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Verminous Pneumonia of Cattle

ROBERT RUBIN

MOST internal parasites of cattle live in the stomach or intestine, but the worms known as *Dictyocaulus viviparus*, which live in the lungs, in many respects are the most injurious.

They are 2 to 3 inches long, white, and threadlike. They live in the medium and small air passages of the lungs. The females lay eggs, each containing a very small worm (first-stage larva). The larva usually hatches in the lungs.

First-stage larvae are coughed up and swallowed and then pass out in the droppings. An infected animal can pass 5 million larvae in a day—an important fact, for 5,000 fully developed larvae can kill a young calf.

On the ground the first-stage larvae develop but do not grow. When a certain stage is reached, the small worms (third-stage infective larvae) are able to reach the lungs and develop into mature worms if they are taken into the body of a susceptible calf, yearling, or adult.

The time required for first-stage larvae passed in the feces to reach the infective stage depends on the weather. Only a week is needed in a warm, wet period. Larvae do not develop in extremely cold or dry weather. The larvae can survive for weeks or months in the infective stage. They reportedly can survive an entire winter in parts of Great Britain, but in the United States the infective larvae have not been known to survive more than a few months at any time of year.

There are many degrees of infection. Even a lightly infected animal may be quite dangerous—it may show only slight or possibly no effects, but it might produce larvae enough to infect an entire calf crop or a number of yearlings or adults. The only way such an animal can be detected is by finding larvae in the droppings. That requires skill, because the larvae cannot be seen without a microscope.

An animal that ingests a large number of infective larvae becomes sick in a short time, usually within 2 weeks. The first noticeable sign is a cough, which becomes more and more frequent. Breathing is faster and more forceful. Often a loud grunt follows each breath. The sick animal may stand away from the herd with head lowered and extended.

As breathing becomes more difficult, as it does in severe and fatal cases, the animal breathes with its mouth open and tongue protruding—much as a dog pants. At that stage, coughing apparently has become too difficult or too much of a discomfort, and is seldom heard. A foamy and sometimes bloody material may collect around
the muzzle. The animal is reluctant to move, usually has a fever, is off feed and water, and becomes gaunt.

The worms usually are directly responsible for the symptoms. Large numbers of them in the air passages can cause a blockage sufficient to prevent enough air from reaching the lung tissue. Suffocation can result. The tissues also may react to the irritating presence of adults, eggs, and larvae by producing fluid (pulmonary edema), which collects in the small airspaces (alveoli) and keeps air from reaching the vital tissues. A fatal bacterial or viral pneumonia can develop because lungworms reduce resistance.

The stock owner can detect symptoms in a sick animal indicative of lungworm disease, but he must find larvae in the feces to be certain.

Only in animals that survive for about 4 weeks after their initial infection is it possible to detect larvae, because it takes that time for third-stage larvae to become adults; the latter produce first-stage larvae. Many infected animals become sick well in advance of this period, and in them a diagnosis cannot be made unless the animal dies or is destroyed and lungworms are found at autopsy.

A further complication is that an animal may die so soon after infection that the worms in the lungs are still microscopic and may be overlooked. Such cases are perplexing to the veterinarian, because the only way to differentiate lungworm (verminous) pneumonia from other lung diseases at autopsy is to find the worms.

Because there is no good treatment, control must be approached with the idea of preventing infections.

Pasture rotation, barn sanitation, and isolation of young stock are helpful but not completely effective. The climate in the Southeastern and Pacific Coast States, the major lungworm sites in the United States, favors the propagation of lungworm disease.

A different approach to control there and in the other regions may be to use phenothiazine, which in proper amounts reduces the number of larvae that become infective. As phenothiazine is effective against other internal parasites, the best recommendation that can be made is to administer phenothiazine, under veterinary supervision, to all in an infected herd.

The preceding facts refer to animals that are susceptible to lungworm infection. There are varying degrees of susceptibility, just as there are degrees of susceptibility or resistance to bacterial, viral, and other parasitic diseases. Such resistance is termed natural resistance.

A definite resistance develops in an animal that can survive a lungworm infection. Once an animal has recovered, adult worms usually do not develop in its lungs; it may pick up infective larvae and some of them may reach the lungs, but usually they do not become adults. These larvae in the lungs cause the animal some trouble, such as rapid breathing for a few days, an increase in temperature, and loss of appetite, but usually that is far less severe than the first time it became infected. That is active acquired resistance. Animals with this type of resistance have survived 15 times the number of infective larvae that have killed susceptible animals.

The blood of animals with an active acquired resistance apparently contains substances—antibodies—that, if put into the blood of a susceptible animal, can help protect it from the serious effects that would naturally accompany an initial infection. This type of resistance is passive acquired resistance. The potential value of active and passive acquired resistance could have great importance in control of this disease.

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