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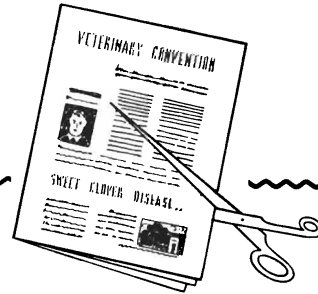
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ABSTRACTS



THE OCCURENCE OF VIBRIONIC ABORTION IN AN ILLINOIS DAIRY HERD. A pure culture of *Vibrio fetus* was obtained from the routine culturing of the stomach contents, liver, and kidneys of aborted fetuses from a large Illinois dairy herd. The portion of the herd concerned showed no evidence of the presence of *Brucella abortus* or *Trichomonas fetus* for 15 years, nor was there any evidence of a deficiency in the level of beta-carotene in the blood. The abortion rate in this herd, in which good husbandry methods had been used, had risen steadily until a peak of 12.5 percent had been reached in a period of 12 years.

The organism was recovered in 3 instances only from the stomach contents of the aborted fetus, and only on tryptose crystal-violet agar plates incubated in an atmosphere of 10 percent carbon dioxide. Photomicrographs made by the means of an electron microscope demonstrated the morphological forms of *Vibrio fetus*. The development of *Vibrio fetus* upon primary isolation seems to be more rapid than that of *Brucella abortus*, but the viability of the former on laboratory media is of short duration. Attempts to artificially transmit *Vibrio fetus* to 3 pregnant cows by intravenous injection, intra-uterine inoculation, and by oral administration were all negative. The organisms used for inoculation purposes may have been attenuated when they were cultured on artificial laboratory media.

So far as can be determined, this is the first report of the occurrence of vibrionic

abortion in cattle in Illinois. Strict sanitary and control measures are now being used on this farm, but the effect has not been evaluated as yet.

[H. E. Rhoades and Harry Hardenbrook, Jr. *The Occurrence of Vibrionic Abortion in an Illinois Dairy Herd: Cornell Vet.* 37:8-13.]

THE FATE OF ENDOCARDIAL VEGETATIONS FOLLOWING PENICILLIN TREATMENT OF BACTERIAL ENDOCARDITIS. The apparent curability of both acute and subacute bacterial endocarditis through penicillin therapy has been well established clinically. Several clinics have reported disappearance of all symptoms, signs of infection, and sterilization of the blood following its use. It has been suggested by some investigators that these lesions may not be healed, but are merely examples of the rare or hypothetical "bacterial-free" stage of the disease. Proof that endocarditis has been cured, and is not merely a "bacteria-free" stage of the disease, lies in the demonstration of unequivocally healed, sterile remnants of vegetations in previously treated cases, following death from other causes.

After death, from causes other than endocarditis, 4 human patients previously treated with penicillin for endocarditis, were presented for post-mortem

examination. Each of the individuals had suffered from bacterial endocarditis. Blood cultures of 3 of the patients yielded *Staphylococcus aureus* and one *Streptococcus viridans*. Following treatment with penicillin, the blood of all 4 patients became sterile. Cultures of the heart following death yielded no growth in 3 of the cases, but yielded growths of *Candida albicans* in one of the patients. None of the deaths could be directly attributed to bacterial endocarditis. However, the congestive failure of the heart was probably due to the aortic valvular location of the bacterial endocarditis accompanied by the sudden strain thrown on the left ventricular musculature.

The 4 cases constitute proof that the infective component of bacterial endocarditis is curable with penicillin. The nature of the healing process in its several stages was studied with histological studies of the vegetations. A mass of granulation tissue and hyalinized connective tissue, surrounded by eosinophilic material and covered by endothelium, represented an early stage. Also seen early, was the organizing mass of fibrin, erythrocytes, and leucocytes that composed the vegetation. More advanced organization and the deposition of calcium within the vegetation indicated a later stage. Finally, the completely healed lesion was marked by a pale, hard, smoothly endothelialized mass of dense connective tissue and areas of calcification. The absence of thrombotic accretions on the vegetations implies that anticoagulant medication is not an essential adjunct of therapy.

Obvious implications of the studies made were: (1) Cases of bacterial endocarditis can be effectively treated with penicillin if the treatment is started early. (2) Convalescence in patients with vegetations on the aortic valve should be prolonged. (3) Return to physical activity should be undertaken slowly.

[Arthur J. Geige, M.D. and Stanley H. Durbachor, M.D. *The fate of Endocardial Vegetations Following Penicillin Treatment of Bacterial Endocarditis: Am. Jour. of Pathology (Nov., 1947):23, 6, 1023-1036.*]

Spring, 1948

INFECTION AND TRANSMISSION OF BRUCELLA BY ECTOPARASITES.

On some occasions, the transmission of brucellosis to either man or animals can not be satisfactorily explained. Occasionally human beings became infected who have not been in contact with infected animals or have not consumed raw milk. Such cases suggest the possibility of a mechanism of infection other than the ones already known. Using the theory that ectoparasites might serve as vectors of the *Brucella* organisms, Dr. Tovar conducted experiments in which ticks, bedbugs and fleas were infected. All of these ectoparasites were found to be susceptible to the 3 species of *Brucella*—*Brucella melitensis*, *Brucella abortus*, and *Brucella suis*. *Brucella suis* was found to be the most infective for all. Fleas were the first to eliminate the *Brucella* organisms in the feces after the infective meal, while ticks and bedbugs were eliminating *Brucella* in the feces 3 months after infection. Cultures of all the infected ectoparasites yielded growth of *Brucella*. An attempt was then made to transmit *Brucella* by allowing the infected ectoparasites to feed on previously normal guinea pigs. Ticks were the only parasite found able to transmit the infection by biting, and then only if they were uninterrupted in their feeding. Ticks were also able to transmit the organism through their eggs and larvae. Bedbugs were not able to transmit the infection by their bite, but they eliminated the organisms in their feces for a considerable period of time. They may infect human beings by direct contact through the skin and mucous membranes, or by the digestive tract. Contaminated feces may also infect through the respiratory tract. Infected fleas eliminated great quantities of *Brucella* in the feces and, as they usually defecate close to the area they bite, the patient could easily infect himself through scratching.

After obtaining the above results, a careful search of naturally infected ectoparasites was made. Ten lots of *Cimex lectularis*, collected in Mexico City from the bedsides of Brucellosis patients, and 5 lots of *Boophilus annulatus*, the cattle tick,

collected from the state of Guanajaato were studied. Guinea pig inoculations were made with solutions made from the infected ectoparasites. Two lots of *C. lectularis* were found to be infected with *Brucella melitensis* and 1 lot of *B. annulatus* was infected with *Brucella abortus*. Two lots of ticks produced fatal infection in guinea pigs within 72 hours. The causative agent which was not *Brucella* has not been identified.

In relation to the finding of Dr. Tovar, brucellosis patients should be considered a possible source of infection and should be freed of ectoparasites.

[Paul M. Tover, D. F. Mexico. *Am. Jour. Vet. Research* (Jan., 1947): 8, 138-140.]

SOME OBSERVATIONS ON BEEF CATTLE AFFECTED WITH GENERALIZED EDEMA OR ANASARCA DUE TO VITAMIN A DEFICIENCY.

The fact that prolonged vitamin A deficiency in cattle may result in generalized edema has been recognized for some time. However, in view of the large number of carcasses that have been condemned in past years by federal meat inspectors, the condition is fairly common. During the year 1943, 1,169 carcasses were totally condemned for anasarca, and this figure does not include the portions of carcasses that were condemned for localized edema. It was found that a fewer number of carcasses were condemned during the years when the grain fed was light or the feeding of short duration. More carcasses were condemned during the latter part of each year.

Two farms that were having difficulty with anasarca were investigated by the authors. On one of these farms 30 of the steers being fed had swollen legs and enlarged briskets, one had been sent to market but had been condemned for anasarca. It was found that the only roughage that had been fed to the animals the previous 3 months was oat straw. A correction in the diet was suggested to

include alfalfa hay. Eight weeks after the feeding of the alfalfa hay was started, the entire lot was sold on the market without evidence of anasarca. On the other farm, 135 steers were being fed. Three months after the animals had been on a diet consisting of oat hay as the only roughage, several of the animals were sent to market and 3 had been condemned because of anasarca. When the authors visited the farm, the animals had been on the diet for 4 months. Several of the steers showed marked symptoms of vitamin A deficiency, blindness, and severe edema of the legs, brisket, and shoulder. Again, alfalfa hay was used in correcting the diet, and shortly, symptoms of anasarca disappeared.

Experimentally, cases of anasarca were produced by feeding either a carotene deficient ration or a ration containing a large amount of yellow corn. Edema was readily produced when the animals were confined to these rations for several months. In a number of instances, after the appearance of anasarca, the diet was supplemented with one of the several sources of vitamin A, such as alfalfa leaf meal, carotene concentrate, crystalline carotene, cod-liver oil, or distilled vitamin A in natural ester form. All symptoms of anasarca with the exception of blindness were relieved with the altered diet.

Several samples of blood, both from clinical and experimental cases were studied. The only significant change found was a distinct lowering in the serum albumin content of the blood. This could hardly be blamed for the formation of edema, however, as there was an increase in total protein which was sufficient to compensate for the decrease in albumin content. Therefore the colloid osmotic pressure was approximately normal. There were deficiency levels of vitamins A and C. Calcium and phosphorus decreased slightly, phosphatase activity was reduced, while magnesium remained unchanged.

Losses due to anasarca associated with vitamin A deficiency are economically important. Never-the-less, they can be prevented by proper diets. Supplying cattle with a small amount of well cured

alfalfa hay, and any other hay having a good green color, well made ensilage, green pasture, or a marine oil, such as cod-liver oil will avoid anasarca due to a deficiency of vitamin A.

[Louis L. Madsen, I. P. Earle. *Some Observations on Beef Cattle Affected with Generalized Edema or Anasarca Due to Vitamin A Deficiency: The Jour. of Nutrition* (Dec., 1947):34, 6, 603-620.]

CHEMOTHERAPY OF VIRUS INFECTIONS. Chemotherapy has become increasingly effective in attenuating the striking power of protozoa and bacterial diseases, but this approach to the control of virus diseases has been unsuccessful except in a few conditions, such as trachoma and mouse pneumoitis, which are susceptible in varying degrees to sulfonamides and penicillin.

Many medicinal agents, which might be chemotherapeutic or antiseptic in inactivating the virus, either while in transit to its final cell, or after entry into this cell, has been studied in the past with disappointing results. These drugs were of such classes as antibiotics, sulfones, sulfonamides, alkaloids, metals, essential oils, detergents, acridine dyes, and many others which were tried either in vivo, or in vitro.

Attempts by the authors to find a chemotherapeutic agent against a virus followed the general plan of previous workers, in which the agents were tested against vaccina, influenza, and herpes simplex viruses in chick embryos and mice. Trial of over 150 compounds, which were classed as antibiotics, dyes, metallics, sulfonamides, and others gave negative results.

Due to the apparent ineffectiveness of the chemical and therapeutic agents, substances of another category, which are agents of a biologic nature, were studied with a limited amount of success realized. These are agents that allow the cell to survive the competition of the parasitic virus by altering the relationship between the cell, virus, and substrate. Instead of injuring the virus by chemical poison, an approach on the basis of the competition

between the virus and the cell for necessary substances or mechanisms was explored. Alteration of the cell-virus-substrate relationship might allow predominance of the cell over the virus. Thus, the cell might withstand a deficiency of a necessary element of the substrate, while the virus could not stand this deprivation. In the presence of an abundance of a necessary substance, the cell might be able to outlast heavy demands by the virus for this substance. With either mechanism, the virus might be extinguished or might become a symbiotic in the cell.

On the basis of the possible mechanism just outlined, trials with deficiencies and excesses of biologic materials were made. Thiamine deficiency proved to be relatively effective in protecting mice against herpes simplex. An attempt to deepen the thiamine deficiency by the administration of pyridoxine did not enhance the action. An excess of riboflavin also offered protection of a slight degree against herpes simplex in mice. However, certain amino acids, intermediates in carbohydrate and fat metabolism, and agents containing labile methyl groups were ineffective.

Another group of amino acids, vitamin analogues, and substituted compounds derived from nucleic acid were tried. None was effective in herpetic infection in mice, although several prolonged the lives of chick embryos infected with vaccina.

Thus, it appears that although no highly therapeutic compounds have been found, minor effects can be produced, and that virus infections appear to be subject to influence by deficiencies and excesses or alterations in their intracellular substrate.

[R. H. Driesbach, R. M. Halper, N. E. H. Irwin, D. W. Jenkins, F. Proescher, H. B. Trippi. *Chemotherapy of Virus Infections: Jour. of Immunology* (Dec., 1947): 57, 12, 379-390.]

Tuberculosis was the most serious disease of cattle in Iowa in 1895. Tuberculin tests had revealed the presence of the disease in many supposedly disease-free herds.

HYPERKERATOSIS OR X-DISEASE OF CATTLE.

A chronic disease characterized by a keratinized thickening of the skin or the mucosa of the mouth and by papillary proliferation of unknown origin has been observed. These were found in several herds of cattle scattered widely in New York, Pennsylvania and Maryland. The outbreak of this disease, which affects young stock more readily than older cattle, appeared during late winter or spring in herds fed a variety of rations. Some of the rations included mineral mixtures containing many of the trace minerals. Other rations contained roughage of an inferior quality, which resulted in the cattle living almost entirely on a portion of the grain ration.

Since this disease is so chronic in nature, the initial clinical manifestations were not observed, but owners reported that a watery discharge from the eyes and nose was the earliest change seen. This was followed by depression and stupidity, accompanied by loss of appetite and loss of condition. The pulse and respiration were not increased and the temperature was not elevated. When cows near freshening were affected, they would exhibit acetoneuria, mastitis, developmental failure of the mammary glands, and numerous obstetrical difficulties, such as retained placentae and septic metritis.

The lesions were found mainly in the skin, mouth, tongue, and esophagus. The skin lesions were found primarily over the withers, on the sides of the neck, over the cheeks, and back of the shoulders. Occasionally the brisket and the inside of the thigh would become involved, but usually the ventral portion of the body was not altered. The skin lesions were characterized by a loss of hair and thickening of the skin caused by an accumulation of keratinized material on the surface. No attempt was made by the animals to rub or lick the affected parts.

The lesions of the mouth, tongue, esophagus, and occasionally of the omasum consisted of raised, rounded, papillary proliferations of variable sizes. Occasionally, *Actinomyces necrophorus* lesions were also found on the tongue and cheeks.

Other common lesions that were ob-

served included mucoid proliferations of the lining of the large bile ducts, fibrosis of a part of the liver, fibrosis of the pancreas, enlargement of the kidneys and a thickening of the mucosa of the intestinal tract, especially in the cecum and colon.

Transmission experiments were carried out on over 20 animals in which such methods as large blood transfusions, introduction of normal animals into affected herds, and intimate contacts in small stalls failed to transmit this disease.

A consideration of the differential diagnosis of this disease shows that it may, upon superficial examination, be confused with mange and with the recently described virus diarrhea. Hyperkeratosis can be distinguished from mange by the presence of the mouth lesions, and the absence of mange mites. Virus diarrhea is an acute disease whereas hyperkeratosis is chronic. Punched out ulcers occur in the tongue in virus diarrhea, the mortality is low, and recovery is fast; whereas in hyperkeratosis, the lesions are proliferative, and the animals almost always die. Virus diarrhea is not characterized by skin lesions of any kind or by lesions of the liver and kidneys, as is hyperkeratosis.

Numerous attempts at treating hyperkeratosis with such drugs as penicillin, sulfonamides, vitamins A, B, C, niacin and riboflavin have been without favorable results.

[Peter Olafson. *Hyperkeratosis (X Disease) of Cattle: The Cornell Veterinarian* (Oct., 1947):37, 4, 279-291.]

Deterioration of Vitamin A in The Presence of Fats

Vitamin A may disappear quickly from various types of oils, or when added to mixed food in the presence of rancid fats. Ingredients added to rations as vitamin A carriers may lose most of their value during transportation or storage.

This vitamin retains its potency much longer in the form of gelatin capsules, Dr. Morris advised.