THE GID PARASITE (*Coenurus Cerebralis*): ITS PRESENCE IN AMERICAN SHEEP.

BY

B. H. Ransom, B. Sc., A. M.,
Scientific Assistant in Charge of the Zoological Laboratory,
Bureau of Animal Industry.

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LETTER OF TRANSMITTAL.

U. S. DEPARTMENT OF AGRICULTURE,
BUREAU OF ANIMAL INDUSTRY,
Washington, D. C., October 21, 1904.

SIR: I have the honor to transmit herewith a manuscript entitled "The Gid Parasite (Crenurus cerebralis): Its presence in American Sheep," by B. H. Ransom, B. Sc., A. M., scientific assistant in charge of the zoological laboratory of this Bureau.

This parasite in its bladderworm stage (Crenurus cerebralis) causes the disease called gid, otherwise known as "sturdy," "staggers," etc., which principally affects sheep, although cattle and other herbivorous animals are also susceptible. It is transmitted to stock through pasture or water which has been contaminated by the eggs of a certain species of tapeworm (Tienia crenurus) parasitic in the intestines of dogs, the latter having acquired the parasite by feeding upon the carcasses of stock infested with the bladderworm stage. Although a common disease in Europe, where it frequently occasions losses of fully 10 per cent in flocks of sheep, gid has until very recently been unknown in the United States. Early in the present year, however, a well-authenticated outbreak occurred in the sheep-grazing regions of the West, and it was, consequently, deemed expedient to publish such facts as would help our stockmen to protect themselves against possible outbreaks in the future.

The information concerning gid herein set forth is of much practical importance to sheepmen, as it enables them to recognize the disease should it make its appearance, and indicates the measures best calculated to prevent its spread. I therefore recommend that the work be published as Bulletin No. 66 of the series of this Bureau.

Respectfully,

D. E. SALMON, Chief of Bureau.

Hon. James Wilson,
Secretary of Agriculture.
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THE GID PARASITE (COENURUS CEREBRALIS): ITS PRESENCE IN AMERICAN SHEEP.

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Scientific Assistant in Charge of Zoological Laboratory, Bureau of Animal Industry.

INTRODUCTION.

The disease, popularly known by English-speaking peoples as gid, sturdy, staggers, turnsick, etc., which affects sheep, and, more rarely, cattle and other animals, is a common disease of stock in Europe. It is said to occur also in South America, and is now known to occur in this country. The cause of this disease, a characteristic symptom of which is vertigo and turning of the affected animal, is the presence of a parasite, the gid bladderworm (fig. 1), in the brain or spinal canal.

The gid bladderworm, or Coenurus cerebralis as it is known technically, is the larval stage of a tapeworm, Tenia canum (fig. 6), which is found in the intestines of dogs. When the brain of a "giddy" sheep is eaten by a dog, the bladderworm develops into tapeworms in the intestines of the latter. The tapeworms produce eggs, which pass out of the dog’s intestines and fall to the ground, where they may lie amid the grass or be washed by rains into pools of water. Sheep and cattle while grazing or drinking are liable to swallow these eggs, which then hatch out, and the embryos, boring out of the intestines, wander to the brain or spinal cord, where they undergo further development and give rise to the condition known as gid.

It seems reasonable to suppose, in view of the large numbers of sheep and dogs which have been imported into the United States from countries where gid is common, that the disease should have become more or less prevalent here also, but for some reason the gid parasite has never gained a foothold in North America, and until very recently, so far as it has been possible to determine, gid has been entirely unknown in this country. A possible exception may be based on the mention made by Leidy (1856) of "Coenurus cerebralis Rud. in the sheep, Capra aries," but neither the time nor the place of the collection of the specimens referred to by that author is known. Although heretofore no cases of gid in this country have been placed on record, it seems hardly probable, in view of our present knowledge, that the disease
has been altogether absent. More likely, cases have occurred from time to time and passed unrecognized, as evidence has come to hand which shows that the disease is now present in the United States, cases having developed recently which, as the attendant circumstances show, must have resulted from infection in this country.

**Presence of Gid in the United States.**

A number of native sheep died at Bozeman, Mont., in January, 1904, with the characteristic symptoms of gid. These sheep were brought to the notice of officials of the Montana Agricultural College. On postmortem examination, bladderworms were found in the brain, and through the courtesy of Professor Cooley two specimens (B. A. I. Collection, No. 3644, figs. 3, 4) have been placed at the disposal of this laboratory. These specimens agree in all essentials with the European *Cesurus cerebralis*, so far as may be determined from published descriptions of the latter, no specimens for comparison being at present available. The salient characteristics of the American form are as follows: Each specimen is nearly spherical, with a diameter of about 25 mm. (about 1 inch). There are no secondary or daughter bladders, either internally or externally. The wall of the bladder consists of a thin semitransparent membrane, and its surface is marked by several groups of little white spots, the invaginated heads, or scolices, of the larval tapeworms, which number over a hundred in each specimen. Each head is supplied with a double crown of 30 to 32 hooks, of two sizes, arranged alternately (figs. 3, 4). The larger hooks measure 160 to 170µ (13/5 to 13/0 inch), and the smaller hooks 114 to 130µ (22/8 to 18/5 inch) in length. In the latter the ventral root is slightly bifid.

Although at present the gid parasite may be causing but little damage in the United States, the fact should be remembered that its history in other countries has shown it to be one of the most dangerous parasites to which sheep are subject, and the possibility kept in view that it may in the future become more widespread and prevalent in this country, and that, in this event, serious losses are liable to occur. Stock raisers, veterinarians, and all persons interested in live stock, especially sheep, should therefore be on their guard against the gid parasite, and, by taking proper preventive precautions—to be discussed later—avoid in this country a possible repetition of the disastrous experience of stock raisers abroad.

**Historical Review of Gid.**

Gid has been known for nearly three hundred years, and possibly longer, it having been traced in literature as far back as 1634. In that year a European surgeon named Reutten observed a sheep
affected with vertigo, and another surgeon, Scultetus, on making a postmortem examination of the animal, discovered in the brain a vesicle filled with a limpid fluid. After this date the disease and the vesicle in the brain with which it is associated were noted by various authors, who attempted to explain in various ways the cause of the disease and advanced numerous theories to account for the presence of the vesicle. Methods of operation for the removal of the vesicle were also described, but not until the latter part of the eighteenth century was the true nature of the watery vesicle in the brain of "giddy" sheep and cattle pointed out. In 1780 Leske demonstrated the animal nature of the vesicle by the discovery of tapeworm heads attached to its surface. Many years later another step was accomplished in solving the question of the etiology of gid when Siebold (1852) discovered that if the gid bladderworm is fed to a dog it develops into mature tapeworms in the intestines of the latter. The following year Küchenmeister (1853) showed further that the eggs from tapeworms thus produced in dogs, when fed to sheep, develop into bladderworms in the brain. Since that time the experiments of Siebold and Küchenmeister have been many times repeated, and the relation between Coenurus cerebralis in the brain of sheep and other animals and Taenia coenuros in the intestines of dogs, as larval and adult stages, respectively, in the life history of a single species of parasite, has been most firmly established.

ECONOMIC IMPORTANCE OF GID.

Gid was soon recognized in Europe, after its first discovery, as one of the most important diseases of sheep; large numbers of animals were lost annually from this cause, and at times in some localities the flocks were almost annihilated.

The following quotations from the literature on the subject will serve to indicate the important nature of the disease, and will give some idea of the damage which has been caused by the gid parasite in countries where it has been prevalent. It will be noticed that most of the authors quoted refer to conditions which existed before the life history of the parasite was known and before the proper means for its prevention were recognized. The percentages given will, in general, probably not apply at the present time, as many stock raisers of Europe have long since realized the practical value of preventive measures, and this has resulted in a material reduction of the general mortality from gid, although in isolated cases where proper precautions are neglected the former high percentage of mortality is still the rule.

Wepfer (about 1659) reported an epizootic of the disease which occurred in the year 1650 among cattle in a certain locality of Germany. In 1752 there were great losses from gid among both sheep
and cattle in Iceland. Sooner or later the disease attracted attention in almost every country in Europe.

Tessier (?1810), according to Numan (1850), placed the annual loss of sheep from gid in France at about 5 per cent. Kuers (1840) estimated the loss in Germany at about the same figure, and Fricke (1844) stated that in his own flocks the losses at one time reached as high as 10 per cent. Numan (1850) states that, while the disease is well known in Holland, it is not so common as in some other countries, although at times it assumes the character of an epizootic. He further states that in some parts of Germany and Austria the loss is 10 per cent, and that in many localities the sheep industry has declined seriously in consequence.

Fleming (in Neumann, 1892) quotes a statement of Youatt that in France 1,000,000 sheep die annually from gid, and states that Gasparin has put the losses in Germany at 15 per 1,000 in the first year of life, 5 in the second year, 2 in the third year, and 1 in the fourth. In England, where the disease is more or less prevalent, the number of sheep lost in some flocks has reached as high as 35 per cent (Veterinarian, 1871, p. 547).

Armatage (1895) estimates the annual losses in Great Britain at about 10 per cent, and states that gid "always prevails in some districts, particularly in Scotland, and upon all uninclosed lands where sheep are gathered in large flocks and attended by dogs."

As an illustration of the extent to which a single flock may be invaded by gid, an instance observed by Brunet (1875) in the Bas Berry region, France, may be referred to. In this case over 80 sheep in a flock of 212, or about 40 per cent, were affected.

DESCRIPTION OF THE GID BLADDERWORM (COENURUS CEREBRALIS).

(Figs. 1-4, 10-12.)

_Cœnurus cerebralis_, the larval stage of the gid parasite, is most frequently found in the brain of sheep and more rarely in the spinal cord. It has also been found in other herbivorous animals—ox, goat, reindeer, roe deer, antelope, dromedary, and horse. No authentic case of _Cœnurus cerebralis_ in man has been recorded. A number of isolated cases are on record in which bladderworms resembling _Cœnurus cerebralis_, and probably identical with it, have been found in locations other than the brain. Eichler and Nathusius have reported such bladderworms in the subcutaneous tissue of sheep, and Heincke (1882) has
recorded the presence of a *C. cerebralis* in the eye of a horse. Rabe (1889) discovered in the brain, muscles, lymphatic glands, and thyroid body of an antelope specimens of a bladderworm which he identified as *C. cerebralis*. In general, however, *C. cerebralis* fails to develop in any other location than the central nervous system, and the embryos which do not succeed in reaching the brain or spinal cord soon degenerate.

The bladderworm (figs. 1, 2) consists of a membranous vesicle, the wall of which bears numerous small invaginations resembling tapeworm heads (fig. 3), each one of which, when swallowed by a dog, is capable of transforming into an adult tapeworm, *Taenia cancrum*. In its growth the vesicle tends to assume a spherical form and attains a size varying from that of a hazelnut to that of a hen's egg. The form of the bladder is modified according to its location. When situated in the spinal canal it becomes much elongated. The wall of the bladder is very...
thin, translucid, and contains contractile fibers. It is filled with a colorless, watery fluid containing 1 to 2 per cent of animal matter and less than 1 per cent of inorganic salts. The heads of the larval tapeworms, which appear as little white spots arranged in irregular groups and usually confined to one region of the vesicle, probably corresponding to the posterior part of the embryo, may number more than 500 in a single specimen, but usually they are not so numerous. They are generally found invaginated and project into the interior of the vesicle; but Davaine (1858) has observed that they are capable of evagination, and thus become an active source of irritation to the brain. Not all of the heads are found equally developed. Some of them can not be distinguished from the head of the adult, while others are rudimentary.

The hooks, suckers, and other details of the head are similar in the larva and in the adult, and will be described in connection with the description of the latter.

DESCRIPTION OF THE GID TAPEWORM (TENIA CENURUS).

(Figs. 6-8, see also figs. 3-5, 9.)

Tenia cenuurus, the adult stage of the gid parasite, is found in the intestines of dogs, and is said to occur also in foxes, and probably also in wolves.

It is not known to occur in other animals. Another tapeworm of the dog, Tenia serialis, is often mistaken for it. (Compare figs. 4, 5, 8 c, 9.)

Omitting an extensive discussion of the anatomy of the worm, the essential characteristics of Tenia cenuurus are as follows:

The average length is 40 to 60 cm. (16 to 24 inches) and the maximum 1 meter (40 inches). It possesses a head, or scolex, an unsegmented neck, and a long segmented portion consisting of two hundred to two hundred and fifty segments, or proglottids (fig. 6).

The fully developed head is pyriform and measures about 0.8 mm. ($\frac{1}{8}$ inch) in diameter. It is supplied with four suckers, 0.30 mm. ($\frac{1}{30}$ inch) in diameter, and a rostellum, also about 0.30 mm. in diameter, bearing a double crown of 22 to 32 hooks (fig. 4) of two kinds, large and small, arranged alternately. The suckers and rostellum
serve to attach the worm to the intestinal wall of its host. The large hooks measure 150 to 170 μ (1/8 to 1/6 inch) and the small hooks 90 to 130 μ (2/10 to 1/5 inch) in length.

The neck is slender and 2 to 3 mm. (1/8 to 1/3 inch) long. In the region of the neck the segments (Fig. 6) are very short and narrow, but toward the posterior end of the worm they gradually increase in size. The last twelve to fifteen segments are much longer than broad, in form somewhat resembling cucumber seeds, and measure 8 to 9 mm. (about 1 inch) long by 3 to 5 mm. (1/4 to 1/2 inch) broad. Near the middle of the right or left lateral margin of each segment is a small opening—the genital pore.

During the life of the worm new segments are continually being added in the neck region by a process of intercalation. The segments when first formed are small and sexually undeveloped. Gradually, however, they are pushed backward by still younger segments which develop in front of them. They grow larger in all dimensions, and the sexual organs develop (Fig. 7), reach maturity, and produce eggs, which are stored up by thousands in the uterus of each segment. Finally the gravid segments break away from the end of the chain and pass out of the dog's intestine. It results from this manner of growth that the small segments farthest anterior are the youngest, and the
large hindermost segments are the oldest; thus, in a single worm, segments in all stages of development may be found.

The most conspicuous structure in the posterior segments (fig. 8 C) is the uterus filled with yellowish or brownish eggs. The gravid uterus has a median stem with eighteen to twenty-six lateral branches on each side. The latter are almost parallel to each other, and are unbranched or only slightly branched.

The eggs, found in the gravid uterus, contain embryos with six hooks and measure from 31 to 36 μ (about \(\frac{1}{400}\) inch) in diameter.

**LIFE HISTORY OF THE GID PARASITE.**

As already stated, the gid bladderworm, *Ctenurus cerebralis*, and the tapeworm, *Taenia cœnurus*, are intimately related, the former being the larval stage of the latter.

In detail the life history is as follows: A dog infected with *Taenia cœnurus* scatters the eggs and gravid segments of the tapeworm over the ground. Those eggs which fall into moist places may live for several weeks, but those exposed to hot, dry weather preserve their vitality only a few days at most. Sheep and cattle while grazing or drinking are liable to swallow some of the eggs thus spread broadcast by the infested dog. By the action of the digestive juices the shells of the eggs are dissolved and the embryos contained set free. The embryos then bore out of the intestine by means of their hooks and wander among the tissues. This migration is undoubtedly aided by the blood current in vessels penetrated by the embryos, which are thus carried passively to various parts of the body. Normally only those embryos which reach the central nervous system continue their development. Others failing to reach this location may live for a while, but generally die and soon disappear. After reaching the
brain or spinal cord, which may occur within eight days after infection, the embryo loses its hooks and transforms into a small cyst. The young eceenurus preserves its power of locomotion for some time and may burrow along the surface of the brain, leaving a shallow, sinuous furrow (fig. 10) in its wake, but within a few days it becomes stationary. These furrows are to be found fourteen to thirty-eight days after infestation; near the termination of each is a small vesicle, the young gid bladderworm. As determined experimentally by Baillet, the vesicle, two or three weeks after infestation, has attained a diameter varying from 0.6 to 3 mm. (√₄ to ½ inch); on the twenty-fourth day it is as large as a pea, but the wall still remains thin and transparent. The heads begin to appear by the thirty-eighth day, when the vesicle has reached the size of a cherry, but they do not seem to attain their full development before the end of two or three months. The vesicle, however, continues to grow, and new heads are in continuous process of formation, so that in addition to fully formed heads, very rudimentary heads and intermediate stages between the two are likely to be found on the surface of the same bladder.

When a brain containing a bladderworm is eaten by a dog, the wall of the vesicle is digested, but the heads remain uninjured by the digestive juices and attach themselves to the wall of the intestine and develop into the adult tapeworm. In one to two months the posterior segments of the worm become gravid with eggs, and breaking away from the rest of the chain begin to pass from the dog's intestine.
Susceptibility of Sheep and Other Animals.

Gid is exceptional in sheep over two years old, the great majority of animals which present symptoms of the disease being lambs and yearlings. The disease is more rare among cattle and goats, and still more rare in other animals. Cattle seem to be susceptible for a longer period than sheep, and although gid is more frequent in calves and yearlings, animals as old as five or six years are still subject to infestation.

Certain breeds of sheep seem to be more susceptible than others, and in some cases this has been explained by the fact that some breeds graze closer to the ground and thus are more liable to take in tape-worm eggs and segments than others. The greater susceptibility of young animals has not been fully explained, but it has been supposed that the embryos of the worm are unable to penetrate the firmer tissues of adult animals, and consequently do not reach the brain.

Symptoms.

The following account of the symptoms of gid is based mainly on the description of the disease in sheep, given by Neumann (1892). The symptoms in cattle and other animals are of the same general character as those in sheep, showing minor differences probably in correspondence with the different natures of the animals affected. The type of symptoms varies according as the bladder worm is located in the brain or spinal cord. If the parasite is located in the brain it gives rise to cephalic gid; if in the spinal canal, to medullary gid.

Cephalic gid.—Unless the infestation is very heavy no symptoms are likely to appear during the stage of invasion of the parasites, or they may be slight and pass unnoticed. When early symptoms occur they appear generally in the second or third week following infestation. The affected animal shows signs of congestion of the brain, there is indifference and weakness, the head is of unusually high temperature and is held in an abnormal position, and the eyes are inflamed. Peculiar actions may become evident.

Frequently the animal turns in circles, or wheels about in one spot as on a pivot. In some cases it staggers about as though intoxicated, stumbles, and often falls. The eyes are turned in or out, there is grinding of the teeth, and sometimes convulsions. The animals may die in a few days in severe cases, but generally the symptoms disappear in eight or ten days, and a period of apparent recovery follows, corresponding to the period of growth of the parasite. In about 2 per cent of animals attacked the parasites apparently die, as no later symptoms develop, but in the vast majority of cases, at the end of four to six months, symptoms appear which mark the final stage of the disease, leading almost invariably to a fatal termination within a few
weeks at most. Usually these symptoms are the first to be noticed, as the initial symptoms are generally so slight that they pass unrecognized. Prior to the onset of the final symptoms and during the growth of the parasite certain slight signs of an abnormal condition of the brain will be noticed from time to time by the careful observer, especially in stormy weather. The time at which the symptoms of the final stage of the disease appear varies somewhat and according to the time at which infestation occurred, but it is generally toward the end of winter or in the spring. The symptoms at this stage may be considered as due in part to the pressure of the coenurus bladder on the brain, and in part to the active irritation produced by the larval tape-worm heads which are capable of being thrust out from the surface of the bladder. The symptoms vary according to the region of the brain occupied by the parasite.

Generally, however, the affected sheep holds his head in an unusual position, his eyes are fixed, with dilated pupils, and he pushes against obstructions. He becomes feeble, loses his appetite, and lags behind the flock. He moves in circles which gradually become smaller and smaller until he turns as on a pivot, and he usually turns toward the side on which the brain is compressed. If the parasite is located in the anterior part of the brain, the sheep is likely to move straight forward, holding his head down and lifting his feet high. A great variety of other movements may be executed, the type of movements depending upon the locality of the brain affected. A characteristic of all these peculiar actions is their automatic and intermittent nature. They recur several times during the day, and may continue to appear during a period of four to six weeks, at the end of which the animal dies from paralysis or exhaustion.

Medullary gid.—When the gid parasite is located in the spinal cord it generally occurs in the lumbar region, and the disease is then termed lumbar gid, or hydatic paraplegia.

The chief symptom of lumbar gid is the gradually increasing weakness and paralysis of the hind quarters. The bladder and rectum become paralyzed, the wool is shed, and the animal becomes progressively thinner, although he may retain his appetite and eat enormously. This condition may persist for several months before death occurs from general debility and exhaustion.

**PATHOLOGY.**

(Figs. 10-12.)

Neumann (1892) gives the following account of pathological lesions occurring in gid:

Pathological anatomy.—At the autopsy of animals which have died of gid one or more Coenures are found in the cranium, as much more developed as the disease has been prolonged. When speaking of its etiology, and of the experiments which have
established this, the developmental phases of the parasitic vesicle were described. In practice, the number of Coenures is generally limited, though Huzard has counted more than thirty in the head of a lamb. Their size and development are generally in inverse proportion to their number. Huzard has seen some which occupied nearly one-half of the cranial cavity.

These vesicles are found at various parts of the brain. When they are small and deeply situated they are surrounded by a pseudopurulent exudate, which is yellow and creamy in places. If the Coenurus is voluminous the brain substance in its vicinity is depressed, atrophied, wasted, and forms a more or less regular pouch, the wall of which is formed of flexuous, interrupted, or broken nerve tubes that are less numerous than in the normal substance; by nerve cells which are no longer in communication with the nerve tubes; by a large quantity of amorphous substance and molecular granules; and lastly, by calcareous crystalline particles. Capillary vessels traverse this layer, and are continuous with those of the cerebral substance (Robin, quoted by Reynal). This pouch contains a grumous purulent matter, and the peripheral cerebral substance is inflamed, dense, and granular.
When the Ctenurus is in a ventricle, the roof of this is thinned and sometimes reduced to its envelopes; the septum lucidum, posterior pillar of the fornix and the corpus callosum are also thinned, and even perforated, and pushed toward the hemisphere or into the opposite ventricle, into which the vesicle sometimes protrudes.

The old and voluminous Ctenures, situated on the superficial parts of the brain, have, by compression, caused absorption and attenuation of the roof of the cranium, which yields to pressure of the fingers (fig. 12).

In medullary gid the Ctenurus is elongated, fusiform, and from 3 to 5 cm., and even a foot long (Numan). Usually there is only one and it is lodged in the lumbar region; it may, however, occupy the cervical region (May), or the posterior part of the medulla oblongata (Störing). In some cases it has undergone calcareous degeneration and is only recognizable by its hooks, which have persisted (Röll). It sometimes only occupies one-half of the spinal cord, sometimes the two divisions, or it is intermediate, and completely separates them (Yvart). It may be situated deeply or only be beneath the arachnoid. The medullary substance is atrophied and hyperemic where it is located or it is softened. The muscles of the hind quarters are wasted and otherwise show the alterations of cachexia.

In a large number of animals there are found in various organs—and especially the heart, lungs, liver, spleen, mesentery, and muscles—some round or ovoid greenish corpuscles from 1 mm. to 4 mm. in diameter. They are composed of an enveloping membrane and granular contents rich in fat globules. These are atrophied and degenerated Ctenures, the remains of erratic embryos which have not met with the conditions favorable for their development. Natimsius and Eichler have seen somewhat large Ctenures in the subcutaneous connective tissue of the calf and sheep.

The malady having generally a chronic course there are observed more or less everywhere the lesions of pronounced cachexia.
DIAGNOSIS.

In diagnosing gid care should be taken not to confuse it with vertigo due to heat, epilepsy, blindness, false gid due to grubs in the nasal cavities, and inflammation of the intracranial sinuses. Likewise, medullary gid should not be confounded with the trembling disease (Scotch louping-ill) or lumbar prurigo.

In some respects the symptoms of gid present many similarities to those of the so-called loco disease of the Western States, and it does not seem unlikely that in some cases the two diseases have been confused. The latter disease, however, is more chronic in its course and does not present the acute symptoms of gid. It is also likely to become evident in animals too young to allow of sufficient time for the development of symptoms of gid, except in cases where early symptoms occur corresponding to the stage of invasion of the parasite. Loco disease, moreover, rarely seems to be a direct cause of death, and the condition of "loco" when once established persists indefinitely.

When gid is suspected in a flock the best means for a positive diagnosis consists in killing one of the affected animals and examining the brain and spinal canal for the presence of the bladderworm.

TREATMENT.

Treatment of gid with drugs is useless, and, on account of the location of the parasite, the various methods of operation which have been practiced are also of little practical use. Trephining and removing the parasite, cauterizing, several days refrigeration of the cranium with ice, or continuous irrigation of the skull with cold water for a period of two weeks, has resulted in a cure in some cases. An operation may be attempted in the case of an especially valuable animal, but in general the surest economy is the slaughter of the animals as soon as symptoms become evident. In case the disease is recognized during the period of invasion the animals may be fattened and killed before the final stage of the disease begins or immediately upon the appearance of the final symptoms.

PREVENTION.

Prevention of gid is, comparatively speaking, a simple matter. With our complete knowledge of the life history of the gid parasite, it is possible to prescribe certain rules which, if observed, will effectually prevent the occurrence and spread of the disease.

All superfluous dogs should be killed. It is hardly necessary to insist upon the destruction of wolves and foxes, on account of the possibility of infestation from this source, since they are already sufficiently under the ban for other reasons. A certain number of dogs are, of course, indispensable, and these animals should be treated sys-
tematically at intervals for tapeworms. While the dog is undergoing treatment he should be kept tied up and all feces collected and burned or buried in quicklime. Prior to the administration of the vermifuge, a laxative, such as castor oil, should be given to empty the bowels, and the animal then starved or fed on milk or soup until the following day.

In giving fluid medicine to a dog the cheek should be pulled away from the teeth at the angle of the mouth, the medicine poured into the pouch thus formed, and the dog's head held until he swallows the dose. If the animal is refractory a cord thrown in two half-hitches over his nose will be of assistance in holding him. After the administration of the dose the dog should be tied short enough to prevent his lowering his head and getting rid of the medicine by vomiting.

Preliminary treatment with a laxative not only serves to clear out the intestines so that the vermifuge will act more vigorously, but usually also expels fragments of tapeworms, from the number of which some idea of the degree of infection may be gained. An experienced observer will also be able to determine by study of these fragments (figs. 8, 9) the species of tapeworms to which they belong. Whether segments appear in the dog's feces or not, the practice of occasional vermifugal treatment should not be neglected. To render more certain the expulsion of all tapeworms the treatment may be repeated in two weeks or a month.

The following notes on vermifuges and their administration are taken from Stiles (1898):

In selecting a remedy it is well to consider the following drugs. The doses (apothecaries' weight) here given and the remarks on the drugs are abstracted from French (1896).

The doses of pelletierine tannate are, for adults, 5 to 15 grains; puppies, one-half to 5 grains. Pelletierine is undoubtedly the most efficient and innocent taniacide for the dog we possess, but is not much used on account of its expense. French has frequently found it most useful when the stomach has refused to retain other remedies. It should be administered in gelatin-capsular form in conjunction with powdered purgatives.

*Aspidium* is perhaps the most reliable of all the vermifuges with the exception of pelletierine. For everyday practice it is to be preferred to all other remedies when given in the form of oleoresin. Doses: For adults, 15 to 40 minims; puppies, 5 to 15 minims. The dose of the liquid extract is the same.

*Kamala* is a very efficient taniacide with drastic purgative properties. Given in small amount as an adjunct to other taniacides, particularly to the oleoresin of male fern, it will be found a very valuable remedy. Doses: Adults, 15 to 30 grains; puppies, 3 to 15 grains.

*Brayera* (U. S. P.), *Cusso* (B. P.), yields kosin or konssin, to which it owes its taniacidal properties. It is one of the best and safest taniacides, its action being directly toxic to the worm, but it is too expensive for ordinary practice. The infusion (*Infusion brayerae*, U. S. P.) and fluid extract (*Extractum brayerae fluidum*) are both too bulky and disagreeable for administration to dogs. Kosin may be given in capsules in doses—adults, 10 to 40 grains; puppies, 10 to 20 grains. The drug usually acts as its own cathartic, but it is better to employ some adjunct for this purpose.

Powdered *areca nut*, when freshly ground, is a very good remedy for tapeworm.
When old, it will generally be found inert; consequently, it is best always to purchase the nut and grind or grate on an ordinary nutmeg grater. It is still largely used by British veterinarians and is a favorite with some Americans, but it can not be regarded as being either as effectual or easy of administration as the two preceding drugs. Its effects on puppies are not unattended with danger, on account of its great astringency; but with due regard to subsequent purgation it is a perfectly safe remedy. Mayhew's method of prescribing 1 to 2 grains to every pound weight of the dog is usually followed, but the smaller quantity will generally suffice, provided the powder is freshly ground. It may be conveniently given in gelatin capsules, accompanied or followed by a purgative.

Turpentine is a powerful remedy against tapeworms, but it is regarded as being somewhat dangerous from its liability to produce strangury and renal inflammation. These effects are said to be less pronounced after large than after small doses, but large doses are more liable to cause gastric and enteric inflammations. It can hardly, therefore, rank with the best remedies. Administer in emulsion with white of an egg, mucilage, milk, or oil. Doses: Adults 10 to 15 minims; puppies, 3 to 10 minims.

Doctor Hoskins has had very satisfactory results with this drug in puppies under 6 months of age and has never noticed any gastric or renal results. In very young puppies he rarely gives over 2 minims, carrying it up to 10 minims and repeating for two or three days on an empty stomach in the morning, allowing no food for an hour or two after its administration.

The following suggestions as to doses, compiled from various sources, are taken from Curtice (1890):

1. Allow 2 grains of freshly powdered areca nut for each pound of the dog's weight; administer dose in soup or milk, stirring it well, or by mixing it in butter or molasses. Follow in two hours with a tablespoonful of castor oil for a moderate-sized dog, giving the oil alone or in three times its quantity of milk.

Zürn advises 4 drams of areca nut for a large dog, 2½ drams for a medium-sized animal, and 1 dram for a small dog.¹

2. One teaspoonful of turpentine and two tablespoonfuls of castor oil given in a cup of milk. The final dose of physic is not given in this case.

3. Twenty drops of oil of male shield-fern, 30 drops of turpentine, and 60 drops of ether. Beat together with one egg and give to the dog in soup.

4. Hagen advises 80 grains of oxide of copper with 40 grains each of powdered chalk and Armenian balsam. Mix with sufficient water to make an adherent mass and divide into 100 pills. Administer one pill three times daily for ten days, in meat or butter.

5. Röll prescribes the following dose for large dogs; smaller doses should be given in proportion to the size of the dog:

(a) Two drams each of extract of male fern and of powdered male fern; or

(b) Decoction of 2½ ounces of pomegranate-root bark in water, reduced to 6 fluid ounces, to which add 1 dram of extract of male fern. Give in two doses, at intervals of one hour; or

(c) One-half to 1 ounce of konoso, made into pills, with honey or molasses and a little meal; or

(d) From 1½ to 2½ drams of kamala, stirred with honey or water, and given in two doses inside of an hour.

[a, b, and c should be followed in two hours with castor oil, but this is not necessary for d.]

¹The doses given by Zürn are two to three times larger than those prescribed by American authorities for freshly powdered areca nut, and should be used cautiously.
In addition to treating dogs with vermifugues to rid them of the gid tapeworm, and thus removing the source from which sheep and cattle become infested, it is also essential that precautions be taken in turn to prevent the dogs from acquiring the parasite. This is accomplished by preventing dogs from eating those portions of the carcasses of "giddy" animals—namely, the brain and spinal cord—in which the bladderworm is located. These portions should be destroyed by burning, or rendered, or they may be thoroughly boiled, and can then be fed with safety to the dogs.

Not only is it a matter of immediate economy to slaughter animals affected with gid as soon as the symptoms indicating the final stage of the disease appear, and while they are still in good flesh, but it is also advisable from the prophylactic standpoint in that it removes a source of infestation to the dogs, and thus indirectly protects sheep and cattle from infestation, provided, of course, that the heads of the slaughtered animals are properly disposed of as suggested above.