do not reproduce the distinctive features of the natural disease. The experimental disease, like the chlamydial infection in sheep, enzootic abortion of ewe (E.A.E.), produces placentitis and usually only minor nonspecific fetal changes.

Cows experimentally inoculated with chlamydial agents respond with increase in CF titers and more specifically the experimentally aborted fetuses also has specific CF fixing antibody. This contrasts with the naturally occurring EBA abortions.

It seems likely from this evidence that the disease recognized as EBA in California is not caused by chlamydial organisms. If chlamydia produce a specific, naturally-occurring disease in cattle resulting in abortion, it has not been characterized or proved.

EBA is reported to occur outside of California. I am not sure if it does or not but I would be very interested to hear of any outbreak and would appreciate the opportunity to study tissues.

I am not sure I have shed much light on the "unexplained" abortion. I hope I have explained why the explanations are hard to come by and why, in spite of the fact that our last study appeared to move us back 10 years. I think more investigation is needed.

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Weak Calf Syndrome

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Weak Calf Syndrome is a specific disease of the bovine species characterized by abortions, stillbirths, neo-natal death and weak calves with the impairment of body functions of some animals visible throughout their natural life.

History

Since 1963 this disease entity has been recognized in beef and dairy herds in this author's practice area, causing tremendous economic losses to individual ranchers and in certain years (1969) reaching epidemic proportions. Since 1969 the disease entity has been recognized by other veterinarians and ranchers in neighboring areas and states (Chart IV).

Due to failure to make a diagnosis of any known diseases, it has been tagged E.B.A. (variant), Bitter Root Crud, "Ward's Disease" and Polyarthritis.

Herd History

A herd that is infected endemically will be plagued by a few scattered abortions during the last trimester of pregnancy involving approximately 1.5 to 2% of the total cow herd. .

The second observation is a 6-8% death loss of calves shortly after birth, plus the same number of calves (6-8%) showing some form of weakness, diarrhea, stiffness and failure to do well as will be described.

The disease can be very sporadic or a very constant problem in a given herd during calving. Calf losses may be heavy at the beginning of calving, then stop; or, it may not become evident until one third or one half of the cows have calved. These factors depend on introduction of replacement animals, concentration, trucking, trailing and climatic conditions during the last 60 days of pregnancy.

The losses due to this syndrome will vary greatly depending on the above mentioned factors and natural immunity received from previous years of exposure.

Etiology

It is my belief that this disease is caused by a

heretofore unrecognized agent, and not any of the recognized agents for the following reasons:

1. Negative serology of the dams for Brucella and Leptospirosis (10 serotypes).
2. Negative bacterial culture from aborted fetuses.
3. Proper immunization of dams, lack of clinical evidence, and negative sera of dams and negative precolostral sera of calves for IBR and BVD (9).
4. Analysis of feeds for protein, minerals, vitamins and trace elements and lack of toxic or subtoxic levels of nitrates.
   a. Parenteral administration of selenium to dams and newborn calves (1).
5. Lack of any positive results during feed trials with broad spectrum antibiotics at levels of 1 gm/per/head/day or higher, starting 60 days prior to calving to control the disease problem.
6. Negative serology for chlamydia from dams and failure in repeated attempts to isolate the organism (9).
7. Identification of two mycoplasma isolates from an affected calf and a placenta but failure to make other isolation in eggs or culture media (7).
8. Blood counts of affected calves at birth showing varying degrees of leukopenia and some showing anemia, suggesting a viral agent (Graph I).
9. The disease syndrome occurring in dairy and beef herds that are not exposed to pine needles or other toxic material.
10. Herd records indicate a very high percentage of the dams develop immunity after abortion or giving birth to a typical affected calf, suggesting an infectious agent.

Since January 1970, attempts at viral isolation on tissue culture have been attempted from affected calves. Those isolated to date have been a Reo virus, PI_3 virus and BVD virus but only in calves four days old or older (9).

Isolation of a viral agent from the thymus, salivary gland and kidney of fetuses, affected newborn, and aged calves showing clinical evidence of the disease and adult cows at random has been accomplished since 1971 (11).

The agent is 80-100 milli-microns in diameter, producing eosinophilic, intranuclear inclusion bodies in tissue culture.

The physical and chemical properties suggest that the virus probably belongs to the herpesvirus group. The nucleic acid content appears to be DNA, and the agent is ether and acid sensitive, but heat resistant. No complement fixation antigen has been detected to date. It is unable to agglutinate red cells (4).

Probably of great importance is the virus' incognetic properties or its ability to transform cells *in vitro* (3).

A similar agent has been isolated from sheep exhibiting a very similar syndrome (4,8).

Clinical Symptoms

A few cows have been observed upon examination to have temperatures of 103° to 106°, with anorexia. Examination of the respiratory system shows a mild asthmatic type breathing with moist rales. Some cows will respond to tetracycline therapy but will abort or give birth to a weak calf in approximately 10 days to two weeks. Dams may show a leukopenia.

Some affected dams, especially heifers, expire with symptoms of meningoencephalitis after exhibiting the above symptoms.

Prior to and during calving we are presented with a number of emphysematous obstetrical cases. These fetuses and the uterine discharge will have a very characteristic and disagreeable odor, not at all like the usual emphysematous fetus.

An apparent uterine inertia is noted in certain cows. Cows weighing 1100 to 1200 pounds, 6-10 years old, have been noticed to begin labor, but after four to six hours have made no progress in delivery. Upon examination a considerable amount of membrane is found to have been expelled, with the fetus in normal presentation. After stimulating labor, very little assistance is necessary to deliver the fetus.

Retained membranes may or may not be a problem. Certain membranes are very edematous and large amounts of fluid can be expressed from the placenta. Periodically, certain sections of the placental membranes will show a very dry leathery appearance.

Necrotic areas of a brownish color may be observed in certain cotyledons. Metritis with large amounts of dark reddish fluid is a common sequal with very slow involution of the uterus. The affected cow becomes very thin and gaunt. Recovery takes from two to three months with no milk production.

Other cows have been noticed to give birth to calves and rise and walk away, paying no attention to the calf. Many times these are weak calves that die in a short time or take hours to obtain their feet.

Many calves will expire a short time after delivery when mild forced traction is used, especially with first-calf heifers. These calves, and

also calves delivered from older cows, apparently cannot withstand much stress from a difficult delivery. These calves may or may not show gross pathology.

Gross Pathology of Fetuses

The closer to term the less gross pathology seen. The most characteristic lesions are hemorrhage and edema of the subcutaneous tissues, especially the extremities.

Aborted fetuses are usually aborted from seven months pregnancy to term. These fetuses will appear to be full of fluid inside and out. Upon palpation they are very edemotous and jellylike. Subcutaneous edema will be present over the extremities, larynx and neck region along the rib cage, sternum and in the fascial planes of the heavy muscles of the hind limbs and forelimbs.

The pleural and peritoneal cavity are usually filled with a port wine-colored watery fluid. The liver may suggest a granular appearance.

Edema of the diaphragm near attachment to the sternum is common, with the pericardium affected at times. When the fetus is opened and left in one place, the fetus will exude fluids for long periods of time.

Gross Pathology of Calves — Term and Older

The most constant gross lesions are diffuse hemorrhages and edema, but to a lesser degree. The skin of the calf must be reflected from most of the entire body, especially all four legs down to the coronary band. **Hemorrhages and/or edema of the extremities are the most constant finding.** In older calves (two-six weeks) the hemorrhages tend to be organized.

These lesions will be seen on the lateral surface of the tarsus and tendon of the gastrocnemius, proceeding down over the metatarsus to the fetlock joint and coronary band. Some subcutaneous edema may be seen within the fascial planes of the semimembranosus and semitendinosus muscles. (Do not confuse this with I.M. injections of medications prior to death.) The same lesions may be found over the carpus, the metacarpus, and proceeding down the metacarpus to the coronary band. The above lesions are usually bilateral and should not be confused with injury from the calf being down for long periods of time. Some hemorrhages may be found over the lateral stifle area, in the flank area, along the rib cage and over the elbow joint. Subcutaneous edema and/or hemorrhage may be found in the muscles of the neck over the laryngeal area and masseter muscles.

Petechial hemorrhage of the third eyelid, sclera and conjunctiva are often present. The muzzle and lips usually show a bright orangish-red erythema. (Difficult to note in black pigmented calves.) Hemorrhage of the undersurface of the tongue may be found. The esophagus and trachea show petechial hemorrhages at times. Very small petechial hemorrhage are usually present in the thymus. The supra scapular and prefemoral lymph nodes may be enlarged and edematous.

Edema of the gall bladder, bile duct and attachment of the duodenum are very constant findings. The peritoneal cavity may contain varying amounts of straw-colored fluid. The spleen may show "paint brush" hemorrhages. There may be edema of the kidney fat and edema in certain areas of the mesentery. A very common finding is a mild to severe gastroenteritis with enlarged mesenteric nodes in calves that have lived only a minute or two. Petechial hemorrhages are often found in the abomasum. Intestinal cultures may yield *E. Coli.* In theory this accounts for the diarrhea observed in calves a few hours old up to several days. It is felt by this author that the *E. Coli* is secondary to an original infection of the fetus *in utero.* Calves two to four days old may exhibit small hemorrhagic ulcers of the abomasum which is believed to lead to chronic or perforating ulcers a month or two later.

The pleural cavity may contain small amounts of straw-colored fluid. There may be excessive pericardial fluid in the pericardeal sac. Epicardial hemorrhages may be very striking to nil. The lungs usually show very little pathology, but periodically will show some hemorrhage and edema. Some calves show strong evidence of a chronic heart failure. The musculature of the diaphragm may be edematous.

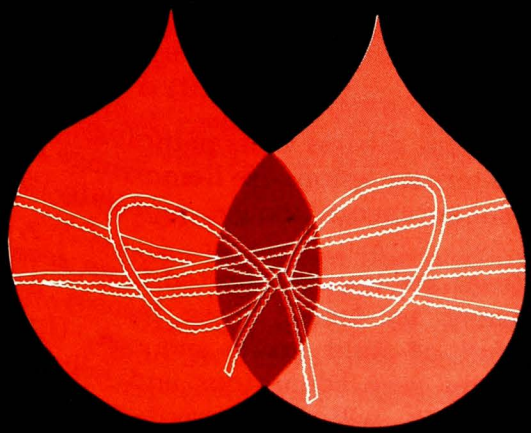
Some calves four days of age and older reveal an involution of thymus as compared to calves the same age dying from other causes. This probably is the most important finding concerning calves that live and are "poor doers" or "runts." The involution of the thymus is thought to cause a lack of the T-Rymphocyte system.

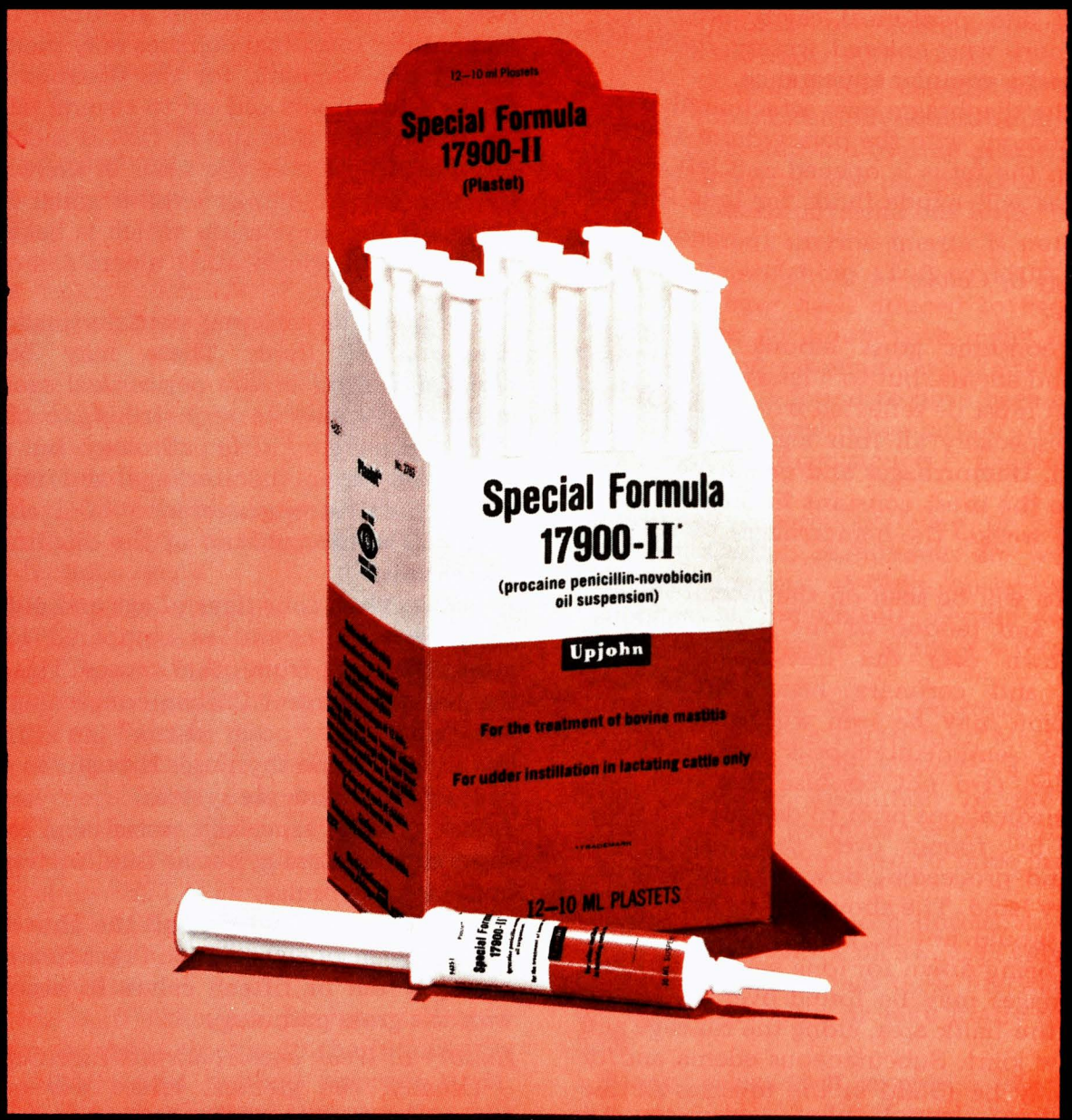
The joints, especially tarsal and carpal, may show a red tinged synovial fluid or fibrin deposits in the joint capsules.

Do not expect to find all the above mentioned pathology in one specimen. It is often necessary to necropsy ten or fifteen calves to become familiar with the gross pathology.

History of Weak and Abnormal Live Calves

Usually, the greatest losses will occur at the beginning of the calving period and taper off as calving proceeds.

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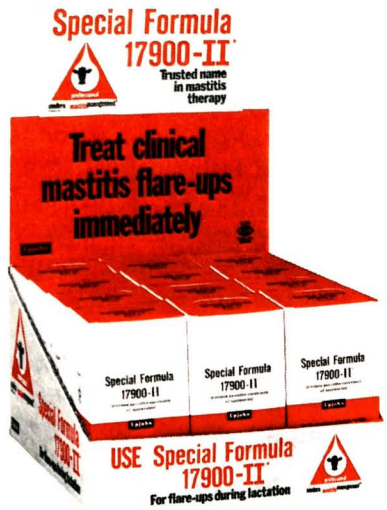

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Some calves may expire within minutes after normal delivery, others will be very weak and are unable to rise unless taken into a warm shelter and given mild stimulants, electrolytes and dextrose, or stimulated by some other method. It may take up to 12 to 24 hours before these calves are able to stand. Many of these calves may show a bright reddish-orange erythema of the muzzle which persists and the muzzle becomes encrusted. The sclera may show large diffuse hemorrhages. The third eyelids may show petechial hemorrhages.

At times the edema of the extremities can be palpated externally. This disturbance of circulation makes the extremities very susceptible to freezing at even mild freezing temperatures (25-32°F). Parts of the extremities may slough at four to six weeks of age even when the temperatures are above freezing.

Calves several days old will be noted walking stiffly and excessive heat and swelling may be palpated over the cannon bones and fetlock joints.

Some will have a diarrhea at birth or within a few hours.

Other calves may be very depressed, have temperatures of 103-104°, have a drawn appearance and exhibit pain on palpation of the abdomen, suggestive of a peritonitis. The respiratory movements may suggest a pneumonia. These calves will expire approximately the third day of life but exhibit very little internal gross pathology.

Certain calves (dummies) will not nurse unless assisted by the owner. It may take several lessons to teach the newborn calf to nurse.

Most of these calves are very susceptible to secondary bacterial enteritis. Many will die as a result of dehydration and shock no matter what type of medicinal care is used.

On necropsy there is evidence of organized hemorrhages of the subcutaneous tissue of the extremities. Calves presented for necropsy examination for baby calf diarrhea from one day up to six weeks of age may show certain and varied degrees of gross pathology mentioned previously. Some will be thin, dehydrated and appear to be starving. The muzzle will be encrusted and peeling, the mesenteric lymph nodes may be enlarged and edematous. The hair coat is long, ragged, and the animal walks with the appearance of weakness, stilted gait and slight incoordination.

Blood Studies

In the spring of 1969 a few blood studies were made on newborn calves, but only in a few cases because of time restrictions. But these were revealing.

In 1970 a project was instigated to establish a set of normal blood values and compare them with blood values from the clinically abnormal calves.

A senior high school student, Mr. Jim Kautz, accepted this as a high school science project with the assistance of R. J. Brophy, D.V.M., and Mrs. Jane Evans, medical lab technician.

Five cc of whole blood was collected in E.D.T.A. tubes and submitted to the laboratory immediately. W.B.C., differential, hematocrit, and platelet count was performed on each sample.

The results of the blood studies showed a mild to severe leukopenia at birth, shifting to a leucocytosis as the calf became older, and exhibited a mild drop in thrombocytes in clinically abnormal calves (Graph No. 1).

Immunological Aspects of the Disease

In the belief that the disease was an infection *in utero*, transfer of passive immunity from previously affected dams to affected calves was attempted.

Five hundred cc of whole blood was drawn from dams that had aborted or given birth to a typical weak calf at least 30 days prior to bleeding. The blood was intravenously administered to calves ranging in age from newborn in age from newborn to ten days. C.B.C.'s were taken from calves to evaluate the degree of clinical illness.

Clinical response was used as a guide to evaluate the response to blood transfusion. Approximately 70% of these animals survived. Some, perhaps 35%, would have survived without blood. Many survivors became "poor doers" or "runts." Immune serum has been used as a preventive at birth with varying degrees of clinical response similar to whole blood.

During the past several years this technique has been used as treatment for many calves exhibiting clinical signs of the weak calf syndrome in neighboring practices.

Some of these clinically affected calves have been observed for growth rate and used for immunological studies. An experiment was designed to test the immunological competence of affected animals.

In March of 1973, preliminary studies were begun on a group of 18 yearlings—10 normal heifers, seven abnormal heifers, and one abnormal bull. The group also included one abnormal two-year-old heifer. All were given a subcutaneous injection of five cc of Brucell Strain 19 vaccine. Three weeks later, the animals were bled, injected with 0.1 cc Brucella standard plate antigen intradermally in the caudal fold, and weighed. All animals reacted to the Brucella plate test with 1:40

Chart III

Conception Rates – Cows and Heifers
Natural Service

	Total cows tested	Preg.	Open	Preg. Culls	% Open	% Culls
1964	1570	1456	82	37	5.2	2.25
1965	1679	1522	62	95	5.7	5.8
1966	1731	*1542	95	94	5.5	5.5
1967	1734	1523	101	110	6.0	6.5
1968	1728	1562	90	76	5.2	4.4
**1969	1772	1561	95	116	5.4	6.5
1970	1750	1490	160	100	8.1	6.0
1971	1759	1601	103	55	5.9	3.1

*Pregnant heifers sold to University of Illinois
**40 cows were not tested.

Chart IV

Percentage of losses in Age Groups of Cows

Total abortions and calf losses for 1969 (Jan. 1 to Dec. 31, 1969) was 203 head.

From necropsies it was established 154 or 75% was due to this disease entity–with the same percentage of loss of the total attributed to the disease entity the past five years.

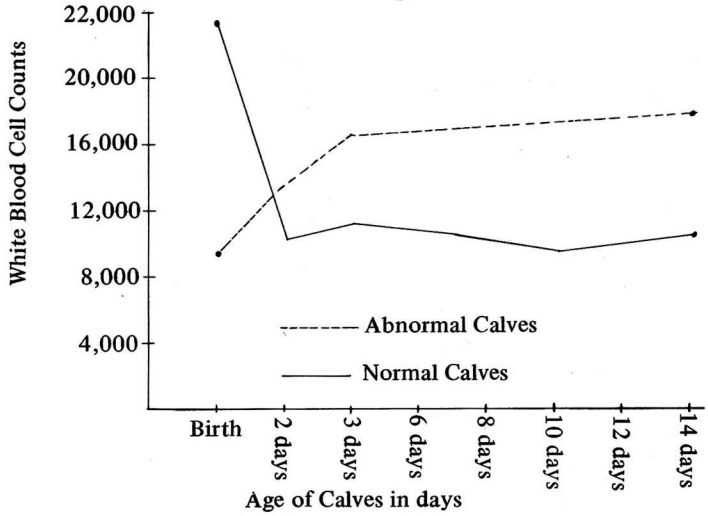
2 yrs.	3 yrs.	4 yrs.	5 yrs.	6 yrs.
17.4%	8.0%	10%	10%	11%
7 yrs.	**8 yrs.**	**9 yrs.**	**10 yrs.**	
10%	7.0%	5.0%	5.0%	

Economic losses 1969

Losses of 154 calves @ $150 = $23,100.

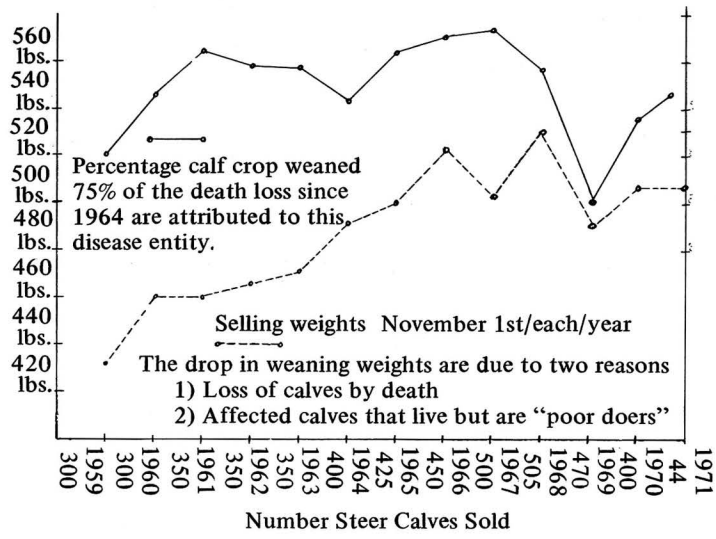
Drop of 30 lbs. in weaning weights on other calves
30 X 35 cents X 900 head = $32,450 on 1600 cows
$55,640 divided by 1600 head = $34.77 loss per cow
The figure $34.77 applied to brood cows in an area is a considerable figure.

Graph I



Spring – 1970
Normal & Abnormal Blood Counts

Graph II



Number Steer Calves Sold

to 1:160, with the exception of the two-year-old heifer which had been vaccinated 15 months previously with Brucella strain 19. The chronic or poor doing animals showed little or no reaction to the intradermal test while the normal animals showed very severe reactions to the intradermal test.

All animals were run together under the same conditions during the summer for breeding purposes.

The animals were injected intradermally with 0.1 cc Brucella standard plate antigen in August 1973. A poor response to the skin test was noted in all normal and abnormal animals. Inflammation and swelling was decidedly different from the first reading six months previously. All normal heifers were found to be pregnant at this time.

It has been noted in the treatment of Herpes viral infection with B.C.G. vaccine in women that during pregnancy or while on the "pill" these patients fail to respond to the tuberculin skin test, but will show a reaction at the site of injection after giving birth or going off the "pill" (6,9).

In review, clinical observations of this disease, preliminary immunological studies, and recent reports in this area show:

1. A viral agent has been isolated from the thymus of affected animals.
2. In affected animals, the thymus involutes at an early age.
3. In some animals, growth rate and immunological response are severely affected in a direct relationship to the amount of irreparable damage to the thymus.
4. Affected animals show a poor immunological response as do pregnant animals.

All of this suggests to this author that perhaps many of these affected animals and many pregnant

Correlation of Immunological Response and Subnormal Growth Rate

Normal Animals					Brucella Plate Test and Brucella Antigen Intredermal			
No.	Breed	Humoral[1] Response 3-15-73		Cellular Response (inches)	Wt. 3-15-73	Conception 10-21-73		Wt. 9-18-73
R4	H/Angus	1:40		1.25 x 1.0	525 lbs.	Pregnant		
R1D	H/Angus	1:80		1.0 x 1.0	565 lbs.	Pregnant		
R12	H/Angus	1:40		1.25 x 1.3	585 lbs.	Pregnant		750 lbs.
R16	H/Angus	1:40		1.0 x 1.3	555 lbs.	Pregnant		690 lbs.
R17	H/Angus	1:80		.75 x .9	480 lbs.	Pregnant		
R21	Ang/SH	1:80		1.8 x 1.0	590 lbs.	Pregnant		
R26	H/Angus	1:40		1.7 x 1.8	545 lbs.	Pregnant		
R36	H/B.S.	1:80		2.2 x 2	565 lbs.	Pregnant		
R37	H/Angus	1:40		2.25 x 1	530 lbs.	Pregnant		
R40	H/Angus	1:80		.75 x .9	500 lbs.	Pregnant		
1W	Hereford	1:160		2.2 x 1.5	525 lbs.	Pregnant		650 lbs.
			Avg.	1.41 x 1.15	543.5 lbs.		Avg.	676 lbs.
B47	Hereford	1:80		.4 x .3	295 lbs.	Open		
B53	Hereford	1:80		.6 x .1	410 lbs.	Pregnant		
B59	Hereford	1:40		.75 x .1	325 lbs.	Pregnant		
B73	Ang/Hereford	1:20		.75 x .25	325 lbs.	Open		470 lbs.
B81	Hereford	1:160		.7 x .8	485 lbs.	Open		
B88	Here/Angus	1:80		.7 x 1.25	365 lbs.	Pregnant		
B113 (2 yr)	Angus	1:10		–0–	500 lbs.	Open		
1D (male)	Hereford	1:160		.3 x .2	320 lbs.	Open		405 lbs.
1C	Hereford	1:80		.3 x .3	325 lbs.	Open		370 lbs.
			Avg.	.5 x .23	Avg. 372 lbs.		Avg.	416 lbs.
			Diff. Avg.	.91 x .92	171.5 lbs. in wts. @ 1 year			261 lbs. @ 18 mos

animals may well be more susceptible to infections of all types and may also fail to respond to our common vaccines for various bovine diseases.

Treatment

Treatment of affected calves is time consuming and often very disappointing. Using the whole blood, immune serum, fluids, and antibiotics, approximately 70% of the calves presented are returned to their owners, only to have some die at a later time, during the summer, and some become chronic "poor doers."

Summary

I have attempted to describe a disease entity of abortion, weak and abnormal calves seen during a normal calving season in well managed cow-calf operations from a clinical aspect, hoping that other practitioners may become aware of certain aspects of the disease.

In the diagnosis of this disease one must consider herd history, clinical observations, eliminations of other abortion disease by bacterial culture, blood serology and elimination of nutritional deficiencies and toxicities, and complete necropsies of aborted fetuses and abnormal calves.

This paper is based on clinical observations and necropsies of fetuses, calves and cows the past ten years. Approximately 750 necropsies were completed in a four-month period, February 1 to May 30, 1969.

Dairy cattle are not immune to the problem. The disease entity has been observed in dairy herds the year round.

It is estimated that 75% of the losses on many ranches in the area are due to this problem, and it is felt very strongly that it has a tremendous bearing on our baby calf scours in beef and dairy herds and a decided effect on weaning weights and % calves weaned (Graph II).

After becoming established in a herd it becomes an endemic problem. Apparently the problem has very little effect on infertility on a herd basis.

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Viruses as a Cause of Neonatal Calf Losses

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Numerous disease agents are responsible for neonatal calf losses. Recent studies on neonatal calves with diarrhea have been reported in Nebraska (1,2,3,4,5) as being caused primarily by two viral agents. These are a reovirus-like agent and a coronavirus-like agent. Previous workers (6,7,8) have reported other viral agents as being significant in neonatal calf enteritis (scours). These agents are a virus which causes calf pneumonia-enteritis and strains of bovine viral diarrhea virus (B.V.D.).

Mebus and co-workers (1) produced neonatal calf diarrhea in experimental gnotobiotic and germfree calves, one to five days of age, with field fecal material and with bacteria-free filtrates prepared from feces of the above experimental diarrheic calves. The period of incubation, age of calf, clinical signs, and mortality were identical to those observed in diarrheic calves on many of the ranches and in dairy herds. The incubation period was 12-18 hours. The clinical signs, when calves were exposed to the reovirus-like agent, were characterized by depression, inappetance, and profuse watery, yellow feces. Most affected calves drooled saliva from the commissures of the lips. Fecal material from these calves, when examined by electron microscopy, contained reovirus-like virions with an approximate diameter of 65 nm (1,9). When fecal material or sections of the small intestine were stained by fluorescent antibody (FA) technique (1) immunofluorescent cells were observed. These cells in the feces were cast off epithelial cells. This procedure (10) is recommended for diagnosis and differentiation of reovirus-like infections and/or coronavirus-like infections in calves.

Nebraska workers (4,11) reported that the incubation period in calves for coronavirus-like infection varied from 19-24 hours. Clinical signs observed were mild depression, dehydration, anorexia, and yellow diarrhea. They indicated that the major difference between reovirus-like and coronavirus-like infections was the continuation of diarrhea in the coronavirus-like infected calves. After several days of diarrhea, the feces had a curdled appearance, contained mucus, and dehydration was greater in coronavirus-like infected calves. Coronavirus-like infection in field outbreaks usually occurs in calves seven to 14 days of age or older; whereas reovirus-like infections are observed at one to seven days of age.

When sections from the small and the large intestine were stained by the FA technique with corona conjugate, epithelial cells were observed to fluoresce. When reovirus—like conjugate and B.V.D. conjugate were used on these same areas, negative results were obtained.

Limited experimental studies (12) in newborn calves exposed orally to an attenuated reovirus-like agent propagated in bovine kidney cells were protected when challenged 72 hours post-vaccination with homologous virulent virus. These findings led to the commercial production of a vaccine. In 1972, a field survey was made on a total of 64 beef herds. Eight thousand twenty-six calves and 35 dairy herds with 806 calves were vaccinated. The median calf morbidity rate before