Internal Parasites of Sheep and Goats

BY G. DIKMANS AND D. A. SHORB

AS EVERY SHEEPMAN KNOWS, internal parasites are one of the greatest hazards in sheep production, and the problem of control is a difficult one. Here is a discussion of some 40 of these parasites, including life histories, symptoms of infestation, medicinal treatment, and preventive measures.

WHILE SHEEP, like other farm animals, suffer from various infectious and noninfectious diseases, the most serious losses, especially in farm flocks, are due to internal parasites. These losses result not so much from deaths from gross parasitism, although fatalities are not infrequent, as from loss of condition, unthriftiness, anemia, and other effects. Devastating and spectacular losses, such as were formerly caused among swine by hog cholera, among cattle by anthrax, and among horses by encephalomyelitis, seldom occur among sheep. Losses due to parasites are much less sensational, but they are constant, and especially in farm flocks they far exceed those due to bacterial diseases. They are difficult to evaluate, however, and do not as a rule receive the attention they deserve.

The principal internal parasites of sheep and goats are roundworms, tapeworms, flukes, and protozoa. Their scientific and common names and their locations in the host are given in table 1.

Another internal parasite of sheep, the sheep nasal fly, the grubs of which develop in the nasal passages and head sinuses, is discussed at the end of the article.

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<td>Globidium getui</td>
<td>Wall of fourth stomach and small intestine.</td>
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1 No common name.

**GEOGRAPHICAL DISTRIBUTION**

While parasites may be found wherever sheep and goats are kept in the United States, they occur with the greatest frequency and are of greatest importance in the farm-flock area as distinguished from the western or range area. The farm-flock area lies roughly east of a line drawn from the western border of North Dakota to the Gulf of Mexico in the vicinity of Corpus Christi, exclusive of South Dakota. According to estimated figures of the Agricultural Marketing Service of the Department of Agriculture for 1940, there are in this area about 14,659,000 stock sheep and lambs, of which 7,255,000, or roughly 50 percent, are found in Ohio, Missouri, Iowa, Kentucky, and Michigan. Parasitism is worst under farm conditions because as a rule sheep in farm flocks remain concentrated on smaller areas for longer periods.
than under range conditions. The climatic conditions prevailing in this area are also more favorable for the development of worm eggs on pastures and the maintenance of pasture infection than are those prevailing in the range States.

In addition to the sheep in the farm-flock area, there are, according to Baker (3), about 500,000 goats, the majority of them being located in southeastern Georgia, central Tennessee, and southern Missouri.

**GENERAL FEATURES OF LIFE CYCLES**

The life histories of all the common roundworms of sheep and goats, with the exception of *Muellerius capillaris, Elaeophora schneideri, Gongylonema pulchrum*, and *G. verrucosum*, which are discussed elsewhere in this article, are direct; that is, no intermediate hosts are required for their development. Tapeworms, flukes, and some of the parasitic protozoa, on the other hand, require intermediate hosts for the completion of their life cycles, details of which will be given separately under the discussions of the particular parasites.

Roundworms reproduce by means of eggs. With the exception of those of lungworms, which hatch in the lungs (the larvae being discharged with the feces), the eggs of the roundworms parasitic in sheep and goats pass out in the droppings of infected animals. With a few exceptions, which will be noted in the discussion of the parasites to which they apply, the eggs develop on pasture into infective larvae in periods varying from a few days to a few weeks, depending upon conditions of temperature and moisture. In warm weather the eggs hatch in a few hours. If the temperature is below 40° F., the eggs remain dormant; if it is below freezing, the vitality of many of the eggs is eventually destroyed. Dryness also destroys many eggs, while moisture favors their development. The larvae as they hatch from the eggs are very susceptible to freezing and drying. In very warm weather they complete their development in 2 to 4 days, provided enough moisture is present. In cooler weather the time required for development is longer; at temperatures below 70° F., 10 days to several weeks may be necessary. Neither the eggs nor larvae in the early stages develop when ingested, or taken into the body, by sheep and goats; only when the larvae have reached the third stage do they become infective to their host. Hence this is known as the infective stage.

In the infective stage the larvae of many different species of these worms migrate to the stalks of grass or to other plants, becoming active whenever the air is saturated with moisture, as from rain, fog, or dew. When the air becomes dry and the moisture evaporates from the grass, the young worms cease their activity, and they resume their migrations only when the air again becomes laden with moisture. Unlike the eggs and the larvae in the early stages, larvae that have developed to the infective stage are able, as a rule, to survive long periods of freezing and dryness. They reach maturity and begin producing eggs about 2 to 6 weeks after they are taken up by their host.

*Italic numbers in parentheses refer to Literature Cited, p. 900.
SYMPTOMS OF WORM INFECTION

The general symptoms of worm infection are unthriftiness, loss of weight, diarrhea, anemia manifested by paleness of the visible mucous membranes of the eyes and mouth, development of potbelly, or enlargement of the abdomen, and often a soft swelling under the jaw, referred to as bottle jaw or poverty jaw. Since animals showing such symptoms are commonly infested with several species of parasites, it is impossible to determine from observation which species are responsible.

Experiments undertaken to determine the effects on sheep and goats of infections with single species of worm parasites have shown that animals infected only with the twisted stomach worm, *Haemonchus contortus*, show a marked anemia, similar to that produced by bleeding, caused by hemorrhage into the stomach. No diarrhea occurred. Experimental infection with small trichostrongyles, *Trichostrongylus* species (1), produced a marked, and in some cases prolonged, watery diarrhea. The infected animals appeared gaunt and depressed, showed evidence of abdominal pain, and refused to eat. Animals dying of this infection were emaciated, and the carcasses showed evidence of dehydration, or loss of water, resulting from the continuous diarrhea. There was little or no evidence of anemia. In Australia the diarrhea caused by infection with these small trichostrongyles is referred to as black scours because of the dark color of the feces passed by the infected animals. This black color, which was not observed in experimentally infected sheep and goats, may be due to the fact that the animals were on pasture or perhaps to blood in the feces resulting from a simultaneous infection with *H. contortus*.

Sheep experimentally infected with *Cooperia curticei*, another member of the group of worms commonly referred to as small intestinal trichostrongyles, did not show any serious clinical effects (2).

Symptoms shown by animals infected with hookworms, *Bunostomum trigonocephalum*, are similar to those generally found in hookworm infection in other animals.

While the twisted stomach worm, *Haemonchus contortus*, is the most common stomach worm of sheep and goats in the eastern half of the United States, the medium stomach worms, *Ostertagia* species, appear to be the most prevalent forms found in some areas in the western part of the country. The symptoms attributed to infection with these worms are the same as those attributed to infection with *H. contortus*, but they do not appear to develop as rapidly or to be quite so marked. Experimental infections with *Ostertagia circumcincta* (51) did not produce any marked clinical symptoms except in some animals to which enormous numbers of infective larvae had been administered.

Symptoms shown by animals infected with nodular worms are diarrhea, abdominal pain, and loss of flesh. These symptoms occur chiefly during the time that the larvae penetrate into and remain in the wall of the intestine.

Opinions differ as to the effect on sheep and goats of infection with the thread-necked strongyles, *Nematodirus* species. Such experi-
mental evidence as is now available (29) appears to indicate that these worms cannot be considered as producing any noticeable symptoms in infected animals.

Experimental infections with Chabertia ovina (18, 28) indicate that these worms may be responsible for symptoms of severe bloody diarrhea during their development to adult worms in the host. The effect of infection with adult worms has not been definitely determined.

PROTOZOA

A large number of protozoa have been reported as parasites of sheep and goats, but only a few of these are known to be harmful. They are all of minute size, too small to be seen with the unaided eye.

Coccidia

A number of species (7) of coccidia, all belonging in the genus Eimeria but varying greatly in size and certain other characters of form and structure, have been reported as parasites of sheep and goats. They occur in the wall of the small intestine and cause a disease known as coccidiosis.

The resting, or spore, form of these parasites, which is the form usually found in microscopical examination of fecal specimens, is ellipsoidal or ovoid (football-shaped).

The life history of these parasites is very complicated. The resting, or spore, forms, known as oocytes, pass out of the body with the feces. Under favorable conditions (8) four bodies known as sporocysts develop within the oöcyst. Within each sporocyst two sporozoites develop, so that at maturity the oöcyst contains eight sporozoites. This is the infective stage of the parasite. When taken up by a susceptible animal with feed or water, these sporozoites are liberated in the intestine and invade the intestinal wall. The preferred site in which the parasite undergoes its development is in the cells composing the inner lining of the small intestine. Here the parasites multiply, at first asexually and later sexually. At the beginning of an infection the asexual cycle is more prevalent, whereas later the sexual forms are more frequently found. The sexual reproduction results in the formation of oocytes, which pass out of the body with the feces and, after maturing outside the body, become infective to a new host.

Little is known concerning the occurrence and actual importance of coccidial infection among farm sheep in the United States. Examinations made in 1937 by the Zoological Division of the Bureau of Animal Industry at Beltsville, Md., of over 100 miscellaneous adult ewes and lambs revealed that all of them were infected with coccidia, thus confirming similar observations made in other parts of the country and showing that healthy adult sheep are carriers of coccidia. During this study the course of coccidial infections was followed in 9 spring lambs by means of frequent fecal examinations from shortly after birth through the following several months. All 9 lambs acquired infection early, as shown by heavy oöcyst production beginning
during the fifth and sixth weeks of life. In 5 of these animals that were followed until they were 7 months old, the infection remained relatively high for 6 to 8 weeks after the oöcysts first began to appear in the feces, then declined and remained at a low level during the summer months. These apparently heavy infections caused no noticeable inconvenience to the animals other than mild scouring during the first few days of heaviest oöcyst discharge.

It appears likely that as a result of the intimate relationship between young lambs and ewes, all normally reared lambs acquire coccidial infection at an early age from oöcysts discharged by the adult carriers, but as a general rule thrifty, well-nourished lambs survive these early attacks without suffering any ill effects, and the infections do little or no harm to the great majority of the animals.

According to Thorp (50), severely affected animals (fig. 1) become dull and lethargic, lose their appetites, and suffer from diarrhea, the discharges often becoming bloody. The diarrhea may continue for several days, and in some animals for several weeks, causing weakness, loss of flesh, and death. Enteritis (inflammation) may be present in both the small and large intestines. The diagnosis rests on the demonstration of coccidia in the feces of affected animals.

The disease as it occurs in feed lots has been described by Thorp (50), Deem and Thorp (15), and Christensen (9). According to these authors virtually all of the lambs are discharging some coccidial

Figure 1.—Sheep affected with coccidiosis.
oöcysts when they arrive at the feed lots. When the conditions are favorable, clinical symptoms of coccidiosis begin to manifest themselves about 2 weeks later. Deem and Thorp (15) reported that in a number of outbreaks most of the cases occurred between 2 and 3 weeks after the lambs were placed in the lots.

Christensen (9), studying an outbreak of this disease in a group of approximately 16,000 lambs, states that death losses from coccidiosis, together with widespread scouring and heavy oöcyst discharge in the feces, began 12 to 16 days after the lambs had been placed in the feed lots. Heavy death losses continued for about 2 weeks, after which they gradually subsided, but many animals continued to suffer from diarrhea for several weeks. During this outbreak 540 lambs, constituting 3.4 percent of the total number under observation, died as a result of coccidiosis, and 1,563 required special care and feeding because of emaciation resulting from excessive scouring.

Christensen demonstrated that in the particular case under consideration the source of infection was in the lambs themselves and not in the old, previously contaminated feed lots.

During this investigation Christensen noted that the corn silage that was used as part of the ration became heavily contaminated with sheep feces and that it was customary to replenish the supply in the feed troughs twice a day by adding fresh material to the uneaten portions of the previous feed. No attempt was made to clean out the troughs by removing unconsumed feed, and there was a gradual accumulation of uneaten silage beneath the fresh additions. Whole and fragmented fecal pellets containing coccidial oöcysts were found scattered throughout this silage in the feed troughs. Because of its high moisture content, the oöcysts found favorable conditions for sporulation, or spore production, in the silage.

Christensen concluded that in this case the contaminated corn silage was the principal source of the infective coccidia responsible for the enormously increased numbers of oöcysts found in the lambs 2 weeks after they were placed in the feed lots and for the severe clinical cases of coccidiosis in the group.

Coccidia occur in practically all lambs and sheep examined and under ordinary circumstances apparently cause little or no disturbance. The factors or combination of factors responsible for severe outbreaks of coccidiosis are not well known. The extent to which external factors, such as abrupt changes in feed or sudden marked changes in temperature, influence the development of coccidiosis by lowering the resistance of the animal is not known. Whether in outbreaks in feed lots the ingestion of increased numbers of oöcysts is the principal factor or whether the change of feed is a contributing factor of major importance remains to be determined.

There is no specific treatment for coccidiosis. Christensen (9), however, reports that the daily administration of small amounts of a combined copper sulfate and ferric sulfate solution over a period of 5 weeks from the beginning of confinement in feed lots significantly lowered the discharge of coccidial oöcysts in one group of healthy lambs as compared with that in another group not receiving such treatment. While this test suggests the possibility that there may be
some benefit in the use of these chemicals as preventives, there is abundant evidence to indicate that in the actual treatment of the disease, these and other chemicals are of no value.

GLOBIDIUM GILRUTHI

Another protozoan parasite, Globidium gilruthi, occurs in the fourth stomach and small intestine of sheep. According to Wenyon (55) these parasites, which are probably related to sarcosporidia, occur as sickle-shaped spores enclosed in membranous cysts or sacs, in the wall of the fourth stomach and small intestine. These cysts appear as little white opalescent elevations of the mucous membrane. Within the mature cyst is a mass of spores. One end of the cyst is blunt and the other pointed.

The infection is not uncommon in sheep, but usually only a few cysts are present, and no clinical symptoms or disease condition referable to the infection can be noted. In some cases, however, the infection is heavy, and according to Marsh and Tunnicliff (33) such infections may cause a severe enteritis, or intestinal inflammation, manifested by pronounced diarrhea and accompanied by the usual depression and loss of weight. There is no fever, and no blood appears in the feces except where there is a hemorrhagic condition of the rectum. Since nothing is known of the life history of the parasites, no recommendations for prevention or control can be made, and medicinal treatment has, so far as is known, not been attempted. It is possible that the administration of intestinal astringents such as those used by Christensen (9) in outbreaks of coccidiosis may have some beneficial effects.

FLUKES

Sheep and goats in the United States may be infested with one or more species of flukes, which are flat, leaflike parasites. Two kinds of flukes may occur in the liver, and two other kinds are found in the rumen, or paunch. Flukes reproduce by means of eggs, which pass out with the feces and hatch in water, releasing ciliated embryos—that is, embryos equipped with hairlike structures for moving around—known as miracidia. The miracidia penetrate certain species of snails and develop into cercariae (flukes in the larval stage), which leave the snails and encyst on vegetation. Encysted cercariae are ingested by sheep and goats in grazing and develop into mature flukes.

THE COMMON LIVER FLUKE

The common liver fluke, Fasciola hepatica, found in the liver, bile ducts, and gall bladder of sheep and goats is a flattened, leaflike,

3 At post mortem examination of an animal dying from the infection, these authors found the small intestine studded along its entire length with small white cysts, some on the surface of the mucous membrane and others deeply embedded in it. The mucous membrane was hyperemic and had a somewhat furry appearance. In another case the infection was found to be greatest in the posterior portion of the small intestine, the cysts being less than 1 millimeter apart. Microscopic examination of affected areas of the small intestine showed large numbers of Globidium cysts, in different stages of development, in the mucosa, destruction of the epithelium of the villi, and infiltration of the tissue by large numbers of monocytes and lymphocytes.
brown worm, usually about an inch long. There is a sucker on a cone-shaped extension at the anterior, or front, end, and just behind this is a ventral sucker. Through the skin, or cutícula, the branching intestine and the uterus filled with eggs can be seen.

The cercaria, which develops within a snail (fig. 2), resembles a small fluke and is provided with a tail by means of which it swims about. It finally loses its tail and encysts, or becomes enclosed in a cyst. The encysted cercariae may float about on water or attach themselves to grass blades or other vegetation. When swallowed by sheep or other suitable host animals, the larval flukes, on reaching the small intestine, bore through the wall to the body cavity. Here they wander over the surface of the viscera and walls of the body cavity, finally perforating the capsule of the liver and reaching the extremities of the biliary (bile) ducts. In the liver the young flukes grow to maturity in the larger bile ducts.

The distribution of the common liver fluke is world-wide wherever low, wet pastures and the suitable intermediate snail hosts occur. In the United States it is most prevalent on the west coast and in the Rocky Mountain States, the Southwest, and the Southeast.

The symptoms associated with liver fluke infection are, in a general

![Figure 2](image-url)

_Figure 2.—Snails serve as intermediate hosts of the liver fluke, _Fasciola hepatica_. These snails were on the ground near a temporary pool in a herd pen in Texas. (Photograph by O. W. Olsen, Angleton, Tex.)_
way, similar to those attributed to other worm infections—unthriftness, anemia, etc. According to Clunies Ross and Gordon (12), in acute cases resulting from massive invasion of the liver by large numbers of young flukes, death may occur after an illness of only a few days and without any symptoms that can be definitely attributed to fluke infection. Sheep suffering from chronic fluke disease appear languid and are easily fatigued when driven. When such animals are examined the visible mucous membranes will be found to be pale and anemic. As the disease progresses the gait becomes stiff, the loss of condition becomes more marked, and dropical (edematous) swellings develop under the jaw. According to Shaw and Simms (42), these swellings, commonly called bottle jaw, occur in sheep suffering from infection with immature flukes as well as in those affected with chronic fluke disease. These authors also report that the distention of the abdomen resulting from the accumulation of fluid and commonly referred to as potbelly (fig. 3) develops during the early stages of the infection when parasites are present in large numbers. The condition later disappears and is not present when the parasites have developed to maturity.

The lesions found in the sheep at post mortem examination depend upon the developmental stage of the fluke. If the infection is heavy, there is considerable damage to the liver from penetration by the immature flukes. This penetration leads to hemorrhage into the abdominal cavity and an accumulation of fluid within it. Acute inflammatory changes occur in the liver substance, and the liver becomes enlarged owing to engorgement with blood. As already noted, infected animals may die as a result of massive invasion of the liver by young flukes without showing any marked symptoms attributable to fluke infection. After the flukes become mature in the bile ducts, these ducts become greatly thickened as a result of continuous irritation. In advanced cases the ducts stand out prominently and, owing to a deposit of calcium salts, become hard and gritty, as may be observed on cutting them.

The Large Liver Fluke

The large American liver fluke, Fascioloides magna, is found in the tissues of the liver, commonly in cysts which usually contain, in addi-
tion to the flukes, a quantity of dark-colored fluid and debris. The fluke may attain a length of 7 centimeters (2¾ inches). The large liver fluke resembles an overgrown specimen of the common liver fluke except that the anterior, or front, sucker is not carried on a distinct cone. The life histories of the two are essentially the same. The same snails serve as intermediate hosts for both, and their development in the snail is the same.

So far as known, deer and sheep are the principal propagators of the large liver fluke. It appears to be indigenous to North America and is principally a parasite of deer, but specimens have been collected from sheep in Montana and Idaho.

According to Swales (48, 49), this fluke infects cattle as well as deer and sheep, but in cattle the parasites become encapsulated, or enclosed in a capsule, by the fibrous tissue formed around them through the reaction of the tissues. This prevents undue damage to the liver and the entrance of the eggs into the bile ducts, and it also precludes the continuance of the life cycle of the parasite. In deer a balance appears to have been established between host and parasite, the host reaction being adequate to prevent undue tissue damage but not to prevent reproduction. In sheep severe damage results from the almost unchecked migration of the parasite in the liver tissue. Sheep are the only hosts in which this parasite produces a severe clinical disease. Experimentally infected sheep became greatly emaciated and very weak, even though only a few flukes were found in the liver at post mortem examination.

The symptoms of infestation by the large liver fluke are similar to those associated with infection by the common liver fluke, and the control measures for the latter, discussed later, also apply to the large liver fluke. In addition, however, sheep should be prevented from grazing on snail-infested land occupied by deer.

**THE RUMEN FLUKES**

Two small flukes, *Paramphistomum cervi* and *Cotylophoron coto-
lophorum*, one-fifth to one-half inch in length, are found in the rumen, or paunch, usually near the opening into the reticulum, or honeycomb stomach.

The bodies are pinkish when alive. They are convex on the back (dorsally) and slightly concave on the under side (ventrally). Shaped more or less like a cone, they are broad and blunt at the hind end and rather pointed at the front end.

The life history of one of these flukes, *Cotylophoron coto-lophtorum*, has been studied in detail (5) and found to be essentially the same as that of the common liver fluke, the same snails serving as inter-
mediate hosts of both.

These flukes have been reported from sheep in Australia, South Africa (32), and India, but they occur most commonly in cattle. There is no record of their occurrence in sheep under natural conditions in the United States.

The adult parasites appear to be relatively harmless, and no defi-
nite lesions, or tissue injuries, have been ascribed to them. It has
been reported, however, that in the immature stages they are definitively disease-producing. The symptoms described are very similar to those of roundworm infestation and the lesions occur chiefly in the small intestine.

Since snails act as intermediate hosts of these parasites, the methods of prevention later advocated in the case of the liver fluke may prove to be beneficial.

TAPEWORMS

Sheep and goats in the United States may harbor both adult and larval tapeworms. The adult tapeworm has a head provided with four suckers and a body consisting of a number of flat segments joined together to form a chain. Tapeworms produce eggs of microscopic size which pass out in the feces of the host animal, together with gravid segments (segments containing eggs), which, when dry, liberate more eggs. On being ingested by a suitable host the eggs develop into an intermediate stage—a larva known as a cysticercoid. Sheep and goats become infested by swallowing such larvae.

The larval tapeworms that occur in sheep and goats are commonly known as bladder worms; they are usually located in the tissues or cavities of the body. The bladder worm consists of a head and neck inverted in a membrane containing a clear fluid, the whole structure resembling a bladder. When a bladder worm is eaten by the final host, the head is extruded, or pushed out of the membrane, and passes to the small intestine of the host, where it develops into an adult worm by the growth of segments back of the head.

THE MONIEZIAS

Two species of tapeworms, Moniezia expansa and M. benedeni—long, flat, ribbonlike worms, which sometimes attain a length of several yards and a breadth of three-fourths of an inch—are known to occur in the small intestines of sheep and goats.

The eggs and segments containing eggs pass out with the feces onto the pasture. According to Stunkard (47) the eggs contained in the gravid segments are expelled as the segments dry. On the grass of the pasture the eggs are accessible to certain free-living mites known as oribatid mites and also as beetle mites, which serve as intermediate hosts of the tapeworms. The eggs contain six hooked embryos known as oncospheres. In the mite the oncospheres develop into cysticercoids, or tapeworm larvae, in 15 to 16 weeks. Field experiments indicate that during the summer this development may be completed in 6 to 8 weeks.

Large numbers of different species of oribatid mites may occur on pastures, but only a few of these, all belonging in the genus Galumna and present on the grass during the cooler parts of the day and on warm, cloudy days, serve as intermediate hosts of the sheep tapeworm. During periods of intense sunlight and when strong,
drying winds prevail, the mites leave the grass and are found at the grass roots and in the soil. Saturation of the soil due to heavy or continued rains causes the mites to migrate onto the grass. They may be found throughout the year, but according to Krull (31) in the vicinity of Beltsville, Md., they are most abundant during April and May.

The tapeworms of sheep are cosmopolitan in distribution. They are commonly believed to be serious parasites of sheep, and various conditions, such as unthriftiness, loss of weight, anemia, and digestive disturbances manifested by diarrhea, have been attributed to tapeworm infection. The reasons for this belief are largely indirect but quite obvious. Tapeworm segments are often found singly or in strings of various lengths in the droppings of sheep running on pastures and showing clinical evidence of parasitic infection. On post mortem examination of such sheep, tapeworms are usually found in the small intestine, and the condition of the animals before death is naturally attributed to the tapeworms. The presence of other parasites, particularly small roundworms, is usually not taken into consideration.

Through experimental infection of lambs with tapeworms (Moniezia expansa) only, uncomplicated by roundworm infection such as is commonly found under natural conditions, Shorb (43, 44) has shown that the effects of the former have been generally overrated. The principal effects observed were a slight intestinal disturbance, manifested by the passage of softened feces instead of the usual formed pellets, and a slight retardation of growth. The severity of the effects depends apparently on the age and size of the infected animal and the number of tapeworms present. Young, undersized lambs from 1 to 3 months of age are more seriously affected by a given number of tapeworms than well-grown, vigorous lambs about 6 months old, and in lambs of comparable ages and sizes those harboring the greater number of tapeworms showed the more marked effects.

Under natural conditions lambs become infected with tapeworms at an early age, and this infection, added to the roundworm infections usually acquired at the same time, may result in serious retardation of growth.

No recommendations as to prevention can be given until detailed information on the life history of the intermediate host has been obtained.

**The Fringed Tapeworm**

Another tapeworm, which appears to be confined to range sheep, is known as the fringed tapeworm, Thysanosoma actinioides. It derives its common name from the fact that each of the segments has a fringe on its posterior, or rear, border, which distinguishes it from all other tapeworms. If the segments are put in water, the fringes float out and can be easily seen.

The fringed tapeworm is a parasite of western sheep and is found in the East only when sheep shipped from the West are slaughtered at eastern abattoirs.
On post mortem examination of infected animals the tapeworms are found in the small intestine, the bile ducts, the gall bladder, and sometimes the pancreatic duct. At times they may be numerous enough to apparently occlude, or close up, these ducts, and many serious pathological conditions and marked clinical symptoms, based largely on deductions from the post mortem findings, have been attributed to them. After examining a number of infected sheep and comparing their condition with that of similar uninfected sheep, however, Christensen (10) came to the conclusion that, at least so far as adult sheep are concerned, no serious effects can be ascribed to this tapeworm.

The life history of the fringed tapeworm is completely unknown, and until it has been ascertained and pure infections have been established, no definite statement concerning its effects on sheep are warranted.

At the present time the most serious loss resulting from infection is due to the fact that under Federal meat-inspection regulations, all sheep livers found on post mortem examination to be infected are condemned as unfit for human consumption.

**BLADDER WORMS**

The bladder worms found in sheep and goats, of which there are four kinds, are the larval or immature stages of tapeworms found in dogs and related carnivores. Their names and usual locations are given in table 1, page 860.

Sheep and goats become infected with these bladder worms by ingesting the eggs voided by dogs and related carnivores on pasture or range.

**The Thin-Necked Bladder Worm**

The thin-necked bladder worm, *Cysticercus tenuicollis*, has the appearance of a large sac filled with clear fluid. A white object, which is the head or neck, projects into it at one end. The bladder is usually about 1 inch in diameter and may attain a length of several inches. The bladder worm proper is surrounded by a cyst wall which is developed by the host animal as a protective measure against the parasite. When this cyst is broken, the parasite usually rolls out and is seen to be a thin-walled structure. By appropriate treatment the head and neck of the tapeworm may be pushed out and made to protrude from one end of the bladder.

When one of these bladder worms is ingested by a dog, the cyst wall is digested, and the tapeworm head and neck passes into the small intestine, where it develops into a mature tapeworm (*Taenia hydatigena*) by the growth of segments back of the neck. Egg-bearing segments develop in 10 to 12 weeks. When infested dogs run over pastures used by sheep and goats they may leave feces containing tapeworm eggs and segments on the pasture, and these eggs may be spread by rain onto the grass and into streams and puddles from which the animals drink. When sheep ingest the eggs the embryo escapes from its shell, makes its way to the liver, and begins to
develop. In the course of time it escapes from the liver and becomes attached to the mesenteries or omenta (abdominal membranes). At first it is a bladder without a head, but later a head and neck develop, and the fully formed cysticercus is ready to infect any susceptible dog that eats it.

This parasite is quite generally distributed over the United States. It is most likely to be present where sheep are associated with dogs or where stray dogs commonly run over pastures and where animals are slaughtered on farms or at small country slaughterhouses at which little care is exercised in disposing of the viscera and of diseased portions of the carcasses.

Light infestations produce little damage, but severe ones may make an animal very sick and even cause death. In such cases the animal shows symptoms of dullness, weakness, loss of appetite, and fever. These symptoms are due to hemorrhage from the liver, caused by the wandering larvae, and the peritonitis, or inflammation of the abdominal lining, that results. The symptoms appearing in the early stages of the infestation are seldom associated with the invasion of tapeworm larvae.

On post mortem examination the bladder worms may be found in the liver or in the abdominal cavity attached to the mesenteries or omenta. No particular lesions are associated with light infestations. In animals dying of hemorrhage from the liver and peritonitis resulting from massive invasion, the liver surface shows a series of short ridges or serpentine markings running in all directions and most numerous near the thin edge. On cut sections the liver substance shows many burrows caused by the wandering tapeworm larvae.

**Sheep Measles**

_Cysticercus ovis_, the bladder worm responsible for "sheep measles," is most commonly found in the heart or diaphragm but occurs not infrequently in the muscles of mastication and the tongue and sometimes in other locations in the muscular system. It may occur in the lungs, the walls of the esophagus, and the wall of the stomach. This is a small oval bladder worm with the head and neck invaginated about midway between the ends instead of at one end as is the case of other bladder worms. The membrane of the bladder is very thin, with small mammillate projections. Sheep and goats become infected by swallowing the eggs produced by the adult tapeworm in the intestines of dogs and voided together with segments containing eggs, on pastures or elsewhere. After the eggs are swallowed, the shells are digested, and the embryos, armed with six hooks, penetrate through the wall of the intestine and reach the liver with the blood stream. They pass through the liver into the veins and thence to the heart. From the heart they pass by means of the general circulation to those parts of the body in which they are found at post mortem examination. They develop into mature bladder worms in about 2½ to 3 months.

This parasite is more or less generally distributed throughout the western part of the United States and may occur wherever sheep are attended by dogs, particularly where dogs have an opportunity to
devour dead sheep. Ransom (41) recorded in 1912 that in that year 20,000 sheep carcasses were retained under Federal meat inspection at various abattoirs on account of infection with Cysticercus ovis.

No symptoms have been attributed to infection with these parasites, the infection usually being discovered only at the time of slaughter. Ransom records, however, that of six lambs experimentally infected with tapeworm eggs, five died in 13 to 23 days after the eggs had been fed them. The animals died approximately in the order of the size of the doses of tapeworm eggs given, those receiving the smallest doses surviving longest. Three of them received only the eggs contained in a single segment and the other two received 3 and 10 segments respectively. The sheep that survived received only one-half of a segment and was sick for a period corresponding to that during which the embryonic worms were invading the muscles. The nature of the symptoms was not described.

This bladder worm of sheep was formerly considered to be rare, and when it was found, it was confused with the bladder worm of swine, Cysticercus cellulosae, the larval form of Taenia solium, a tapeworm of man. (See Internal Parasites of Swine, 745.) In accordance with Federal meat inspection regulations governing the disposition of swine carcasses infected with C. cellulosae, carcasses and parts of carcasses of sheep found to be infected with muscle cysticerci were condemned as unfit for human consumption. Eansom (Jfl) demonstrated that the muscle cysticerci found in sheep were the larvae of T. ovis, a tapeworm of dogs not transmissible to man, and he also showed that muscle cysticerci of sheep, instead of being rare, occurred in a large number of sheep slaughtered.

At the present time the parasite is important chiefly from the point of view of meat inspection. Heavily infected carcasses are condemned or rendered into inedible products. The infected portions of a lightly infected carcass are removed and condemned, and the remainder is passed for food. This disposition is made of infected material not because of any danger of human infection but because of the appearance of the meat.

The Gid Bladder Worm

The gid bladder worm, Coenurus cerebralis, occurs in the brain or spinal cord of infected animals as a large cyst, or bladder, attaining the size of a hen's egg or larger. It consists of a thin membrane containing a rather large amount of fluid. On the membrane there are a number of small white objects about the size of a grain of wheat, which project into the fluid. These are the tapeworm heads.

Sheep and goats acquire the infection in a manner similar to that described for the other bladder worms—by ingesting the tapeworm eggs voided by infected dogs or related carnivores on pasture or range. The embryos that emerge after the eggshells have been digested make their way through the wall of the intestine and are carried by the blood stream to various parts of the body, but only those that reach the brain or spinal cord are able to complete development, the others dying and degenerating before full larval development has been completed. Those that reach the central nervous system at first move
about on or in the brain and spinal cord, forming curving channels. After a while the young worm comes to rest, and the bladder worm develops, completing its development in about 7 to 8 months.

At the time when the young worm gets to the brain there are usually slight symptoms of fever and restlessness, which are easily overlooked. If, as a result of severe infection, the sheep dies at this time, an examination of the brain will show a number of curving channels on its surface. If the infected animal does not die, the symptoms of this stage abate, and there is no further indication of the presence of the parasite until it has grown to the point where the heads form.

Infection with this parasite gives rise to certain characteristic symptoms (fig. 4), their occurrence and severity depending on the number of worms present. According to Neveu-Lemaire (38), animals harboring 10 to 12 or more parasites in the brain show, shortly after the beginning of the infection, symptoms of depression, somnolence, and loss of appetite and lose flesh rapidly. Disturbances of vision are noted, some animals becoming completely blind and running into objects in their path. The visual disturbances are accompanied by disturbances in locomotion. The animals stumble, fall down frequently, settle on the hindquarters or forequarters, and may lie down for whole days as if paralyzed. Death, resulting from acute encephalitis, or brain fever, follows in about a month. There is no evidence of turning or circling disease.

Animals infected with only a few parasites in many cases do not show these early symptoms; in others the symptoms occur, but they are less marked and disappear after about 8 to 10 days. For the next 4 to 6 months the animals show little evidence of infection. After that, disturbances in vision are noted, and they are accompanied by locomotor disturbances manifested by turning movements. The animals walk in circles either to the right or to the left, or they may pivot in one spot; others walk with the head high and with high knee action, or with the head low and a stumbling gait, the character of the movements depending on the location of the cysts in the brain. The animals refuse feed and water and become greatly emaciated. They may move about continuously or stop at times and gaze fixedly at nothing in particular. They are difficult or impossible to herd and tend to lag behind the flock or become lost. When the parasite is located in the spinal cord there is paralysis of the hindquarters.

![Figure 4.—A "giddy" sheep. (Photograph by M. C. Hall.)](https://example.com/figure4.jpg)
the rectum, and the bladder. At first the hind legs are brought up convulsively; later they drag, and the muscles atrophy.

In sheep that die in the early stages of a massive infection the aborted worms that have lodged at places unfavorable for their development may be detected by the presence in the muscles of sinuous channels, whitish or yellowish in color and enlarged at one end, where the aborted larvae may be found. Similar tracks, representing the wanderings of the worms, may be found on the brain in 2 to 4 weeks after it is invaded by the parasites. The pressure of the cysts causes atrophy, or wasting away, of the neighboring nervous tissue and the overlying bone. The bone becomes thin and may even be perforated.

**The Hydatid**

Hydatids (*Echinococcus granulosus*) occur most frequently in the liver (fig. 5) and the lungs, but they may be found in practically any organ or tissue. The cysts may vary from less than 1/4 inch to more than 6 inches in diameter. They may be single or multiple, sterile or fertile. When located on the surface, the cysts form spheroidal protuberances. According to Mönnig (35) only 8 percent of

**Figure 5.**—Liver of sheep showing hydatid infestation.
hydatids in sheep are sterile. When sterile, they contain fluid only; when fertile, they contain minute objects resembling grains of sand attached to the cyst wall or lying unattached in the fluid. These grains are broad capsules, and each of them may contain a number of very small tapeworm heads.

While this tapeworm of dogs and related carnivores has a worldwide distribution, there is very little definite information on the frequency of its occurrence in dogs and its geographical distribution in the United States. Meat-inspection records covering the period 1930–38, inclusive, indicate that approximately 1,200 cattle livers were condemned each year for hydatid infestation. The locations of the meat-inspection establishments at which these livers were condemned indicate a wide distribution of the parasite throughout the United States.

The symptoms shown by animals affected with hydatids depend on the location of the parasites and their size. If the parasites are small or do not crowd important organs, few or no symptoms will be manifested. When the parasites are located in the heart or brain or some other vital organ, there may be marked symptoms or even death from pressure or from rupture of the cysts. As a rule infestations are not detected or diagnosed during life but are found only on post mortem examination.

**ROUNDWORMS**

Except in areas where liver flukes are prevalent, the roundworms are the most serious parasites of sheep and goats. Roundworm parasites occur in the respiratory tract, all parts of the digestive tract except the second and third stomachs, the circulatory system, and the eye and lachrymal (tear) ducts. The only roundworms occurring in the esophagus, or gullet, and the rumen, or paunch, are species of Gongylonema, which occur principally in the lining membranes of these organs and, so far as is known, have no marked harmful effects on their hosts.

**ROUNDWORM PARASITES OF THE ABOMASUM, OR FOURTH STOMACH**

*The Twisted Stomach Worm*

The twisted stomach worm, *Haemonchus contortus*, is the most serious worm parasite of sheep and goats. It is from ¾ to 1 ½ inches long and about as thick as a pin. The females are larger than the males, and when alive, their bodies are marked with a spiral striping, which is responsible for the common designation, twisted stomach worm. On post mortem examination of animals that have died of stomach worm disease, these parasites are found, sometimes in enormous numbers, in the fourth stomach.

The general facts of the life histories of most of the common roundworms parasitic in the gastrointestinal tracts of sheep and goats have been given (p. 861). The life history of the stomach worm, *Haemonchus contortus*, as determined by Veglia (52), is here
presented in detail for the purpose of illustration. Veglia's observations on the influence of temperature and moisture on the development of eggs and larvae of *H. contortus* are also noted.

The eggs are produced in the stomach and pass out with the feces. Under favorable conditions of temperature and moisture they hatch in a few hours, but unfavorable conditions may delay hatching for several days. In the course of the next few days the larvae that develop from the eggs molt twice and are transformed into third-stage, or infective, larvae. These third-stage larvae are enclosed in the skin of the second molt and are therefore also known as ensheathed larvae. They do not feed and are quite resistant to unfavorable climatic conditions. When the weather is warm and the grass is wet with rain or dew they climb up on grass blades, with which they are swallowed by host animals in grazing. Shortly after being ingested, they shed the skin of the second molt and reach the fourth stomach of the host animal, where, after another molt, they develop into the fourth larval stage. The fourth molt is completed between the ninth and tenth days after infection, and with the completion of this molt the worms reach the adult stage. They become mature in the course of 2 to 3 weeks. The period required for development, from the time the larvae are swallowed until the adult stage is reached, is variable, but it averages about 15 days. According to Veglia (52) eggs not uncommonly appear in the feces on the fifteenth day after infection, indicating that the adult stage was reached earlier. On the other hand oviposition (depositing of eggs) may be retarded for 10 days or more.

Larvae introduced into sheep by means of water reach the abomasum and there undergo the second molt. Larvae introduced with solid food start the second molt at once, that is, in the mouth. The larvae possess a distinct biotactism, or affinity, for the mucous membrane of the fourth stomach and do not remain in the rumen for any great length of time. After reaching the fourth stomach, they lodge between the minute epithelial processes of the mucous membrane without actually piercing it.

Veglia gives a detailed account of conditions affecting the development of the preparasitic, or free-living, stages of *Haemonchus contortus*. The results of his work on the factors of air, temperature, moisture, and light in relation to development and survival of eggs and larvae may be applied to field conditions as follows:

1. Constant warm dry weather, such as occurs during periods of drought, is unfavorable to the development of eggs and larvae of *Haemonchus contortus*.
2. Constant moist or cloudy warm weather is favorable to the development of the eggs and larvae.
3. Constant or prolonged cold wet weather delays development but does not prevent it, provided the moisture is not excessive.
4. Constant dry cold weather, especially if the temperature falls to freezing or below for long uninterrupted periods, kills both eggs and larvae.
5. No definite information can be given as to the effect of variable weather. A very high percentage of eggs and of freshly hatched
larvae are killed if during the first 2 days following their deposition on soil the air is dry and warm, provided the soil also is dry. If infected feces are deposited on dry soil, numerous eggs die, even if the first day is cloudy, whereas if the feces are dropped after a heavy rain, numerous eggs survive, especially in grass, even if the first day is sunny and warm.

6. The nature of the soil covering, that is, the pasture and the soil itself, are factors to be considered.

7. The great majority of the eggs passed by infected animals fail to reach maturity.

Various symptoms have been associated with stomach worm (*Haemonchus contortus*) infections, but experimental infection with this worm has demonstrated that the principal symptom is a severe anemia manifested by pallor of the skin and the visible mucous membranes (fig. 6). According to Clunies Ross and Gordon (12), lambs harboring considerable numbers of worms may exhibit little obvious evidence of ill health provided feed conditions are satisfactory. Even in massive infections no obvious symptoms may be noted for some time, but when the symptoms develop they do so rapidly and are severe. Infected animals become weak, are disinclined to move, and develop the characteristic swelling known as bottle jaw. In animals experimentally infected with the *H. contortus* worms, there was no evidence of diarrhea, a condition formerly commonly associated with such infection. This has also been noted by Clunies Ross and Gordon, who state that in the great majority of severe cases of stomach worm infection there is no diarrhea but on the contrary the feces are harder and drier than normal and greatly reduced in quantity. Lambing ewes, ewes with lambs at foot, and older animals may be severely affected, particularly late in the summer and in the fall when pasturage becomes short. Infected animals that show no marked evidence of stomach worm infection as long as they remain relatively quiet may die when driven for any distance. Fat lambs and even adult sheep may die suddenly when heavy infections are rapidly acquired. Such animals may literally bleed to death before they have had time to lose condition.

The lesions found in animals dying of stomach infection are those
of severe anemia. The blood is thin and watery, and there is a gelat-ino us infiltration of the omenta and mesenteries. On examination the blood shows marked changes, the number of red cells being greatly reduced. The anemia found in stomach worm infection is due to hemorrhage. There is no evidence that the worms secrete any toxins that affect the sheep adversely \( (12) \). In experimentally infected ani-mals blood appeared in the feces in 6 to 10 days after the animals were infected, several days before the worms had reached the egg-laying stage.

**ROUNDWORM PARASITES OF THE FOURTH STOMACH AND SMALL INTESTINE**

*The Small Trichostrongyles*

Sheep and goats are often infected with a number of smaller worms, collectively known as small trichostrongyles, and sometimes suffer severely as a result.

**THE MEDIUM STOMACH WORMS, OR BROWN HAIR WORMS**

The medium stomach worms, *Ostertagia circumcincta* and *O. trifur- cata*, are found in the abomasum, or fourth stomach, generally at the end nearest the small intestine. They are small, brownish, hairlike worms about one-half inch long.

The life history of these worms is direct. The eggs are deposited by the female worms in the fourth stomach and pass out with the feces. Under favorable conditions of temperature and moisture they hatch in about a day and infective larvae develop in 5 to 6 days. The infective larvae are taken into the body during grazing, and after reaching the fourth stomach they penetrate into the wall of this organ. Further development to the adult stage takes place in about 15 days, and eggs are found in the manure of infested sheep about 18 days after experimental infection.

As a result of the penetration of the larvae the stomach wall be-comes inflamed, dotted with small white elevated areas, and marked with minute hemorrhages. The white spots which contain the worms, increase in size and become nodular—knotlike or lumpy—as the worms grow. With maturity the worms emerge from these nodules, which recede and disappear.

Since under natural conditions infestation with *Ostertagia* is usually accompanied by infestation with the common stomach worm, *Haemonchus contortus*, as well as with other worms, it is rather dif-ficult to describe any specific symptoms. Sheep primarily infested with *Ostertagia* species are said to show progressive loss of condition with intermittent diarrhea and a stunted appearance.

**THE SMALL STOMACH AND INTESTINAL WORMS**

Several species of small trichostrongyles (*Trichostrongylus axei*, *T. colubriformis*, *T. vitrinus*, and *T. capricola*) are found in the stomach and small intestine of sheep and goats. *T. axei* occurs for
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the most part in the fourth stomach, while the other three species are generally found in the small intestine, *T. capricola* occurring more frequently in goats than in sheep.

These trichostrongyles are small, hairlike worms about one-fourth to one-third of an inch long. They are usually present in fairly large numbers, but because of their small size they are easily overlooked on post mortem examination. The easiest way to find them is to wash out the first 20 feet of the small intestine in a glass dish and then examine the washings against a dark background. Another method is to scrape the lining of the small intestine and examine the scrapings for the worms.

The life history of these small trichostrongyles is essentially similar to that of the common stomach worm, differing only in the resistance of the embryonated eggs (those containing embryos) to drying; they have been shown to be able to survive drying for 15 months under experimental conditions. This is of great importance since it enables the eggs to survive long periods of drought and resume their development as soon as sufficient moisture becomes available, thus continuing pasture infections that might otherwise be terminated by the dry weather.

There are no noticeable gross lesions associated with trichostrongylosis (infection with trichostrongyles). It may be differentiated from haemonchosis (a disease due to infection with *Haemonchus contortus*) as follows: Trichostrongylosis is essentially a slow, protracted disease of young animals, especially lambs and yearlings, which appears shortly after weaning and continues through the summer and the next winter. It is marked by unthriftiness, pronounced diarrhea (fig. 7), progressive weakness, and loss of appetite. Anemia is not noticeable at first but becomes evident as the disease progresses; potbelly and bottle jaw are absent in pure infestations. Infection with stomach worms, on the other hand, is a rapidly fatal disease affecting all classes of animals, showing no noticeable stunting or consistent scouring—that is, scouring may or may not be present, or it may be intermittent. Anemia appears early in the course of the disease, and potbelly and bottle jaw are commonly present.

Bone weakness, manifested by lameness and spontaneous fracture
of the leg bones, especially the femur and the humerus (the upper bones of the hind and front legs), has been reported occasionally in sheep heavily infested with small trichostrongyles.

The Australian workers emphasize that the course of this disease is markedly influenced by the nutritional condition of the animal. Losses from trichostrongylosis occur during the dry summer months when the grass becomes scarce.

**The Cooperias**

The species of *Cooperia* commonly found in the small intestine of sheep and goats are *C. curticei* and *C. oncophora*; *C. punctata* and *C. pectinata* have also been reported from sheep, although they occur more commonly in cattle. All of them are relatively small, hairlike worms about one-fourth to one-half inch long and of a brownish-red color when freshly collected.

The preparasitic stages of these nematodes are similar to those of *Haemonchus contortus* and of trichostrongyles. According to Andrews (2) the infective larvae lose their sheaths soon after being ingested and migrate into the crypts of the mucous membrane of the small intestine about 3 days after entering the host. They do not, however, penetrate into the mucous membrane. They undergo the third molt on the fourth day after entering the host, grow rapidly, and return to the lumen of the intestine on the fifth day. They pass through the fourth molt about the ninth day, and eggs may be found in the feces on the fifteenth day after infection.

Experimental infection with *Cooperia curticei* produced no marked symptoms or lesions in lambs receiving good care and an adequate amount of feed, although fatal infections in young goats by members of the genus *Cooperia* have been reported by Edgar (18). Andrews (2) reported that experimental infections with this worm decreased the ability of the infected animals to convert their feed into gain in weight, but no symptoms referable to the worm infection were noted and no lesions were found at post mortem examination.

**The Sheep Hookworm**

The sheep hookworm, *Bunostomum trigonocephalum*, is a relatively large white worm. The male is about half an inch and the female about three-fourths of an inch to an inch long. The worms are about one-half to three-fourths as thick as an ordinary pin.

The preparasitic, or free-living, stages of this worm are similar to those described for the other strongyle worms, except that the infective larvae, in addition to being able to enter the body by way of the mouth, are also able, according to Beller (4) and Ortlepp (39, 40), to penetrate the intact skin. Beller reported finding hookworm eggs in the feces of a sheep 17 days, and in those of a goat 15 days, after having applied infective larvae of *Bunostomum trigonocephalum* to the skin.

In 1939, on the basis of his investigations, Ortlepp (40) gave a detailed description of the life history of the sheep hookworm, which
may be summarized as follows: Under favorable conditions of temperature and moisture the eggs hatch in 24 hours, and the larvae reach the infective stage in 5 days. These larvae can infect sheep either through the mouth or through the skin. When applied to the skin, they penetrate it and are carried, presumably by the blood stream, to the lungs, which they reach in 6 days. They remain in this organ for about 5 days. During this time they grow, pass into the fourth stage, and are provided with a provisional mouth capsule. They molt and migrate from the lungs to the small intestine by way of the esophagus and stomach. They reach the small intestine as fourth-stage larvae about 11 days after infection. About 4 weeks after infection they reach the adult stage, and about 5 weeks later they reach maturity, the females at this time containing segmented eggs.

Ortlepp's experimental results are in agreement with field observations to the effect that mature hookworms do not occur in very young lambs.

While infections with this worm are usually complicated by the presence of stomach and other worms, it may be assumed that it does the same damage as other hookworms. This damage consists in the abstraction of blood from the host and the secretion of a hemolytic substance—one that destroys red corpuscles and prevents coagulation of the blood—which causes prolonged hemorrhage at the point of attachment when the worms move from one point to another. Animals infected with this worm manifest the characteristic anemia and edema generally resulting from hookworm infection and infection with other bloodsucking internal parasites.

The clinical symptoms of hookworm infection are identical for the most part with those of stomach worm infection, namely, anemia, edema, and unthriftiness. A clinical diagnosis that will differentiate one condition from the other is practically impossible.

The Thread-Necked Strongyles

The thread-necked strongyles, *Nematodirus spathiger*, *N. filicollis*, and *N. abnormalis*, are found in the small intestines of sheep.

The anterior portion of these worms is slenderer than the posterior, and the head and neck are thin and transversely striated, or marked with narrow circular bands. In *Nematodirus spathiger* the male is about three-fifths of an inch and the female about an inch long. These nematodes can be differentiated only by microscopic examination.

Their life history differs in some respects, according to Boulenger (6), from that of the other nematodes mentioned. The rather large eggs pass out with the feces, and an embryo develops in them. This embryo, instead of emerging from the shell and molting like the

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5 In a preliminary report (59) Ortlepp stated that he was able to infect 24 out of 27 sheep with *Bunostomum trigonocephalum* by placing infective larvae of this hookworm on the skin behind the ear. The worms, however, did not reach the egg-laying stage until 10 weeks after the larvae had been applied. In a sheep infected with hookworms in this way, Ortlepp found at post mortem examination, 8 weeks after infection, only adolescent hookworms, no females having reached the egg-producing stage.

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larvae of most other strongyles, molts twice within the shell. Under the influence of alternate moistening and drying or of temperatures varying from 75° to 90° F., the infective larvae emerge from the shells and, like the larvae of stomach worms, crawl up on blades of grass and enter the body of the host animal with the feed.

According to available information, the distribution of these worms in the United States seems to be largely confined to the central and northern parts of the country.

No symptoms or lesions have been definitely attributed to these worms. Recently reported experiments (29) appear to show that these worms are not of any great importance as disease producers, at least so far as sheep are concerned, except perhaps when present in very large numbers. Sheep harboring up to 6,500 adult worms in the small intestine showed no clinical symptoms that could be attributed to infestation with *Nematodirus*, although on post mortem examination minute hemorrhages were found in the mucous membrane of the small intestine at the sites where the worms were found.

**Parasites of the Cecum and Colon**

**The Whipworm**

The whipworm *Trichuris ovis* is usually found attached to the wall of the cecum, or blind gut.

The body is made up of a thin anterior and a thick posterior portion. The anterior part may be compared to the lash of a whip and the posterior to the stock or handle; hence the name whipworm. The worms are white, and the eggs are brown and characteristically lemon-shaped.

So far as is known, this worm has a simple life history. An infective embryo develops in the shell, and sheep and goats are infected by swallowing infective eggs in grazing.

There are no well-defined clinical symptoms associated with whipworm infestation in sheep and goats, but it has been found that in man whipworm infection sets up a low-grade inflammation with distinct symptoms of discomfort and distress. In camels a severe whipworm infection has been found to cause a decided thickening of the wall of the cecum with an excessive secretion of mucus, and it is quite possible that in other animals heavily infected with whipworms similar pathological conditions may be produced. The anterior part of the worm is usually found deeply buried in the mucous membrane lining the cecum, or blind gut, and the most common lesions consist of inflamed and thickened areas surrounding the points of attachment.

**The Nodular Worm**

The adult nodular worm, *Oesophagostomum columbianum*, is found in the cecum and colon of sheep and goats. The larvae are found in nodules in the cecum and large intestine and in heavy infections may be distributed throughout the intestinal tract. The nodular worm, *O. columbianum*, is a fairly large, white worm, about five-eighths of
an inch long. The head is usually bent, forming a hook with the rest of the body.

The free-living phase of the life history of this worm is similar to that of the other strongyles inhabiting the digestive tract of sheep. In lambs a considerable number of the larvae may leave the wall in 5 to 8 days, though some may remain much longer (53). In older sheep several months may be spent in the wall of the bowel. When the larvae return to the intestinal cavity they undergo their fourth and final molt and become adults. The first eggs are passed about 40 days after infection. According to Veglia (54), the adult worms may live for 21 months, but the majority probably die and pass out of the body in 9 to 12 months.

The chief symptom shown by infected animals during the time that the larvae are invading the wall of the bowel is diarrhea, the feces containing considerable quantities of mucus. According to South African workers, during this stage infected animals may show evidence of “rekziekte,” or stretching disease, manifested by a characteristic position, with the legs, especially the hind legs, extended and the back hollowed. Clunies Ross and Gordon (12) report that in the more chronic stages due to adult worms, sheep show progressive weakness with intermittent characteristic diarrhea containing much mucus. The sheep lose weight, and there is some evidence of anemia.

The larvae cause local injury to the mucous membrane, but in initial infections of lambs in which the larvae remain in the wall of the intestine for only a short while, there is little evidence of nodule formation. In older sheep, a more severe local reaction follows the penetration of the wall of the intestine, and marked nodules develop. These nodules at first contain a mass of creamy, greenish material which gradually becomes first cheeselike and later hard and calcareous. While the nodules, which may extend throughout the length of the intestine, are the most noticeable lesion, it has not been demonstrated that they are the principal cause of the harmful effects produced by infection with these parasites. It is true that interference with bowel function may result when nodules are large and very numerous, and complete impaction (stoppage due to a hardened mass of material) and intussusception (telescoping of one part over another) may occur when the lumen, or interior, of the bowel is excessively narrowed; but serious effects are reported (12, 54) to have resulted from chronic infections in which the animals had survived the early stages and adult worms had developed. These effects apparently do not show up until some months after infection.

Nodular disease of sheep was first described in 1890 by Curtice, who at that time reported that it caused heavy economic losses both through its effect on the health of the sheep and because it rendered sheep intestines valueless for making sausage casings. While the disease is seldom fatal, it causes considerable loss of mutton and wool and an impairment of breeding stock. According to Curtice, sheep raising has had to be abandoned in certain areas on account of the nodular worm.

Theiler noted that in certain parts of South Africa two complications of nodular worm disease are found fairly often. One is an in-
vagination of the ileum (infolding of the last section of the small intestine), called knopziekte or rekziekte, the symptoms being disinclination to move, standing in a stretching position with the back hollowed, and the presence of blood in the feces or stoppage of feces. The other is a septic (poisonous) infection of the peritoneal, or abdominal, cavity. Neither of these conditions is associated with nodular worm disease in the United States.

The Large-Mouthed Bowel Worm

The large-mouthed bowel worm, Chabertia ovina, is commonly found in the coiled portion of the colon of sheep and goats. The parasites are relatively large, creamy-white worms, up to three-fourths of an inch long. They are confined to the Temperate Zone; the records of the Bureau of Animal Industry, in fact, show no specimens collected south of Tennessee.

The development of eggs and larvae to the infective stage is similar to that of other strongyles and trichostrongyles. According to Clunies Ross and Gordon (12), however, eggs and larvae are capable of development at low temperatures; mature larvae will develop even when eggs have been kept continuously at about 40° F. in the refrigerator. After ingestion, the infective larvae migrate to the large bowel, the skin of the second-stage larvae being shed immediately and the third molt taking place after some hours. The fourth-stage larvae have a prominent mouth capsule. The fourth molt is not undergone until nearly 2 months after infection, and the first eggs are not passed until 9 to 10 weeks after infection.

In experimental infections, diarrhea, often of an irregular character, usually develops about the fourth week, the feces containing much mucus and blood. The growth rate is slowed, and some animals may show extreme weakness and emaciation. As a rule, however, symptoms gradually disappear, and the animals recover after a period of 4 to 6 weeks.

The adult parasites are always associated with visible lesions, the wall of the colon being thickened and edematous or swollen, and the mucous membrane showing patchy congestion and even hemorrhage. According to Clunies Ross and Kauzał (13) developing worms may cause even more marked lesions, the colon being congested throughout its length. The immature worms are pinkish, owing to ingested blood. In heavy infections with immature worms there is a definite reduction in hemoglobin and red-cell count, and the white cells tend to increase. The immature worms appear to be more injurious than the adults, but the adults are probably not without effect on the host.

Roundworm Parasites of the Respiratory Tract

Lungworms

In the United States sheep and goats may be infected with two species of lungworms, the thread lungworm, Dictyocaulus filaria, and the hair lungworm, Muellerius capillaris. Other species of lungworms, belonging in the genus Protostrongylus, have been reported as para-
sites of sheep and goats in other parts of the world, but thus far nematodes of this genus have been found only in deer, mountain sheep, and rabbits in North America.

Thread lungworms are whitish worms up to 4 inches in length. They may be easily seen on opening the air passages of the lungs of infected animals.

The eggs, which already contain embryos when laid, hatch in the lungs. The larvae reach the pharynx, in the upper part of the throat, and may be expelled when the animal coughs or swallowed and passed out with the feces. They molt twice in the course of the next few days, the time varying with the temperature and moisture, and under ordinary circumstances reach the infective stage in about 10 days. According to Clunies Ross and Gordon the infective larvae are not very resistant to drying but are able to develop at relatively low temperatures, that is, about 40° F. After being swallowed by the sheep the larvae bore through the wall of the small intestine and, according to A. Hobmaier and M. Hobmaier (24), pass by way of the lymph stream into the lungs. In 2 to 5 days after infection they are found in large numbers in the mesenteric lymph nodes. Here they undergo the third molt, and the fourth-stage larvae then leave the glands and pass with the lymph stream to the heart and then with the blood to the lungs. They pass through the walls of the alveoli, or air cells, into the larger air passages. Adult male and female worms develop in the lungs about 18 days after infection, and eggs may be produced in 26 days but usually do not appear in less than 5 weeks.

The worms and their eggs and larvae set up an irritation, and the first symptom noted is a cough. While this may occur as early as the sixteenth day after infection (25, 26, 27), it usually starts about 30 days after infection and coincides with the development of adult worms in the lungs and the passage of larvae in the droppings. The cough is usually strong and harsh in light infections and may be very soft or even absent in heavy infections (12). In severe infections the breathing becomes rapid and shallow owing to the obstruction of the air passages. Heavily infected animals become weak and listless and lose condition rapidly, and death may occur about 2 months after infection. The effects produced by the adult worms, especially when they are numerous, appear to be largely mechanical. The masses of worms and eggs and the exudate, or discharge, produced by the irritated mucous membranes lead to complete blockage of the air passages and the consequent collapse of the lung tissue supplied by them. Large areas of the lungs may thus be rendered functionless, and in massive infections the principal cause of death appears to be suffocation from mechanical blocking of the air passages. The irritation of the small and large bronchial tubes by the worms may lead to bronchitis, which may spread to the lung tissues around the bronchial tubes and give rise to pneumonia. Secondary infection of the collapsed portions also may lead to pneumonia.

In light infections the lesions are usually confined to the lower parts of the lungs.

The hair lungworm, *Muellerius capillaris*, occurs in the small bronchial tubes and in the lung tissue. It is a small, very fine worm, sel-
dom seen against the background of lung tissue in which it occurs.

The eggs are unsegmented when laid but develop and hatch in the lungs. The larva has a characteristic undulating tail with a small spine on the upper side. Larvae either migrate to or are coughed up into the pharynx, where they are swallowed, and then pass out onto the pasture with the feces. They can survive considerable periods of dryness and freezing temperatures in spite of the fact that they have no protective sheath. It has been shown experimentally by the Hobmaiers (23) that these larvae can penetrate and develop to infective larvae in land snails. It is assumed that sheep become infected by ingesting the snails in grazing. After being liberated, the infective larvae reach the lungs in the same manner as the larvae of the thread lungworm, *Dictyocaulus filaria*.

No specific symptoms are attributed to infection with these worms. The presence of the worms and their larvae is indicated by raised, grayish areas on the surface of the lungs. These areas are usually rounded and vary in size from $\frac{1}{4}$ to 1 inch in diameter. On being cut through, they are seen to be greenish gray owing to the accumulation of eosinophiles (certain white blood corpuscles). At times definite pneumatic changes are associated with these lesions.

**Roundworm Parasites of the Eyes of Sheep**

Small roundworms, about one-third to four-fifths of an inch in length, may occur in the ducts of the tear glands, between the eye and the lids, or under the nictitating membrane—the so-called third eyelid—of domestic and wild ruminants. These worms belong in the genus *Thelazia*, and one species, *Thelazia californiensis*, has recently been reported as occurring in the eyes of sheep and deer in California.

These worms normally occur in the ducts of the tear glands, but they may escape from their usual location and be found on the surface of the eyeball beneath the lids, under the nictitating membrane, or even in the eyeball. They are white, slender, and tapering at both ends. The males are one-third to one-half and the females one-half to three-fourths of an inch in length. Nothing is known of the life history of the worms, but in view of the fact that a closely related eye worm of chickens requires an insect intermediate host in its life cycle and that putting larvae of *Thelazia calliptena*, an eye worm of the dog, directly into the eye of a dog failed to produce an infection (19), it is assumed that the life history of the eye worm of sheep also is indirect and that an intermediate host is required in its life cycle.

According to Stewart (49) excessive watering of the eyes, or lachrymation, engorgement of the blood vessels of the membranes and of the eyeball, and small areas of hemorrhage on the membranes and under surface of the lids were observed in infected sheep in California. The author notes, however, that some of these symptoms may have been due to infectious keratitis, or inflammation of the cornea, with which some animals in the flocks examined were affected. In other animals from which these worms have been reported, it has been found that they cause irritation of the conjunctiva, profuse lachry-
Internal Parasites of Sheep and Goats

mation, sensitiveness to light, and cloudiness, followed later by defi-

tive opacity, of the cornea. As a result of invasion by pus-produc-
ing organisms the cornea may become ulcerated, and this in turn

may lead to an inflammation of the iris and other structures of the

eye. If the worms are not removed, the eyelids and the nictitating

membrane may become swollen, and, owing to the drying of the

purulent, or puslike, discharge exuding between the eyelids, the lids

stick together. Mechanical injuries due to loss of sight and the

fixation of the nictitating membrane, which is unable to function

because of its swollen condition, may aggravate the primary difficulty.

The presence of these eye worms may be suspected when one or

more animals in a flock show signs of sensitiveness to light with pro-

cuse watering of the eyes, or lachrymation. The parasites are most

easily found in animals showing the earliest stages of the clinical

manifestations, namely, lachrymation with slight opacity of the

cornea and little, if any, purulent discharge. The septic, or poio-

soning, processes due to secondary infection appear to kill off the

nematodes, or at least to confine them to the depths of the tear

ducts. The worms are not always seen in a cursory examination of

the eye, because of their unusual habitat. It is necessary, therefore,
to examine the parts thoroughly by exposing the under surface of

the nictitating membrane and the eyelids, when the worms can be
detected by their active wriggling movements in the lachrymal secre-
tion. They appear to be washed up with the tears from the

lachrymal duct, as any manipulation or application of dressings that

tends to increase the lachrymal secretions facilitates the recovery

of specimens from infected animals. The instillation into the eye of

a few drops of a 1-percent solution of cocaine or other suitable local

anesthetic facilitates the examination of the eye. According to

Stewart (46) this procedure also causes the worms to crawl out of

the inner cornea of the eye and makes it easier to remove them.

Until the life history of these worms has been worked out, no

preventive or control measures can be recommended.

The treatment consists in the mechanical removal of the worms

from the eye. After the worms have been removed the eyes should
be treated as in cases of inflammation due to other causes.

**Roundworm Parasites of the Circulatory System**

The roundworm, *Elaeophora schneideri*, has been found in the

arteries (carotid, mesenteric, and iliac) of sheep. It is a slender

white worm. The male is about 60 millimeters (2.5 inches) and the

female 110 to 120 millimeters (4.5 to 5 inches) long, tapering at both

ends.

These roundworms, or nematodes, have been found in sheep in

Catron County, N. Mex., and in deer in Utah. Lesions similar to

those caused by the microfilariae, the larvae of these nematodes,

have been noted in sheep in Arizona and Colorado.

The life history of the worms is not definitely known, but since

they are filarids and all filarids require an intermediate host in their
life cycles, it is assumed that some arthropod (insect or insectlike animal) acts as their intermediate host.

The adult worms produce no known symptoms in infected animals, but Kemper (30) reports that the microfilariae cause a dermatitis, or skin inflammation, usually involving the skin in the region of the poll (fig. 8) and in a few cases extending forward over the face to the nostrils and lips. Lesions sometimes occur also on the foot used to scratch the head and on the abdomen where the affected

![Figure 8](image_url)

**Figure 8.**—Head of a sheep, showing lesions caused by microfilariae of *Elaeophora schneideri*. (From Kemper.)

foot has come in repeated contact with the skin, perhaps while the sheep was lying down. The lesions are primarily those of a chronic dermatitis with excessive growth of tissue and the formation of numerous small abscesses. The extent of scratching and rubbing, apparently a result of intense itching, more or less determines the ultimate size of the lesion.
THE CONTROL OF INTERNAL PARASITES OF SHEEP AND GOATS

As previously noted, the internal parasites of sheep and goats include protozoa, flukes, tapeworms, and roundworms. Control depends on effective anthelmintic medication—the use of effective worm medicines—and a thorough knowledge of the life histories of the parasites. There are two phases in the life histories of all of these parasites. One, the reproductive phase, is spent within the body of the sheep, and the other is spent either free in the natural environment of the sheep or in some intermediate host. The intermediate hosts do not serve merely as mechanical carriers, since a definite part of the life history of the parasites is completed within them. A knowledge of the life cycles of the intermediate hosts is generally necessary before effective control measures can be formulated for the parasites that require such hosts.

CONTROL OF THE PROTOZOA PARASITES

The protozoan parasites of sheep that are known to be of importance in this country are various species of coccidia and *Globovibium gilruthi*. They are all microscopic parasites too small to be seen with the naked eye. Seven different species of coccidia have been recently reported by Christensen (7) as parasites of sheep in this country. Whether there is any difference among these species in their effect on sheep is not definitely known at present.

As already pointed out, there is no specific medicinal treatment for coccidiosis. Affected animals should be removed from the flock and isolated, and the exposed but healthy individuals should then be allowed to run over a large acreage to lessen the chances of infection. All straw, litter, or other material soiled by the discharges of affected animals should be burned or disposed of in such a manner as not to form a source of infection for other animals. The premises occupied by infected animals should be thoroughly cleaned and disinfected before being used for healthy animals. In the cases described by Christensen (9), referred to earlier in this article, it is obvious that either some method of feeding should be devised which will prevent the contamination of the feed with sheep feces, or the unconsumed portions of feed should be removed from the feed troughs and the troughs thoroughly cleaned before fresh supplies are placed in them. The extent to which such measures can be applied will depend on the stockman’s interest and on circumstances. Practical methods of using copper sulfate and ferric sulfate mixtures as a means of lowering the level of infection remain to be devised. If, as suggested by Christensen, these substances can be used in powdered form in the feed or in solution in the drinking water and be so distributed as to insure each animal’s receiving the required amount, sufficient protection may be afforded to obviate serious outbreaks of the disease.
CONTROL OF FLUKES

In common with all other flukes, the flukes of sheep and goats require a snail intermediate host for the completion of their life cycles. It is obvious, therefore, that the control of these parasites in sheep and goats depends on the control of the snails. A thorough knowledge of the environment, distribution, etc., of the intermediate hosts is necessary to accomplish this control.

The most effective medicinal treatment of sheep and goats for liver fluke infection is the administration of carbon tetrachloride. For convenience in administration, this drug can be incorporated in a suitable vehicle, such as liquid paraffin or raw linseed oil. Carbon tetrachloride, however, is effective only against the adult parasites in the bile ducts. Immature flukes which may be present in the liver tissues or which have only recently arrived in the bile ducts are not affected by the treatment, and it should therefore be repeated at intervals to remove the flukes as they mature. Although the carbon tetrachloride treatment is normally safe for sheep, it may, at times, according to Clunies Ross and Gordon (12), be followed by heavy losses 3 to 4 days after the drug has been administered. Affected animals show evidence of acute abdominal pain, and at post mortem examination acute inflammation of the fourth stomach and intestine are noted. Scattered hemorrhages are found in the mesenteries and pleura—the membrane lining the thorax and covering the lung. These authors also report that some sheep lose their wool 2 to 3 weeks after treatment.

While the reasons for this occasional toxicity of carbon tetrachloride for sheep are not well known, certain general precautions should be observed: Only reliable preparations of carbon tetrachloride, free from phosgene and sulfur impurities, should be used; when liquid paraffin is used as a vehicle in the administration of the drug, it should be of a high medicinal quality; where the drug has not previously been employed, a trial group of sheep should be treated a few days ahead of the whole flock; abrupt changes of feed prior to treatment should be avoided; feeding of concentrates should be discontinued at least a week before treatment; sheep should never be drenched in cold; inclement weather; if there is any indication of mineral deficiency, sheep should be given access to mineral supplements containing a mixture of steamed bonemeal and salt and ground limestone or dicalcium phosphate and salt (12).

The destruction of the snails which are the hosts of the intermediate stages of the fluke is the most important control measure. This may be accomplished by drainage of wet areas where snails propagate, by the use of copper sulfate (bluestone, blue vitriol) for their destruction, or by a combination of the two. Since snails require a certain amount of water in which to live and propagate, drainage is the preferred method. Complete drainage so changes the environment as to make it impossible for snails to survive. If the drainage ditches are kept open and clean, the drained area will

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remain unfavorable for snails and will require no other attention. If the ditches become infested, treating the banks with copper sulfate will destroy the snails. Drainage is not always possible, however, because of the cost or engineering difficulties; though in figuring the cost of any drainage project, the increased value of the land and of the improved forage that can be produced should always be considered. Wherever drainage is either impractical or impossible the treatment of snail-infested areas with copper sulfate should be considered.

Copper sulfate has proved to be very effective for killing snails. While this chemical, diluted 1 part to 1,500,000 parts of water, will kill them overnight, the thorough distribution of such small amounts is difficult to accomplish, and it is usually applied in much greater concentrations.

Copper sulfate kills the snails, the miracidia, and the free-swimming cercariae. It does not injure the fluke eggs or the encysted cercariae; hence, to prevent infestation, the snails should be destroyed before the water and grasses become infected. As used in dilutions for killing snails, copper sulfate is not injurious to grasses or flowering plants and will not poison livestock. It will, however, kill the lower forms of plant life, such as the algae and mosses, and it may kill fish.

The method of application varies with the movement and distribution of the infested waters. In a stream originating on a stockman's land and with the water confined within definite banks, sacks containing large crystals of copper sulfate may be placed in the headwaters. To reach the snails on the banks, dams may be thrown across the stream at various places, depending on the fall of the stream, to raise the treated water to cover the snails. Overflow lands, stagnant backwaters, marshes, pools, and similar wet places are best treated by broadcasting powdered copper sulfate. The water troughs, and especially the puddles around the troughs, should not be overlooked. It is more economical to use the powdered than the crystalline copper sulfate for treating these places. The chemical may be mixed with a carrier such as sand or land plaster in the proportion of 1 part of the copper sulfate to 4 to 8 parts of the carrier, depending on the volume of water to be treated. The land plaster has the advantage of marking the treated area. The airplane has been found to be practical in spreading copper sulfate over large swampy areas, but when applied in this way the copper sulfate and carrier mixture must be fine enough to be applied as a dust.

To estimate the amount of copper sulfate needed to treat the water in a stream, it is necessary to know the flow in cubic feet per second. To determine this, select a uniform section of the stream about 50 feet long, measure the width and the average depth of the flowing water, and multiply them to get the area of the cross section in square feet. Mark off 50 feet, throw a chip in the water at the upper end, and ascertain the number of seconds required for the chip to float the 50 feet. Divide this by 50 to obtain the velocity in feet per second. The number of square feet of cross section multiplied by the velocity in feet per second equals the approximate flow in cubic feet per second.
For example: A stream 6 feet wide has an average depth of 6 inches; the area of cross section equals 6 times 0.5, or 3 square feet. If it takes a chip 25 seconds to float 50 feet, the rate of flow is 2 feet per second. Multiply the cross section (3 square feet) by the velocity (2 feet per second) and the result is a flow of 6 cubic feet per second. For a 24-hour treatment at a dilution of 1 to 500,000 parts of water, 12 pounds of copper sulfate are required for each cubic foot per second of flow. Therefore, the amount of copper sulfate required for this stream would be 6 times 12, or 72 pounds.

Where the amount of copper sulfate required to treat the water in a lake would be excessive or where the lake is used as a fish preserve, the copper sulfate may be broadcast along the banks and on the water a few feet out from the bank.

A few days after treating the water it is well to make a careful investigation of the effect on the snails. Usually one treatment at the right time is enough for a year, but if live snails are found after an application of copper sulfate, the waters should have another application. When once a range is infested, it is necessary to repeat the copper-sulfate treatment once a year until all snails are killed. The best time to treat is when the greatest number of snails can be reached; usually this is after the spring rains.

Often a combination of drainage and the use of copper sulfate will bring about the desired results. Where complete drainage is either impractical or impossible, partial drainage may prove to be of value in reducing the size of the wet area. The remaining undrained portion can then be treated with copper sulfate. The methods used will depend entirely on local conditions.

Control of Roundworms

Except in the areas where liver flukes occur, the roundworms are the most important parasites of sheep and goats. With the exception of *Elaeophora schneideri*, a threadworm found in the blood vessels of sheep, *Gongylonema pulchrum* and *G. verrucosum* (the gullet worms), and *Muellerius capillaris*, one of the lungworms, the life histories of all the roundworm parasites of sheep are direct, no intermediate hosts being required.

The only known effective method of attacking the parasites in the parasitic phase of their existence consists in anthelmintic medication. Many drugs have been tried and recommended as anthelmintics, but, for all practical purposes, there are at the present time only three drugs and one combination of drugs available for effective treatment: Phenothiazine, copper sulfate, tetrachlorethylene, and a combination of copper sulfate and nicotine solution.

Phenothiazine has a very high degree of efficacy when used in the treatment of sheep for large stomach worms, *Haemonchus contortus*; hookworms, *Brunostomum trigonocephalum*; nodular worms, *Oesophagostomum columbianum*; and large-mouthed bowel worms, *Chabertia ovina*. It has given satisfactory results when used in treating sheep for the small trichostrongyles, *Ostertagia, Trichostrongylus*, and *Cooperia* species (fig. 9, A and B). It appears to be ineffective, however, in the treatment of sheep for the removal of thread-necked
Internal Parasites of Sheep and Goats

Strongyles, *Nematodirus* species; whipworms, *Trichuris* species; and lungworms and tapeworms.

Copper sulfate alone is effective against the common twisted stomach worm, *Haemonchus contortus*, but it is not very effective against the other worms found in the stomach and small intestines. Copper sulfate and nicotine solution, commonly referred to as Cu-Nic, is as effective against the common stomach worm as copper sulfate alone and is more effective than copper sulfate alone against the other worms found in the stomach and against some of the worms in the small intestine. Tetrachlorethylene appears to be effective against stomach and intestinal worms, and it is apparently more effective against the small trichostrongyles than either copper sulfate or a mixture of copper sulfate and nicotine solution. The thread-necked strongyles, *Nematodirus* species, are only slightly affected by treatment. However, so far as is known at present, these parasites do not produce any serious disturbance in sheep in spite of the fact that they may be present in large numbers. The nodular worm, *Oesophagostomum columbianum*, and the large intestinal worm, *Chabertia ovina*, are not amenable to treatment with either Cu-Nic or tetrachlorethylene. South African workers, however, report good results from a treatment consisting of a mixture of copper arsenic, 2 parts; calcium hydroxide, or slaked lime, 3 parts; and copper tartrate, 5 parts. The dose of this mixture is 1 gram (15 grains) for lambs 3 to 6 months old; 1.8 grams (27 grains) for lambs 6 to 18 months old; and 2.5 grams (37.5 grains) for animals over 18 months old. Two doses are given on consecutive days, and each treatment is preceded by the administration of 2.5 cc. of a 10-percent solution of copper sulfate. The treatment is administered as follows:

Figure 9.—A, Bottle jaw in a sheep suffering from general gastrointestinal parasitism; B, the same sheep 19 days after treatment with phenothiazine. (From Habermann and Hunt.)
The sheep's mouth is opened wide and the copper sulfate solution is poured alongside the tongue so that it runs down into the throat; then before the sheep's mouth is closed, the proper dose of the copper arsenate, lime, and copper tartrate mixture, which is made up in the form of a powder, is placed on the back of the tongue. The treatment is repeated the next day. Sheep should not be given grain or be permitted access to a salt lick for 2 days before and 1 day after the treatment. If the pasture is dry, green feed should be furnished for a few hours before dosing. Sheep must not be kept from feed and water before treatment. After treatment they may graze immediately but should have no water for 1 to 2 hours.

Suckling lambs should not be treated unless this is necessary. If treatment is necessary the lambs should not be permitted access to water from 4 hours before to 4 hours after the treatment. Weak lambs should receive only one dose, and weak sheep and those that are small for their ages should get a smaller dose on the second day.

From this brief review it is quite clear that from the viewpoint of anthelmintic treatment it does not make any great difference with what particular roundworms the sheep are infected. Two things are necessary: (1) An accurate diagnosis of worm infection should be made, and (2) anthelmintic treatment should be given as and when necessary.

The prevention of infection with gastrointestinal roundworms depends upon a thorough knowledge of the preparasitic, or free-living, stages of these parasites—the egg and the first, second, and third (infective) larval stages. As already noted in connection with roundworms in general and 

From reports of some recent observations on the longevity of infective larvae of various species of parasitic roundworms indicate that the major portion of these larvae died within a few weeks after they were placed on grass plots outdoors and that only a comparatively small number survived for as long as 4 months. Other reports show that the infective larvae of some species may survive much longer. There is a marked difference in the ability of the eggs and infective larvae of the various species to withstand unfavorable environmental conditions, and much more detailed information is necessary before general recommendations for the prevention of parasitic infection applicable to various parts of the United States can be made. Present indications, however, are that the majority of the infective larvae die in periods of 6 to 12 weeks, but since infective larvae develop under favorable conditions in 4 to 10 days after the eggs reach the pasture, it is clear that pasture rotation alone at intervals of 6 weeks and over cannot be relied on to control parasites.

There is no completely satisfactory medicinal treatment for the removal of lungworms from sheep and goats. The intratracheal injection of various drugs and combinations of drugs, the inhalation of chloroform and sulfur fumes, and the administration of certain volatile compounds that are excreted through the lungs, have been recommended by various authors. Clunies Ross and Gordon state...
that in Australia the intratracheal injection of a mixture of 1 part creosote, 1 part chloroform, 2 parts turpentine, and 4 parts olive oil, in doses of 4 milliliters (1 dram) for lambs, is recommended. In other countries intratracheal injections of pyrethrin in olive oil and Lugol's solution of iodine in glycerin are advocated, but Mönnig (36) reports that intratracheal injections with the latter two substances are of little value.

The development from eggs to infective larvae can take place through quite a wide range of temperature, and the results of cultural experiments indicate that lungworm larvae are well adapted to temperate climates. Although the infective larvae are quite resistant to unfavorable environmental conditions, the eggs and the first- and second-stage larvae are destroyed by drying, and it is a common observation that lungworm infections by *Dictyocaulus filaria* are intimately connected with low-lying, wet pastures. The use of such areas as pastures, wherever the topography permits of a choice, should therefore be avoided. This applies also, of course, to the prevention of infection with other worm parasites. Removal of lungworm-infected animals from infected pastures to dry lots to avoid constant reinfection is also to be recommended wherever it is practicable.

As previously noted, the gullet worms *Gongylonema pulchrum* and *G. verrucosum*, the hair lungworm *Muelleiriis capillaris*, and *Elaeophora schneideri*, a roundworm found in the blood vessels of sheep and deer, require intermediate hosts in their life cycles. There is no medicinal treatment for the removal of either gullet worms or hair lungworms. Kemper (30) reports that the administration of fuadin was of benefit in the case of sheep infected with *Elaeophora schneideri*. The administration of 88 cubic centimeters or more of fuadin intramuscularly in doses of 4 cubic centimeters daily was followed by complete healing of the skin lesions caused by the microfilariae of this parasite.

The tapeworms *Moniezia expansa*, *M. benedeni*, and *Thysanosoma actinioides* also require intermediate hosts in their life cycles. The combination of copper sulfate and nicotine solution has been reported as effective for the removal of tapeworms (*Moniezia* spp.) from sheep (20), but there is no effective medicinal treatment for the removal of the fringed tapeworm, *Thysanosoma actinioides*.

The prevention of infection with bladder worms obviously consists in the proper disposal of all infected sheep carcasses and parts of carcasses, the elimination of stray dogs, and the examination and proper anthelmintic treatment, whenever necessary, of all dogs that can come in contact with sheep or wander over the pastures on which the sheep graze.

Where sheep are housed during the winter months it is a good practice to treat them once every month in order to reduce the infection to a minimum. When such sheep and their lambs are placed on pasture in the spring, most of the pasture infection will have been eliminated; and, with only a few parasites in the old sheep, it will take some time for a pasture infection to build up to a point where it becomes dangerous. Frequent rotation of pasture, with the sheep
not returning to any one pasture in less than 3 months, will be a material aid in controlling infection.

The well-known recommendation for early lambing is based on the known influence of climatic conditions on pasture infection. Low temperatures retard development of eggs and larvae, and temperatures sufficiently low and prolonged will kill them. Lambs dropped early in the season and well cared for will either be ready for market before the favorable season for parasitic development arrives, or if they are retained for flock replacements, they will be in a better condition to withstand parasitic infection than lambs dropped later in the season and going on pasture when the weather favors development of parasites.

One of the most beneficial results of pasture rotation lies in the improved grazing furnished by the pasture as a result of rest and the consequent improved feeding conditions for the animals. While more information is needed concerning the relation of nutrition to parasitic disease, common observation and such experimental evidence as is available indicate very clearly that the nutritional state of the animal has a decided influence on its susceptibility to parasitic attacks. Clunies Ross and Gordon (11) found that resistance to stomach worm infection in older sheep was broken down when the sheep were kept for a considerable time on a diet low in protein and minerals.

THE SHEEP NASAL FLY

The condition in sheep commonly referred to as grub-in-the-head is due to the larvae or grubs of the sheep nasal fly, *Oestrus ovis*, also called the sheep gadfly and the head maggot fly. This fly is somewhat larger than the common horsefly, dull yellow or brownish in color, and hairy (22).

The flies are active during the warm part of the day and rest in warm corners and crevices during the cooler parts. They are viviparous—that is, they bear living young instead of eggs—and deposit their larvae around the nostrils of sheep. In attempting to deposit the larvae, the flies cause great annoyance to the sheep. The animals stop feeding, become restless, press their noses against the ground or against other sheep, and huddle together under buildings, rock ledges, and shade trees along fences.

After being deposited, the minute larvae migrate into the nasal passages. They remain on the nasal mucous membranes for varying periods of time before migrating to the frontal sinuses, where they complete their development as larvae. The mature larvae then leave the sinuses, return to the nasal passages, and drop to the ground, where they burrow into the soil and pupate. The period of pupation may last from approximately 3 weeks to 2 months, depending on soil temperature and moisture. At the end of this period the adult flies emerge from the pupal cases, crawl to the surface, and become active. According to Mitchell and Cobbett (34), larvae deposited in the nostrils of spring lambs early in the season may complete their development in 2 1/2 to 3 1/2 months. Since such larvae may pupate in 12 to 72 hours after reaching maturity and the period of pupation may
last from 17 to 57 days, a full life cycle may be completed in 3 to 5½ months. In New Mexico the flies are active during the late spring, summer, and early fall, and many larvae develop to maturity and are expelled in 25 to 35 days. Many do not develop during the season in which they are deposited, but remain in the nasal chambers throughout the late fall and the winter and do not migrate to the frontal sinuses until the next spring. Cobbett (14) expresses the opinion that the flies overwinter in the nasal cavities of sheep as first-stage larvae.

As the larvae crawl about on the nasal mucous membrane they set up an irritation that results in an increased flow of mucus from the nose, resembling that accompanying a cold in the head. This discharge is thin and clear at first, but as a result of bacterial infection, it soon thickens and becomes discolored, presenting the condition commonly called snotty nose, the most generally recognized symptom of grub infestation. Other symptoms shown by affected animals are frequent sneezing and difficulty in breathing. The eyes become inflamed, and the head is carried low. The animals grate their teeth and lose their appetite, or at least there is interference with feeding.

According to Mitchell and Cobbett (34), post mortem examination of the heads of infested sheep shows little pathological change in the tissues affected by live larvae other than increased mucous secretion, even when the head cavities contain many larvae. Only when dead larvae were encountered in the frontal sinus was there evidence of inflammation of the affected tissues, with discoloration and thickening of the mucous membrane and the presence of a thick, discolored secretion.

Many attempts have been made to treat or to prevent grub infestation of sheep. They may be summarized as follows: (1) Attempts to kill the grubs in the frontal sinuses of the head by injecting materials through perforations in the frontal bones; (2) attempts to kill the grubs by injecting materials into the nasal cavities; (3) attempts to remove the grubs by introducing irritants into the nasal passages for the purpose of causing the animal to sneeze and expel the grubs. Efforts to prevent infestation have been largely confined to placing repellents on the nasal openings. Critical examination has shown that the repellents employed thus far are of little or no value.

Stewart (45), Du Toit (16), and Du Toit and Clark (17) report having obtained satisfactory results in treating sheep for grub infestation by injecting various preparations into the frontal sinuses through openings made in the frontal bones. They described the technique of the operation and reported that they observed no deleterious effects from the treatment. They also noted that in sheep slaughtered a few days after the treatment had been given, no dead larvae were found in the sinuses, apparently demonstrating that the treatment completely eliminated grubs from the head. Gildow and Hickman (21), Du Toit (16), and Mönnig and Du Toit (37) have described a treatment that consists in the introduction of larvicides into the nasal cavities. The animals to be treated were restrained on their backs with the heads held at an angle of 45 degrees with the ground.
Cobbett (4) recently reported having obtained satisfactory results from the injection under pressure into the nasal cavities of a 3-percent solution of a saponified cresol preparation known as saponated solution of cresol. The chief difference between this treatment and those previously described consists in the fact that Cobbett's treatment is mainly directed toward killing the small larvae in the nasal cavities before they reach the sinuses, whereas the purpose of the other treatment is to kill the larvae in the frontal sinuses.

Cobbett reports that irrigation of the nasal mucous membranes of sheep with this solution caused no injury to the animals treated. Some sneezing and coughing occurred immediately after the treatment, but these and the subsequent nasal discharge were of short duration. Occasionally an animal held its breath immediately after treatment and fell to the ground, but such animals recovered promptly when assisted to their feet. The author calls attention to the fact that this treatment is effective as a control measure only in areas where the temperature during the winter months is sufficiently low to kill the adult flies and prevent any adult-fly activity.

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