Department of Agriculture. Each of these READEO's is under the direction of a veterinarian from the Veterinary Services Regional Director's office. He is assisted by State veterinarians representing the specific States affected and/or under quarantine.

The Director maintains a close liaison with the military, legal staff, Meat and Poultry Inspection personnel, information specialists, and wildlife specialists. He also works closely with a disease specialist, who is considered to be an authority on that specific disease problem.

Each foreign animal disease outbreak presents its own unique problems:

1. How do we control milk and/or assure it is safe?
2. How do we control meat and/or assure it is safe?
3. What role does wildlife play in the scenario?
4. If vaccination is indicated, how do we distribute and monitor administration?

All of these problems are being discussed with the affected industries. Based on these discussions, the eradication procedures and policies are being updated with the hope of developing a workable plan which will eradicate the disease agent and yet let business go on as usual as much as possible.

In addition, we are looking for alternatives to the policy of diagnose and slaughter. For example, in a FMD campaign, one might consider the use of vaccine. It is for this reason that USDA has decided to purchase 2 million doses each of Types A, O, and C from Bayer laboratories in Cologne, Germany, and has asked them to stockpile it as an antigen for possible future use during a FMD outbreak.

When will the next foreign animal disease strike? We do not know. With the rapid transportation systems available today, it is only a matter of time. But, we do know that with the help of every veterinarian, animal health official, and the livestock industry, we will be prepared.

Foot-and-Mouth Disease in Cattle— Some Relationships Between Pathogenesis and Epizootiology

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Definition

Foot-and-mouth disease (FMD) is one of the most infectious of all of the animal diseases. It is viral in origin and occurs principally in cattle, swine, sheep, goats, and other cloven-footed animals, domesticated and wild. It was first described in Italy in 1546. The causative agent was isolated and determined to be a virus which was later shown to be approximately 23 millimicrons in diameter. Its high infectivity in several species; ability of the virus to spread rapidly; its widespread distribution; and its plurality of serotypes, are some of the characteristics which made FMD difficult to control.[1]

Geographic Distribution

It occurs in all of the large land masses of the world with the exception of North America, Central America, Panama, Australia, and New Zealand. It has not occured in North America since 1953 when it was eradicated in Mexico. It last occurred in Canada in 1952, in the United States in 1929, and in Australia in 1872 (ref. 1).

Economic Considerations

Wherever it has existed, it interferes with import and export trade in animals and animal products. Entry of products from enzootic areas into FMD free countries is either prohibited or so severely restricted that the price is affected. Because of the interference from FMD in world trade, the disease has on occasion been called a political disease; however, the problems which result from controlling FMD are real as well as political. Each new epizootic is widely publicized and often those repsonsible for its eradication are criticized. In spite of the fact that the disease has a long history, the public awareness of it and quarantine methods used by many countries, world-wide, effective control is in the distant future (ref. 1).

Host Range

While natural infection is limited to cloven-footed animals, domestic and wild, experimentally the virus can be propagated in other species including dogs, cats, chickens, rats, mice, rabbits, and guinea pigs. The horse has never been

infected naturally or experimentally. The disease exists in a wide variety of wildlife including deer, antelope, pigs and buffalo—any of which may pose a threat to control of infection during an epizootic. Man is rarely affected; thus, the disease is not considered a public health problem (ref. 1).

Pathogenesis

Much of the information on the course of the disease as well as clinical signs has accumulated over the decades and even over centuries ago and has been repeated in textbooks without reference to the original source. Until recently, it was commonly thought and so stated in the literature, that natural infection of cattle takes place by the oral route. While this is still considered to be one of the routes of infection, in the last 15 to 20 years more and more investigators have been accumulating evidence to lead us to the belief that the most important means of transmission of FMD virus is by aerosol which causes an initial infection in the upper respiratory tract.[2,3,4,5] Results with intranasal instillation of FMD virus in our laboratory have lead us to agree with the suggestion that this is the common or natural route of infection.[6] During the last two and one-half decades, there have been instances where the virus has been known to spread by aerosol from infected premises to adjoining lots where normal cattle were being held. In one instance the virus escaped from an infected premises and set up infection in cattle approximately two miles away. There was no direct contact between the premises. In our own laboratory building aerosol transmission as far as 200 feet has been demonstrated.[7] Virus introduced by this means sets up infection in the pharyngeal region of cattle and may be recovered from these areas as early as two hours following infection.[8] In these animals' throats the virus replicates in the soft tissue of the palate, tonsilar tissue, the posterior nares, and in the esophagus.

While there is now ample evidence that transmission by aerosol is probably the most natural means, we must not overlook the fact that FMD virus may be transmitted by many other means including ingestion, through the eye,[9] through the mammary tissue such as might happen by contaminated milking machines, through the vagina (natural or artificial insemination), and by inoculation of almost any portion of the exterior body surface. There is ample evidence that the virus can enter the animal's body by any of the means reviewed above.

Following replication of the virus in the tissues of the nasopharyngeal area, it is transferred to the associated pharyngeal or submaxillary lymph nodes where it begins to multiply further, and once entering the lymphatic system, it may spread throughout the animal's body. After further replication in the lymphatic system, the virus is then transported through the blood to predilection sites such as the tongue, other areas in the bucal cavity, the feet, and any of the several other body organs or areas in which or where the virus might replicate, such as heart muscle, pillars of the rumen, mammary tissue, and skin. Cottral has studied the viremia in cattle which were inoculated in the tongue with FMD virus. Under these conditions viremia was found as early as two hours but could also be delayed for as long as fourteen. The differences depend upon the virus dose, virulence, and host influence. Undoubtedly, had he studied it, the intranasal instillation would have produced other circumstances. He also concluded that the length of time viremia precedes clinical signs and lesions may vary from 8 to 40 hours. If the virus dose is high, the initial viremia may be predominantly composed of the inoculated virus particles. When lower virus doses are given, the host replicated virus particles probably are the main constituents of the viremia. The virus levels used also will influence the peak viremia titer. In his study, however, he concluded when all of the data was considered, the peak of viremia was between 40 to 42 hours post-inoculation. The variations in viremia titer are probably more influenced by variations in host response, than by virus strain differences. As to the duration of viremia, Cottral found viremia in cattle as short as three days and the longest was for five days. Regardless of the virus dose or route of inoculation, viremia persisted for no longer than five days.

When cattle were inoculated intramuscularly or by aerosol the starting time for viremia was delayed one or more days but the duration still did not exceed five days. By the time the virus is present in all of the body fluids and tissues, the gross lesions on the predilection sites are most likely at their height. Infection in these areas usually begins as a small blanched area in the epithelium which subsequently fills with fluid forming a vesicle. The vesicle may enlarge and may coalesce with other vesicles. The vesicles may rupture and the fluid escape through cracks in the epithelial covering. At this stage the covering may come off leaving an ulcer or eroded area. A grey colored fibrinous coating forms over the lesions. The coating becomes discolored—yellow, brown, or green. Following this the epithelium is restored but lines of demarcation develop because of color differences between the old and new tissues. Gradually the lines of demarcation fade away so that scars in some instances do not remain. The period of time from the beginning to the end of a lesion of FMD is influenced by many factors including such things as general health of the animal, feed, and especially bacterial contamination or secondary infection in the primary lesion site. On an average, however, it is 15 to 30 days before the new epithelium is generated to cover the eroded area that is left when the vesicle bursts or when sloughing of the epithelium occurs (ref. 3).

During the period the lesions are present, the animal will salivate profusely, and this is frequently seen hanging from the corners of the mouth as a ropy viscous material. At this stage of the infection, this saliva is virus laden. In addition, the animal is seen to lacrimate severely, and there is a nasal discharge. If lesions develop on the feet, the animal walks with difficulty and usually appears lame. It is prone to lie

down, and when it is forced to stand, it usually puts all four feet under its body so as to better distribute the weight. During these times, animals move only with a great deal of difficulty. About the time the vesicles rupture is the time that the fever ends. This is followed by the end of viremia, and it is at this stage that you begin to see neutralizing and other types of antibody. As antibody develops, there is a decline of virus titer in the tissues and other body fluids. Healing of the lesions takes place and the animal resumes eating. There is a gradual disappearance of virus in the tissues and body fluids and eventual healing of the lesions. From start to finish, the clinical signs will last from 15 to 30 days depending upon the various factors mentioned above.

Carriers

For many years livestockmen, researchers, and control officials were convinced that once the bovine had recovered from FMD that such animals were an important factor in epizootiology of the disease. There are various reports in the literature on the role of such animals in spreading infection. Other researchers were successful in recovering virus from animals that had recovered. It was not, however, until van Bekkum and coworkers in the Netherlands published their work on carriers that substantial information concerning the carrier became evident. **These workers found that a large percentage of animals carried the virus in their throats for varying period of time following infection.**[10]

Now, research workers in laboratories around the world as well as control officials are well aware that cattle which are infected with FMD may become carriers of the virus for long periods of time. It is also common knowledge that animals which have been vaccinated but which are subsequently exposed to the virus may also become carriers and not show evidence of the disease. While carriers may be readily detected the true role of such carriers in the spread of the disease is not yet known because no one has yet demonstrated that such carriers are responsible for infecting other cattle. However, many workers conclude that this is only because the carrier has not been caused to shed enough virus during given stress periods or the susceptible animal has for some reason not come in adequate contact with the virus given off by the carrier or there is a combination of these circumstances.[11,12] While spread has not been demonstrated from these carriers, most workers consider them as potential hazards, and consequently great effort is made to exclude such animals in the international movement of livestock from countries where FMD exists to those that are free and from countries where maybe on type of the virus exists to a country where that given type is not know, such as occurs when zebu are shipped from Brazil to Venezuela.

Following infection the recovered animal is usually immune to the type of virus from which it has recovered. Neutralizing antibody may be detected as early as four days after infection and peak antibody levels usually develop at least by 21 days after infection.[13]

The usual or typical form of FMD has been described. It should, however, be remembered that there are several other forms of the disease which livestock officials should be knowledgeable about. This includes the form of the disease where the virus replicates; lesions may or may not develop, but if they do, they are not in an area where they may be observed. The other form is that which results when animals are inoculated intranasally. In summary, one should remember that in cattle FMD or the signs, or pathogenesis of the disease will vary depending upon many factors including the type and subtype of virus inoculated, quantity of virus inoculated, species, condition, age, and health of the recipient with special reference to inapparent infections.[14]

Transmission

As indicated above, after inoculation the virus begins to replicate sometimes as soon as two hours. In other instances replication is not detected for days.

Burrows has studied excretion of Type O virus prior to the development of lesions. In his study groups of cattle, sheep and pigs were placed in an isolation compound with cattle which had been infected by inoculation. The test animals were sampled daily for evidence of virus in the blood, milk, pharynx, rectum, and prepuce. Virus was recovered from the pharyngeal samples of the majority of the animals several days before clinical signs of the disease appeared. Virus was also recovered from the blood, milk, rectum, prepuce, and vagina before signs of the disease. In this study some cattle and sheep were shown to shed virus for periods of up to five days and pigs for 10 days before the disease was diagnosed (ref. 4).

Sellers and Parker studied airborne excretion of FMD virus from infected cattle, sheep and pigs.[15] Excretion was highest from pigs per volume of air exhaled. Maximum recovery occurred 41 hours after infection in pigs and cattle, when lesions had generalized, and 17 hours after infection in sheep, before lesions were observed. These workers suggested that the site of production of virus excreted as aerosol is the upper respiratory tract. They further postulated that under conditions of low temperature and 70 percent relative humidity survival of virus for distances of 100 km is likely to occur. There is some evidence to support this theory in the 1967-68 outbreak in Great Britian where spread was reportedly by the air.[15] On the other hand, workers in hot and dry endemic areas tend to discount transmission by airborne means and probably for good reason.

Cottral, *et al.* studied FMD virus in semen from bulls experimentally infected with 6 of the 7 immunologic types of virus and demonstrated virus in semen as soon as 12 hours after inoculation and several hours prior to signs of the disease. Virus was found in 58 of 71 semen samples from 16 bulls for as long as 10 days. Heifers artificially inseminated with semen from infected bulls developed FMD. It was concluded from this study that semen of bulls may contain

FMD virus prior to signs of illness and that the disease could be transmitted by aritificial insemination.[16]

Netto has studies FMD virus in bull semen in Brazil and reported isolation of virus from 7 to 22 randomly selected ampules of semen to be used for artificial insemination. Five virus isolates were Type C and two were Type O.[17]

Knowledge that milk from cows with FMD may contain FMD virus has been known for many decades.[18] LeBailley, 1920, called attention to the fact that FMD virus maybe in milk before appearance of signs of illness. It was not, however, until the 1967-68 outbreak in Great Britian that infective milk was found to be a factor in spread of the disease.[20] In addition, levels of virus were found in milk prior to diagnosis of the disease and presented a problem in control. These workers concluded infective milk was undoubtedly responsible for numbers of outbreaks during the enzootic.

Following these observations, Burrows, in 1971, studied the excretion of FMD virus in milk from cows experimentally infected by contact exposure and by inoculation of virus into the udder.[21] Virus was found in milk several hours before signs of the disease, when the animals were exposed by contact and by udder inoculation; surprisingly, one animal continued to shed virus for 23 days after inoculation, and convalescing animals were susceptible to re-infection by udder isoculation.

The evidence available on the involvement of milk in the spread of the disease will cause animal disease officials to re-examine control measures to include better surveillance of milk from animals in the area where the disease is appearing. Since it is now evident that cattle shed in milk sometimes for 2 and 3 days before clinical signs of the disease appear, the area surrounding the actual outbreak where control measures on milk are enforced obviously will have to be expanded.

Following the 1967-68 outbreak in Great Britain, Sellers studied the rate of inactivation of FMD virus in milk at various temperatures and pH and found that both factors strongly influence the results.[22] At pH 6.7,99.999% of virus was inactivated at 6 minutes at 56°C; a minute at 63°C; 17 seconds at 72°C; and less than 5 seconds at 80 and 85°C. When the pH of the milk was u.6, the time to inactivate to a survival of 0.001% was 30 seconds at 56°C; w minutes at 63°C; 55 seconds at 72°C; and less than 5 seconds at 80°C. Traces of virus were detected after these times.

In practice, the virus content and pH of infective milk would be affected by the extent to which the milk is diluted with uninfected milk either at the farm, or in bulk handling procedures. The virus in infective milk may be in the form of free virus or in cells; in which event, the virus would survive longer. Little research has been done on the survival of the virus in infective milk under the conditions of drying as practiced in industry. Most milk that is dried is first pasteurized, and the volume is subsequently reduced to one-half by evaporation. Following this the milk is dried either on a roller drier or by spraying droplets in a hot atmosphere. The survival under such commercial practices should receive attention for such products may be dangerous, especially if they are used in free areas in animal feeds.

Cottral *et al.* conducted tests to study the survival of FMD virus in cured and uncured meat.[23] In their studies, samples of muscle, blood, and lymph nodes were tested, and virus was demonstrated in all fresh samples and in such samples stored at 16, 30 and 50 days at 4°C.

Carcasses tested after storage at 4°C contained demonstrable virus in rib bone marrow at 14, 60 and 73 days and in lymph nodes, blood, and muscle at 60 days. The chemical changes that take place during the ripening of meat inactivate the virus in muscle tissue but do not appreciably affect virus in lymph nodes, large blood clots, or morrow. Because of these and other studies and observations, some FMD free countries that now import meat from infected countries require that such meat be boned prior to shipment and there is no question but what this procedure lessens the risk of such importations.

In a joint study between representatives of the Government of Argentina and the United States, it was clearly shown that vaccination very markedly reduced the chances of recovering virus from lymph nodes and other tissues at the time of slaughter of cattle exposed to virus 32 hours previously. This is but another example that as the immunity is built up in cattle populations, the risk of spread of the disease by various means including meat is lessened.[24]

Gailiunas and Cottral studied the presence and persistence of FMD virus in bovine skin and showed that all 7 antigenic types of the virus have consistent affinity to all areas of bovine skin. Considerable amounts of virus were present in skin of 13 different body areas irrespective of presence of hair. In skins of some animals, FMD virus persisted for as long as 5 days after cessation of viremia. This observation also is of epizootiological importance for hides offered international trade, because FMD virus located intracutaneously is more difficult to inactivate than virus adhering to hide surfaces.[25]

In further studies on the survival of FMD virus in cattle hides held at lower temperatures, information has been developed that the virus persists for months at lower temperatures; this further complicating the epizootiological impact of such products.

Scott *et al.* studied FMD virus content in the pituitary gland and secondary nervous system of infected cattle and found high titers in these organs of cattle during the early clinical, clinical, and early convalescent stages of the disease. This information of pathogenicity of the virus in these two tissues is but further indication that FMD virus finds its way through the circulatory system or may replicate in almost any animal tissue or organ in the body. Special care must be exercised when such products are used for production of pharmaceuticals which may then be used in livestock. There are cases in the literature where pituitary extracts imported from countries where FMD exists into a free country were responsible for an epizootic of the disease. The extraction

process of the hormone from this and other glands does not inactivate the virus of FMD, and thus, when such products are produced from organs of animals, infected with the disease and are subsequently used in livestock, they pose a risk of introducing the disease.[26]

Summary

Foot-and-mouth disease virus may be transmitted by many means, but the two most common routes are thought to be by **aerosol** and **ingestion.** Virus penetration of cells takes place rapidly, and the primary sites of virus replication are probably in the cells of the naso-pharyngeal area. From there the virus enters the lymphatic and blood circulatory systems, and replication then takes place in the predilection sites followed by replication in many other areas of the animal's body. Factors that influence this include the virus characteristics with which the animal is infected, environment, and the host. Infected animals may transmit to other animals by exhaled air, saliva, urine, feces, and milk. Most body tissues may contain the virus and thereby pose a threat to spread of the disease.

Recovered cattle may be carriers of the virus; however, such animals have thus far not been caused to transmit virus to susceptible contact in laboratories. On the other hand, there is circumstantial evidence that this has occurred in the field. Because of the existence of carriers and the threat that products from infected animals pose to FMD free meat importing countries, the price of animals and animal products that are offered on the world market are influenced by the presence or absence of FMD in the exporting country.

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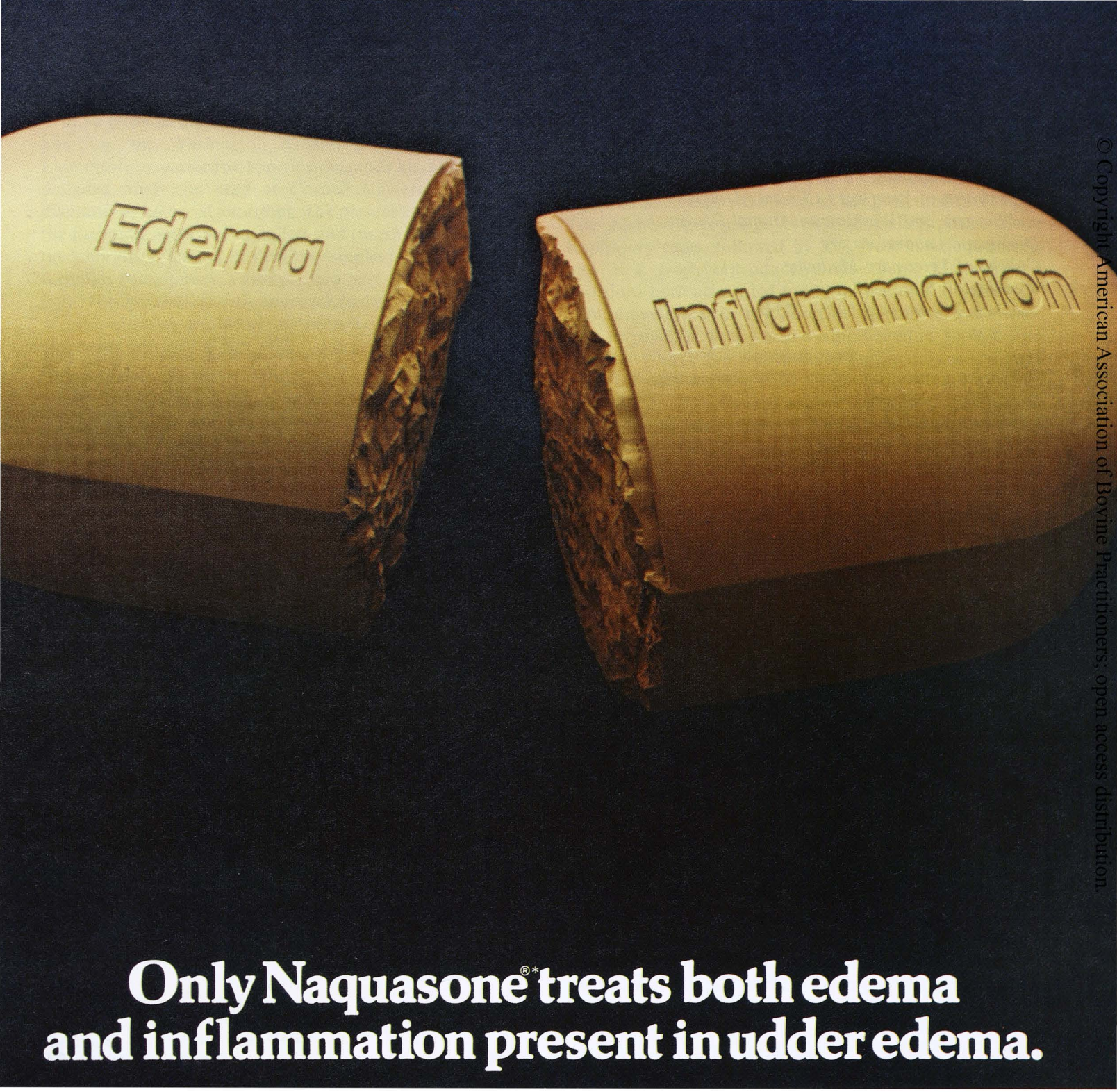

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