Liver Fluke and Stomach Worm of Sheep

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Liver fluke of sheep and cattle is becoming an increasingly important problem in California. This disease, known as "leaches," "liver rot," "rotten liver," and "flukes," as well as by its technical name of hepatic distomiasis, is caused by a flattened, leaflike, brownish-gray worm about one inch in length that is known as Fasciola hepatica.

The severity of the disease depends on the condition of the infested animal and the number of flukes that it is harboring. One rancher on the Pacific Coast is reported to have lost 1,500 animals last year (1927). The economic loss entailed by the animals that are fatally attacked, great though this may be, is but a part of the total loss. Animals that are infested even moderately fail to make proper weight, are poor mothers, and fall easy prey to diseases such as pneumonia, hemorrhagic septicaemia, and lung worms. Although beef cattle are seldom affected as severely as sheep, the loss of parasitized livers condemned at the slaughter houses on account of fluke lesions amounts to a formidable figure.

Until relatively few years ago it was believed that flukes were endemic only along the Gulf Coast states and in isolated spots along the Pacific Coast. At present we know the disease to be present on the Pacific Slope in Washington, Oregon, California, Idaho, Montana, Utah, and Colorado.

The Parasite

Flukes (fig. 1) are animals belonging to a queer group of flat worms known as trematodes. They are leaflike instead of rounded like roundworms or elongated like tapeworms. At the anterior or wider end a conical projection occurs; this supports a round "sucker" or pore by which the fluke holds itself in position. On the lower surface of the organism at the base of this projection is a second or ventral sucker. The sheep fluke is hermaphroditic, i.e., both male and female generative organs are found in the same animal. The male elements develop first and later disappear to make room for the development of the eggs, which finally fill the fluke body completely.
Fig. 1.—(a) Adult liver fluke enlarged five times. Ventral view. (b) Actual size of the fluke shown in (a). Dorsal view. (c) Adult fluke cleared and prepared for microscopical study, showing internal anatomy. × 5.

**LIFE HISTORY**

The adult flukes at rest in the larger bile passages of the liver give rise to myriads of eggs which pass into the small intestine with the bile and are voided with the feces of the infested animals. The eggs are yellowish and thin-walled, contain numerous embryonic cells and are fitted anteriorly with an opereulum or cap to facilitate hatching (fig. 2a). The embryo (fig. 2b) matures very rapidly in warm, moist surroundings, and in the presence of moisture it hatches, liberating a young fluke that is known as a miracidium (fig. 2c).

The miracidia are microscopic in size, a little more than twice as long as wide, and clothed with cilia or hairlike structures that help it to swim about in the water. The minute organisms swim about in search of their next host which, if we are to accept European observations as applicable to American conditions, is a snail of the genus *Lymnaea*. This genus of snails is characterized by its cornucopia-
shape and by having its opening to the right as one faces the opening. Burrowing into the body of the snail, the miracidium settles down in the tissue, usually in the pulmonary chamber. The parasite now becomes encysted and saclike and its contents divide and redvide until the sac or sporocyst (fig. 2d) contains from three to eight separate cylindrical organisms known as rediae.

Fig. 2.—Developmental stages of the liver fluke that take place outside the body of the sheep. (a) Egg shortly after being voided from the sheep's body with the feces; (b) egg ready to hatch, containing the fully formed miracidium; (c) miracidium attacking the body wall of a snail; (d) sporocyst which has developed from the miracidium in the pulmonary chamber of the snail and contains developing rediae; (e) redia from the liver of the snail containing developing cercariae; (f) free-swimming cercaria which has emerged from the snail's body. (Redrawn in part from Thomas.)
The rediae (fig. 2e) now begin to move about and finally rupture the sporocyst wall and migrate to some other organ in the snail’s body, generally the liver. The reproductive cells in the body of each redia now begin to develop and soon each redia gives birth to from eight to ten immature flukes known as cercariae (fig. 2f).

These cercariae (approximately 300 for each original miracidium that entered) leave the body of the snail and, swimming about in water, eventually work their way up blades of grass or other vegetation. Certain cells on the surface of the body produce a tough, parchment-like material which covers the body of the cercaria and glues it to the surface of the vegetation. Here it remains alive as long as a small amount of moisture is supplied by the vegetation upon which it is encysted, or until it is eaten by one of its many hosts, such as sheep, cattle, goats, hogs, rabbits, horses, mules, or even man.

When eaten by its primary host, the cyst is quickly dissolved, and after passage through the stomach the young fluke burrows through the intestinal mucosa, wanders about the body cavity for a time and in the majority of cases finally attacks the liver, burrowing through the capsule and functional tissue until it reaches one of the minute bile capillaries down which it progresses as it grows older and larger. Eventually the bile ducts are reached. Here the fluke comes to maturity and liberates eggs over long periods of time—variously estimated at from six to twelve months—after which, its function in life accomplished, it flows into the duodenum with the bile and is voided from the body of its victim in a partly digested condition.

THE COURSE OF THE DISEASE IN SHEEP AND CATTLE

An infestation of sheep fluke is commonly known among veterinarians as distomiasis, so-called because at the time the term was coined the fluke was known technically as Distomum hepaticum. At present the technical name is Fasciola hepatica, and there are many references in the literature to the disease as fascioliasis.

After gaining entrance to the liver, it is very probable that the small flukes excrete a definite poison which destroys the active liver cells. As the cells break down, areas of dead cells often become cavities, and these, at first minute, gradually become extended until blood vessels are tapped and more or less serious hemorrhages are produced. Sometimes these areas may progress to the surface of the organ and by draining into the body cavity produce acute peritonitis. If the condition does not become serious enough to produce death a
chronic state of inflammation is set up. Scar tissue then forms in much greater profusion than is needed for actual repair, with the result that the bile ducts become greatly enlarged. The abnormal growth of this tissue in the supporting framework of the liver causes a condition known as cirrhosis.

At autopsy, the first point that will be noticed is the swollen condition of the bile ducts, which often stand up like ropey swellings on the surface of the liver. These will be found plugged in many cases by cheesy masses of semi-solid bile. If the bile ducts are found in this swollen condition it is generally possible to find adult flukes present also, but in recovered or convalescent cases all the flukes may have matured and disappeared. In severe cases the liver tissue has the appearance of being decomposed, which gives the name "liver rot." This condition may have progressed from merely slightly discolored areas to the almost complete supplanting of normal liver tissue by masses of blood in various stages of coagulation and decomposition.

A serious condition known as parasitic anaphylaxis is often attributed to fluke infestation in yearlings and aged ewes. The animals affected will drop out of the flock suddenly and stand with lowered head. The respiration will increase rapidly, diarrhea will be present, the temperature becomes sub-normal, and in a few hours the affected animal will die in a short convulsion. On post-mortem examination no abnormal conditions will be found except the presence of minute flukes, scarcely $\frac{1}{8}$ to $\frac{1}{4}$ inch in length in the liver, and old, healed scars of a previous infestation of flukes. It is believed that the shock producing death is caused by the introduction into the blood stream of the toxin or poison produced by the young flukes and its combination or reaction with some substance formed by the previous infestation of flukes. Fortunately this condition is rare.

**SYMPTOMS**

The first indication of infection generally manifests itself about a month after infestation with the cercariae. A tendency to lag behind the flock, a paling of the exposed mucous membranes and a slight edema or watery swelling beneath the jaw and under the eyes are all that may be noted at this time. Curiously enough, the animal apparently gains weight during this period. This is explained by the increased flow of bile caused by the invasion of the parasites. This increased bile in turn facilitates the digestion and assimilation of fats.

Shortly after this first preliminary manifestation, the length of time depending on the severity of the infestation, the gain in weight
stops and a loss is noticeable. The animal becomes more languid, the watery swelling under the jaw becomes more pronounced and pendulous and may be accompanied by an extensive edema under the eyes and along the belly. The so-called "black" diarrhea may be present. The animal entirely loses its appetite. Fatal cases terminate apparently in exhaustion. The duration of the disease is exceedingly variable but very rarely exceeds six months, death or recovery having ensued in the meantime.

**TREATMENT**

A thorough discussion of the different "cures" that have been suggested from time to time would be too lengthy for a circular of this kind; consequently a statement of the methods known to be of economic importance is all that will be attempted.

**Male Fern.**—In 1884, Grassi and Calandruccio, two Italian investigators, reported favorable results from the use of male fern in sheep affected with liver flukes. Several years later patent preparations of male fern appeared in many European countries under trade names and since that time the literature is filled with references to male-fern treatment under the disguises of "Filmaron," "Fasciolin," "Avisciolina," "Danistol," and "Distol"—all male-fern extractions containing approximately 22 to 26 per cent filicine, the active agent in the male-fern preparations.

Extensive trials of male-fern extract in the British Isles have shown it to be effective against adult flukes and to afford adequate treatment in cases where the infestation was not too heavy. In these latter cases death was sometimes caused in lambs by the hordes of immature flukes too small to be reached by the male-fern treatment.

The recommended method of treatment is to administer 4 grams (1 teaspoonful) of extract of male fern containing 25 per cent filicine in 10 cc (21/2 teaspoonfuls) of nonpurgative oil on five successive mornings at least two hours before the animal is allowed to feed.

**Kamala.**—Crude kamala had been used for many years as a general vermifuge. Its rated efficiency was not high, however, on account of the small dosages of the crude drug generally recommended. After the advent of male fern considerable impetus was added to its sale and use by the work of Marek, who reported very favorably on its use as compared with other types of treatment. It was recommended by Hall that the crude drug be given as follows: "To yearlings and older sheep give 15 grams divided into two doses of 7.5 grams (2 drams) each, and given at 12- to 24-hour intervals. The entire
15 grams may be given in one dose to a strong animal or divided into five doses for weak ones. After treatment sheep are dull for 3 to 5 days, they lie down a great deal, eat little or may stop eating for a day or two, and have a diarrhea. The flukes are said to die in 3 to 8 days.'"

*Carbon Tetrachloride.—* In 1921 Hall of the United States Department of Agriculture proposed the use of carbon tetrachloride (CCl₄) as an anthelmintic for intestinal roundworms. In 1926 Montgomerie in Wales reported that carbon tetrachloride was an efficient agent in the destruction of adult flukes. Doses of one-half cubic centimeter administered in capsules effected a complete eradication of adult worms. Later work led to the general recommendation of the administration of one cubic centimeter for all sheep. Only the pure drug is used, since the commercial grade is far less effective. Seddon in New South Wales has administered the dosage of one cubic centimeter diluted with four cubic centimeters of liquid petrolatum as a drench, but the experience in this country is limited to the capsule method of administration.

Soft gelatin capsules each containing one cubic centimeter of the carbon tetrachloride are now procurable. These are administered by means of a capsule "gun" or long forceps. No preliminary fasting is necessary. Shaw and Sims in Oregon report very satisfactory results with carbon tetrachloride for liver-fluke infestation in goats and have since extended their work to sheep with highly efficient results. Thousands of sheep have now been treated in Oregon and California and no word of failure has been published. The simplicity of treatment and its apparent success leave little to be sought in the way of treatment. It can be administered throughout pregnancy with safety. Tetrachlorethylene (C₂Cl₄) which has largely supplanted carbon tetrachloride for intestinal worms is not effective for flukes.

One of the main contraindications for administering carbon tetrachloride to human beings has been the presence of liver lesions. Consequently it was with some doubt that the drug was first used in a sheep disease where liver lesions are the most prominent defects noticeable. However, the results of treatment have shown no untoward effects in sheep, although cattle are reported to show toxic results in some cases when treated with this drug for liver fluke.

The answer to this variability was doubtless explained in Minot's work with dogs. He found in dogs with a low calcium content, that approximately twelve hours after administration of carbon tetrachloride the animals died with all the symptoms of infantile tetany caused by lack of calcium. Various bile pigments such as bilirubin
are known to combine with calcium in the blood; in fact, this is a normal procedure wherein the calcium in the blood protects the body from excess of bile. However, calcium when combined with bilirubin is no longer available to the body, and if there is no ionized or free calcium available the animal suffers from calcium deficiency and dies in tetany. It is also known that the administration of carbon tetrachloride produces an abnormal amount of bilirubin in the blood. He was able to restore dogs to a normal condition in a few moments by injecting calcium chlorid intravenously. Cattle are apparently drained of their available calcium far more readily than are sheep, and consequently the bilirubin poured into the blood stream by the action of the drug combines with the small available supply of calcium more completely than is the case with sheep that apparently have a larger reserve of calcium. It is possible that sheep pastured on stubble and hay pasturage free from legumes might be reduced in calcium to the point where carbon tetrachloride would be toxic. Attempts to produce this condition artificially are now being conducted at this station.

To summarize the recommendations for treatment, it appears that sheep may be treated for liver fluke with safety and efficiency by the administration of one cubic centimeter of carbon tetrachloride in capsules, observing the necessary care not to break the capsule in the mouth, where the fumes could be inspired. Calcium deficiency is not as important in sheep as in cattle. The latter are susceptible to calcium-deficiency tetany following treatment with carbon tetrachloride and should be treated with care by a veterinarian and only after reinforcing their feed with assimilable calcium such as marl for at least a week. In treating sheep or goats that have not had access to pasturage or feed containing legumes, such as bur clover, alfalfa, or other source of calcium it is well to make a preliminary treatment, under the observations of a veterinarian, of a few trial animals.

**PREVENTION**

"'Permanent pastures perpetuate pernicious parasites'" is a jingle that every flockmaster should take as his litany and heed as no other great truth of his industry. In the days of the wide ranges the infestation of the pasture with parasitic eggs and larvae was so scattered that the parasites that obtained a host were few, and infestations of severe nature were consequently very scattered. With our present practice of small permanent ranges, which are often overstocked, the infective forms of the parasite become concentrated in
relatively small areas, and it is almost impossible to avoid infestation of the host animals. Infestation probably occurs throughout the year in California, although late summer seems to be the commonest time.

Attempts have been made to rid the ranges of snails by adding copper sulfate (bluestone) to the standing water present at the rate of one part of the sulfate to a million parts of water (one ounce to 7,800 gallons) and repeating at the end of a month to kill those snails that have hatched in the interval, but this is impractical under most California conditions.

Some owners have decreased the amount of infestation remarkably on relatively dry ranges by fencing off the marshy areas and supplying water in elevated troughs. Wherever this procedure is practical it pays large dividends, because not only fluke but stomach-worm, lung-worm, and intestinal-worm infestations have their origin in marshy areas and stagnant pools.

STOMACH WORMS

California sheepmen have congratulated themselves for years that "stomach worms," probably the greatest single enemy of sheep in the United States, was, for some unknown reason, absent from California. For several years reports of infestations that were indicative of stomach worms have been received, but no actual specimens were identified at the Experiment Station from California until 1927, when specimens from four separate localities were found to be true stomach worms. Since that time numerous reports verified by competent veterinarians have been received, indicating that this infestation is becoming a common source of trouble in California.

THE PARASITE

The stomach worm of sheep, *Haemonchus contortus*, is a slender, hairlike roundworm found in the fourth stomach. The females are approximately one inch in length with a backward-directed flap covering the genital opening, which is located one-fifth the length of the worm from the tail end. The males are shorter ($\frac{1}{2}$ to $\frac{3}{4}$ inch) with two rounded lobes at the posterior end of the body, which are strengthened by six fingers or ribs in each; in addition, the right lobe bears at its base a small accessory lobe with a bifurcated rib. The females have a peculiar twisted arrangement of the genital tubes which shows through the integument, giving a spirally-banded effect.
The general color of these parasites ranges from a pure white to blood red, the average specimens appearing pinkish as they are seen at autopsy.

The stomach worm of cattle, *Ostertagia ostertagi*, has been reported from sheep and confused with the sheep stomach worm. The former is brown, not over one-half inch in length and hairlike in thickness.

**LIFE HISTORY**

Adult females in the fourth stomach of the sheep produce eggs, which pass to the exterior with the feces of the animal. In the presence of water these eggs develop and hatch as motile larvae, after which, without growing larger, their skin is molted but is not cast off, thus producing the so-called 'ensheathed larva,' which is the infective stage of the parasite. The time necessary for the production of this stage ranges from approximately three days at 95° F to three weeks at 50° F. The ensheathed larva may float about in stagnant water or crawl up on grasses to await the time when it is eaten by another host.

Sheep are the normal hosts, but goats and cattle have also been found to be infested.

**COURSE OF THE DISEASE**

Infested animals exhibit the same general symptoms seen in fluke disease, except that there is no preliminary gain in weight. The emaciation is progressive, the mucous membranes become pale, edema is noticeable in the ‘bottle-jaw’ swelling under the jaw, and the animal refuses to eat, dying in a condition of extreme emaciation.

At autopsy in cases of heavy infestation the entire contents of the fourth stomach may appear to be in writhing motion from the wriggling of the worms. In lesser infestations it is sometimes necessary to flood the walls of the organ with clear water after washing over the contents to detect the parasites adhering to the mucosa. In nearly every case the tiny, bright red pin points on the mucosa (lining of the stomach) will indicate the presence of these parasites because they change their feeding points at frequent intervals, leaving these marks as evidences of their points of attachment.
TREATMENT

A one-per-cent solution of copper sulfate (bluestone) in water (¼ pound to 3 gallons) administered as a drench in doses of 100 cc (approximately 3 ounces) to yearlings and older sheep and 50 cc to lambs over three months of age forms a very satisfactory treatment. In heavily infested territory this must be repeated every month as long as the mean temperature remains above 50° F. Use only bright blue crystals, discarding any that have turned white. Crush these and add to some of the water that has been heated. When completely dissolved add the remainder of the water. Do not use exposed-metal containers. Allow three gallons of the solution for each hundred adult sheep.

The actual administration is most easily accomplished with a rubber tube, enamel-ware funnel and a small cup holding a single dose. The funnel is fitted into the outer end of the tubing to receive the dosage. The end which is placed in the sheep’s mouth may be fitted with a piece of metal tubing or passed through a wooden block 1 in. × 4 in. to prevent the animal from biting down on the flexible tubing. The tubing is often stapled to a post at a convenient height with enough left free at its lower end to facilitate treatment. By dipping a cupful (choose a small cup holding 100 cc or mark the 100 cc volume on a larger cup) from an open container and pouring it into the funnel after the tubing has been adjusted into the mouth of the animal considerable speed may be developed in treatment. Outfits which depend on passing the solution through a graduated glass cylinder by means of pinchcocks are perhaps slightly more accurate where many lambs are included in the treatment.

In treating, allow the animal to stand in a normal position with its head parallel with the ground. Do not tilt the muzzle up as this may prevent the animal from swallowing and allow the solution to enter the windpipe.
PREVENTION

Marshy swales, seepage areas, or stagnant water in some form are absolutely essential to the growth of the parasite from the time the egg leaves the sheep’s body until it becomes an ensheathed larva. The sources of infestation are more apparent under California conditions than elsewhere on account of the lack of summer rainfall. It is self-evident that if sheep could be restrained from frequenting such areas by fencing them off, reinfestation would be prevented automatically. If this is done and drinking water supplied in elevated troughs the latter must, of course, be so constructed that they do not overflow and produce a muddy bog.

In many cases this procedure is impractical and pasture rotation becomes the only available procedure. At temperatures below 50° F a change of pasture every month is sufficient, but as the temperature rises it is necessary to move more frequently until at 95° F it is possible to use the same area only three or four days. Put the lambs on the cleanest pasture as they are more seriously affected than the older animals.

Cultivation apparently destroys the infective larvae. Cattle should not follow infested sheep, as they too are subject to infestation. Hogs and horses are apparently not affected.
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