity, and composition of gain. Composition of gain simply means whether cattle are putting on more muscle or more fat. For example, protein requirements will be higher for young cattle because they are gaining more muscle than fat. When cows need to gain weight to increase their body condition score, this is also considered growth.

Reproduction. Adjustments to requirements for reproduction are based on expected calf birth weight and stage of gestation. Usually, pregnancy does not significantly affect requirements until the last three months of pregnancy when the fetus is growing rapidly.

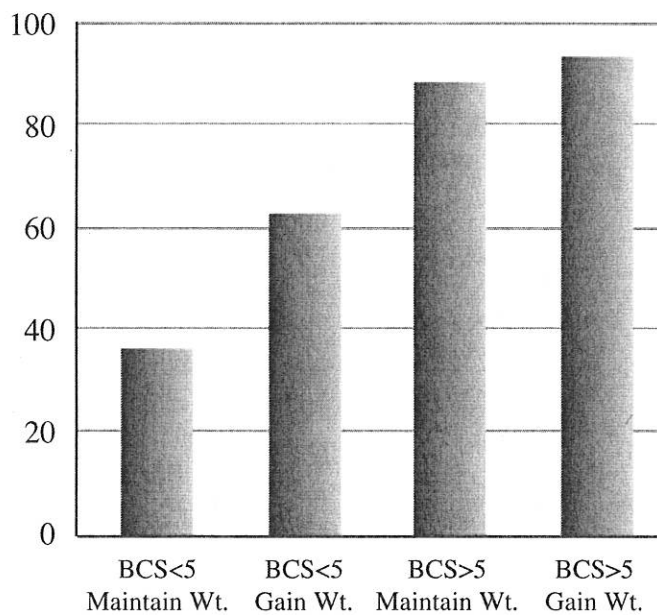


Figure 2. The effects of body condition at calving and postpartum gain on conception rates in heifers.

How to use requirement tables or calculate requirements

There are two ways to determine the nutrient requirements of beef cows and calves. The first and most useful for most producers and Extension personnel is to use pre-calculated tables of nutrient requirements derived from the Nutrient Requirements of Beef Cows (NRC, 1996). Except for unusual circumstances, these tables give sufficiently accurate requirements for beef cows, heifers, and young calves. Tables 1-4 contain simplified tables for the major classes of cattle and nutrients. More detailed tables in terms of milk production and physiological status are available from the Arkansas Cooperative Extension Service (Publication MP 391). Tables 1-4 will provide sufficient accuracy to design feeding programs for most producers. Note that the diet nutrient density requirements in the tables are on a dry matter (DM) basis.

The second method is to use the new Nutrient Requirements of Beef Cows computer program. Nutrient Requirements of Beef Cows (NRC, 1996) brought about dramatic changes in the power, flexibility, and accuracy of determining the nutrient requirements of beef cows. The new formulas and computer program can take into account many factors including breed, weight, body condition, physiological stage, milking ability and composition, environment, etc. Although very powerful, this new program is very complex and cumbersome for producers and Extension personnel who have not had extensive nutritional training or training with the program. If you are interested in using this program, you should contact a trained Extension professional or nutritionist to assist you.

To use Tables 1-4 to determine nutrient requirements of cows use the following steps: 1) Locate the table with the type of animals you want requirements for (i.e. Mature Cow, Pregnant Heifer, etc). 2) Pick the production period of the animal (i.e. Gestation, Post-

partum, etc). 3) Locate the average body weight of the animal and read across. This gives you the animal's daily nutrient needs in pounds per head per day. 4) Look at the required nutrient density line at the bottom of the requirements for that particular production period. This gives you the minimum nutrient density or concentration of nutrients needed in the diet.

Either an Animal's Daily Nutrient Needs or Diet Nutrient Density can be used to design diets to meet the nutritional needs of beef cattle. Because cows are generally allowed to eat all they want, the Diet Nutrient Density Requirements in dry matter are most useful. Basically, if a cow eats all she can consume of a diet containing the required percentage of a nutrient, she will consume the needed amount of that nutrient each day.

Tables 1-4 also indicate a dry matter intake requirement or figure. This figure is a guide to how much 100% dry feed an animal could or should eat. It is not the total pounds of feed in its normal or as fed form an animal could eat.

For example, an 1100 pound cow in the pre-calving period would need to eat 22.7 lbs (dry matter basis) of a feed that was 54.6 TDN and 8.6% crude protein to meet her requirements. You have hay on farm that was 85% DM, 55% TDN, and 10% CP. This meets her needs for energy and exceeds her need for protein; so how much do you need to feed her? Use the following formula:

$$\frac{\text{Lbs DM required}}{\% \text{ DM of the feed}} = \text{Lbs of feed needed}$$

$$\frac{22.7 \text{ Lbs. DM}}{.85} = \text{so you would feed her 26.7 or 27 lbs of hay.}$$

For more assistance with calculating diets or evaluating feeds, contact your County Extension Animal Science Agent.

Example Diets for Beef Cattle

Late gestation cows (1200 lb. Last 60 days of gestation)

1. 30 lbs good quality hay*
2. 30 lbs fair quality hay* plus 1 lb corn
3. Stockpiled fescue
4. 25 lbs 80% poultry litter 20% corn plus 5 lbs poor hay
5. 60 lbs corn silage plus 1 lb protein supplement
6. Good quality fall pasture

Lactating cows (1200 lb. Average milking ability)

1. 32 lbs good hay plus 1 lb corn plus 1 lb protein supplement
2. 32 lbs fair hay plus 5 lbs corn gluten pellets
3. Spring pasture, good quality summer pasture or excellent stockpiled fescue
4. 28 lbs 80% poultry litter 20% corn plus 5 lbs hay
5. 68 lbs silage plus 4 lbs protein supplement

Gestating cows (Mid Gestation)

1. Stockpiled fescue
2. Moderate quality pasture
3. 25 lbs average quality hay
4. Grazing corn stalks plus 1 lb cottonseed meal

Pregnant replacement heifers (Late Gestation)

1. Good quality grass – legume pasture
2. 21 lbs good quality hay plus 6 lbs cracked corn and 1 lb soybean meal
3. 21 lbs good quality hay plus 7 lbs barley
4. 40 lbs corn silage plus 4 lbs whole cottonseed

Pregnant heifers (Mid Gestation)

1. Good quality pasture
2. Stockpiled fescue
3. 22 lbs good quality grass hay plus 3 lbs barley
4. 34 lbs corn silage plus 2 lb soybean meal plus hay

Lactating 1st calf heifers

1. 24 lbs good hay plus 3 lbs corn plus 2 lbs soybean meal
2. 24 lbs good hay plus 6 lbs corn gluten pellets
3. Abundant spring pasture

Young herd bulls (12-24 months)

1. High quality pasture plus 12 lbs corn
2. 20 lbs grass legume hay plus 12 lbs corn
3. 80 lbs corn silage plus 2 lbs protein supplement

Mature herd bulls

1. High quality pasture plus grain if needed
2. 30 lbs of good quality hay plus grain if needed
3. 70 lbs corn silage plus 1.5 lbs protein supplement

* Good quality hay = >56% TDN; >10% CP

Fair quality hay = 50 - 55% TDN; 8 - 9% CP

Table 1.

Daily Nutrient Requirements and Diet Nutrient Densities for Mature Cows

Post Partum - Early Lactation Through Breeding

Body Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	26.2	15.5	15.7	2.75	0.08	0.05
1200	27.6	16.2	16.3	2.82	0.08	0.06
1300	29.1	17.0	17.2	2.91	0.09	0.06
1400	30.4	17.6	17.6	3.01	0.09	0.06
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		59.2	0.60	10.5	0.30	0.20

Lactating & Pregnant - Late Lactation to Weaning

Body Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	25.1	13.8	13.3	2.18	0.06	0.04
1200	26.5	14.5	14.0	2.28	0.06	0.05
1300	28.0	15.2	14.8	2.35	0.07	0.05
1400	29.4	15.9	15.3	2.44	0.07	0.05
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		55.1	0.53	8.7	0.24	0.17

Gestation - Weaning to 60-90 Days Before Calving

Body Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	21.3	10.1	8.7	1.41	0.03	0.03
1200	22.8	10.8	9.3	1.50	0.04	0.03
1300	24.3	11.5	10.0	1.60	0.04	0.03
1400	25.6	12.1	10.5	1.69	0.04	0.03
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		47.4	0.41	6.6	0.17	0.13

Pre-Calving 60 - 90 Days Before Calving

Body Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	22.7	12.3	20.7	1.93	0.06	0.04
1200	24.4	13.2	22.2	2.07	0.06	0.04
1300	25.8	14.1	23.5	2.22	0.07	0.04
1400	27.3	14.9	25.1	2.35	0.07	0.04
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		54.6	0.92	8.6	0.26	0.16

Table 2.

Daily Nutrient Requirements and Diet Nutrient Densities for 1st Calf Heifers**Post Partum - Early Lactation Through Breeding**

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	22.4	13.6	13.9	2.35	0.07	0.04
1200	23.7	14.3	14.7	2.44	0.07	0.05
1300	25.0	15.0	15.5	2.53	0.08	0.05
1400	26.2	15.7	16.2	2.62	0.08	0.05
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		60.6	0.62	10.5	0.31	0.19

Lactating & Pregnant - Late Lactation to Weaning

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	22.1	12.6	12.4	1.97	0.06	0.04
1200	23.5	13.4	13.2	2.07	0.06	0.04
1300	24.8	14.1	13.9	2.16	0.06	0.04
1400	26.1	14.8	14.6	2.24	0.07	0.04
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		57.0	0.56	8.9	0.25	0.17

Gestation - Weaning to 60-90 Days Before Calving

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	20.5	10.4	9.4	1.48	0.04	0.03
1200	21.9	11.1	10.3	1.60	0.05	0.03
1300	23.3	11.8	11.0	1.70	0.05	0.03
1400	24.6	12.5	11.6	1.80	0.05	0.04
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		50.9	0.47	7.3	0.22	0.15

Pre-Calving 60-90 Days Before Calving

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	21.8	12.6	12.4	1.94	0.06	0.04
1200	23.3	13.5	13.5	2.10	0.07	0.04
1300	24.8	14.4	14.4	2.23	0.07	0.04
1400	26.2	15.3	15.2	2.36	0.08	0.05
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		58.3	0.58	9.0	0.30	0.18

Table 3.

Daily Nutrient Requirements and Diet Nutrient Densities for Pregnant Replacement Heifers

Early Gestation - Breeding through Preg Check (1 lb Gain/Day)

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	18.5	9.3	8.5	1.33	0.041	0.032
1200	19.8	10.0	9.1	1.42	0.046	0.036
1300	21.0	10.6	9.8	1.52	0.049	0.038
1400	22.3	11.3	10.5	1.61	0.054	0.040
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		50.8	0.47	7.2	0.24	0.18

Mid Gestation(1-1.25 Lb. Gain/Day)

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	20.1	10.4	9.7	1.48	0.044	0.034
1200	21.5	11.1	10.3	1.58	0.047	0.037
1300	22.9	11.8	11.0	1.68	0.050	0.041
1400	24.2	12.5	11.6	1.78	0.054	0.044
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		51.7	0.48	7.4	0.22	0.18

Late Gestation - Pre-Calving (1.5-2.25 Lb. Gain/Day)

Estimated Mature Weight	Dry Matter Intake, (lb)	Lb Per Animal Per Day				
		TDN	NEm	CP	Ca	P
1100	22.2	12.7	12.6	1.95	0.069	0.050
1200	23.7	13.4	13.2	2.05	0.073	0.053
1300	25.2	14.2	13.9	2.17	0.076	0.056
1400	26.6	15.0	14.7	2.27	0.081	0.059
Required Diet		% TDN	NEm	% CP	% Ca	% P
Nutrient Density		56.2	0.55	8.0	0.30	0.22

Table 4.**Daily Nutrient Requirements and Diet Nutrient Densities for Breeding Bulls****1700 Lb Mature Weight Bull Gaining 1.5 Lbs/Day**

Dry		Lb Per Animal Per Day					
Current Weight	Matter Intake, (lb)	TDN	NEm	NEg	CP	Ca	P
1300	30.7	18.4	10.6	4.6	1.85	.057	.037
1400	32.4	19.4	11.2	4.9	1.88	.057	.039
1500	34.1	20.5	11.8	5.1	1.92	.058	.040
1600	35.8	21.5	12.4	5.4	1.95	.059	.041
Required Diet		% TDN	NEm	NEg	% CP	% Ca	%P
Nutrient Density		60.0	0.61	0.35	6.0	.19	.12

1700 Lb Mature Weight Bull Gaining 0-0.5 Lbs/Day

Dry		Lb Per Animal Per Day					
Current Weight	Matter Intake, (lb)	TDN	NEm	NEg	CP	Ca	P
1600	33.9	17.0	12.4	1.36	1.84	.054	.039
1700	35.8	17.8	13.0	1.42	1.91	.052	.040
Required Diet		% TDN	NEm	NEg	% CP	% Ca	%P
Nutrient Density		50.0	.45	.20	5.5	.16	.12

2000 Lb Mature Weight Bull Gaining 1.7 Lbs/Day

Dry		Lb Per Animal Per Day					
Current Weight	Matter Intake, (lb)	TDN	NEm	NEg	CP	Ca	P
1500	34.1	20.5	11.8	5.1	2.10	.066	.043
1600	35.8	21.5	12.4	5.4	2.10	.066	.044
1700	37.5	22.5	13.0	5.6	2.15	.066	.046
1800	39.1	23.5	13.5	5.9	2.18	.067	.047
1900	40.8	24.5	14.0	6.1	2.2	.068	.047
Required Diet		% TDN	NEm	NEg	% CP	% Ca	%P
Nutrient Density		60.0	0.61	0.35	6.0	.19	.12

2000 Lb Mature Weight Bull Gaining 0-0.5 Lbs/Day

Dry		Lb Per Animal Per Day					
Current Weight	Matter Intake, (lb)	TDN	NEm	NEg	CP	Ca	P
1900	36.6	19.3	14.1	1.54	2.10	.063	.047
2000	37.2	20.1	14.6	1.60	2.10	.063	.047
Required Diet		% TDN	NEm	NEg	% CP	% Ca	%P
Nutrient Density		50.0	0.46	0.20	5.5	.16	.12

Body Condition Scoring Your Beef Cow Herd

Resource: Karla Wilke, Travis Mulliniks and Kacie McCarthy, University of Nebraska-Lincoln.

Body condition scores (BCS) describe the relative fatness or body condition of a cow herd through the use of a nine-point scale. A body condition score five (BCS 5) cow is in average flesh and represents a logical target for most cow herds. A BCS 1 cow is extremely thin while a BCS 9 cow is extremely fat and obese.

Impact on subsequent rebreeding performance

Body condition score (BCS) of beef cows at the time of calving has the greatest impact on subsequent rebreeding performance (Table 1). The postpartum interval is the length of time from calving to first estrus (heat) after calving.

For a cow to maintain a 365 day calving interval, she must rebreed by 82 days after calving (283 day gestation + 82 day postpartum interval = 365 days). On the average, cows that calve in a BCS 3 or 4 have difficulty exhibiting their first heat by 80 days after calving. Whereas cows that calve in BCS 5 or 6 tend to exhibit heat by 55 days after calving and; therefore, have a better opportunity to maintain a 365 day calving interval. Although cows that calve in a BCS of 7 have a short postpartum interval, it is not economical to feed cows to a condition score of 7.

Thin cows at calving (BCS 4 or thinner) produce less colostrum, give birth to less vigorous calves that are slower to stand and these calves have lower immunoglobulin levels (Table 2, below), thus impairing their ability to overcome early calf-hood disease challenges. This illustrates the importance of targeting mature cows to calve in a BCS of at least 5. Because 1st-calf-heifers have only reached about 85% of their mature weight after calving and require additional nutrients to support growth, they need to be fed so they are a BCS of 6 at calving.

BODY CONDITION RELATES TO INTERVAL FROM CALVING TO FIRST HEAT		
BCS	Post-Partum Interval (Days)	
3	89	80
4	70	
5	59	55
6	52	
7	31	

Table 1. Body Condition Score Pre-calving and impact on days from calving to estrus (post-partum interval, PPI). Houghton et.al., 1990. Effects of body composition, pre- and postpartum energy level and early weaning on reproductive performance of beef cows and preweaning calf gain.

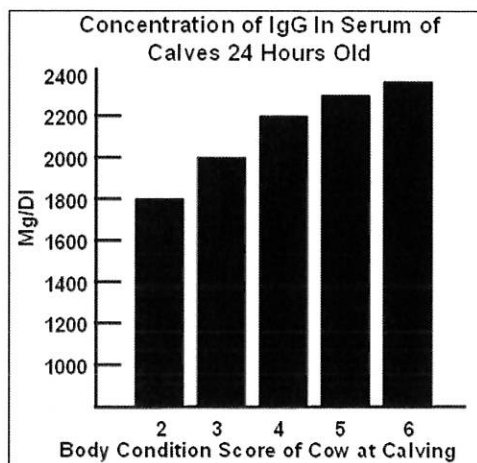
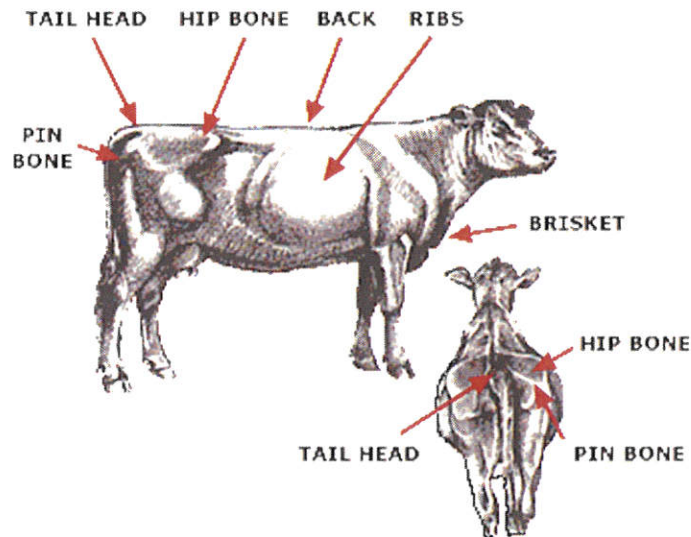


Table 2. IgG in Beef Calves 24 hours after birth whose dams are in different body condition. Odde, K.G. 1997. Reproductive efficiency, precalving nutrition, and improving calf survival. Proc. Bovine Connection, pg. 860-092.

The Look and Feel of Body Condition Scoring

Body condition scoring can be done using only visual indicators or a combination of visual and palpation of key bone structures for fat cover. Palpation can be done during routine processing of cows through a chute. The key areas for evaluation are the backbone, ribs, hips, pinbones, tailhead, and brisket. Palpating cows for fatness along the backbone, ribs, and tailhead will help refine your skill to visually assess body condition.



If body condition scoring is new to you, just focus on separating cows into thin, moderate, and fat groups without worrying about the numerical score. With experience, you will connect the "look and feel" of your cows to a body condition score that you can consistently determine.

Body condition scores should be recorded so that links to productivity and herd management (particularly nutritional management) can be examined. Several years of such information could reveal, for example, needed management changes for a given age group (i.e., thin three-year-olds) of cows or might identify a sire group of females that simply didn't fit your resources.

When visually scoring body condition, you must "look through the hair coat". Sometimes this is difficult due to a long winter hair coat. It is good training to re-evaluate your body condition scores when cattle are wet. You may be surprised at the impact hair coats can have on visual scores. Long, thick winter hair coats are obviously highly desirable (at least in the Northern plains), thus actual palpation for fatness of cows may be the best choice to produce consistent body condition scoring. Drawings of cows in BCS 1 to 9 can give an indication of how these cows would look if they were without hair.

Other factors in addition to hair coat that can affect visual body condition scores are age of cow, rumen fill, and stage of pregnancy. The goal of body condition scoring is to evaluate fatness independent of these factors. At first, one or more of the above factors may mislead you, but careful study of your herd through the production year will sharpen your focus so that body condition can be scored independent of the above factors.

The same techniques are used to condition score cattle that have Bos Indicus genetics. Depending on the percentage of Bos Indicus genetics, the skin appears to be wrinkled or folded. Determine degree of condition at the same locations and assign a score based on the 1 to 9 scale.

When to Condition Score Cows

The greatest single factor influencing rebreeding performance of beef cows is body condition at calving, especially for spring-calving females. However, if producers wait until calving to manage body condition of their cows, they will find it very difficult and expensive to increase the body condition of a lactating cow.

Although evaluation of body condition can be looked at as an ongoing process, there are several key times when body condition scoring should be considered:

Late Summer Early Fall

This is an important time to condition score cows in drought years or in systems where females are managed almost entirely on vegetative or dormant grazed forage. If cows are thin, early weaning should be considered. Non-lactating cows may pick-up condition by grazing forage alone or by feeding a small amount of supplement along with the grazed forage. If young cows are thin and grass in the pasture is decreasing in nutrient quality, strategically wean calves.

Weaning Time

Pay particular attention to young cows weaning their first calves, as they are most likely to be thin at this time. For young cows, you may need to consider early weaning calves and giving cows access to higher quality forage.

45 Days after Weaning

This will give you a good idea how fast cows are "bouncing back" after weaning. Thin cows should be gaining back condition if cow type is matched with the feed resources.

90 Days before Calving

This is the last opportunity to get condition back on cows economically. This would be the time to separate thin cows from cows in good condition.

Calving Time

If cows are thin, producers may want to change the pre-calving feeding program. Because of the nutritional demands of lactation, it is difficult to get cows to condition economically after calving.

Beginning of Breeding Season

Thin cows at this time may indicate a poor match of calving season to feed sources. Maybe calving occurs too early in the spring. The period from weaning to 90 days pre-calving is the best time to get serious about body condition scoring and planning the nutrition/management program because the manager's strategy can have great impact. The period from calving to re-breeding may help explain the productivity (or lack thereof!) but it is likely too late to have much impact on herd productivity and profitability at this point. If cows are thin management options include early weaning when the youngest calf is 45 days old or 48 hour calf separation. Both of these management techniques will help initiate estrous cycles in beef cows.

Nine Point Body Condition Scoring for Beef Cow Herd

Resource: Karla Wilke, Travis Mulliniks and Kacie McCarthy, University of Nebraska-Lincoln

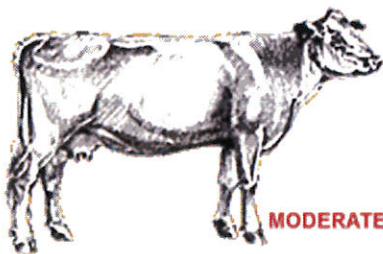
Nine Point Body Condition Scoring



1. Bone structure of shoulder, ribs, back, hooks and pins are sharp to the touch and easily visible. No evidence of fat deposits or muscling. Cattle in this condition are weak, near death and have trouble standing or walking.

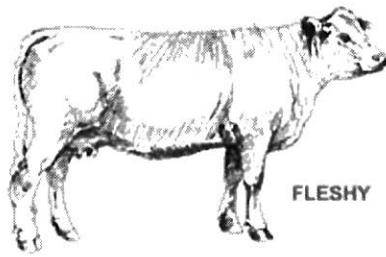
2. No evidence of fat deposition and muscle loss in the hindquarters. The spinous processes feel sharp to the touch. The spinous processes and the spaces between them are easily seen.

3. Very little fat cover over the loin, back and fore-ribs. The backbone is still highly visible. Processes of the spine can be identified individually by touch and may still be visible. Spaces between the processes are less pronounced.



4. Fore-ribs and 12th and 13th ribs are still noticeable to the eye. The transverse spinous processes can be identified only by palpation (with slight pressure) and feel rounded rather than sharp.

5. The 12th and 13th ribs are slightly visible to the eye. The transverse spinous processes can only be felt with firm pressure and feel rounded but are not noticeable to the eye. Spaces between the processes are not visible and are only distinguishable with firm pressure. Areas on each side of the tailhead and the fore-rib, behind the shoulder are starting to fill.



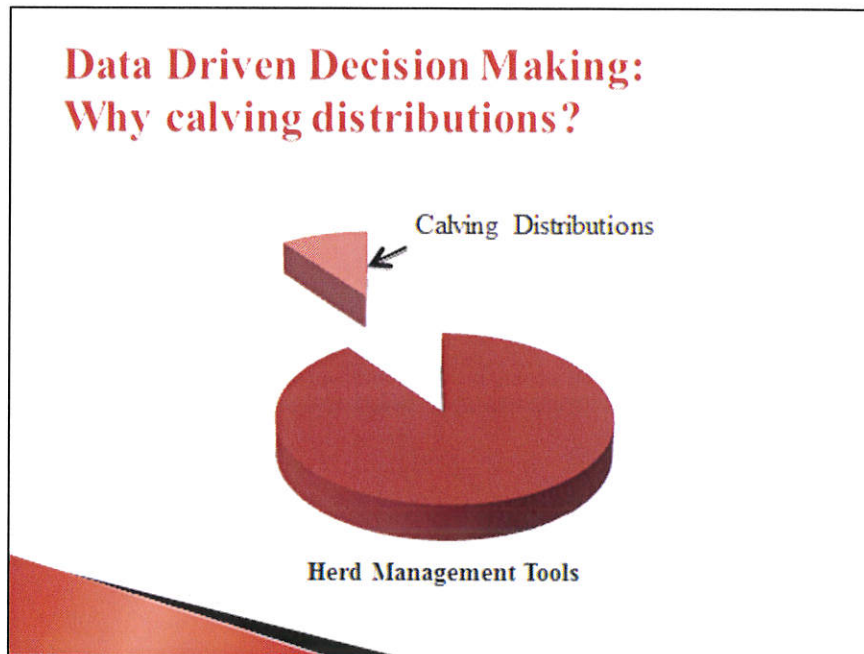
6. Ribs are fully covered and are not noticeable to the eye. Hindquarters are plump and full. Noticeable springiness over the fore-ribs and on each side of the tailhead. Firm pressure is now required to feel the transverse processes. Brisket has some fat.

7. Ends of the spinous processes can only be felt with very firm pressure. Spaces between processes can barely be distinguished. Abundant fat cover on either side of the tailhead with evident patchiness. Fat in the brisket.

8. Animal takes on a smooth, blocky appearance. Bone structure disappears from sight. Fat cover is thick and spongy and patchiness is likely. Brisket is full.

9. Bone structure is not seen or easily felt. The tailhead is buried in fat. The animal's mobility may actually be impaired by excessive fat.

Calving Earlier In the Calving Season: Using calving distributions as a component of production management

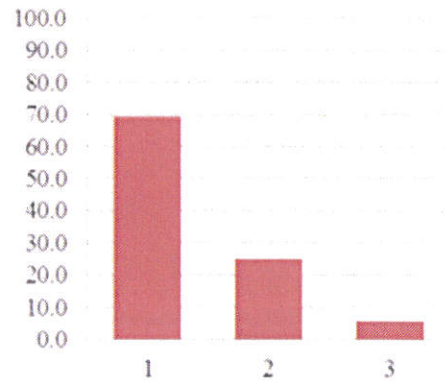


Calving distributions are simple to construct and provide useful information for management decisions. That said, a distribution is only a very small piece of the management pie, and other components, such as pregnancy, calving, and weaning percentages, or pounds weaned per calf exposed offer even more insight. The end goal is to increase reproductive efficiency and herd productivity. Veterinarians should play a primary role in achieving this goal, and data analysis and interpretation is a means to increase involvement and impact.

An advantage for the veterinarian in constructing and discussing calving distributions with clients is that it may offer a gateway for veterinarians to become more involved in management practices. Ideally, this could enable a veterinarian to create a “team” approach for the producer and increase the relevance of a veterinarian to the operation. Capturing this type of data can provide a producer with a clearer understanding of his/her economic position, allow for goal assessment or modification, and provide a means to measure improvement in efficiency and overall operation health.

Calving Distributions

- ▶ 21 day periods during the calving season
- ▶ Start date: 3rd term calf
- ▶ Minimum:
 - #born for each period
- ▶ Better:
 - Cow ID
 - Cow age
 - Calf birth date
 - Calf ID



Adapted from BIF Guidelines, 2002

The construction of a calving distribution: 1st day of the calving season begins with the 3rd calf (term) born. This is the standard used in the current study. Similarly the Standard Production Analysis (SPA) guidelines could also be used: 1. The first 21-day period starts when the third mature cow (3-years-old or older) has calved, or; 2. The first 21-day calving period starts 285 days after the start of the breeding season.

The most basic form is simply counting the number of calves per 21 day period (See form on page 10). To get more repeatable data, and to monitor progress for individual herds, inclusion of cow age and ID, actual calf birth date, and calf ID are needed.

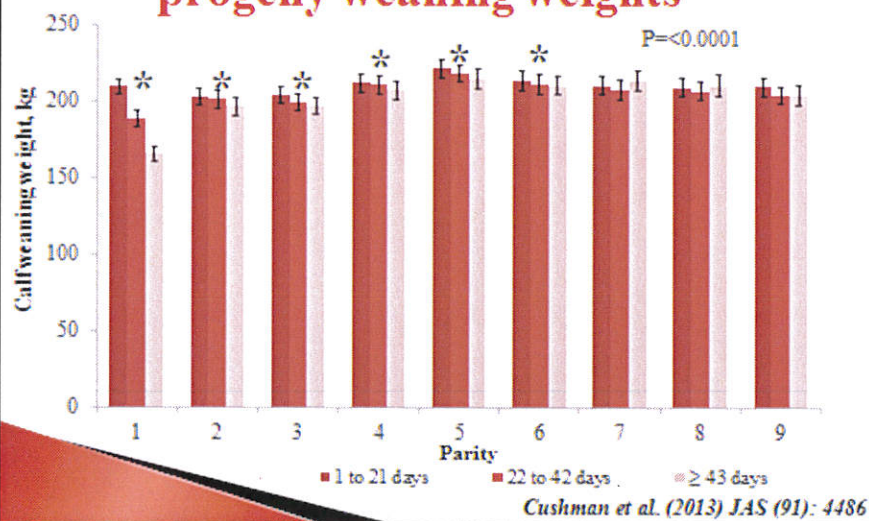
Calving Earlier in the Calving Season: Effects on performance, productivity, and longevity

Weaning Weight (lb) Differences

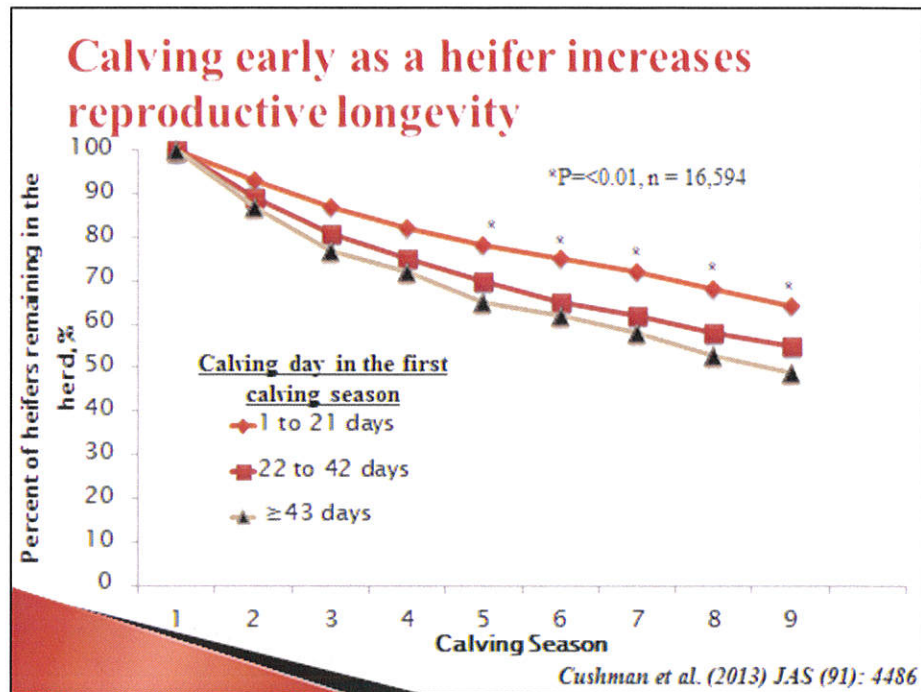
	Calving Period 1	Calving Period 2	Calving Period 3
Lesmeister et. al, 1973	0	-46	-71
Funston et. al, 2012 (steer calves)	0	-28.7	-74.7
Cushman et. al, 2013	0	-45.64	-97.02
NDSU Dickinson Research Center, 2002	0	-40	-88

**First calf heifer progeny*

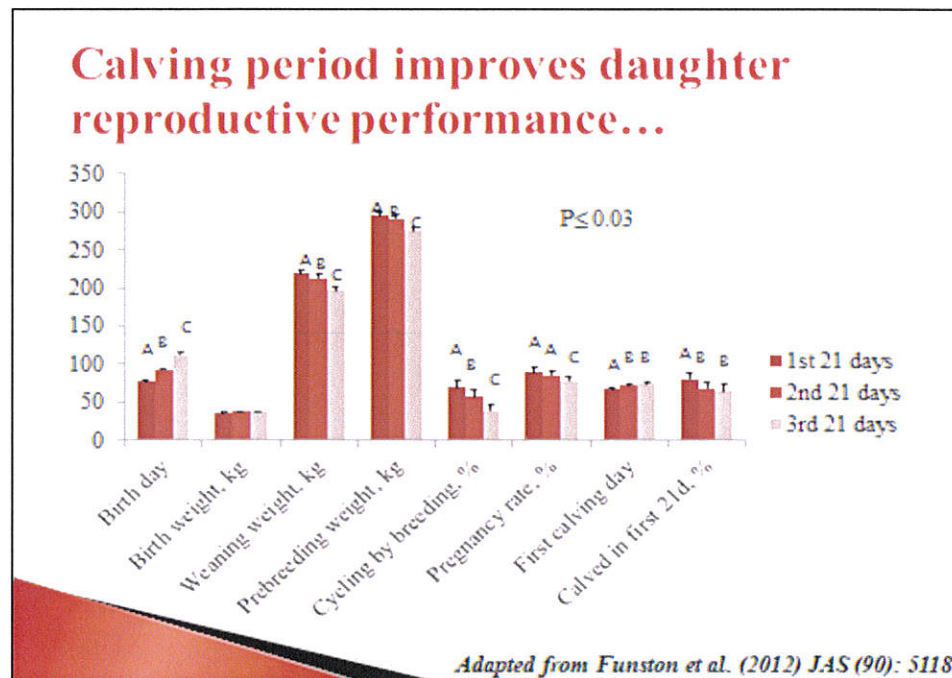
Calving early as a heifer increases progeny weaning weights



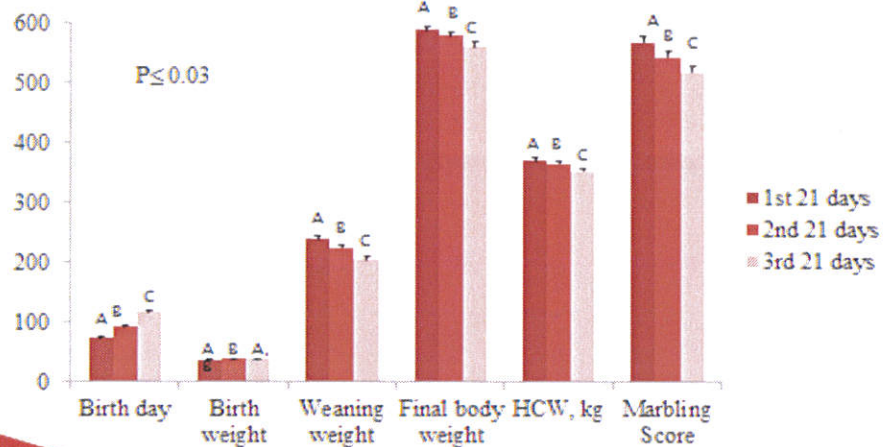
In addition to a longer lifespan, those cows calving in the first 21 days as a heifer weaned heavier calves through the 6th calving season. This difference equates to approximately 1-2 extra calves over her lifespan. Thus the effect of calving early is two-fold: longer productive lifespan and more beef per calving season. This effect was first documented in the 1970's by Lesmeister and is still true today. Weaning weights are a function of both management and genetics.



There were no differences in birth weight between the three groups. Birth dates of heifers in the first 21 days were 2 and 3 days older than heifers calving in the second and third 21 days ($P < 0.05$).

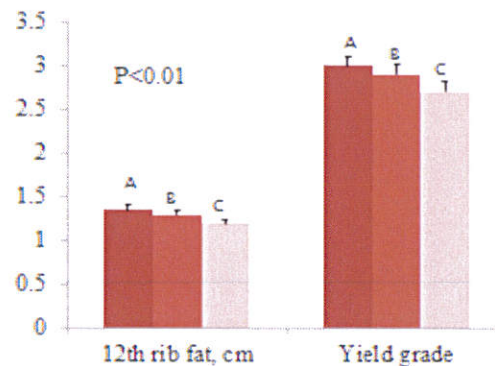
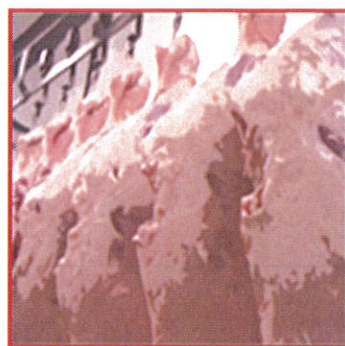


...and son performance on the farm, the feedlot and at the packing plant



Adapted from Funston et al. (2012) JAS (90): 5118

...and son performance on the farm, the feedlot and at the packing plant



Adapted from Funston et al. (2012) JAS (90): 5118

This study demonstrated a difference in carcass value from calving period 1 to calving period 2 of \$24.00, and a difference from period 1 to 3 of \$75.00

Calving Distribution Collection Form

The following information is requested to compile a simple distribution of calf births through the calving season to better evaluate and potentially improve reproductive efficiency.

Cows

- 1 Total number calves born in calving season
- 2 Enter date the third cow in the herd calved
 - a. Date must be within 10 days of bull turnout date plus 283 days.
- 3 Enter the number of calves born the first 21 days of calving season beginning with the date in 2) above. Include the 2 calves born prior to the date in 2) above
- 4 Enter the number of calves born the second 21 days.
- 5 Enter the number of calves born the third 21 days.
- 6 Enter the number of calves born the fourth 21 days.
- 7 Enter the number of calves born the fifth 21 days.
- 8 Enter the number of calves born the sixth 21 days.
- 9 Enter the remainder of calves born.

Heifers

- 1 Total number calves born in calving season
- 2 Enter date the third cow in the herd calved
 - a. Date must be within 10 days of bull turnout date plus 283 days.
- 3 Enter the number of calves born the first 21 days of calving season beginning with the date in 2) above. Include the 2 calves born prior to the date in 2) above
- 4 Enter the number of calves born the second 21 days.
- 5 Enter the number of calves born the third 21 days.
- 6 Enter the number of calves born the fourth 21 days.
- 7 Enter the number of calves born the fifth 21 days.
- 8 Enter the number of calves born the sixth 21 days.
- 9 Enter the remainder of calves born.

Whole Herd

- 1 Total number calves born in calving season
- 2 Enter date the third cow in the herd calved
 - a. Date must be within 10 days of bull turnout date plus 283 days.
- 3 Enter the number of calves born the first 21 days of calving season beginning with the date in 2) above. Include the 2 calves born prior to the date in 2) above
- 4 Enter the number of calves born the second 21 days.
- 5 Enter the number of calves born the third 21 days.
- 6 Enter the number of calves born the fourth 21 days.
- 7 Enter the number of calves born the fifth 21 days.
- 8 Enter the number of calves born the sixth 21 days.
- 9 Enter the remainder of calves born.

Mature Cows

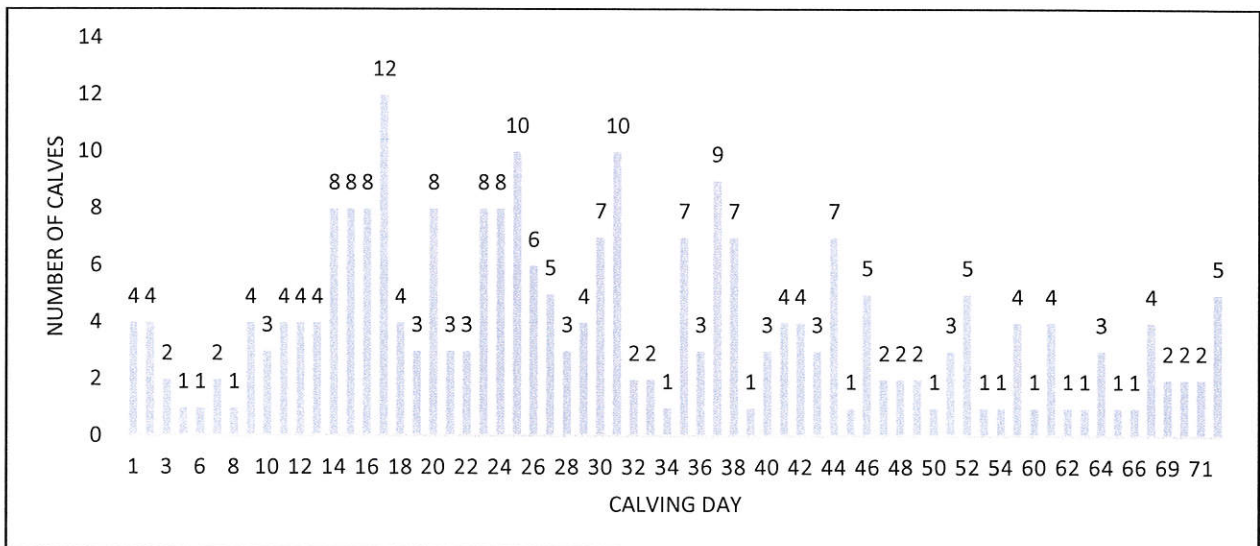
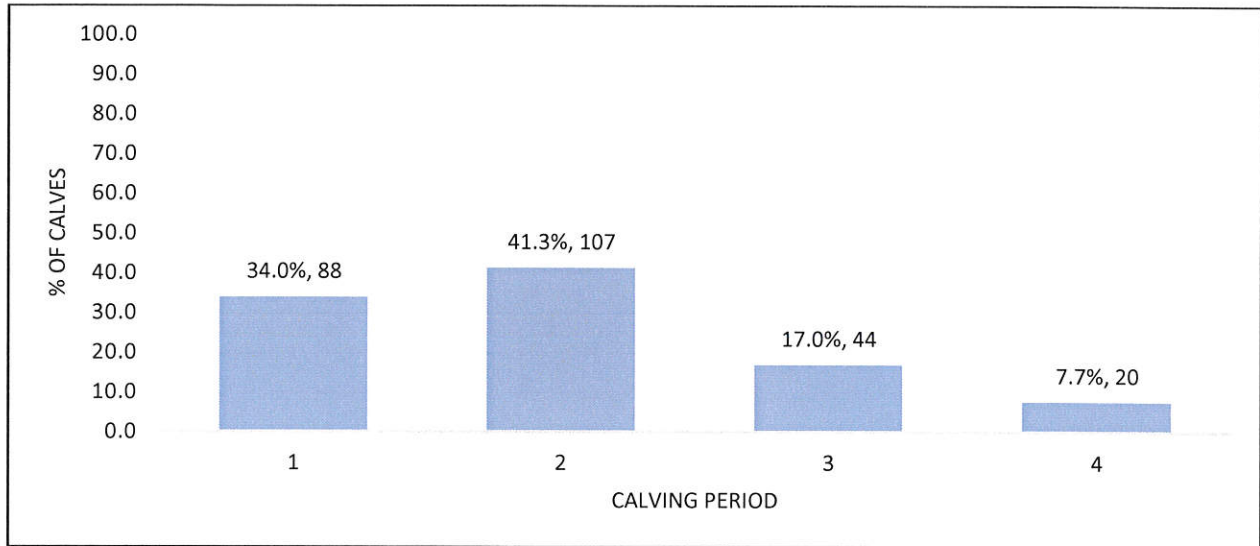
Average Calving Day: 31.3 +/- 1.1

Median Calving Day: 28

Start of Calving (Day 1): 2/17/2014

End of Calving (Day 73): 5/1/2014

Total Number of Calves: 259



Time Investment: Input

Non-computerized= 25 herds (62.5%)

	Paper	Redbook	Form	Total	Range
Herds	4	12	9	25	
Total Head	436	1803	3002	5241	15-761
Time/hd (Seconds)	25	26.5	2.7	18.6 Hours	1.2-54.6
Cost/hd (\$15/hr)	\$0.11	\$0.11	\$0.01		
Cost/herd	\$11.38	\$16.65	\$3.75		\$2.50- 37.50
Total cost/herd (Input+Analysis)	\$20.75	\$28.83	\$15.83		\$10.00- \$52.50

Analysis of collection data included format of data submitted, time it took for data to be received, time spent entering data and creating reports, and costs. Once data was converted into Excel, there was little variation in report generation time. Overall, it took 83 days from first to final submission, and two-thirds of the data (25/40 herds) came in a paper format, primarily copies of IRM Redbook pages.

On average, it took about 25 seconds per head for data entry and report generation. The average time per herd was 1.5 hours (35 minutes-3.5 hours). The costs of input for non-computerized formats are summarized above. Note that even though the fillable form was the quickest and least expensive, the type of data generated is more limited and has less value year over year.

Summary Statistics

	Tally/Average	Range
Participating Herds	40	
Total Study Population	8696	
2 Year Olds	1560	
3 Year Olds	1242	
Matures	5894	
Average Herd Size*	217	15-761
Herd Size ≥ 100	30	104-761
Herd Size < 100	10	15-84
Average Calving Season (days)	86	18-154
Herd Size ≥ 100	91	53-154
Herd Size < 100	71	18-125
Heifers calve before/after/with cows (herds)	16/5/19	

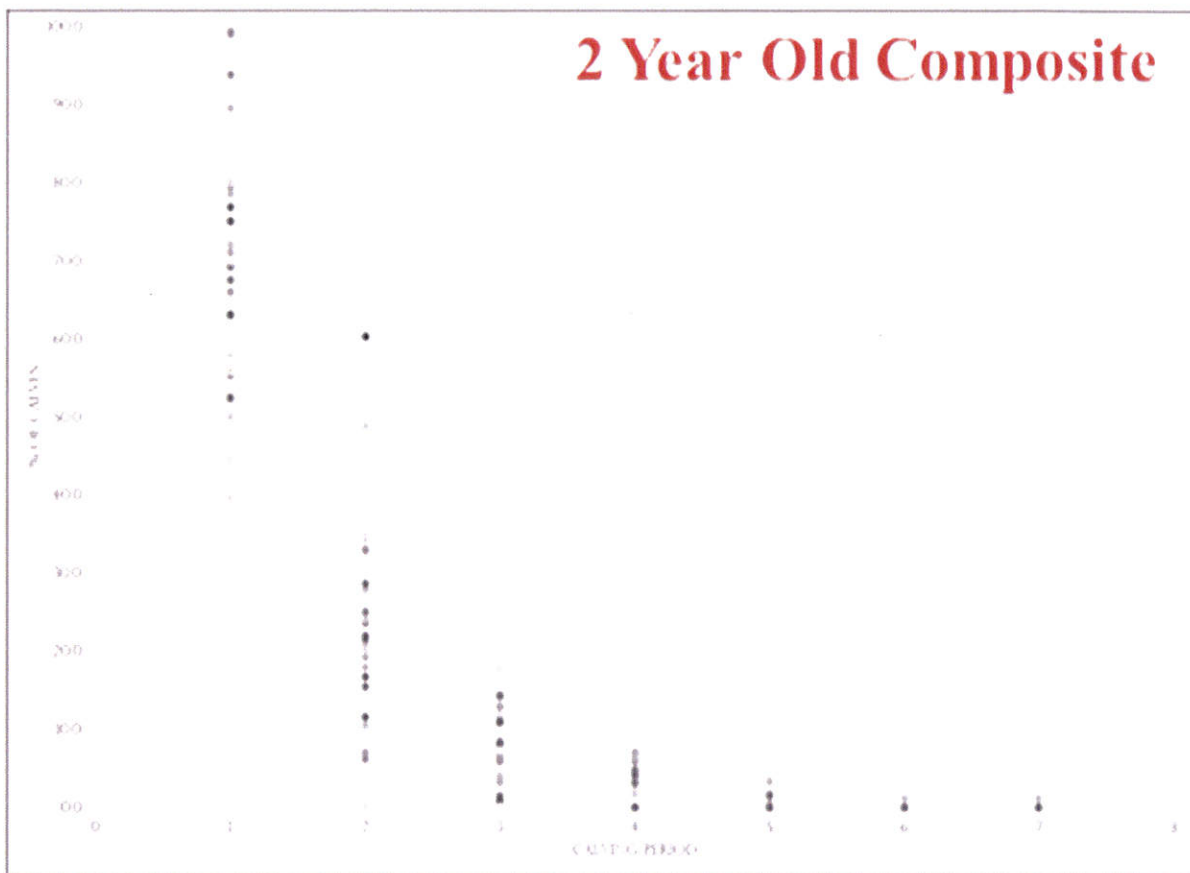
*Average herd size in Nebraska: 94 (www.nebeef.org, 2014)

Participating clinics were from various regions of Nebraska (western, central, north central, south central), one from North Dakota, and one from South Dakota. Clinics submitted from 1 to 10 herds. All cattle were included in the above summary statistics; individual statistical analysis was dependent upon available data. Mean and median calving day could not be constructed for herds grouped in 21 day periods alone; if ages were not known, all females were considered mature cows.

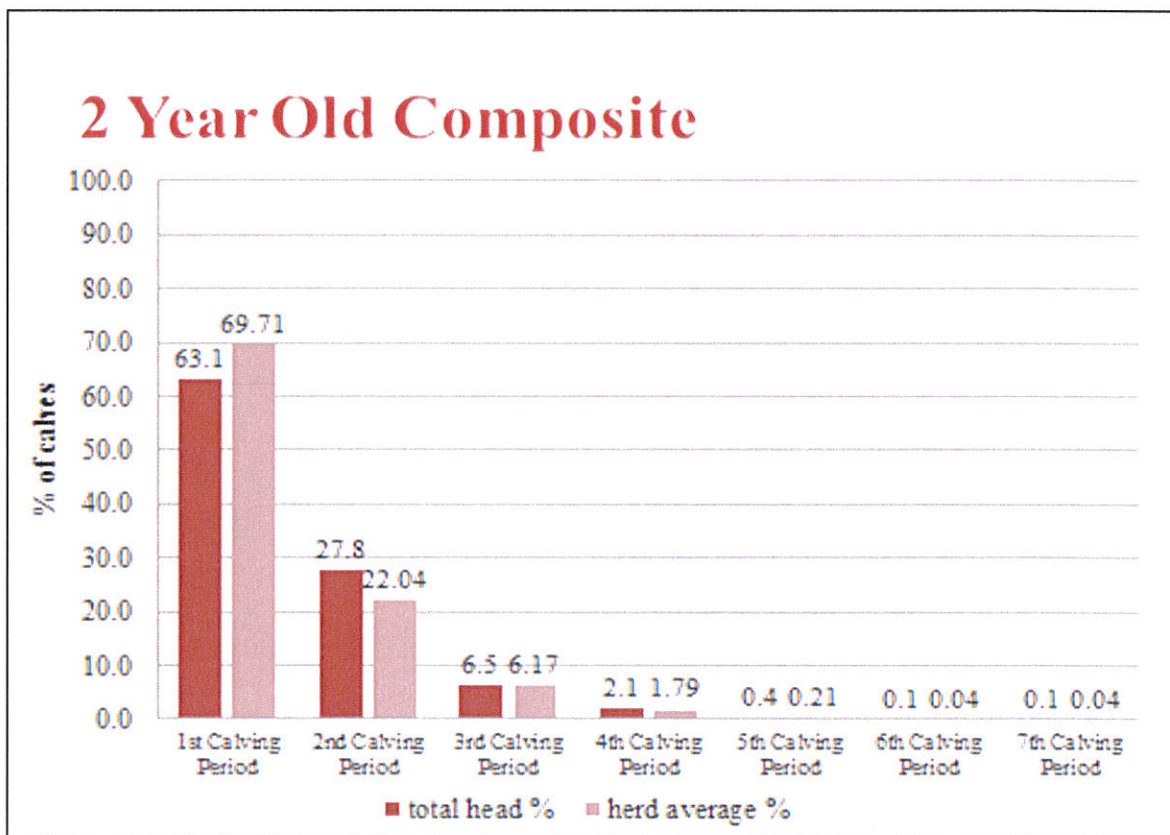
The following charts are the cumulative results from the study. It is important to remember that this is the data as is; synchronization, herd location, management strategies, etc., are not accounted for in the analysis. Dot plot charts give an illustration of herd groupings within age categories, and averages with ranges are listed below the charts, as well as in the study summary sheet.

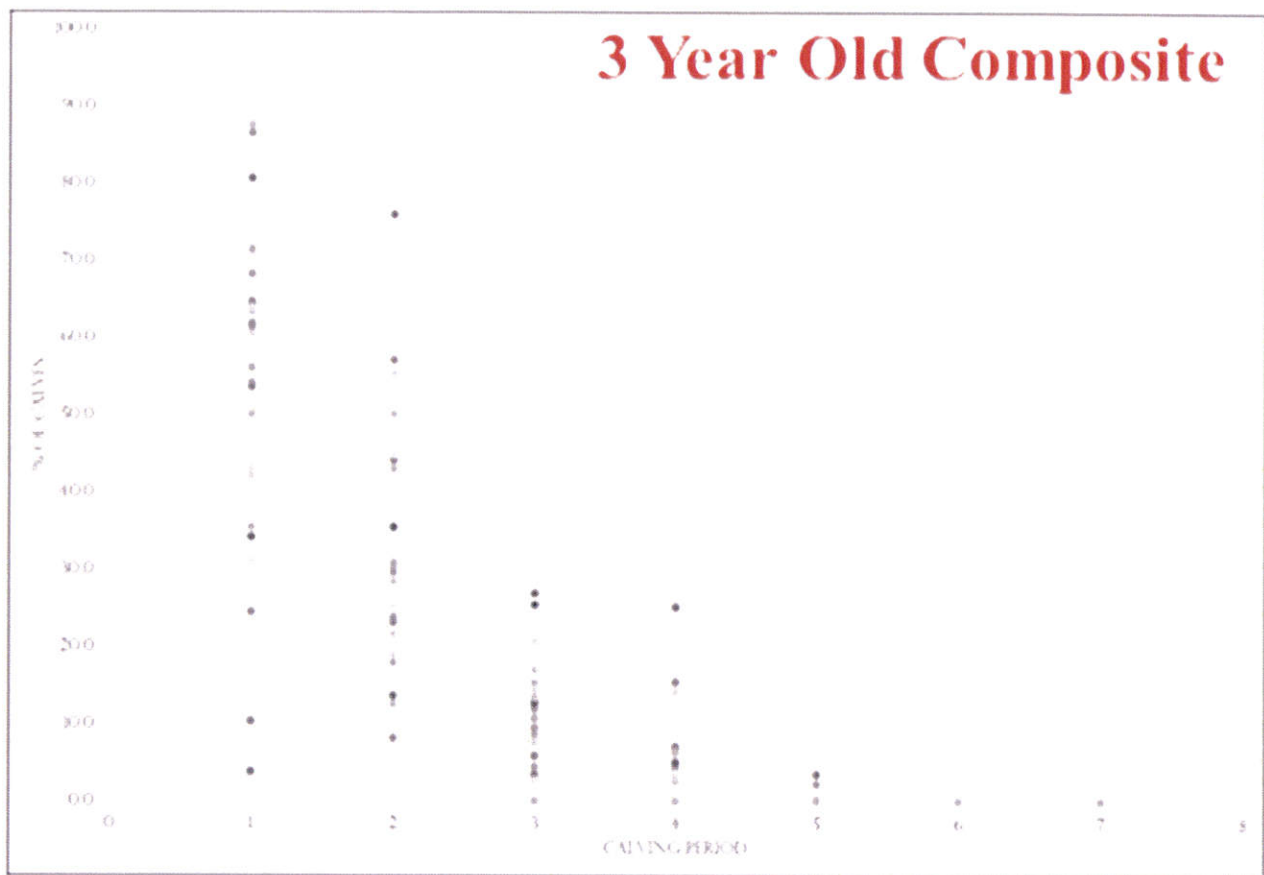
The cumulative composite consists of the distribution of the entire study population (dark bar) and the average of the participating herds (light bars). The average is calculated in an attempt to remove herd size as an influence. Caution must be taken when using these values for benchmarks. In herd evaluations, individual goals and year-over-year herd statistics are more precise guidelines to determine production efficiency.

**Percent of calves is listed on the y axis, and calving period is listed on the x axis.*

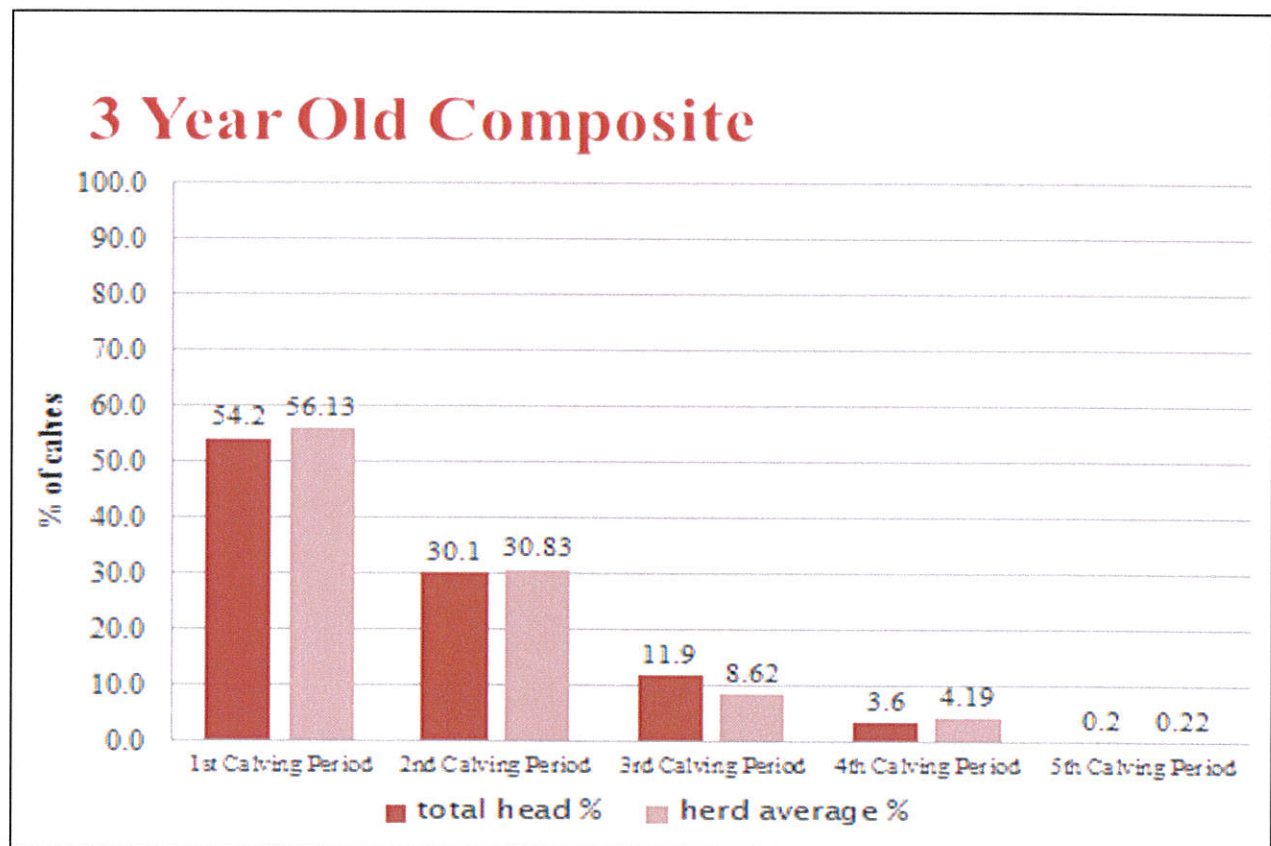


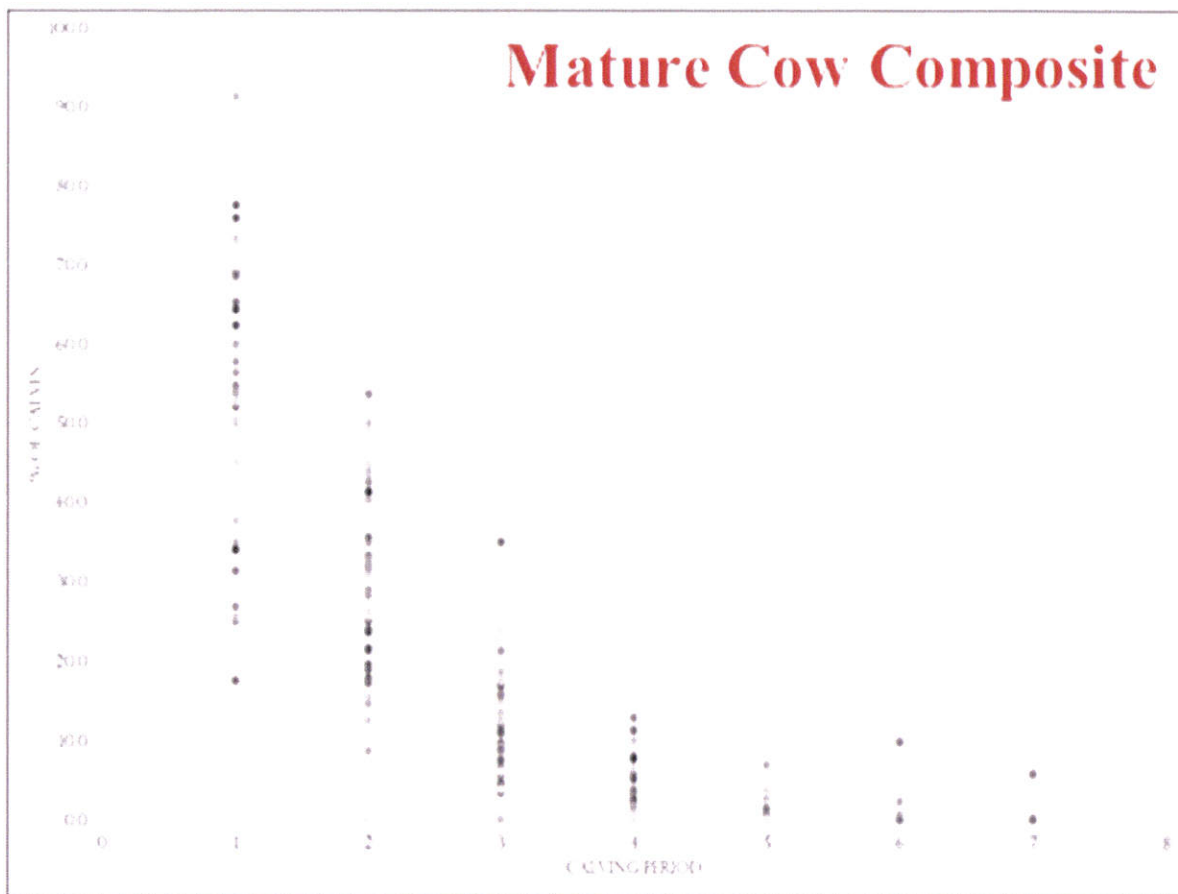
CP 1: Range of population 39-100%; Top 25% Range 79.4-100%, Average 92.84%



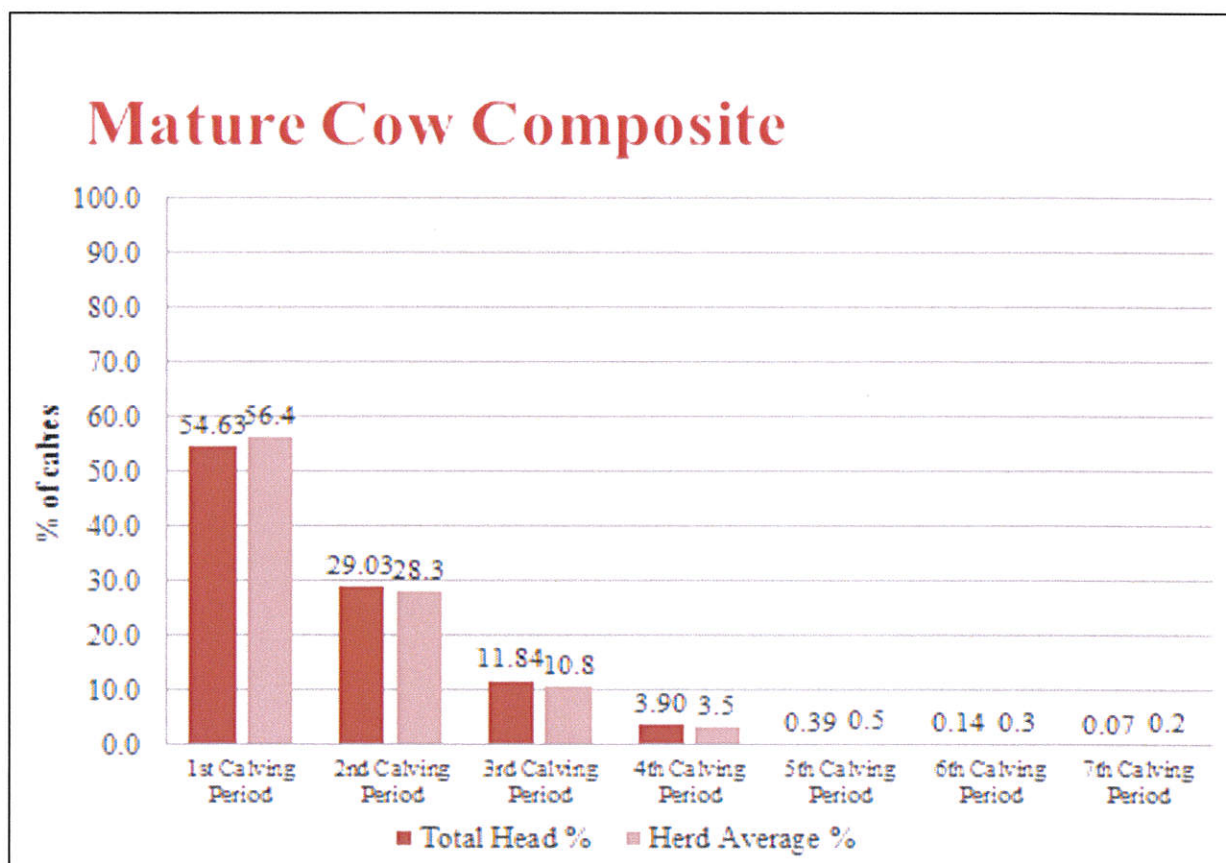


CP 1: Range of population 3.8-93.1%; Top 25% Range 70.6-93.1%, Average 80.31%





CP 1: Range of population 13.9-100%; Top 25% Range 69.1-100%, Average 79.9%



Economic Implications

Illustrations on pages 18-20 demonstrate the economic impact that calving earlier in the calving season may have on an individual producer, as well as the study population at hand. Keep in mind that this is an oversimplified view, but it does help to emphasize the importance and potential impact of this concept.

For this analysis, the following assumptions were made:

- Individual herd-470 head; overall population 8,696 head
- All calves born are weaned
- Weaning dates are the same
- No differences in birth weights between 2yo and 3yo; cow +5
- No differences in birth weights between CPs
- ADG increases between age groups
 - 2.0-2.2-2.4

The first two slides show the distribution and weaning weights of the individual operation; the next two slides show the “missed opportunity” of the operation if the distribution were adjusted to a reasonable target. The final page shows the difference in distributions and total weaned pounds of the study population from the current distribution to one that comes in line with the top 25% of herds in the study.

Study Summary

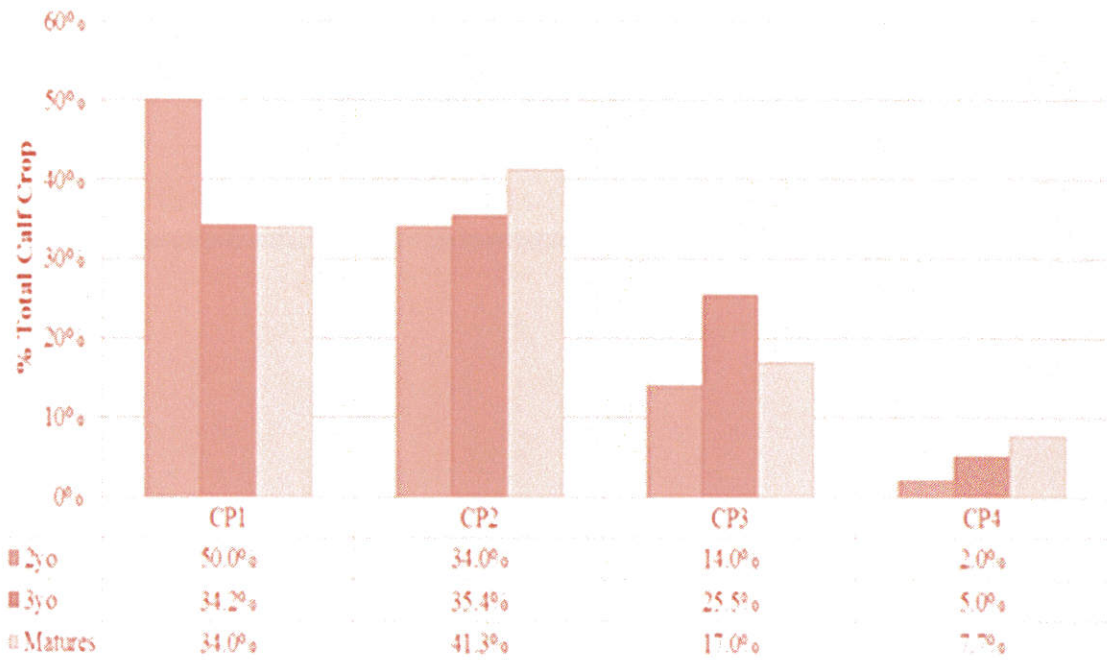
Pilot Study

It was clear that data collection from producers and veterinarians had challenges. Participating veterinarians expressed their concerns with ease of collection, contact with producers, confidentiality, and overall relevance of the data collected. Resistance to computerized forms of collection was evident, although only from the veterinarian’s perspective. Other limitations may include available facilities, weather, labor, feed resources, marketing goals, and above all else, tradition. Working through these producer concerns, as well as increased time and/or monetary investment from the clinics, is all necessary to sustain the data collection and analysis.

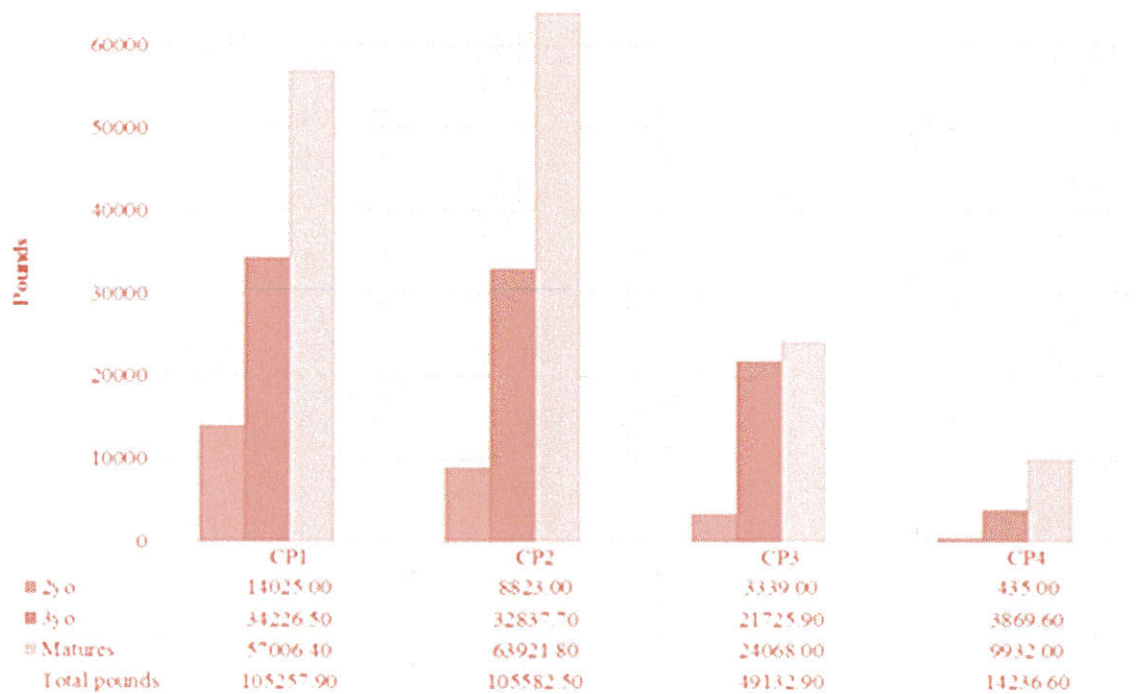
Distribution Data

While limited in terms of power, the study showed that there was a wide variation in not only distributions, but management practices. No doubt, for many herds there is a great opportunity to improve reproductive efficiency by simply measuring and evaluating calving data, and then implementing changes to primarily increase weaning weight. While this is the most immediate impact, the effects on cow productivity and longevity cannot be overlooked, and may be vastly more important.

Initial calving distribution-Individual herd

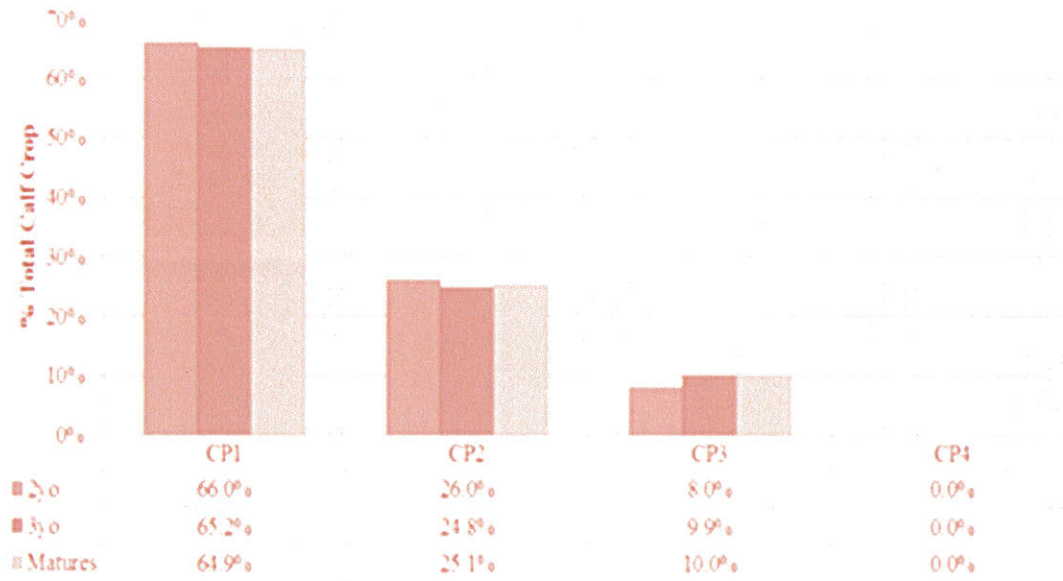


Initial pounds weaned



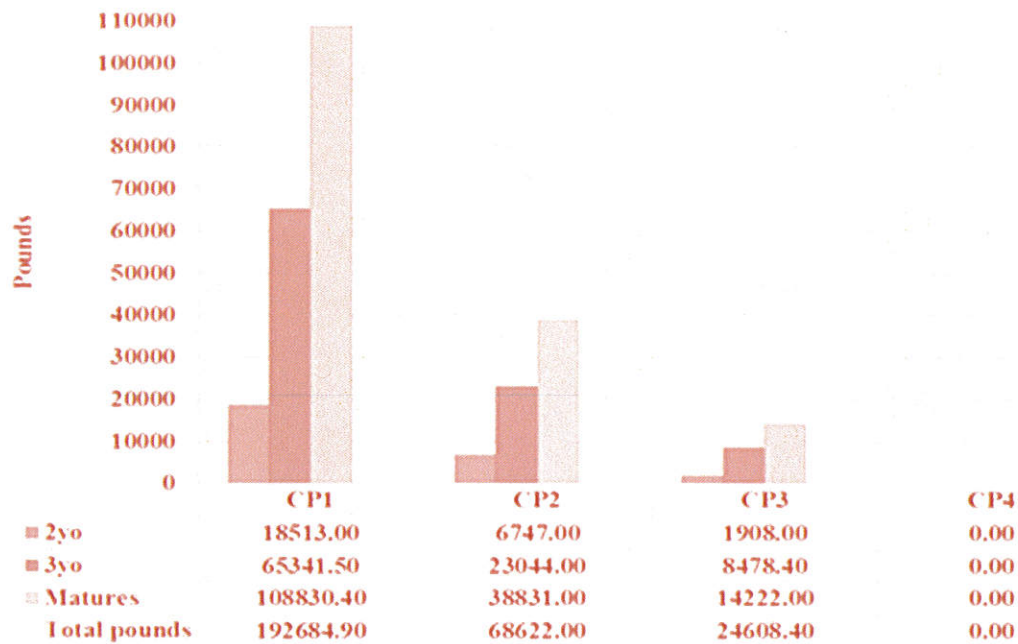
Missed Opportunity

Goal Calving Distribution



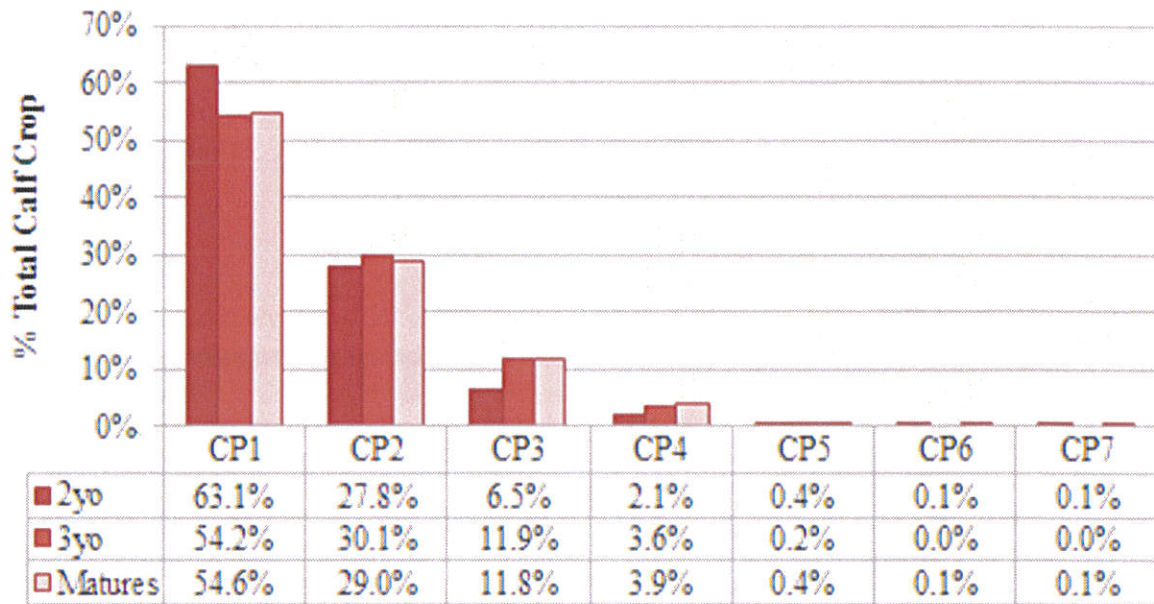
Missed Opportunity

Goal Total Pounds Weaned



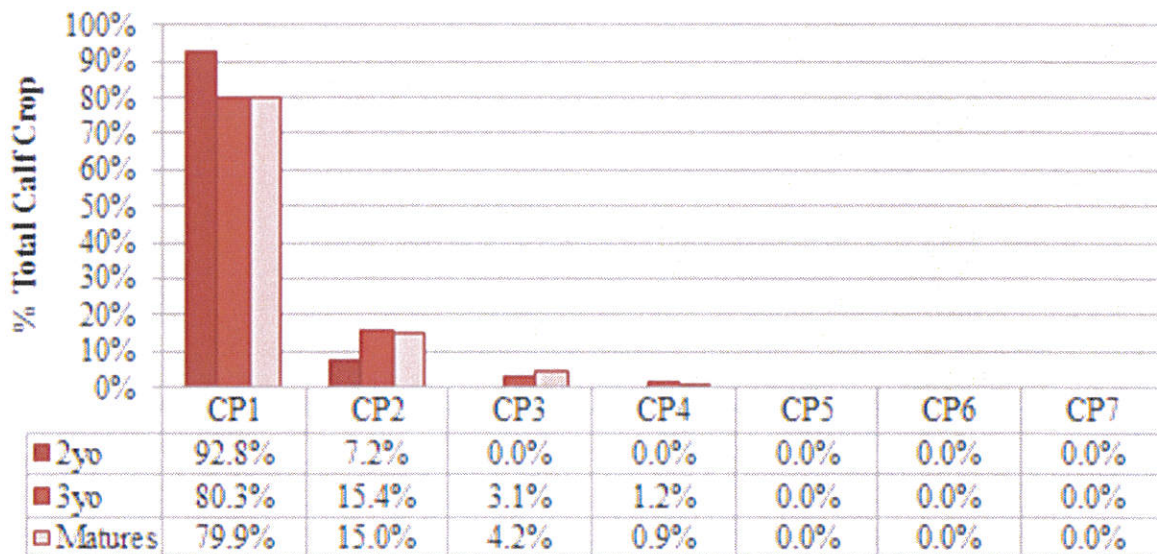
The difference in total pounds weaned for this operation was **11,705.40**. While there is certain price variation, it is clear that there is a certain economic advantage for this producer to utilize technologies to increase the number of females calving at the beginning of his/her calving season

Current Calving Distribution



Total Pounds Weaned: 5,145,902.20

Top 25% Calving Distribution



Total Pounds Weaned: 5,317,972.60

The difference in total pounds weaned for the study population was **172,070.40 lbs.** While the target for this example may seem high, it was achievable within this population. Depending on optimal goals of an operation, the movement from a seemingly acceptable calving distribution to one that is shifted even earlier in the calving season may still be advantageous.

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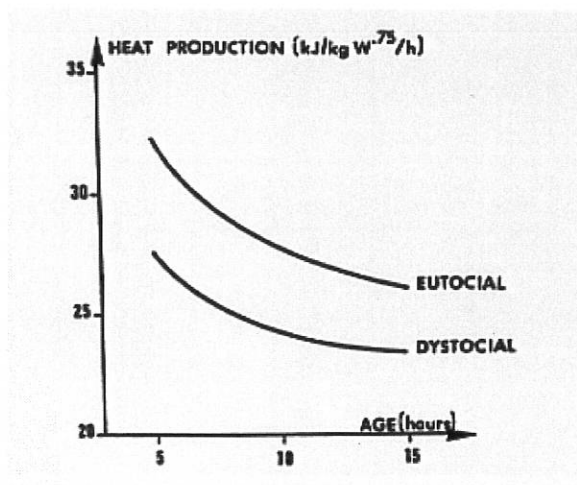
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CALVING AND HANDLING CALVING DIFFICULTIES

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Introduction

The most common reason for calf losses in the beef cattle industry is still calving difficulty. Looking only at the effects of calving difficulty on the calf, we find the following four relationships well established in the literature. First, the more difficult the calving difficulty the greater risk for infectious disease. Typically, this is reflected by higher incidences and death loss associated with either diarrhea or respiratory disease. Second, the more difficult the calving difficulty, the harder it is for the calf to maintain its body temperature following calving. This is illustrated in Graph 1:



Graph 1: Relationship between calving difficulty and body heat production of the calf

The third relationship with degree of calving difficulty is the decrease in absorption of protective antibodies with more difficult deliveries. The fourth relationship with calving difficulty that has been firmly established is the increased infertility losses in the dam. To these losses must be added the increased maternal deaths, treatment costs and diminished productivity of the dam. In a survey of the effects of dystocia on fertility in 1889 cows, Laster et al. (1973) found that cows which experienced calving difficulties had a delay in resuming estrus and showed 15.9% reduction in conception rate compared with cows which had calved normally.

Commonly, producers and veterinarians alike, feel that successful dystocia management is achieved with a calf that is alive at birth. I would offer that the successful management of calving difficulty is achieved when we optimize calf survivability and dam reproductive performance. Thus, the goal

of providing assistance is to minimize stress on the calf and dam. This article describes some useful guidelines and obstetrical techniques that can help you reduce the losses due to dystocia.

The basics of normal calving

The recognition of dystocia comes first from a basic understanding of normal calving. From this understanding, you can establish guidelines for frequency of observation of cattle during calving and when to provide assistance during the delivery process. The rancher and/or his personnel must use good judgement in deciding when to intervene and when to call for professional help if calf losses are to be reduced.

Calving is a complex process. Many mechanisms affect the process, but none completely control it. As the fetus matures and the uterus enlarges, the capacity of the placenta to respond to additional demands of the fetus may be surpassed. The placenta may begin to function less efficiently due to limiting morphologic changes, which occur during the latter part of pregnancy. These or other undefined stimuli cause a fetal stress reaction. In cattle, this results in an increased production of glucocorticoids such as cortisol and steroid precursors to estrogens from the fetal hypothalamo-pituitary adrenal systems. These steroids in turn enable the feto-placental unit to produce estrogens and prostaglandins. Endometrium layer in the uterus may also produce prostaglandins. Concurrently, production of progesterone is decreased, probably at least in part due to the luteolytic effect of the prostaglandins on the corpus luteum of the ovary. The estrogens and prostaglandins in turn stimulate maternal release of oxytocin, sensitize the uterus to the effects of oxytocin, and cause the cervix to dilate. The uterus is thus released from inhibition by progesterone and made sensitive to the stimulatory effects of prostaglandins and oxytocin, and to stimulation mediated through the autonomic nervous system. Uterine muscles, which have increased contractility in late pregnancy due to stretching, begin to contract regularly as the cervix dilates. When the cervix is dilated, fetal parts are forced into the birth canal. These produce point pressure in the vagina, further stimulating release of oxytocin and initiating the abdominal press. The process appears to have a cascade effect and is irreversible. The fetus must be delivered or death of either the fetus and/or the dam or both are likely to occur.

Signs of labor

From the practical viewpoint, the time sequences involved in calving are more important than the biological process. Prediction of time of calving would be of value under certain conditions, but it is difficult to predict time precisely on the basis of clinical signs. Criteria that have been used in attempts to identify the onset of labor in cattle include changes in body temperature, respiration and heart rates, "springing" or relaxation and enlargement of the vulva, udder changes including enlargement, tenseness and filling of the teats, changes in quantity and viscosity of vaginal secretions, relaxation of the sacro-sciatic ligaments, and dilation of the cervix.

Two criteria, relaxation of the sacro-sciatic ligaments and cervical dilation, are more reliable, but difficult to apply on a practical basis for beef operators. Relaxation of the sacro-sciatic ligaments can be palpated best by inserting one hand into the rectum and placing the other on the caudal border of the ligament from the outside. Displacement of the ligament can be estimated when

pressure is placed against it from the inside. Several days before term, the ligament can be displaced up to 2.5 cm (1 inch). This relaxation should not be confused with the progressive relaxation that occurs just before calving, allowing displacement of the ligaments 5 cm (2 inches) or more. Successive palpation will help define this stage, which indicates that calving will usually occur within 24 hours.

Dilation of the cervix begins shortly before calving. It is usually closed prior to calving; although up to four fingers can be inserted part way in some cows. Normal dilation preceding calving can be identified by a progressive, conical dilation of the cervical canal with the apex of the cone toward the internal Os. When uterine contractions begin, mechanical forces are applied to the internal Os and enlargement of the cervical canal proceeds throughout its length. Once cervical dilation is initiated, calving usually occurs within 24 hours, sometimes in as little as 6 hours in mature cows. Cervical dilation is very rapid in most cows after it has opened enough to allow passage of the hand. Normal calving is a continuous process, but is often divided into three stages for the purposes of description. These stages are arbitrary but fairly well defined. They usually follow one another in the sequence given, but sometimes, when dystocia is present, fetal membranes are expelled or at least freed from their maternal attachments before a dead fetus is delivered. Dystocia occurs when any stage is slow developing or fails to progress normally.

Stage 1

Visible signs of labor may be scant or absent in mature cows, but more evident in the first-calf heifers. The pastured cow will usually seek an isolated place and vaginal discharges increase in liquefaction and expulsion of the cervical plug. The cow (particularly first-calf heifers) will show signs of uneasiness and pain.

Occasionally she will kick at her belly and wring her tail. Restlessness and a tendency to lie down and get up frequently are also often observed. Stage 1 begins with contraction of the longitudinal and circular muscle fibers of the uterus and ends when the cervix is fully dilated and fetal parts enter the birth canal. Uterine contractions first occur about every 15 minutes, but by the end of stage 1, they occur about every 3 minutes. As the first stage progresses, the contractions become strong enough to cause the cow to arch her back and strain slightly. In cattle, the normal duration of stage 1 is 2-6 hours, sometimes longer in heifers.

What is happening inside the cow's uterus at this stage? Each time the uterus contracts, the cow feels a slight, sharp pain which produces her uneasiness. With each uterine contraction you have to realize that some separation of the normal strong attachment of the placenta to the cow's uterus is being weakened. Thus, the supply of oxygen may be decreasing with each uterine contraction. With each uterine contraction the cervix is also progressively dilating. Normally, the first water sac (chorioallantoic sac) is forced into the dilating cervix and breaks during stage 1 and the rancher may observe that the water has broken. Certain abnormal deliveries are characterized by a failure of the heifer or cow to progress into stage 2 and the calf may be dead before the decision for intervention is made. Thus, if you really suspect a heifer or cow has been in stage 1 too long and not progressed into Stage 2, intervention is recommended.

Stage 2

Second stage labor begins when the cervix is fully dilated and the second water sac (amniotic sac), plus fetal parts enter the birth canal further stimulating stronger uterine contractions. The unbroken water sac is often forced through the vulva after the cow has been in labor a short time. For the producer, the observation of the water sac is probably the most practical indication the animal is in stage 2 labor. When point pressure is applied to the birth canal by fetal parts, the abdominal press accompanies its uterine contractions. The pains of uterine contraction at this point usually force most cows to lie down. The abdominal press is exerted more frequently as labor progresses until it occurs up to 1-3 times per minute.

At this point it is appropriate to introduce the terms of **presentation**, **position**, and **posture** of the fetus during a delivery.

- **Presentation** refers to whether the calf is coming frontward, backward, or transverse.
- **Position** refers to whether the calf is right side up or upside-down with only right side up being considered normal.
- **Posture** refers to the relationship of the calf's legs and head to its own body.

The most frequent calf delivery is a frontward presentation, right side up position, and a normal posture of both front legs and head extended into the birth canal.

Sometimes a backward presentation may occur and may be deliverable if we have right side up position and the posturing being with both hind limbs in the birth canal. Nevertheless, a backward presentation should be considered a high-risk delivery a grounds for intervention. All other presentations are considered abnormal.

During delivery, a series of frequent abdominal presses followed by a short period of rest is characteristic. The greatest frequency and force is achieved when the fetal head is being forced through the birth canal and vulva. Following delivery of the head, a short period of rest may occur. Strong expulsive efforts are required again to force the shoulders and chest of the calf through the birth canal. Sometimes the cow will stop straining for a short time following delivery of the chest, allowing the rear legs to rest in the birth canal.

At this point, usually the umbilical cord may be compressed shutting off the oxygen supply to the calf from the dam. It is not unusual to observe the calf establishing its own breathing at this point. Occasionally at this point, the sac is still over the head of the calf, and the calf could suffocate if the sac is not broken. Delivery of the hips and legs is usually uneventful; occurring soon after the chest passes through the vulva. Second stage labor lasts from .5 to 4 hours in the cow, but intervention guidelines suggest assistance at not over 2 hours, and earlier if it is not progressing normally.

Stage 3

The placenta or fetal membrane is usually expelled within 8-12 hours after delivery of the calf.

Recognizing deliveries that may need assistance

At the time of calving all preventive procedures have been exhausted and the producer is left with human judgement as the biggest single factor in successful deliveries. **Four managerial decisions that dramatically effect the outcome are:**

- **Frequency of observation**
- **When to intervene**
- **Can the calf be delivered by forced extraction, and**
- **When to call for professional assistance**

After a thorough understanding of the stages of labor involved in a normal delivery, it is possible to establish a recommended frequency of observation of cows and heifers during the calving season or period. It is recommended that frequency of observation be a **minimum of three hours apart**. This recommendation may need to be modified to fit within the economic restraints of the individual ranch operation, but should be weighed heavily in favor of calf survivability. Large cow/calf operations usually can provide almost full-time observation of their heifers, but may fall short of adequate in mature cows. Smaller operations may find it more difficult to have the level of observation that would be optimal. Nevertheless, efforts need to be made to come as close as possible to these guidelines if losses are to be decreased.

The decision to intervene should be made based on sound judgement. An understanding of the rationale behind these guidelines is essential for all personnel who may be involved in the calving crew. Recommended guidelines based on stage of labor are:

Stage 1

If you suspect the cow has been in stage 1 of labor for over 8 hours, intervention is indicated. Some abnormal deliveries do not allow the cow to progress into a normal stage 2 of labor. In other cases, the cow may be in a state of uterine inertia and will not go into stage 2 of labor.

Stage 2

Intervention is indicated if any of the following conditions of stage 2 exists:

- **If the water sac is visible for 2 hours and the cow is not trying.**
- **If the cow has been trying for over 30 minutes and making no progress.**
- **If the cow has quit trying for over a 15-20 minute period of time after a period of progress. Breaks normally should not exceed 5 to 10 minutes unless fatigue or uterine inertia is involved.**
- **If the cow or calf is showing signs of excessive fatigue and stress--like swollen tongue of the calf or severe bleeding from the rectum of the cow.**
- **If from an observational standpoint you determine that you have an abnormal delivery from the presentation, position, and posture standpoint.**

Stage 3

If the cow has not passed fetal membranes within 12 hours of calving, intervention may be necessary. If they are retained, treatment may be indicated. In no instances, however, is manual removal of the fetal membranes advocated, as this is detrimental to subsequent reproductive performance.

These specific guidelines for intervention in protracted labor will be adequate in most instances. However, the stockman should realize that interruption of normal progress of labor at any stage or time is sufficient justification for intervention. Early intervention appears to be of the greatest benefit for calf survivability and reproductive performance of the heifer or cow. Early intervention is defined as 30 minutes after presentation of the water sac with feet outside the vulva. Late intervention was 1 hour. This is particularly significant when a recent survey of national cattle producers revealed that they would not intervene in Stage 2 of labor until 3 hours had elapsed (Beef'97; NAHMS)

Table 1: Effect of intervention time on calf survivability and cow reproductive performance

Item	<u>Intervention time</u>	
	Early	Late
Calf vigor (1-3)	1.1	1.1
Calving Difficulty Score (1-5)	2.1	1.3
Postpartum Interval (days)	52	54
1 st Service CR (%)	75	60
% Heat begin breeding season	91.4	81.7
% Pregnant	90	76

Bellows, RJ. 1984

Physical facilities and equipment for handling dystocia

The design of the physical facilities should allow easy entry of animals and minimize the stress of handling and restraint during assistance. Preference on most operations, unless on very small herd size, is to have a separate delivery and post-delivery areas. This is advocated from both the physical handling of the delivery and the potential for build-up of disease producing organisms. For the calf, dam, and attendant providing assistance, protection from the elements is the most desirable. Being dry and warm will go a long way in encouraging the use of proper techniques in dystocia management. The delivery area should have a straight-sided head catch with side gates that are hinged on each side of the head catch to swing freely to either side with sufficient room to the sides and rear to allow assistance with the needed personnel or fetal extractor. This allows the cow to go down and still breathe comfortably.

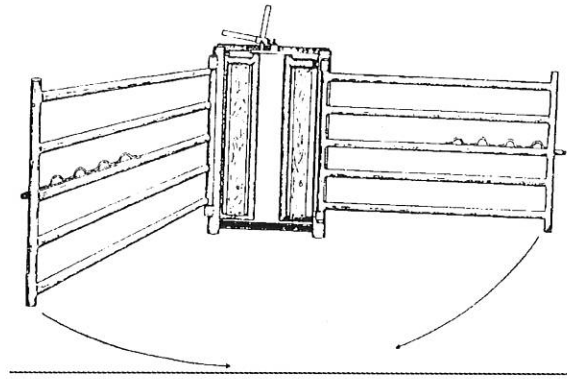


Figure 1: Suggested delivery restraint facility

Handling dystocia in squeeze chutes is to be avoided. A cement floor is recommended for cleaning purposes with access to both hot and cold water preferred. This may seem like a luxury, but may make the difference in optimizing calf survival from the standpoint of accurate decision making during the delivery. Obstetrical chains are preferred to rope when traction is required because they are more easily sanitized. Also, handles are available which attach anywhere along the chains and make traction easier to apply. Nylon web obstetrical straps are available. These may be less traumatic to the fetus, but must be sanitized very carefully between deliveries. Fetal extractors are an essential component of the calving shed, but are a dangerous item of equipment if misused.

How to examine the cow

Keeping in mind:

- Obstetrics is a branch of surgery and therefore cleanliness, asepsis, and gentleness are of prime importance.
- Whereas the treatment must be humane and should be carried out with adequate available help to realize the best outcome.
- Obstetric cases should always be regarded with urgency but the actual intervention requires patience, bearing in mind the normal duration of the different stages of labor.

First, take time to scrub the hands and arms thoroughly with soap, warm water and antiseptic. Now wash the vulva and anal area plus an area lateral to both. Use of plastic palpation sleeves is recommended in most instances in the examination process--especially in geographical areas with herds who have a Brucellosis problem.

Insert your hand slowly into the vagina, and don't rupture the water-sac unless the cervix is fully dilated. Use lubrication as necessary and determine the presentation, position, and posture of the calf. If the calf's presentation, position or posture is not normal, you may want to seek help particularly if you encounter a delivery you have not seen before. Then, the presentation, position, and posture of the fetus should be assessed and a determination made as to whether it can be handled within the capabilities of the assistant. Luckily, the handling of most dystocia problems is

within the capabilities of the stockman. If assistance is to be provided, it is essential that the assistant have a thorough understanding of the amount of traction, direction of pull, and limitations of assistance in the delivery process. If not, more qualified help should be sought immediately. The methods presented in the following paragraphs are for the Utrecht method of handling dystocia.

Before attempting assistance it is best to determine if the calf is alive or dead and the relative strength of the calf before attempting correction and manipulations. **The reflexes used for determining viability are:**

- **Withdrawal reflex-** Pinching between the digits of the hoof, and the calf withdraws its limb in response.
- **Suckle reflex-** Placing hand in mouth you can feel mouth close or tongue move.
- **Poke in eye-** Calf usually responds by withdrawing head.
- **Check for heartbeat-** For a frontward calf, run hand down along side of the chest and feel for the heart beat, or in a backwards calf feel for pulsation in the umbilical cord.
- **Rectal reflex-** In backward calf, sticking finger in rectum should elicit contraction on the finger in a good strong calf.

These reflexes themselves are helpful in determining if the calf is strong enough to withstand correction and/or delivery by forced extraction. They are generally unnecessary to apply if you have observed good observation times and intervene according to the given rules. The second point to be made relative to providing assistance in correction is to understand that the uterus has contracted down around the calf from all directions. This has decreased the amount of room within the uterus for corrective purposes. It is generally advantageous to apply lubrication liberally within the uterus of the cow before correction is started. In some instances, the use of 4 to 5 gallons of warm water with lubricant in it will also help distend the uterus to give you the extra room needed for correction.

Lastly, the following guidelines are recommended to call for professional help to maximize the opportunity for a live calf. Professional assistance needs to be defined as someone who knows more about handling the problem than you do. The different level of experience among individuals will dictate what problems you are requiring assistance in. Regardless of the experience level, if these rules are followed the survivability opportunities of the calf and dam are increased. **The suggested guidelines are:**

- **Don't know what problem they are dealing with!**
- **Know the problem and the solution, but know they are unable to handle the problem!**
- **Know the problem and the solution; have tried and simply made no progress in a 30-minute period! Further delays will simply put the calf in jeopardy.**

Causes of dystocia

The **most common** cause of dystocia is a relatively oversized frontward-presented calf for the size of the birth canal. This calf is also in the normal position (right side up) and posture (both front legs and head presented in birth canal). This accounts for 90% or more of the assisted births at the

producer level. Another 5% of dystocia are due to abnormal presentation, position, and posturing of the calf. The remaining 5% of dystocia is due to the cow herself and not the calf. This is in most instances uterine inertia due to fatigue, disease, or metabolic problems. Failure to follow recommended guidelines would result in more dystocia due to uterine inertia than would be expected.

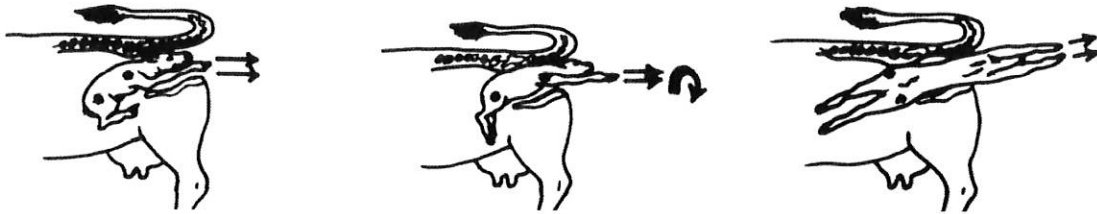


Figure 2: Sequence of delivery by forced extraction of calf in frontward presentation with normal position and posturing

Oversize calf

The clinical diagnosis "oversize calf" includes "relative oversize" in which the calf is of normal dimension but the maternal pelvis is too small, "absolute oversize" in which the maternal pelvis is normal but the calf is abnormally large. In cases of oversize it may not be always be obvious to the obstetrician whether the calf is too large or the pelvis too small, but the technique of delivery is the same irrespective of the source of the trouble.

Delivery of the frontward calf by forced extraction

The question for the producer is whether the calf is deliverable by forced extraction or not. After examination and a determination that the calf is in the correct position and posture, chains or straps should be placed on both forelimbs. They may be placed either above the fetlock joint or below or above the hoof of the calf or a combination of the two. In most instances, it is recommended to place the chains above the fetlock and take a half hitch below the fetlock joint as well. In placing above the fetlock, please insure you are where the bone is decreasing in size on the leg so you are above the growth plate of the bone. Traction should only be applied when the cow is assisting with an abdominal press. You are now at a point to determine if delivery by forced extraction is possible. (Figures 2 & 3) This test for delivery is valid only if certain criteria are followed relative to position of cow, type and amount of traction, and direction of pull. Traction should only be applied when the cow is assisting with an abdominal press. In the frontward presentation with normal position and posture of the fetus, this guideline is whether both shoulders of the calf can pass through the pelvis of the cow using recommended traction techniques. To actually determine this, the cow should be down, on her **right side**, and traction should be applied to one leg at a time (unilateral traction) to walk the shoulders through the pelvis of the cow. Positioning of the cow on her right side allows the frontward calf to enter the pelvis of the cow relatively straight. In a difficult delivery, this is important. Type and amount of traction should be no more than the force of one person per leg and the direction of pull should be straight out. Research in England has demonstrated that direction of pull straight out reduces the amount of force need by about 30%.

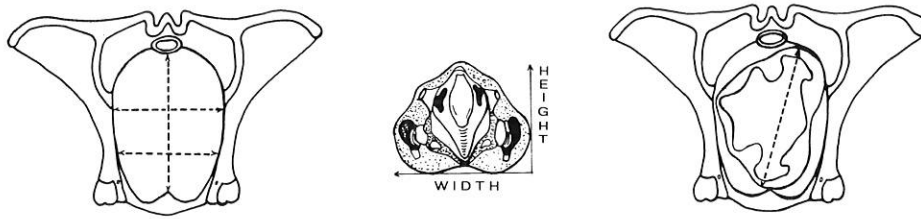


Figure 3: Illustration of relationship between pelvic opening shape of the cow and cross-section of the hips of a calf during delivery process

This is very advantageous in reducing stress to the calf. It is preferable to start with the down leg (left) of the calf. This usually comes through easily, so **the actual test for delivery is if you can get the second shoulder past the cow's pelvis**. You should be able to feel the shoulder move past the pelvis as you are applying traction. However, a suggested rule to determine if the shoulder of the calf is past the pelvis of the cow is if the calf fetlock joint is one hand's breadth or about 10 cm outside the vulva of the cow. Once the first shoulder is through the pelvis of the cow, it should be held in place and traction applied to the other leg. The amount of traction should be limited to the force of one man per leg. Two strong men can exert a force of from 400-600 pounds while erroneous use of a fetal extractor could exceed 2000 pounds of pressure. Thus, good clinical judgement in the application of traction is important and necessary. Our goal is to deliver a live calf with the maximal opportunity for survival. Exceeding this rule may result in the delivery of the calf, but will markedly increase the chances for the loss of the calf during delivery or subsequently to disease, cold, or starvation.

Once the shoulders of the calf are through the pelvis of the cow, delivery by forced extraction is possible. If not, call for professional assistance, as a C-section is

recommended if you want a live calf. Bilateral traction can be exerted at this point to further pull the calf before the pelvis of the calf enters the pelvis of the cow. As in the normal delivery, this is when the umbilical cord is compressed and the cow usually takes a break for a short period of time. This is a point when the calf should be allowed to breathe on its own or oxygen can be administered. It is also a point with the oversized fetus where rotation of the calf should occur. This rotation is necessary to bring the widest part of the calf pelvis through the widest diameter of the cow's pelvis. Once breathing has been established, completion of delivery is possible in most instances. Occasionally, calves are lost because of failure to allow the calf to breathe. Constant pulling on the calf at this point will not allow the calf to expand its chest and take in any oxygen and it is possible to lose the calf if breathing is not allowed.

Delivery of the backward calf by forced extraction

The test for delivery of a calf in the backwards presentation but normal position and posture differ in that the fetus should be first rotated 45-90 degrees by crossing the legs before attempting delivery to take advantage of the widest diameter of the cow's pelvis. In addition, the direction of pull on the calf is in a direction that is slightly up from a line straight out from the back of the cow. (See Figures 3 & 4)



Figure 4: Sequence of delivery of calf in backward with normal position and posturing

Bilateral traction can be applied in the amount of two men and should be applied bilaterally (both legs at the same time). The test for delivery is if both hips of the calf can pass through the pelvis of the cow. This is determined in most instances by the extension of the hocks of the calf beyond the vulva. If this is easily accomplished, possible delivery can be made. However, now we have very little time left to accomplish rotation of the fetus to a right side up position for the chest of the calf to come through the pelvis of the cow and deliver the calf. We have probably no more than 2-3 minutes to complete the delivery. If the test fails in either case, call for professional assistance as surgical delivery is probably indicated.

There is a common misunderstanding, that calves need to be pulled out very rapidly, otherwise they will die. One must remember that the calf's life will not be compromised until its umbilical cord becomes trapped against the maternal pelvis. In practical terms, therefore, traction should be slow and controlled until such time as the calf's tail head and anus begin to emerge from the cow's vulva. Once this point is reached, delay should be avoided.

Common abnormal presentations, positions and postures

Abnormal presentations, positions, and postures are best corrected while the cow is in the standing position. Once corrected, the tests for delivery can be applied as previously described. Try to carry out all these operations when the cow is not straining vigorously.

Elbow lock posture

If one or both of the forelimbs are not extended as they come into the pelvic inlet,

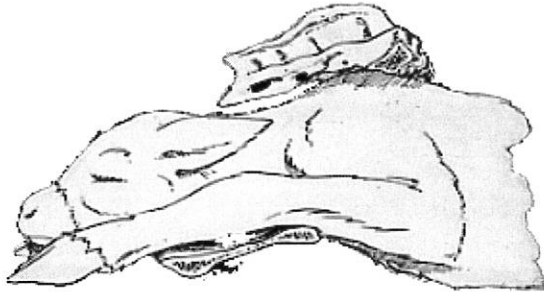


Figure 5: Calf in elbow lock posturing

the partly flexed elbows may lock on the brim of the pelvis and cause elbow lock. This is an easily corrected problem requiring repulsion of the body of the calf while simultaneous traction is exerted on the affected limb.(Figure 5)

Deviation of the head

If the head cannot be felt, do not assume that the calf is coming backward. The two front legs may be presented and the head deviated to the side or down between the front. Before pulling on the limbs, distinguish between forelimbs and hind limbs as described earlier. If the head is bent back into the right flank of the cow it will be easier to correct if the left hand is used and vice versa. By grasping the muzzle or by placing the thumb and middle finger in the eye sockets, the head can be raised and directed into the pelvis (Figures 6, 7, & 8). A loop of



Figure 6: Correction of deviated head by grasping the muzzle or nose of calf

soft rope or chain placed in the mouth and looped up around the poll of the head behind the ears will sometimes be helpful. The honda of the rope may be placed next to the mouth and the rope placed above the tongue of the calf. In some instances, looping the rope around the lower jaw may be used instead. However, it is easy to use excess traction and fracture the lower jaw, so this should be avoided unless absolutely necessary. In all these cases, the head can be brought up and straightened more easily if the body of the calf is at the same time repelled further back into the

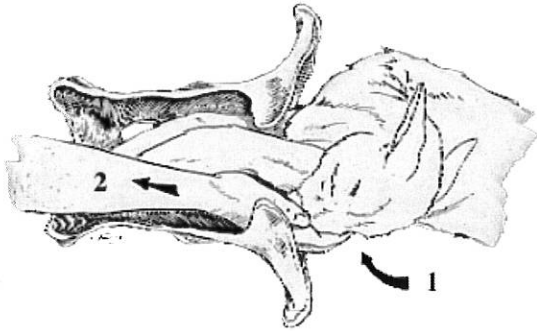


Figure 7: Correction of deviated head by swinging head into correct posture before extraction is attempted

uterus. This can be done by placing the hand between the front legs and pushing back the chest while the head is being pulled into the pelvis at the same time. In some instances, it is necessary to create even more room to correct the head. This may require one of the front legs to be pushed back into the uterus to create a retained front leg that is flexed at the shoulder. In the case where the head is between the front legs this needs to be done first. This would allow you to manipulate the head into first a lateral deviation and then correct as previously described.

It should be pointed out clinically that many of the calves with the head deviated between the front legs are dead or weak before you even start. In addition, many of the calves with a deviated head will still fail the test for delivery for using forced extraction once they are corrected.

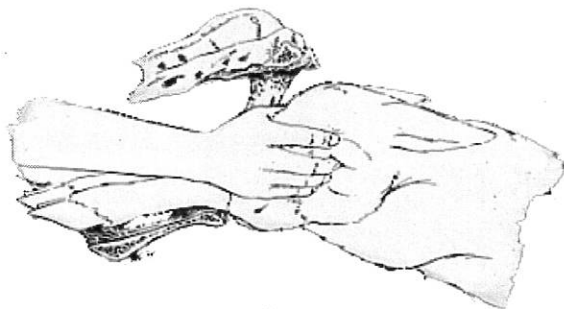


Figure 8: Alternative use of gripping the orbits of the eye in correcting a head deviation

Therefore, good judgement needs to be used before putting excess stress on the calf during assistance.

Retention of one or both forelimbs

The calf may have the head out, but one or both forelegs retained. Secure the head by placing a chain behind the poll and through

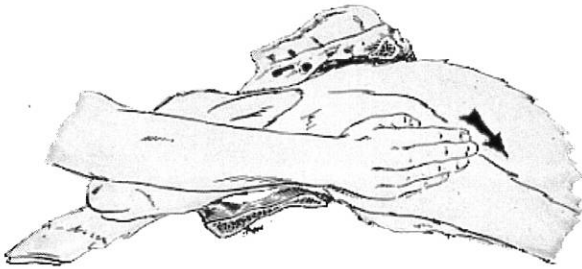


Figure 9: Correction of retained forelimb step 1. Slipping hand down below elbow to convert leg to flexed knee or carpus

the mouth, then lubricate the head and push it back into the uterus. Then search for the limbs one at a time. If fully retained, the limb should be grasped just below the knee (carpus) and the limb be pulled until bent at the knee. Once this is accomplished you can generally slip a hand down the limb and grasp the hoof. It is necessary to cup the hoof such that you are providing protection for the uterus of the cow as you continue in the correction process. (Figures 9 & 10)

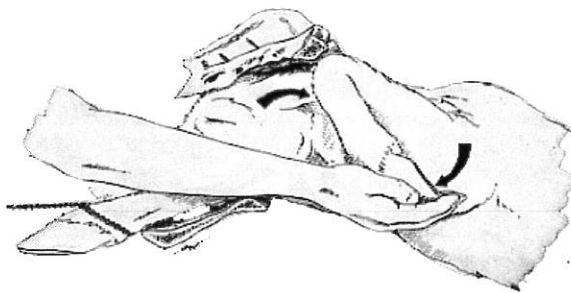


Figure 10: Step 2 of correction. Simultaneous movement of hoof toward midline of calf and knee laterally before pulling leg into extended posture

To correct, now opposing forces need to be applied simultaneously. The knee should be repelled by one hand in a forward-upward-lateral direction and traction on the hoof in a medial-backward direction by the other hand.

These directions are relative to the cow. It may be necessary to use a small rope or chain and place around the leg above the fetlock and between the digits of the hoof if getting both arms in the cow is a problem. If the other leg is retained, it is corrected in a similar fashion.

Retained hindlimb or Backward presentation, breech posture

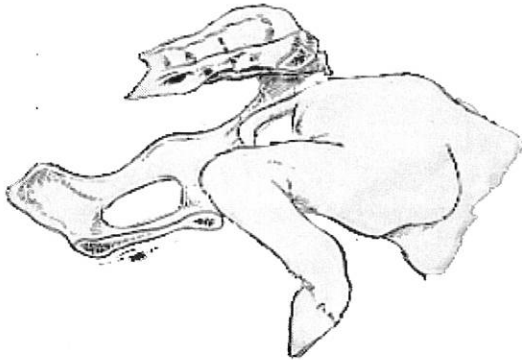


Figure 11: Retained hindlimb in flexed hock posture

The correction of this abnormal posture is the same as the retained forelimb. First, you find the hock and pull it until it is in the flexed position. Then, you slip your one hand down to cup the hoof. The hock should be

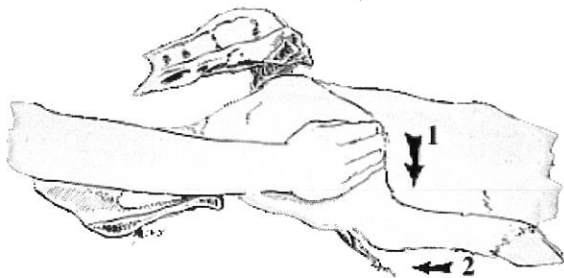


Figure 12: Step 1 in correction of retained hind limb. Sliding hand down leg to level of hock then converting limb to flexed hock

repelled by one hand in a forward-upward- lateral direction and traction on the hoof in a medial-backward direction by the other hand.

In some instances, the calf has to be repelled back into the uterus before correction can be made. Occasionally, it is difficult to get both hands into the cow for correction. In these instances, the use of a toilet plunger a repulsion device against the rump of the calf has worked effectively. (Figures 11,12, & 13)

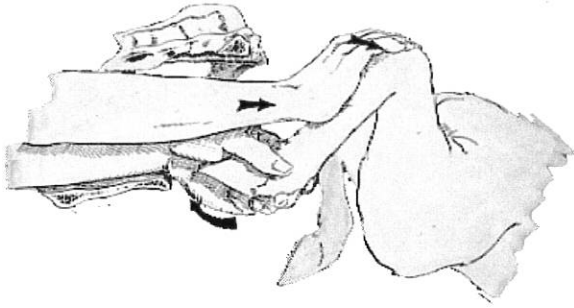


Figure 13: Correction of flexed hock by medial-posterior movement of hoof while lateral-forward repulsion of the hock

Transverse presentation

Occasionally, calves lie with their back against the pelvic opening or with all four limbs extended into the birth canal. Determine the hind from the forelimbs and if possible, deliver hind limbs first so you don't have to worry about the head. Since the calf is on its side, it's easier to rotate the calf's body by the hind legs than the forelegs. This requires repulsion of the forelimbs of the calf and usually the trunk of the calf as well. In most instances, this is a difficult correction to make. (Figure 14)

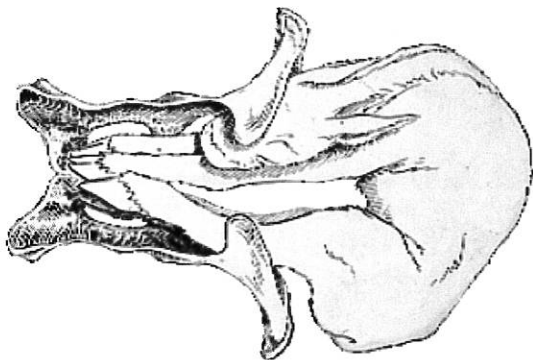


Figure 14: Calf in transverse presentation

Twins

If twins enter the vagina one at a time, there is generally no problem due to a smaller size. However, occasionally twins are presented together and block the birth canal. In most of these cases, one comes frontward and the other backward. Extract the closest twin first. If in doubt, first extract the twin presenting hind legs, after first repelling the other twin back into the uterus. (Figure 15)

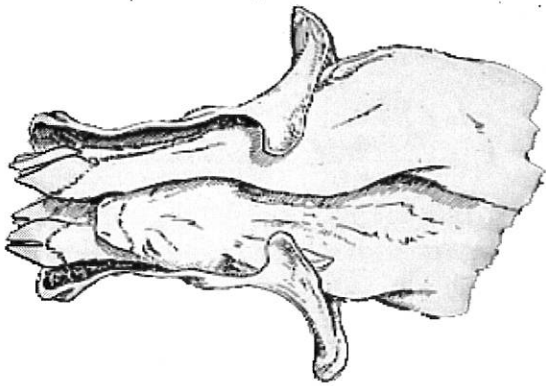


Figure 15: Twins with one each of frontward and backward presentation

C-section and fetotomy

Cesarean section is now a routine obstetric procedure in cattle practice. **It is the method of choice when you are dealing with a live calf and want to optimize calf survivability. In those cases where the calf is already dead, fetotomy is the method of choice due to optimal cow survivability.** Remember, C-section is the most invasive of the two procedures.

The reasons for surgery include the most causes of dystocia but analysis of published cases shows that the following five major indications account cumulatively for 90% of all C-section or fetotomy procedures:

- Fetal oversize
- Incomplete dilation of cervix
- Irreducible uterine torsion
- Fetal deformity or monsters
- Uncorrectable abnormal presentation, position or posture of fetus.

It has been my observation that the success of the surgical procedure has been more due to the timely decision-making of the producer than to the surgical skills of the veterinarian. Therefore, I recommend you make the decision in a timely fashion for greatest survivability of the calf and the cow.

Good luck with your calving problems!

Assisting the Beef Cow at Calving Time

Richard F. Randle, Extension Beef Veterinarian

Aaron L. Berger, Extension Educator

Six to ten percent of all calves born in beef cow herds in the U.S. die at or soon after birth. Approximately half of those deaths are due to calving difficulty (dystocia). This multimillion dollar annual loss is second only to losses from cows failing to conceive.

Factors Causing Calving Difficulty

About 80 percent of all calves lost at birth are anatomically normal. Most of them die because of injuries or suffocation resulting from difficult or delayed parturition (calving). Factors contributing to calving problems fall into three main categories: calf effects, cow effects, and fetal position at birth.

Calf Effects — Heavy birth weights account for most of the problems related to the calf. Birth weights are influenced by genetics of the sire and dam, sex of the calf, age of the cow, environmental temperature conditions, and, to a slight degree, nutrition of the cow. Shape of the calf also may have a small effect on calving problems.

Cow Effects — Several factors associated with the cow influence dystocia, the major ones being her age and pelvic size.

Age — Two-year-old heifers require more assistance at calving than do cows because these females usually have smaller pelvic areas.

Pelvic Area — Pelvic area (birth canal) increases as the female develops to maturity. Thus, a higher proportion of calving difficulty in 2- or 3-year-old cows is due to smaller pelvic openings. Heifers and cows with small



Minutes after giving birth, a 2-year-old beef cow attends to her newborn calf. Photo by Scott Bauer, USDA.

pelvic areas are likely to require assistance at calving. However, even heifers with large pelvic areas may need help in delivering large calves. The calf's birth weight and cow's pelvic area have a combined effect on dystocia. Degree of dystocia is determined primarily by the size of the calf in relation to the size of the cow's pelvic area. Therefore, calving problems can be reduced by decreasing calf birth weight and ensuring adequate growth and

development of replacement heifers from weaning to calving.

Fetal Position at Birth — About 5 percent of the calves at birth are in abnormal positions, such as foreleg or head turned back, breech or rear end position, sidewise or rotated, etc. (Figure 1). This requires the assistance of a veterinarian or an experienced herdsman to position the fetus correctly prior to delivery. If fetal position cannot be corrected, the veterinarian may have to perform a caesarean section.

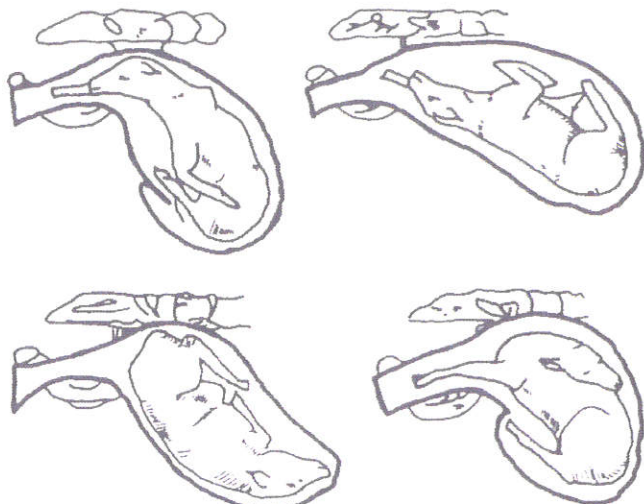


Figure 1. Abnormal positions of the calf for delivery.

Stages of Calving

Parturition, the stages of calving, is a complex, dynamic process initiated by the fetus through hormonal signals transmitted to the dam as well as mechanical and neural stimulation in the uterus. Normal calving can be divided into three general stages: I - preparatory, II - delivery of fetus, and III - expulsion of the placenta or afterbirth. The time interval of each stage varies among types and breeds of cattle and among individuals of the same breed. A general understanding of the birth process is important to proper calving assistance and is summarized in Table I.

Table I. Stages of Calving

Stage and time	Events
I - Preparatory (2 to 6 hours)	Calf rotates to upright position Uterine contractions begin Water sac expelled
II - Delivery (1 hour or less)	Cow usually lying down Fetus enters birth canal Front feet and head protrude first Calf delivery complete
III - Cleaning (2 to 8 hours)	"Button" attachments relax Uterine contractions expel membranes

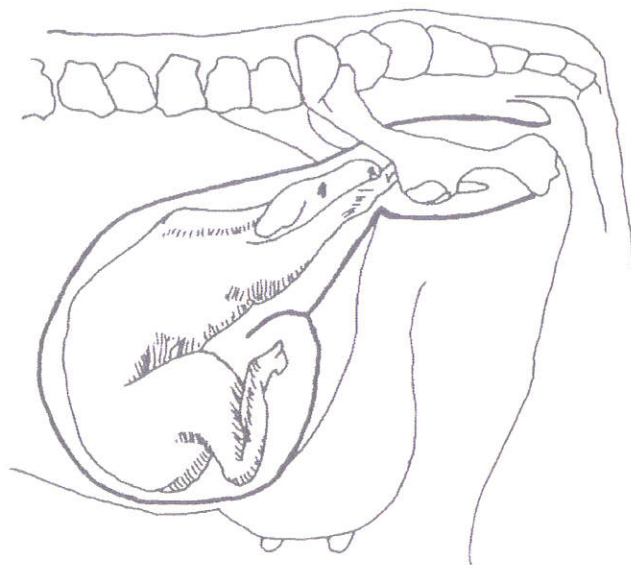


Figure 2. Normal position of the calf just prior to delivery.

Stage I - Preparatory (two to six hours) — During pregnancy, the fetal calf resides in a large fluid-filled sac and can be found in many positions. Just prior to labor, it rotates to an upright position with its forelegs and head pointed toward the birth canal (Figure 2).

In the preparatory stage, the cervix dilates and rhythmic contractions of the uterus begin. Initially, contractions occur at approximately 15-minute intervals. As labor progresses, they become more frequent until they occur every few minutes. These contractions begin at the back of the uterine horn and continue toward the cervix, forcing the fetus outward. Any unusual disturbance or stress during this period, such as excitement or even movement, may inhibit the contractions and delay calving.

At the end of the preparatory stage, the cervix fully dilates, allowing the uterus and vagina to become a continuous canal. A portion of the placenta (water sac) is forced into the pelvis and aids in the dilation of the cervix. This water sac usually ruptures and the membranes hang from the vulva until Stage II.

Stage II - Delivery (one hour or less) — Delivery begins when the fetus enters the birth canal, and usually occurs while the cow is lying down. Uterine contractions are now about every two minutes and are accompanied by voluntary contractions of the diaphragm and abdominal muscles.

Surrounded by membranes, the calf's forelegs and nose now protrude from the vulva. After the nose is exposed, the dam exerts maximum straining to push the shoulders and chest through the pelvic girdle. Once the shoulders have passed, the abdominal muscles of the calf relax, and its hips and hind legs extend back to permit easier passage of the hip region.

The calf is normally born free of fetal membranes (placenta) because they remain attached to the cotyledons or “buttons” of the uterus. This ensures an oxygen supply for the calf during birth. Upon passage through the vulva, generally, the umbilical cord breaks and the lungs become functional.

Delivery is normally completed in one hour or less. Special assistance is warranted if this stage goes beyond two to three hours.

Stage III - Cleaning (two to eight hours) — The caruncle-cotyledon, or button attachment between uterus and placenta, relaxes and separates after parturition. The placenta is then expelled by continued uterine contractions. Cows normally expel the placenta within two to eight hours.

Preparing for Calving Assistance

Normal delivery should be completed within two to three hours after the water sac appears in heifers, and one to two hours in cows. If prolonged, the calf may be born dead or weak.

Because timing is vital to providing proper assistance, frequent observations are a must. Assisted deliveries should not be attempted without proper preparation of facilities and equipment. A clean, well-lighted maternity stall with head catch facilitates examination. Head catch facilities should include removable or fold-out sides that allow a cow to lay down with ample room for those assisting once the “pulling” of the calf begins.

Obstetrical (OB) equipment such as chains and handles should be placed in a bucket of water with disinfectant before use to reduce bacterial contamination. Disinfectant, soap, and lubricant should be in plastic squeeze bottles to enhance use.

Check with your veterinarian for advice on when to assist a cow alone and when to call him or her. Experience will help determine if the calf can be delivered with assistance or if a caesarean is necessary. Determination is usually made on initial examination. The goal is to deliver a live calf from every cow.

Steps in Calving Assistance

1. After observing a delay in delivery, a pelvic examination should be done to determine the extent of cervical dilation. The cow’s vulva and rectum should be scrubbed. You should scrub your hands and arms, and wear plastic shoulder-length OB sleeves. Lubrication should be applied to the OB sleeves.

2. Determine the position of the fetus (*Figures 1 and 2*). If it is in an abnormal position, experience and judgment must be used to determine if a correction can be made or if professional help should be summoned.
3. Examine the size of the calf relative to the birth canal. A large calf forced through a small pelvic opening may result in death of the calf and injury (including paralysis) to the cow. If this examination is made when the head and front feet are still in the birth canal, the opportunity for a successful caesarean section exists.
4. Attach the obstetrical (pulling) chains to the front legs of the calf, placing the loop of each chain around each leg. Then slide the chains up on the cannon bone 2 to 3 inches above the fetlocks (ankle joints) and dew claws. Place a second loop between the fetlocks and the top of the hoof (*Figure 3*). Make sure the chain pulls from either the top of the leg over the fetlocks or the bottom of the leg (dew claw side).

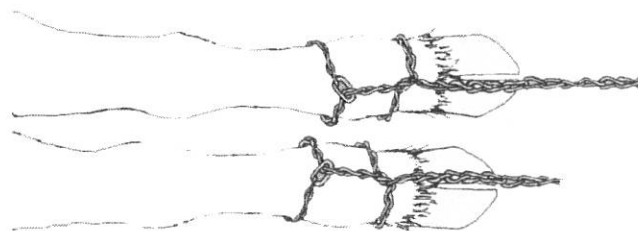


Figure 3. Proper attachment of the pulling chains.

5. Attach the obstetrical handles and pull gently, making sure the chains have not slipped. Some simple guidelines can be used to determine if the calf can pass through the pelvic canal. First, by pulling on the front legs, the entire head of the calf should enter the bony pelvic canal. Second, continue to pull on one front leg. The first joint (fetlock) of that leg should extend at least one hand’s width past the vulva of the cow. Third, pull on the opposing leg. The fetlock of this leg also should extend at least one hand’s width past the vulva of the cow. If all three of these guidelines cannot be accomplished, you should be concerned that the calf might be too large to successfully pass through the pelvic canal, and a caesarean section may be needed. If all the guidelines are met, then continue to deliver the head and shoulders.

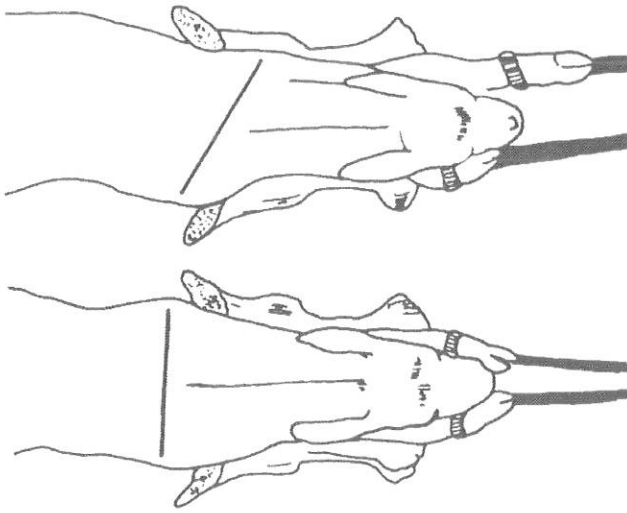


Figure 4. "Walking out the shoulders."

6. Although some calves can be delivered by pulling both legs evenly, it's usually best to alternately pull on one leg and then the other a few inches at a time (Figure 4).

This is called "walking out the shoulders."

7. Once the head and shoulders are exposed, pull the calf downward at a 45 degree angle.
8. Hip lock, which occurs when the calf's hips are horizontal to the cow's pelvis, can result in loss of the calf. If hip lock occurs, push the calf back a short distance and rotate the exposed portion of the calf 90 to 120 degrees while continuing to pull downward at a 45 degree angle. If the cow is lying down, roll her on her left side before rotating the calf. Make sure the calf begins breathing normally as the umbilical cord will be pinched closed. Call your veterinarian if the hip lock cannot be readily delivered.
9. Posterior presentations (backward calf) occur in less than 5 percent of calves born. The posterior presentation is a problem because the calf's hind legs and hips do not dilate the cervix as well as the front legs and head. Due to premature rupture of the umbilical cord, early assistance and rapid delivery are needed. A backward calf in the setting position with feet and legs up under him (breech presentation) must be detected early in labor and corrected. Cows will start labor but nothing will show externally except, occasionally, the tail of the calf. These cows often appear to be in labor for a period of time and then quit as exhaustion occurs.

10. Cows with torsion of the uterus (posterior uterus and cervix twisted) will act similarly to cows with a breech presentation; however, they usually will show much more pain. On examination, the calf is difficult to palpate and the twisted opening can be determined. If detected early, the torsion can be corrected or a caesarean performed to obtain a live calf.
11. A calf puller should be used correctly and only by experienced people. A calf puller can apply traction equivalent to the pull of seven men. First examine the cow, making sure the calf is in the proper presentation and position, lubricate the vagina, and then apply gradual traction. Maintain the butt plate of the puller just below the vulva opening and the jack end of the puller at or below the level of the cow's hocks. Excessive traction may kill the calf, traumatize the cow, and both may be lost.
12. Correcting abnormal presentations and positions after extended labor usually requires professional help. Remember: be clean, know your capabilities, and learn when to call for help.

Strategies to Use if the Calf Is Not Breathing

Once delivered, clear any mucus from the calf's mouth and throat with your hand. Then, if necessary, stimulate the calf to breathe by either rubbing it briskly or tickling the inside of a nostril with a straw.

Artificial respiration can be applied to the calf as follows: place a short section of garden hose into one nostril and hold mouth and nostrils shut so air enters and leaves only through the hose. Then alternately blow into the hose and allow expiration of air. Repeat at five-to seven-second intervals until the calf begins to breathe. Another method is to alternate pressure and release on the rib cage. Commercial respirators also are available and may be a wise investment in larger herds.

Potential Post-Delivery Problems

Uterine Prolapse. This is an inversion of the uterus that can occur following calving. Prolonged labor, difficult birth, excessive traction, and subclinical milk fever are predisposing factors. Uterine prolapse should be treated as an emergency with early intervention by a veterinarian.

Retained Placenta. The placental membranes are normally expelled within two to eight hours after birth. Occasionally, they fail to separate from the uterus. If not treated, this condition may pose a health threat to the cow and cause problems in rebreeding. The reason for retained placentas is not known, but high incidence may indicate disease. They also commonly accompany difficult births, multiple births, short gestations, and bull calf births.

Research has shown that manual removal of retained placentas will decrease fertility. The recommended treatment is to wait 24 to 48 hours after birth and then treat with injectable antibiotics along with hormonal therapy as advised by a veterinarian. Observe the cow closely for signs of illness.

Summary of Calving Management Recommendations

- Observe the herd closely during calving season, especially first-calf heifers, as they likely will require the most assistance. Be there and be an astute observer.
- Have the proper equipment and facilities available and in clean working order prior to calving.
- Give assistance during delivery or call a veterinarian when needed. Do not wait more than two to three hours after labor begins to act.
- Correct any abnormal fetal positions in the early stages of delivery.
- When pulling a calf, double loop the chain or rope above and below the ankle joint. Apply gentle traction on one leg at a time to facilitate passage of the shoulders through the birth canal.

- Remove mucus from the calf's nose and mouth immediately after birth. If the calf does not start to breathe normally, stimulate the calf to breathe by rubbing it briskly, tickling the inside of a nostril with a straw, or applying artificial respiration.
- Disinfect the navel cord with iodine to prevent infection. Make sure the calf nurses within an hour after birth or give colostrum to weak calves.
- Keep birth weight and ease-of-calving records to identify the sires and dams responsible for calving problems. This information is especially important for selecting sires to breed yearling heifers. When possible, cull females with a history of calving problems and avoid selecting replacement heifers from such cows. *Table II* shows a simple calving ease scoring system.

Table II. Calving Ease Scoring System

Score	Description
1	No difficulty, no assistance
2	Minor difficulty, some assistance
3	Major difficulty, assistance with jack or puller
4	Caesarean birth
5	Abnormal presentation

Acknowledgment

Gene H. Deutscher, former extension beef specialist, and the late Donald B. Hudson, former extension veterinarian, contributed to the original preparation of this publication.

This publication has been peer reviewed.

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A modified method for complete bovine fetotomy

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SUMMARY

A modification of the "Utrecht technique" for complete bovine fetotomy was developed. In cranial presentation, the first step is decapitation, then oblique indirect section through the neck and thorax to remove 1 forelimb with a small part of the thoracic wall. This is followed by thoracic and abdominal evisceration. The final step is direct oblique section of the fetal pelvis or, alternatively, deep detruncation followed by bisection of the pelvis. In caudal presentation, the first step is removal of a hindlimb, then abdominal and thoracic evisceration, followed by thoracic detruncation and oblique section for removal of 1 forelimb and most of the remaining thorax. The neck and remaining forelimb are removed together to complete the procedure.

AN EFFECTIVE TECHNIQUE for complete bovine fetotomy has been developed at the University of Utrecht, The Netherlands. There, partial fetotomy is usually avoided in the oversized bovine fetus. Once begun, fetotomy is carried to completion in a systematic way.¹ With the Utrecht technique, delivery of smaller pieces of the fetus is less likely to damage the birth canal of the dam and time is not wasted in futile efforts to deliver a partly dismembered fetus before it is small enough.

Survival and convalescence of the dam were important concerns with Utrecht clinicians. With a live fetus, cesarean section was chosen. However, survival and convalescence of cows following fetotomy on a dead fetus was better than with cesarean section. Consequently, they used fetotomy almost exclusively when the fetus was dead. Work in Australia has yielded similar results.²

In many cows in dystocia, the disproportion of fetal size to maternal pelvic dimensions is not so great that all cuts in the Utrecht fetotomy need be made. Consequently, modifications of the Utrecht fetotomy in both cranial and caudal fetal presentations have been developed in a mock uterus and tried

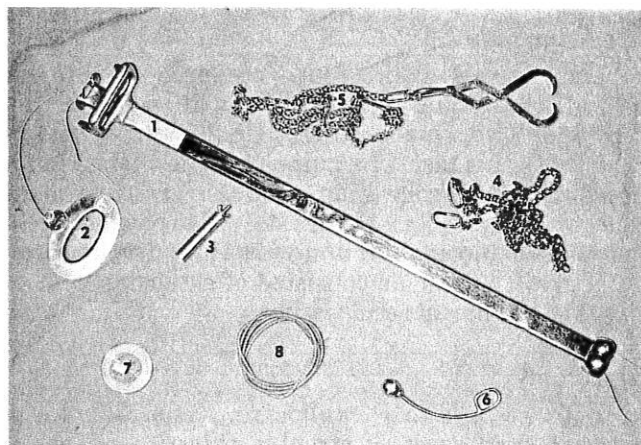


Fig 1—Equipment used in fetotomy. 1=fetatome; 2=spool handle for wire saw; 3=locking handle for wire saw; 4=obstetrical chain used in application of counterforce; 5=Krey's hook with obstetrical chain attached; 6=wire saw introducer; 7=spare wire saw; 8=fetatome wire saw threader with cleaning brush on one end.

in the field in approximately 40 cows. In most instances the method was successful; in 2 cases more complete fetotomy was required.

Fetotomy Equipment

Good equipment is necessary for effective fetotomy.¹ A double-tubed fetatome (Fig 1,1) is better than a fetatome with a single tube because the likelihood of fouling of the wire saw is decreased. The head and butt of the fetatome should be hardened, and the wire saw openings should be rounded. The fetatome head should be smooth and rounded, beginning at the wire saw outlets and continuing around the outside to the attachment of the head to the barrel, thus preventing fraying and breakage of wire during use.

One wire saw handle should be constructed to allow spooling of wire (Fig 1,2). If the wire begins to fray during a cut, additional wire can be played out from the spool and an equal length can be removed at the other handle. The second handle should attach to the wire saw by clamp or other secure means (Fig 1,3). We have found quick release handles to be unsatisfactory.

Other necessary tools include obstetrical chains (Fig 1,4), a Krey-Schöttler hook (Krey's hook) with chain attached (Fig 1,5), and a wire saw introducer (Fig 1,6). Good quality wire saw (Fig 1,7), a fetatome

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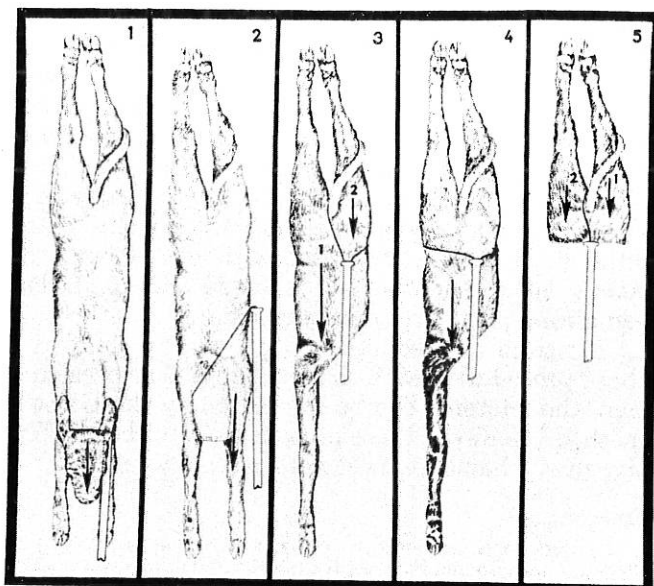


Fig 2—Steps in modified fetotomy, with fetus in cranial presentation—fetatome and wire saw placement. 1 = head removal; 2 = oblique section of the forelimb, neck, and part of thorax; 3 = section of pelvis; 4 = alternate method leading to section of pelvis—deep section of the fetal trunk; 5 = bisection of the pelvis.

wire saw threader (Fig 1,8), and a pair of side-cutting pliers (not shown) are also necessary.

Fetotomy, Cranial Presentation

If the fetus is in cranial presentation, a loop of the wire saw is worked around the fetal neck, the fetatome head is placed alongside or beneath the fetal jaw, and the head is removed (Fig 2,1). This cut makes available room for additional fetotomy. The second cut runs obliquely across the fetal neck and thorax (Fig 2,2), as described by Australian workers,³ and it creates an opening into the thoracic cavity large enough to admit a hand. This cut requires counterforce. In general, counterforce is applied by attaching a chain to the fetal part being removed, placing the fetatome head in position, then attaching the other end of the chain into one of the niches in the fetatome buttplate (Fig 1,1). Counterforce is necessary to prevent displacement of the fetatome head caudally when the wire saw is angled sharply away from the head of the fetatome as the cut is being made. To remove the neck and forelimb, the head of the fetatome is placed about 4 inches caudodorsal to the scapula of the fetus, and a counterforce chain is applied to the limb being removed. Tension can be maintained on the neck stump by securing it with a Krey's hook to which traction is applied to stabilize the neck and to prevent wire slippage. The wire saw should pass obliquely across the sternum between the manubrium sterni and the xyphoid cartilage to ensure making a large hole into the thoracic cavity.

After the forelimb and neck are removed, the fetus may sometimes be extracted by traction on the remaining forelimb. If the fetus is still too large, it is extracted until it is snug in the maternal pelvis, then it is eviscerated through the thoracic opening. Access

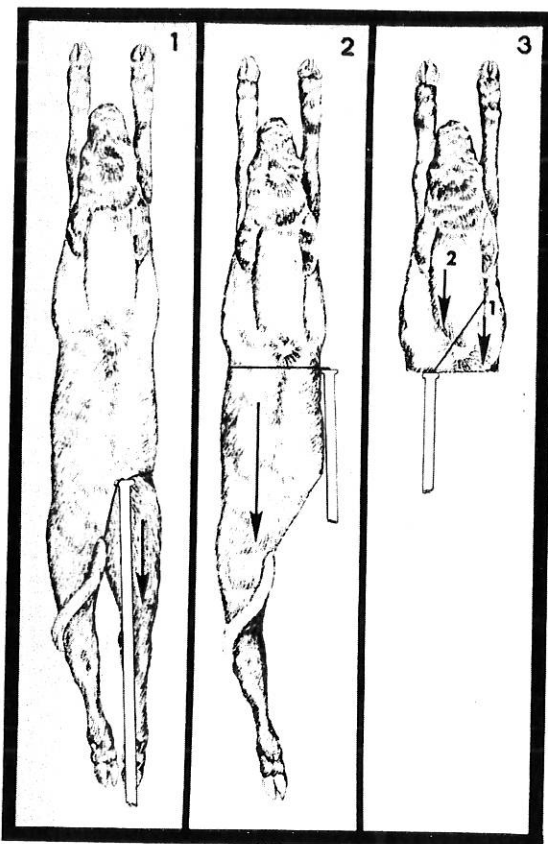


Fig 3—Steps in modified fetotomy, with fetus in caudal presentation—fetatome and wire saw placement. 1 = section of hindlimb; 2 = section of the caudal part of the fetal trunk; 3 = section of the neck and cranial portion of thorax.

to the abdominal contents is gained by manually rupturing the diaphragm. The birth canal should be guarded by hand from the cut ribs as the fetal thorax is pulled through the dam's pelvis. The opening into the thorax of the emphysematous fetus allows escape of gases that may otherwise prevent its extraction, and evisceration allows the thorax and abdomen to collapse as the fetus is pulled into the pelvic canal.

If hip lock becomes a problem, 2 alternatives are available. In one, the wire from a half-threaded fetatome is attached to the wire introducer and passed between the hindlimbs of the fetus. The fetatome head is positioned above the paralumbar fossa of the fetus, with the wire saw in the ischial arch, and the fetal pelvis is transected (Fig 2,3), or the fetus may be detruncated cranial to its pelvis and then the pelvis bisected (Fig 2,4 and 2,5).

Fetotomy, Caudal Presentation

If the fetus is in caudal presentation, with hindlimbs extended, an oblique cut is made through the fetal pelvis. The head of the fetatome is placed near the lumbar vertebrae (Fig 3,1), counterforce attachment is completed, and the wire is positioned in the ischial arch for the cut. If a hindlimb is retained in hip or hock flexion, the wire saw from a half-threaded fetatome is passed around the limb and the cut is made obliquely across the pelvis by holding the head of the fetatome lateral to the ischial tu-

berosity on the side opposite the limb being removed. If both hindlimbs are retained, the second limb often can be exteriorized following removal of the first one. If that is not possible, the second limb is removed together with the pelvis by making a transverse cut through the lumbar region.

Removal of the first hindlimb, whether presented or retained, allows for evisceration through an opening made by blunt dissection through the abdominal wall. The fetus may be extractable following evisceration. If not, the next cut is a transverse one through the thorax, just caudal to the scapula (Fig 3,2). The fetatome head is maintained in proper position for this cut by applying a counterforce chain from above the hock to the butt plate. If the trunk is too large to come into the pelvis, the half unthreaded wire saw is introduced through the hole in the flank, brought back on the outside of the fetal trunk, and threaded. The costovertebral attachments are transected on one side. This cut reduces the diameter of the torso because it can be rolled into a tube by overlapping the cut side over the fetal back. This last procedure is seldom necessary because the torso of the fetus

usually collapses as it is pulled into the pelvic inlet after evisceration.

The last cut is an oblique one through the remaining part of the thorax. The half unthreaded wire is passed around the forelimb, then rethreaded. The transected spine is fixed with a Krey's hook, the fetatome head is placed on the side of the Krey's hook opposite to the limb being removed, the shank of the fetatome is grasped together with the body of the Krey's hook, and the cut is made (Fig 3,3). The remaining parts are then removed.

If size is not reduced enough at any time with these procedures, with the fetus in either presentation, the fetotomy can be completed by the Utrecht method.¹ However, these procedures have been effective in our hands in most field cases.

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Endotoxin-induced hemodynamic changes in dogs

Plasma concentrations of thromboxane and prostaglandin I_2 (PGI_2) before and after IV injection of endotoxin and resulting hemodynamic changes in dogs were evaluated. Effects of flunixin meglumine on plasma concentrations of these prostaglandins and the related hemodynamic changes were also determined.

Shock was induced in 2 groups of anesthetized dogs. Four dogs were given endotoxin only and 4 dogs were given endotoxin and then were treated with flunixin meglumine. Arterial blood pressure (BP), cardiac output (CO), and heart rate were measured, and blood samples were collected at postendotoxin hours (PEH) 0, 0.1, 0.25, 0.5, 1, 2, 3, and 4.

Plasma thromboxane and prostaglandin I_2 concentrations were increased in canine endotoxic shock. Thromboxane concentration was highest early in shock, and appeared to be associated with an initial decrease in BP and CO. The increased concentration of prostaglandin I_2 was associated with systemic hypotension at PEH 1 to 2. Treatment of dogs with flunixin meglumine at PEH 0.07 prevented further increase of thromboxane and blocked the release of prostaglandin I_2 , resulting in an increased CO, BP, and tissue aerobic metabolism.—G. D. Bottoms, M. A. Johnson, and O. F. Roesel in *Am J Vet Res* 44 (Aug 1983): 1497.

Immediate Postpartum Care of the Dam

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The relief that accompanies the delivery of a calf following dystocia should not divert attention away from the cow, which should be the object of primary economic concern to the herdsman or rancher. Parturition is both a culmination and a beginning. The postpartum cow is in metabolic transition between the demands of late pregnancy and ensuing lactation.

Mismanagement of added stress caused by regrouping, diet change or sequelae to dystocia can spell the difference between profit and loss. The veterinary obstetrician bears responsibility for evaluation of the dam and for initiation of therapy when it is needed to maximize subsequent fertility, lactation or, at least, salvage value.

Trauma to the Birth Canal

Assisted delivery increases the risk of trauma to the soft tissue of the birth canal. This seems obvious in cases of fetal/maternal disproportion with normal presentation, but the mere fact that the calf is aided with traction rather than pushed out elongates and constricts the vagina (consider the principle of novelty store "Chinese handcuffs"). Assisted delivery may also deny the cervix, vagina and vulva the aid in dilation caused by the progressive pressure of the calf. All assisted deliveries, even with the absence of hemorrhage, deserve an immediate, clean, manual examination and systematic evaluation of the uterus, cervix and vagina.

The vulva and perineum should be cleaned with soap and water. The examiner should assure the cleanliness of his own arm and with adequate lubrication should examine, by progressing inward, the entire circumference of the birth canal. While knowledge of the obstetric procedures performed can guide the thoroughness of the examination, the possibility of trauma from prior assistance should be kept in mind. The presence of a remaining calf should always be ruled out. Soluble oxytetracycline powder (Vetquamycin 25, Rochelle Laboratories, Inc., Long Beach, CA 90801) in large gelatin capsules provides some measure of antibiotic protection against the contamination inherent in any manual exam. The number of boluses used should reflect the depth and duration of manipulations and degree of sepsis. An attempt should be made to place the boluses between the membranes and endometrium, lest they be expelled rapidly by the contracting uterus. Intramuscular oxytocin at a dose of 60 to 100 IU (3-5 ml), depending on the size of the cow, will aid in contracting the uterus and in expelling the fetal membranes.

The management and prognosis for trauma to birth canal depends on location, severity and existence of sepsis. Thus, a small dorsal uterine laceration sustained during delivery of a fresh calf may be managed with oxytocin and systemic antibiotics and have a favorable outcome. Conversely, comparable vaginal lacerations or bruising during fetotomy of an emphysematous fetus may result in severe perivaginal cellulitis and pelvic inflammation, with death occurring in a stressed, toxic cow not receiving aftercare.

The vulva is frequently lacerated in first calf heifers even during unassisted delivery. These lacerations are generally dorsal and heal without consequence. Lateral laceration of the labia can occur and may, if severe, be sutured to restore vulvar function.

The vagina may suffer longitudinal lacerations during assisted delivery of large or an inadequately lubricated calf. The caudal vaginal wall frequently splits when a wave of perivaginal fat preceding a large calf meets the vestibulovaginal sphincter. This fat may prolapse following delivery and needs to be differentiated from any other abdominal structure that may have prolapsed through a more cranial vaginal defect. Lacerations of the vagina are generally left unsutured, as healing by first intention is rarely achieved. Concurrent systemic antibiotic therapy may lessen perivaginal cellulitis and abscessation. Prolapsed fat can be manually removed or trimmed without serious hemorrhage.

The effaced cervix at parturition is subtly hoop-like on manual examination per vaginam. The cervical rings are reduced to ripples of mucosa at the junction of the smooth vaginal epithelium with the endometrium, which is identified by the presence of caruncles. Interfering fetal membranes should not cause confusion in the detection of myometrial defects, which may be longitudinal or circular, paralleling the dilated cervix. Simultaneous examination per vaginam and per rectum may be necessary. Defects diminish in size as the uterus contracts; therefore, examination should be performed prior to administration of oxytocin.

Uterine laceration may have resulted from mutation of limbs with inadequate uterine relaxation or lubrication, or from injudicious use of fetotomy instruments. Cows with long-standing dystocia with a dry fetus and contracted uterus or fetal emphysema with metritis and severe uterine distention are at increased risk of uterine rupture.

Uterine body and cervical lacerations may be sutured per vaginam with a hand-held Loopuyt's needle and catgut. Any enhancement of apposition will aid the fibrin seal, which, with systemic broad spectrum antibiotics, is the real salvation of the cow. Lacerations in the uterine horn may require an abdominal approach via an inguinal or caudal midline incision. Alternately, the uterus may be manually prolapsed during the 12 hours immediately postpartum following slow intravenous administration of 10 ml of 1:1000 USP epinephrine, which causes temporary complete myometrial relaxation. Caruncles at the tip of the horn of pregnancy are grasped per vaginam and gently withdrawn, producing inversion. Prolapse is aided by the cow's straining as the uterine mass enters the vagina. This procedure should not be attempted following epidural anesthesia or oxytocin administration.

Hemorrhage

Vaginal lacerations are a frequent source of acute hemorrhage. Some fresh blood is always present at the vulva owing to rupture of the calf's umbilicus. Bleeding can also be caused by laceration of the cervix or uterus, forcible separation of membranes or laceration of a caruncle during fetotomy. Hemorrhage from the deeper structures may not be evident at the vulva but noticed only after manual examination. Bleeding from a ruptured broad ligament may be intra-abdominal and therefore occult unless a peritoneal tap is performed. The source of serious hemorrhage should be determined. Large vaginal bleeders may be ligated, or a hemostatic

forceps may be left in place for 24 to 48 hours. Bleeding of uterine origin may be decreased by administration of oxytocin. The cow's membrane color and respiratory and heart rates should be noted to follow the progress of hypovolaemic shock. Whole blood transfusion may be indicated.

Evaluation of Other Body Systems

A rapid but systematic appraisal of the dam following obstetric intervention will often reveal problems in other body systems, which can seriously alter the outcome of the case. At the very least some appraisal needs to be made of the musculoskeletal system and, particularly in dairy cows, the udder, for evidence of mastitis.

Within a half-hour postpartum the cow should be encouraged to rise. Footing should be good and assistance available at the tail, if needed. The cow should be assessed for strength and proprioception (knowing the location and position of feet and legs) in the hind limbs. Weak animals may have some degree of paralysis due to nerve damage. Hypocalcaemia could be a concurrent problem and should be suspected if the cow is unable to rise, especially if the dystocia was caused by ineffective labor. Cows with weakness are at an increased risk for further injury, such as a hip luxation or rupture of the gastrocnemius muscle.

Cows unable to rise need nursing care. Crush syndrome of the down side rear limb ensues rapidly if the animal is not well bedded and turned side-to side at several hour intervals. Food, water and shelter should be provided. Affected cattle need frequent reevaluation, as injury such as hip dislocation often occurs following paresis. The hind legs may be hobbled to prevent spreading beyond one meter. If considerable improvement has not occurred after parturition, recovery is unlikely in the first two weeks.

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Resuscitation and Intensive Care of the Newborn Calf

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In dairy practices, the death of a calf within minutes of birth is a well-known occurrence. Data collected on Michigan dairy herds has indicated that approximately 6 percent of calf losses occur at the time of birth.

Large animal practitioners often work hard to correct a complicated dystocia only to have the calf die before establishing adequate ventilation. When delivered, these calves are usually fully developed, have normal reflex responses and have strongly beating hearts. Typically, attempts to clear the airway and stimulate breathing are not successful in saving the calf. Some of the calves that die at birth can be saved if proper resuscitation procedures are followed. The techniques are simple and easily performed without expensive equipment. Although many factors may prevent success, there are several reasons for always making an attempt at resuscitation.

First, many calves asphyxiate because of a failure to establish adequate ventilation. Since endotracheal intubation is an easily learned skill, no animal should ever die of primary ventilatory failure without an attempt by the veterinarian to perform intubation and to assist in ventilation.

Second, attempting to intubate and to ventilate all dying newborn calves will improve the skills of the practitioner and will increase the frequency of successful resuscitation. The few minutes required to prepare and administer emergency procedures will be many times repaid by the positive professional image created by the attempted and successful resuscitations.

This article will discuss the physiology of the near-term fetus, the changes that occur during parturition and the steps that can be taken to support weak and dying calves. These physiological principles and the resuscitation techniques can be applied to all mammalian species.

Intrauterine Physiology

In order to help a newborn calf establish respiratory and circulatory independence, it is essential to understand fetal and neonatal physiology and anatomy. Tremendous changes occur in both of these organ systems at the moment of birth. The events that occur are a carefully developed cascade of steps. Each event must occur in order to allow the next step to proceed.

During intrauterine development the nutrients required by the fetus are provided by the maternal circulation through the placenta. Nutrients, including oxygen, are carried from the

placenta via the umbilical veins. The umbilical veins deliver the oxygenated blood into the caudal vena cava by means of the ductus venosus. The blood from the postcava is primarily diverted through the foramen ovale and into the left atrium. Blood from the fetal head and right atrium enter the right ventricle. Blood leaving the right ventricle flows into the pulmonary artery, but because of high pulmonary vascular tone, the majority of the blood does not perfuse the lungs. Instead, the blood passes into the aortic arch via the ductus arteriosus.

The circulatory pathway effectively delivers blood that is high in oxygen and nutrients to the fetal heart and brain. The lungs that are unable to oxygenate the blood are perfused with only enough blood to meet the needs of the pulmonary tissue. At parturition, this circulatory pattern must change if the calf is to survive in the extrauterine (outside) environment.

The partial pressure of oxygen in the fetal blood is lower than that required by adult animals. This relative hypoxic condition is compensated for by an increased affinity for oxygen by fetal hemoglobin and by an increased ability to release oxygen to the tissues.

In the lungs, the lower oxygen tensions cause a constriction of the vasculature. This contracture produces high pulmonary vascular resistance and prevents blood flow through the nonventilated lungs. This phenomenon is called *hypoxic vasoconstriction* and remains as a characteristic of the pulmonary vasculature throughout adult life.

Although the fetal lungs are not involved in the elimination of carbon dioxide and the absorption of oxygen, they serve several special functions. The lungs produce amniotic fluid to cushion the fetus and they act as an important glycogen reservoir during late pregnancy. As the end of fetal development approaches, the lungs begin to produce the surface-active substance that will be required to stabilize the air-inflated lungs. This substance is called *surfactant* and is an essential requirement for alveolar stability in the newly inflated lungs of the newborn.

The bovine lung is highly lobulated and lacks collateral channels for ventilation. The calf must inflate each lobule of its lungs independently of the adjacent lobule. This lack of interdependence may predispose the calf to greater problems with *atelectasis* if small airways are occluded.

Near parturition, the fetal lung becomes primed for the initiation of breathing. The muscles used for respiration contract and relax as the functional development of the respiratory center reaches maturity. The ventilatory efforts are weak because of the mild stimuli that reach the calf through the intrauterine environment. Another indication of fetal lung maturity is the lung size. The fetal lung becomes filled with surfactant-rich fluid to a volume comparable to the functional residual capacity of the inflated neonatal lung. This ensures that the lungs will have sufficient size to sustain the newborn immediately following birth.

Parturition-Induced Changes in the Neonate

The birth of the fetus causes the separation of the fetal life line and initiates the sequence of events necessary for the independent life of the neonate. During the birth process, the calf is forced through the birth canal. Umbilical blood flow may become reduced, resulting in lower oxygen tension and higher carbon dioxide tension in the fetal blood. Central and peripheral chemoreceptors become activated with changes in the gas tension and blood pH resulting in secondary stimulation of the respiratory center of the brain. Additionally, ventilation is stimulated by the change in ambient temperature and by the tactile stimulation caused by the new environment. All of these factors are normal respiratory center stimuli.

The first breath marks the end of fetal life and the beginning of the postnatal period. Lung inflation, in response to the numerous strong stimuli, must occur within a few minutes of placental separation. Measurements of the effort needed initially to inflate the fluid-filled fetal lung have shown that intra thoracic negative pressures in the range of -60 to -80 cm H₂O are required. Once air is retained within the lung and the fluid is absorbed, the effort of breathing is reduced to the range of -8 to -12 cm H₂O pressure.

The inflation of the lungs is the first and the most important step of the cascade. If ventilation occurs spontaneously, the calf will quickly be able to sustain itself. With air in the lung, the pulmonary vascular resistance will rapidly decrease as the vessels dilate in response to the increased oxygen and the decreased carbon dioxide tensions. *The reduction in pulmonary vascular resistance and the concomitant increase in pulmonary blood flow are the second step of the cascade.* Vasoactive kinins, which are released from the aerated lung, further cause the *pulmonary vessels to dilate* and also cause *constriction of the ductus arteriosus and umbilical vessels.*

As the resistance to blood flow into the lungs decreases, right atrial pressure also decreases. Simultaneously, increased blood flow into the left atrium and increased systemic vascular resistance due to constriction in the umbilical arteries cause *functional closure of the foramen ovale.* At this point, the newborn calf has switched from the fetal circulatory pattern to the independent pulmonary and cardiovascular systems of the adult.

Newborn Respiratory Distress of the Calf

Full-term, normally developed calves may exhibit signs of respiratory distress immediately following birth. Some of the causes for this syndrome have been identified. As indicated previously, the respiratory center of the brain is stimulated by changes in the blood's oxygen and carbon dioxide levels. Dystocia can cause the calf to become severely hypoxic and acidotic. With severe hypoxia or acidosis the ability of the calf to respond and to initiate ventilation diminishes until the calf is unable to generate the large negative pressures needed to displace the lung fluid. Weakened calves are often delivered following dystocias or when cesarean sections have been performed because of the large calf size.

Another cause of weak, ineffectively ventilating calves is the breech presentation. It is believed that placental blood flow becomes disrupted while the calf's head is still within the uterus or vagina. When delivery is prolonged, the blood gas changes become severe, resulting in the unresponsive weakened calf. Any factor that causes disruption of placental blood flow during the birth process will lead to fetal depression.

In humans, respiratory distress syndrome of the newborn is associated with immaturity of the lung. This is most frequently a problem with premature infants. The immature infant lacks sufficient amounts of surfactant in the lung. This deficiency causes the alveoli to be unstable. In order to sustain these infants, they are intubated and maintained on mechanical ventilators until the lungs produce enough surfactant to remain inflated. Some apparently full-term calves may be born lacking surfactant. The initial resuscitation procedures will be the same but continued mechanical ventilation will be required. This type of intensive neonatal support is beyond the scope of this article and of most veterinary hospitals. These calves not only fail to initiate adequate ventilation, but they also die if ventilation support is terminated.

Resuscitation Procedures

As soon as the fetus has been delivered, a series of evaluations need to be performed. If the calf begins to fail in its conversion from a fetus to a neonate, resuscitation must be instituted immediately. After initially cleaning the face, nares and oral pharynx of mucous and fetal membranes, the heart rate, mucous membrane color and respiratory efforts should be evaluated. The results of the initial evaluation will be quite variable, depending on the vitality of the calf, but it is important to establish the basis for future evaluations.

Calves with weak or slow heart beats and pale mucous membranes and lacking any ventilatory effort should be immediately resuscitated. If ventilation is shallow, erratic or labored, the calf should be closely monitored and the veterinarian should be prepared to intubate the calf. Strong vigorous calves should be rechecked every 30 seconds and intubated if respiratory distress begins to occur. The success of the resuscitation is dependent upon early support before cardiovascular failure occurs.

The first step in ventilatory support is to place an endotracheal tube into the trachea. Attempts to inflate the lungs by blowing through the calf's nose or by using a mask will result in air filling the stomach, since the resistance to stomach inflation is less than the resistance to displacement of the lung liquid.

In order to quickly and atraumatically intubate a calf, it is important to understand the anatomy of the upper airway. The bovine larynx is dome shaped and easily moveable. The rounded edges of the arytenoid cartilages allow the endotracheal tube to slide into the esophagus. This is especially true if the alignment of the larynx and the endotracheal tube is not straight. The calf's head should be positioned so that the tip of the nose is in a straight line with the thoracic inlet. Extension of the head will straighten the airway. Care must also be taken to keep the head and chest in the same plane. Intubation around a bend is usually difficult to accomplish. Placement of the calf in sternal recumbency with the head pulled upward provides the best visualization for intubation. This position does require an assistant to hold the calf. Intubation

of the trachea can be performed with the calf in lateral recumbency. The head and neck must still be extended to straighten the airway.

Blind intubation in the calf is more difficult than intubation done while visualizing the laryngeal opening. For this reason, it is recommended that a simple laryngoscope be used to ensure rapid, atraumatic placement of the endotracheal tube. An equine sweat scraper can be used as the laryngoscope blade. The aluminum scraper should be straightened slightly and then covered with Elasticon tape to prevent the sharp edges from cutting the delicate tissues of the calf.

Several light sources are available to illuminate the larynx during intubation. A long-shaft pen light provides the best illumination without blocking the clinician's visualization of the larynx. Summit Hill Labs (P.O. Box 1, Avalon, NJ 08202) and Concept Inc. (21707 US 19 South, Clearwater, FL 33516) each sells this type of light. The lighted tip of the laryngoscope blade should be placed on the base of the tongue. The epiglottis and larynx will be seen immediately in front of the light.

The endotracheal tube is passed through the mouth and placed on top of the epiglottis. Gentle but forceful advancement of the tube will place the endotracheal tube tip against the larynx. Often, the endotracheal tube will slide into the trachea without resistance, but occasionally gentle pressure and rotation are necessary for the tube to separate the vocal folds and to enter the trachea.

To facilitate manipulation of the tube, a rigid wire stylet inside of the endotracheal tube is necessary. Stylets can be made out of an aluminum splint rod or coat hanger wire. A rubber stopper should be used on the stylet to prevent the wire from extending beyond the cuffed end of the tube.

Most calves have tracheas large enough to accommodate a 7 mm ID (inside diameter) endotracheal tube. However, very small calves may require a 5.5 mm ID tube. Both size tubes are inexpensive and available from many sources.

Following intubation, the calf should be ventilated. Inflation of the fetal lungs will be difficult until the lung liquid is absorbed through the alveoli. Several commercial ventilators are available and are described in this book in the section on resuscitation of foals. However, it is possible to inflate the lungs of the neonate by blowing through the endotracheal tube. This is often the only readily available source for positive pressure ventilation.

Most full-term calves will readily convert to the neonatal circulatory pattern following several deep breaths. A few calves will require sustained ventilatory support for the lungs to remain filled with air. Once the calf begins to breathe on its own and is able to move a sizable forceful volume of air through the endotracheal tube, ventilatory support can be stopped. The endotracheal tube should remain in place until the calf strongly objects to the presence of the tube. Swallowing, coughing and the ability to hold its head up are signs that the endotracheal tube is no longer needed.

Once intubation and lung inflation have been accomplished, additional methods of postpartum care can be utilized. Since heat loss can rapidly occur in newborns because of evaporation and convection, care should be taken to keep the calf warm and dry. Hypothermic infants are unable to respond to normal amounts of stimulation and frequently will not suckle or breathe adequately.

Dextrose-containing fluids can be given intravenously to the calf to provide a readily available energy source. Forced feeding of calves that lack strong reflex responses may result in pulmonary aspiration of the colostrum. Furthermore, if the calf is hypothermic, its ability to digest and to absorb milk-based nutrients will be markedly slowed.

In summary, weak calves should be intubated and ventilated to facilitate the conversion from the fetal to the neonatal circulatory pattern. Newborns must be kept at their normal body temperature and may require intravenous dextrose fluids for energy support.

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Health and Management of the Nursing Calf

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Proper nutritional and health management of the nursing calf is important not only because it results in the most pounds of beef to sell at weaning, but also because it sets the calf up for achieving optimal postweaning performance.

Prenatal Nutrition and Health of the Cow

The health and well-being of the nursing calf starts with the health and nutritional status of the cow prior to the birth of the calf. Nutrient needs of the cow increase during the last trimester of gestation, and by the last month prior to calving, the fetus is gaining approximately 1 pound per day. In addition to this late-term fetal growth, the cow is also preparing for lactation. Research has shown that cows who are thin (body condition score ≤ 4) have a decreased concentration of immunoglobulins in the colostrum compared with cows in a body condition score of 5–6 (1–9 scale).

Additionally, calves born to very thin cows may be weak and slow to nurse, also reducing the colostrum they consume and making them more susceptible to disease. For more information on the nutrient needs of the cow, see NebGuide G2268, *Supplementation Needs for Gestating and Lactating Beef Cows and Comparing the Prices of Supplement Sources*. For assistance determining body condition score of beef cows, see Extension Circular EC281, *Body Condition Scoring Beef Cows: A Tool for Managing the Nutrition Program for Beef herds*.

A Healthy Start for the Newborn Calf

In addition to a healthy mother, the newborn calf needs a clean environment. Healthy adult cattle often shed very low levels of bacteria and viruses that can cause scours and other diseases in calves. Manure and mud provide an ideal environment for disease-causing bacteria and viruses. Early in the calving season, calves are exposed to these pathogens and often develop minor, undetectable infections; however, these young calves amplify the pathogen load in the environment faster than do adult cattle. As the calving season progresses, newborn calves are challenged with increasingly higher levels of pathogens. Infection with high doses of bacteria or viruses combined with other risk factors like overcrowding, temperature extremes, and precipitation can quickly overwhelm calves' defenses, and clinical cases of scours may develop. Long calving seasons further exacerbate the situation by providing a steady supply of new calves that are susceptible to infection over a long period of time. These conditions often lead to scours outbreaks, which cause great financial loss due to increased labor, reduced weight gain, and even calf death loss.

Extension veterinarians at the University of Nebraska–Lincoln have found that segregating cow/calf pairs by the age of the calf has helped reduce the incidence of scours outbreaks. When possible, calving areas should be divided into large lots. After a week to two weeks of calving, cows that have not calved should be moved to the clean area.



Figure 1. Ideally, a cow in good body condition gives birth to a vigorous calf and quickly stimulates the calf by licking it clean. (Photo: Groves Ranch)

This process should be repeated every week to two weeks so that the older calves are not housed with the younger calves, as it is the older calves that infect younger calves.

After the youngest calves are 4 weeks old, all calves can be comingled. Managing breeding to achieve a short, intense calving season enables optimal management by reducing the number of lots needed to calve out a herd as well as by shortening the time susceptible calves can be born, become infected, and further contaminate the environment.

The timing and the amount of colostrum consumption is critical for the newborn calf as well. Ideally, newborn calves need to stand and nurse within the first few hours of life to maximize colostrum absorption and immunity. The best case scenario occurs when a cow in good body condition gives birth to a vigorous calf in a clean environment and promptly stimulates the calf by licking it clean. Vigorous calves quickly nurse a large colostrum meal. The first meal a calf consumes is one of the most important meals of its life because a sequence of gut changes begins with that meal. The intestines of a newborn calf have the ability to carry their contents across the wall of the intestine directly into the blood. This ability is critical to move colostrum antibody from the intestines into the blood; however, the process is not selective for antibodies and can allow pathogens into the blood as well.

To protect the calf from pathogens, the gut begins to “close” (loses its ability to take contents directly across into the blood) as soon as the calf’s first meal is introduced to the intestinal tract. As a result, less and less antibody can be absorbed from each subsequent meal until gut closure is complete. A common misconception about the window of

time available to deliver colostrum to calves is that a calf always has 24 hours. In reality, the window depends on when the calf first consumes any kind of meal. If a calf has nothing to eat, it can still absorb some antibody at 24 hours, but if the calf consumes anything, gut closure begins immediately and can be complete before 24 hours has passed. Unfortunately, “anything” can be a dose of colostrum that is too small, milk replacer, or debris nursed from a dirty udder or environment. All of these meals will initiate gut closure and decrease the amount of antibody absorbed.

Worse yet, bacteria nursed from a dirty environment, including the udder, can be directly absorbed into the blood and cause severe disease. Good passive transfer is accomplished most effectively by implementing preventive management strategies. These include maintaining adequate BCS in cows, providing a clean calving environment, and routinely observing calves to make sure they have paired up and nursed. Calves born following prolonged labor, especially if the calf had to be pulled or removed via caesarian section, are at high risk for failure of passive transfer. Calves born to first-calf heifers are more vulnerable as well. Close observation of these pairs can allow early intervention to maximize passive transfer.

Interventions depend on producer preferences as well as recommendations from local veterinarians. In cases where the calf cannot nurse colostrum from its own dam, the following options are available:

1. Milk out dam and feed with bottle or tube feed.
2. Feed colostrum banked from other cows that have lost their calves.
3. Feed commercial colostrum replacement product.
4. Feed colostrum banked from neighboring herds (beef or dairy if available).
5. Feed colostrum supplement.

These options are listed in order of decreasing efficacy and increasing risk of introducing new diseases. Resident cattle have the ideal colostrum for their own calves because the antibodies match the pathogens present in the herd and the surrounding environment. Disease transmission is also less likely if colostrum from within the herd is used.

Commercial colostrum replacers are available, are effective, and come from carefully tested herds. These products are usually costly and must be mixed carefully according to the provided instructions. Feeding colostrum banked from neighboring herds can be effective, but dramatically increases the risk of introducing diseases into a



Figure 2. Many ranchers provide a shed or other shelter for their calves to reduce environmental stresses. (Photo: Groves Ranch)

producer's herd. Colostrum supplements are relatively safe in terms of disease transmission but typically do not contain a high enough concentration of antibody to guarantee adequate passive transfer. Visit with your local veterinarian when considering these options.

Regulating body temperature can be a challenge for the very young calf. Depending on the time of year a calf is born and the current weather conditions, the challenges can vary. Producers need to be aware of what those might be and plan accordingly. Calves born in cold and wet conditions may need a dry, warmer place to acclimate. Initially, this may be a barn with the dam. However, many ranchers have devised innovative ways to make a shelter or hutch that calves can access without their dams to reduce cold and wind stress. Conversely, calves born in the summer months may need access to shade and plenty of easily accessible water to combat heat and humidity.

Feed and Water for the Young Calf

While milk intake is very important to the nutritional status of the young calf, it is not the only source of nutrition that must be made available. When a calf nurses, the esophageal groove closes and the milk bypasses the rumen and enters the abomasum. This allows this highly digestible nutrient source to be used for skeletal and muscle growth of the calf rather than be used by rumen bacteria. However, it is critically important that the young calf begin rumen development, which requires both a solid food source, such as grass or hay, and water. Young calves start to nibble at feed within the first week or so of life. Research has shown they are eating 1 percent of their body weight by the time they are 3 months old. The development of the rumen is

critically important for both preweaning and postweaning weight gain. Therefore, making sure easily accessible, palatable feed is available to the calf as early as possible is important.

Water is an important nutrient often overlooked, even in adult animals. For the young calf it is critically important for both health and rumen development. Water intake and dry matter intake are highly correlated. Beef calves that start feed consumption early will gain weight quickly and tend to thrive. Iowa dairy researchers found that dry matter intake explained more than 60 percent of the variation in free choice water intake of bottle-fed dairy calves.

Dairy industry researchers also determined that when temperatures increase, water intake increases exponentially rather than linearly in a nursing calf. Careful attention should be given to water access for calves, especially when daily temperatures begin to increase. Producers should consider pushing dirt up around tanks that have blown out to ensure calves can reach the water. Additionally, they should evaluate the water flow into the tank. If the cows drink the tank down to a level below what the calf can reach, how quickly does the tank refill? If the producer has small automatic waterers, then observing whether the cows are pushing calves away from the water source is important. If this is happening, the calves may need an additional water source in a creep area where cows cannot reach it.

Calfhood Vaccinations and Other Management Practices

Calves should be processed early in life to maximize growth and minimize disease and stress. Processing can include vaccination, castration, use of growth implants, and branding. Vaccination is an important tool to prevent disease in calves before and after weaning. Early vaccination, even in calves as young as a week old, has been shown to generate protective immunity. Each producer's vaccination program, including the products and timing of vaccinations, should be built with the help of a local veterinarian who understands disease pressure in the area, vaccine use and efficacy, and the producer's goals.

Castration, and if necessary, dehorning should be done as early as possible. Complications associated with castration increase dramatically as calves get older and heavier. Many studies have shown that calves castrated early in life will achieve the same or higher weaning weights as calves castrated later because the detrimental impacts of late castration far outweigh any increase in growth associated with maintaining bull calves intact. Castration near the

time of birth is ideal and should be completed in the vast majority of herds by the time calves are 2 months old. Pain management should be discussed with a local veterinarian, especially if late castration is unavoidable.

Growth implants in nursing calves have been shown to increase weaning weights by 15 to 30 pounds. If given once at approximately 30 days of age, they have not been shown to negatively impact reproductive performance in heifers. Implants should not be given to bull calves intended for reproduction. Steer calves should be at least 30 days old when given the first implant. Calves should be given a growth implant approved for nursing calves, and all label instructions should be followed regarding proper ear placement, and hygiene of the implant needle.

Preconditioning Calves for Weaning

Good nutrition is a key component of a good immune system. Therefore, nutritionally preparing calves for a stressful experience such as weaning is as important as a good vaccination program. Zinc and copper are minerals that play an important role in immune function. Therefore, prior to weaning calves should have access to a good trace mineral that supplies those nutrients. As discussed earlier, feed that allows for good rumen development is important prior to weaning since the calf is going to transition to a milk-free diet. When possible, calves should be fed a diet of high quality, long stem hay or allowed to graze grass in an area where they become familiar with the water source before the cows are removed. When practical, introducing calves to supplements containing coccidiostats (monensin, lasalocid, and decoquinate) prior to weaning can help calves transition to bunk feeding and prevent clinical coccidiosis. Keeping things as common and familiar as possible when

cows are removed helps reduce stress. For more information on weaning procedures see NebGuide G2057, *Management, Health, and Nutritional Considerations for Weaning Calves*.

Processing, either before, or at the time of weaning can reduce disease risk and increase growth performance of calves. As is the case with early vaccination, a protocol should be designed to match the characteristics of the producer's herd as well as augment calf value in the post-weaning phase of development. Coordination between the producer, the local veterinarian, and, when possible, the stocker or feedlot producer taking possession of the calves postweaning is important for planning vaccination programs to match disease risk and desired outcomes. Other processing events should also be considered at this time. Antiparasitic treatment is important in calves because they tend to be more susceptible to parasites than older cattle. Internal parasite burdens not only decrease feeding efficiency, but also have been linked with decreased immune function. Reimplanting at preconditioning/weaning can be valuable and should be considered.

Conclusion

Providing proper nutrition, a clean environment, and proper vaccination protocols prior to and after calving helps ensure a healthy, thriving calf crop at weaning. Understanding the importance of a good source of colostrum, the timing of delivery, and the development of gut function in the newborn will help producers give their calves a healthy start. Developing and executing a management protocol with a local veterinarian and extension personnel will help guard against local pathogens and assist in setting calves up for preweaning and postweaning success.

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COLOSTRUM

“THE BEST INSURANCE FOR A LIVE HEALTHY CALF”

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Making certain each calf that is born alive remains alive is one of the “critical success factors” for a cow/calf enterprise. The most important step that can be done to insure life, health and productivity of each calf is to make absolutely certain that it receives adequate amounts of good colostrum within the first hours following birth. The focus of this paper will be to briefly review the important points related to colostrum and the newborn calf.

DISEASE ASSOCIATED WITH FAILURE TO OBTAIN ADEQUATE COLOSTRUM

Mortality of calves is related to inadequate transfer of passive immunity from colostrum (Table 1). Postnatal disease resulting from inadequate colostral immunity has been divided into two major clinical categories. Septicemia is the invasion and rapid spread of microorganisms (usually coliform bacteria in calves) through the blood, to all tissues of the body. Septicemia is usually related to complete or nearly complete lack of colostral antibody uptake by the calf. The disease is highly fatal in spite of treatment and death usually occurs at an early age.

The second, more insidious form of inadequate colostral intake is that of calf scours. This broad term reflects the most common sign, diarrhea that is observed in the sick calves. Calves may suffer from excessive fluid and electrolyte loss, varying degrees of intestinal damage, endotoxemia from bacterial toxins produced in the gut, and a more limited bacteremia with localization and infection in one or two organs of the body. Because of the wide array of clinical signs and time of occurrence, it is easy to lose sight of the fact that the basic cause is related to a common but simple management failure, to obtain adequate colostrum. Although many of these affected calves are seen shortly after birth or within a few days of age, conditions such as “joint ill” may not be observed until several weeks or months of age and result in severe lameness or even paralysis, if it involves the spinal vertebrae. An increase in respiratory disease such as pneumonia has also been shown to occur as a result of inadequate passive immunity.

Other conditions associated with inadequate colostrum are more difficult to detect. These may be associated with micro nutrient deficiencies such as vitamins and minerals or the major nutrients, protein/energy malnutrition. Once calves have burned up body stores of energy it is essential that they keep receiving a high quality nutrient source from the dam. If they miss colostrum within the first few hours of life they become high risk calves.

THE IMPORTANCE OF COLOSTRUM FOR THE NEWBORN CALF

When a calf is born it is highly susceptible to many infectious organisms in the surroundings, to which other animals are immune. More than 70 years ago, scientists demonstrated the fact that

the newborn calf is not capable of responding to the challenge of many relatively common organisms carried and shed by other animals in the same environment, unless they receive adequate colostrum. This has been explained primarily by a lack of antibodies that the mother provides for her calf through nursing first milk commonly known as colostrum,. It is called passive immunity because the calf receives antibody that was developed outside of its body (by the dam) rather than by the calf itself. The calf is born without the ability to completely respond in an active manner to challenges by outside organisms due to immaturity. It also requires days or weeks to mount an active response to a disease challenge. Although the entire process seems simple enough, in actual fact the series of events necessary for the calf to obtain high quality colostrum often fails.

Some of the contents in colostrum are listed in Table 2. We know that milk contains high quality nutrients needed for growth but colostrum contains many additional factors essential for the calf's health. There is approximately five times as much protein, most of which is antibody (immunoglobulin) and other immune related protein such as complement, interferon, lactoferrin, and lactoperoxidase. Several important vitamins are deficient in the new calf at birth and are available only after the calf receives the dam's colostrum in adequate amounts. There is twice as much fat (energy source and milk laxative), calcium, phosphorus and magnesium. Through colostrum, the calf receives a very important survival package from its mother and if it is to be valuable it is needed immediately after birth.

WHY DOES THE SYSTEM FAIL?

Following a normal birth, the cow rises and begins to lick the calf and encourage it to rise and nurse. This should occur within the first hour after birth. All of the important ingredients in colostrum are received in a short time and nearly simultaneously as the calf enters its new environment. Therefore, the calf is off to a healthy start. Unfortunately, there are a number of barriers where the natural system can go wrong. This may happen in a very high percentage of the calves if the herd is malnourished or only a portion of the calves, such as those experiencing dystocia. When failure occurs these calves end up with little or no colostrum and become a high risk for the producer. By far, the majority of calves that develop serious diseases are those that fail to develop a solid colostral immunity.

There are a number of complex physiological processes that underline this seemingly simple series of events. The dam produces colostrum during the last few weeks of pregnancy and stores the colostrum in the udder. If the cow is not in good body condition or is being fed a deficient ration the colostrum she produces will be deficient and her calf may lack vigor (Table 3). If her first milk is lost for any reason, such as another calf happens to suck the cow prior to her own calf, the colostrum will not be replaced and the offspring will be deficient. If the dam has mastitis, the quality of colostrum from that quarter will be adversely affected.

The next phase of the process where passive transfer of colostrum can fail is related to the management of calving within the herd. If the dam or first calf heifer experiences calving difficult, the prolonged delivery and resulting stress upon the dam and calf results in decreased

mothering ability, a weakened calf that frequently fails to nurse adequately, and lower passive immunity (Table 4).

Another category for failure of the system is when the calf fails to utilize colostrum even if obtained. The calf may become chilled or stressed due to injury or any number of causes and after laying quietly following birth, its body temperature drops. Following this, if colostrum is fed, normal absorption may be depressed because of decreased blood flow to the digestive system. Also, within this category another major cause of failure is related to administering colostrum too late. If a disease process starts before the calf receives colostral protection its value is minimal. The amount of colostral antibody the calf can absorb from the gut decreases from good to poor during the first 24 hours of life. If the calf is near 24 hours of age before receiving colostrum it will likely absorb very little immunoglobulin. Supplemental colostrum should be given within two hours of birth if possible. Any additional recommendation must be “the earlier....the better!”

Although there have been other documented causes for the lack of proper colostral utilization by the newborn calf these cover the majority of common situations. The important fact to keep in mind is that nearly all of the causes of failure of the calf to receive and utilize colostrum can be overcome by good management.

GOOD CALVING MANAGEMENT RELATED TO COLOSTRUM

Proper year round herd management is essential for a successful calving program. This pertains to adequate nutrition and selection within the herd to prevent excessive calving problems and to wise calving management related to the delivery of a live vigorous calf when assistance is necessary. This should be followed by insuring that plenty of rich colostrum is obtained by each calf immediately following birth whether assisted or unassisted. Table 5 demonstrates the reduction in protein and total solids over 36 hours following milking at 6 hour intervals. This information documents that providing second or third milk to the calf instead of first milk does not accomplish the same task. In fact, by 36 hours the composition is nearly identical to that of milk in later lactation.

Two common questions are, what is good colostrum and how much should a calf receive? The answer to the first question is relatively simple. Good colostrum is golden colored and varies from a thick material that will not flow, to a heavy syrupy consistency. As a general rule, the thicker the colostrum, the higher the antibody concentration and the less volume required for the calf since thickness nearly always varies with volume. Most low to moderate milk producing beef cattle, especially first calf heifers produce low volumes of concentrated colostrum and it is important that the calf receive the entire first milking obtained. When the volume of first milking exceeds two quarts, it is best to administer this first milking in several feedings. As a general rule, calves absorb approximately one-half of the immunoglobulin in colostrum when it is administered in large amounts, although the calf is probably more efficient in obtaining antibody if it suckles small amounts over the first 24 hours. In any case, the calf requires a minimum of 1 gram of immunoglobulin for each pound of body weight. Table 4 indicates an average of 1062 milliliters of first milk obtained from crossbred first calf heifers. The concentration averaged approximately 12 grams of immunoglobulin per 100 milliliters which provided about 120 grams in each quart of colostrum. Heavy milking cows produce much more

dilute colostrum and may require as much as 4 quarts to give the calf the same amount of immunoglobulin. If frozen colostrum is used, only the first milking should be stored . Although there is additional benefit from the second and third mild the total value declines rapidly when compared with first milk.

Figure 1 indicates the level of serum immunoglobulin and the length of time it provides protection in the newborn calf. If the initial level is high, the protection is adequate for an extended period of time. In the calf, several weeks are required before it can actively produce immunoglobulin in adequate amounts to compensate for the gradual decline in antibody received from the dam. Because of this gradual decay or loss of antibody, it is important that the calf receive as high a level as possible at birth. It should be emphasized that getting adequate colostrum into each newborn calf is the single most important criteria related to the health and productivity of the calf.

SUMMARY

Good health-management programs for beef cattle operations insure that each calf receives adequate colostrum shortly after birth. This may require milking and administration of first mild to all animals in question in addition to any cases where the management knows the calf requires direct assistance. Guesswork about whether a calf obtains colostrum is of little value after the calf is more than 24 hours old and the calf failed to receive adequate colostrum. If a calf receives colostrum early the prognosis for a productive life is good.

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Table 1. Correlation of Serum Immunoglobulin and Mortality of Market Calves*

No. of Calves	Serum Ig (mg/ml)	Mortality (%)	Cause
180	<8	61	Septicemia (50%)
80	8-17	23	Other
165	>17	4	Other

*Data from Fisher EW, Selman IE, McEwan AD, De La Fuente GH: Some causes of and some factors affecting mortality in newborn calves. In: Fifth International Meeting on Disease in Cattle. Opatija, Yugoslavia, p. 41, 1968.

Table 2. Representative Values for the Composition of the First 24 hours of Colostrum

Constituent		Cow
Fat	(g/100g)	3 · 6
Non-fatty solids	(g/100g)	19 · 5
Protein	(g/100g)	14 · 2
Lactose	(g/100g)	3 · 1
Ash	(g/100g)	1 · 0
Carotene	(µg/100g)	150 · 0
Vitamin A	(µg/100g)	140 · 0
Thiamine	(µg/100g)	60 · 0
Riboflavin	(µg/100g)	500 · 0
Nicotinic acid	(µg/100g)	100 · 0
Pantothenic acid	(µg/100g)	220 · 0
Vitamin B ₆	(µg/100g)	50 · 0
Biotin	(µg/100g)	4 · 0
Folic Acid	(µg/100g)	2 · 0
Vitamin B ₁₂	(µg/100g)	1 · 0
Ascorbic Acid	(mg/100g)	2 · 5
Vitamin D	(i.u./100g)	4 · 0

Table 3. Effects of Restricting Protein to Prepartum 2-Year-Old Beef Heifers

	Adequate	Restricted	P Value
Protein intake (kg/day)	0.62	0.38	
Metabolizable energy (mcals/day)	14.63	14.03	
Colostrum quantity (ml)	2585.6	1913.1	0.15
Colostrum IgG (mg/dl)	5856.1	7076.1	0.05
Colostrum IgM (mg/dl)	190.6	330.7	0.48
Total colostrum IgG (g)	157.1	136.6	0.89
Total colostrum IgM (g)	13.1	12.0	0.95
Calf serum IgG, 24 hours (mg/dl)	1111.8	1683.8	0.01
Calf serum IgM, 24 hours (mg/dl)	112.9	181.2	0.07
Heat production (Kcal/MBS)	118.2	104.3	0.04
Interval from calving to standing (min)	66.0	97.4	0.12

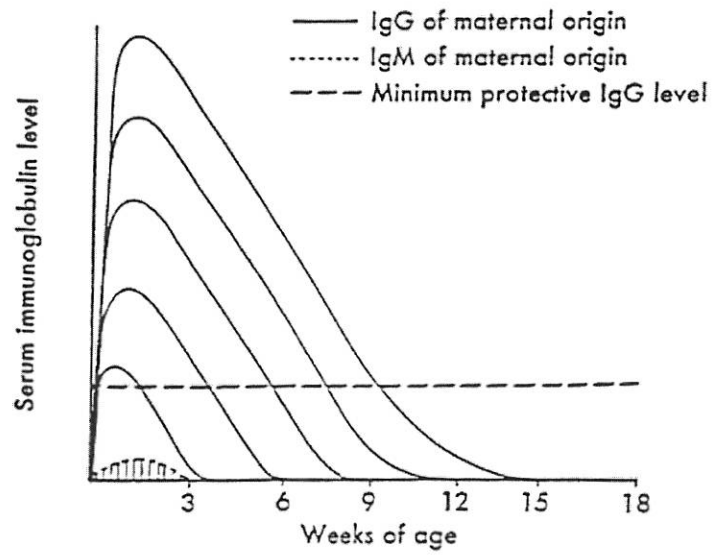
Table 4. The Relationship of Calving Difficulty and Passive Immunity

Calving Ease	Total protein (0 hours)	Total protein (12 hours)	Total protein (48 hours)	Volume of colostrum	Time In labor (hrs)
1 (42)	3.55	6.14	6.57	----	----
2-5 (17)	3.47	5.50	5.88	1062	2.9

Table 5. Composition of Colostrum from Holstein Cows (After Garrett and Overman)

A. Gross Composition						
Time After Calving	Specific Gravity	Total Solids (%)	Ash (%)	Protein (%)	Fat (%)	Lactose (%)
At parturition	1.0537	27.42	1.37	13.97	8.45	3.63
6 hours	1.0345	27.47	1.07	9.34	13.02	4.04
12 hours	1.0316	15.63	0.89	4.77	5.68	4.29
18 hours	1.0308	14.56	0.87	4.25	5.26	4.18
24 hours	1.0297	13.98	0.87	3.99	4.88	4.24
30 hours	1.0304	13.41	0.87	4.09	3.88	4.57
36 hours	1.0304	13.54	0.86	3.85	4.08	4.75

Figure 1.



Association of neonatal serum immunoglobulin G1 concentration with health and performance in beef calves

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RUMINANTS

Objective—To evaluate associations between neonatal serum IgG1 concentration and pre- and postweaning morbidity and mortality rates and average daily gains (ADGs) in beef calves and define a cutoff point for serum IgG1 concentration necessary for optimal health and performance of beef calves.

Design—Nonconcurrent cohort study.

Animals—1,568 crossbred beef calves.

Procedure—Single radial immunodiffusion was used to quantitate IgG1 concentration in sera collected from calves between 24 and 72 hours after birth. Logistic regression, ANCOVA, and likelihood ratios were used to analyze data.

Results—In the preweaning period, lower perinatal IgG1 concentrations were significantly associated with higher morbidity rates, higher mortality rates, and lower ADGs. Calves with serum IgG1 concentration < 2,400 mg/dL were 1.6 times as likely to become ill before weaning and 2.7 times as likely to die before weaning as calves with higher serum IgG1 concentrations. Calves with serum IgG1 concentration of at least 2,700 mg/dL weighed an estimated 3.35 kg (7.38 lb) more at 205 days of age than calves with lower serum IgG1 concentration. No significant association of serum IgG1 concentration with feedlot morbidity, death, or ADG was identified.

Conclusions and Clinical Relevance—By use of likelihood ratios, the threshold of serum IgG1 concentration for optimal health and performance of calves was higher than values reported previously. Implementation and maintenance of management and intervention strategies designed for early detection and treatment of calves at risk for failure of passive transfer will likely result in increases in preweaning health and performance parameters. (*J Am Vet Med Assoc* 2006;228:914–921)

passive transfer of immunoglobulins from maternal to fetal circulation.¹ This renders the newborn calf essentially agammaglobulinemic. Adequate and immediate intake of colostrum rich in immune factors and antibodies is essential to confer protective immunity to young calves.

The general positive influence of colostrum on calf health has been widely recognized.^{2–7} Although much has been discovered in the last century about colostrum composition and the physiologic process of absorption from the gastrointestinal tract, relatively little has been done to quantify the effects of different concentrations of IgG1 absorption on subsequent health and performance, especially in beef calves. Many factors, in addition to intake of colostrum, such as pathogen challenge, environment, and herd management practices, play vital roles in calf health.^{8,9} Quantitative information regarding performance, health, and economic benefits is critical to appropriately integrate recommendations on colostrum intake into an effective management program to optimize calf health and performance. The objectives of the study reported here were to evaluate and quantitate associations between serum IgG1 concentration and pre- and postweaning morbidity and mortality rates and ADGs in beef calves and, by use of likelihood ratios, define a cutoff point for serum IgG1 concentration necessary for optimal health and performance of beef calves.

Materials and Methods

Selection of cattle—A nonconcurrent cohort design was selected for this study. Calves from beef-breed dams that were 4 years of age or older and had been bred to purebred Charolais or Belgian Blue-cross bulls were eligible for inclusion in the study. Calves born with no or only minimal assis-

ADG	Average daily gain
OR	Odds ratio
CI	Confidence interval
LHR	Likelihood ratio

Bovine fetuses, like all ruminants, have a syndesmochorial type of placentation that does not permit

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Published as a journal series article No. 14580 of the University of Nebraska Agricultural Research Division, Lincoln, Neb.

Presented in part at the Conference of Research Workers in Animal Diseases Annual Meeting, St Louis, November 2002 and at the American Association of Bovine Practitioners Annual Meeting, Madison, Wis, September 2002.

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tance on February 28 through May 20 in each of the years 1996, 1997, and 1998 at the Roman L. Hruska US Meat Animal Research Center were included. All calves were maintained with similar management practices and levels of intervention. Calves were excluded from the study if they were not sired by the prescribed set of bulls, were fostered away from their dams, or left the herd for unknown reasons; there were no available data regarding dam line; or a blood sample had not been obtained between 24 and 72 hours after birth. Within 24 hours of birth, each calf was individually identified with 2 ear tags. For each calf, the identification number, sire line, date of birth, sex, birth weight, dam's degree of difficulty during parturition (calving difficulty score assigned), calf's coat color, and type of birth (single or twin) were recorded.

Data pertaining to weather conditions during the 24-hour period in which each calf was born were obtained from the High Plains Regional Climate Center, Lincoln, Neb. The source of the measurements originated within 4 miles of the facility where the cattle resided, and data were recorded each day at the same time. Standards and guides from the National Weather Service were utilized in collection of data. From the data collected during each 24-hour period, the minimum temperature, mean temperature, amount of precipitation, minimum wind chill index, mean wind chill index, and relative humidity were obtained for use in the study.

Determination of neonatal IgG1 status—A blood sample was collected from a jugular vein of each calf between 24 and 72 hours after birth. This time period for blood collection was selected to allow adequate time for calves to absorb a maximum amount of IgG1 from ingested colostrum.^{10,12} Clotted blood samples were centrifuged, and sera were collected and stored at -80°C until assayed. Single radial immunodiffusion⁴ was used to quantitate IgG1 concentration in each serum sample. One hundred thirty-six serum samples had IgG1 concentrations < 412 mg/dL, which were beyond the range of the assay; therefore, the IgG1 concentration in those samples could not be precisely estimated. For statistical analysis, the IgG1 concentrations in these samples were identified as < 412 mg/dL. By use of criteria suggested by McGuire and Adams,¹³ calves were classified according to their measured serum IgG1 concentration into 1 of 3 groups: calves with failure of passive transfer (all calves with serum IgG1 concentration ≤ 800 mg/dL), calves with marginal passive transfer (all calves with serum IgG1 concentration of 801 to 1,600 mg/dL), and calves with adequate passive transfer (all calves with serum IgG1 concentration $> 1,600$ mg/dL).

Prewaning morbidity, death, and performance—Trained personnel monitored calf health by performing daily herd checks and were not aware of the calves' IgG1 status. Calves were observed for signs of illness, such as malaise, diarrhea, dyspnea or increased respiratory effort, trauma, physical deformities, mentation abnormalities, nasal discharge, and increased rumen fill (bloat). When signs were observed, an individual morbidity event record was created for that calf that included diagnosis, date of diagnosis, and prescribed treatment. All personnel identified illnesses and diagnosed diseases on the basis of 1 set of standard guidelines, which included case definitions and prescribed treatment protocols. For example, a person examining a lame febrile calf with swollen joints and a swollen umbilicus would, based on the guidelines, assign a diagnosis of "joint ill" (ie, septic arthritis and omphalitis) to the calf and proceed with the prescribed treatment protocol. The treatment assignments were made in accordance with a preestablished treatment protocol. Complete postmortem examinations by veterinarians or a qualified veterinary technician were conducted on all calves that died. Necropsy findings, supple-

mental tests, and diagnoses were recorded and subsequently entered into a database.

A preweaning morbidity event was defined as any recorded disease event during the period between birth and weaning for which a treatment was administered. Morbidity events associated with a traumatic cause were excluded from analyses. Classification of preweaning deaths was based on postmortem records. Deaths attributed to traumatic causes, including drowning and lightning strike, were excluded from analyses. Prewaning performance was determined by calculating individual ADG from birth to weaning. Calves were weaned at approximately 200 days of age, and actual weights at weaning were recorded. Adjusted weaning weights were also calculated for 200-day weights by use of the Beef Improvement Federation guidelines that adjusted for age of the dam and sex of the calf.

Postweaning morbidity, death, and performance—Postweaning morbidity rates were assessed in a 2-stage process. Calves were moved to a feedlot environment at weaning where experienced feedlot personnel monitored calves daily for indications of illness. Morbidity events included signs of depression, decreased rumen fill (anorexia), bloat, droopy ears, ocular discharge, head tilt, lameness, nasal discharge, increased respiratory effort, or dyspnea. Calves exhibiting ≥ 1 of these symptoms were sent to the feedlot hospital for further assessment, and preestablished protocols were used to make diagnoses and assign treatments. Diagnoses were established on the basis of 1 set of standard guidelines provided to all personnel. The assignment of a particular diagnosis to an ill calf was based on signalment and clinical signs. For example, a person examining a lame calf that had signs of infection between the digits with swelling, heat, or drainage from an open wound would, based on the guidelines, assign a diagnosis of foot rot (interdigital necrobacillosis) to the calf. When signs were observed, an individual morbidity event record was created for the calf that included diagnosis and prescribed treatment; date of diagnosis; and type, amount, and routes of administration for each drug. All calves that died at the feedlot underwent a postmortem evaluation. Deaths were classified by use of database records supplemented with original copies of postmortem examination records. Deaths resulting from trauma or other atypical events (such as anaphylactic shock) were excluded from further analyses. Final weights (feedlot-out weights) of calves were measured and recorded approximately 7 to 10 days before slaughter. Postweaning performance was measured by calculating individual ADG from weaning to feedlot-out weight.

Statistical analysis—Descriptive statistics were generated for pre- and postweaning health and performance outcomes. The primary risk factor of interest was the immunoglobulin status of perinatal calves. Serum IgG1 concentration was examined as a continuous variable and as a trivariate categorical variable with regard to the success of passive transfer (failure, marginal, or adequate). When serum IgG1 concentration was considered as a continuous variable, the serum samples that had < 412 mg of IgG1/dL were classified as 411 mg/dL.

Individual weaning weights and ADG from birth to weaning and from weaning to feedlot-out weight were outcomes of interest. These variables served dual purposes because they were also covariates in the analysis of other outcomes. In our analyses, ADG was considered a continuous variable. Deaths and morbidity events were used as dichotomous variables. Other variables were included in the analysis to control for confounding and interaction and included characteristics of the dam and the calf itself and climatic factors at the time the calf was born.

Continuous predictor variables used in the analysis included serum IgG1 concentration, calf birth date, calf birth weight, weaning weight, adjusted weaning weight, and feedlot-out weight. Except for precipitation, all climatic variables were treated as continuous predictor variables in the analysis. Precipitation was analyzed as a continuous variable and also as a categorical variable. Precipitation was analyzed categorically by sorting all days (24-hour periods) in which a measurable amount of precipitation had occurred into 1 group and classifying days (24-hour periods) in which precipitation had not occurred into another group.

To control for dam line, this variable was broadly categorized and analyzed as a fixed effect. Categories of dam-breed influences included British ($n = 341$), Brahman (269), Boran (282), Tuli (301), Belgian Blue (311), and Piedmontese (64). Dam morbidity was analyzed as a categorical predictor variable, and health records were obtained and examined for all dams of calves in the study population. Any recorded treatment associated with a morbidity event for the dam from 60 days before calving until it weaned its calf was noted.

Other categorical predictor variables included serum IgG1 concentration coded trivariately, dam line, sire line, sex of calf, type of birth (single or twin), preweaning treatments for illness, feedlot treatments for illness, and dam's calving difficulty score. A calving difficulty score of 0 or 1 was assigned to each calf. All calves that were born without assistance received a calving difficulty score of 0, and calves of dams that had minimal levels of dystocia received a score of 1.

Logistic regression^b was used to examine the associations of serum IgG1 concentration with morbidity and death. Potential confounders were screened and included for consideration in multivariable modeling if their independent association with health yielded a value of $P < 0.25$. Dam and sire lines and year of birth were included in all models. Serum IgG1 concentration was forced into all models and its significance examined. The Wald χ^2 statistic (Wald statistic < 0.05) was used to evaluate each possible experimental model. The best 5 models, ordered from highest to lowest χ^2 value, were obtained. Possible models ranged from a 1-variable model to a maximum model size of 15 effects. The Hosmer-Lemeshow statistic was used to assess goodness-of-fit. The most parsimonious model that contained significant covariates (Wald statistic < 0.05) was selected, and ORs and their 95% CIs were determined.

Multiple regression analysis^b was used to assess pre- and postweaning gain. Serum IgG1 concentration was forced into all models and its significance examined. Independent variables were entered in a best R^2 selection method, and possible models were ranked according to R value. This method works by identifying subsets of independent variables of various sizes. Members of each subset contain equal numbers of variables, and the models are sorted in decreasing order of magnitude of R value. Although each subset contains equal numbers of variables, the variables may differ in size. Criteria for entering or leaving the model were set at $P < 0.05$. Once the best model for continuous variables was ascertained, an ANCOVA was used to allow inclusion of both quantitative and qualitative predictor variables. Effects of dam and sire lines and year of birth were included in every model as fixed effects. Criteria for entering or leaving the model remained at $P < 0.05$. Comparisons of the adjusted R^2 and P value were used to distinguish the model most suitable for each outcome.

Likelihood ratios were generated by incremental classification of serum IgG1 concentrations and subsequent treatment of the upper limit of each IgG1 classification as a cutoff value. A series of LHRs were generated by successively dichotomizing serum IgG1 values more or less than 412 mg/dL, more or less than 500 mg/dL, and more or less than each 100 mg/dL level from 500 to 2,800 mg/dL.¹⁴ Positive and negative LHRs (LHR+ and LHR-) and their 95% CIs were cal-

culated separately for each of the 6 health and performance outcomes of interest (pre- and postweaning morbidity, death, and ADG) at each cutoff point in the range of serum IgG1 concentrations.¹⁴ Pre- and postweaning morbidity and death were coded as qualitative dichotomous variables. Pre- and postweaning ADGs were also coded as dichotomous variables by calculating the overall means for pre- and postweaning ADG. Animals were then assigned to 1 of 2 groups: calves with ADG below the population mean for daily gain or calves with ADG equal to or greater than population mean for daily gain. For each serum IgG1 cutoff value, the frequencies of true-positive, false-positive, true-negative, and false-negative results were determined.

For purposes of this study, LHR+ (ie, $\text{LHR+} = \text{sensitivity}/[1 - \text{specificity}]$) described how many times more likely a calf that had the outcome of interest would have a serum IgG1 concentration at or below the cutoff value than would a calf that did not have the outcome of interest. An LHR- (ie, $\text{LHR-} = [1 - \text{sensitivity}]/\text{specificity}$) described how many times more likely a calf that had the outcome of interest would have a serum IgG1 concentration above the cutoff value than would a calf that did not have the outcome of interest.

For each outcome, magnitude of LHR and limits of the 95% CIs were examined. Further investigation ensued if the LHR+ was > 1.0 and 1.0 was not contained within the surrounding confidence limits. If these conditions were met, modeling of the outcome with the appropriate covariates and the selected IgG1 cutoff value was performed to determine significance ($P < 0.05$). When comparing cutoff values, the magnitude of LHR+ Wald P values (< 0.05) was considered, and those associated with higher R^2 values were considered optimal.

Results

In the present study, 798 calves were sired by Charolais bulls and 770 calves were sired by Belgian Blue-cross bulls. Most calves (1,558/1,568 [99.4%]) were single births. The mean birth weight for calves in the study was 39.9 kg (88 lb), and weights ranged from 20.9 to 66.2 kg (46 to 146 lb). Of the 1,568 calves included in the study, 796 (51%) were heifers and 772 (49%) were bull calves. In the study group, 1,529 calves were born without assistance (calving difficulty score of 0). Only 2% (39/1,568) of calves were assigned a calving difficulty score of 1 because their dams required assistance during parturition. Of the 1,568 dams, 28 were treated because of a morbidity event that had occurred within the period of 60 days prior to parturition until weaning. On the day the calves were born, minimum recorded temperatures ranged from -26.16° to 14.17°C (-15.1° to 57.5°F). Mean temperatures ranged from -18.78° to 18.78°C (-1.8° to 65.8°F), and relative humidity ranged from 26.8% to 100%. On days the calves were born, the lowest recorded wind chill factors ranged from -36.5° to 51.0°F , whereas mean wind chill factors ranged from -20.8° to 58.6°F . The amount of precipitation varied from 0.0 to 0.85 inches, and 303 of the study calves were born on days that had a measurable amount of precipitation.

Prewaning morbidity among beef calves—After excluding 12 traumas and atypical morbidity events, 1,556 calves were entered in the preweaning morbidity analysis. At least 1 morbidity event prior to weaning was recorded for 12.0% (187/1,556) of the study population. Morbidity events and deaths were often asso-

ciated with disease of the respiratory or gastrointestinal tract. For example, diagnoses of pneumonia (73/187 calves) or scours (25/187) accounted for more than 50% (98/187) of the recorded preweaning morbidity events. Calves with serum IgG1 concentrations ≤ 800 mg/dL had a preweaning morbidity rate of 21.7% (48/221), compared with a rate of only 13.4% (18/134) for calves with serum IgG1 concentrations from 801 to 1,600 mg/dL (Table 1). A preweaning morbidity event was diagnosed in only 10.1% (121/1,201) of the calves that had serum IgG1 concentrations $> 1,600$ mg/dL. Dam line, calf's year of birth, and sire line were included a priori in the preweaning morbidity logistic regression model. Sex of the calf and calving difficulty score were considered as covariates on the basis of univariate screening; however, calving difficulty was not included in the final model. Thus, the final model included calf sex and serum IgG1 concentration as independent variables. Adequate goodness-of-fit was determined by use of the Hosmer-Lemeshow goodness-of-fit test ($P > 0.50$), and the model accounted for 10.6% of the variability associated with preweaning morbidity. Calves classified as having failure of passive transfer (serum IgG1 concentration ≤ 800 mg/dL) were 2.24 (95% CI, 1.52 to 3.29) times as likely to have a preweaning morbidity event, compared with calves classified as having adequate passive transfer (serum IgG1 concentration $> 1,600$ mg/dL). Calves classified as having marginal passive transfer (serum IgG1 concentration of 801 to 1,600 mg/dL) were not (OR = 1.38; 95% CI = 0.80 to 2.39) more likely to have a preweaning morbidity event, compared with calves classified as having adequate passive transfer.

When considering a serum IgG1 cutoff value for preweaning morbidity based on LHR+ > 1.0 with 1.0 not contained within the surrounding CI, 2 possible cutoff values were identified: 2,400 and 2,500 mg/dL. Covariates included in the logistic regression model were identical to those used in the final model for preweaning morbidity. Model convergence and the goodness-of-fit criteria were satisfied for each model. Serum IgG1 concentration was significantly negatively associated with preweaning morbidity when calves were classified by dichotomizing serum IgG1 concentration at 2,500 or 2,400 mg/dL. When compared with 2,500 mg/dL, the

threshold of 2,400 mg/dL resulted in a slightly higher OR (1.65 vs 1.5). Calves with serum IgG1 concentrations $\leq 2,400$ mg/dL were 1.6 (95% CI, 1.19 to 2.28) times as likely to develop illness before weaning as calves with IgG1 concentrations $> 2,400$ mg/dL. This model accounted for 9.4% of the variability associated with preweaning morbidity rate.

Preweaning death among beef calves—Nine calves were excluded from this analysis because their deaths were a result of trauma or an extraordinary event. After these exceptions were removed, 1,559 calves were entered in the analysis of deaths. Of the 1,559 calves, 42 (2.7%) died before weaning (Table 1). Two hundred twenty-one calves were classified as having failure of passive transfer (serum IgG1 concentration ≤ 800 mg/dL), and of these, 19 (8.6%) died before weaning. In the group of calves classified as having marginal passive transfer (serum IgG1 concentration of 801 to 1,600 mg/dL; $n = 135$), there were only 3 (2.2%) preweaning deaths. Of the 1,203 calves classified as having adequate passive transfer (serum IgG1 concentration $> 1,600$ mg/dL), 20 died before weaning; this group had the lowest rate of mortality (1.7%) among the 3 groups. Dam and sire lines and calf's year of birth were included a priori in the preweaning death logistic regression model. Lowest recorded temperature on the day the calf was born and serum IgG1 concentration were included in the final model. Model convergence criteria were satisfied in the final model, and the Hosmer-Lemeshow goodness-of-fit test had a P value of 0.95. The final model accounted for 11% of the variability in preweaning mortality rate. Calves that were classified as having failure of passive transfer were 4.9 (95% CI, 2.48 to 9.52) times as likely to die before weaning than calves classified as having adequate passive transfer. Compared with calves that had adequate passive transfer, calves with marginal passive transfer were not (OR, 1.20; 95% CI, 0.35 to 4.14) more likely to die before weaning. When the marginal passive transfer category was combined with the failure of passive transfer category and compared with the adequate passive transfer category, the OR was reduced to 3.4 (95% CI, 1.79 to 6.45).

After LHRs were generated and examined for values with an associated LHR+ > 1.0 with 1.0 not con-

Table 1—Number of beef calves with various serum concentrations of IgG1 after ingestion of colostrum that had at least 1 morbidity event or died before weaning or during the interval after weaning until removal from the feedlot.

Serum IgG1 concentration (mg/dL)	No. of calves (%) that had ≥ 1 preweaning morbidity event	No. of calves (%) that died before weaning	No. of calves (%) that had ≥ 1 morbidity event at the feedlot*	No. of calves (%) that died at the feedlot*
≤ 800	48/221 (21.7)	19/221 (8.6)	20/202 (9.9)	2/202 (1.0)
$\leq 1,200$	57/274 (20.8)	21/276 (7.6)	25/254 (9.8)	2/254 (0.8)
$\leq 1,600$	66/355 (18.6)	22/356 (6.2)	36/333 (10.8)	3/333 (0.9)
$\leq 2,000$	78/461 (16.9)	23/463 (5.0)	49/437 (11.2)	3/439 (0.7)
$\leq 2,400$	92/631 (14.6)	28/635 (4.4)	67/632 (11.1)	4/604 (0.8)
$\leq 2,800$	113/836 (13.5)	31/843 (3.7)	95/806 (11.8)	5/808 (0.6)
$\leq 3,200$	130/1,006 (12.9)	32/1,013 (3.2)	116/973 (11.9)	5/976 (0.5)
Total No. of calves (%)	187/1,556 (12.0)	42/1,559 (2.7)	175/1,513 (11.6)	6/1,517 (0.4)

*Period from weaning until removal of calf from the feedlot.

tained within the surrounding CI, 4 potential cutoff values were selected for further evaluation: 2,400, 2,500, 2,600, and 2,700 mg/dL. When these cutoff values were analyzed, covariates incorporated in the model were identical to those used in the final preweaning death model with the more conventional cutoff value (serum IgG1 concentration ≤ 800 mg/dL) and also included sex of the calf and the recorded minimum temperature on the day of the calf's birth. Dam and sire lines and calf's year of birth were included a priori in the model. When serum IgG1 cutoff values of 2,600 and 2,700 mg/dL were analyzed, *P* values were < 0.05 but the lower CI value was very marginal at 1.02. Analysis of serum IgG1 concentration cutoff values of 2,400 or 2,500 mg/dL resulted in *P* values indicating significance (ie, values < 0.05) and more reliable CIs surrounding the ORs. When serum IgG1 concentration of 2,400 mg/dL was used as a cutoff value, calves with serum IgG1 concentrations $< 2,400$ mg/dL were 2.7 times (95% CI, 1.34 to 5.36) as likely to die before weaning, compared with calves with higher concentrations.

Preweaning performance parameters—Individual calf preweaning ADGs were calculated for calves that survived from birth to weaning. Gains ranged from 0.42 to 1.26 kg/d (0.93 to 2.77 lb/d). The mean ADG for all calves from birth to weaning was 0.92 kg/d (2.03 lb/d). Overall, 1,511 animals were included in this analysis.

Dam and sire lines and calf's year of birth were included in the multivariable logistic models. Calf's birth weight, sex, and calving difficulty score were also significantly associated with gain in some of the models. Serum IgG1 concentration was analyzed as a continuous variable and as a categorical variable but was not significantly associated with ADG from birth to weaning in any of the models.

After screening potential serum IgG1 concentration cutoff values for LHR+ > 1.0 with 1.0 not contained in the surrounding CI, values from 2,200 to 3,200 mg/dL were selected for further evaluation. Covariates were identical to those used in the final model for preweaning ADG with the more conventional cutoff values and included dam and sire lines; calf's year of birth, birth weight, and sex; and calving difficulty score. In contrast to the analysis that involved conventional serum IgG1 cutoff values, results derived from examination of LHR uncovered significant effects of serum IgG1 concentration on gain (Table 2). The R^2 values were similar among models and accounted for 29.0% to 29.2% of the variability in preweaning ADG. Analysis of all nonconventional serum IgG1 concentration cutoff values indicated that there was significant association between lower serum IgG1 concentration and lower ADG with similar point estimates of -0.014 to -0.018 kg/d (-0.03 to -0.04 lb/d).

Postweaning morbidity among beef calves—After excluding atypical morbidity events, 1,513 heifers and steers were included in the postweaning morbidity analysis. Overall, 175 (11.6%) calves of that population were treated at least once while in the feedlot. Of the 202 calves classified as having failure of passive transfer, 20 (9.9%) had at least 1 morbidity event in the

feedlot (Table 1). Among 131 calves categorized as having marginal passive transfer (serum IgG1 concentration of 801 to 1,600 mg/dL), 6 had a morbidity event; the rate of morbidity in these calves was higher (12.2%) than the rate (9.9%) in calves classified as having failure of passive transfer (serum IgG1 concentration ≤ 800 mg/dL). The morbidity rate for calves with adequate passive transfer (serum IgG1 concentration $> 1,600$ mg/dL) was intermediate at 10.5% (139/1,319 calves).

Use of logistic regression satisfied the convergence criterion, and after adjusting for effects of dam and sire lines and calf's year of birth, the only significant factor in the model was the calving difficulty score. No significant effects of serum IgG1 concentration on feedlot morbidity rate were evident in any of the models. In addition, some models identified slightly lower risk for groups with higher serum IgG1 concentration, whereas other models identified slightly higher risks in those groups. There were no significant differences among serum IgG1 concentrations and no significant association of decreasing morbidity rate with increasing serum IgG1 concentration. No additional serum IgG1 concentration cutoff values were identified or evaluated because no values satisfied both criteria (LHR+ and lower limit of the CI > 1.0) for further screening.

Postweaning death among beef calves—Only 0.4% (6/1,517) of the calves died while in the feedlot. Less than 1% (2/202) of calves classified as having failure of passive transfer (serum IgG1 concentration ≤ 800 mg/dL) died while in the feedlot. Of the 132 calves with marginal passive transfer (serum IgG1 concentration of 801 to 1,600 mg/dL), only 1 died (Table 1). Of the 1,183 calves with adequate passive transfer (serum IgG1 concentration $> 1,600$ mg/dL), only 3 (0.25%) died while in the feedlot. This low number of cases prevented use of regression modeling of postweaning death, and no association with serum IgG1 concentration was detected.

Postweaning performance parameters—Individual animal ADG ranged from 0.5 to 1.9 kg/d (1.1 to 4.2 lb/d). When all values were averaged, the mean ADG from weaning to feedlot-out weight was 1.1 kg/d (2.5 lb/d). Feedlot-out weights ranged from 320 to 730 kg

Table 2—Results of logistic regression analysis of serum IgG1 concentrations dichotomized to new cutoff values for ADG among 1,511 beef calves from birth to weaning.

Serum IgG1 concentration (mg/dL)	Point estimate of ADG (lb/d) if serum IgG1 concentration \leq cutoff value	R^2
$\leq 2,200$	-0.03336	0.2916
$\leq 2,300$	-0.03344	0.2917
$\leq 2,400$	-0.03440	0.2919
$\leq 2,500$	-0.03160	0.2913
$\leq 2,600$	-0.03481	0.2921
$\leq 2,700$	-0.03600	0.2924
$\leq 2,800$	-0.03194	0.2913
$\leq 2,900$	-0.03180	0.2911
$\leq 3,000$	-0.02860	0.2903
$\leq 3,100$	-0.02819	0.2901
$\leq 3,200$	-0.02760	0.2900

(704 to 1,605 lb; mean feedlot-out weight, 535 kg [1,176 lb]). Mean age of cattle at time of shipment to slaughter was 16 months (480 days; age range, 13 to 19 months [384 to 569 days]).

By use of multiple logistic regression, all point estimates for serum IgG1 concentration were not significant ($P > 0.05$), and no further analysis was pursued. Significant covariates included sex of calf and birth and weaning weights.

Discussion

The association between preweaning morbidity and death in calves with low serum IgG1 concentrations was consistent with other reports^{2,6,15-22} in the literature. In the present study, we extended evaluation of the effects of serum IgG1 concentration after colostrum ingestion to examine whether additional benefits for calf health were accrued at values greater than the traditional serum IgG1 concentration cutoff values of 800 and 1,600 mg/dL. Calves with serum IgG1 concentrations $< 2,400$ mg/dL were almost 3 times as likely to die, compared with calves with greater IgG1 concentrations. The detection of continuing negative effects of low serum IgG1 concentrations on preweaning morbidity and mortality rates, even in calves with 2,400 mg of IgG1/dL of serum, suggests that negative effects of inadequate passive transfer may still occur at serum concentrations of IgG1 that are higher than those examined in other studies. This may provide added impetus to intercede and provide supplemental care to promote calf health.

The level of passive immunity neither guaranteed nor completely prevented preweaning morbidity or death among beef calves. Most calves that had been classified as having failure of (≤ 800 mg/dL) or marginal (801 to 1,600 mg/dL) passive transfer survived (333/355 [93.8%] calves) and remained healthy (289/355 [81.4%] calves) until weaning. In our study, 121 of 1,201 (10%) calves classified as having adequate passive transfer had at least 1 recorded preweaning morbidity event. Twenty of the 1,203 (1.7%) calves classified as having adequate passive transfer died before weaning, illustrating that adequate passive transfer (serum IgG1 concentration after ingestion of colostrum $> 1,600$ mg/dL) is not fail-safe protection against death before weaning among beef calves.

The risk for developing an illness in calves with low serum IgG1 concentrations was not as great as those in the study by Perino et al.⁶ In that study, beef calves categorized as having failure of passive transfer (serum IgG1 concentration < 800 mg/dL) were 9.4 times as likely to become ill in the preweaning period as calves with higher serum IgG1 concentrations. However, that study group⁶ was composed of first-born calves from crossbred beef cows, among which there was a high rate of twins (43%). Twins have been found to be at greater risk of death within the first 21 days of life than single-born calves.²³

In the present study, overall preweaning mortality rate (2.6%) was low, compared with other reports.²⁴⁻²⁶ This remained true even when the 12 animals that had died but had been excluded from analysis of preweaning death were included in another calculation of over-

all mortality rate. Several factors may have contributed to the low overall mortality rate in our study population. The calves of the present study were maintained by knowledgeable personnel and provided with high-quality management and regular veterinary care. In addition, all dams were multiparous and calves were at least 24 hours of age before inclusion in the study. Overall mortality rates were likely decreased by the exclusions of atypical preweaning deaths and calves born to heifers.^{24,25}

In addition to the importance of serum IgG concentration, other key covariates were identified in the analysis of preweaning deaths. The maternal genetic description (dam line) and the recorded minimum temperature on the day the calf was born were factors that were significantly associated with death before weaning. To our knowledge, no other studies to investigate the combination of these variables and their effects on preweaning death have been reported. In addition to adjusting estimates of the effects of serum IgG1 concentration for these important factors, our data have provided new insights regarding combinations of factors that may be managed to reduce the frequency of preweaning death among beef calves.

One shortcoming of the present study was that some calves may have had morbidity events that were unnoticed during herd checks. Because morbidity events were defined as those calves needing treatment, some calves may have had undetected morbidity events or subclinical morbidity events that did not require treatment. However, all herd health checks and observations for morbidity events were conducted by trained and experienced personnel. Because 136 serum samples in which IgG1 concentration was < 412 mg/dL could not be accurately assayed, they were simply classified as < 412 mg of IgG1/dL for categorical analysis and as 411 mg of IgG1/dL for the analysis of serum IgG1 concentration in a continuous manner. In the continuous analysis of serum IgG1 concentration, this categorization likely underestimated the effect of serum IgG1 concentration on health and performance of beef calves.

When modeling preweaning gain with traditional cutoff values for serum IgG1 concentration after ingestion of colostrum, IgG1 concentration did not significantly influence ADG from birth to weaning. Further investigation and modeling of other serum IgG1 concentration cutoff values selected after examination of LHRs revealed a significant relationship between serum IgG1 concentration and preweaning gain. When the point estimate of -0.02 kg/d (-0.036 lb/d) was multiplied by 205 days, the mean weight of calves with serum IgG1 concentration $\geq 2,700$ mg/dL would be expected to be 3.4 kg (7.4 lb) more at 205 days of age than the weight of calves with serum IgG1 concentration $< 2,700$ mg/dL. Kyuma et al.,²⁷ Robison et al.,⁴ Odde,²⁸ and Fiems et al.²⁹ have also reported significant correlations between serum immunoglobulin concentration and subsequent weight gains. To our knowledge, our study is the only investigation involving beef cattle raised in an environment typical for North American cattle that has established a significant effect of serum IgG1 concentration on preweaning gain. It is possible that other studies that

did not determine that serum IgG1 concentration was significantly associated with preweaning gain would reveal significance if higher serum IgG1 concentration cutoff values were explored.

The improved ADG associated with higher serum IgG1 concentrations may have been related to a decreased rate of morbidity. In addition to improved gain, calves with high serum IgG1 concentrations also developed less illness. Wittum et al²⁶ reported that calves that had a preweaning morbidity event weighed less at weaning than calves that did not become ill. Dollars lost as a result of reduced weight gain attributed to low serum IgG1 concentration and subsequent preweaning morbidity in beef calves may be recovered through effective management and intervention strategies designed to optimize acquisition of passive immunity in a timely manner.

In our study, there was no significant effect of serum IgG1 concentration on feedlot health or performance parameters. Although the study of this report represents what we believe to be the largest investigation to date that was designed to measure the association of serum IgG1 concentration with feedlot morbidity and death and ADG, the mortality and morbidity rates were low. Competing risks may also have contributed to the lack of effect of serum IgG1 concentration on feedlot health and productivity. Calves that would have been at higher risk in the feedlot may have already died during the preweaning period. Calves that continued from the preweaning phase into the feedlot component of our study would be those that had managed to mount a competent active immune response to infectious challenges.

Lack of power in our study may also have contributed to the lack of significance between serum IgG1 concentration and postweaning morbidity, postweaning death, and ADG. For the threshold of 1,600 mg of IgG1/dL of serum, the analysis of feedlot morbidity events had only 5.5% power. To obtain 83% power, diagnoses of 25 additional morbidity events (total of 61 cases) in the low serum IgG1 concentration category would be necessary. This would be true if the proportion of morbidity events did not change for the other groups. The analysis of death had only 45% power. To increase this power to 79% ($\alpha = 0.05$), 5 additional deaths (total of 11 cases) in the low serum IgG1 concentration category would have been necessary.

The lack of associations between serum IgG1 concentration and postweaning morbidity, postweaning death, and ADG is in contrast to findings of a study by Wittum and Perino,²² which is the only other published study of which we are aware that addresses the association between serum IgG1 concentration and feedlot health and performance. The study herd investigated by those investigators was characterized by a high rate of twinning and maternal dairy influence. Calves in that study also had a much higher rate of morbidity (53%) than calves of the present study. It is possible that the inordinately high disease challenge in the study of Wittum and Perino may have overwhelmed the slightly compromised but normally competent immune system of calves that had been classified as having failure of or only marginal passive transfer. That

high morbidity rate may also have created enough power to result in a level of statistical significance that studies with more typical morbidity rates could not achieve. Repeated observations in different study groups and use of various feedlot scenarios would add additional credence to the hypotheses and strengthen the consistency of the causal association.

Identification and quantification of the effects of low concentrations of serum IgG1 after ingestion of colostrum in beef calves form the basis for recommendations to ensure early acquisition of an adequate mass of colostral IgG1. In the present study, use of LHRs identified a cutoff value for serum IgG1 concentration that was much higher than has been typically used in other studies investigating risk for morbidity. To our knowledge, this method of analysis has not been previously applied to define a cutoff value for serum IgG1 concentration after ingestion of colostrum in beef calves. Our data expand the body of evidence supporting the importance of timely acquisition of an adequate mass of colostral immunoglobulins for optimizing preweaning health and performance parameters in beef calves. Early detection and treatment of calves at risk for failure of passive transfer is likely to result in improved preweaning health and increased performance parameters within herds.

a. VMRD Inc, Pullman, Wash.

b. SAS for Windows, version 8.2, SAS Institute Inc, Cary, NC.

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Selected abstract for JAVMA readers from the *American Journal of Veterinary Research*

Cardiovascular effects of desflurane in mechanically ventilated calves
Robert D. Keegan et al

Objective—To determine cardiovascular effects of desflurane in mechanically ventilated calves.

Animals—8 healthy male calves.

Procedure—Calves were anesthetized by face mask administration of desflurane to permit instrumentation. Administration of desflurane was temporarily discontinued until mean arterial blood pressure increased to ≥ 100 mm Hg, at which time baseline cardiovascular values, pulmonary arterial temperature, end-tidal CO_2 tension, and end-tidal desflurane concentration were recorded. Cardiac index and systemic and pulmonary vascular resistances were calculated. Arterial blood gas variables were measured and calculated. Mean end-tidal concentration of desflurane at this time was 3.4%. After collection of baseline values, administration of 10% end-tidal concentration of desflurane was resumed and calves were connected to a mechanical ventilator. Cardiovascular data were collected at 5, 10, 15, 30, and 45 minutes, whereas arterial blood gas data were collected at 15 and 45 minutes after collection of baseline data.

Results—Mean \pm SD duration from beginning desflurane administration to intubation of the trachea was 151 ± 32.8 seconds. Relative to baseline, desflurane anesthesia was associated with a maximal decrease in arterial blood pressure of 35% and a decrease in systemic vascular resistance of 34%. Pulmonary arterial blood temperature was decreased from 15 through 45 minutes, compared with baseline values. There were no significant changes in other measured variables. All calves recovered from anesthesia without complications.

Conclusions and Clinical Relevance—Administration of desflurane for induction and maintenance of general anesthesia in calves was smooth, safe, and effective. Cardiopulmonary variables remained in reference ranges throughout the study period. (*Am J Vet Res* 2006;228:387-391)



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Dynamics of Colostral IgG, Absorption in Beef Calves After Bottle Feeding, Stomach Tubing and Dam Suckling

L. Hungerford, R. Dewell, G. Dewell, G. Rupp, W. Laegreid and K. Shuck

Abstract

Many factors can affect passive transfer of immunoglobulins, including age of calf, total mass of immunoglobulins ingested, method of ingestion, breed, environmental temperature, calf vigor and cow mothering ability. Most field studies have used 12-48 hour IgG or total protein values to evaluate immune status (e.g. less than 800=inadequate, 800-1600=marginal, greater than 1600=good) and have found improved health among calves with increasing serum levels (Wittum and Perino, 1995). A few studies have found 12-24 hour immunoglobulin values to be less predictive (Barber, 1978; Rea et al., 1996). The time between birth and achievement of adequate immunoglobulin levels may affect the disease incidence, especially due to enteric organisms, even if calves eventually receive colostrum. Experimentally, calves inoculated with *E. coli* before receiving colostrum were found to be as susceptible to disease as colostrum-deprived calves, regardless of the amount of colostrum subsequently provided (Logan et al., 1977). Early ingestion of colostrum should give calves a better chance to resist pathogenic microbes in their environment. There is wide variation in published curves for IgG absorption over time and many studies have used dairy calves. Our objectives were to determine how quickly IgG and total protein values would rise in unstressed, beef calves in a production setting and to compare the dynamics of absorption following colostrum administration via stomach tube, bottle feeder or natural suckling.

Forty calves, from mixed breed beef cows in body condition 6+ with unassisted calving during the fall of 1998, were used. Calves were weighed and randomly assigned, within each sequential group of four, to the immediate stomach tube (IST), later stomach tube (LST), bottle feeder (BF), or dam suckling (DS) groups. A colostrum sample was collected from each dam. Calves in the IST, LST and BF groups received 40 ml/kg colostrum or the entire first milking from their own dams: within 30 minutes after birth for the IST and BF groups and at 4 hours after birth for the LST group. Calves

were then muzzled or separated from the dam by a pipe gate to prevent nursing but allow close contact for 6 hours post feeding. Suckling times were recorded for calves in the DS group through 6 hours. Blood samples were collected at times 0, +0.5 hr, +1 hr, +1.5 hr, +2 hr, +3 hr, +4 hr, +5 hr, +6 hr, +7 hr, +8 hr, +12 hr, +24 hr post-colostrum. Packed cell volumes (PCV) and plasma total protein (TP) levels were determined. Single radial immunodiffusion was used to quantitate IgG, levels in serum and colostrum (VMRD, Inc, Pullman, WA.). Absorption curves were compared using repeated measures ANOVA (PROC MIXED, SAS Institute, Inc.; Littell et al., 1996). Individual 4 hour TP levels were compared to the average of all TP measurements made at birth and ROC analysis used to determine the size of the difference which best predicted colostrum intake. P values of ≤ 0.05 were considered significant.

The IgG and TP absorption curves differed significantly between treatment groups (see figure for IgG). However, ranges of values were almost as wide within groups as between groups. Although the DS group had the highest IgG levels, it did not differ significantly from the IST or LST groups. Therefore, tube feeding provides an excellent alternative if suckling is delayed. Since BF led to significantly lower absorption than the stomach tube groups combined, there is no justifiable clinical advantage to this method.

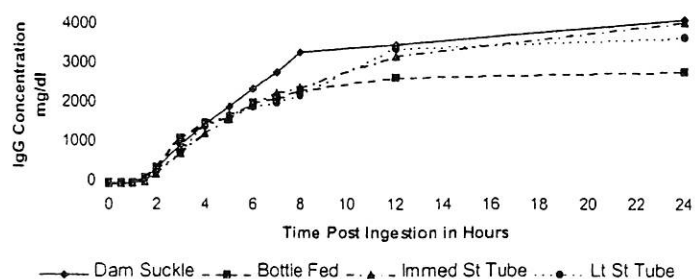


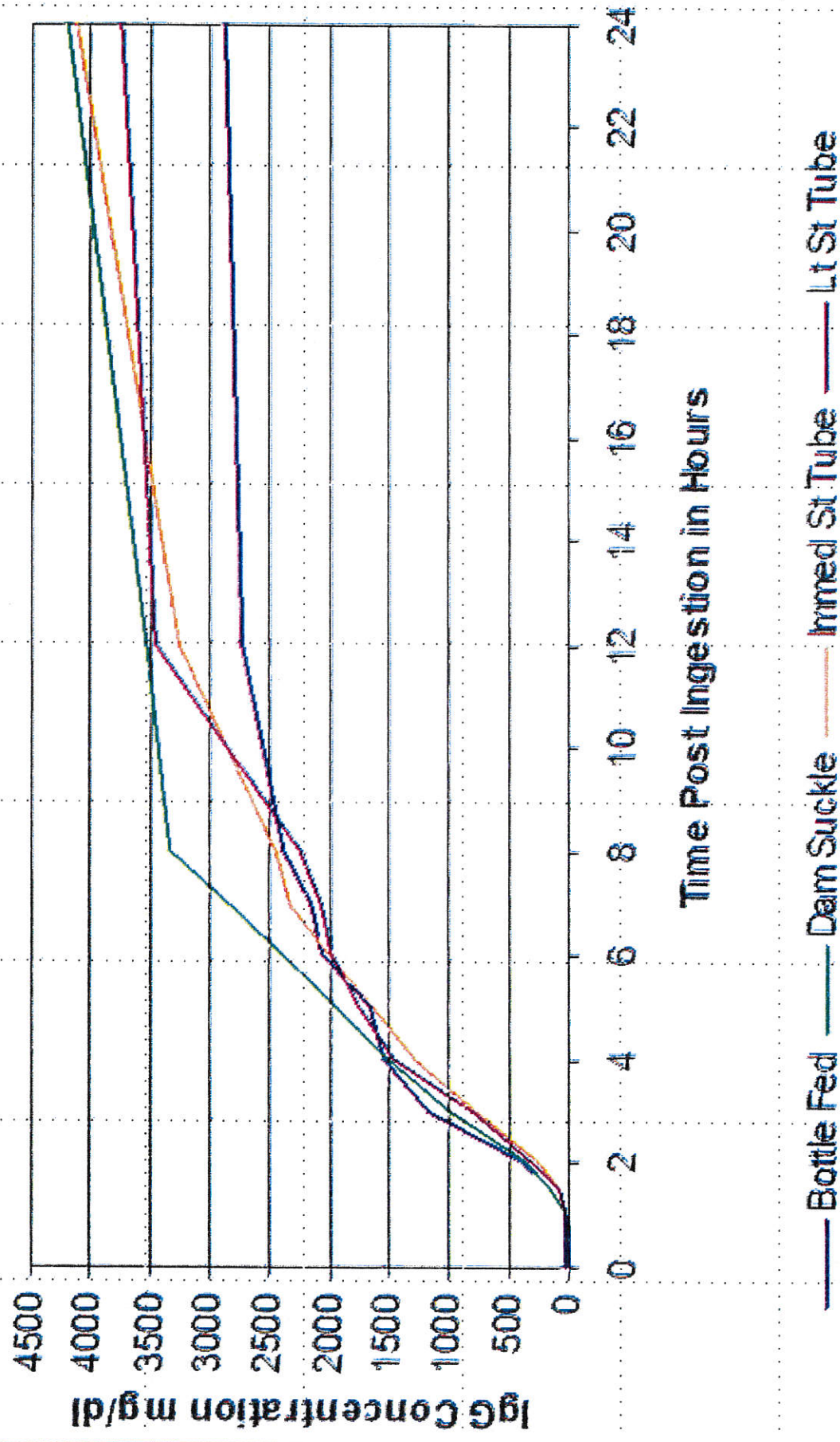
Figure 1. Change in immunoglobulin G1 concentration following colostrum ingestion—mean values for groups.

Although there were differences in the IgG and TP absorption curves, calf IgG levels reached 800 mg/dl in an average of 3-1/2 to 4 hours post-colostrum, regardless of group. Since calves which had not received colostrum had less than a 0.5 gm/dl increase in TP at 4 hours over the herd pre-colostral average (Sensitivity=87%, Specificity=89%), this provides a quick and easy means of identifying calves requiring supplementation. Delaying administration of colostrum until 4 hours of age did not affect the absorption rate, but did, of course, increase the total time period until the calf had passive IgG protection. This extended period of susceptibility could not be detected by IgG differences at 12 or 24 hours of age. This may explain the variation in neonatal morbidity among calves with adequate 24 hour IgG levels (Rea et al., 1996). Delays in acquisition of colostrum may critically jeopardize calf health.

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**Change in Immunoglobulin G1 Concentration
Following Colostrum Ingestion
Mean Value for Group**



VETERINARY

FOR THE PRACTICING VETERINARIAN

Quarterly

Winter 2009

Volume 12, Number 1

Environment, dam, management: Factors influencing passive transfer of immunoglobulins to neonatal calves

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The bovine fetus has a type of placentation that does not permit contact between maternal and fetal blood and therefore does not permit passive transfer of immunoglobulins (Ig) from the mother to her fetus. The bovine calf is born agammaglobulinemic and depends on the intake of an adequate amount of maternal colostrum to provide antibodies and other immune factors that confer protective immunity against environmental pathogens from the early postnatal period until the third or fourth month of life. Many factors related to the calf, the environment, the dam and management can influence the passive transfer of Ig to neonatal calves (5, 9, 24, 46).

Inadequate intake of colostrum immediately after birth, whether in quantity or quality, results in partial or complete failure of passive transfer (FPT) of Ig, which has been considered the most important risk factor for morbidity and mortality in neonatal beef and dairy calves (5, 9, 14, 16, 22, 30, 36, 46, 47). Various methods

used to diagnose FPT have been reviewed by others (24, 30). The level of IgG in serum has been considered the most accurate method to diagnose FPT in calves. The range of values considered for failure are < 8 g/L for total failure, >8 but <10 g/L for partial failure, and >10 g/L for adequate transfer of IgG (30). Some authors consider only > 10 g/L for adequate transfer and <10 g/L for inadequate transfer of IgG (5, 9, 14, 15, 18, 22, 46).

Various studies have shown that the prevalence of FPT in dairy calves may be around 10-35% (24, 30, 46). One study from USDA reported that more than 40% of dairy heifer calves have < 10 g/L in serum in the first day of life (51). In beef cattle, around 11-31% of calves reportedly achieved < 8 g/L during the first day of life (16, 29, 36).

There are direct associations between FPT and presentation of disease (septicemia, diarrhea, pneumonia) and mortality in calves during the pre-weaning period. Some studies have shown that calves with FPT (< 8 g/L) are between 1.6 and 9.5 times more likely to become ill, and between 2.7 and 5.4 times more likely to die than calves with adequate passive transfer during the first months of life (16, 36, 37, 40).

At the same time the performance of calves that had FPT is decreased compared to calves with adequate transfer of Ig. The average weight at weaning for calves with adequate Ig transfer can be 6-34 lb more than calves with FPT (14, 16, 17, 36, 40). All of this leads to economic losses due to

reduced growth, suboptimal reproductive performance, treatment costs and death losses.

An adequate colostrum management program and the early detection and treatment of calves suspected of FPT is required to improve the pre-weaning health and performance of the young stock in dairy and beef herds. The following will discuss in depth the factors that influence the Ig absorption process and different management and product alternatives that producers have to prevent FPT in calves.

Immunoglobulin Absorption

Absorption of immunoglobulins and other macromolecules in the small intestine of the newborn calf is a short-term, non-selective process which is induced and also stimulated to close by feeding (45, 49). The small intestinal absorptive cells in the neonate are induced to absorb macromolecules like immunoglobulins, other proteins like albumin and casein, and even microorganisms like *E. coli*, by pynocytosis once they have contact with any feed after birth. This process is non-selective, with each type of macromolecule having the same opportunity to be absorbed, therefore competing with Ig for receptors or binding sites in the enterocytes (45, 46). Once inside enterocytes the proteins absorbed are transported by vacuoles through the cytoplasm to the basal membrane and from there to lymphatics and the portal system where they enter the general circulation. Absorption of proteins

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seems to be higher in the jejunum than in the other portions of the small bowel (46). The cessation of the absorptive capabilities of the enterocytes is stimulated by feeding, especially by any protein (immunoglobulin, albumin, casein) in the feed. It seems that protein can activate an enzymatic digestive process in the cell that breaks down the vacuoles involved in transportation, thus impeding the process (45, 46, 47, 48). Other factors like maturation of small intestine cells, increased abomasal acidity, and development of intestinal secretions (digestive enzymes) could be involved in the cessation of absorption of Ig (45).

Therefore the efficiency of IgG absorption depends on the time of first colostrum feeding, the concentration of IgG in the colostrum, and the total amount of colostrum fed per unit of body weight (9). Some authors have established a formula to calculate an Apparent Efficiency of Absorption (AEA) of IgG in calves as follows: $AEA = \text{plasma IgG at 24 h (g/L)} \times \text{Body Weight (kg)} \times 0.092 / \text{total IgG intake in grams}$ (13). The average of AEA IgG level is around 20 to 35% (13, 44, 50).

Factors influencing the passive transfer in calves

Many factors have been established to affect the absorption of IgG in neonates, but the age (in hours) at first ingestion of colostrum, the concentration of IgG in the colostrum ingested, and the total amount of colostrum at first ingestion are the primary influencing factors.

Once enterocytes come in contact with feed (protein) the closure process begins. Some research has stated that the first 4 hours of life are the most critical period to absorb large quantities of Ig, and that after only 8-12 hours of life the absorption capacity is severely diminished. (27, 28, 46). Even if the calf has not been fed, a spontaneous closure (cessation of uptake of macromolecules by intestinal cells) occurs by 24 hours of life (46).

There is also evidence that the higher the concentration of IgG in colostrum, the higher its absorption (12, 14, 22). There are major differences in colostrum IgG concentration between cows. A primary factor influencing IgG concentration is breed, with dairy breeds with higher total milk volume at first milking typically having a lower Ig concentration per unit

of colostrums than other breeds with lower total volume at first milking (12, 14, 22, 39, 46, 47, 53). Some authors have stated that the colostrum of younger cows and first-calving heifers has lower IgG concentration than colostrum from older cows (30, 37, 39). Similarly, calf serum IgG concentrations have been found to increase as the dam's age increase, reaching a maximum level at the 3rd or 4th calvings (34, 36, 37, 53).

Differentiating a high IgG concentration colostrum from low concentration colostrum is problematic (53). Because colostrum is also composed of other proteins, lipids, carbohydrates, minerals, hormones, growth factors and cells besides Ig, the Ig / total solids ratio becomes important because the higher the concentration of Ig the higher its potential for absorption (46, 47, 22, 48, 22, 23, 24). A lower ratio of Ig / total solids increases the competition for absorptive binding sites in the small intestine and the efficiency of absorption of Ig will be diminished.

Some works have shown that the natural act of suckling compared with bucket, nipple bottle or esophageal tube administration of colostrum enhances and increases the efficiency of absorption of Ig. This appears to be related to a larger amount of colostrum being consumed by the calf and also to the closure of the esophageal groove stimulated by suckling that favors Ig reaching the absorptive cells in the intestine more rapidly (48, 36, 24).

Conversely, some authors have shown high rates of FPT in dairy calves that are left with the dam and allowed to suckle naturally (1, 9), the explanation being that suckling dairy calves consume less concentrated colostrum. It is important to state that many times if the quality of colostrum is low (< 50 g/L IgG) the administration of 2 L of colostrum is probably insufficient to achieve adequate Ig transfer to calves. For that reason it is important to promote adequate suckling or ensure feeding of a sufficient amount of colostrum to reach the targeted quantity of Ig consumption during the first 4 hours of life.

Other factors like dystocia, environmental temperature, factors associated with the pre-partum period and nutrition management of the dam have been related with FPT (12, 34, 36, 43).

Dystocia or delayed parturition has been reported to diminish passive transfer of Ig to calves. Hypoxemia in the dystocic calf affects the absorptive capability of the small intestine (36, 53, 18). Also, affected calves sometimes have problems related to inability to stand and suckle (15, 36). Some studies have found intestinal lesions in neonatal calves that have suffered a prolonged dystocia, resulting in impairment in the absorptive capabilities of the enterocytes (26).

The environmental temperature the day of calving may influence the absorption of Ig. Some works have shown that high temperatures or "heat stress" decrease the rate of absorption of Ig (49, 15). Similarly, low temperatures or cold stress can negatively affect the rate of absorption because hypothermia decreases the blood flow to the small intestine and diminishes absorption capability of enterocytes (35, 15, 18, 21).

Another factor implicated has been the pre-partum nutrition of the dam. Some work has shown that the concentration of Ig in colostrum is 50% lower in overconditioned beef cows compared with properly conditioned beef cows (43). Other work has indicated that increased body condition score (>5) in first-calf beef heifers is associated with increased IgG levels in calf serum (34). Calves born to cows with poor udder conformation or a history of mastitis also should be categorized as high risk for FPT (37, 52).

Alternatives for prevention of failure of passive transfer:

Ongoing problems with FPT have created a renewed emphasis on colostrum-management programs and caused producers to look for alternative solutions such as colostrum supplements or colostrum replacers to provide passive immunity to neonatal calves.

Other on-farm colostrum

A common practice on some dairy and beef farms, and considered to be the best option, is the utilization of fresh or pre-collected frozen colostrum as the primary source of passive immunity to the calf. The objective is to offer an adequate amount of excellent quality colostrum shortly after birth. Since parity and lactation level can influence the colostrum

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concentration of Ig, it is important to always harvest the initially-available colostrum and determine whether it has sufficient Ig concentration to meet the needs of the calf. It is considering good quality colostrum when it has IgG levels > 50 g/L. An acceptable on-farm method to measure the IgG concentration is the use of a colostrometer or hydrometer to check the colostrum density. Some works have shown that a colostrum density > 1050 correlates with > 50 g/L of IgG (4, 38, 44).

The general recommendation is to administer about 200 g of IgG totally to achieve > 10 gr of IgG in the calf's blood; therefore, approximately 4 L of 50 g/L colostrum should be administered during the first 4 hours of life (19, 24, 44).

Maternal or on-farm pooled colostrum provides other nutrients and beneficial components in addition to its role as a source of IgG. Carbohydrates, fat, minerals, growth factors, cytokines, lactoferrin, and viable leukocytic cells are present in natural colostrum and play an important role in the development of immunologic capabilities of the neonate. It also provides specific antibodies against common on-farm and environmental pathogens, as well as vaccines to which the dam has been exposed, making it more effective in protecting the calf (4, 41, 50, 54).

Colostrum supplements and colostrum replacers

In cases of absence of the mother, poor colostrum quality, little frozen colostrum reserve or high resident herd incidence of potentially colostrum-transmitted infectious diseases such as John's Disease, Bovine Leukosis Virus (BLV), Salmonellosis, etc., producers may choose to use colostrum supplements or replacers to provide exogenous sources of IgG (13, 16, 24, 44).

Colostrum supplements and replacers are commonly derived from bovine lacteal secretions (milk whey or colostrum), eggs, bovine serum or from concentrated IgG from bovine plasma, all of which are available commercially (with the exception of products derived from concentrated IgG from bovine plasma). The IgG provided by these products are not specific against the common farm pathogens, but can provide generic protection against several key pathogens. Research studies done with these products show variable results – some works show an acceptable transfer of

immunoglobulins to calves (13, 19, 25, 40, 42, 44), while other commercial products tested did not provide adequate IgG transfer (4, 32, 33, 41, 54).

Factors like source of IgG, method of fractionation of IgG, non-Ig protein concentration, IgG/total solids ratio and inability to provide viable leukocytes, growth factors, cytokines and lactoferrin may affect the efficiency of absorption of colostrum supplements/replacers and may impair their ability to provide adequate immune transfer (13, 24, 33, 44, 46). Some authors have shown a decrease in live animal performance when maternal colostrums-fed calves were compared with colostrum supplement-fed calves (25).

Colostrum supplements

The colostrum supplements are commonly derived from whey and cow colostrum and usually contain very low concentrations of IgG per dose, usually 17-50 g IgG. They are not recommended to replace colostrum, but are recommended to mix with and improve low-quality colostrum (24). Generally the non-IgG protein concentrations of these products are very high and the ratio of IgG/total solids is very low. Many studies have shown a decreased efficiency of absorption of Ig when colostrum supplements are administered alone or when mixed with maternal colostrum (13, 15, 24, 32, 33, 41, 4). However, a few studies have shown adequate passive transfer with the use of colostrum supplements (5).

Many works have shown that the low amount of IgG compared with the high non-Ig protein and total solids present in these products compete and interfere with the efficiency of Ig absorption in the small intestine in the neonate (13, 41, 54). Also the addition of more colostrum supplement or the administration of two doses to increase the amount of IgG offered to calves decreases the efficiency of absorption even more. Some works have shown that this practice can reduce the Apparent Efficiency of Absorption (AEA) in 37% of calves so treated (13, 24). Reportedly the addition of bovine serum albumin to maternal colostrum reduced serum IgG concentration in newborn calves from 9.3 to 6.9 g/L (8).

Other work has shown that colostrum supplements as a single dose feeding or as a mixture with colostrum never achieve 10 gr of Immunoglobulin G in serum of

neonatal calves after 24 hours of life and that the average daily gain from 0-3 weeks of life is about 175 g/day less for the colostrum supplement-fed calves (32).

Some colostrum supplements derived from hyperimmunized chicken eggs containing immunoglobulin Y have shown low intestinal absorption in the calf and also a low levels in circulation.

Colostrum replacers

Colostrum replacers normally are derived from bovine serum or from IgG concentrates from plasma, and usually contain > 100 g of IgG per dose. These products are formulated to replace colostrums. Some studies have shown that colostrum replacers derived from concentrated IgG from bovine plasma can achieve high levels of IgG in newborn serum (13, 19, 40, 42). The explanation for the improved efficiency of absorption for colostrum replacers compared with colostrum supplements are the source and the concentration of IgG present in these products.

Some studies utilizing bovine serum-derived colostrum replacers have shown that a single dose will not achieve > 10 g of IgG in serum of neonatal calves, and that doubling the initial dose will further reduce the efficiency of absorption (40). This is possibly due to the effects of increase total solids and non-Ig protein in the total mass of product and its interference with the absorption process in the small intestine (4).

Other studies have used a non-commercially available, concentrated IgG product derived from bovine plasma, and reported adequate transfer of immunoglobulins with a single dose, and also an increase in the serum level of IgG from 11.6 to 13.6 g/L with a second dose of the product (50). These findings indicate that the colostrum replacers derived from concentrated IgG from bovine plasma are effective in preventing FPT in calves (13, 19, 25, 40, 42).

The most recent studies testing commercially-available colostrum replacers under field conditions have shown that they are not effective in achieving adequate serum levels of IgG in newborn calves, and in one of these studies 93.1% of the calves were considered FPT (< 10 g/L IgG) compared with maternal colostrum-

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fed calves. The consumption of roughly 170 g of IgG in the colostrum replacer was able to increase the serum IgG level only to 8 g/L on average (44, 50).

In various studies where colostrum replacers or colostrum supplements have been utilized, there have not been significant differences in the presentation of disease, frequency of treatments or mortality rates between calves fed these products when compared to maternal colostrum (32, 44, 50). It is possible that the total IgG level achieved by these products provide some degree of protection from disease. It is also possible that the calf's level of exposure to challenge microorganisms in these trials have been reduced.

Conclusions

The absorption of immunoglobulins by the newborn calf is a complex, nonselective process that can be influenced and affected by many factors; the most important of which are the amount and concentration of IgG in colostrum and the time at first feeding. The most effective method to prevent failure of passive transfer in calves is a colostrum-management program that assures the adequate amount of colostrum intake during the first 4 hours of life by promoting suckling or force feeding. The use of colostrum supplements or replacers should be restricted to special or emergency circumstances when maternal or on-farm colostrum is not available, and then a product with a high concentration of IgG must be selected. Additional research is needed to identify the interactions between the colostrum supplements and replacers and the absorptive process in the intestine to get a truly workable solution for the problem.

Factors like dystocia and environmental temperature (heat or cold stress) have to be considered as they increase the risk for failure of passive transfer. A good dam nutrition and vaccination program can also help to prevent the condition in neonatal dairy and beef calves.

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Vaccination debate prompts new strategies for dogs, cats

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Within the last decade, the frequency of small animal vaccinations, the efficacy of certain vaccines and the patient's need for various vaccines has become a topic of considerable debate. The impetus for the discussions was emerging evidence linking vaccines with fibrosarcomas in numerous cats. The ripple effect quickly spread to include dogs in the discussions although there was less specific evidence of long-term vaccine associated diseases in that species.

The increasing number of non-core, novel vaccines in the marketplace fueled over-vaccination concerns. The ensuing vaccine-related concerns have focused on the necessity of routine annual vaccines in adult dogs and cats combined with mounting evidence that vaccines can cause serious systemic diseases in both species.

Vaccinations are an important part of maintaining a healthy animal, however vaccine risk awareness, cost factors, antigen overload considerations, and regional disease incidences now require the owner and veterinarian to make rational and balanced vaccine selection decisions based on risk assessment of each patient. Among vaccine experts in academia, the trend has been to promote the concept of vaccine risk assessment for each patient, i.e. every patient does not automatically need every vaccine every year. Not surprisingly, many of the biological manufacturers have not endorsed or embraced this idea.

Allergic reactions to vaccines are relatively rare. Reactions occur as a result of an immediate or delayed hypersensitivity reaction to the antigenic component(s) of the vaccine. In addition, vaccine suspensions also may contain allergic components associated with production methodology that may include proteins from tissue culture or egg yolks. The allergic reaction also can occur from the antibiotics or preservatives present in the vial.

Allergic reactions can result in milder symptoms (localized swelling, systemic signs of depression, vomiting and/or diarrhea), to severe systemic shock and possible death. In addition to the more

obvious vaccine-induced allergic reactions is the less definitive group of systemic diseases that have also been postulated as being linked to vaccines. Unlike allergic reactions, the cause and effect of various canine vaccines with these diseases are less clear-cut, and the scientific basis for the relationship is often anecdotal.

Unfortunately the duration of immunity for each vaccine is a complex issue involving the particular antigen used, for example, MLV vs. killed agent; various manufacturing techniques including the adjuvant used; the individual patient's response to the vaccine, age of the patient, and previous vaccine history.

Traditionally, veterinarians have relied on vaccine manufacturers to provide the duration of immunity (DOI) data to the profession. Ironically the one-year re-vaccination recommendations for most canine and feline vaccines were not determined by a scientifically validated study. Except for rabies vaccination, we don't know the exact duration of immunity for most current canine vaccines produced because most DOI studies are based only on one-year serum titers extrapolations and not on timed experimental challenge.

Fortunately in the last four years, several companies have accepted the challenge of developing and gaining USDA approval of canine and feline three-year duration of immunity core vaccines. These vaccines have demonstrated both protective titers at three years post vaccination, and animals have withstood pathogenic viral challenges at three years post vaccination.

Although many veterinary clinics still recommend annual re-boosting to protect against the core diseases, the more progressive practices are now employing a three-year re-boost schedule advocated by most veterinary teaching hospitals, the AVMA, and recently the American Animal Hospital Association (AAHA). The basis for changing the traditional annual vaccine protocol recommendations to three-year programs and eliminating unnecessary vaccine was based on the premise that active immunization to most viral antigens probably persists for several years or perhaps even throughout the life of the patient. Arguably Leptospirosis,

Corona virus, Giardia, Lyme, and Bordetella vaccines are good for one year, if that long.

Selected Reading

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Winter hay feeding practices affect stable fly numbers

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It's time to remind cattlemen that stable flies attacking pastured cattle this spring and summer may have come from sites where round hay bales were fed to cattle during the winter. Over the last three decades there has been a dramatic increase in the population of these blood-sucking flies.

Formerly a major pest of livestock in confined-feeding operations such as dairies and feedlots, stable flies have extended their host range to attack pastured and range cattle. Cattle waste up to 50% of the hay when feeding on round bales. Mixed with cattle feces, wasted hay develops into an ideal habitat for stable fly larvae. As a result, stable fly populations reach major

damaging levels for about 8 weeks during spring and early summer and can cause a loss of 0.5 lb/head/day. Lower fly densities before and after this period also detrimentally affect cattle performance. Recent estimates of of stable flies' combined economic impact on dairy and pastured and confined beef cattle production show it to be greater than \$2 billion a year in the United States.

There is no effective method for controlling stable flies attacking pastured and range cattle. Cultural methods that reduce larval media by decreasing the amount of wasted hay at the round bale feeding site are recommended. Suggestions include the following:

- Use a hay feeding ring rather than placing the bale on the ground.

- Use cone feeders, which have a demonstrated ability to reduce the amount of wasted hay.
- Move hay feeders frequently to prevent accumulation of the hay-manure medium in one spot over time.
- Unroll the round bales on pastures, changing sites each time.
- Spread accumulated hay-manure medium to allow it to dry out.

Although stable flies are good fliers capable of dispersing up to 155 miles on prevailing winds, population levels pestering a given herd depend on the number of flies emerging from round bale feeding sites in the vicinity. Cultural methods may not be effective if neighboring ranches do not prevent the development of large populations of stable flies on their premises.

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The Kansas State University Diagnostic Laboratory and Department of Animal Sciences and Industry at Kansas State University greatly appreciates the sponsor(s) of the Kansas Veterinary Quarterly Newsletter. These sponsorships in no way imply the Departments' endorsement of the products and services offered by the sponsors. The Departments welcome inquiries from other individuals, associations and firms that may be interested in cosponsoring this publication.

DESIGNING BIOSECURE SYSTEMS TO PREVENT NEONATAL CALF DIARRHEA

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Diarrhea remains an important cause of morbidity and mortality of neonatal beef calves. The economic effects of neonatal calf scours can be profound. Some beef cattle herds annually experience mortality rates of 5 to 10 percent or greater, sometimes with near 100 percent morbidity. Economic costs to the disease include loss of performance, mortality, and the expense of medication and labor to treat sick calves. In addition, herd owners and their employees often become disheartened after investing long hours to treat scouring calves during an already exhausting calving season.

The Sandhills System for Calf Scours Prevention

Our objective was to prevent neonatal calf diarrhea in ranch systems typical of the Nebraska Sandhills. Our strategy was to design calving systems to prevent calves from making effective contact with scours pathogens. An effective contact is an exposure to pathogens of a dose-load or duration sufficient to cause disease. Effective contacts can be prevented by physical separating animals, reducing the level of exposure (e.g. through the use of sanitation or dilution over space), or minimizing contact time. These actions have been successfully applied in calf hutch systems to control neonatal diseases in dairy calves. The actions we defined as the Sandhills Calving System (Figure 1) were to prevent effective contacts by:

- 1) Segregating calves by age to prevent direct and indirect transmission of pathogens from older to younger calves
- 2) Routinely moving pregnant cows to new calving pastures to minimize pathogen dose-load and contact time

Case Herd #1 was an 800-900-cow beef herd. Calving began each year in early March. Cows typically calved in calving lots and the calf and dam were “paired-out” into larger pastures. The mortality rate due to scours was 14 percent and 6.5 percent in 1995 and 1996, respectively. In 1999 the scour mortality rate was 8.2 percent. Medical records were not available for 1997 and 1998 but the ranch owner reported that losses due to scours were also within this range. Veterinary expenses during the calving season averaged \$3114.18 per year from 1995 to 1999. Veterinary expenses were primarily for antibiotics and fluids for the care of scouring calves. Calf scours mortality rates were unaffected by supplementing the cow herd with trace minerals.

The Sandhills Calving System was adopted in 2000 and this plan was continued in 2001 and 2002. In the new system large contiguous pastures were used as calving pastures. Cows

were turned into the first calving pasture (Pasture 1) as soon as the first calves were born. Calving continued in Pasture 1 for two weeks. After two weeks the cows that had not calved were moved to Pasture 2. Cow-calf pairs remained behind in Pasture 1. After a week of calving in Pasture 2 the cows that had not calved were moved to Pasture 3 and cow-calf pairs born in Pasture 2 remained in Pasture 2. Each subsequent week cows that had not calved were moved to a new pasture and pairs remained in their pasture of birth. The result was multiple pastures each with calves within one week of age of each other. Cattle from different pastures could be commingled after the youngest calf was four weeks of age.

In the three years since implementing the Sandhills Calving System illness and death due to calf scours decreased significantly ($p < 0.01$). No calves died from neonatal calf scours in since beginning the system. Four calves were treated for scours in 2000 and no calves have been treated for scours since. Veterinary expenses incurred during the calving seasons of the past 3 years have averaged \$128.83 per year, a 24-fold reduction from previous years ($p < 0.01$). The owner estimated savings of \$40,000 to \$50,000 per year attributable to greater numbers of weaned calves, improved calf performance, and reduced expenses for treatment.

Case Herd #2 was a 400-cow beef herd using rotational grazing and early summer calving. Calving in this herd occurred as the cows moved through a series of pastures every two to four days. This herd experienced 6.5 percent (28 deaths /433 births) and 11.9 percent (48 deaths/402 births) mortality in 1999 and 2000, respectively. Deaths were primarily due to neonatal calf scours.

The Sandhills Calving System was adopted in 2001 and continued in 2002. The system differed slightly from that of Case Herd #1 to meet the requirements of the intensive pasture grazing system. Groups of cattle moved to different pastures throughout the calving season as appropriate for forage utilization. However, every 10 days or whenever 100 calves were born the herd was divided by sorting cows that had not calved from the cow-calf pairs of the preceding group. In this manner the number of calves within any pasture group never exceeded 100 and all calves within a group were within 10 days of age of each other. Pasture groups were commingled after the youngest calf was four weeks of age.

Death loss was significantly reduced in 2001 and 2002 compared to previous years ($p < 0.01$). No calves died of neonatal scours in 2001 or 2002. Total death loss was 2.3 percent (8 deaths /398 births) and 1.5 percent (5 deaths /340 births) in 2001 and 2002, respectively.

Discussion

Neonatal calf scours is a multifactorial disease. Agent, host, and environmental factors play important roles in the occurrence of disease and knowledge of these factors become the basis for intervention to control the disease. Numerous infectious agents have been recovered from scouring calves. Common agents of neonatal calf scours include bacteria such as *Escherichia coli* and *Salmonella*, viruses such as rotavirus and coronavirus, and protozoa such

as cryptosporidia. Bovine rotavirus, bovine coronavirus and cryptosporidia are ubiquitous to most cattle populations and can be recovered from calves in herds not experiencing calf diarrhea. Further, multiple agents can be recovered from herds experiencing outbreaks of calf diarrhea suggesting that even during outbreaks more than one agent may be involved.

Calves acquire passive immunity against the common agents of calf scours after absorbing antibodies from colostrum or colostrum supplements. The quality and quantity of colostrum ingested largely influences the level of passive protection. The presence of antibodies in colostrum directed against specific agents requires prior exposure of the dam to the agent. Vaccines are often used to immunize the dam against specific agents and some commercially available colostrum supplements contain polyclonal or monoclonal antibodies directed against specific agents. Vaccination or the use of colostrum supplements has not been universally successful for controlling neonatal calf scours.

The age of the host appears to be an important factor of neonatal calf scours. Calves become ill or die from scours within a small range in age (Figure 2). Age-specificity may not be explained solely by the incubation period of the agents, because disease is observed in colostrum-deprived calves within 48 hours of virus inoculation regardless of age. It is possible that calves become more susceptible to disease as the amount of antibodies bathing the gut wanes.

The conditions of the environment may influence both the level of pathogen exposure and the ability of the calf to resist disease. Ambient temperature (e.g. excessive heat or cold) and moisture (e.g. mud and snow) are important stressors that impair the ability of the calf to resist disease. Environmental exposure to pathogens may occur through direct contact with other cattle or contact with contaminated environmental surfaces. The exposure level (i.e. dose-load) of pathogens in the environment is a function of both animal density and the multiplier effect of sequential infections. Crowded conditions facilitate the number of effective contacts with infected animals or contaminated surfaces. Over time environmental pathogen contamination may increase especially when conditions favor survival of the agent such as during wet, cool weather common to springtime calving. Further, with sequential infections (e.g. dam to calf, older calf to younger calf...) the level of pathogen shedding may increase to greater levels. Eventually the dose-load of exposure may exceed the calf's ability to resist disease. These factors alone or in combination may explain observations that calves born later in the calving season are at greater risk for disease or death (Figure 3).

The Sandhills Calving System as applied to these herds was designed to prevent effective contacts by using clean calving pastures, preventing direct contact between younger calves and older calves, and preventing later born calves from being exposed to an accumulation of pathogens in the environment. However, the actions taken to implement the system differed slightly between herd to meet the specific needs of each production system. Key component of the systems in both herds was age segregation of calves and the movement of gravid "heavy" cows to new pastures rather than "pairs". Age segregation prevents the serial passages of

pathogens from older calves to younger calves. The design of the system to routinely move heavy cows to new calving pastures prevents the build up of pathogens in the calving environment over the course of the calving system and the resultant exposure of the latest born calves to an overwhelming dose load of pathogens.

The Sandhills Calving System afforded some additional benefits to management. For example Herd #1 realized some labor efficiency because cattle movement could be scheduled once a week when additional labor was available. Moving cattle without calves to a new pasture was easier than moving individual cow-calf pairs. Also, the workload was partitioned between pasture groups such that cows at risk for dystocia were together in one pasture while the calves at risk for scours risk were in another. Information from pregnancy examination, when available, enabled sorting cows into early and later calving groups. Cows expected to calve later in the season can be maintained elsewhere and added to the calving pasture as appropriate, thereby reducing the number of cattle moving through the initial series of pastures.

After implementing the Sandhills Calving System in these two ranch herds we observed important reductions in the morbidity and mortality due to neonatal calf diarrhea. The reduction in illness and death has been consistent over five calving seasons. We concluded that the Sandhills Calving System effectively prevented illness and death due to neonatal calf diarrhea.

Week 5

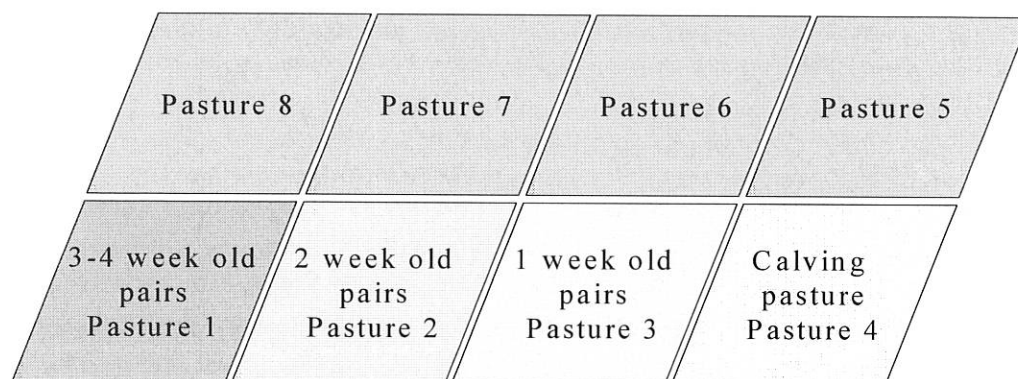


Figure 1. Schematic of the Sandhills Calving System in the fifth week of the calving season. During Week 5 cows are calving in the 4th pasture and calves born in the previous pastures remain behind in age-related groups.

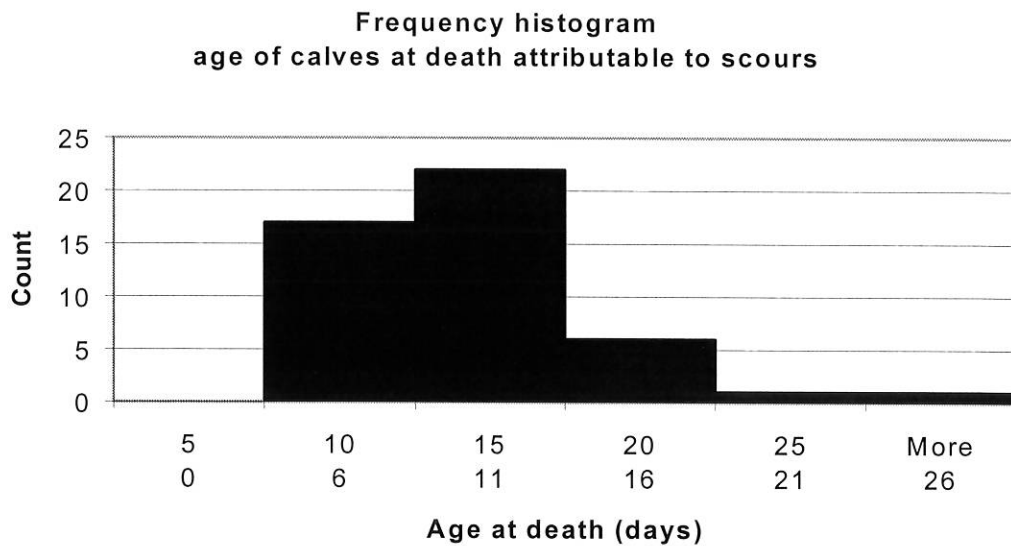


Figure 2. Frequency distribution of the age calves died from scours. Most calves died between 6 and 15 days of age. Data are from Case Herd #2 prior to implementing the Sandhills Calving System.

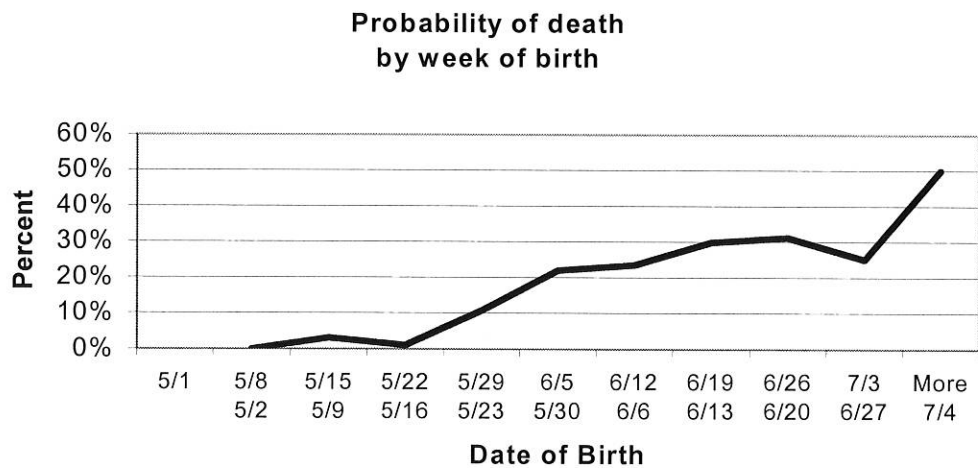


Figure 3. The proportion of calves born each week that subsequently died due to scours. Calves born later in the calving season had increasingly greater risk of death. Data are from Case Herd #2 prior to implementing the Sandhills Calving System.

NEONATAL CALF SCOURS: PATHOPHYSIOLOGY AND THERAPY

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INTRODUCTION:

Neonatal calf scours is a problem encountered by many practicing veterinarians who provide veterinary care for beef and/or dairy cattle operations. Therapy of individual sick calves may result in favorable outcomes if an understanding of the disease processes and their therapies are known. In this presentation, pathophysiology of disease, clinical signs, diagnosis, and fluid resuscitation therapy will be discussed. Etiologic agents associated with neonatal calf scours will be individually discussed; however, the interaction of multiple pathogens in inducing disease will be emphasized.

Diarrheal disease of young calves in a herd is a complex, multifactorial problem that is complicated by the interplay of multiple causative agents, host immunity, environmental factors, nutrition factors, and management conditions. Truly, to say that agents cause disease in neonatal calf scours is an error, as many of our cattle operations possess these pathogens that can cause disease. The presence of disease is often the result of environment and host factors. Example host factors include passive transfer status of calf, cow vaccination status, age of dam, nutritional status of cow and calf, including vitamin and mineral nutrition.

Risk factors associated with neonatal calf scours from an environmental perspective include housing conditions and atmospheric conditions such as ambient temperature, precipitation, wind chill, and ventilation. The physical environment, housing density, general hygiene, and hygiene related to feeding practices all impact upon whether disease will occur or not. Some things are under the control of management. Others are not, such as weather.

The most important host factor associated with neonatal calf scours is failure of passive transfer. Feeding the proper amount of colostrum soon after birth will prevent significant mortality in most dairy farm situations. Proper nutrition of the dam during late gestation is another area of concern for development of neonatal calf scours.

CAUSATIVE AGENTS OF NEONATAL CALF DIARRHEA

Numerous etiologic agents have been associated with causation of neonatal calf scours. We often separate these pathogens into 3 major groups: viral, bacterial, and protozoan. Bacterial pathogens include *Escherichia coli*, *Salmonella* spp, *Clostridium* spp, *Bacteroides fragilis*, *Campylobacter fetus ss jejuni*, *Enterococcus durans*, and *Yersinia enterocolitica*. Viral pathogens include rotavirus, coronavirus, atypical rotaviruses, parvoviruses, caliciviruses, astroviruses, and BVDV. The primary protozoal parasites include *Cryptosporidium parvum* and *Eimeria bovis* and *Eimeria zurnii*.

Although many different etiologic agents can cause diarrhea, most cases of diarrhea are caused by 5 major pathogens, and these include *Escherichia coli*, *Salmonella* spp

rotavirus, coronavirus, and *Cryptosporidium parvum*. Table 1 provides a listing of these 5 pathogens along with their characteristic clinical findings, methods for diagnosis, and common age group of calf affected.

Table 1: The 5 major causative agents of neonatal calf scours

Agent	Age	Appearance of Feces	Pathophysiology	Diagnostics
ETEC	1-3 days to 7 days	Watery	Secretory	Culture; slide agglutination; ELISA; DNA probes; PCR; histopathology; IFA
<i>Salmonella</i> spp.	Peracute: 5-10 days; acute 5-10 days; chronic > 10 days	Systemic signs Watery; hemorrhagic; dysentery	Inflammatory; heat-labile toxin (secretory)	Culture; serology; PCR
Rotavirus	< 3 days nonclinical carriers; 4-7 days mild diarrhea; > 7 days mild to moderate diarrhea	Slight diarrhea to profuse watery diarrhea	Malabsorptive; maldigestive	ELISA; electron microscopy; IFA
Coronavirus	Usually > 10 days	Moderate to severe diarrhea	Malabsorptive; maldigestive	Electron microscopy; IFA
<i>Cryptosporidium parvum</i>	4-21 days	Mild to severe diarrhea	Malabsorptive; secretory ??	Direct microscopy; FA; ELISA; diagnostic stains

***Escherichia coli*:** Enteric colibacillosis has for many years been considered a common clinical disease of neonatal calves. Strains of enterotoxigenic *E. coli* (ETEC) cause the disease. Two essential virulence properties are expressed by ETEC: 1) expression of a fimbrial antigen that allows the bacteria to attach to intestinal epithelial cells, and 2) elaboration of exotoxins that influence intestinal excretion of fluid. Both virulence factors are necessary to establish a secretory diarrhea. Other strains of *E. coli* are associated with calf diarrheal diseases, but the ETEC are the most common. These other strains include attaching and effacing *E. coli* (AEEC), necrotoxicogenic *E. coli* (NTEC), and enterohemorrhagic *E. coli* (EHEC), which include O157:H7 and others which are recognized as human pathogens.

The clinical presentation associated with colibacillosis is characterized by a profuse, effortless, watery diarrhea. Most calves with colibacillosis are less than a week of age, and some calves may show clinical signs of disease within 24 hours of birth. In the acute stages, affected calves become dehydrated rapidly, and develop signs referable to hypovolemic shock. These signs include subnormal body temperatures, tachycardia, weakness, depression, and recumbency. Clinical pathologic evaluation usually reveals a metabolic acidosis, pre-renal azotemia, and hyperkalemia.

Detection of colibacillosis requires special laboratory assays to identify the specific *E. coli* strains and their virulence factors. The most practical procedure, yet least reliable, is demonstration of the fimbrial antigen on strains isolated from feces or intestinal tissues. An enzyme immunoassay is available to detect the common fimbrial antigen, K99.

Therapy for the individual calf often relies on intravenous fluid therapy if the animals are recumbent and severely ill. Oral rehydration solutions can be utilized in less severe cases or in cases that are early in their clinical progression.

Vaccines are available for prevention and control of colibacillosis. Pregnant cattle can be immunized in late gestation with specific fimbrial antigen containing vaccines. The effect of this vaccination is to booster specific immunoglobulins in the colostrum. As expected, these vaccines are only as effective as the colostrum program. In calves, monoclonal antibodies are available against the K99 fimbria, and these prevent fimbrial attachment to epithelial cells.

***Salmonella* spp:** *Salmonellae* infections are considered the most important bacterial cause of enteric diseases in calves greater than 10 days of age. There are several different manifestations of disease in calves: peracute septicemia; enteric (acute to subacute diarrheal disease); and chronic. Over 2,200 different serotypes of *Salmonella enterica* exist, but most infections in cattle are due to *S. dublin* and *S. typhimurium*. *S. dublin* is the host-adapted serotype for cattle, whereas *S. typhimurium* lacks host specificity.

Clinical signs of disease can be quite diverse. Enteric salmonellosis is characterized by watery diarrhea, central nervous system depression, fever, anorexia, and dehydration. The feces may have a putrid odor and may contain blood, mucus, and intestinal casts. These signs are more characteristic of *S. typhimurium* infection, while *S. dublin* often presents as the septicemic form in neonatal calves.

Detection involves culturing the organism from tissues or feces using selective enrichment media. If salmonellosis is suspected, submitting 2-5 grams of fresh feces collected from the rectum into sterile containers provides the best chance for identifying the organism. In chronic cases, submission of multiple fecal samples may be necessary for identification. With enteric salmonellosis, the presence of a pseudomembranous colitis often provides evidence.

Therapy involves supportive care, including fluids. There is significant debate as to whether to use antibiotics in salmonellosis suspect cases. There is concern that antibiotics may prolong the carrier state. In addition, there are concerns about resistance patterns in the *Salmonellae*, and it is known that *Salmonella* spp rapidly develop R-factor resistance to various antibiotics.

Rotaviral enteritis: Rotavirus is considered to be the most common causes of diarrhea in neonatal calves. In general, rotaviral enteritis is a high morbidity, low mortality disease. Group A and B rotavirus can infect calves, but most infections occur with group A rotaviruses. Rotaviruses are nonenveloped double-stranded RNA viruses. Due to the fact, rotaviruses are nonenveloped, they can survive in the environment longer than other viruses.

Clinical signs of disease vary considerably. Rotaviruses can be found in the feces of most adult animals without overt clinical disease. Variations in clinical disease are likely due to differences in virulence, age and immune status of host, the dose of the inoculum, and environmental stressors. In addition, clinical disease with rotavirus is more likely to occur in the presence of mixed infections with other pathogens, specifically *Cryptosporidium parvum*. Fortunately, most rotaviral enteritis cases are mild and self-limiting.

Detection often involves demonstration of viral particles using electron microscopy or using rapid diagnostic EIA tests (Rotazyme EIA tests). Atypical rotaviruses are often undetectable using the commercial EIA tests. For postmortem intestinal tissues, immunofluorescent antibody procedures are available to rapidly diagnose rotaviral enteritis.

Therapy is often directed at restoring hydration, correcting acid-base disturbances, and correcting electrolyte abnormalities.

Vaccines are available for prevention and control of rotaviral enteritis. Pregnant cattle can be immunized in late gestation in order to booster specific immunoglobulins in the colostrum. As expected, these vaccines are only as effective as the colostrum program. In calves, modified live vaccines are available to stimulate specific local immune responses. These vaccines must be given prior to the administration of colostrum, as immunoglobulins in colostrum will neutralize the vaccine virus.

Coronaviral enteritis: Coronavirus is less widespread than rotavirus. The pathogenesis of coronaviral enteritis is similar to that of rotavirus. Coronaviruses are enveloped, RNA viruses. Coronaviruses replicate in the villus epithelium and crypt epithelium of the small and large intestine, resulting in a malabsorptive, maldigestive type of diarrhea. Due to the fact coronavirus infects crypt epithelia, longer rejuvenation times for re-epithelialization of the intestine may be observed.

Clinical signs of disease are usually more severe and longer-lasting than that observed with rotavirus. Calves are commonly affected between the ages of 5-21 days, but in general, coronavirus commonly affects calves older than what would be expected with rotavirus. Diarrhea and depression proceed through a course of 4 to 5 days. Mild respiratory disease can be observed concurrently in populations of calves affected with coronaviral enteritis.

Detection of coronavirus can be accomplished using electron microscopy of fresh feces or immunofluorescent antibody examination of chilled intestinal specimens. For coronavirus, sections of spiral colon are preferred.

Therapy for coronaviral enteritis is similar to that for rotavirus. Fluid therapy, often for a prolonged period of time or repeatedly may be necessary to treat calves affected by coronaviral enteritis.

Vaccines are available for prevention and control of coronaviral enteritis. Pregnant cattle can be immunized in late gestation in order to booster specific immunoglobulins in the colostrum. As expected, these vaccines are only as effective as the colostrum program. In calves, modified live vaccines are available to stimulate specific local immune responses. These vaccines must be given prior to the administration of colostrum, as immunoglobulins in colostrum will neutralize the vaccine virus.

Cryptosporidium parvum: *Cryptosporidium parvum* has become one of the most important food animal pathogens due to enteric infections in calves but also due to the potential for human disease. *C. parvum* is considered a ubiquitous pathogen, as estimates indicate greater than 90% of cattle operations harbor this parasite. Four characteristics distinguish *Cryptosporidium parvum* from other coccidian protozoa: 1) they lack host specificity; 2) they have an intracellular yet extracytoplasmic location within host cells; 3) oocysts contain four naked sporozoites; and 4) oocysts are fully sporulated before passage in the feces. Oocysts of *C. parvum* are small (4-5 μm), and the parasite can survive for prolonged periods in the presence of organic debris. In addition, most conventional disinfectants are ineffective in destroying oocysts.

The clinical signs of disease are similar to what has been described for the other pathogens. Diarrhea is frequent in infected calves, along with increased frequency of defecation, tenesmus, anorexia, emaciation, and dehydration. Neonatal animals are most susceptible, and resistance

develops with increasing age. In addition, more severe clinical disease is observed in younger calves.

Detection of *C. parvum* is accomplished by demonstration of oocysts in feces or examination of stained intestinal tissue. Fecal examination is performed on fecal samples collected from the same animal on a daily basis. Some animals shed oocysts intermittently. Mixed infections are quite common with *C. parvum*, and it is common to find ETEC, rotavirus, and coronavirus in affected calves.

Therapy involves supportive care and intravenous fluids. Since the disease is malabsorptive and maldigestive in nature, it is important to provide nutritional support to affected calves.

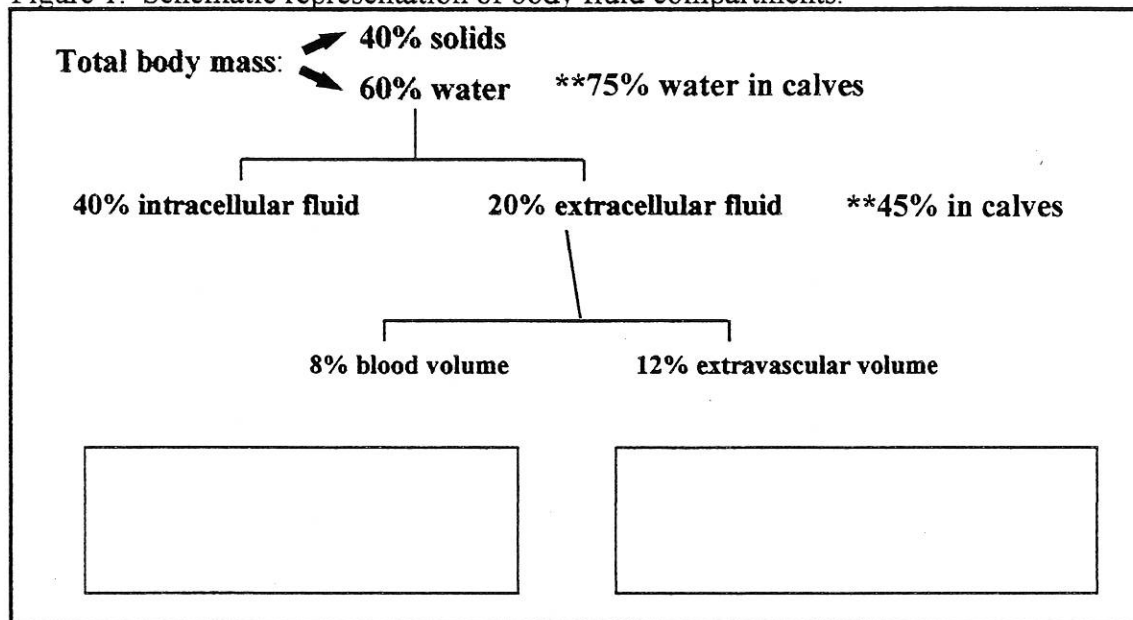
THERAPY FOR THE INDIVIDUAL CALF WITH NEONATAL SCOURS

PATIENT ASSESSMENT:

The physiology of fluid: The immediate objective in dealing with a sick, dehydrated neonatal calf is to restore the diarrheic calf to a normal systemic state. First and foremost is to provide fluid therapy. Indications for fluid therapy include: severe dehydration, severe acid-base and/or electrolyte disturbances, septic shock, blood loss, and diuresis. The goals of fluid therapy are as follows: restore fluid volume, correct metabolic acidosis, correct mental depression, restore suckle reflex, correct electrolyte abnormalities, correct energy deficit, and facilitate repair of damaged intestinal surface.

In order to administer fluids and electrolytes, one must understand body fluids. Total body water exists as extracellular fluid and intracellular fluid. The majority of fluid exists intracellularly with approximately half as much in the extracellular fluid space. When compared to adult cattle, calves are in essence, composed of more water per unit weight.

Figure 1: Schematic representation of body fluid compartments.



Within the fluid compartments, electrolyte differences also exist. Potassium is the major intracellular ion, while sodium and chloride are mainly found in the extracellular fluid. When fluids are administered, we are replacing losses of fluid from the extracellular fluid compartment,

and thus, the fluids being administered must contain concentrations of ions similar to what is found in the extracellular fluid compartment.

Handling the sick diarrheic calf: Regardless of the specific inciting cause of diarrhea, 4 specific metabolic changes can be expected in calves with neonatal scours:

- 1) dehydration
- 2) acidosis
- 3) electrolyte abnormalities
- 4) negative energy balance (hypoglycemia). NOTE: Of these 4 abnormalities, I have found that most calves with diarrhea are not hypoglycemic, but they are in the process of breaking down fat for energy. They are in negative energy balance.

Therefore, when discussing the specific therapy for the individual sick calf, all 4 of these metabolic derangements need to be addressed. This proceedings material now focuses on each of these 4 derangements.

Patient assessment of dehydration: In calves with diarrhea, there is a net loss of fluid in the form of fecal fluid loss. This loss of fluid can approach 13% of their body weight over the course of 48 hours. Diarrheic calves lose fluid from the extracellular fluid space first. The body initially responds to this fluid loss by withdrawing fluid from tissues in order to maintain an effective circulating blood volume. This “pulling” of fluid from the interstitial and intracellular fluid spaces results in loss of skin elasticity, dry mucous membranes, and sunken eyeballs.

The methods to assess hydration status in calves are semiquantitative at best. Most often, we assign calves a percent dehydration based upon clinical examination. Measuring the packed cell volume (PCV) and total plasma proteins (often referred to as total solids) are sometimes used, but these can often be misleading since the normal ranges for PCV and TPP in young calves is quite wide. In addition, the TPP can vary based upon the amount of colostrum intake. Since this is largely unreliable, we simply use clinical assessment as follows:

Table 1: Assessment of hydration status in calves.

% Dehydration	Eyeball sunkenness	Neck skin tent (seconds)	Mucous membranes
0	None	<1	Moist
1-5	None/slight	1-4	Moist
6-8	Slight separation of eyeball and globe	5-10	Tacky
9-10	Gap, <0.5 cm, between eye and orbit	11-15	Tacky to dry
11-12	Gap, 0.5-1.0 cm, between eye and orbit	16-45	Dry

Patient assessment of acidosis: Assessing the acid-base status of young neonatal calves is more difficult from a clinical assessment perspective. A quick and accurate assessment of acidosis can be achieved through the use of a blood-gas analyzer. Most practices do not possess a blood-gas analyzer, and if so, there is limited use of such equipment in the field. Therefore, in most situations, the degree of acidosis will be estimated. Based upon evaluation of many diarrheic calves, Dr. Jonathan Naylor (University of Saskatoon, Saskatchewan, Canada) developed a clinical scoring system for degree of acidosis. Young calves (calves less than 8 days of age) will

generally have **less severe** acidosis than calves greater than 8 days of age. Calves with severe diarrhea that are semi-comatose and recumbent will have greater acidosis than calves still standing and nursing. In general, calves with severe diarrhea that are less than 8 days of age will have a base deficit of 10-15 mEq/L, whereas older calves with the same degree of diarrhea and clinical signs will have a base deficit of 15-20 mEq/L (See Table 2).

Table 2: Assessment of acidosis in neonatal calves.

Clinical Description	Base deficit for a calf less than 8 days of age	Base deficit for a calf greater than 8 days of age
Standing, strong suckle reflex	0	5
Standing, weak suckle reflex	5	10
Sternal recumbency	10	15
Lateral recumbency, semi- comatose	10	20

Patient assessment of electrolyte abnormalities: Assessment of electrolyte derangements is very difficult without the use of laboratory analyses. We therefore, make several assumptions when dealing with calves with diarrhea in field situations. Most calves with neonatal diarrhea will have normal to low sodium (hyponatremia), normal to low chloride (hypochloremia), and elevations in blood potassium (hyperkalemia). By providing isotonic saline, the sodium and chloride may be addressed. With respect to potassium, calves are usually hypernatremic as a result of acidosis, yet they have a total body depletion of potassium due to loss of potassium in the fecal fluid. We therefore will often empirically administer potassium once the acidosis is corrected.

It is important to note that assumptions such as these can also lead to treatment failures. An example is the calf that has received inappropriate mixtures of oral electrolytes and the calf has extremely high blood sodium (hypernatremia).

Patient assessment of hypoglycemia: Most calves that have neonatal calf scours are in negative energy balance because of malabsorption and maldigestion, inadequate energy intake due to inappetance, and an increased metabolic demand such as encountered in cold weather. When referred to a hospital, most calves are not hypoglycemic though. Clinical signs of hypoglycemia include weakness, lethargy, convulsions, and eventually coma and death. Inclusion of dextrose and oral energy sources once a suckle reflex is restored is usually part of the therapy.

THE FLUID THERAPY PLAN:

Once assessment of the patient has been completed, the next step is to devise a plan of action for restoring the dehydrated, acidotic, hypoglycemic calf to a normal systemic state. Remember to consider the 4 major metabolic derangements when working up the neonatal calf with diarrhea: dehydration, acidosis, electrolyte abnormalities, and hypoglycemia.

Correction of dehydration: When devising a fluid therapy plan, it is important to consider 3 main ingredients: *replacement fluids, maintenance fluids, and fluids associated with ongoing losses in diarrhea*. Calculation of replacement fluid therapy is easy. The first priority for treating the dehydrated calf is to restore the extracellular fluid volume back to normal. Simply use your assessment of dehydration and multiply by the body weight in kilograms. This will give you the amount of fluid in liters that is required to restore an animal to a normal hydrated state.

$$\text{Replacement} = \% \text{ dehydration} \times \text{body weight (kg)}$$

Calculation of maintenance fluids is based upon the physiologic requirements of the animal. It is critical to include both maintenance and ongoing fluid losses in the calculations, because many an animal will slip back into a dehydrated state if fluids for maintenance and ongoing losses are not accounted for.

$$\begin{aligned} \text{Maintenance} &= 50 \text{ ml/kg/day} \\ \text{Or} \\ &20\text{-}30 \text{ ml/lb/day} \\ \text{OR} \\ &1 \text{ ml/lb/hour} \end{aligned}$$

Ongoing losses from diarrhea can be substantial in calves with profuse, watery diarrhea. Calves with enterotoxigenic *E. coli* can lose additional fluid in the fecal volume even when rehydration has been performed.

$$\text{Ongoing losses in diarrhea} = 1\text{-}4 \text{ L / day}$$

As an example, a 9 day-old purebred Angus calf is brought to your clinic for neonatal scours. The calf is in lateral recumbency, with severely sunken eyes. You estimate the calf to weigh 125 pounds (60 kilograms). Based upon physical examination, you estimate the calf to be 10% dehydrated. The replacement fluid needed to restore this calf to normal hydration would be: $0.10 \times 60 \text{ kg} = 6 \text{ liters}$. The daily maintenance fluid requirement for this calf would be: $50 \times 60 \text{ kg} = 3000 \text{ ml}$ (**3 liters**). Ongoing losses for this calf would be anywhere from **1-4 liters**. So, over the course of 24 hours, this calf would require roughly 13 liters of intravenous fluids.

Correction of metabolic acidosis: Once the fluid volume depletion has been addressed, the next item to care for is acidosis. Providing fluids will help correct metabolic acidosis, but calves with diarrhea have an absolute requirement for alkalinizing agents such as bicarbonate. The reason for this is the significant loss of bicarbonate in the feces of calves with diarrhea. Acidosis can be corrected by providing intravenous bicarbonate and alternatives to bicarbonate such as lactate (such as is found in lactated Ringer's solution), acetate, gluconate, propionate, and citrate. When it is all said and done, bicarbonate is still the best choice for treating acidotic calves.

Several different formulations of bicarbonate exist for intravenous fluid administration. Sodium bicarbonate (NaHCO_3) intravenous solutions can be purchased at two different concentrations: 5% NaHCO_3 and 8.4% NaHCO_3 . Isotonic bicarbonate is 1.3%, so both of the sterile, bottled bicarbonate sources are hypertonic. The quantity of bicarbonate ions in the 5% solution is 0.6 mEq/ml, whereas the quantity in the 8.4% solution is 1 mEq/ml.

Calculating the quantity of bicarbonate needed to restore the patient to a normal base status can be done by clinical examination. See table 2 above. The following formula is important for determining the quantity of bicarbonate needed to restore the patient to a normal base status:

$$\text{Bicarbonate needed} = \text{Body wt. (kg)} \times 0.5 \text{ (liters/kg)} \times \text{Base Deficit (mEq / liter)}$$

=mEq of bicarbonate needed to restore neutrality
=This quantity to be given IV to correct acidosis

The base deficit is determined from the clinical examination using table 2. The 0.5 value in the equation is an estimate of the extracellular fluid volume space for a calf (see figure 1).

Let's go back to our Angus calf example. The calf is in lateral recumbency and semi-comatose. Using table 2, you estimate the calf to have a base deficit of 20 mEq, since it is greater than 8 days of age and is in lateral recumbency. The amount of bicarbonate needed to restore this calf to a normal base status is: $60 \text{ kg} \times 0.5 \times 20 \text{ mEq} = 600 \text{ mEq}$ of bicarbonate.

Correction of electrolyte abnormalities: As stated previously, most calves with diarrhea are hyperkalemic, but actually have a total body depletion of potassium. In most situations, we assume these calves have a total body depletion of potassium and we supplement potassium in the fluids. Potassium can be cardiotoxic. So long as potassium administration does not exceed **0.5 mEq/kg/hour**, it is safe to give calves potassium. Most frequently, potassium is administered into the fluids at 20-40 mEq of potassium per liter. There is little danger in adding 20mEq of potassium per liter of fluids.

Correction of hypoglycemia: Administration of solutions containing dextrose will correct hypoglycemia. Addition of glucose to intravenous fluids does seem to provide clinical benefit. Bottles of 50% dextrose can be purchased, and dextrose can be added to fluids to make a 2.5% or a 5% solution. Isotonic dextrose is 5%.

FORMULATING A SOLUTION FOR INTRAVENOUS ADMINISTRATION:

Numerous solutions are available for intravenous fluid therapy in calves. Several important criteria exist for choice of fluids, or how fluids are made. Intravenous fluids must have an osmolality equivalent to plasma. This means the sodium and chloride levels should be very close to what is found in plasma, as sodium is the major component in serum osmolality. There are numerous fluids available, and the following table lists the main choices and components in each. Most of these fluids can be purchased from veterinary distributors at reasonable prices.

Table 3: Available fluids

Fluid type	Na ⁺	K ⁺	Ca ⁺⁺	Mg ⁺⁺	Cl ⁻	HCO ₃ ⁻	Osmolality
<i>Plasma</i>	<i>133-144</i>	<i>3-5</i>	<i>2.5-4</i>	<i>0.8-3</i>	<i>93-104</i>	<i>22-28</i>	<i>280-300</i>
LRS	130	4	3	0	109	28 (lactate)	274
0.9% NaCl	154	0	0	0	154	0	308
5% Dextrose	0	0	0	0	0	0	250
1.3% NaHCO ₃	156	0	0	0	0	156	312
PlasmalyteA	140	5	0	3	98	27,23 (acetate, gluconate)	294

At times, we deal with animals of limited financial value. In situations such as these, the price of fluid therapy for the disease can be more than the inherent value of the calf. In these situations, fluids can be mixed with readily available ingredients with less cost than purchasing preformed fluid products. At the Kansas State University Teaching Hospital, we often formulate our own intravenous fluids. All that is needed are: gallons of distilled water, baking soda, noniodinized table salt, 50% dextrose bottles, and reagent grade potassium chloride. The methods to formulate these homemade fluids are presented in table 4. **NOTE: It is critical that these solutions are made appropriately, as administration of hypotonic or hypertonic fluids may results in severe electrolyte derangements and possible death of the patient.**

Table 4: Formulation of "homemade" intravenous fluids

Fluid	Composition per liter	Formulation per liter	Formulation per gallon
Isotonic saline	156 mEq Na ⁺ 156 mEq Cl ⁻	7 cc table salt per liter	28 cc table salt per gallon
Isotonic sodium bicarbonate	154 mEq Na ⁺ 154 mEq HCO ₃ ⁻	13 cc baking soda per liter	52 cc baking soda per gallon
5% dextrose	50 grams dextrose	100 mL of 50% dextrose per liter	400 mL 50% dextrose per gallon
0.45% saline and 2.5% dextrose	77 mEq Na ⁺ 77 mEq Cl ⁻ 25 grams dextrose	3.5 cc table salt and 50 mL of 50% dextrose per liter	14 cc table salt and 200 mL of 50% dextrose per gallon
Hypertonic saline (7.2% NaCl)	1232 mEq Na ⁺ 1232 mEq Cl ⁻	56 cc table salt per liter	224 cc table salt per gallon

For the calf with neonatal calf scours, it is advantageous to develop a fluid therapy protocol and implement it. First and foremost is to do a clinical examination. Assessing body weight, assessing hydration status, and assessing acid-base status are critical components of the exam. A calf that weighs 50 kilograms (110 pounds) and is 10% dehydrated will need 5 liters of fluid to replace the deficit, 2.5 liters of fluid for maintenance, and 4 liters for ongoing losses for the first 24 hours (Total = 11.5 liters). If the calf is in lateral recumbency and is severely depressed and is greater than 8 days, the base deficit will be 20 mEq, so the amount of bicarbonate to be given is 50 x 0.5 x 20 = 500 mEq. For this particular calf, I have the following fluid therapy protocol:

- 1) Initially provide 1-2 liters of lactated Ringer's solution as a rapid bolus. This provides immediate expansion of their extracellular fluid. Correct dehydration.

- 2) Next, administer 1 gallon of 1.3% sodium bicarbonate. This is given to provide additional expansion of their extracellular fluid space and correct the acidosis. There is approximately 3.8 liters of fluid in a gallon. This will provide 585 mEq of bicarbonate, so by administering the entire gallon, the animal should be returned to normal acid base status. In addition, the 3.8 liters of isotonic bicarbonate plus the 2 liters of lactated Ringer's given initially should correct the calf's dehydration.
- 3) Next, administer second gallon of 0.45% saline and 2.5% dextrose with 80-160 mEq of potassium added to the gallon. One gram of potassium chloride provides approximately 14 mEq of potassium, so I simply add 14 grams of potassium chloride to the fluids. This will address maintenance fluids and ongoing losses. In addition, this will address the hypoglycemia and low total body potassium issues that were discussed previously.
- 4) Next, switch the calf to oral electrolytes and oral milk as soon as the suckle reflex is restored. Provide small quantities of oral fluids frequently throughout the next days.

CASE EXAMPLE

You are presented with a 3 day-old Simmental calf with severe diarrhea. The calf is in lateral recumbency with a rectal temperature of 99.0°F. The calf is lethargic and doesn't have any suckle reflex, but the respiratory rate is elevated at 50 breaths per minute. The skin tents for greater than 15 seconds, and there is a gap between the eye and eyelid about 0.5 cm. You estimate the calf weighs approximately 100 pounds (45 kilograms). This is a commercial calf, so the owner has agreed to \$50.00 as a ceiling for costs incurred for treatment.

What is the estimated % dehydration? _____

Why is the rectal temperature low? _____

What is the replacement fluid volume for this calf? _____

What is the maintenance fluid volume for this calf over 24 hours? _____

How much bicarbonate should this calf receive? _____

Outline a fluid therapy protocol for this calf.

Ruminant Abortion Diagnostics

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KEYWORDS

• Ruminants • Abortion • Diagnostics

KEY POINTS

- Abortion rates vary between producers, production systems, and management styles, but in most situations, a rate much higher than 5% to 8% is usually deemed unacceptable.
- Costs of diagnostic services for abortion disease diagnosis can vary greatly among laboratories, but are often significant.
- Numerous improvements in test development have given the diagnostician powerful tools for etiologic diagnosis.
- Practitioners must understand the process and inherent limitations of abortion diagnostics, be able to help the producer determine if and when an investigation is warranted, and submit appropriate samples to a laboratory that specializes in diagnosis of reproductive failure in livestock.
- Successful abortion diagnosis in ruminants involves input from the producer, practitioner, and diagnostician.

Reproductive failure due to abortion disease remains a significant revenue drain in many ruminant livestock production systems. Abortion rates vary among producers, production systems, and management styles, but in most situations, a rate much higher than 5% to 8% is usually deemed unacceptable and worthy of investigation. Given currently high commodity prices for beef and lamb, the tolerated abortion rate may be much lower. Abortion storms such as were historically seen with infectious bovine rhinotracheitis (IBR), and more recently with *Neospora caninum*, can affect up to 10% to 40% of the pregnant animals and be devastating to the economic health of the producer.

Costs of diagnostic services for abortion disease diagnosis can vary greatly among laboratories, but are often significant. Implementing intervention strategies to impact ongoing abortion is usually limited, costly, and often of questionable efficacy.

The author has nothing to disclose.

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Vet Clin Food Anim 28 (2012) 407–418
<http://dx.doi.org/10.1016/j.cvfa.2012.07.007>

vetfood.theclinics.com

0749-0720/12/\$ – see front matter © 2012 Elsevier Inc. All rights reserved.

Preventive programs may need modifications, but in reality, most producers already have basic vaccination programs in place. The search for a definitive diagnosis, or even an “educated maybe,” is often difficult in light of the many diagnostic challenges that are incumbent in abortion diagnostics. Therefore, practitioners must understand the process and inherent limitations of abortion diagnostics, be able to help the producer determine if and when an investigation is warranted, and submit appropriate samples to a laboratory that specializes in diagnosis of reproductive failure in livestock. Although ideal conditions rarely present themselves in the field, the practitioner and producer must be willing to work with the laboratory and diagnostician to find answers (if possible), hopefully in an economically feasible manner.

From a laboratory perspective, numerous improvements in test development have given the diagnostician powerful tools for diagnosis. Immunocytochemistry and new bacterial identification systems are rapid and highly sensitive. New multiplex polymerase chain reaction (PCR) formats are highly sensitive and allow rapid detection of multiple agents in a single test.¹ Histopathology, routine culture, fungal culture, fluorescent antibody (FA) tests, and virus isolation are still common and form the foundation of the approach to abortion diagnosis. Diagnostic laboratories are constantly evaluating new technologies for their ability to provide new diagnostic information. At the same time, diagnosticians must also be aware that these new techniques incur new costs that must eventually be passed on to the producer.

The development of new vaccines and improved vaccination strategies has reduced the impact of the once-major reproductive infectious diseases, such as IBR, bovine viral diarrhea virus (BVDV), brucellosis, and leptospirosis.^{2,3} These once-major players are being replaced by an increase in opportunistic pathogens that seem to be emerging with changing production and management systems. The widespread use of total mixed rations and hay processing equipment in the upper Midwest ensures that any poor-quality feedstuff is incorporated into the total ration and consumed. The environmental bugs that used to be left in the moldy or rotten hay are now all but guaranteed entrance into the animal. The bottom line is that every year, significant financial losses from reproductive failure still occur despite vaccine and management improvements. The practitioner is faced with the dilemma of trying to find answers for these losses when often none exist. The diagnostician is often faced with trying to make a definitive diagnosis when none is possible. The cycle tends to repeat itself every year during “abortion season.” Autolysis and incomplete submissions are known to be common challenges that face the diagnostician, but practitioners are limited by monetary constraints that dictate the diagnostic path to follow. Idiopathic abortion is a code phrase for “we just don’t know,” and is often the result of a variety of factors beyond the diagnostician’s control. This article outlines some of the basic mechanisms and resulting pathology of abortion in ruminant livestock species and approaches for abortion diagnostic investigations that have evolved over the past 18 years of handling thousands of cases of reproductive wastage submitted to the diagnostic laboratory at South Dakota State University.

ABORTION VERSUS STILLBIRTH VERSUS LIVEBORN

The terminology of reproductive failure is often ignored by practitioners and producers. Embryonic mortality (up to 45 days) is often unnoticed and results in open animals or extended calving, lambing, and kidding intervals. These early fetal losses are associated with a wide range of physiologic, nutritional, environmental, and noninfectious causes that often go unrecognized.⁴ Infectious causes of fetal

loss during early gestation traditionally include *Tritrichomonas foetus*, *Leptospira borgpetersenii* serovar hardjo type hardjo-bovis (*Leptospira hardjo*), and BVDV.⁵ In most circumstances, embryonic loss occurs without recovery of a conceptus. Abortion implies expulsion of a fetus before full term and viability outside of the uterus. Still-birth or premature delivery is expulsion of a term fetus that is considered viable. Near-term fetuses, it is necessary to determine if the fetus was viable at expulsion or had been dead in utero. Antepartum death is characterized by variable degrees of autolysis, accumulations of blood-tinged fluids in body cavities, soft autolytic kidneys, and variable degrees of liquefaction of the brain. Tissues develop a uniform red-brown appearance from hemoglobin staining. Deaths associated with the parturition process are often less autolytic and display evidence of viability such as hemorrhage (functioning circulatory system), partial aeration of the lungs, meconium staining of the perineum and skin, swelling of the head and cervical region, subcutaneous edema, and fractures of ribs and limbs associated with the fetal expulsion process. Animals that have survived the birth process and died shortly after will have blood clots in umbilical vessels, aerated lungs, and minimal free fluid in body cavities.

Routes of Infection

The routes through which infectious agents reach the fetus include hematogenous spread through the placental-maternal interface where the placental chorioallantois attaches to the lining of the uterus at the caruncle. Additionally, ascending infection from the vagina through the cervical os can result in placental infection.⁶ Infectious agents can colonize the placenta, penetrate into the amniotic fluid, and be swallowed by the fetus. Fungal organisms can penetrate the placenta and result in colonization of the fetal skin. Hematogenous spread results in passage through the liver and to the remaining tissue through the vascular system. Fetal pneumonia in these cases results in interstitial accumulation of organisms and inflammatory cells. For example, abortion associated with *Listeria monocytogenes* presents with massive bacterial growth, with organisms present in blood vessels in most fetal tissues. With this infectious species, inflammation is generally mild compared with the massive number of organisms present in tissues. Organisms can also enter the lung through the airways by inhalation of infected amniotic fluid. This amniotic fluid will often contain clumps of meconium, indicating advancing fetal stress caused by hypoxia.

Fetal hypoxia can result from maternal hypoxia, maternal circulatory system failure, or interference with oxygen transfer through the placental interface, most often associated with placentitis or premature placental separation. If possible, fetal compensatory mechanisms shunt blood to vital organs in an attempt to maintain normal oxygen levels. Fetal respiration increases in an attempt to compensate for hypoxia. This labored breathing is often associated with the aspiration of amniotic fluid. If the placenta is compromised because of slow-growing opportunistic bacteria or fungi, and the fetus is not immediately overwhelmed by the infection, the slowly advancing placental damage will suffocate the fetus from lack of oxygen or starve it from lack of nutrient transfer across the fetal maternal interface. If the fetus is not yet viable, abortion occurs; if the fetus is still viable but weakened from hypoxia, low nutrient transfer, and the possible deleterious effects of chronic infection, the outcome is often a still-born or weak-born calf.

Clinical History

Appropriate collection and submission of samples for abortion diagnosis is critical for diagnostic success. A complete history, although often excluded on most submission forms, can be the first critical component to that success.

The following information that should be included:

- Size of the herd or flock, subgroups within the herd or flock, number of abortions (sporadic or epidemic), age of aborting animals, trimester in which abortions are occurring based on breeding dates or crown-rump measurements, recent purchases or whether it is a closed herd or flock, when and where any new additions were purchased from, previous reproductive history, natural service or artificial insemination, when the bulls or rams were pulled, exposure to other herds or flocks, and whether animals were clinically ill before or at the time of abortion
- Health management practices, including vaccination history, recent vaccinations, types of products, recent use of any modified live vaccines, recent treatments including feed-grade antibiotics, and treatments of clinical disease in the herd, flock, or affected individual animal
- Nutritional management, including types of feed; feed quality issues; feeding practices, including processing, feeding on the ground versus bunks, and trace mineral practices that may lead to deficiencies; potential toxic exposures to plants; nitrates/nitrites in feedstuffs; excessive minerals in feedstuffs (selenium); and water quality issues
- Environmental conditions, including heat or cold stress, overcrowding, and severe storm events

Unfortunately, nearly blank submission forms are often presented. Fortunately, the nervous producers or practitioners can often be consulted by telephone to fill in the gaps.

Sample Submission

Collection and submission of inappropriate or unsuitable samples is a disservice to the producer because it incurs needless costs and usually results in no useful information on which to base treatment or prevention strategies. Sample quality issues are a constant problem in abortion diagnostics. Aborted fetuses are often retained in utero, macerated, mummified, severely autolytic, partially eaten, covered in mud and manure, buried in bedding, frozen solid, or rotten from extreme heat. Superficial contamination can be rinsed away. Unfortunately, rotten is still rotten. Some samples are just unsuitable for evaluation. Gross lesions in abortion diagnostics are rare, and the submitted tissue is often soft, homogenous in color, and often bathed in red-black fetal fluid. Brain tissue is often liquefied and may pour out through the foramen magnum.

The whole fetus and complete placenta are considered ideal samples for submission if the laboratory is located in proximity to the producer. Fetuses are often at diagnostic laboratories at minimal or no additional charge. Crown-rump length is recorded as an estimation of fetal age, the overall stage of fetal development ([Table 1](#)) is noted, and the overall postmortem condition of the fetus is assessed.⁷ External congenital anomalies are recorded and photographed ([Fig. 1](#)). Body or tissue weights are rarely collected unless a congenital disease is suspected and the fetus or individual organs are substantially smaller than expected. Necropsy procedures involve exposure of the thoracic and abdominal cavities, removal of the brain, and collection of appropriate tissues and body fluid, as listed in [Table 2](#). These tissues can be collected easily in the field or veterinary clinic.

Placenta

The placenta is most significant tissue for abortion diagnosis. If unavailable, the probability of diagnosis is significantly reduced. A whole, intact placenta is rarely received

Table 1 Estimation of fetal gestational age			
Crown-Rump Length (cm)	Age	Comparative Size	Physical Characteristics
1	30 d		
10	60 d	Mouse	
20	90 d	Rat	
30	120 d	Small cat	
45	150 d	Large cat	
60	180 d	Small dog	
80	210 d	Large dog	Hair around eyes, tail, muzzle
100	240 d		Hair on body, incisors slight eruption
>100	270 d		Near-term, incisors erupted

for examination. Often only a small portion of placenta is recovered and may be devoid of any cotyledonary structures. Rarely are these samples diagnostically useful. Histologic changes in placenta are often multifocal in distribution, requiring examination of multiple sections to give the diagnostician the best chance of detecting subtle areas of placental damage. Placentitis results in disruption of placental functions, including oxygen transport and exchange, nutritional support for the fetus, and hormone and growth factor production, which can affect normal parturition and fetal development. Chronic inflammation associated with release of cytokines and proinflammatory factors alters normal physiologic processes that occur at the fetal–maternal interface. Fetal macrophages within the placenta are rare in the early gestational fetus, but by 8 months’ gestation, they have increased 10-fold. These macrophages are numerous within the allantoic stroma in areas of inflammation, and often seem to contain debris or organisms in their cytoplasm. Their role in cell defense against infectious agents and in dissemination of organisms is unknown.⁸ The author believes that a significant number of stillborn or weak-born calves and lambs that are presented every late winter and spring are the result of placental dysfunction, often associated with chronic



Fig. 1. Near-term bovine fetus with multiple congenital anomalies, including angular limb deformities and vertebral malformations. Suspected complex vertebral malformation in a Holstein calf.

Table 2 Samples to submit for ruminant abortion diagnosis			
Whole fetus and placenta if proximity to laboratory is convenient; fresh (chilled) not frozen tissue samples, if entire fetus and placenta cannot be submitted:			
Fresh ^a		Formalin-fixed ^b	
Lung (anterior lobes)	BV	Lung	HP
Kidney	VB	Kidney	HP
Liver	V	Liver	HP
Spleen	V	Spleen	HP
Heart	V	Heart	HP
Thyroid gland (ovine)	V	Thyroid (ovine)	HP
Placenta	BVM	Placenta	HP
		Skeletal muscle	HP
		Thymus	HP
		Brain	HP
		Ear notch	IHC
Fetal stomach content and bowel movement: collected with sterile syringe and submitted in snap cap tube			
Fetal thoracic fluid/heart blood: collect with sterile syringe and submitted in snap cap tube			
Ocular fluid for nitrate/nitrite analysis: collect with sterile syringe and submitted in snap cap tube			
Maternal blood for serology ^c			
Other: feed and water samples			

Abbreviations: B, bacteriology; HP, histopathology; IHC, Immunohistochemistry; M, mycology; V, virology.

^a Adequate fresh sample should be placed in leak-proof bags and chilled or frozen if delivery is delayed.

^b Fix in adequate (10x) volume 10% buffered neutral formalin, submit in leak-proof sealed container.

^c Maternal blood can be collected and serum harvested and saved frozen for future use.

placentitis. The outcome of pregnancy (abortion, stillborn, or weak-born) often depends on how long the fetus can survive with a damaged placenta.

A complete placenta is a large tissue, and placental lesions are often focal to multifocal in distribution. Therefore, evaluation of a single small section of placenta may miss significant changes and result in a missed diagnosis. The author is constantly reminded by clients that the placenta often disappears shortly after birth for several reasons, but is satisfied if the client realizes that the diagnostic success rate is significantly reduced without the placenta. In the laboratory, the placenta is rinsed and cleared of contaminating debris and spread out for examination. It should be examined for gross changes, including the presence of exudate or thickening of intercotyledonary spaces or discoloration of cotyledons. The normal placenta is thin and transparent in the intercotyledonary areas, and the cotyledons are dark red-brown. The size and distribution of cotyledons should be noted.

Maternal and Fetal Serology

Single serum samples from the dam are often submitted with abortion investigations, but they are usually of little value in abortion diagnosis. Positive serology for an individual animal at best indicates exposure to a specific agent or antigens to a specific

agent in the form of vaccine. Separating the 2 responses is often impossible. Knowledge of vaccination history, types and brands of vaccine used, and baseline serologic data from the specific laboratory performing the test is crucial to any serologic interpretation. In many laboratories, a positive serology result only means the animal has mounted a detectable immune response to the agent, and cannot separate actual exposure from vaccination. Most of the opportunistic infections, including environmental bacteria and fungi, do not have validated serologic tests. Many infectious agents stimulate titer increases that predate expulsion of the fetus. Therefore, using paired serum samples on individual animals to detect changes in titers is also rarely useful for demonstrating evidence of specific abortion agents. A serologic profile comparing aborted animals with normal controls is more often recommended. Serologic profiling on a significant number of animals in a herd may provide data on vaccination status for a given antigen and suspected exposure based on markedly elevated titers in the abortions versus the normal controls. Fetal serology may be useful in some instances. If the fetus is old enough to be immunocompetent, fetal immunoglobulin G (IgG) levels can be significantly elevated in fetal fluids in some infectious abortions. If IgG is elevated, then individual serologic tests can be performed as appropriate.⁹ For example, indirect FA is a useful serologic test to detect antibody to *Toxoplasma gondii*. In *N. caninum* abortions, fetal and neonatal serology was used to detect in utero infections in aborted fetuses or precolostral calves.

Diagnostic approach

When conducting abortion diagnostic workups, most laboratories tend to perform a standard battery of tests to cover the major bacterial, viral, fungal, and protozoal abortion diseases for the species submitted. Numerous excellent reviews on the complete list of potential agents are available and recommended for review.^{9–11} History and gross examination may indicate a particular agent, but in practice, following a standard abortion protocol and performing additional tests as the investigation warrants is more practical. Ideally, one could test for every possible agent on each case, but financial considerations dictate that the diagnostic tests should be ordered selectively.

Bacterial infections

Most bacterial causes of abortion are opportunistic pathogens. These organisms are not infectious, and are common inhabitants of the host or its environment. These bacteria gain entrance to the bloodstream of the dam and occasionally introduce an infection in the placenta. *Arcanobacterium pyogenes* and *Bacillus* spp, followed by *Escherichia coli*, *Histophilus somni*, *Pasteurella* spp, *Listeria* spp, *Staphylococcus* spp, *Streptococcus* spp, and basically any other bacteria that can find its way into the bloodstream, can be opportunistic pathogens. These opportunists are usually associated with sporadic abortions, unless specific risk factors give a particular organism the chance to affect multiple animals. Cattle with abscesses or a history of feet problems seem to be affected by *A. pyogenes*. Cattle exposed to processed bales with a great deal of soil-associated spoilage can have increased problems with *Bacillus* spp. *Listeria* spp is usually associated with poorly fermented silage feeding.

Most opportunists can cause abortion at any stage of gestation, but most are associated with late second to third trimester abortions. Gross lesions are rare but can include exudate on the placenta surface, or possibly increased fluid in body cavities, occasionally with fibrin. Histologic lesions include suppurative fetal pneumonia, mild perivascular inflammation in the epicardium and, to a lesser extent myocardium, increased portal inflammatory cells in liver, and inflammatory cell pooling in blood vessels in the brain and other tissues. A variable severe, multifocal, necrotizing, and

suppurative placentitis is a common lesion if adequate placenta is examined. Numerous intralesional bacteria are often observed histologically, especially in the case of *A. pyogenes*-induced abortion. Bacterial culture of these organisms is usually straightforward, until one realizes that rarely do autolyzed fetuses yield pure growth of a single organism.

Campylobacter jejuni, *C. fetus* subspecies *fetus*, and *Salmonella* spp are similar to *A. pyogenes* in that numerous intracellular bacterial colonies are usually evident in sections of placenta, often associated with vigorous inflammation. These organisms are normal or transient inhabitants of the dam's intestinal tract and travel to the placenta during periods of bacteremia. *Campylobacter* spp and *Salmonella* spp are more commonly associated with abortions in sheep. Gross lesions, if present, are confined to the placenta and include accumulation of exudate or discoloration of cotyledons. Special culture media is required for *Campylobacter* spp, but *Salmonella* spp grows rapidly on conventional media. Brucellosis associated with *Brucella abortus* and other *Brucella* spp is rare in the United States. The most common member of this genus, *B. ovis*, the agent associated with ram epididymitis, has been rarely associated with abortion in sheep. Serologic monitoring tests are available to detect cattle that have been exposed to *B. abortus*. Special culture media is usually recommended for *Brucella* spp; however, culture specifically for this organism is not routinely attempted unless brucellosis is suspected.

Multiple species, serovars, and types of leptospira, including hardjo type hardjo-bovis, *pomona*, *icterohaemorrhagiae*, *grippityphosa*, and most likely many others, can be involved in bovine embryonic loss and abortion.¹² Specific gross and histologic lesions have been described historically, but leptospira-induced abortions are so infrequent today that many diagnosticians would not recognize them. In the upper Midwest, the near-universal use of multivalent vaccines for *Leptospira* spp have significantly reduced its diagnosis associated with abortions in cattle. Culture of this organism is not practical because of time and cost constraints. Microscopic detection through dark-field examination of fetal fluids or silver-stained histologic sections is occasionally used, although the sensitivity of the techniques is low. A common technique, FA staining of kidney homogenates with multivalent antisera, is frequently used. Again, the sensitivity may be low, especially with host-adapted *Leptospira* spp, such as harjo type harjo-bovis. New PCR tests are currently in use and have the benefit of speed, specificity, and sensitivity. The PCR format is routinely used to detect carrier cows that shed the harjo-bovis organism in urine, and is becoming more common for detecting leptospira organisms in abortions.

Chlamydophila abortus associated with enzootic abortion in ewes is a significant cause of abortion in range sheep flocks, or farm flocks that buy range ewes for replacements. Gross lesions include thickening of the intercotyledonary spaces around affected cotyledons. Histologic lesions are most common in the placenta and, to a lesser degree, the liver. Placental lesions include a suppurative and necrotizing placentitis with marked stromal thickening and inflammation. The liver will rarely contain multifocal areas of necrosis. Diagnosis is accomplished routinely through immunocytochemistry of affected placenta. Serologic methods can detect specific antibody to *C. abortus* in fetal thoracic fluid or heart blood. Although this organism can be cultured in embryonated eggs or in cell culture, very few laboratories still attempt isolation. PCR is available for *C. abortus* at some laboratories, although the advantage PCR over immunohistochemistry is questionable in most routine circumstances.

The role of *Ureaplasma* spp and the agent associated with epizootic bovine abortion in bovine abortion seems to be significant in some geographic regions. Most

laboratories do not routinely screen for these agents unless requested or lesions are present.

Viral Infections

Viral causes of abortion include bovine herpesvirus type 1, the cause of IBR, BVDV, and, to a lesser extent, bovine herpesvirus type 4 (BHV-4). Numerous references are available that describe these agents in detail.^{2,5,9-11} IBR-associated abortions have decreased dramatically since the introduction of effective vaccination procedures. Recently, increased numbers of IBR abortions have been reported in unvaccinated or questionably vaccinated cows exposed to modified live vaccines during gestation. Gross and histologic lesions are commonly observed with IBR and can include pale foci in the liver that correspond with the multifocal necrotizing lesions that are present in several fetal tissues, including liver, lung, and spleen. Similarly, the incidence of BVDV has also decreased in the past several years, most likely because of increased vaccination. BHV-4 is considered an opportunistic viral pathogen and its role in abortion is difficult to determine. Diagnostic procedures for viral abortion agents vary among laboratories. Fluorescent antibody tests are rapid and usually of acceptable sensitivity. Virus isolation is considered a tried and true method, especially for discovery of new agents, but it is very expensive and time-consuming, and requires technical expertise. The advantage of virus isolation is that an isolate is available for further study or vaccine production at the end of the procedure. Molecular PCR-based tests have replaced other techniques in many laboratories because of their speed, specificity, and sensitivity. Multiplex PCR is currently available for IBR and BVD. Other viruses have been reported in certain geographic regions as causes of abortion and congenital anomalies. This group includes many arthropod-borne viral agents, such as bluetongue and Cache Valley virus.¹³ Other viruses in the group are not routinely found in the United States, or require special diagnostic procedures performed at reference laboratories. Congenital anomalies can be associated with early bluetongue virus, Cache Valley virus, or BVDV infections.

Mycotic Infections

Mycotic abortion is common worldwide. The common agents include *Aspergillus fumigatus*, *Aspergillus* spp, *Candida* spp, and a variety of environmental species.^{14,15} These organisms are ubiquitous saprophytes in the environment and often increase in numbers in moldy feedstuffs or bedding. Abortions usually occur when cattle are fed high concentrations of moldy stored or processed feedstuffs. The conidia from these organisms enter the respiratory tract or digestive tract, gain entrance into the bloodstream, and spread to the uterus and placenta. Gross lesions include thickening and roughening of cotyledons and intercotyledonary spaces. Lesions are often localized and may not be present if only a small portion of placenta is submitted. Histologic lesions, if present, will confirm a severe necrosuppurative placentitis and stromal arterial vasculitis. Fungal hyphae are often associated with these necrotic lesions.

Mycotic abortion can be diagnosed using fluorescent potassium hydroxide (KOH) staining procedures on placental scrapings to allow visualization of fungal elements. Special histochemical stains are also useful for histologic identification of fungal elements. Culture of fungal organisms from stomach content and placenta requires special media with added antibiotics to suppress bacterial growth. When a mixed growth of fungal organisms is isolated, the significance of the results should be questioned, but not dismissed. Multiple fungal species are often present in feed stuffs, and therefore dual infections cannot be completely eliminated as a possible diagnosis. If any particular fungal organism is isolated in heavy growth, or isolated in heavy growth

from fetal stomach content and placenta and compatible placental lesions exist, then causality can be considered.

Protozoa

Protozoal agents associated with abortion include *N caninum*, *T gondii*, and *T foetus*.¹⁶ *N caninum* is vertically transmitted from dam to congenitally infected normal offspring, and horizontally transmitted through ingestion of infective oocysts shed by the canine definitive host. Epidemic abortions were more common historically when most cattle were naïve to infection. The most common presentations associated with *N caninum* today are sporadic or endemic abortions. The dam is clinically normal, and most abortions occur between 5 and 7 months' gestation. Compatible lesions include multifocal necrosis and gliosis, and nonsuppurative epicarditis, myocarditis, and myositis. Occasionally, similar focal lesions are present in other tissues. Immunohistochemistry is used for detecting the organism in the context of the histologic lesion. PCR and several serologic tests are also available for diagnosis. Caution should be used if lesions are very mild or nontypical, because most calves born to seropositive dams will be congenitally infected, and the abortion could have been caused by other agents.

T gondii is similar to neosporosis but is primarily a problem in sheep and goats. The definitive host is the cat, and infective oocysts are usually consumed in contaminated feed stuffs. Mummification is common in *Toxoplasma*-induced abortion, and fetuses of various stages of development are often presented (Fig. 2). Histologic lesions include multifocal necrosis and gliosis in the brain and a nonsuppurative epicarditis. Oocysts of *T gondii* can occasionally be observed in routine histologic sections. Immunohistochemistry can improve detection of the organisms if needed. Indirect FA procedures can accurately detect antibodies specific for *Toxoplasma* in fetal fluids from aborted lambs or kids.

T foetus is most often associated with early embryonic death and early abortions in cattle. The organism can be cultured in special media from carrier bulls and occasionally from infected cows or recovered fetuses. PCR techniques have also been developed. In aborted fetuses, a mixed pneumonia is present, and occasionally protozoa compatible with *T foetus* can be found. Immunohistochemistry is available for diagnosis in fixed tissue.



Fig. 2. Abortions associated with toxoplasmosis. Note the various stages of fetal development. All fetuses came from a single Finnsheep ewe that died from complications related to pregnancy toxemia.

Noninfectious Abortion

Noninfectious causes of abortion are often lumped together and include a variety of genetic, nutritional, and environmental factors associated with reproductive failure. This category is often a catch-all and is often overlooked in most diagnostic scenarios. Genetic causes of early embryonic mortality often go unnoticed. Embryonic loss associated with chromosomal defects or lethal mutations are rarely detected. Obvious congenital anomalies that present at birth often fit in 1 of 2 categories. The first includes animals with established genetic conditions, often with known genetic defects and testing strategies to eliminate the trait from the breed. The second and most common includes all other animals with a congenital anomaly. Caution is warranted in using the word *genetic* too early when investigating congenital malformations.¹⁷ Many of the animals involved in these situations are extremely valuable, and data must be collected carefully and thoroughly before reaching any conclusion. Most nongenetic causes are probably still unknown, but nutritional factors, toxic plants, chemical exposure, and viruses should be considered as possible suspects. Toxic plant exposure during the first trimester can result in limb deformity, cleft palate, and spinal column abnormalities. Alkaloid-producing plants, such as the lupines, have been proven experimentally to cause malformations. Similar evidence exists for poison hemlock.¹⁸ Exposure to mycotoxins has been suggested to contribute to limb and jaw anomalies. The challenge is determining which potentially toxic plant was present during the summer when the fetus was at 60- to 80-days' gestation when one is examining a calf submitted to the laboratory in March when 2 feet of snow are covering the ground. Nutritional factors, including trace mineral, vitamins, protein, and energy, can contribute to increased fetal loss and poor postnatal survival. Although many of these causal links are difficult to prove, the possibility of a nutritional component should be considered, if for no other reason than to give the producer the opportunity to evaluate and correct nutritional problems before they get worse. Exposure to parasiticides or other chemicals have been reported to have deleterious effects on fetal development, although the data are incomplete.

If genetic problems are suspected, diagnosticians should ensure they are dealing with purebred animals, offspring from a single sire, or offspring from very closely related sires, and that the defect occurs in expected frequencies. In reality, most genetic conditions that result in lethal outcomes in popular breeds cannot be hidden forever.

SUMMARY

Successful abortion diagnosis in ruminants involves input from the producer, practitioner, and diagnostician. Unfortunately, despite best efforts, many investigations still result in a diagnosis of idiopathic abortion. If this diagnosis is made after a complete and systematic investigation of appropriate and reasonably preserved samples, some comfort can be taken that practitioners and diagnosticians did their best for the benefit of the producer. As new diagnostic technology is developed for abortion diseases, hopefully the best will only get better.

DEDICATION

This work is dedicated to the late Dr Clyde Kirkbride, Professor, South Dakota State University, Animal Disease Research and Diagnostic Laboratory. Clyde was my mentor and friend. His desk and file cabinet, which is filled with a career's worth of knowledge on abortion disease in all species, still sit in my office. I keep them there

as a reminder of his legacy and contribution to the field of abortion diagnosis. He was truly a pioneer.

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Diagnosis and Control of Viral Diseases of Reproductive Importance

Infectious Bovine Rhinotracheitis and Bovine Viral Diarrhea



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KEYWORDS

- Biosecurity • Bovine herpesvirus • Bovine viral diarrhea virus • Surveillance
- Testing • Vaccination

KEY POINTS

- Both bovine viral diarrhea (BVDV) virus and bovine herpesvirus 1 can have significant negative reproductive impacts on cattle health.
- Vaccination is the primary control method for the viral pathogens in US cattle herds.
- Polyvalent, modified-live vaccines are recommended to provide optimal protection against various viral field strains.
- Of particular importance to BVDV control is the limitation of contacts of pregnant cattle with potential viral reservoirs during the critical first 125 days of gestation.

Viral infection and disease can have significant negative impacts on the reproductive efficiency of cattle herds in both the beef and the dairy industries. Consequences of infection range from abortion outbreaks that can affect a large proportion of the pregnant herd to more subtle syndromes (eg, impaired conception, early embryonic death) that may go unnoticed or undiagnosed. Diagnostic tests must be used in a way that conforms to the overall biosecurity program of the operation, and when applied correctly, should be viewed as an economic asset and not a liability. Control programs should be designed and implemented to prevent introduction and/or spread of the viral pathogens with particular attention to the periods in the

Drs B.W. Newcomer and M.D. Givens have received research funding from Boehringer Ingelheim Vetmedica, Inc and Zoetis.

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Vet Clin Food Anim 32 (2016) 425–441

<http://dx.doi.org/10.1016/j.cvfa.2016.01.011>

vetfood.theclinics.com

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production cycle when cattle are most susceptible to the consequences of disease. Such schemes must be implemented in harmony with the variance in management schemes and production goals of individual producers. This review focuses on (a) the potential reproductive consequences of bovine viral diarrhea (BVD) and infectious bovine rhinotracheitis (IBR); (b) surveillance schemes to assess the level of infection at the herd level and diagnostic assays for detection of infection in the individual; and (c) vaccination and biosecurity programs to prevent or mitigate the effects of infection in replacement heifers, the mature herd, and animals newly introduced to the farm.

REPRODUCTIVE CONSEQUENCES OF INFECTION WITH BOVINE VIRAL DIARRHEA VIRUS

Although capable of manifesting in any number of bodily systems, the reproductive consequences of BVD are the most costly on dairies and cow-calf operations.¹ Cattle that are infected shortly before the breeding period have reduced conception rates.^{2,3} Decreased conception rates may result from impairment of fertilization or early embryonic death but may be, at least in part, mediated by alterations in ovarian function. Transient infection with bovine viral diarrhea virus (BVDV) can result in oophoritis and subsequent ovarian dysfunction,⁴ resulting in impaired fertility and repeat breeder syndrome.

Viremia subsequent to infection of a naïve, pregnant animal allows the virus to readily cross the placenta of pregnant animals and infect the growing fetus; the effect on the growing fetus depends largely on the stage of gestation at which the infection occurs.⁵ A naïve cow infected during the first month and a half of gestation may suffer early embryonic death, due to endometrial inflammation resulting from the viral infection or direct viral effects on the developing embryo. Infection between 3 and 5 months of gestation, while the fetus is undergoing the final stages of organogenesis, is associated with a variety of congenital defects, most commonly involving the central nervous system. Cerebellar hypoplasia is the most notable developmental defect, but other common defects include hydranencephaly, microphthalmia, hypotrichosis, and brachygnathism. If infection occurs after the completion of organogenesis and the development of fetal immunocompetence, the calf may mount a protective immune response as demonstrated by a precolostral antibody titer to BVDV. However, infection during this period can result in abortion of the pregnancy or, less commonly, the birth of weak calves.

The most important consequence of intrauterine infection is the creation of the persistently infected (PI) animal. In utero exposure to noncytopathic strains of BVDV before development of fetal immunocompetence (generally before 125 days of gestation) can result in a calf that is PI with the virus.⁶ PI calves are often weak at birth, and most will die before 1 year of age. However, others may not show signs of disease but continuously shed virus and are epidemiologically important due to efficient transmission of BVDV. Superinfection of PI calves with homologous cytopathic strains of BVDV may result in mucosal disease, which is almost invariably fatal. Calves born to PI cows or heifers will consistently be PI themselves. Thus, preventing the creation of PI animals is essential to control of the virus.

Effects of infection in the bull are less noticeable than in female cows. Infectious virus is shed in the semen of transiently infected or PI bulls.⁷ Although shed in lower levels, semen from transiently infected bulls, as well as from PI bulls, is capable of infecting naïve cattle, resulting in seroconversion and the potential birth of PI calves.^{8,9} Less commonly, persistent testicular infection has been reported; such bulls consistently shed high amounts of live virus in the semen despite high serum antibody titers

and a lack of viremia.^{10,11} Although commonly overlooked, the bull should not be ignored when seeking to diagnose and control disease due to BVDV.

REPRODUCTIVE CONSEQUENCES OF INFECTION WITH BOVINE HERPESVIRUS 1

Like BVDV, bovine herpesvirus 1 (BHV-1) is capable of causing a variety of clinical reproductive syndromes. The virus is ubiquitous in cattle populations, and disease may be seen following acute infection or after viral recrudescence. Latency commonly occurs following natural infection or vaccination with attenuated strains; recrudescence is thought to occur following periods of stress. The genital forms of the disease, infectious pustular vulvovaginitis and infectious pustular balanoposthitis, were commonly seen in Europe but only rarely appreciated in the United States. Infected bulls shed live virus in the semen. Endometritis, infertility, and altered estrus cycles can be seen in cattle inseminated with infected semen.¹² Mucopurulent discharge may also be observed in cattle, and affected bulls may suffer from epididymitis.

More commonly, BHV-1 is associated with late-term abortions and infertility in North America.¹³ Abortion generally occurs within a few weeks of exposure but may be delayed for up to 4 months if viral latency occurs in the placenta.¹⁴ Recrudescence of the virus may subsequently infect the fetus, and thus, abortions may appear to be associated with vaccination if natural exposure occurred previously. Abortion is often accompanied by retained placenta, but subsequent infertility is not commonly seen. Occasionally, fetal infection results in the birth of stillborn or weak calves with increased mortality during the first week of life.

DIAGNOSIS

Herd Surveillance Testing

The presence of PI animals in the herd may often go unnoticed because such animals do not always show clinical signs and thus often serve as the viral reservoir to expose and infect susceptible herd mates. Consequently, herd surveillance testing at routine intervals is recommended to identify and cull PI animals from the herd. In the United States, herd surveillance for BHV-1 is less commonly used because the virus is often ubiquitous in cattle populations. The primary tests used for BVDV surveillance include the bulk tank milk (BTM) test on dairy operations and pooled ear-notch testing in non-lactating animals. In addition, feed trough sampling may be used to detect PI animals within a group of nonlactating animals, and sentinels can be used to detect circulation of either virus within the herd.

Bulk tank milk testing

The consistent shedding of high amounts of live virus in all bodily secretions, including milk, enables routine testing of BTM samples to readily identify herds containing lactating PI cows.¹⁵ Testing BTM samples provides an economical, simple, and rapid way to determine the presence of PI animals in the lactating string.¹⁶ Most commonly, somatic cells are collected from submitted BTM samples and subsequently tested by polymerase chain reaction (PCR) to detect viral RNA. Combining the testing of BTM samples by PCR and by virus isolation increases the sensitivity of detection¹⁷ but adds expense and a delay in assay results. Although recommendations will vary between laboratories, PCR-based testing is sensitive enough to identify a single PI animal diluted 1:600 with milk from BVDV-negative animals.¹⁷ When testing large herds, it is advised to contact the testing laboratory to determine their recommended number of cows per sample. String samples may be submitted in lieu of BTM samples when the number of total cows exceeds the recommended number of allowable cows per

sample. Specific laboratories should also be contacted for the desired specifics on sample handling and shipping. Alternatively, BTM samples may be assessed for the level of BVDV antibodies^{18,19} or by using an antibody enzyme-linked immunosorbent assay (ELISA) test^{20,21} for surveillance and determination of herd infection status. However, antibody-based surveillance is complicated by herd vaccination status, and results should be interpreted with caution in vaccinated herds.²²

It is important to remember which animals are not included in the sample when testing a herd for BVDV. Obviously, only lactating animals will be tested; the status of replacement heifers, the most common class of animal brought onto dairy operations, will not be assessed until they enter the lactating herd. Bulls, dry cows, and cows in the hospital pen/string are groups commonly overlooked using BTM BVDV testing. Most cattle will be included in subsequent samples but transmission of the virus may occur before the PI animal is identified in future tests, especially in the case of introduced animals. Consequently, it is important to sample these animals by another method to assay their BVDV status, particularly in the face of an outbreak, or when the presence of a PI individual is suspected in the herd.

When a positive result on a BTM BVDV assay is encountered, additional testing is necessary to identify the source of the positive test result. The testing program that should be pursued will depend on the reason for the initial testing and the biosecurity/biocontainment goals of the dairy. At a minimum, the individual cow or cows responsible for the positive test and her offspring should be identified and removed from the herd (**Fig. 1**).

Pooled sample testing

The advent of molecular diagnostics has allowed the testing of pooled individual samples to detect the presence of PI animals within the tested group. Using PCR, pooling either blood^{23,24} or ear-notch samples^{25,26} provides a rapid and economical surveillance tool for BVDV. Using ear-notch samples has the benefit of being less invasive, and individual samples composing a positive pooled sample can be subsequently examined to positively identify the PI individual within the group. Pools of up to 100 samples have demonstrated high sensitivity in detecting PI animals, but the number of samples pooled may vary by laboratory depending on individual assay validation and the regional prevalence of disease. A smaller number of samples included in the pool become increasingly more cost-effective as the prevalence of disease increases because of the need for subsequent individual sample testing.²⁷ Pooled testing is currently the method of choice for BVDV surveillance testing in groups of nonlactating animals.

Antibody detection in sentinels

Screening sentinel animals for the presence of antibodies to BVDV has been proposed as a potential surveillance option for the detection of PI animals within the herd.^{28,29} In one study involving 5 nonvaccinated calves of at least 6 months of age in 47 cattle groups, the presence of PI animals within the herd was accurately predicted if at least 2 sentinel animals had virus neutralization titers of 128 or greater.³⁰ However, in another study involving 27 cow-calf herds, finding a titer 1000 or greater in at least 3 of 10 sentinel calves accurately predicted the presence of a PI only 53% of the time.³¹ In a surveillance program, sensitivity is valued over specificity; sensitivity of surveillance schemes involving antibody titers in sentinel animals is maximized by decreasing the threshold antibody titer or decreasing the number of calves required to have the threshold titer.³⁰ Precolostral antibody detection in dairy calves may be a more cost-effective surveillance tool than testing all calves for the presence of PI

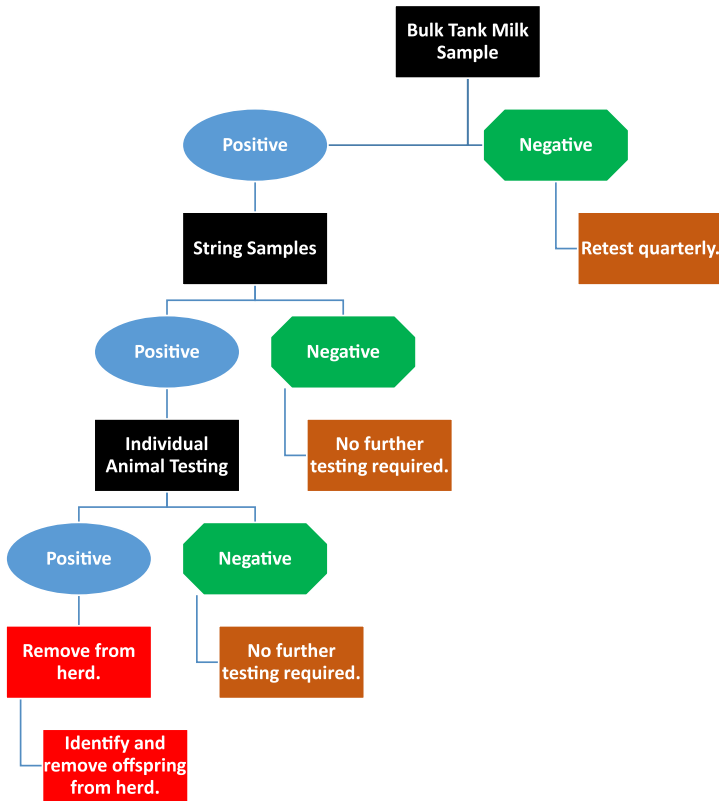


Fig. 1. Testing strategy to identify and remove animals PI with BVDV from the milking herd using BTM testing.

animals because more seropositive calves are likely to be born than PI calves in infected herds.³² Precolostral surveillance has the added benefit of detecting circulating infections in nonlactating heifers that would be missed by BTM sampling.

Feed trough sampling

A diagnostic test using reverse transcription PCR (rtPCR) has been described for the detection of PI animals in a group of nonlactating animals that does not require individual animal handling.³³ By swabbing consumption surfaces within 6 hours after feeding and assaying the swabs by rtPCR, the investigators were able to detect the presence of PI animals in a larger group of cattle. The assay successfully differentiates between transient and persistent infection. The assay provides a potential alternative to the BTM BVDV test for use on beef cattle populations or in groups of replacement dairy heifers.

Individual Animal Testing

Mature animals

Individual animals are rarely tested for BHV-1 because the virus is nearly ubiquitous in North American cattle; exceptions are in the case of introduced animals or abortion outbreaks. Testing for BVDV in individual mature animals is most commonly performed to identify PI animals for removal from the herd, often after a clinical outbreak

of BVD or following the detection of a PI animal on surveillance screening. Several tests are available, and selection of the appropriate test will be dictated by several factors, including the management system of the farm, financial constraints, and availability of tests at a given laboratory. Because not all available tests are appropriate for each clinical situation, care should be made when selecting a test in order to reach a successful solution quickly and efficiently.³⁴ Historically, isolation of live virus from tissues or secretions of infected animals is considered the “gold standard” diagnostic test for both BVDV and BHV-1 but is infrequently used in diagnostic laboratories due to the expense and time needed for the assay; isolation of virus from samples obtained from an animal at least 3 weeks apart is indicative of BVDV persistent infection.

Although molecular techniques have become the screening method of choice for BVDV and BHV-1, the demonstration of viral DNA does not confirm active infection. Most US cattle are latently infected with BHV-1, and disease most commonly results from viral recrudescence. The detection of BVDV antigen is preferred to confirm the PI status of individual animals. Commonly used antigen detection methods include antigen capture ELISAs (ACE) and immunohistochemistry (IHC) techniques. Additional testing may be warranted in certain circumstances due to the occurrence of false positives.³⁴ When pooled sampling detects a PI animal in the group, subsequent testing of the individual samples is used to positively identify the affected animal. Alternatively, several studies have demonstrated a high level of sensitivity for the commercial antigen capture ELISA kits when used as a screening test to detect PI animals.^{35,36} Commercial ACE kits and IHC techniques rely on monoclonal antibodies targeting the E^{ms} glycoprotein of BVDV; consequently, the potential exists for a rare and uniquely divergent strain to escape detection by these tests.³⁷ Identification of mature PI cows necessitates the removal of any replacement offspring as they will also be PI.

Calves

Testing of individual calves for PI status is performed following a known or suspected exposure of the dam during pregnancy. Antigen detection assays are the most reliable methods for consistently detecting PI animals.³⁶ Identification of PI animals is ideally performed soon after birth to limit the potential exposure to naïve contacts. This identification is imperative in herds wherein PI calves will be in contact with other animals in early- to midgestation potentially resulting in the creation of additional PI animals such as in cow-calf herds with poorly defined calving seasons. Precolostral antibody titers to BVDV represent in utero exposure to the virus and may be useful in surveillance as described above but are less useful in identifying PI animals because such animals are immunotolerant to the virus.

Diagnosis of abortion

Diagnosis of bovine abortion can be challenging because a definitive diagnosis is obtained in only 20% to 30% of submitted cases. A recent review on ruminant abortion diagnostics is recommended for a complete discussion of the diagnostic workup.³⁸ When abortions due to BVDV or BHV-1 are suspected, submission of the aborted fetus and the entire placenta provide the best opportunity for establishing a diagnosis of viral infection. Both samples, but particularly the placenta, may be unavailable for submission for a variety of reasons, but it is important to communicate to the owner or herdsman the decreased opportunity for obtaining a diagnosis when either of these samples is unavailable. With the widespread use of viral vaccines in the United States, single serum samples from aborting dams are not often helpful in determining a diagnosis. An increase in titer as demonstrated in paired samples taken at least 4 weeks apart will be of greater benefit, but the results must be interpreted in light of the animal's vaccine history.

CONTROL**Vaccination**

In North America, vaccination is the primary means of controlling reproductive losses due to the viral pathogens. Vaccination programs for BHV-1 and BVDV should have several goals: (1) prevention of acute disease in the vaccinee; (2) prevention of reproductive losses in the vaccinee; and in the case of BVDV, (3) prevention of the creation of PI animals. Fetal and abortive protection of vaccination against BHV-1 and BVDV is critical to the success of herd health programs, whereby eradication is not feasible or not practiced.³⁹ Although field protection is not complete,^{40–42} challenge studies consistently demonstrate a significant decrease in fetal infection following virulent challenge for both BVDV (**Table 1**) and BHV-1 (**Table 2**) vaccines. A recent meta-analysis on the efficacy of BVDV vaccines to protect against reproductive loss found

Table 1
Primary studies to evaluate the efficacy of fetal protection of BVDV vaccination following viral challenge

Reference Number	Study Size	MLV/Inact	Interval (d)	Fetal Protection (%)
Brownlie et al, ⁷³ 1995	18	Inact	25–90	100
Ficken et al, ⁷⁴ 2006	58	MLV	370	100
Frey et al, ⁷⁵ 2002	15	Inact/MLV	122	100
Givens et al, ⁷⁶ 2012	29	MLV	102	100
Meyer et al, ⁷⁷ 2012	22	MLV	120	100
Patel et al, ⁷⁸ 2002	18	Inact	187	100
Rodning et al, ⁷⁹ 2010	47	MLV	91	100
Schnackel et al, ⁸⁰ 2007	65	MLV	110–150	98
Fairbanks et al, ⁸¹ 2004	55	MLV	120–132	97
Xue et al, ⁸² 2011	35	MLV	121	96
Ellsworth et al, ⁶⁵ 2006	30	MLV	490	95
Kovacs et al, ⁸³ 2003	35	MLV	88–146	95
Arenhart et al, ⁸⁴ 2008	28	MLV	138	94
Brock & Grooms, ⁸⁵ 1996	18	MLV	100–112	92
Dean et al, ⁸⁶ 2003	36	MLV	121	92
Brock et al, ⁸⁷ 2006	54	MLV	125–135	89
Mcclurkin et al, ⁸⁸ 1975	27	Inact	30–60	89
Rodning et al, ⁷⁹ 2010	28	Inact	91	89
Leyh et al, ⁸⁹ 2011	50	MLV	124	85
Cortese et al, ⁹⁰ 1998	18	MLV	107	83
Grooms et al, ⁹¹ 2007	29	Inact	69	79
Xue et al, ⁹² 2009	83	MLV	191–205	78
Ficken et al, ⁹³ 2006	74	MLV	125–146	76
Harkness et al, ⁹⁴ 1985	21	Inact	85–90	64
Brock & Cortese, ⁹⁵ 2001	25	MLV	120–150	58

Study size describes the number of animals included in the study. MLV/Inact is the type of vaccine administered to experimental groups. Interval (d) is the interval in days between the administration of vaccine and the challenge of pregnant animals. Fetal protection is the percentage of vaccinees that produced calves free of infection.

Abbreviation: Inact, inactive.

Table 2 Primary studies to evaluate the efficacy of fetal protection of bovine herpesvirus 1 vaccination following viral challenge				
Reference Number	Study Size	MLV/Inact	Interval	Fetal Protection (%)
Smith et al, ⁹⁶ 1978	31	MLV	282–316	100
Givens et al, ⁷⁶ 2012	29	MLV	102	100
Pospisil et al, ⁹⁷ 1996	40	Inact	21	97
Saunders et al, ⁹⁸ 1972	33	MLV	159–219	94
Cravens et al, ⁹⁹ 1996	20	MLV	219	90
Zimmerman et al, ⁶⁶ 2013	51	MLV	240–390	88
Zimmerman et al, ¹⁰⁰ 2007	35	Inact	230	86
Ficken et al, ¹⁰¹ 2006	29	MLV	365	84

Study size describes the number of animals included in the study. MLV/Inact is the type of vaccine administered to experimental groups. Interval (d) is the interval in days between the administration of vaccine and the challenge of pregnant animals. Fetal protection is the percentage of vaccines that produced calves free of infection.

Abbreviation: Inact, inactive.

that vaccination was associated with an overall decrease in abortions of nearly 50% and a nearly 85% decrease in fetal infection rate in vaccinates.⁴³ Consequently, a sound vaccination program will reduce reproductive losses but may not completely prevent all reproductive consequences of viral infection.

Selecting an appropriate vaccine type depends on the strengths and weaknesses of modified-live (MLV) or inactivated vaccines in light of the herd history and current management goals of the farm.⁴⁴ In general, MLV vaccines stimulate higher production of neutralizing antibodies and longer duration of protection than inactivated vaccines.^{45,46} In addition to inducing significant antibody production, MLV vaccination also stimulates cell-mediated immunity.^{47,48} Although peak immunity following vaccination with inactivated vaccines is seen only after the initial dosing schedule is complete, partial and complete protection from experimental BVDV challenge has been demonstrated at 3 and 5 days, respectively, after an initial dose of MLV vaccine.⁴⁹ Inactivated vaccines are generally safe for use in pregnant cattle with no, or unknown, vaccine history; however, several MLV vaccines have been developed for administration to pregnant cattle when label conditions are met. Even when used according to label conditions, MLV vaccines may be associated with subsequent abortions involving BHV-1 in a small number of pregnant cows (eg, 1 in 235 heifers aborted with detected BHV-1 in 1 study).⁵⁰ Because of demonstrated efficacy, the use of MLV vaccines is strongly encouraged when vaccinating nonpregnant cattle at least 30 days before breeding. Significant protection can still be achieved using inactivated vaccines: a meta-analysis of BVDV vaccines demonstrated a 34% decrease in abortion rate and a 76% reduction in fetal infection rate in vaccinated cattle compared with unvaccinated controls.⁴³

The existence of multiple genotypes for both BVDV and BHV can pose a challenge to providing optimal vaccinal coverage. Both genotypes of BVDV (BVDV1 and BVDV2) are common in US cattle populations⁵¹ and are clinically indistinguishable. Although BHV-1 has historically been the primary herpesvirus of concern in cattle, there is evidence that BHV-4^{52–54} and BHV-5⁵⁵ may also cause reproductive disease in cattle populations. Although vaccination with heterologous strains provides some degree of protection against other genotypes,^{56–59} immunity is generally inferior to vaccination with homologous strains or unable to prevent infection.^{60–63} Currently, vaccines

for BHV genotypes other than BHV-1 are not commercially available in the United States; however, polyvalent BVDV vaccines are commonplace. Inclusion of the 2 BVDV genotypes in a vaccine generally provides superior protection against the varied BVDV isolates to which cattle may be exposed. Thus, the use of vaccines containing type 1 and type 2 BVDV is recommended to help prevent disease caused by the varied strains encountered in the field.

Replacement heifers

Replacement heifers should receive their full complement of vaccinations for BVDV and BHV-1 at least 30 days before the onset of the breeding period. In a group of BVDV-challenged cattle near the time of breeding, conception rates were 78.6%, 44.4%, and 22.2% for cattle that seroconverted before, during, or after breeding, respectively.² When the second dose of an MLV polyvalent vaccine was given to 60 heifers seronegative to both BVDV and BHV-1 at least 10 days before synchronized natural breeding, no negative effects on reproductive performance were observed.⁶⁴ The researchers evaluated the duration of interestrus intervals, proportion of heifers responding to synchronization, serum progesterone concentrations, pregnancy rates, and pregnancies during the first 5 days of the breeding season. Consequently, vaccination of replacement heifers before the onset of breeding is both safe and effective. It is the authors' preference that replacement heifers be vaccinated using a multivalent MLV vaccine for optimal efficacy of protection.

Mature herd

Vaccination of the mature herd should be timed to match the peak of vaccinal immunity to the highest risk of reproductive loss due to viral challenge. In the cow-calf herd, revaccination is often performed annually, at least 2 weeks before the breeding season. Thus, after-vaccinal antibody titers are expected to persist through the breeding season and the crucial first 125 days of gestation. In dairy operations, cattle are typically in various stages of gestation so whole-herd vaccination schemes can be more problematic, particularly when MLV vaccines are used. Alternatively, an event-driven vaccination protocol may be desirable; cattle are vaccinated after a specific event (eg, dry-off, prebreeding) rather than a specific time of year. Most vaccines are labeled for annual revaccination, which may pose a problem for event-driven programs because the average calving interval on US dairies increases. Duration of fetal protection often extends beyond 12 months,^{65,66} but adherence to all label directions is encouraged.

Biosecurity

Preventing the untimely introduction of BVDV and/or BHV-1 to naïve herds is essential to maintain herd health and limit reproductive losses in breeding female cows. Both viruses are spread through bodily secretions, including nasal exudate, genital secretions, semen, and respiratory droplets. In particular, PI animals shed high levels of infectious virus in all bodily secretions, which can be transmitted by fomites for a short period. Airborne transmission in the absence of direct contact is not a major route of transmission and occurs only over very short distances. However, it is recommended that groups more likely to contain individuals undergoing active infection (eg, young stock, introduced animals) not be housed adjacent to pregnant cattle whenever possible.

Cattle represent the major reservoir for spread of both viruses, although infection may be seen in multiple species. Cross-species transmission of BVDV has occurred in specific experimental situations,^{67,68} although the risk of interspecies transmission

remains largely uncharacterized. Prevention of exposure of pregnant cattle is a key factor to limiting reproductive losses from BHV-1 and BVDV.⁶⁹ Younger stock represents the cattle population most likely to be experiencing active infection with either virus; thus, limiting commingling and fence-line contact between these groups and pregnant cattle is recommended. Breeding bulls should also be protected from potential exposure because both viruses are capable of being passed in the semen following natural infection.^{8,70}

Introduced animals

Introduced animals represent the greatest biosecurity risk for the introduction of infectious pathogens to a clean herd. Introduced cattle should be quarantined for a minimum of 3 weeks unless the animal has been shown to be free of active viral infection. The quarantine area should not be located adjacent to, or in contact with, areas holding pregnant cattle. Quarantining newly acquired animals is often overlooked or ignored by producers, but the risks posed to the reproductive health of the herd due to lack of quarantine should be effectively communicated.⁷¹ Minimally, purchased animals should be tested to ensure they are not PI, preferably using an antigen detection assay as described above. It is important to note that most tests will not detect transient infections; thus, a quarantine period is still recommended even if the animal is shown not to be PI. Vaccination of introduced animals will depend on the vaccination history and gestational stage, if pregnant, of the animal. If the animal is not pregnant and has an unknown history, the authors prefer 2 doses of a multivalent, MLV administered per label instructions before introduction to the herd. If the animal is pregnant, a multivalent inactivated vaccine may be substituted.

Bred replacement heifers pose a potential double threat because the fetus may also serve as a reservoir of BVDV, even if the dam is not infected. An animal purchased in midgestation carrying a PI calf represents a significant threat for the introduction of BVDV even if the recommended quarantine period is observed for the dam. Currently, there is no practical way to determine the BVDV status of a gestating fetus; percutaneous aspiration of fetal fluids can be used to identify PI fetuses, but the procedure may be associated with increased fetal loss.⁷² Consequently, testing of any newborn calves of introduced animals at the time of birth is a critical component of the biosecurity plan, particularly in cow-calf operations where the calf will likely not be weaned until dams are in midgestation of the next pregnancy,⁶⁹ creating the potential for additional PI births.

SUMMARY

Both BVDV and BHV-1 can have significant negative reproductive impacts on cattle health. Although latent BHV-1 infections are nearly universal, the potential for PI animals to silently introduce and/or spread BVDV within the herd warrants herd surveillance; molecular technologies have made such surveillance rapid and economical with minimal labor involvement. Introduced animals should also be BVDV tested. Vaccination is the primary control method for the viral pathogens in US cattle herds. Polyvalent, MLV vaccines are recommended to provide optimal protection against various viral field strains. However, the importance of biosecurity in the control of BVDV and BHV-1 cannot be overstated. Of particular importance to BVDV control is the limitation of contacts of pregnant cattle with potential viral reservoirs during the critical first 125 days of gestation. In addition, introduced animals should be quarantined and tested for persistent infection before introduction to the herd.

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Diagnosis and Control of Bovine Neosporosis



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KEYWORDS

• *Neospora caninum* • Abortion • Transmission • Prevention • Parasite • Review

KEY POINTS

- Neosporosis is one of the most widespread and frequent causes of bovine abortion.
- The two major methods of parasite transmission to cattle are ingestion of oocysts shed by infected canids and transplacental transmission from infected dams.
- Effective vaccines have not yet been developed.
- Good management practices can help control neosporosis in herds, but complete eradication is usually impractical.
- The long-term key to avoid or reduce high infection prevalence, is to protect Total Mixed Rations and drinking water from contamination by canine feces.

PUTTING NEOSPOROSIS IN PERSPECTIVE

A recent analysis estimated that neosporosis costs the US dairy industry \$546 million and the beef industry \$111 million per year.¹

Worldwide, neosporosis ranks among the most widespread and difficult-to-control causes of bovine abortion. For comparison, consider 4 other common infectious causes of abortion. Bovine *Brucellosis* has been eradicated from most wealthy nations; elsewhere, it may be possible to eliminate it from closed herds, and in other cases, abortion may be prevented or at least partially controlled by vaccination. Bovine *Pestivirus* (BVD virus) infection can be eliminated from closed herds and has even been eradicated from a few European countries, and vaccines are available in most countries. Bovine *Herpesvirus*-1 (IBR virus) has been eradicated from some European countries, and elsewhere, abortion can be prevented by vaccination. *Leptospirosis* control is challenging because there are many different serovars that may be transmitted by various wild or domestic animals; nevertheless, vaccines are available to provide short-term protection against the most important serovars, and antibiotic treatment can clear carrier cattle or entire herds from infection with bovine-adapted serovars.²

The author has nothing to disclose.

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Vet Clin Food Anim 32 (2016) 443–463

<http://dx.doi.org/10.1016/j.cvfa.2016.01.012>

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In contrast, neosporosis occurs in all countries; no vaccine is currently available, and latent infections cannot be cleared by antimicrobials. Maintaining a closed herd cannot guarantee freedom from infection because the causative parasite may be transmitted in feedstuffs or water, and the parasite naturally cycles within wildlife.³

Critically, brucellosis and leptospirosis are zoonotic, whereas neosporosis is not. Furthermore, neosporosis is seldom if ever a cause of regional or international trade restrictions, unlike brucellosis, bovine pestivirus, and bovine herpesvirus. The economic importance of neosporosis lies simply within its effect on the reproductive performance of breeding cows.

CLINICAL MANIFESTATIONS OF BOVINE NEOSPOROSIS

Most infections in cattle are subclinical, but there are frequent exceptions. Abortion is the only major problem, which is generally not associated with other signs of illness in dams. Abortions may occur between 4 months of gestation and birth, but most occur in months 5 through 7. Neosporosis is not a significant cause of infertility or early embryonic resorption. Retained fetal membranes and metritis may be secondary complications that follow abortion.^{4,5}

In addition to abortion, bovine neosporosis is associated with stillbirths or with the occasional birth of premature or neurologically impaired calves.^{6–8} Clinically affected calves may have normal size or be notably small, and signs range from being neurologically moribund to having partial spinal deficits (**Fig. 1**) with poor conscious proprioception of the rear limbs and inadequate balance.

In dairy cattle, one of the costs associated with abortion (from any cause, not just from neosporosis) is reduced milk production. Reduced milk production is expected to occur because of interference with the timing and length of the lactation and dry periods, body conditioning, and udder health.

UNCLEAR ASSOCIATIONS WITH MILK PRODUCTION AND GROWTH RATES

When abortion has not occurred, there are contradictory studies regarding a possible effect of *Neospora* serologic status (ie, the presence of a detectable antibody titer) on milk production. However, the largest studies, involving thousands of dairy cattle and hundreds of herds, indicate that *Neospora* serologic status does not directly reduce milk production. A study in Ontario⁹ concluded that loss of milk production was associated with abortion rather than with simply being seropositive, and a study in the



Fig. 1. Neurologic impairment in calves infected with *Neospora caninum*. The beef calf at left was born following a neosporosis abortion outbreak. It was undersized, had weak hindlimbs, and a conscious proprioceptive deficit that is here demonstrated by the dorsal placement of the left rear hoof. The dairy calf at right was unable to stand, maintain sternal recumbency, or elevate its head.

Netherlands¹⁰ concluded that subclinical or endemic neosporosis is not associated with reduced milk production.

About 15 years ago, a research group in the United States reported that in comparison with uninfected herd mates, *Neospora*-seropositive steers had mildly decreased growth rates and feed efficiency, and increased average time spent in sick pens.^{11,12} However, later studies in Canada and Argentina did not find these problems,^{13–15} and there has been no further published evidence to support an effect on meat production.

THE CAUSATIVE PARASITE

Comparison with the familiar life cycle of a butterfly, which passes through egg, caterpillar, and chrysalis stages before emerging as a sexually competent adult, can help make the life stages of protozoa a bit easier to comprehend. Several microscopic forms of *Neospora caninum* reside inside of cells of infected animal hosts (Fig. 2). The parasite reproduces sexually within the intestinal tract of canid definitive hosts and is then shed in feces as environmentally hardy oocysts (pronounced Ō ō sists).

Intermediate hosts of *N. caninum* are prey animals that become infected either by ingestion of oocysts that contaminate dust and water following decomposition of

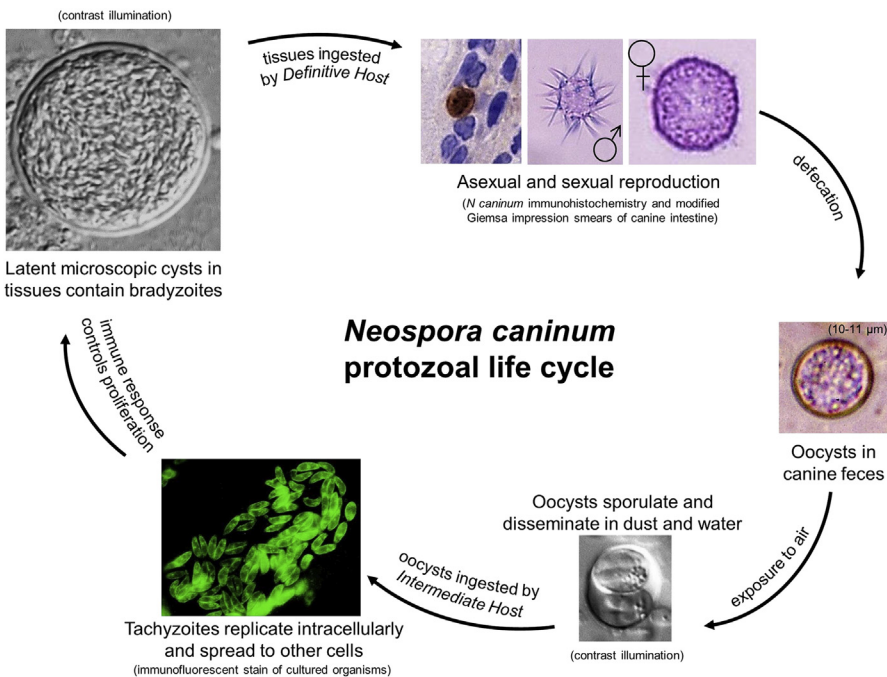


Fig. 2. *Neospora caninum* life stages. (upper left) Photomicrograph of a squash preparation showing a spherical, thick-walled cyst containing numerous bradyzoites; this latent stage survives in tissues for prolonged periods. (upper right) Asexual and sexual reproductive forms of the parasite that occur in canine intestine after ingestion of infected tissues. (right) Egg-like oocyst, shed in canine feces. (bottom right) Sporocysts containing sporozoites have developed within an oocyst, which is now infectious. (bottom left) Upon ingestion of infectious oocysts by an intermediate host animal, sporozoites are liberated by digestive juices, invade host cells, and become rapidly dividing tachyzoites; if unchecked by the immune response, tachyzoites may cause clinical disease. Otherwise, they convert to latent bradyzoites and the cycle is completed.

infected canine feces or by transplacental transmission from the mother. Regardless of the method of infection, there is an initial period of rapid asexual replication of *tachyzoites* within host cells, and then the host cells die as the tachyzoites rupture out and spread to infect new cells.

If clinical disease occurs, it is caused by tachyzoites. However, in most cases, tachyzoites fall under the control of the animal's immune response and then convert into relatively dormant *bradyzoites*, which reside within microscopic intracellular cysts. These cysts often endure for the life of the intermediate host. When an infected animal is preyed upon by a definitive host canid, then ingested bradyzoites become activated by gastric digestion, infect the canine intestinal tract, and the cycle is renewed.

RELATED ORGANISMS

Although there are several closely related parasites including *Toxoplasma gondii*, only *Neospora hughesi* shares *N. caninum*'s genus name. *N. hughesi* has been found in horses, in which it may cause spinal infections and ataxia, but much less is known about it. In this article, all further references to "Neospora" or "neosporosis" will be used to indicate the parasite *N. caninum* and associated disease conditions, without further consideration of *Neospora hughesi* or other related parasites.

TRANSMISSION CYCLES IN CATTLE

There are at least 3 methods of transmission of *Neospora* infection to cattle (**Fig. 3**):

1. Cattle may become infected at any time by ingestion of oocysts. This occurrence may be referred to as horizontal transmission. Upon infection, there will be a period of tachyzoite proliferation, an antibody titer will develop, and then the organisms will convert into bradyzoites within latent intracellular cysts.
2. If a naive heifer or cow first ingests oocysts when she is pregnant, then the infection may breach the placenta and be transmitted to the developing fetus; this is termed

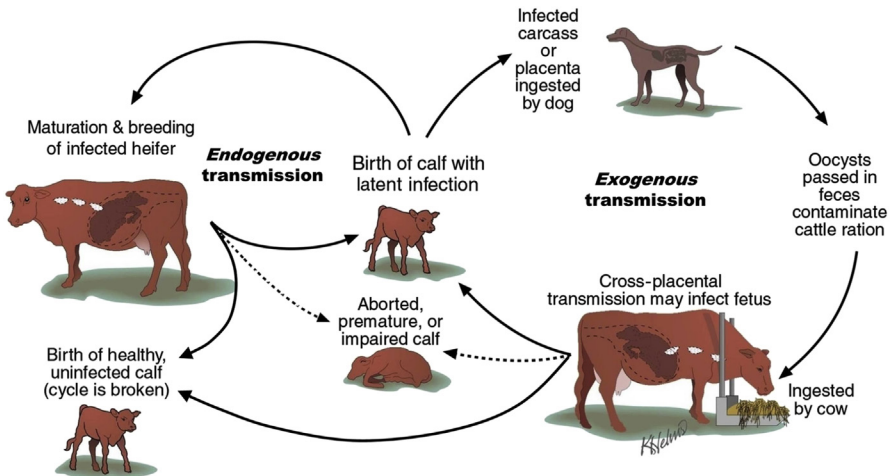


Fig. 3. Transmission of *Neospora caninum*. Cattle may become infected "horizontally" by ingestion of oocysts at any time of life (not only when pregnant). Congenital infection may result if infection is first acquired by a dam during pregnancy (exogenous transplacental transmission), or by reactivation of organisms from a latently infected dam (endogenous transplacental transmission). Artist: Kerry Helms.

*exogenous transplacental transmission*¹⁶ (hereafter shortened to exogenous transmission). Exogenous transmission is a subtype of horizontal transmission in which both the dam and the offspring become infected from the same event.

3. If a female heifer or cow has a latent infection and later becomes pregnant, organisms may reactivate and cross the placenta; this is termed *endogenous transplacental transmission*¹⁶ (hereafter shortened to endogenous transmission). Endogenous transmission may occur in multiple pregnancies of the same dam or may occur across several generations to transmit infection within maternal lines of cattle.

Confusingly, the *Neospora* literature often refers to “vertical” transmission. Technically, vertical transmission indicates transmission from mother to offspring, but this occurs in both endogenous and exogenous transmission; thus statements about vertical transmission are often ambiguous.

NEOSPOROSIS IN OTHER DOMESTIC ANIMALS AND IN WILDLIFE

As with cattle, *Neospora* infection in other animals is usually subclinical, but there are many exceptions. Dogs with clinical neosporosis may show a wide range of problems, such as hindlimb paresis or ataxia with muscle atrophy, myocarditis, dermatitis, or diarrhea. Clinical neosporosis in puppies may affect individuals or litters.¹⁷

Neosporosis abortion or neonatal illness is known to occur in a variety of large herbivores, including goat, sheep, llama, alpaca, several species of deer, lesser kudu, horse, and white rhinoceros.³ Naturally occurring *Neospora* parasitism, but mostly without reports of disease, have been detected in adults of many other species of wild, feral, captive, and domestic animals, including rodents, lagomorphs, kangaroos, sparrows, parrots, and chickens. The possibility that these animals may serve as efficient intermediate hosts of the parasite, inducing patent infections when they are eaten by canids, is plausible but speculative.

Dogs, gray wolves, and Australian dingos (all subspecies of *Canis lupus*) and coyotes (*Canis latrans*) are known to be definitive hosts of *N. caninum*.^{18–21} Red foxes (*Vulpes vulpes*) have also been found to have small numbers of *N. caninum* oocysts in feces and therefore may also be a definitive host²²; however, experimental confirmation has not been achieved and thus the role of fox is uncertain.^{23,24}

There is good evidence that *Neospora* actively cycles between gray wolves and their cervid prey.^{19,25} However, deer also have high seroprevalence rates in regions without wolves, thus suggesting that coyotes and dogs may be sufficient to maintain this wild cycle. Dogs have been induced to shed *Neospora* oocysts after consuming hunter-killed white-tailed deer.²⁵

EPIDEMIOLOGIC PATTERNS IN CATTLE

Three major patterns of neosporosis have been observed in herds of breeding cattle: abortion outbreaks (epidemic pattern), increased annual abortion losses (endemic pattern), and subclinical infections.

Epidemic Pattern

Outbreaks of neosporosis may occur in which a high proportion of pregnant cows abort within a short time period. Outbreaks are generally suspected to have resulted from an event in which the pregnant herd's feed or water has been contaminated with *Neospora* oocysts. This contamination is difficult to prove in retrospect; nevertheless,

several lines of supporting evidence have been obtained from studies of outbreaks. Epidemic curves observed in outbreaks are consistent with point-source exposure events, such as contamination of a batch of mixed feeds, dietary supplements, or drinking water.^{5,26,27} Specialized avidity serologic techniques have provided strong evidence that infections in cows were acquired recently in relation to the time of the abortion outbreak,^{28–31} which is consistent with exogenous rather than endogenous transmission (see Fig. 3). Finally, epidemiologic studies have linked the odds of epidemic neosporosis with the presence and number of dogs on cattle farms, consistent with transmission to cattle by ingestion of oocysts.^{32,33}

Experiments using oocysts are difficult, time-consuming, and expensive. First, infected tissues must be produced by inoculating animals (usually calves) with cultured organisms; then these tissues are fed to dogs to induce production of oocysts, and finally, the oocysts are administered to pregnant cows, which are followed to term. As a result, there have only been 3 small-scale experiments using oocysts in pregnant cattle,^{34–36} so the following observations could use strengthening and refinement by additional investigation. Nevertheless, a pattern begins to emerge from examination of the combined results of the 3 available studies (Table 1). Administration of *Neospora* oocysts to 7 cows in the first trimester of pregnancy, even with the highest dose of 70,000 oocysts, did not result in transplacental infection of any calf. This transmission barrier began to break down a bit later in gestation; of 9 cows administered oocysts between 120 and 130 days of gestation, 6 gave birth to uninfected calves, but the other 3 had transplacental infection with 2 abortions (39–44 days after exposure) and 1 stillbirth. Another 7 cows were exposed in the late-second to early-third trimester, and 6 of their 7 calves were born with congenital infections, even with the lowest dose of approximately 127 oocysts, but were all clinically healthy. This experimental pattern roughly corresponds with the timing of neosporosis abortions in the field. This pattern also resembles transplacental toxoplasmosis in humans, in which the likelihood of transplacental transmission is lowest in the first trimester (but with the most severe consequences) and increases into the third trimester (and is often subclinical).³⁷ Thus, ingestion of *Neospora* oocysts by naive pregnant cows does appear to be capable of inducing an abortion outbreak, provided that exposure occurs

Table 1 Results of administration of <i>Neospora caninum</i> oocysts to pregnant cows, compiled from the only 3 published experiments of this type			
	Day of gestation that oocysts were administered to cows		
	70	120–130	162–210
Number of cows that became infected and were followed to parturition	7	9	7
Number of offspring that became infected	0	3	6
Proportion of infected cows having infected offspring	0.00	0.33	0.86
Number of cows having abortions or stillbirths	0	3	0
Number of aborted fetuses or stillborn calves that were infected	—	3	—
Proportion of infected offspring that were aborted or stillborn	—	1.00	0.00

Data were included from all cows that became infected and that were followed until abortion or birth. All negative control cows gave birth to healthy uninfected calves.
Data from Refs.^{34–36}

within an as-yet imprecisely defined gestational window of susceptibility, at a time when infection may cross the placenta but before the fetus has matured sufficiently to be able to defend itself (Fig. 4).

Endemic Pattern

In herds with endemic neosporosis, seropositive animals are often related along maternal lines that may span several generations.^{38,39} Infected dams may give birth to one or more congenitally infected offspring, which in turn may enter the breeding herd and continue the cycle of endogenous transmission (see Fig. 3). There is a greater relative risk of abortion in *Neospora*-seropositive dams than in seronegative dams. For dairy cattle, a median relative risk of abortion of 3.5 was obtained from a compilation of numerous studies from 10 representative countries.¹ This relative risk indicates that seropositive dams are approximately 3.5 times more likely to suffer an abortion than are seronegative dams; for example, if the background rate of abortion in seronegative cattle is 2% or 3%, then about 7.0% to 10.5% of *Neospora*-seropositive cattle may abort. The data become more complicated when considering the effect of parity and of any previous abortions. For congenitally infected dams, the highest relative risk of having an abortion (7.4-fold in one study) occurs in the first pregnancy.⁴⁰ If the first parity is successful, then the relative risk of abortion drops considerably for future pregnancies, but if the heifer aborts her first pregnancy, then her risk of aborting future pregnancies remains high.⁴⁰

In high seroprevalence dairy herds with year-round breeding programs, endemic abortions will occur more or less randomly throughout the year, although there may be minor seasonal fluctuations.⁴¹ This problem may be so stable that the owner accepts it as normal for the herd, when in fact it represents a persistent drain upon profit.

It is possible for beef or dairy herds with endemic neosporosis, but which practice seasonal instead of year round breeding, to experience a pattern of abortion losses similar to an epidemic. Because most abortions occur between 5 and 7 months of gestation, annually increased losses in mid-gestation may cluster together within a short range of dates.

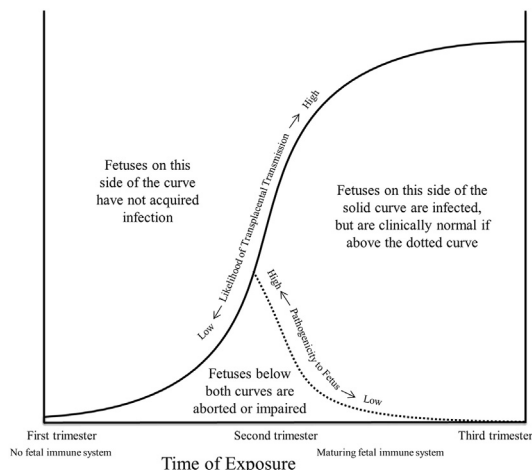


Fig. 4. Relation between the time of maternal exposure to *Neospora caninum* oocysts and the outcome of pregnancy in naive cattle.

Although endemic abortions are often assumed to be from endogenous transmission, horizontal transmission usually contributes to the maintenance of high seroprevalence in endemically infected herds.⁴² Consistent with this, epidemiologic studies reveal statistical associations between the presence and number of dogs and the occurrence of *endemic* bovine neosporosis.^{43,44} Because *Neospora* infection increases the likelihood of abortion and associated culling, a prolonged lack of horizontal transmission in a herd should result in the gradual depletion of seropositive animals, even if endogenous transmission were to occur in 100% of pregnancies (which is seldom the case). Horizontal transmission within endemic herds can occur as an irregular trickle that infects individual cattle at different times (perhaps from grazing), or as very infrequent exposure events that may infect a cohort of cattle (perhaps from contamination of a mixed ration or water).⁴²

The efficiency of endogenous transmission, defined as the percentage of pregnancies of previously infected dams that give rise to congenitally infected offspring, has been the subject of many investigations. **Table 2** lists the 8 largest studies found in a literature search, each reporting the *Neospora* serologic status of at least 100 offspring of seropositive dams. Simple ratios (number of seropositive offspring/number of seropositive dams) vary between 41% and 86%, and the cumulative total provides an average of 63% efficiency of endogenous transmission. However, each of these studies included evidence of horizontal transmission (either postnatal or exogenous transplacental), which in 2 studies exceeded 20% per year.^{45,46} Horizontal transmission causes upward skewing of the simple ratios that are used to calculate endogenous transmission, because some seronegative offspring will become seropositive from postnatal exposure, which gives the appearance of endogenous transmission.⁴² One of the studies in **Table 2** provided a statistically corrected estimate of 45% for the rate of endogenous transmission, down from the listed simple calculation of 62%.⁴² The true efficiency of endogenous transplacental transmission is likely to be somewhat lower than is reflected in the table, but a high degree of variability makes it impossible to be precise.

Table 2 Endogenous transplacental transmission rates of <i>Neospora caninum</i> in dairy cattle			
Citation	Location	Seropositive Offspring/ Seropositive Dams	Simple Calculation of Endogenous Transmission Rate, %
Pan et al, ⁶⁶ 2004	Ontario	252/619	41
Bartels et al, ⁴² 2007	Netherlands	325/526	62
Dijkstra et al, ⁶⁷ 2003	Netherlands	363/500	73
Frössling et al, ⁶⁸ 2005	Sweden	316/369	86
Romero & Frankena, ⁴⁶ 2003	Costa Rica	168/285	59
Dijkstra et al, ⁴⁵ 2001	Netherlands	163/204	80
Bergeron et al, ⁶⁹ 2000	Quebec	64/144	44
Paré et al, ⁷⁰ 1996	California	93/115	81
	Combined	1744/2762	63

Studies were included if they tested the offspring of at least 100 seropositive dams. These calculations have not been adjusted to account for horizontal transmission, which could cause upward skewing.

Endogenous transmission does not require that the dam herself was infected congenitally. This point was illustrated by an intensely investigated herd of beef cows that had suffered a neosporosis abortion outbreak. Avidity serology provided compelling evidence that most cows in the herd had acquired infection recently, consistent with a point source exposure event.³⁰ However, in the following 2 years, the calculated rate of endogenous transmission in that same herd was 85%. This finding is evidence that the initial episode of horizontal infection of pregnant dams, and the occurrence of exogenous transmission, later resulted in a high rate of endogenous transmission in those same cows.⁴⁷

It is likely that long-term endogenous consequences of horizontally acquired *Neospora* infection in female cattle may depend on several variables, perhaps including the age and physiologic state of the animal at the time infection is acquired (eg, not pregnant, pregnant in early term without transmission to the fetus, or pregnant in later term with infection of the fetus), characteristics of the parasite strain, and infectious dose.

Although seropositive dams have a higher relative risk of abortion than do seronegative dams, this only holds true if the cows are not exposed to a horizontal challenge. Conversely, when herds of pregnant cattle have neosporosis abortion outbreaks, avidity serology shows that the previously uninfected cattle are at risk of aborting, whereas previously infected cattle appear to resist the horizontal challenge.^{30,31,48} Experimental infectious challenge of latently infected, pregnant cattle also shows that they resist abortion.⁴⁹

Subclinical Pattern

Overall, *Neospora*-seropositive dams have an increased relative risk of abortion compared with seronegative dams, but this does not hold true for every herd. A large-scale postnatal exposure event has been documented in which abortions were not noted.⁵⁰ A small number of studies have not shown a link between serologic status of dams and the relative risk for abortion. These circumstances are unlikely to be noticed unless performing serologic surveys.

EFFECT OF PRODUCTION TYPE

Overall, the prevalence of neosporosis is higher in dairy than in beef cattle.¹ This prevalence is probably related to production and management factors that affect the odds of exposure to the parasite, rather than a difference in susceptibility. Dairy cattle are more likely than beef cattle to be fed total mixed rations (TMR), which provide opportunities for point source exposure of herds to various ingested pathogens such as *Neospora* oocysts. Also, dairy cows require more feed and water than beef cows, increasing the opportunity to consume a larger dose of an infectious agent. Nevertheless, when they are seropositive, beef cows have a relative risk of abortion that is at least as high as for seropositive dairy cows.¹

There are many more reports of abortion in European breeds of cattle (*Bos taurus taurus*) than in tropical Asian breeds (*Bos taurus indicus*). In one study, purebred Holsteins had a greater abortion risk than did Holstein-Zebu crosses.⁴ Similarly, water buffalo (*Bubalus bubalis*) have infrequently been documented to suffer neosporosis abortion even though they may have a high prevalence of infection.⁵¹ Because of this history of published reports, it is suspected that European cattle are more susceptible to neosporosis abortion in comparison with Asian cattle and water buffalo. However, it is also possible that abortions have simply been underinvestigated and underreported in tropical countries where zebu and buffalo are more common.

DIAGNOSIS

Neosporosis should be included in the differential diagnosis for either endemic or epidemic problems with bovine abortion, together with several other infectious and toxic causes of abortion, depending on the geographic location. Neosporosis abortions will not be associated with current or recent history of systemic illness in the dams, unlike some conditions such as nitrate poisoning, salmonellosis, or leptospirosis. Usually, the best diagnostic approach for an abortion problem is to perform a general workup, rather than to single out any one cause for diagnostic attention. For herds that clearly have an elevated proportion of abortions, diagnostic attempts have a good success rate. Conversely, results are often nondiagnostic for examinations of random abortions from herds that do not clearly have a significant abortion problem, and these may not be worth the trouble and expense of investigation.

So where should the line be drawn between normal background and elevated abortion losses? For dairy cattle, the US National Animal Health Monitoring Service (2007) has suggested a goal of 2% abortions per year, although stating that up to 5% was normal, with national averages of 5.0% for dairy cows and 3.3% for dairy heifers.⁵² For beef cattle, the same service reports abortion prevalence of 0.5% for cows and 0.7% for replacement heifers.⁵³ Some investigators have suggested targets of 5% for dairy and 2% for beef, with annual abortion losses above this triggering investigation and corrective action.⁵⁴ Veterinarians should advise farmers on the establishment of targets that are appropriate for their situation.

Abortion diagnostics can be approached using serologic screening of dams to detect evidence of specific pathogens, or necropsy of aborted fetuses, or a combination of these. Serologic surveys have the advantage of being relatively easy to perform, but a disadvantage is that they can only detect evidence of a few specific agents. Examination of aborted fetuses provides the best opportunity to detect a wide variety of problems, not only common problems but also uncommon but important abortifacients such as *Salmonella dublin* and *Coxiella burnetii*. Disadvantages of this approach include the need to find aborted fetuses, the greater expense of examinations, and greater logistical issues in handling and shipping specimens.

Serologic Screening

At the time of abortion, affected dams usually can be expected to have a high level of specific antibodies against the causative pathogen. For example, it is possible for a single serologic examination of an aborting cow to provide a presumptive diagnosis of neosporosis or leptospirosis, but only if the result is quite high. Just what constitutes "quite high" for neosporosis will vary among diagnostic laboratories, because several different types of *Neospora* antibody detection methods are used, variously reported as titers, optical densities, or sample/positive ratios. As an example, the author considers an indirect fluorescent antibody test titer of 1:400 to be consistent with but not diagnostic of neosporosis abortion, 1:1600 as a likely association, and 1:6400 as a highly probable association. Keep in mind that the cutoff titer between a seronegative and a seropositive result can be set as low as 1:25, but that it is possible for a latently seropositive animal to abort from other causes.

The same serologic specimens can be used to examine titers for *Leptospira* serovars, viral antibody, or antigen tests, and for tests of other pathogens depending on regional considerations.

A more powerful serologic approach is to examine several dams that have *recently* aborted and compare them with a similar number of dams that have not aborted. The greater the number of animals tested, the greater the power of the comparison. For large

herds, a rule of thumb is to test 10 or more animals per group. Results consistent with neosporosis, or of another condition such as leptospirosis, will show that most or all of the aborting dams are seropositive, whereas a lower proportion of the normal animals may be seropositive. There usually are seropositive animals in the normal group, because not every animal that is infected will abort. Statistical comparison can also be performed of the mean antibody levels in aborting and normal groups, with higher levels expected in clinically affected animals; this comparison may be essential in herds that have a very high seroprevalence (or, in the case of leptospirosis, to distinguish between infection and vaccination). Simple statistical procedures, such as the Fisher exact test (seropositive vs seronegative) or Mann-Whitney *U* test (level of titers), are sufficient for these comparisons, and many free online statistical calculators are available.

Examination of Aborted Fetuses

Necropsy of aborted fetuses provides the greatest chance for arriving at a diagnosis, regardless of the cause of the abortion problem. Diagnostic rates will be low when examining randomly lost fetuses within normal background levels of abortion, and many are presumed to have suffered genetic, placental, or hormonal defects. However, in the face of an abortion outbreak, the odds of achieving a meaningful diagnosis are much higher. Despite this, examination of more than one fetus is often needed. The diagnostic value of any one fetus cannot be guaranteed, in part because there is great variability between the time that the fetus dies and when it is expelled from the uterus, so that autolysis and putrefaction can be advanced. Nevertheless, accurate diagnosis has often been achieved from autolyzed or even mummified fetuses, so almost all fetuses have potential diagnostic value.

If there is convenient access to a veterinary diagnostic laboratory (VDL), then timely delivery of the entire chilled fetus may be the best option. When possible, include placenta (especially including cotyledons) and a serum sample from the aborting dam.

Alternatively, the veterinarian should examine the fetus, collect specimens, and ship these to a VDL. [Table 3](#) provides a general list of specimens to collect. Histology often

Table 3 Generally suggested specimens to send for bovine abortion diagnostics			
	Fresh Tissues	Fluids	Formalin-fixed Tissues
From head	Eyeball	—	Half of brain (even if soft) Cross-section of tongue
From thorax	Lung	Fetal fluid from any site (thorax, abdomen, or pericardium)	Lung Thymus Myocardium Diaphragmatic muscle
From abdomen	Liver Spleen Kidney	Abomasal fluid collected with sterile technique Fetal fluid if needed as listed above	Spleen Liver Adrenal Kidney
Placenta	+/-	—	Especially cotyledon
From dam	—	Serum	—
Information	Proportion affected; time course in herd; the presence of clinical signs in cows; a finding of icterus, mummification, or other lesions in fetus or placenta; recent movements, feed changes, or procedures; conditions suspected		

Specific consultation is recommended with the servicing VDL to correspond with the types of tests that they use.

provides the best value for money as a general surveillance technique that includes options to add additional tests using immunohistology and some types of polymerase chain reaction (PCR). For bacterial culture, abomasal fluid is an excellent specimen, easily collected using a syringe and needle without contamination, and transferred into a sterile vacuum tube or similar device for shipping. Abomasal fluid in part consists of swallowed fluid from the amnion and may contain pathogens that have caused placentitis or fetal pneumonia, yet abomasal fluid will remain free of the environmental contamination that affects the placenta upon abortion.

Safe secure packaging, chilling, and rapid shipment of biological specimens are essential, as is the inclusion of adequate information (something that is too often neglected!) (Fig. 5).

Aborted fetuses are often soft from autolysis, because fetal death typically occurs well in advance of expulsion. If the fetus is retained in the uterus for an unusually protracted period after death, then it may become dehydrated and shrunk; this “mummification” cannot occur with most bacterial infections, so neosporosis is one of the most common causes of this uncommon occurrence.⁵⁵ Other gross lesions of the fetus may be observed in a variety of abortifacient conditions that are beyond the scope of this discussion.

Examination of Farm Dogs

Diagnostic efforts in farm dogs are usually of questionable value, because there is not a clear strategy about what to do with the information. Serology can be performed. Seropositive dogs were infected at some time in the past, and they may have shed oocysts. However, the period to shed oocysts is usually (not always) less than 2 weeks, so seropositive dogs are not more likely to shed *Neospora* oocysts than are seronegative dogs. Seronegative dogs may be naive to the parasite and thus susceptible to acquiring infection and shedding oocysts. Parasitologic examination of canine feces may be attempted to observe oocysts, but because the period of shedding oocysts is likely to be brief, there is always a low likelihood of observing oocysts in any one specimen, whether the cattle herd has a high seroprevalence or not. The oocysts are about $10 \times 11 \mu\text{m}$ in diameter (see Fig. 2), which presents approximately 2% of the surface area of *Toxocara canis* ova, so they are easily missed. Furthermore, oocysts of a closely related but nonpathogenic organism, *Hammondia heydorni*, are nearly identical in appearance (although $12 \times 14 \mu\text{m}$), so positive identification of *Neospora*-like oocysts must be confirmed by PCR or other specialized laboratory techniques. For comparison, oocysts of *Cryptosporidium parvum* in the feces of diarrheic calves are even smaller, $5 \times 7 \mu\text{m}$, and *Giardia* cysts in feces are about $9 \times 13 \mu\text{m}$.

WHY NEOSPOROSIS IS SO PREVALENT TODAY

Historically, all breeding cattle were managed extensively. Even dairy cattle were kept in small enough groups that could be milked by hand. *Neospora* oocysts in canine feces would be deposited in the great outdoors, where gradual weathering would have slowly disseminated the oocysts into the immediately surrounding dirt or surface water. Weeks or months later, individual cattle could become infected if they happened to graze the contaminated spots. Small groups of cattle might acquire infection by drinking together from a small water hole, but in larger bodies of water the dilutional effects would be too great to enable efficient transmission. As a result, *Neospora* would tend to infect individual animals. The prevalence of bovine neosporosis was probably much lower under those circumstances.

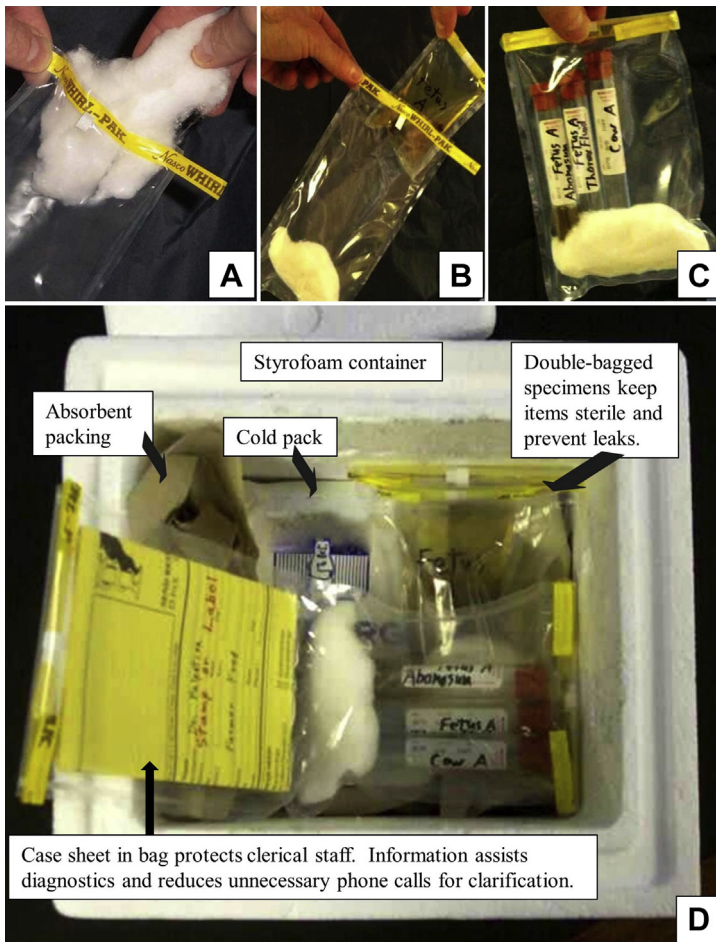


Fig. 5. Submitting specimens for abortion diagnostics. (A) Wet specimens of any type should be packaged within 2 containers, an inner primary and an outer secondary container, with absorbent material in between. Here, absorbent material is placed in the bottom of a biological specimen bag (not a zip-closing sandwich bag) to provide reliable secondary containment. (B) Tightly closed primary specimen containers are suitable for formalin specimens or fresh tissues. (C) Vacuum serum tubes are suitable sterile containers to hold liquid specimens including abomasal fluid, fetal fluids from thorax or abdomen, and serum from the aborting dam. (D) A package ready to be sealed and shipped.

In recent decades, and accelerating since the time that bovine neosporosis abortion was first described in 1988,^{56,57} cattle enterprises are increasingly mechanized. Tractors and milking machines have enabled farmers to keep more and more cows in smaller and smaller spaces. As a consequence, it is no longer practical for large dairies to send cows to pasture during the day, but instead all feedstuffs are harvested and brought to the farm. Nutritional advances have led to the widespread adoption of TMR that contain a variety of feedstuffs and additives. One of the most commonly produced feedstuffs is silage, which decades ago tended to be stored within secure silo structures that could not be accessed by dogs or other animals. However, solid erect silos are expensive and can be slow to unload; as herd sizes began to increase, other

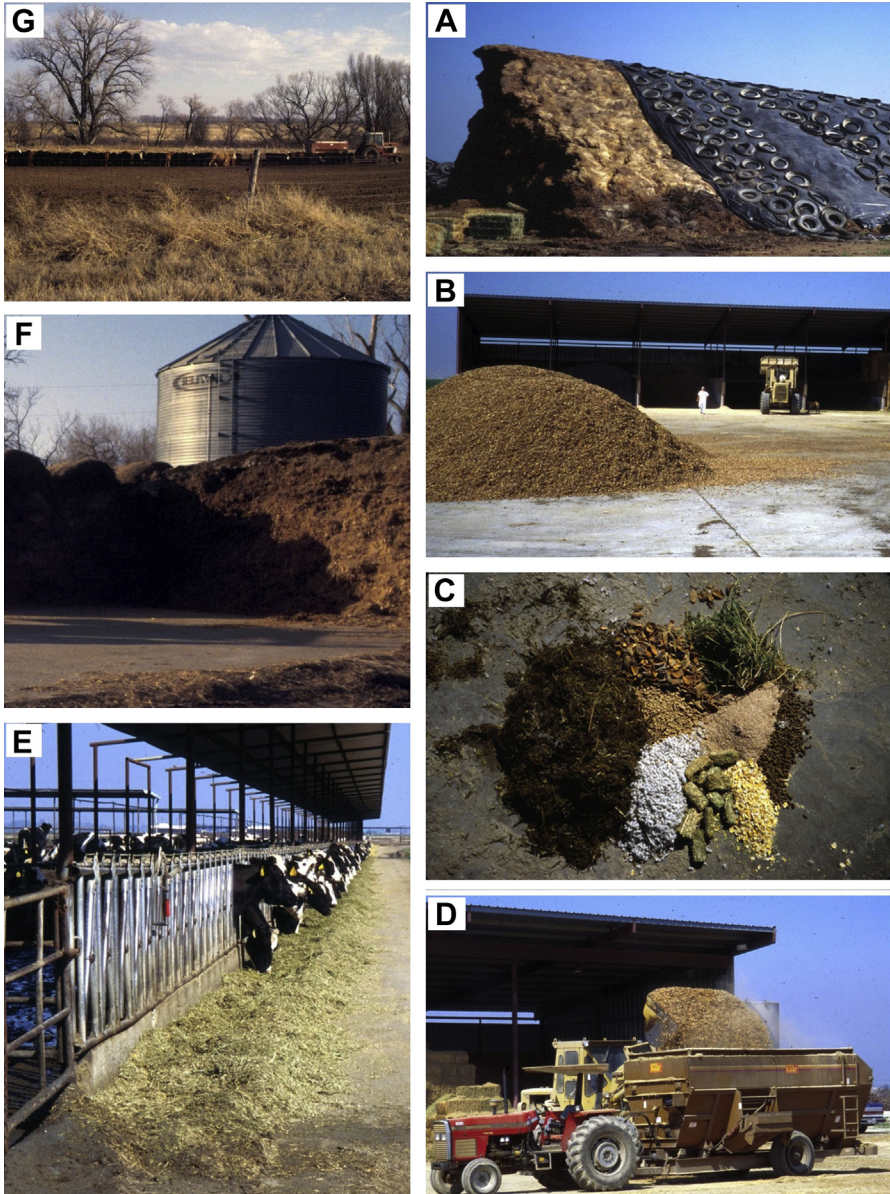


Fig. 6. Potential contamination of feedstuffs with *Neospora caninum* oocysts and dissemination in mixed rations. Representative dairy (A–E) and beef (F, G) farms are depicted that each had a recent neosporosis outbreak. Clockwise from upper right: (A) Above ground ensiling practices increase the opportunity for rodents to frequent or inhabit the silage, especially at the leading edge and around the sides, where discoloration reveals that anaerobic conditions have not been adequately maintained. When examined, rats and mice were flushed out of the silage pile. Dogs and other canids are attracted both to open silage and to rodents. (B) Examples of ration components piled in open bays or outside at a large dairy farm. (C) Various components of the mixed ration. (D) Use of a front-end loader and mixer wagon to prepare a TMR. If any component is contaminated with *Neospora* oocysts (or

forms of ensiling large amounts became more common, such as above ground or in pits. The variety of feedstuffs used in a typical TMR increased with computerization and development of least-cost ration analyses, and this in turn required that a greater variety of feedstuffs be purchased and stored until used. As a result of these technological advances and increases of scale, opportunities for widespread transmission of ingested pathogens have magnified (including *Neospora*, *Leptospira*, and *Salmonella*). Simply by contaminating a pile of exposed feedstuffs, a herd or pen of cattle may become exposed when the ingredient is mixed into a TMR and delivered for feeding (Fig. 6). If the cattle are not mature, or are not pregnant, or are not in a susceptible stage of pregnancy, then abortions do not occur. If some or most of the cattle happen to be in a susceptible stage of pregnancy, then abortions may occur (see Fig. 4). Either way, the prevalence of infection is augmented. Because infections last for life, and because many infected dams will endogenously transmit infections to future generations, the augmentation of seroprevalence has a prolonged effect.

In turn, maintaining a herd that has a high *Neospora* seroprevalence increases the opportunities for dogs or other canids to consume infected tissues, from either consuming placentas,⁵⁸ dead stock, or discarded offal, and then shed oocysts, thus increasing the frequency of environmental contamination and the associated likelihood of horizontal transmission.

CONTROL OPTIONS

The long-term key to avoid a high prevalence of infection, or to reduce a prevalence rate that is already high, lies in the protection of feedstuffs from contamination with canine feces. The historical, low-level, random acquisition of *Neospora* by grazing an infected spot of pasture cannot presently be prevented. It should be possible to reduce the prevalence of infection in intensively managed cattle, but not below the levels of infection that commonly occur in extensively managed cattle. Complete elimination of *Neospora* from a herd is not a recommended goal unless there are exceptional circumstances.

Feedstuffs used in mixed rations need to be better protected, so as to inhibit visits by dogs or wild canids. In small enterprises, this could be accomplished by maintaining feedstuffs in bins, silos, or behind closed doors. For large mechanized dairies, investment is needed to protect feedstuffs. The simplest solution may be use of dog-proof fencing. However, fences are no better than their gates, and it could become quite tiresome to have to manually open and close large gates for the heavy machinery involved in delivering feedstuffs, mixing rations, and feeding cattle. Gates could be left open during the day when predators are less likely to visit and operators are more likely to be aware. However, the best solution could be installation of automatic gates.

For operators that do not have a dedicated feed storage area, erection of electrified predator fencing around stored feeds could be used to inhibit canine access without the need to install permanent fencing.

other orally transmitted pathogens such as *Leptospira* or *Salmonella*), then it will become distributed throughout the TMR. (E) Feeding the TMR to a pen of dairy cows. Potential exists to deliver pathogens to many animals at a time. (F) This outdoor silage pile on a beef farm was not covered, and animal tracks and scats of many types were present, potentially increasing attractiveness to dogs and coyotes. (G) Provision of mixed silage and chopped hay to the beef cow herd, which were periodically brought in from winter pasture for supplemental feeding.

Similar consideration should be given to the protection of drinking water. The most likely sources of contamination would be small surface ponds or bogs, which could become contaminated with runoff from surrounding ground. Watering from elevated troughs should reduce risk and is a practical solution for most intensively managed herds.

Unfortunately, epidemiologic studies are clear that the presence of dogs on breeding cattle premises is a risk factor for bovine neosporosis, and therefore, removal of all dogs may be considered as a potentially effective method to reduce this risk. However, the author does *not* recommend a blanket ban on farm dogs and thinks that it is possible to reduce the risk of horizontal transmission of *Neospora* to cattle without the removal of all dogs. First, the farm should only keep the dogs that they really want on the property. Larger numbers of dogs on a property increase the risk of bovine neosporosis, and probably the greatest risk occurs when there are litters of puppies about. Compared with adult dogs, puppy litters are more likely to be naive to the parasite, and thus, they have greater potential to produce oocysts on first exposure to an infected meal. Therefore, breeding dogs and raising puppies on a cow-calf or dairy farm may be stretching luck a bit far. Second, be a good neighbor and keep an eye on where your dogs go and request the same in return. If unwanted stray dogs frequent your farm, then ask for help from local authorities. Third, even if a dog becomes infected and sheds *Neospora* oocysts for a period of time, this could be relatively unimportant as long as the dog's feces do not end up in the mixed ration; this gets back to the issue of fencing and the use of secure containers.

Another consideration is that some dogs will actively guard their territory and thereby reduce visits by wild and stray canids, thus providing some measure of protection from exposure to *Neospora*. Evidence consistent with this possibility was found for beef cattle,⁵⁹ in which exposure probably occurs in individual animals during grazing, rather than from mass exposure of a contaminated TMR. An infected working dog will only shed oocysts for a brief period, which is unlikely to infect a large number of grazing cattle. Afterward, the dog will probably be refractory to oocyst production,⁶⁰ while throughout its lifetime that same dog may reduce visits from many wild or feral canids.

What can an enterprise do if it already has a high prevalence of neosporosis abortions? In the author's opinion, any management strategy must include protection of feedstuffs. Without such protection, any reduction in the seroprevalence of cattle may not endure. However, if appropriate measures have been implemented to protect feedstuffs, and the farmer does not wish to wait for the seroprevalence to slowly reduce over the years, then consideration may be given to performing *Neospora* serology on all cows and heifer calves. Seropositive dams can be presumed to have a high (possibly 63%; [Table 2](#)) rate of endogenous transmission, so one possible method to speed a reduction in herd prevalence is to not retain heifer calves that are born to seropositive dams. A suggested variant of this strategy is to inseminate all seropositive dams using beef semen, and it has even been suggested that hybrid pregnancies are less susceptible to abortion.⁶¹ A second but similar approach is to perform serology on all potential replacement heifers, regardless of the serologic status of their dams, and then retain only seronegative animals to enter the breeding herd; this serology could be performed from precolostral blood specimens obtained at birth, but more commonly, it would be performed after maternal immunity has waned, perhaps at 6 months of age.

Finally, if a seropositive cow or heifer has particularly valuable genetics, then the farmer may wish to consider using embryo transfer to ensure that endogenous transmission does not occur.⁶² Surrogate cows should be selected after careful screening

for *Neospora* and other unwanted pathogens such as bovine viral diarrhea virus, bovine-adapted *Leptospira* serovars (hardjo-bovis and hardjo prajitno), *S dublin*, and bovine leukemia virus.

No matter how well-designed and conscientiously practiced, neosporosis management programs may not achieve or sustain complete elimination of neosporosis from a herd. Certain factors are beyond control, such as whether a particular feedstuff or additive could have been contaminated before delivery, a stray or wild canid defecates in a pasture that is used for grazing or to make haylage, or if there is a chance contamination event of water. There may even be additional methods of parasite transmission that have not been described; for example, it is plausible that tissues of infected rodents could be chopped and mixed into a TMR, and such mechanically assisted carnivorousness could be sufficient to infect cattle.

FUTURE PROSPECTS FOR VACCINES

Effective vaccines for bovine neosporosis are sorely needed. Although a killed vaccine was previously available in the United States, it was not reliable and is no longer in production.

A highly effective live-attenuated vaccine for a very similar condition of sheep, caused by *T gondii* rather than *N caninum*, has been used in New Zealand and parts of Europe since 1988.⁶³ There is good experimental evidence that bovine neosporosis could also be controlled by a similar vaccine containing attenuated organisms⁶⁴; however, to date, none has been developed and commercialized. Government-sponsored research of animal diseases has been markedly curtailed in the United States⁶⁵ and currently tends to favor conditions that are bioterrorist threats, have zoonotic potential, or that restrict international trade. Neosporosis does not meet those funding criteria, despite its economic importance to the cattle industry in general and to intensive dairy enterprises in particular, and this has inhibited research efforts in the United States over the last 15 years.

Development of an effective neosporosis vaccine for dogs would also be a most welcome aid for the control of bovine neosporosis and could alleviate concerns about the traditional place of pet and working dogs around breeding cattle.

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