Meningo-Encephalitis.
(BLIND STAGGERS.)

BY

THOS. P. HASLAM,
Assistant in Corn Mold Investigations.

MANHATTAN, KAN.
SEPTEMBER, 1910.
DEPARTMENT OF VETERINARY SCIENCE.

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Work of the Department of Veterinary Science.

SCHOOL OF VETERINARY MEDICINE:
   Giving a full four-year course of instruction and graduating students with the degree of Doctor of Veterinary Medicine.
   Investigating corn mold and troubles caused by diseased corn and stalks.
   Production of anti-hog-cholera serum.
   Production of blackleg vaccine.
   State veterinary work of investigating contagious diseases.
   Veterinary work connected with the State Live-stock Registry Board.
   Supervision of testing cattle for tuberculosis.

   Special lines of investigation are taken up from time to time, as necessity demands.
   Bulletins are issued on these subjects, which, with the bulletins of other departments of the Station, may be obtained free of charge by residents of the state, by addressing the Director, Experiment Station, Manhattan, Kan.
Meningo-Encephalitis (Blind Staggers).

By Thomas P. Haslam, Assistant, Corn Mold Investigations.

This disease (I), commonly known as staggers, blind staggers, sleepy staggers and mad staggers, has occurred, in outbreaks of greater or less severity, in many sections of the United States. Among a large number of states in which losses have occurred, Kansas, Texas, Louisiana, North Carolina, Delaware and Arkansas have experienced the most trouble.

Previous to the establishment of the Veterinary Department of this Experiment Station we have no written record of the trouble in this state, but various farmers and veterinarians seem to have encountered cases at a very early date. In 1891, soon after assuming his duties as veterinarian of this Station, Doctor Mayo investigated a severe outbreak that extended over almost the entire state. He states that owing to the dry weather the corn crop was almost a total failure, and that a certain green mold, Aspergillus glaucus, had badly damaged the small amount of corn that was grown. To this mold he attributed the death of the horses. In 1902, and again in 1906, there were severe outbreaks in various portions of the state. Besides these general outbreaks there seems to be a continual loss of horses from staggers in any locality in which much corn of an inferior grade is fed.

During the outbreak of 1906 the writer began an investigation of this trouble under the direction of Dr. F. S. Schoenleber, veterinarian of the Kansas Experiment Station. Examination of corn from the seat of the outbreak failed to show the presence of Aspergillus glaucus, but did show three molds: Mucor rhizopodoformis, Rhizopus nigricans, and Fusarium sp. Accordingly an investigation of the properties of these molds was carried out by the writer for the next two years, and continued by Dr. P. J. Meenen during the years 1908 and 1909. Owing to insufficient funds progress was slow until July, 1909.

Note.—We are indebted to Dr. M. A. Barber of the Kansas University Medical School for many valuable suggestions, and to Dr. C. E. Basseler of Greensburg and Dr. F. O. Chase of Haviland, Kansas, for much aid in our field investigations.
when the writer was employed to devote his entire time to re-
search work upon this subject.

In addition to the results recorded in this bulletin, there has
accumulated a large amount of material of a purely scientific
nature which will be published later. While the exact cause
of the trouble has not as yet been discovered, there seems to
be no doubt that it can be produced by immature ears of corn
infected by the molds described later in this bulletin. Experi-
ments are being carried out at present to determine whether
such immature ears free from molds will produce the same re-
results, or whether these molds themselves produce a poison capa-
able of causing the staggers. When these experiments have
been completed, several points which are not as yet clear will
probably be decided. While in this state we have few authentic
records of cases occurring when the horses have not been fed
on corn, yet in other states severe losses of horses have oc-
curred when the grass in the pastures becomes moldy. Since
the disease in this state has occurred almost exclusively where
the horses have been fed upon this immature moldy corn,
these results are published as a preliminary report, at this
time, in order to prevent further loss of horses from this cause.

The work has been done under the direction of Dr. F. S.
Schoenleber, veterinarian of this Station. Much valuable as-
sistance has been given by Dr. L. W. Goss, pathologist of this
department; the autopsies on all of the cases recorded in this
bulletin having been held by him. Martin Dupray has been
for the past year a constant and efficient aid in these investi-
gations, and the analyses of the moldy feed were largely car-
rried out by him. Cultures of many of the molds encountered
in this work have been sent to Dr. Chas. Thom, of Storrs,
Conn., who has given much valuable aid in their identification.

SUMMARY.

The contents of this bulletin may be briefly summarized as
follows:

1. Feeding horses upon immature corn badly infected with
molds and worm dirt, with its accompanying bacteria, pro-
duces typical cases of staggers.

2. The extract of such corn is rapidly fatal to rabbits.

3. Aspergillus flavus, Aspergillus niger and Rhizopus
nigricans have been found abundantly on the corn only in lo-
calities which lose horses from staggers.
4. Corn containing any molds should be thoroughly cleaned or "floated" before feeding.
5. Mixing corn with bran and oats seems to lessen the danger.
6. Treatment to be effective must be begun early.
7. This department will analyze and report upon samples of corn free of charge. About one peck is needed, which should be sent, prepaid, to the Veterinary department of the Kansas State Agricultural College.

**DIAGNOSING THE TROUBLE.**

During the course of these investigations it was found that in some localities cases which were reported as staggers were in reality typical cases of parasitism produced in horses by the palisade worm (*Sclerostoma equinum, or Strongylus ar-matus*) in the intestines and blood vessels, the error arising from the similarity of some of the symptoms of the two diseases. Those familiar with the course of either disease rarely make this mistake. To avoid this error some of the symptoms seen in each trouble are given below in parallel columns:

<table>
<thead>
<tr>
<th>STAGGERS.</th>
<th>PALISADE WORM.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Previous history:</strong></td>
<td><strong>Previous history:</strong></td>
</tr>
<tr>
<td>Animal has been running in a stalk field or eating wormy or moldy corn, smutty grass, or decayed food.</td>
<td>Animal has been in pasture containing stagnant water, or has been watered from a dirty trough.</td>
</tr>
<tr>
<td><strong>Beginning:</strong></td>
<td><strong>Beginning:</strong></td>
</tr>
<tr>
<td>If the animal has been carefully watched some loss of appetite may have been noticed, otherwise the first symptoms observed will be that the horse stumbles over ordinary low objects. If the animal has not been worked these symptoms may be overlooked. Eyes either dull or excited.</td>
<td>The first symptoms may be any one or a combination of the following: Colic, restlessness, or weakness of the back.</td>
</tr>
<tr>
<td><strong>Development:</strong></td>
<td><strong>Development:</strong></td>
</tr>
<tr>
<td>A few hours after showing the above symptoms the horse will either develop (a) &quot;mad staggers&quot; or (b) &quot;sleepy staggers,&quot; or a combination of the two.</td>
<td>The animal is usually restless, falling or throwing itself down and getting up again in a few minutes. When no longer able to get up it struggles violently at variable intervals and paws so vigorously that it ultimately digs a half circle around itself in the dirt. Often it bites at its sides.</td>
</tr>
</tbody>
</table>

(a) **Mad staggers:**
The horse becomes very violent, runs through fences, into trees, machinery, etc., usually traveling
STAGGERS.

in circles. It cannot be driven or led through the barn door without running against one side. After a few hours the horse becomes quiet and the symptoms resemble those of sleepy staggers.

(b) Sleepy staggers:
The patient, either with or without having shown the above symptoms, becomes stupid and sleepy, leans against the side of the barn, or pushes against the manger; stands with legs spread apart in a characteristic manner; refuses to be led; but some cases can be made to go backwards.

Treatment:
If the intestines can be emptied and excretion stimulated soon after the first symptoms are noticed many of the cases will recover.

Recovery:
Few cases recover without treatment.

Duration:
Usually from six or eight hours to days.

Post-mortem:
Congestion of the intestines, brain and its coverings (meninges), are usually observed. (Some writers report softened areas in the brain.)

PALISADE WORMS.

Treatment:
After the symptoms appear treatment cannot greatly alter the course of the disease. The parasites should be removed from the intestines.

Recovery:
It is not unusual for cases to recover without treatment if the parasite does not invade the circulation to any great extent.

Duration:
Some cases die almost instantly, others show symptoms for months.

Post-mortem:
The finding of large numbers of palisade worms in a part of the large intestine (caecum), or enlargements (aneurisms) of the anterior mesenteric or other arteries.

RELATION TO CORNSTALK DISEASE.
The observations of this department have not been sufficiently extensive to determine the relation between staggers and the so-called cornstalk disease. The latter is the name popularly applied to many disorders of the digestive tract in cattle and horses, some of which are clearly not the result of any poison or toxin. It seems that neither the seasons nor the localities in which many cattle have died in the stalk fields coincide with those in which severe outbreaks of staggers in horses have occurred.
RELATION TO PELLAGRA.

At the first pellagra conference, held in Columbia, S. C. (2), Doctor Wood pointed out the similarity of pellagra in man and staggers in the horse, and expressed the opinion that the diseases are identical. A careful review of the literature, and feeding experiments with various animals carried out by this Station, have indicated that only man and the horse develop a recognized set of nervous symptoms from eating moldy food. In other animals, diseases of the stomach and intestines sometimes result and death occasionally follows from diarrhea and inflammation of the bowels. This is particularly true in the case of white mice, guinea pigs and rabbits. These experimental results agree with the practical observations of many farmers and stockmen. Almost all farmers, even in districts suffering from staggers, feed the moldiest portions of the corn to their hogs, apparently without injurious results. Some feeders exercise no care whatever in selecting the feed for their cattle, or, if they do, they select the best portions for their horses and feed the refuse corn to the cattle. As yet few reports of loss from feeding cattle moldy feed have reached us, though the practice can scarcely be recommended. If, as seems to be the case, man and the horse are peculiarly affected by moldy feed, then experiments upon horses have the greatest significance in the investigation of the very obscure trouble in man known as pellagra. The positive results of the feeding experiments recorded below add strength to the theory that pellagra may be caused by an almost exclusive diet of unsound or immature Indian corn.

CAUSE OF STAGGERS.

Several publications by various experiment stations have dealt with this disease. Their results have been for the most part negative. Foreign investigators have likewise obtained almost uniformly negative results when feeding horses, cattle, sheep or goats upon moldy feed. But Doctor Klimmer (3), of Berlin, one of the leading German veterinary authorities, in commenting upon these negative results, issues a warning against the use of such feed, saying that the frequent losses of stock following the use of moldy feed renders it very probable that the experiments were not sufficiently extensive from which to draw conclusions.

While a large number of experiments have been conducted
by the Veterinary Department of the Kansas Experiment Station during the past three years, only a brief summary of the work will be presented here, leaving a full report until the experiments now in progress are completed.

MOLDS ATTACKING KANSAS CORN.

Black cottony mold (*Mucor rhizopodoformis*), yellow-green dusty mold (*Aspergillus flavus*), grayish cottony mold (*Rhizopus nigricans*), and black dusty mold (*Aspergillus niger*), occur on the corn in great abundance under certain conditions. They have all been shown to produce more or less poison, and until more evidence has accumulated, corn infected with any of the above molds should not be fed to horses. These three molds occur abundantly upon the corn in districts losing horses from staggers, but are rarely found upon the corn where staggers does not exist. They do not seem to possess the power to attack uninjured ears of corn, but follow some injury, usually that produced by the corn-ear worm. If the ear is a thrifty, vigorous one, the growth of the mold will probably confine itself to the vicinity of the injured grains. But if, as in dry seasons, or because of poor soil, the corn is stunted, then these molds are able to cover the entire ear. This is especially true if the dry spell which partially kills the corn is followed by a warm wet spell, which favors the growth of the mold.

EXPERIMENTS WITH MOLDS AND BACTERIA GROWN IN THE LABORATORY.

Rabbits, guinea pigs and horses have been fed, for periods of about one month each, upon food in which was mixed a liberal supply of mold, without results. The molds employed were *Aspergillus flavus*, *Aspergillus niger* and *Mucor rhizopodoformis*. The latter mold kills rabbits in three days when injected into their veins, but three rabbits were maintained for nearly a month upon feed which was black with this mold without noticeable injury.

At the present time this department is feeding horses upon molds grown in the laboratory, and the result will tend to show whether the molds or the immature corn itself is the cause of the trouble.

Of the bacteria found upon the corn, one species (belonging to the *Aerogenes* group) is usually fatal to horses and rabbits when injected into their veins. It does not develop in the blood and often entirely disappears from it if the animal does
not die for some time. Bacteria which have been heated to boiling for half an hour are just as injurious as the living ones. This bacterium is always present in the excrement of the corn-ear worm, and exists in large quantities in the so-called worm dust of corn.

**FEEDING EXPERIMENTS WITH MOLDY CORN.**  

*Analysis of Material Fed.*

The moldiest corn available in the state was obtained and graded as follows: The length, average diameter of each ear, and number of square inches damaged by each species of mold, were measured, and from the data thus obtained the average content, for the entire experiment, of each mold given below, was calculated:

Total area damaged by all molds......... 24.54 per cent.  
Area damaged by *Aspergillus flavus*....... 1.58 "  
" " by *Aspergillus niger*.............. 3.64 "  
" " by *Fusarium* Sp.................. 5.92 "  
" " by *Penicillium glaucum*......... 4.15 "  
" " by *Diplocodia zea*.............. 6.15 "  
" " by *Rhizopus nigricans*........ 2.85 "  
" " by other molds................... 0.254 "

Toward the close of the experiment some of the corn was ground and analysis made by the plate method. This gave about fifty million bacteria per gram of the dry substance, thirty-three million of which were of the species producing the poison.

Analysis of this same sample by plating upon Pasteur agar and corn-extract agar gave the following results:

Total number of mold spores per gram of corn.. .......... 21,000,000  
Number of *Aspergillus flavus* spores per gram........ 6,000,000  
" " of *Aspergillus niger* spores per gram........ 2,000,000  
" " of *Fusarium* Sp. spores per gram........ 12,000,000  
" " of *Penicillium glaucum* spores per gram...... 400,000  
" " of *Rhizopus nigricans* spores per gram..... 600,000  

That only a small portion of the spores present were capable of germinating may be seen from the fact that, by the use of appropriate stains and special counting apparatus, the material could be shown to contain 292 million mold spores per gram. A gram of the ground corn is about the quantity of meal that will lie on the point of a large pocket knife.
Extracting the Poison.

After a series of preliminary experiments, in which alcohol, ether and water were employed, the following method was shown to give the best results: One hundred grams (¼ lb.) of the finely ground corn is mixed with 1000 cc. (1 qt.) of water and heated, with constant stirring, at 70 degrees centigrade (158˚ F.), for four hours, allowed to stand until it has settled and the somewhat cloudy liquid is siphoned off and concentrated at 70 degrees centigrade until it is evaporated to 30 cc. When this concentrated extract in quantities of 30 to 60 cc. is introduced into a rabbit's stomach, the animal dies in from one and one-half hours to two days. Similar tests of sound mature corn have given negative results. Unfinished experiments with pure molds and with immature corn have shown that the extracts of both are capable of producing death in a rabbit when introduced into its stomach.

Results of Feeding the Corn.

On November 8, horses Nos. 1, 2, 3, 5, 6 and 7 were placed upon a diet of this corn. From November 9 to 14, Nos. 2 and 3 received about one kilogram of ear corn (approximately 2½ lbs.) twice daily. All of the horses ate well and seemed in every way normal.

On November 14 a gradual increase was made and continued until Nos. 6 and 7 were receiving 1800 grams (approximately 4 lbs.) twice daily, and Nos. 1, 2, 3 and 5, 2200 grams (approximately 5 lbs.). About the 1st of December No. 7 lost its appetite, and December 12 was taken off the experiment. November 27 No. 4 was placed upon the same corn and the amount gradually increased to 2200 grams (approximately 5 lbs.) twice daily. On December 17, horse No. 6 was found dead at noon, having died apparently without a struggle. Post-mortem showed congestion of the gastro-intestinal tract, but it was impossible to ascertain the cause of death. On the 21st of December, horse No. 1 developed a typical case of staggers. Horse No. 2 developed staggers on December 22, horse No. 3 on December 27, and horse No. 4, which was placed in the feeding experiment November 27, succumbed on January 14 with staggers. On January 13 horse No. 5 was used for another purpose because the supply of corn was exhausted.

Arranging these results in tabular form, we see that four-sevenths, or 57 per cent, of the number of horses started upon
the experiment died of staggers after eating the corn forty to fifty days, and that four-fifths, or 80 per cent, of those which ate the corn for forty days or longer died. Most of the horses used in these experiments were aged animals.

RECORD OF CASES.

The symptoms and post-mortem findings of these horses are given below:

Case No. 1.- On December 21, at 9 o’clock A. M., some difficulty was experienced in leading the horse to water, and the animal seemed quite nervous. At 9:30 A. M. the horse was trembling all over, could not be led or forced to move for some time, and when finally induced to move walked stiff-legged, going sidewise with a very unsteady gait, walking in a semi-circle to the right, from the south to the north end of the barn. After a great deal of trouble the horse was finally placed in a box stall, where he leaned up against the side and kept bumping his head. At about 10 A. M. he went around the stall leaning against the wall all the way. He then began to show signs of great nervousness, exhibited violent symptoms for a few minutes and then floundered down. When down he was very violent, striking with his feet and throwing his head. He drew his head around and bit at his side for several minutes, and then made several unsuccessful attempts to rise, gnawed the ground a while, and then became quiet. Temperature at 10:15 A. M., 100.6 degrees. At 11 A. M. he was resting his nose on the ground, partly closing his nostrils; otherwise about the same as at 10:15 A. M. At noon he had changed his position in the stall by attempting to rise. He lay flat on his side part of the time and attempted to rise, but could not do so Pulse 78, regular; temperature, 100.5. At 12:30 P. M. and at 1 P. M. was lying flat and quiet, but struggled when touched.
At 1:30 p.m. he had passed into a sleepy (comatose) condition, the only response to prodding being twitching and jerking of the muscles (exaggerated reflexes). This condition continued until death took place, at about 6:30 p.m. During the last few hours he breathed with violent flank convulsions and showed a marked jugular pulse. Post-mortem examination was held two hours after death.

Peritoneum: Slightly congested.
Small intestine, lymph glands and mesentery: Ecchymoses just under the peritoneum; mesentery slightly congested; lymph glands normal.
Large intestine and lymph glands: Ecchymoses on wall; contents normal.
Spleen: Slightly congested; hematoma on surface at central part.
Kidneys: Slightly congested.
Liver: Slightly congested.
Pleura: Small amount of organic exudate.
Lungs: Showed petechise upon surface.
Brain and meninges: Slightly congested.
Spinal cord and meninges: Slightly congested.
Other organs or parts: Normal.
Resume of morbid changes: Indications of toxæmia.

Cultures were made from the blood and viscera as follows:

<table>
<thead>
<tr>
<th>Material</th>
<th>Medium</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 cc. of blood from jugular vein a few minutes before death.</td>
<td>Nutrient bouillon</td>
<td>No bacteria developed.</td>
</tr>
<tr>
<td>3 cc. of heart blood 3½ hours after death.</td>
<td>Nutrient bouillon</td>
<td>No bacteria developed.</td>
</tr>
</tbody>
</table>

Portions of the various organs, about .5 cm. in diameter, were placed upon the surface of sterile agar in large test tubes, as follows:

<table>
<thead>
<tr>
<th>Organ</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spleen</td>
<td>No molds.</td>
</tr>
<tr>
<td>Liver</td>
<td>No molds.</td>
</tr>
<tr>
<td>Brain</td>
<td>Aspergillus niger.</td>
</tr>
<tr>
<td>Brain</td>
<td>No molds.</td>
</tr>
</tbody>
</table>

Case No. 2- The first noteworthy symptoms were observed December 22, 1909, although two days previous the animal ate and drank very poorly, and on the 21st did not drink at all. At 1:30 P.M. the animal was found leaning against the side of the stall and showing marked paralysis of the muscles of the nose and neck. When disturbed he would turn in a circle to the left. Breathing was about normal, and the animal responded when spoken to sharply. The temperature was 100.5. The symptoms remained the same, refusing food and water on
account of the paralyzed condition of the muscles of the mouth and throat, until December 25, when he was able to eat and drink a little; but again on the next day he was unable to eat or drink. Respiration still remained normal. Temperature 100.5. These symptoms continued until December 31, the animal having stood for hours at a time leaning against the side of the stall, or pressing its head and nose against the side of the barn. Death occurred without additional noteworthy symptoms.

Post-mortem was held a few hours after death and showed conditions exactly similar to those described in the cases of No. 1 and No. 3. Cultures were made in bouillon and pieces of the various organs were removed, with all the usual precautions, and placed on the surface of nutrient agar in large tubes, with results as follows:

<table>
<thead>
<tr>
<th>Source of material.</th>
<th>Medium.</th>
<th>Results.</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 cc. blood from heart, 2 tubes of nutrient bouillon.</td>
<td>No bacteria after twelve days anaerobic incubation.</td>
<td></td>
</tr>
<tr>
<td>Portion of spleen, 2 tubes of nutrient agar,</td>
<td>No mold.</td>
<td></td>
</tr>
<tr>
<td>Portion of lymph glands, 2 tubes of nutrient agar,</td>
<td>One tube developed Aspergillus flavus.</td>
<td></td>
</tr>
<tr>
<td>Portion of brain, 8 tubes of nutrient agar,</td>
<td>One tube developed small Mucor.</td>
<td></td>
</tr>
<tr>
<td>Portion of lung, 2 tubes of nutrient agar,</td>
<td>One tube showed a Mucor, the other Aspergillus flavus and an unknown white mold.</td>
<td></td>
</tr>
<tr>
<td>Portion of liver, 2 tubes of nutrient agar,</td>
<td>No mold present.</td>
<td></td>
</tr>
</tbody>
</table>

Case No. 3.—On the evening of December 25 it was noticed that he did not eat all of his feed. On the next day, at 8 A.M., he became violent and would pay no attention to his feed. An attempt was made to remove him to the hospital but great difficulty was experienced in doing so. He would not lead, and was finally backed out of the barn, when he fell to the ground, and after numerous unsuccessful attempts to rise finally regained his feet. When an attempt was made to lead him he fell to the ground again and experienced a similar difficulty in regaining his feet. He was then backed to the hospital, a distance of perhaps ten rods, and placed in a box stall. He now showed great nervous excitement and constantly leaned against the side of the stall. Temperature 100.7.
At noon, spells of restlessness alternated with periods of quiet, in which he would lean against the wall of the stall. This condition lasted throughout the afternoon. Toward evening he began stumbling blindly about the stall, butting with great force into everything that came in his way. He finally was allowed to get out, when he would walk rapidly and unsteadily in a large circle to the left, running into a fence several times, and finally falling down. He remained in a passive condition for about ten minutes, after which he rose to his feet. At this time there was only one attendant with him, and two hours were spent in an attempt to get the animal to the hospital again, during which he had spells of restlessness in which he walked in circles, and spells of rest in which he leaned against the fence. The active periods lasted for about ten minutes and the periods of rest for about two to three minutes each. At 11:30 P.M., after a number of unsuccessful attempts to lead him, owing to his tendency to turn to the left, he was backed into the hospital. During the remainder of the night he showed the alternate periods of rest and excitement. At 6 A.M. he went down, unable to rise again. During the latter part of the night he showed paralysis of the muscles on the left side of the tail. When he was down he was very violent, struggling considerably. At 8:30 A.M. his temperature was 103.

Death occurred that afternoon at 4:15 after about thirty hours of illness; violent struggling, groaning and gritting of teeth continuing to the end. Post-mortem was held in a few hours, with the following findings:

- Subcutis: Some bruises.
- Small intestine: Slight congestion; ecchymoses, size of a pea, on wall.
- Mesentery: Yellowish red.
- Large intestine, with contents and lymph glands: Contents normal; a few ecchymoses as on small intestine.
- Kidneys: Slightly congested.
- Mouth cavity wounded.
- Pleura: Congested; fluid contents of the pleural cavity slightly increased.
- Lungs: Some congestion.
- Pericardium: Slightly congested.
- Heart blood vessels and contents: Normal, except ten to twelve Pali-sade worms in the mesenteric artery.
- Brain and meninges: Congested.
- Spinal cord and meninges: Normal.
- Resume of morbid changes: Indications of toxæmia.
Except in the case of the specimens from the lung and spleen, an instrument was employed which was made by grinding a glass tube about one centimeter on the emery wheel to resemble a cork borer. A piston was made by wrapping a wooden rod with cotton. The entire instrument was wrapped in paper and sterilized by steam at fifteen pounds pressure. With these tubes, cylinders of tissue were readily taken from the brain and placed in sterile test tubes. In the other organs the following method was employed:

After searing an area about six inches in diameter, the tube is held at right angles to the organ and by means of a rotary motion is pushed deeply into the tissue. The tube is then inclined until it is almost parallel to the surface of the organ and pushed in for two or three inches. By still further inclining the tube and pushing, the cutting edge will penetrate the sterile surface of the organ, two or three inches from the point of entrance. Still holding the tube parallel to the surface and pulling upwards the tissue between the two points is torn and the tube freed. The cylinder of tissue contained within the glass tube is pushed out directly into a sterile tube with the piston.

Cultures on bouillon were made from the heart blood and viscera as follows:

<table>
<thead>
<tr>
<th>Material</th>
<th>Medium</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 cc. blood from jugular just before death</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Heart blood, 5 cc. taken 3 hours after death, Fluid from ventricle of brain</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>No medium added</td>
<td></td>
<td>Remained clear and showed no organisms upon microscopic examination.</td>
</tr>
</tbody>
</table>
Case No. 4.—Was fed from November 27 to January 14, when at about 10 A. M. it was noticed that the animal was suffering from severe nervous excitement. According to the report of the hospital attendant, when he attempted to lead her from the stall she became very violent and lunged about striking the sides of the stall with great force and experiencing much difficulty in getting through the door. When outside of the barn, the ground being very icy, she slipped and fell and lay struggling violently. After several unsuccessful attempts to help the animal to her feet, owing to the extremely icy condition of the ground, she was placed on a sled and taken to the hospital. About two hours was consumed in this, during which she struggled constantly and frequently struck her head against the ice with great force. When she reached the hospital she was unconscious and died almost immediately. The post-mortem appearances did not differ from those already described.

In the culture tests special emphasis was placed on the search for a bacterium, and a number of anaerobic cultures were made.

<table>
<thead>
<tr>
<th>Source of material</th>
<th>Medium</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 cc. heart blood</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>5 cc. heart blood</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>5 cc. heart blood</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>5 cc. heart blood</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
</tbody>
</table>

(The above tubes were incubated in an atmosphere of hydrogen over alkaline pyrogallic acid, and after a week were removed and examined microscopically.)

<table>
<thead>
<tr>
<th>Source of material</th>
<th>Medium</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 cc. heart blood</td>
<td>No medium added</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>5 cc. heart blood</td>
<td>No medium added</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>5 cc. heart blood</td>
<td>No medium added</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>5 cc. heart blood</td>
<td>No medium added</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>One loop of brain substance</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>One loop of brain substance</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Two loops spleen</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Two loops spleen</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
</tbody>
</table>

(The above four tubes were incubated anaerobically.)

<table>
<thead>
<tr>
<th>Source of material</th>
<th>Medium</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spleen</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Spleen</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Brain</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Brain</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Liver</td>
<td>Nutrient bouillon</td>
<td>No bacteria.</td>
</tr>
<tr>
<td>Ventricular fluid</td>
<td>No medium added</td>
<td>No bacteria.</td>
</tr>
</tbody>
</table>
In the above, as in all other cases, plate cultures from the blood before and after death, and from the liver, kidneys, spleen and brain, on ordinary agar and on corn-extract agar failed to show any bacterium common to all cases. The plates usually remained entirely sterile. In some cases, especially those from the liver, kidneys and spleen, colonies developed, but they showed no uniformity.

**Interpretation of Results.**

From a practical point of view, the relation between corn and staggers has been quite thoroughly demonstrated. The question naturally arises, which of the micro-organisms present on the corn is the specific cause of the trouble, or whether a too exclusive corn diet (4) or the immature corn possesses the injurious properties. These questions must still remain open, but the indications are that the cause of the trouble is contained in the moldy portions, as sound corn from the same bin was fed to more than a dozen horses and mules for months without producing any disorder. The assumption that the symptoms were caused by a soluble poison, and not by the parasitic growth of the micro-organisms in the tissues of the horse, is supported not only by the fact that extensive cultural tests fail to show the presence of any bacteria or molds, but also that experiments now in progress have demonstrated the presence of poisons in this corn.

Dr. Carlo Ceni, of Italy, and his pupils report (5) that the molds are capable of producing poisons, but only at certain seasons of the year, and that in the winter as well as in the middle of the summer they are entirely inactive.

Another fact of great interest has been developed by Dr. M. Otto, of Germany, that while the extracts of two species of *Aspergillus fumigatus* obtained from Italy are very marked poisons, those from the same kind of molds growing in Germany possess little or no poisonous properties. The influence of the time of the year and the locality in which the molds grew may perhaps explain why loss of stock does not always follow the use of moldy feed and why pellagra has not always been found in corn-growing districts.
PREVENTION AND CURE.

Veterinarians accustomed to treating this trouble usually cure a small majority of the cases treated if the treatment is begun before the disease has progressed very far. The most successful line of treatment seems to consist in the subcutaneous administration of arecoline or eserine and pilocarpine, followed by a good antiferment, and perhaps also an aloe bolus. It is very evident that the best means of combating this disorder consists in prevention. No unsound corn should be fed to horses. If it is necessary to feed a poor grade of corn, it should be shelled and thoroughly cleaned with a fanning mill. Very good results have been obtained by the so-called floating of corn before feeding, which consists in pouring it into water. The moldy grains, being lighter, rise to the surface and may be skimmed off. Good results are reported by some who have ground the well-cleaned corn and mixed it with equal parts of bran and oats.

BIBLIOGRAPHY.

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3. Klimer’s Veterinary Hygiene, pp. 204-245.