# **Beef Calf Deficiencies in Practice**

## J. K. WARD, D.V.M. Hamilton, Montana

One of the most serious and economically important deficiencies of beef calves in my practice is selenium deficiency. The area is in a mountain valley in Western Montana. Soils vary greatly within a quarter to half mile.

When I first entered practice in 1958, vitamin E was used widely in capsule form in the prevention of white muscle disease with a fair degree of success, but it failed to control the disease as effectively as desired. During the past 25 years, the deficiency has increased in severity, occurring on ranches that previously were not troubled with the problem. With the advent of commercial fertilizers, primarily phosphates and sulfates, it has been shown that these prohibit the forage from utilizing the element selenium in an already borderline deficient soil. I do not recommend the cessation of using commercial fertilizers because of benefits received in the quantity and quality of feeds.

White muscle disease usually occurs as the cardiac form in calves between 10 days and two months of age. The skeletal form is much less common. Lambs usually exhibit the skeletal form at two weeks of age or older.

Two cases have been diagnosed in foals on one ranch where the problem was very severe in sheep and cattle. The foals showed somewhat different clinical symptoms.

#### Clinical Signs

A few calves are seen alive with the cardiac form, but usually there is too much cardiac damage and the animal succumbs in a few hours. Most cases are diagnosed on postmortem with a history of acute death. Often the owner states that the calf was playing or running and fell over dead. Here the history may be confused with *Cl. perfringens* Type C or a perforating ulcer of the abomasum because of the acute death.

The skeletal form is somewhat different, the calf having a normal appetite with a stilted, stiff-legged walk and lying down often. This will progress over a period of a few days, the animal becoming more severely affected, being unable to rise on its own, but is able to stand with assistance. Appetite and temperature remain normal. The front legs are usually affected to a greater degree than the rear legs, letting the dorsal body of the scapula protrude above the thoracic spine to a height of two or three inches. Without treatment, the calf will be unable to stand even with assistance. Seldom do we see the cardiac and skeletal form in the same animal.

#### Postmortem Findings

Gross lesions vary according to the severity of the problems. The skeletal area shows a few white streaks in muscle fibers to complete muscle groups being affected in the front and hind quarters.

The lesions of the cardiac muscle appear streaked or parboiled in appearance. On cross section as much as one-third to one-half of the cardiac muscle may be involved.

#### Treatment

The skeletal form of white muscle disease responds very well to an injection of five mg. sodium selenite and 4.4 mg vitamin E repeated in two to four days. Treatment of the cardiac form, when diagnosed clinically, is a case of being too little and too late.

### Prevention

In the early sixties we used selenium injections on 15 to 20 thousand calves in clinical field trials and found it very effective. We have carried on this practice since using the commercial products, Bo-Se(R) and Mu-Se(R). Some practitioners make their own, but you may have a toxicity problem if improperly prepared. The dosage we recommend is 2.5 mg. sodium selenite and 2.19 mg. vitamin E (Bo-Se\*) i.m. at birth. Several ranches have had to double this dosage recently in order to control white muscle disease.

Only a few ranchers use the product on pregnant cows because of the cost. We have found Mu-Se very helpful in beef and dairy herds to eliminate retained placentas and fertility problems after ruling out other common infections and nutritional problems.

### Iodine

Iodine deficiency has been known to exist for years. It shows itself in a few isolated cases. These are usually in small herds of two or three cows, but periodically a new rancher forgets to use iodized salt. The calves usually are born weak and die shortly after birth maybe taking a few breaths but not aerating the lungs completely. The region over the thyroid is swollen in varying degrees, sometimes extending to the thoracic inlet. Calves that live are treated with an organic iodine, 1 tsp., orally for five days with gratifying results.

\*BoSe-Burns Pharmaceuticals \*MuSe-Burns Pharmaceuticals

### Postmortem Findings

The major lesions are diffuse subcutaneous edema in the laryngeal area; the thyroid is greatly enlarged and edematous, approximately the size of a goose egg. Prevention is easily obtained by feeding iodized salt with 0.007 percent iodine.

#### **Crooked Calf Disease - Lupine Toxicity**

"Crooked calf disease" possibly should not be included in the deficiency category but I think it should be mentioned. At least three causes are mentioned which should be considered in making a diagnosis. They are: lupine toxicity, manganese deficiency, and a genetically linked problem. There are possibly other causes. The calves are born with mild contracture of the flexor tendons of the forelimbs to very severe and varying degrees of distortion affecting all limbs and the body. These cases may exhibit arthrogryposis, kyphoscoliosis and torticollis. One or all three may be present and/or cleft palate. Many calves are unable to rise because of the severe involvement and have to be destroyed. They also may cause dystocia, leading to partial embryotomies, or caesarean section. Two ranches involved several years ago suffered a 20% loss from this problem.

On postmortem examination, there is contracture of the ligaments and tendons of the extremeties and/or the body. No joint involvement is apparent except they may be offset laterally.

In lupine toxicity the major problems apparently occur between the 60th to 90th day of gestation from ingesting the lupine plants, *Lupinus sericeus* or *L. caudatur*.

Prevention is strictly a management problem, by not grazing land where lupine is part of the natural growth or removal, if possible, from access to this plant during the 40th to 70th days of gestation. Mild forms of malformation may occur outside these stages of gestation. One rancher who had a considerable problem had very good success in controlling the plant on grazing land by using 2-4D weed spray applied by plane.

Genetic linked crooked calves consistently show similar abnormalities, usually a cleft palate and mild contracture and curvature of the forelimbs. Unless a registered herd traces pedigrees and matings of recessive parents, the diagnosis is much more difficult and should be approached with caution.

#### Thiamine

Periodically during the past several years we have been presented dairy calves between two and four months of age that show an incoordinated gait, stiffness of the neck, and on examination, have impaired vision, and a normal temperature. If left untreated for 24 hours they will eventually become recumbent, show opisthotonus, nystagmus, and blindness, with mild, intermittent convulsions. These calves have responded to B-complex and tetracycline, 1 gm. i.v., in 12 to 24 hours. The treatment is repeated in 24 hours and the animals are sent home normal in all clinical aspects.

During the past few years, individual beef calves weighing 300 to 400 lbs. in the fall have exhibited the same clinical syndrome with no other diagnostic signs. These beef calves have responded well to the same therapy as the dairy calves. Because of the clinical signs of CNS disturbance and response to treatment, it is suspected that these animals are exhibiting a thiamine deficiency.

### Energy (TDN Deficiency)

A deficiency often overlooked by veterinarians and stockmen alike is "energy" supplied to the dam during the last trimester of gestation.

The newborn calf deficient in energy at birth may be too weak to nurse, making it much more susceptible to the natural elements and diseases than a vigorous healthy animal.

The energy needs of the dam varies drastically with the change in temperature, shelter provided, and the temperature of her drinking water, etc.

### **Electrolytes and Fluids**

Whether electrolytes and fluids should be included as a deficiency for beef calves is questionable, but in our practice they are responsible for the greatest economic and death loss of beef calves. Of course, this is the result of the complicated scour complex seen in the beef industry today. Control and prevention is very trying and exasperating to the livestock owner and practitioner alike because of the complex nature of the disease. Factors involved are nutrition, concentration of animals, climatic conditions and infective agents. Some are controllable and others are not.

Enough has probably already been said about the subject the past day or two, but I'd like to say a few words concerning treatment after all attempts at prevention have failed.

For a number of years we have been guilty, as have the owners of pharmaceutical firms, of using antibiotics and numerous and sundry scour preparations as a crutch to alleviate a major problem and at times, through bacterial resistance, creating a much bigger one than the dairyman and mastitis.

Our treatment is aimed entirely at replacement of fluids and electrolytes supplemented with antibiotics, intestinal protectives, and any other drug or product that we think will be of benefit.

We start not with a liter, but with liters, usually four, of lactated Ringer's solution or our own mixture, consisting of Eltra Add L.A. (Concentrate) 125 cc and 30 gms. of NaHC O<sub>3</sub> (baking soda) and q.s. to 4000 ccs of tap water given i.v. with an indwelling catheter. For home treatment of calves that are not down or comatosed, the owner is instructed to give a similar solution orally in smaller amount (one-half to one pint) as often as possible until rehydration is established. We look for something practical and economical in time and labor. Other deficiencies that may be considered in newborn calves are gammaglobulins, colostral antibodies and natural steroids and homones and milk. I present these only as food for thought.

In summary, let me say deficiencies of beef calves are wide and varied. My views are those of a practitioner, from clinical observation, experience, experimentation, consultation, laboratory procedures, necropsies, trial and error and thirteen years of many, many frustrations!

#### References

Brophy, R. J. Personal communication and consultation. – Dyer, I. A. Deformed Calves: A Nutritional Problem, Western Veterinarian (1963) 31. – Muth, O. H., Oldfield, J. E., and Weswig, P. H. (Eds.): Symposium; Selenium in Medicine. The AVI Co., Westport, Conn., 1967. – Muth, O. H., Schubert, J. R., and Oldfield, J. E. White Muscle Disease (Myopathy) in Lambs and Calves, Etiology and Prophylaxis. Am. J. Vet. Res.: 22, (1961) 466. – Palotay: Crooked Calves. Western Veterinarian (1958) 16. – Shupe, James L. Personal communication and consultation.