

# Cow - Calf Split Session I

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## Neonatal Calf Management

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Perinatal and neonatal calf mortality account for the second greatest loss to the beef cattle industry; the first being open or late calving cows. Large surveys have shown perinatal deaths to range from 4 to 7 percent and neonatal deaths to range from 2 to 6 percent.<sup>18 27</sup> Certain herds and areas have mortality rates much higher in epizootic situations. The purpose of this presentation is to discuss some of the causes of such losses and management practices to reduce the losses.

### Causes

Congenital anomalies have always been a small source of perinatal and neonatal fatalities. Occasionally the incidence of congenital anomalies can be alarmingly high in an individual herd. This occurs when (1) a teratogenic agent invades the embryo or fetus at a susceptible stage of development or (2) when the gene frequency for a genetic anomaly is very high within the herd. A veterinary practitioner should seek diagnostic assistance before declaring an anomaly is of genetic etiology.

Viral agents such as BVD and bluetongue may cause congenital anomalies if a susceptible dam is infected in the second trimester. These are most commonly exhibited as internal hydrocephalus, cerebellar hypoplasia, and other neurological anomalies. Some calves will be born dead, and others may live but have varying degrees of ataxia. It is important to remember that such conditions are sporadic unless many dams are susceptible and infected at the correct stage of gestation. Losses due to BVD can be prevented by vaccination of all replacements with BVD vaccine. Losses due to bluetongue infections have not been fully assessed, but some investigators feel bluetongue is an important cause of calf losses in endemic areas.<sup>6</sup> There is currently no vaccine for bluetongue.

Plant teratogens which cause lethal anomalies exist in certain localities, but these also are not important on a national scale.

### Neonatal Isoerythrolysis (NI)

In the late 1960's a fatal hemolytic disease of newborn calves was observed with increasing frequency. This

condition was identified to be a disease of passive immunity. Calves born to cows previously vaccinated with anaplasmosis vaccine would develop a hemolytic crisis following nursing. These calves would be normal at birth, stand, and nurse but become weak, collapse, and often die within 24 hours after nursing.

Anaplasmosis vaccine is made from a homogenate of red blood cells from cows acutely infected with anaplasmosis. Antigenic determinants on the red blood cells may be foreign to some cows, so antibodies are produced in response to these RBC antigens. The antigenic determinants are genetic in nature and are linked to those that determine blood types. If the offspring inherits the same blood type from the sire that stimulated antibodies in the dam, NI may occur after colostrum is ingested. The maternal antibodies from colostrum react with the antigenic determinants of the calf's erythrocytes causing hemolysis, anemia, and often death.<sup>24</sup>

**Herds having a high incidence of NI should stop using the anaplasmosis vaccine, and calves should receive colostrum from non-vaccinated cows. The vaccine is very effective in preventing anaplasmosis, so the stockman and veterinarian should judge its value on the basis of total economic return. Current recommendations are to vaccinate no more frequently than every two years and the incidence of NI seems to be decreasing.**

### Weak Calf Syndrome (WCS)

A disease characterized by late abortions, stillbirths, weak calves at birth, and surviving calves which become chronic "runts" was reported in Montana, Idaho, and Oregon in the 1960's. The condition has more recently been reported in Nebraska and other parts of the midwest. The most common sign was weak calves at birth or in the first few days of life. Most calves affected would die.

No etiological agent has been determined to date, and the name 'weak Calf Syndrome' has been adopted to encompass a single or perhaps a multitude of entities.<sup>23</sup>

Symptoms reported include:

1. Stillbirths.
2. Weak calves at birth, depressed, unable or unwilling to

nurse, often dying in a few hours after birth.

3. Diarrhea at birth.
4. Polyarthritis with heat and swelling of carpal and tarsal joints.
5. Affected calves are very susceptible to any stress at birth such as hypothermia and secondary infections. They are refractory to treatment and may become chronic 'runts.'

The most consistent necropsy finds are:

1. Edema and hemorrhage of extremities, especially of the carpus and tarsus.
2. Polyarthritis.
3. Hyperemia of small intestine.
4. Occasionally meningitis.
5. Calves that survive more than a few days show atrophy of fat and atrophy or involution of the thymus.

While no etiological agent has been isolated and identified, the following agents or conditions have been incriminated: (1) Hypothermia,<sup>1</sup> (2) Low prepartum protein for dam,<sup>1</sup> (3) BVD,<sup>23</sup> (4) Bovine adenovirus,<sup>4</sup> (5) Mycoplasma,<sup>23</sup> (6) *Hemophilus somnus*,<sup>28</sup> and (7) Bluetongue virus.<sup>6</sup>

Bull reported in Idaho that cows on very low prepartum protein levels had calves with a higher incidence of weak calf syndrome. He determined that each 0.1 pound of crude protein consumed below two pounds per day resulted in a one percent increase in weak calf syndrome in 12 Idaho herds. The condition was exacerbated by cold weather and did not seem to occur if either prepartum protein intake was adequate or if calves were not born during cold weather.

The signs of WCS have been experimentally produced by intrauterine inoculation with *Hemophilus somnus*, but *H. somnus* has not been isolated from affected calves frequently enough to be identified as the etiological agent. Bovine adenovirus has also experimentally caused the signs and lesions.

Treatment is generally directed towards supportive therapy. It is most important that calves be kept warm and out of drafts. Most have to be fed colostrum with a stomach tube or esophageal feeder. Blood transfusions or plasma from cows that previously produced a weak calf have been reported to be helpful.

Weak calf syndrome may be a 'catch-all' for perinatal deaths in some areas of the United States. The symptoms and proposed etiological agents are varied, but there is little doubt that a specific condition exists in some areas. Besides the identified protein problem in Idaho, there is probably an infectious disease contacted *in utero* because it is seen most commonly in offspring from first-calf-heifers or new additions to the herd. The herds affected in Nebraska were in excellent nutritional status and an infectious agent was suspected.

### Bovine Fetal and Neonatal Immunity

The bovine fetus is immunocompetent during the last trimester, and calves have been experimentally immunized

inutero against *E. coli* by injection of the antigens into the amniotic fluid. These calves withstood the challenge of inoculation with virulent organisms before nursing while controls succumbed to colibacillosis.<sup>2</sup>

Calves are normally born agammaglobulinemic. Immunocompetence is probably the poorest from birth to 2 weeks of age than at any other time. The fetal corticosteroids that are responsible for the initiation of parturition remain at high levels for several days following birth. These high levels of corticosteroids suppress immunogenesis during the first two weeks of life. This immunosuppression is mediated through reduced thymus activity and lymphocyte numbers which results in interference with cellular and humoral immunity.<sup>16</sup> At seven to ten days of age the corticosteroid levels return to normal, and calves have been shown to be able to develop immunity against colibacillosis after this time. Calves that are agammaglobulinemic during this time are likely to die of colibacillosis. On the other hand, properly managed calves that received adequate colostrum soon after birth are likely to survive this critical period and develop immunity through natural exposure.

The most important protection for the neonate remains proper colostrum intake. Hypogammaglobulinemia may result from:

1. Low colostrum intake due either to low volume or inadequate nursing.
2. Poor quality colostrum.
3. Late nursing.
4. Perinatal stress (catch-all).

A calf should consume 6%-10% of its body weight as soon as possible after birth. First, the rate of absorption of immunoglobulins from the gut declines from maximum at birth to no absorption at approximately 24 hours postpartum (Table 1). Second, there is a lag of approximately 5 hours between sucking and 50% maximum antibody levels in the calf's circulation.<sup>12</sup> This means that a calf that nurses at 2 hours of age is still hypogammaglobulinemic at 6-7 hours of age and susceptible to invasion by pathogens.

TABLE 1. Effect of Time of Colostrum Feeding on Ig Absorption.

Time of Feeding (Hrs. PP.)	Plasma Ig Levels mg/ml	% Absorption
6	52.7	66
12	37.5	47
24	9.2	12
36	5.4	7
48	4.8	6

Prepartum nutrition levels have been shown to affect cow colostrum volume and calf serum Ig levels following colostrum ingestion. Cows wintered on free choice silage produced 3.5 pints of colostrum at first milking, while cows wintered on available pasture produced 1.2 pints of colostrum. There was a marked difference in total colostrum Ig levels, and 70% of the calves from the pastured cows were hypogammaglobulinemic after nursing<sup>14</sup> (Table 2).

TABLE 2. Effect of Prepartum Nutrition on Cow Colostrum Levels and Calf Serum Ig Levels

Cows	Prepartum Body Weight	First Milking		Calves Hypo Ig
		Vol. Colostrum	Total Ig	
Silage	1,043	3.5 pts.	108 gm	27%
Pasture	761**	1.2 pts.**	39 gm*	70%

\* — P < 0.01  
 \*\* — P < 0.0005

Regardless of nutritional levels all hypogammaglobulinemic calves were from cows with the lowest levels of Ig in the colostrum. Poor nutrition reduced total volume of colostrum in the pastured cows.

A similar study was conducted in Wyoming where cows were fed 50% energy requirements starting 100 days before expected calving. Thirty days before calving half of the cows were fed 117% energy requirements while other remained on the low level of nutrition. Calves born to nutritionally deprived cows were much more susceptible to diarrhea and mortality was markedly increased<sup>3</sup> (Table 3). Calf immunoglobulin levels were not reported, but prepartum nutrition had marked effects on calf survival and weaning weight.

TABLE 3. Effect of Restricting Prepartum Energy Intake and Elevating it 30 Days Prior to Calving.

	Continuous Low*	Low-High**
	Energy Level	
Cow prepartum Wt. Change	—145	—22
Calf Birth Weight	59	76
Calf Survival to Weaning	71	90
Calf Scours — % Affected	52	33
% Mortality	19	0

\* 50% of energy requirement for last 100 days prepartum  
 \*\* 50% of energy requirement for days —100 to —30 then switched to 117% for last 30 days prepartum.

### Perinatal Stress

In spite of adequate colostrum consumption in the early hours of life approximately 20-30% of neonatal calves remain hypogammaglobulinemic.<sup>16</sup> The reason for this is not known but probably accounts for some of the consistent low levels of colibacillosis in the best managed herds. A generalized category of perinatal stress may explain some of the variation of immunoglobulin levels. These include:

1. Environmental stress.
2. Dystocia.
3. Poor 'mothering-up'.

Arizona calves subjected to extreme heat stress at birth had higher cortisol levels, lower immunoglobulin levels, and higher mortality rates<sup>25</sup> (Table 4). All calves were subjected to heat above the comfort range, but those under extreme stress had lower Ig levels and higher mortality rates.

TABLE 4. Calf Heat Stress — Ig Absorption and Calf Mortality.

	Temperature Humidity Index*		
	81.2	82.4	84.5
No. Calves	36	36	36
Calf Mortality	1 (2.7%)	2 (5.6%)	9 (25%)**
2 Hr. serum Cortisol (mg/ml)	42	42	56**
Ig Conc. 2 days (mg/ml)	25.5	22.0	18.6**

\* Comfort Maximum = 73  
 \*\* — P < 0.01

While the cortisol levels were higher in the extremely stressed calves, the role of cortisol in Ig absorption is not clear in this study. The cortisol level may only be in response to the heat stress. Other studies have indicated that high cortisol levels in calves at birth did not interfere with Ig absorption.<sup>10</sup> Also exogenous corticosteroids used for induced parturition apparently do not hinder absorption, but the early calving may reduce the volume of colostrum.<sup>9</sup>

Dystocia remains the number one cause of perinatal calf mortality. Young in Australia reported that dystocia was responsible for 70% of all deaths at or near birth, and this amounted to 45% of all calf deaths including abortion and postnatal deaths.<sup>29</sup> Patterson and Bellows reported similar figures of 60% and 38%.<sup>18</sup> In all reports most of the dystocia cases are first-calf-heifers.

Stillbirths are not the only result of dystocia, but many calves are injured that later die or are permanently injured. Such injuries reported have been slipped femoral capitulum epiphysis,<sup>7</sup> femoral nerve stretching and paralysis, ruptured quadriceps muscles, meningeal hemorrhage and congestion.<sup>8</sup> Most of the above lesions are due to excessive forced traction where a caesarean section would have resulted in a live calf.

Many factors have been attributed to causing dystocia. Stockmen have traditionally held to the premise that calving difficulty can be controlled by limiting prepartum nutrition so calf birth weight is reduced. Research and experience have conclusively shown that only extremes in body conditions cause high dystocia rates. That is, thin and small heifers have more difficulty because of weakness and small size in spite of somewhat reduced birth weights. Conversely, very fat heifers have more difficulty because internal pelvic fat decreases the pelvic area of the dam. A general rule of thumb is to have heifers at 85% of their mature weight at calving (including calf weight) to minimize dystocia and maximize subsequent breeding efficiency.

Calf birth weight has consistently been shown to be the most important factor associated with dystocia.<sup>22</sup> Birth weight has moderate to high heritability, and the sire effects on dystocia and perinatal mortality are quite variable. This means dystocia and mortality rates can be reduced by selecting bulls whose progeny cause few difficult births.<sup>20 27</sup> This discussion will not dwell further on causes or prevention of

dystocia but the effects of dystocia on neonatal life.

The bovine fetal genotype has recently been shown to have marked effects on the fetal and maternal endocrine systems. This 'control' of the hormone levels influences parturition, milk production, and postpartum fertility. One report showed the effect of progeny from five sites on maternal urinary estrogens and stillbirths<sup>17</sup> (Table 5).

TABLE 5. Sire Effects on Maternal Hormone Relationships to Dystocia and Perinatal Deaths.

Sire No.	Maternal E2 (ub)	Stillbirths in Heifers
	1150*	3.0%
2	1543	5.5%
3	1297	9.1%
4	1098*	20.1%
5	1057*	33.0%

\* — < 0.05

With the exception of sire No. 1 there was a significant relationship between low maternal estrogens and higher dystocia and stillbirth rates. Such a relationship is possible since fetal cortisol initiates the events leading to a normal parturition. This includes elevated maternal estrogens prior to parturition.

O'Brien and Stott showed a difference between prepartum maternal hormone levels in eutocia and dystocia parturitions<sup>15</sup> (Table 6).

TABLE 6. Prepartum Maternal Hormones Related to Dystocia.

	Eutocia	Dystocia
Cortisol	Higher	Lower
Estradiol 17	Higher	Lower*
	(15-20 days prepartum)	
Progesterone	Lower	Higher*

\* — P < 0.01

Other works by Stott also showed that dystocia calves consistently had lower cortisol levels at birth than eutocia calves<sup>25</sup> (Table 7). The question is: Were the cortisol levels low because of the dystocia, or did the dystocia occur because of the low cortisol levels? Birth weight did not seem to vary between the groups. This allows one to theorize that fetal endocrine action (low cortisol) may not have initiated the normal chain of events of increased estrogens, prostaglandin F<sub>2α</sub> production, decreased progesterone, release of oxytocin, and perhaps relaxin with cervical dilation and parturition. Future research may answer some of these questions.

Dystocia calves clinically appear to be slower at standing and nursing and more susceptible to infectious diseases. However, Stott found no difference in immunoglobulin levels when the calves were fed pooled colostrum four hours postpartum. The dystocia calves were sluggish for the first

TABLE 7. Cortisol and IgG, IgM, IgA Serum Levels in Dystocia Calves.

	Hours Postpartum	Dystocia	Eutocia
Cortisol	0	162 mg/ml*	211 mg/ml
IgM	24	2.4 mg/ml	2.1 mg/ml
IgG	24	12.3 mg/ml	12.2 mg/ml
IgA	24	1.9 mg/ml	1.8 mg/ml

\* — P < 0.01

24 hours, and had they not been force fed perhaps there would have been a difference in circulating antibodies at 24 hours.

Recent studies reported by Doornbos in Montana and Putnam in Oklahoma, indicate that the length of Stage II of normal parturition is shorter than classically described in textbooks. Length of Stage II is reported to range from 0.5-4 hours with averages from 1-3 hours depending on the age of the dam. Most texts do not recommend intervention until Stage II has been in progress 2-3 hours without completion.

Both studies defined Stage II as clinically beginning with abdominal press and appearance of fetal membranes (water bag) at the vulva. In Montana the average length of Stage II in unassisted births of first-calf-heifers was approximately 1 hour and in adult cows approximately 0.5 hour.<sup>5</sup>

Putnam reported similar results with first-calf heifers. In this study, heifers that did not deliver after two hours of Stage II labor were to be assisted. Heifers that calved in less than two hours after the appearance of membranes did so in 55 minutes. Heifers that calved in more than two hours did so in 162 minutes<sup>21</sup> (Table 8). Of the 15 heifers that required more than two hours of labor, only four calved unassisted and these were obviously going to calve unassisted at the end of the two hour period.

TABLE 8. Length of Stage II For Heifers Calving Less Than 2 Hours and Those Calving Greater Than or Equal to 2 Hours.

	< 2 Hours (Mins)	> 2 Hours (Mins)
No. Heifers	— 31	15
Average Length	— 55.48	162.33

P < .001

Pelvic measurements were also taken at parturition. While there was no difference in dystocia rates between heifers with below average pelvic areas and those with above average areas, there was a significant difference in the length of Stage II. There was also a difference in the number of calves that needed assistance in nursing, dystocia or not.

Immunoglobulin levels were not determined and there were no neonatal diseases, but what might have happened had there been a higher dystocia rate or if the sluggish calves had not been assisted after two hours of not nursing?

Another question: If eutocia are apparently completed with 30-60 minutes why recommend waiting 2-3 hours after

TABLE 9. Pelvic Area and Length of Stage II.

	Large Pelvic Area	Small Pelvic Area
Number Heifers	23	23
Length (Minutes)	68	113
No. of calves not nursing in 2 hour postpartum	3	7

labor has started to intervene and assist the birth? Doornbos found that intervention upon appearance of fetal membranes did no harm to cow or calf. Also subsequent cycling rates and pregnancy rates were significantly higher in early assisted heifers.

Obviously more work needs to be done with these procedures but from these data it would appear that earlier assistance could improve fertility of the dam and viability of the calf. These observations may warrant recommending intervention within one hour after the appearance of membranes at the vulva, **provided** the cervix is completely dilated.<sup>21</sup>

### Colostrum Management

Recent studies have shown that force feeding one gallon of colostrum (10% of body weight) within six hours of birth reduced the incidence of colibacillosis. The object of forced feeding is to increase passive immunity on a herd basis. This may be practical in dairies but not in beef cattle. The rancher must observe his herd closely at calving for problems other than dystocia.

1. He must see that the calves are 'mothered-up'. Studies have shown that when the dam licks and cares for the newborn calf, immunoglobulin absorption is significantly increased when colostrum is consumed.<sup>25</sup> Calves separated from the dams at birth had reduced antibody levels even when fed colostrum at the same time as the 'mothered-up' calves. Ranchers and veterinarians have observed that the neglected calf is less vigorous and always needs more attention to 'get going'.
2. He must observe the cow and calf to assure that nursing has occurred within six hours of birth. If the calf has not nursed, it must be assisted or forced fed 2-4 quarts of **FIRST MILKING COLOSTRUM**. Many beef cows do not give that much colostrum and there is no way of knowing how much colostrum a beef cow has or how much the calf nursed. Only problem calves can be supplemented, but losses will be reduced by feeding these calves.

Even though many beef calves may be hypogammaglobulinemic, neonatal diseases may be decreased by reduced exposure to organisms. This is achieved by having clean calving grounds and dispersion of pairs into cow-calf pastures. The management goal should be to maximize passive immunity and minimize exposure within the limits of the environment and type of cattle.

The value of hyperimmunizing pregnant cows with *Colostridium perfringens* Type C toxoid to provide calves with passive immunity to hemorrhagic enteritis has long been known. The effectiveness of similar techniques to prevent other enteric conditions is not well documented. Commercial and autogenous *Salmonella sp* bacterins have been used with mixed results. Cows experimentally vaccinated twice in the last 30 days of gestation with *E. coli* antigens produced calves that withstood oral challenge following colostrum feeding.<sup>14</sup> Seventy-seven percent of the calves from vaccinated cows were protected from diarrhea. All calves from control cows suffered severe diarrhea, severe dehydration, and hypothermia. Calves not treated with electrolytes died. Commercial *E. coli* bacterins now available for pregnant cows might be useful in improving the quality of Ig in colostrum. Regardless of the quality of Ig in the colostrum, passive immunity will not be acquired unless adequate volumes are consumed within six hours of birth.

### Evaluation of Passive Immunity

There are several diagnostic aids available for the evaluation of immunoglobulin levels of the newborn.

1. The use of a refractometer to measure serum protein is quick and serum protein levels below 5 mg% can mean that the calf is hypogammaglobulinemic. However, it is the least accurate method.
2. Single radial immunodiffusion and serum electrophoresis are both very accurate but require laboratory facilities and are relatively expensive.
3. The zinc sulfate turbidity test is easy and inexpensive and is accurate enough to be of prognostic value.
4. I prefer the sodium sulfite precipitation test because it is simple, inexpensive, and also accurate enough for a clinic prognosis. Immunoglobulins can be precipitated from serum with concentrations of Na<sub>2</sub>SO<sub>3</sub> ranging from 14 to 18%.<sup>19</sup> Lower concentrations fail to precipitate any protein, and concentrations above 18% will precipitate non-Ig proteins. Concentrations of 14, 16, and 18% Na<sub>2</sub>SO<sub>3</sub> may be prepared using 14, 16, and 18 gm of anhydrous Na<sub>2</sub>SO<sub>3</sub> in a total volume of 100 ml distilled water. Serum (0.1 ml) is added to 1.9 ml of each Na<sub>2</sub>SO<sub>3</sub> solution. Mix samples and allow to stand for 30 minutes at room temperature. Observe samples for precipitation. Precipitation may vary but score positive for any flaking or precipitate. An estimation of serum Ig levels are shown in Table 10. This test does not differentiate between immunoglobulins but is over 90% accurate for total immunoglobulins. Calves with serum Ig levels above 50 mg/ml will precipitate all 3 concentrations and may be considered to have adequate Ig levels. Calves with levels below 50 mg/ml are considered hypogammaglobulinemic.

This is a simple prognostic test which can be run in the field to determine efficiency of colostrum intake and to plan course of therapy for affected calves.

TABLE 10. Estimation of Serum Ig Content From Sodium-Sulfite Precipitation.

Concentration of Ig (mg/ml)	Na <sub>2</sub> SO <sub>3</sub> Concentration		
	14%	16%	18%
< 5	—	—	+
5—15	—	+	+
>15	+	+	+

The administration of whole blood to hypogammaglobulinemic calves may be helpful. Whole blood is suggested because of the difficulty of separating bovine plasma from citrated blood. One to two liters of whole blood can be administered subcutaneously or intraperitoneally with no adverse effect on the calf. Blood can also be administered intravenously but should not exceed 500 to 1000 ml. Intravenous blood is also more time consuming and if the IV use is not necessary the other routes are just as satisfactory.

### Neonatal Nutrition

**The nursing calf that is receiving enough milk needs no interference by man because it is getting what Mother Nature intended.**

A popular belief is that cows fed high energy feeds or on green grass give too much milk thus causing scours. Actually very few beef calves get too much milk. Calves nursing good milking cows not only gain better, but are less likely to contact infectious diseases. Calves that have marginal or deficient milk consumption are more susceptible to infectious diarrhea and pneumonia.<sup>3</sup> These calves are weak, emaciated, and more difficult to treat because they rapidly dehydrate.

Economically there is little that can be done for the beef calf that is receiving marginal amounts of milk. However, the calf that is being starved because its mother does not milk well, has mastitis, sore teats, etc. needs attention. Ranchers should be alert to notice these problems and supplement such calves or find foster mothers. When foster mothers are not available, starving calves can be raised on milk replacer and starter grain just as with dairy calves.

Whole milk is the most desirable food for all calves. Economically this can not be fed to orphan calves so milk replacers are used. Calves should be fed two quarts twice a day (approximately 8-10 percent of body weight). They should also receive free choice clean water. This will meet hydration requirement and if the milk replacer is of high quality the energy, protein, and mineral intake will be adequate. High quality milk replacers will have the following basic properties on a dry matter basis:

- 20-22 percent protein from milk source, with no plant proteins.
- 15-20 percent fat.
- less than 0.5% (preferably 0.3%) fiber.

Other additives such as vitamins and antibiotics will be present, but the real value (and cost) is determined by the

above three factors.

Calves less than 4-6 week of age cannot utilize plant proteins. Many inferior milk replacers contain plant protein to make the product less expensive. Unknowing farmers will often buy these products with resulting starvation of the calves. I have seen starving conditions reversed in approximately two weeks by simply changing milk replacers.

Calves should be offered a highly palatable calf starter. This should be offered free choice in a clean container that the calves cannot soil. After the calves are 6-8 weeks of age they should be consuming approximately 1½ to 2 pounds of starter and can be weaned from the milk replacer. Limitation of milk replacer to one gallon per day encourages feeding on the starter. The neonate bovine is basically monogastric but with this stimulation the rumen soon develops and the calf can be weaned at two months of age.

The fiber content of this feed should be less than 10 percent and the protein content should be 18 percent with no urea. The protein supplement would ideally be soybean meal. Avoid cottonseed meal for the calf starter. Processing of cottonseed meal releases gossypol which is toxic to the preruminant calf. Vitamins 'A' and 'D', Calcium, Phosphorus, and Trace Minerals should be added to balance the calf starter. A study at Oklahoma State University on weaning beef calves at 6-8 weeks of age used the following ration:<sup>13</sup>

Early Weaning Rations Used in 1980 (OSU)

Ingredient	Ration		
	Starter	Ration II	Ration III
	%	%	%
Roller Corn	64.0	56.5	50.1
Soybean meal	20.0	17.0	12.0
Cottonseed hulls	10.0	20.0	33.0
Cane molasses	5.0	5.0	3.0
Dicalcium phosphate	—	—	.5
Limestone	0.5	0.5	0.5
Potassium chloride		0.5	0.5
Salt	0.5	0.5	0.5
Vit A (30,000 IU gm)	1 lb/ton	1 lb/ton	½ lb/ton

Calves were fed the starter for two weeks, Ration II was fed for six more weeks and Ration III was fed until approximately seven months of age. The early weaned calves were 88 pounds heavier (435 lbs. vs 347 lbs.) at seven months when the nursing calves were weaned. While early weaning will not be a routine management practice in beef herds, it can be a feasible salvage practice and should not be overlooked.

**The futility of treating hypogammaglobulinemic calves is well documented. The frustration that owners and veterinarians have when faced with an outbreak of a severe perinatal or neonatal disease has been discussed in many poolrooms and professional meetings. These frustrations can be reduced through preventive medicine. The veterinarian should give timely instructions on preventive medicine**

## management to his clients.

The following procedures should increase perinatal and neonatal survival:

1. Have heifers big at calving
2. Breed heifers to sires selected for 'easy calving'.
3. Provide proper prepartum nutrition.
4. Hyperimmunize dams when indicated.
5. Provide adequate and clean calving facilities.
6. Provide proper surveillance and early assistance at calving.
7. Insure adequate and timely colostrum consumption.

## Bibliography

1. Bull, R.C., Loucks, R.R., Edmiston, F.L., Hawkins, J.N., and Stauber, E.H.: Nutrition and Weak Calf Syndrome in Beef Cattle. Current Info. Series No. 246. U. of Idaho, Coll. of Ag., Sept. (1974). 2. Conner, G.H., Richardson, M., Carter, G.R., and Wamukoya, J.P.O.: Immune Responses of the Bovine Fetus. *J. Dairy Sci.*, 60:289, (1977). 3. Corah, L.R., Dunn, T.G., and Kaltenbach, C.C.: Influence of Prepartum Nutrition on Reproductive Performance of Beef Females and Their Progeny. *J. An. Sci.* 41:819, (1975). 4. Cutlip, R.C., and McClurkin, A.W.: Lesions and Pathogenesis of Diseases in Young Calves Experimentally Induced by Bovine Adenovirus Type 5 Isolated from a Calf with Weak Calf Syndrome. *Am. J. Vet. Res.* 36:1095, (1975). 5. Doornbos, D.E., Bellows, R.A., and Burfening, P.J.: Effects of Obstetrical Assistance on Postpartum Reproduction in Beef Females (In Press). 6. Frey, M.: Personal Communication, (1983). 7. Hamilton, G.F., Turner, A.S., Ferguson, J.G., and Pharr, J.W.: Slipped Capital Femoral Epiphysis in Calves. *J.A.V.M.A.*, 172:1318, (1978). 8. Haughey, K.G.: Meningeal Haemorrhage and Congestion Associated with the Perinatal Mortality of Beef Calves. *Aust. Vet. J.*, 51:22, (1975). 9. Hoerlein, A.B., and Jones, D.L.: Bovine Immunoglobulins Following Induced Parturition. *J.A.V.M.A.* 170:325, (1977). 10. Johnston, N.E., and Oxender, W.D.: Effect of Altered Serum Glucorticoid Concentrations on the Ability of the Newborn Calf to Absorb Colostral Immunoglobulin. *Am. J. Vet. Res.* 40:32, (1979). 11. Logan, E.F.: The Influence of Husbandry on Colostrum

Yield and Immunoglobulin Concentration in Beef Cows, *Br. Vet. J.* 133:120, (1977). 12. Logan, E.F., McMurray, D.H., O'Neill, D.G., McParland, P.J., and McRory F.J.: Absorption of Colostral Immunoglobulins by the Neonatal Calf. *Br. Vet. J.* 134:258, (1978). 13. Lusby, K.S., and Parra, A.A.: Effect of Early Weaning on Calf Performance and on Reproduction in Mature Cows. *An. Sci. Res. Rep. MP-108, Ok. St. U.* p 64, (1981). 14. Myers, L.L., Newman, F.S., Wilson, R.A., and Catlin, J.E.: Passive Immunization of Calves Against Experimentally Induced Enteric Colibacillosis by Vaccination of Dams. *Am. J. Vet. Res.* 34:29, (1973). 15. O'Brien, T., and Stott, G.H.: Prepartum Serum Hormone Concentrations Related to Dystocia in Holstein Heifers. *J. Dairy Sci.* 60:249, (1977). 16. Osburn, B.I., Stabenfeldt, G.H., Adams, A.A., Trees, C., and Sawyer, M.: Perinatal Immunity in Calves. *J.A.V.M.A.* 164:295, (1974). 17. Osinga, A.: Endocrine Aspects of Bovine Dystocia with Special Reference to Estrogens. *Therio.* 10:149, (1978). 18. Patterson, D.J., Bellows, R.A., Burfening, P.J., Short, R.E., and Carr, J.B.: Incidence and Causes of Neonatal and Postnatal Mortality in Range Cattle. {Abst. *J. An. Sci.*, 1979, Vol. 49 (Suppl. 1) p 325} *Proc. 71st Annual Mt. Am. Soc. An. Sci.* 19. Pfeiffer, H.E., and McGuire, T.C.: A Sodium-Sulfite Precipitation Test for Assessment of Colostral Immunoglobulin Transfer to Calves, *J.A.V.M.A.* 170:809, (1977). 20. Philipsson, J.: Studies on Calving Difficulty, Stillbirth, and Associated factors in Swedish Cattle Breeds: III Genetic Parameters. *Acta Ag. Scand.* 26:211, (1976). 21. Putnam, M.R., Rice, L.E., Wettemann, R.P., Lusby, K.S.: Planipart (Clenbuterol) for the Postponement of Parturition and Alleviation of Dystocia in Cattle. *Proc. Soc. Therio.* (1982). 22. Price, T.D., and Wiltbank, J.N.: Predicting Dystocia in Heifers. *Therio.* 9:221, (1978). 23. Stauber, E.H.: Weak Calf Syndrome: A Continuing Enigma. *J.A.V.M.A.* 165:223, (1976). 24. Stormont, C.: Neonatal Isoerythrolysis in Domestic Animals: A Comparative Review. *Cornell Vet.* 25. Stott, G.H.: Immunoglobulin Absorption in Calf Neonates with Special Consideration of Stress. *J. Dairy Sci.* (In Press). 26. Tryphonas, L., Hamilton, G.F., and Rhodes, C.S.: Perinatal Femoral Nerve Degeneration and Neurogenic Atrophy of Quadriceps Femoris Muscle in Calves. *J.A.V.M.A.* 164:801, (1974). 27. Van Dielen, S.W.J.: Calf Mortality at Birth. *Veetelt. en Zwivel Berichten.* 7:20-29. In *An. Brd. Abstr.* 33:1166, (1964). 28. Waldham, D.G., Hall, R.F., Meinershagen, W.A., Card, C.S., and Frank, F.W.: Haemophilus Somnus Infection in the Cow as a Possible Contributing Factor to Weak Calf Syndrome: Isolation and Animal Inoculation Studies. *Am. J. Vet. Res.* 35:1401, (1974). 29. Young, J.S., and Balir, J.M.: Perinatal Calf Losses in a Beef Herd. *Aust. Vet. J.* 50:338, (1974).