infection. When the carrier state is eliminated by treatment, the recovered animal is rendered susceptible to infection.

Vaccination

The use of a vaccine has proved to be a valuable aid in reducing losses from anaplasmosis. Anaplaz®, produced by Fort Dodge Laboratories is the most frequently used vaccine to aid in the prevention of clinical signs of anaplasmosis. While the carrier state may occur in vaccinated cattle following exposure to infection, clinical illness and death losses are absent or minimal. The vaccine has been used in combination with tetracycline therapy to provide both immediate and long term resistance to infection.

Strict adherence to product label directions is of utmost importance. Initially, cattle must be vaccinated twice at not less than four week intervals. The first injection serves as a sensitizing dose, the second injection is the immunizing dose. Ideally, the series should be completed at least two weeks prior to the beginning of the vector season. Animals vaccinated in this manner will develop a positive titer to the complement fixation and card test that may persist for one to four months or longer. Annual 2 cc. booster injections should be given when indicated.

In 1968 neonatal isoerythrolysis was first recognized in calves. The disease was associated with the use of Fort Dodge Anaplaz ® vaccine for immunizing cattle against anaplasmosis. There are several reasons for the syndrome occurring in other species. Reports have been received of the disease appearing in herds that have never been vaccinated for anaplasmosis. Circumstantial evidence indicates there is a risk involved in the use of the vaccine. When an anaplasmosis control program is being outlined that includes the use of vaccine, every aspect of the benefits versus the hazards should be completely discussed and understood. Adverse vaccine reactions should never come as a complete surprise or disappointment to the veterinarian and his client.

Clostridial Infections of Cattle

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I. Clostridia General

A. 1. Anaerobes; 2. Gram positive rods; and 3. Spore formers.

B. Grouping: Invasive group: 1. *C. chauvoei*, 2. *C. septicum*, 3. *C. sordellii*, 4. *C. novyi*, 5. *C. perfringens* (man). Non-Invasive group: 1. *C. perfringens* (man & animals), 2. *C. tetani*, 3. *C. hemolyticum*, 4. *C. botulinum*.

II. Clostridium chauvoei: (feseri) "Blackleg"

A. Pathogenesis:
1. Younger cattle predominantly
2. Taken in orally - crosses intestinal barrier
3. Spores lodge in liver and muscle
4. Area of necrosis formed usually by trauma (Anaerobiasis)
5. Toxemia and death
6. Possible wound infection
7. Usually moderate morbidity

B. Signs:
1. Incubation period of 2-5 days
2. High temperatures of 104-106°
3. Muscular stiffness often with S.Q. crepatation
4. Sudden death

C. Post Mortem Lesions:
1. Lesions of muscles are *usually* found in heavily muscled areas such as thigh, shoulder, back and neck
2. Also found in tongue, diaphragm and myocardium
3. Lesions are dark, dry and contain gas.
4. S.Q. crepatation is present - caution is needed with this lesion

D. Diagnosis:
1. History-Signs-Lesions-Lab confirmation
Prevention & Treatment:
1. Early Penicillin - large doses
2. Formalized bacterins

III. Clostridium septicum: (Malignant edema)

A. Signs:
1. Incubation period of 2-4 days
2. High temperature, depression, edema around wound with subsequent gravitation
3. Death in 12-48 hours - high mortality - low morbidity

B. Pathogenesis:
1. Cattle of any age
2. Wound contaminant - fecal and soil contaminant
3. Spores are introduced into wound, which is anaerobic in some of its parts - Organism vegetates, grows, produces a very powerful and lethal toxin

C. Post Mortem Lesions:
1. Edema, necrosis and hemorrhage in and around

the wound, little gas and foul odor.

D. Diagnosis:
1. History - signs - post mortem lesions - lab confirmation

E. Prevention and Treatment:
1. Formalized bacterins
2. Penicillin - massive doses

IV. Clostridium novyi (oedematiens). Gas Gangrene (cattle) Black's Disease (sheep)

A. Pathogenesis:
1. Actually unknown - Probable wound infection, but can have a similar pathogenesis as Black's Disease in sheep. Forms powerful exotoxins

B. Signs:
1. Incubation period - thought to be relatively short, two to three days
2. Sudden death

C. Post Mortem Lesions:
1. Lesions of massive edema w/necrosis, foul smelling - usually found subcutaneously - resemble *C. septicum* lesions

D. Diagnosis:
History - sudden death - P.M. lesions - lab confirmation on *fresh tissues*!

E. Prevention & Treatment:
1. Formalized bacterins
2. Possibly penicillin - Treatment doubtful

V. Clostridium sordellii

A. Pathogenesis:
1. Unknown - thought to be taken in orally - may be a wound infection
2. More frequently seen in feedlot cattle on a high plane of nutrition (high protein)
3. May be associated with *Hemophilus sominus* infection

B. Signs:
1. Short incubation period - probably 2-3 days
2. Sudden death - rarely found ill
3. Experimentally - no temperature elevation

C. Post Mortem Lesions:
1. Muscle necrosis, edema, hemorrhage, seen most often in the area of the neck or throat and brisket
2. Gas bubbles or pockets not observed
3. Regional lymph nodes severely hemorrhagic
4. Hemorrhage surrounding the tracheal rings

D. Diagnosis:
Vaccination history - sudden death - lesions - lab confirmation

E. Prevention & Treatment:
1. Formalized bacterins
2. Reduce protein intake while immunizing
3. No treatment known

F. Other:
A sordellii enterotoxemia has been reported

VI. Clostridium hemolyticum
Red Water disease - ictero-hemoglobinuria

A. Pathogenesis:
1. Oral-acute duodenitis and systemic involvement to the liver
2. Liver necrosis, flukes or other factors - anaerobiasis and growth
3. Elaboration of powerful toxins causing focal necrosis and massive intravascular hemolysis

B. Signs:
1. Sudden onset - depression - temp. 104-106°
2. Hemoglobinuria - mucous membranes pale and icteric
3. Rapid respiration - early constipation then diarrhea
4. Course 1-4 days - morbidity 10-25% - mortality 95%

C. Post Mortem Lesions:
1. Dehydration - tissues pale and icteric - blood thin
2. Evidence of duodenitis
3. Swollen liver - anemic infarct
4. Kidneys dark - dark urine in bladder

D. Diagnosis:
1. History - Signs - P.M. Lesions - Lab confirmation

E. Prevention and Treatment:
1. Formalized bacterins
2. Massive penicillin injections - supportive therapy

VII. Clostridium tetani (Tetanus)

A. Pathogenesis:
1. Wound invader - small area of anaerobiosis
2. Growth and powerful neurotoxin produced - (tetanospasm)

B. Signs:
1. Incubation period - one to three weeks
2. Ears erect, tail stiff, prolapsed nictitating membranes
3. Violent muscular contractions following external stimuli
4. Mortality Rate - Around 60% in cattle

C. Post Mortem Lesions:
1. None - or perhaps only original wound

D. Diagnosis:
Typical signs - History of wound - Lab confirmation not possible

E. Prevention & Treatment:
1. Antitoxin
2. Toxoids
3. Treatment - High levels of penicillin - tranquilizers - dark quiet quarters, administration of fluids I.V. and liquid nourishment orally. Tetanus antitoxin, although questionable efficacy at this time

VIII. Clostridium botulinum (Botulism)

A. Pathogenesis:
1. Preformed toxin ingested
2. Obtained from carrion or from animal carcass contaminated hay or silage
3. Powerful neurotoxin

B. Signs:
1. Depends on dose taken in
   a. Large dose - found prostrate - or head turned into flank - tongue protruding - death in a

few hours
b. Lesser dose - slowly developing paralysis - beginning with difficulty in masticating feed - drooping eyelids and ears - stiff clumsy gait

C. Post Mortem Lesions - None
D. Diagnosis - Difficult - Signs - Lab confirmation - 66-100 Mortality in cattle
E. Prevention and Treatment:
1. No treatment
2. Remove source of contaminated feed
3. Formalized Bacterin-Toxoid

IX. Enterotoxemia (Clostridium perfringens - Welchii)

A. Criteria for Development:
1. Toxogenic strain
2. Abundance of growth promoting nutrients
3. Partial or complete stasis of the gastro-intestinal tract

B. Major Toxogenic Strains:
Type and Toxin
A - alpha
B - beta and epsilon
C - beta
D - epsilon

C. Diagnosis:
1. History:
a. Sudden death - steers on full feed or best doing calves
2. Lesions:
a. Hemorrhagic enteritis with Type C
b. Mucoid and catarrhal enteritis w/Type D
c. Hemorrhages in the thymus
d. Hemorrhages on intestinal serosa and diaphragm
e. Increased straw-colored pericardial fluid
3. Intestinal Smears:
a. Preponderance of large gram positive rods and absence of normally found gram negative flora
4. Mouse neutralization
5. Urine glucose (Type D)

X. Immunizing Products

A. Bacterins:
1. *C. Chauvoei* Bacterin
2. *C. Chauvoei - C. Septicum* Bacterin
3. *C. Chauvoei - Pasteurella* Bacterin
4. *C. Chauvoei - C. Septicum - Pasteurella* Bacterin
5. *C. Novyi-Sordellii* Bacterin
6. *C. Chauvoei -Septicum-Novyi* Bacterin
7. *C. Chauvoei-Septicum-Novyi-Sordellii* Bacterin
8. *C. Hemolyticum* Bacterin
9. *C. Chauvoei-Septicum-Novyi-Sordellii-Perfringens* - Type C & D Bacterin

B. Bacterin-Toxoids:
1. *C. Botulinum* Type *C Bacterin*-Toxoid
2. *C. Novyi* Bacterin-Toxoid
3. *C. Novyi Sordellii, Perfringens* Type C & D Bacterin-Toxoid
4. *C. Perfringens* Type *C Bacterin*-Toxoid
5. *C. Perfringens* Type *D Bacterin*-Toxoid
6. *C. Perfringens* Type *C & D Bacterin*-Toxoid

C. Toxoids:
1. *C. Perfringens* Type C Toxoid
2. *C. Perfringens* Type D Toxoid
3. *C. Perfringens* Type C & D Toxoid
4. *C. Perfringens* Type D - Tetanus Toxoid
5. Tetanus Toxoid

D. Antitoxins:
1. *C. Perfringens* Type C Antitoxin
2. *C. Perfringens* Type D Antitoxin
3. *C. Perfringens* Type C & D Antitoxin
4. Tetanus Antitoxin

Salmonellosis in the Bovine Animal

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Salmonella organisms are gram negative rods that are parasitic in man and animals and usually are viewed as enteric organisms.

The first "salmonella" organisms were isolated in 1885 by Smith and Salmon from swine. In 1888, the second isolote of the "salmonella" group was from a man who had eaten raw meat from a diseased cow (1). Salmonella was the name proposed for the group of organisms in honor of D.E. Salmon, the first chief of the United States Bureau of Animal Industry.

It is interesting to note that in 1934, 44 serotypes were recognized. This number gradually increased to 962 in 1966 and has continued to some 1,300 now. However, most salmonella isolated now belong to a small number of serotypes (3). The disease is believed to be worldwide and in cattle it appears to have dramatically increased in the last few years. In fact bovine salmonellosis has been recognized as an important cattle disease only since the late 1950's (4). Part of this increase may be more apparent than real because of better isolation techniques, and especially a greater awareness of the disease in cattle. It has, of course, been recognized for a long time as a major dis-