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Cornstalk Disease of Cattle.—Preliminary Bulletin.

CORNSTALK DISEASE.

For many years, ever since corn has been grown extensively in this state, it has been a common practice to gather the corn from the standing stalks, and then turn cattle into the stalk fields, thus utilizing as forage quite a large portion of the stalks that would otherwise be wasted.

Since the practice of pasturing the stalk fields during the fall and winter has been followed, there have been serious losses reported among cattle that were running in the stalk fields. To the disease or condition which has caused the death of cattle under these circumstances the term "cornstalk disease" has been applied. While there are objections urged against this name by those who are of the opinion that the cause of death is indigestion, and also by others who believe that the cause of death is a poison, yet it seems best, by virtue of long usage and common interpretation to use the well-understood term "cornstalk disease" until the real nature of the disease is determined positively, and not manufacture new names which will only serve to confuse.

The cornstalk disease is extremely difficult to study satisfactorily owing to the uncertainty of its occurrence, the varied conditions and circumstances under which it occurs, and the rapid course of

the disease. It is rarely that a person can respond to a call to investigate this disease, if necessary to go any distance, and find an animal sick with the disease, and *post mortem* examinations do not furnish conclusive or satisfactory evidence as to the cause of death.

The loss to the live-stock industry of this state is difficult to estimate, but it is probably greater than that caused by any other disease except hog-cholera. It has been estimated that the loss from this disease in the state of Nebraska during the fall and winter of 1889-'90 was about $60,000. It is probable that this state suffers greater losses than any other on account of the large acreage of corn, the great number of cattle handled, and the usually mild winters, offering favorable opportunities for pasturing cattle in the stalk fields until midwinter or later. While the losses in the aggregate and often individually are severe, it is the opinion of many stockmen of experience that if cattle are carefully handled it pays to pasture the stalk fields; that the gain from utilization of the forage more than compensates for the losses of cattle which die from eating it.

There is a great diversity of opinion as to whether the cornstalk disease is increasing or decreasing in prevalence in recent years. The opinion seems to vary with the seasons; if there have been quite severe losses in a neighborhood in a season the opinion prevails that the disease is on the increase, and *vice versa*. It seems that the losses at the present time are not proportionately as great as they were in former years, but this may be accounted for, partially at least, by the better conditions surrounding cattle at the present time, both as regards food and shelter.

The cornstalk disease is not confined to any particular part of the state, but is liable to occur wherever corn is grown and the stalk fields pastured, especially if the pastures are dry and there is no laxative green food. From reports which I have observed, the disease seems to prevail most extensively in a belt comprising the central two-thirds of the state. The less frequent reports from the extreme west may be accounted for by the small amount of corn grown. In the extreme eastern part of the state, the reports may not have come under my notice, or it may be, as the country is divided into smaller farms and fewer cattle, the conditions for serious outbreaks of the disease would not be so great, while the proportionate loss may be fully equal to any other part of the state. The greater abundance of tame grasses, especially blue grass, which remains green later in the season, may also have some effect on lessening the prevalence of this disease.

The prevalence of the cornstalk disease varies in different years,

being most prevalent in years when there has been a large corn crop. This may be accounted for, partially at least, by the increased growth of stalks, which contain a relatively larger amount of indigestible material; it is noticed that the disease seldom occurs where the stalks are small and fine. More cattle are probably pastured in the stalk fields on account of the larger amount of stalks, and thus increase the fatalities.

Outbreaks of the disease are more likely to occur with, or follow closely after storms, especially cold rain storms. While the disease occurs at other times, the increased prevalence with or following storms seems remarkable. Whether this increase is due to the wet condition of the stalks, or whether it is caused by the well-known tendency of animals to eat large quantities of food just preceding a storm, or whether it is caused by a lowered vitality of the animal brought on by the storm, or to a combination of these, it is difficult to say.

The disease also seems more likely to occur when cattle are just turned into the stalk fields or changed from a stalk field that has been pastured to a new stalk field which has not been pastured. The increased tendency to the disease under these circumstances may be the result of indigestion, which was induced by a change of food when first turned into the stalk fields. It is well known that a sudden change of food in animals is liable to cause indigestion. Or the increase in the disease may be caused by engorgement or from some injurious or poisonous substance which may be upon or contained in the cornstalks themselves.

There is also a tendency for the disease to attack younger cattle, yearlings and two-year-olds in preference to older cattle. Whether this increased mortality is caused by indigestion induced by incomplete mastication, due to shedding of temporary teeth and cutting of the permanent teeth, or whether it may be due to the fact that the digestive organs of young cattle are not mature enough to thoroughly digest such coarse food, or it may be due to the susceptibility of yonger animals to poisons; for it is well established that younger animals require proportionately smaller doses of poison to affect them seriously than older animals.

The cornstalk disease occurs under a great variety and apparently diverse conditions. It may occur in one bunch of cattle, while another bunch of cattle in an adjoining field under apparently the same conditions are not affected. This great variation in the occurrence has resulted in many theories which have been advanced by stockmen and others as to the probable cause of the disease.

CORN SMUT.

A very common theory that has been held for some time, is that the disease is caused by eating corn smut *(Ustilago maidis)*. Corn smut is a fungus which attacks the growing cornstalk or ear of corn. As the corn matures the smut is readily recognized by the presence of large dark brown or black masses of spores. Cattle will often eat this corn smut quite readily, whether it is on cornstalks or corn in the field, or on fodder that has been cut and cured. As the cornstalk disease does not occur in cattle that are fed on corn-fodder even when the fodder contains unusually large quantities of the fungus, indicates that the corn smut is not especially injurious to cattle, and experimental evidence proves conclusively that corn smut is not injurious to sattle.

In 1868, Prof. John Gamgee, in investigating the cornstalk disease, fed experimentally 40 pounds of corn smut to two cows, beginning with six and increasing to 12 ounces daily. The smut was fed with ground grain and chopped hay. To one cow it was given wet, to the other dry. The cow that received the wet ration gained in weight during the trial, the other lost in weight, but both remained well.

Several years ago Mr. John Booth, a farmer living north from Manhattan, being of the opinion that corn smut was liable to produce the cornstalk disease, took pains to gather the smut from the field. One night his cattle broke into the inclosure where the smutty corn and smut had been thrown out and ate all they wished; no injurious effects were noticed.

The Bureau of Animal Industry of the United States Department of Agriculture made two experiments of feeding corn smut to cattle under the direction of Dr. F. L. Kilborne, as follows:

The load of corn stalks affected with smut *(Ustilago maidis)* removed from the field used in the preceding experiment (page 44) were fed to three two-year-old steers placed separately in box stalls in a barn on Mr. McCleary's farm. The animals had been in a corn stalk field for about five weeks prior to the feeding. They were fed exclusively on the smut-laden corn stalks and free smut mixed with a small quantity of a mixture of corn meal and wheat bran for seven days without ill effects. Owing to the absence of necessary appliances the weight of the animals and of the smut fed was not taken. It is safe to say that these animals consumed a much greater quantity of smut than the animals which died in the field. It was observed that the animals did not eat the smut readily in the field, and in this experiment the animals refused it until apparently hunger compelled them to eat. The mixing of mill feed with the pure smut made it more appetizing, and consequently a much larger quantity was eaten than otherwise would have been. The fact should be borne in mind that the smut used in this experiment came from a corn field in which several animals had died within five days after they were turned into it.

(2) In December, 1893, six barrels of corn smut were collected from several corn fields in which cattle had not been turned, near Van Meter, Iowa, and shipped to the experiment stations of this bureau. This material comprised both abortive ears with the husks attacked by the smut and the masses of pure smut found growing on the stalks. The total weight was 280 pounds, of which at least one-half was smut.

Two heifers, Nos. 238 and 284, 15 to 18 months old, were placed in separate box stalls. They were bedded with peat moss instead of straw, in order that their food should consist exclusively of the prescribed ration. They were allowed to run in a small yard (16 by 30 feet) for about five hours daily.

During the experiment the animals were given all the well water they wished. They were not salted. The smut was apparently not relished by the heifers, especially by No. 238, so that it required considerable inducement to get them to eat the desired quantity.

Beginning on the morning of January 17, 1894, and continuing until noon of February 2 (16½ days), the heifers were fed morning and evening with from two to three quarts of a mixture of equal parts by weight of cut hay and a mixture of corn-meal middlings and wheat bran, and 16 quarts of smut. The actual quantity of the fungus consumed by No. 238 was 61 pounds, or a daily average of nearly three and seven-tenths pounds, and by No. 284 67½ pounds, or a daily average of four and one-fifth pounds. The temperatures of the animals were taken every morning and evening. The animals appeared to be perfectly well throughout the time of feeding and continued so for several months, during which time they were kept under close observation.*

In the winter of 1895-'96, the Michigan Experiment Station also made some experiments in feeding smut to cows, some giving milk and others pregnant. They commenced with small doses of corn smut, and increased until enormous quantities were fed. The experiment continued for one month. No bad effects were noticed either on the digestive or nervous systems, and of the animals giving milk, the milk flow was not lessened.

In November and December, 1895, I had some corn smut collected from the College farm and other fields in the vicinity. One lot of 500 grams of pure smut was extracted with alcohol; the alcohol was then driven off by evaporation at the temperature of the room to 30 cubic centimeters. This was tested upon guinea pigs by giving them 2 and 3 c. c. doses. No ill effects were noticed.

Another test was made by taking the corn smut and steeping in distilled water slightly acidulated with sulphuric acid, and after filtration, the filtrate was tested with the following reagents for the presence of an alkaloid or nitrogenous base which might be poisonous: Mayer's reagent, iodine and potassium iodide, sodium phospho molybate, and platinic chloride. No reactions were obtained, nor were any reactions obtained by testing the evaporated

*Bulletin No. 10 U. S. Department of Agriculture, Bureau of Animal Industry. Cornstalk Disease and Rabies in Cattle.

alcoholic residue by dissolving in acidulated water and testing with the same reagents.

Such a mass of evidence from many sources shows conclusively that corn smut is not injurious to cattle and consequently is not the cause of the cornstalk disease.

BACTERIAL CORN DISEASE.

Another theory that has attracted considerable attention in the West is one advanced by Dr. F.S. Billings, formerly of the Nebraska Experiment Station. This theory attributes the cornstalk disease in cattle to eating cornstalks which are affected with a bacterial disease, known as the Burrill bacterial corn disease. When the germs of the diseased cornstalks were taken into the system of cattle with the stalks, the germs then caused a disease of cattle known as the cornstalk disease.

This disease of constalks was first studied and demonstrated by Prof. T. J. Burrill, of the University of Illinois. This disease of cornstalks is quite common in small stunted cornstalks, especially if grown on rather wet soil, and is shown by rusty brown spots on the leaves and stalks.

The theory that these diseased cornstalks produced the cornstalk disease in cattle was attracting considerable attention among farmers and stockmen in 1891 when I first began to investigate the cornstalk disease, so experiments were undertaken to demonstrate what influence, if any, the diseased cornstalks had in the production of the cornstalk disease.

On July 13, 1891, I received from Professor Burrill a culture of the germs or bacteria which produced the disease in growing cornstalks, and also specimens of fresh and dry cornstalks showing the appearance and effects of the germs upon the corn. From the original cultures sent me by Professor Burrill other cultures were made, and from these cultures two rows of growing corn 40 rods long were inoculated. Each stalk was inoculated in several places by making incisions into the stalk, and especially where the leaf sheath surrounded the stalks. The corn was making a vigorous growth and considerable difficulty was experienced in getting the disease to “take,” but by going over the corn several days apart and mutilating the stalks at the point of inoculation most of the stalks became affected with the disease, but not seriously as the growth of the stalks was not materially checked. These inoculated stalks when thoroughly ripened were gathered and fed to a two-year-old heifer which was confined in a stable. For eight days she was fed exclusively upon this inoculated corn fodder.

which she ate readily. Her temperature and pulse were taken twice daily, morning and night. The excretions and general condition were carefully watched, but no deviation from a normal condition was observed.

Cultures of the Burrill corn-disease gems were made in beef broth, and when the broth was decidedly turbid from the growth of germs, the same heifer was given doses of this culture, beginning with 100 c.c. doses twice daily for two days, without any apparent effect. Then two doses of 500 c. c. were given morning and night for one day. Her normal temperature had varied from 101 2-5 deg. F. to 102 3-5 deg. F. On the morning when this large dose was given her temperature was 102 deg., at night 103 l-5; the next morning temperature was 102 2-5 deg. F., and she was affected with a mild diarrhea. Blood was drawn from the ear and examined microscopically and inoculations made in nutrient agar, but no bacteria were found and the tubes remained sterile. The next day her morning temperature was 102 2-5, night 102 3-5. Nothing further abnormal in her condition was noted.

Fields of growing corn in the vicinity of Manhattan were examined to determine the presence of the Burrill bacterial corn disease. In all fields some was found, but it did not seriously affect any. One field near the College, belonging to Mr. McDowell, was found which seemed to have more of the diseased corn than others examined. Most of the diseased corn was along a low place, at times a water-course, that ran through the field. Into this field of about 25 acres 28 head of mixed native cattle were turned, about the middle of November. The field was well pastured off. No ill effects were noticed, the cattle all doing nicely.

The Bureau of Animal Industry of the United States Department of Agriculture, in investigating the cornstalk disease of cattle, also found that cattle ate cornstalks affected with the Burrill corn disease without ill effects:

A drought which extended over the greater part of the state of Illinois in August and September, 1893, dried up the pastures and compelled the farmers to feed their cattle. In many instances cornstalks were the only food given. As all of the corn was found to be affected with this disease, it was evident that no feeding experiment could be more efficacious than those already being made. Several herds of cattle that were fed exclusively on these stalks were carefully watched for a period of several weeks, but no evidence of disease appeared. The cornstalks fed were carefully examined and found to be thoroughly affected with the Burrill disease. It should be noted that the cattle ate these stalks much closer than they did the dried ones in the field later in the fall, and consequently they ingested many more of the diseased areas. It is of interest to add that, so far as it was possible to learn, the cornstalk disease did not appear among cattle in 1893 in the districts visited in the fall

when the mature cornstalks were known to be badly affected with the Burrill disease.

If corn affected with the Burrill corn disease is the cause of cornstalk disease in cattle, it seems probable that the disease would be more likely to occur among cattle that were fed corn fodder which had been gathered and sheltered so that the germs would not be weakened or destroyed by exposure to inclement weather. The cornstalk disease does not occur among cattle fed on corn fodder; on the contrary, it often occurs as late as February where cattle are pastured on stalks that have been exposed for months, the most inclement of the year. I have also examined bacteriologically pieces of tissue from five different animals said to have died from cornstalk disease and all from different outbreaks. The examination included an examination of the tissues microscopically for the presence of pathogenic bacteria, and inoculations of artificial media from the pieces of tissue, to try and grow and isolate some pathogenic or disease-producing germs. A number of varieties of bacteria were obtained, but all proved to be germs of decomposition, and not disease producing. The Bureau of Animal Industry, in the bulletin previously quoted, reports an examination of eight different animals that had died of cornstalk disease and the same results obtained. No pathogenic germs were found.

There are no indications that the cornstalk disease of cattle is a germ disease; in fact, all indications are that it is not a germ disease; and there is abundant evidence that cornstalks affected with the Burrill corn disease do not cause cornstalk disease in cattle, nor do such stalks have any injurious effect upon stock. Neither do large doses of pure cultures of the germs of the Burrill corn disease, when given to animals as a drench, produce serious effects.

Chinch-bugs in the standing cornstalks are occasionally suggested as a possible cause of cornstalk disease, but all the evidence is against such a theory, as much larger quantities of chinch-bugs are often fed in corn fodder and sorghum than could possibly be obtained in the standing stalks, and no ill effects follow. The presence of poisonous plants aside from cornstalks is also occasionally mentioned as a possible cause of death; but as the cornstalk disease often occurs after midwinter, when it is not possible for cattle to procure plants of a poisonous nature, this theory must be abandoned. The relation of salt to the cause of cornstalk disease will be discussed later.

All the evidence at hand seems to indicate that corn or cornstalks are the cause of the disease, and that the cornstalk disease results from indigestion, or irritation from the cornstalks, or the presence of a poisonous substance either in the corn or stalks.

SYMPTOMS.

For the symptoms of cornstalk disease it is necessary to depend upon the farmers and stockmen for descriptions, and these seem to vary as much as the conditions under which the disease occurs. It is quite probable that quite a number of cattle die in stalk fields from other causes than cornstalk disease, and the symptoms shown by such cattle are often confused with those of cornstalk disease. On the other hand, the variety of symptoms reported as those of cornstalk disease would indicate that the cornstalk disease may result from a combination of causes. The principal symptoms of what might be called typical cornstalk disease are as follows:

The disease comes on suddenly with very few premonitory symptoms. If the cattle are in the stalk fields, the affected animal is noticed lying or standing apart from the rest. If standing, the animal is usually "humped up," often switching the tail or twisting the rump in a peculiar manner, sometimes kicking at the belly. There is often a peculiar wild look to the eyes and other indications that the animal does not see readily, or comprehend what it does see. If the animal is disturbed it usually moves reluctantly, though if the brain symptoms are pronounced the animal often starts wildly and appears slightly delirious. If the animal's brain is not affected it usually moves with an uncertain, "wabbling" gait, often smelling of the cornstalks but never eating. As the disease progresses the symptoms of suffering and delirium are greatly increased, struggling, and moaning or bellowing. The respirations are increased and there is often some bloating. Death usually following within 24 hours after the animals are first noticed ailing.

OUTBREAKS REPORTED.

The season of 1895 was noted on account of the unusually large corn crop in this state, and there appeared to be a marked increase in the prevalence of the cornstalk disease in this state. The following reports will give some idea of the circumstances and conditions under which the disease occurs.

Mr. C. S. Jobes, of Attica, Harper county, writes, under date of November 30, 1895, as follows:

> I had an outbreak of the disease in my herd this week. I had 225 head up feeding them on cane, which was cut and cured in August last and has laid in shock ever since. They had been fed on this cane for two weeks or more. On last Monday they broke out of the lot and went at once into stalk field; they were not out to exceed one hour. On Wednesday morning two animals were found dead and by afternoon of Thursday 15 more died. I was away from home at the time and did not wire you as I should otherwise have done. Neither can I give you the symptoms minutely. The first stomach seemed

normal in nearly all cases, the second stomach (omasum or manifolds) was very hard in almost every case. I saved one of the latter and will send it to you. My foreman is firmly of the opinion that the wet, sleet-covered cane which they ate on Monday and Tuesday is responsible for the trouble, while I was equally confident that the stalks did the work. However, I have two steers that go where they please; they are constantly in these cane stalks and have suffered no inconvenience and are doing splendidly. There has been, I think, close to 100 or more head of cattle lost within a radius of five to seven miles of my place, all chargeable to cornstalks.

Mr. Jobes very kindly sent a manifold from one of the dead cattle and also a bundle of cornstalks which he thought caused the trouble.

The omasum (manifolds) was filled very full of partially digested food and was extremely hard and dry. It was impossible to make a dent in the manifolds by pressure with the fingers. It should be stated that this manifold had been removed from the animal six days before, and had probably dried out somewhat. It must also be remembered that the natural consistency of the omasum or "manifolds" is about that of a bag of putty. The partially digested food taken from the manifolds was examined chemically for the presence of alkaloids or nitrogenous bases, by extracting the food both with alcohol and acidulated water and testing with the common reagents. A faint trace of a reaction occurred, but this was probably the result of the decomposition of the animal tissue of the manifolds. This partially digested food was also tested to determine the presence of potassium nitrate (salt peter), but none was found.

The bundle of cornstalks received from Mr. Jobes was also examined for the presence of potash but no appreciable amount was discovered.

The next outbreak to be reported was by Mr. E. F. Gregory, of Garnett, Anderson county. Mr. Gregory is secretary and treasurer of the Enterprise Cattle Company, a close observer and of large experience in handling cattle. He writes as follows:

Garnett, Kas., December 4, 1895.

Dr. N. S. Mayo, Kansas Experiment Station, Manhattan, Kas. Dear Sir.—In compliance with your request published in the "Kansas Farmer," I report to you what we think to be a case of cornstalk disease.

About 10 days ago 75 head of steers were turned into an 80-acre stalk field. Cattle were supplied with plenty of water and salt, and due care taken in putting them onto that kind of feed. The second or third day after sleet encased almost every particle of food and cattle could get but little, so some shock corn was hauled out to them. About the fifth day or soon after ice melted, some of the cattle were noticed getting stiff, particularly in hind parts, getting up with difficulty, etc., but as dung showed quite a little corn and they

had not been used to any, it was thought that slight founder was the trouble or that rotten corn might have something to do with it. As the cattle had been pretty well over the field and the supply of good and rotten corn was pretty well exhausted it was decided to let them remain. The trouble did not cease. however, each day more steers showed stiffness, often falling down and unable to regain their feet without help. Some of the first and worst cases got better without medical aid, but as the number of ailing ones was increasing and some cases getting worse all the time, it was decided this morning that it was genuine cornstalk disease and cattle were taken from the field, leaving one dead, one dying, four unable to get up, and amongst those brought away half a dozen were reeling in their tracks.

I say it was "decided" a case of cornstalk disease, but mean only that in the writer's judgment it was such—think there is not much doubt of it as it certainly is not from impaction in stomach and there was not much corn in the first place, with little or none during last four or five days.

On December 6, 1895, I visited Mr. Gregory's place and found the condition of the cattle as stated in Mr. Gregory's letter. The cattle affected were 3-year-old range cattle, known as "nester" cattle.*

The stalk field was a mile or more from the ranch and feed lots, where all the cattle were, except six that had been left in the stalk field. Of those brought to feed lot three were down and five or six were noticeably affected, as shown by uncertain gait, lack of co-ordination and knuckling of hind legs. The steers that were down had been hauled to sheltered places and were nibbling at millet hay and eating a little corn that was given them. Their temperatures were 102 1-5 deg. F., 102½, 102 1-5 deg. The respirations and pulse were faster than normal but this was probably caused by the excitement of drawing them upon a sled. There was paralysis of the hind quarters.

Of the steers that were necessarily left at the stalk field, one was dead and six had been hauled out of the stalk field and placed on the south side of the haystack in an adjoining meadow. Two of the six head had been able to regain their feet during the night and had wandered away. Of the remaining four, three presented the same appearance and condition as those at the feed lot, except their temperatures were slightly lower, being 101 3-5, 102, 102 1-5. Their bodies had been covered by hay, of which they ate a little and also a little corn that was given them. If approached they would shake their heads and make efforts to rise, but were unable to do so on account of paralysis of the hind parts. All of these steers were lying in normal position upon the sternum or "brisket." Another steer more seriously affected was lying upon the right side, and was

*"Nester" cattle are those raised by men who have small ranches, or "nests" in the big range country. They are not so wild as range cattle but otherwise do not differ.

quite badly bloated. This bloating probably caused from indigestion induced by lying in one position on the side. Tapping the rumen gave temporary relief. This steer appeared to be in no pain. The eyes were congested and were discharging some mucopurulent matter. His temperature was 101 3-5. The dung of all the sick steers was very dark colored and foetid, yet containing quite a little undigested corn. The remaining (seventh) steer was dead. Had been dead two days, but as the weather had been cold decomposition was not marked. The steer was lying in a depression or "draw" that contained some standing water and was quite mirey. An autopsy revealed congestion of the brain and lungs, the latter severe. Rumen was partially filled with well masticated cornstalks. There was no impaction of the omasum (manifolds); the abomasum or true stomach was empty of food but congested and contained a quantity of mucus. Small intestines were congested. Large intestines congested and filled with dark, foetid manure, mixed with considerable mucus. The heart was congested. The liver, spleen and kindneys appeared normal. After this steer was taken ill he was exposed to a cold sleet and snow storm and his death probably resulted from a combination of disease and exposure.

Inoculations of agar were made from blood of heart, also from spleen and liver. Smear cover glass mounts were also made, but no bacteria but those of decomposition were found.

A hasty examination of the stalks in one corner of the field failed to find any noticeable abnormal conditions, although Mr. Gregory mentioned that there had been considerable rotten corn in the field. Under date of December 21, Mr. Gregory wrote as follows:

As promised, I now give you results of disease amongst our cattle supposed to be cornstalk disease and investigated by yourself.

At the time of your visit here, if I remember rightly, there were five steers down in the field, one dead, and at the farm those that fell were being hauled up and made comfortable in the lot.

This continued until some 16 in all were badly affected, and three or four others evidently sick with the disease were left in the corral. We did not interfere with any able to keep their feet and these last named, after being helped up once or twice managed to pull through without further assistance.

Of the five you examined at the hay stack, two got up during the night, reeled about for a few days, and have now recovered, or nearly so. The remaining three died right where they were, but the animal I was about to let you kill for examination (as being the most hopeless) lived longer than any of the rest, and it was 8 or 10 days after your visit before he died.

Of 10 others hauled up to the lot, six died, two are unable to get up, and two have regained their feet and will probably recover. Thus it stands, of the 75 steers, 20 or more were visibly affected, of which number 16 were down

and absolutely helpless, 10 of these died, four may be said to have recovered, and two yet in doubt, with chances that one will die and one recover.

In case of the first steer that died, and on which you made *a post mortem* examination, I am now, in the light of more extended experience, satisfied that death was hastened by exposure in a helpless and wet condition, he having fallen in a bad place and remained there some time before found during severe weather. At least all others have lingered from a few days to two weeks or more. Nearly all the dead have been examined by Doctor Lytle, a local veterinary surgeon, and conditions found about the same as those you found but more intense, which is probably accounted for by death being hastened by exposure in the animal you examined. In all cases the several stomachs of the animals have been in a condition nearly normal, and in much better shape than any of the other organs unless it was the urinary organs. *Heart and lungs always badly congested* (lungs completely filled). Brain and spinal cord congested to a greater or less extent in five or six cases. In one case this appeared normal.

In every case the disease first became apparent by animal's want of control of hind parts, sometimes holding neck as if stiff and head frequently carried unnaturally. Eyes and ears looked all right, and dung, while not just what it should be, was about like what might be expected from animals grazing on dry stalks.

When the animals were able to keep their feet, or, to put it another way perhaps, when the disease did not get hold enough to prostrate, they got over it without assistance or medical aid, but amongst those that "got down" it was quite fatal. In a number of cases it was two or three days after the cattle had been taken from the field before they were sufficiently affected to show it to an ordinary observer.

Most of them bled at the nose, for which the condition of lungs after death would account. After being hauled up and made comfortable, most of them ate and drank quite well, those that got well improved in this respect right along. While those that died gradually gave way, taking less and less nourishment. Their respirations became more rapid and shorter, until just before death the mouth was held open, tongue protuding and respirations only a gasp. The animals in every case reached death by suffocation, through failure of lungs, but presume while it was the direct cause of death it was only secondary.

We treated some of the cases a little, giving first a cathartic drench, adding both a heart and nerve stimulant; supplemented same with injections and cleared out rectum. After that we used *nux vomica* for a constitutional treatment either by drench or hypodermic injections of *strychnia* solutions, but did no good.

Now may I ask a favor, and by way of explanation will state that my partner laughs at the idea of any cornstalk disease except impaction; has seen stalks grazed all his life, etc. I would like something to convince him that if he gets into the wrong field that "salt, sulphur and water" will not cure his cattle. I don't pretend to know anything about it myself, but I do know that the cattle became diseased in the stalk field and that *it was not impaction.* As you have been personally looking the matter up, will you kindly give me result of conclusions?

This outbreak at Mr. Gregory's is a very interesting and peculiar one. It is evidently not the ordinary cornstalk disease or if it is

the cornstalk disease it differs radically in the following particulars:

The disease came on slowly. A well defined paralysis of the hind quarters. Severe congestions of the lungs. Absence of reflex nervous symptoms, such as delirium, etc., and no impaction of the manifolds.

The history and symptoms as exhibited by these cattle would indicate that they were affected by some poisonous substance, which had a paralyzing action upon the nervous system, as shown by loss of control of hind parts, and congestion of lungs, which indicates a weak action of the heart.

The source of this poisonous substance, if such it be, may be in the rotten corn; something of the nature of a ptomaine or nitrogenous base formed in the decomposing corn.

On January 5, Mr. Fred. Koster, of Miltonvale, Kas., wired me that he had lost 15 steers in the stalk fields, and asking me to come and examine them. I was unable to go however, and wrote asking him for particulars. Mr. Koster has much experience in handling cattle, extending over many years. This year he was pasturing in stalk fields three herds of 370 each, Texas steers. Under date of January 14, 1896, Mr. Koster wrote as follows:

> Your letter of January 6 forwarded from Clay Center. I have been pasturing stalks with large herds for the past 10 years and have never met with so heavy a loss as this winter.
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> On December 13, 14, and 15, we lost five steers out of a herd of 368 four-year-old Texas steers. They were on Peach creek, about one mile from Republican river, near Morganville. The creek is very sluggish here, being so close to the river, with heavy walnut timber on its banks, and when the cattle would go into the water and stir it up there was a very bad smell from the water and it was quite black from walnuts and leaves falling in it, there having been no rains this fall to wash it out.
>
> After losing these five steers we took the cattle to the river for one week and dammed the creek up, and since then the water is of sufficient depth so they do not go in it and stir it up. I concluded that they would not drink the offensive water in sufficient quantities to keep them alive on stalks. They are apparently doing all right since the creek was dammed.
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> The next herd was located between Morganville and Clay Center, about four miles from each town, on the Republican river. We lost 11 head on January 3, 4, and 5; seven head on the 3d, and two head on each day following for two days.
>
> We drove these cattle to water at noon and night, they were in a fenced stalk field of 300 acres, but after watering at night they were put in a fenced prairie pasture over night where they could get no water. The cattle had been in the 300 acres of stalks but four days when they commenced to die. I attributed this loss to the fact that there was no water in the pasture where we kept them over night. We then made a corral by the water and have lost none since. We had not salted these cattle for about 30 days until about three days before they commenced to die, when we gave them one bushel of salt.

Many think salt causes their cattle to die when on the stalk fields and do not give salt. The herd in which we lost the five head had not had any salt for 30 days but we began to salt them after the five died and continued until the 11 head died in the second herd (18 days) and then quit.

In the third herd we lost one steer on December 24, three days after salting. They were on the river in a 70-acre field of stalks and were driven to the water noon and night. The water was right in this stalk field and the cattle could go to the water whenever they chose. The cattle were kept in the field night and day.

You will see that my cattle died when they were salted and died when not salted. There are so many farmers that think it is death to cattle to salt when on the stalk fields that I have quit. Yet I have doubts whether salt has anything to do with it.

The first symptoms of sickness is that we find the steer lying down in the herd while the others are grazing. If he is driven up he will walk a short distance, smell of the stalks, but not eat. Sometimes he will look at his sides, twist his tail and soon lie down again. When well advanced in sickness he will stagger and act crazy and if you attempt to drive him will plunge at you and fall down.

We discovered several years ago that to run the steer, on the first symptoms of sickness, until he was thoroughly heated up and scoured out would cure them. Until we made this discovery we lost *every one that was sick.* But since then we have saved a good many and have seldom lost one if he is noticed before the disease is too far advanced. The most of the steers we lost of late we were not able to run when noticed and not a few were found dead in the field or corral in the morning. We have never saved a steer by drenching with oil or anything else. . . . I thought it best to give you my experience and conclusions, whether right or wrong, and perhaps you can give me some light if I am wrong.

We invariably find the small stomach (abomasum or manifolds) dry and the food packed like yeast cakes, the lining coming off with the food. My theory is that the indigestible cornstalks pack in the stomach, fever sets in and destroys the lining of the stomach.

The later part of December, 1890, 300 head of New Mexico three- and four-year-old cattle that had been pastured on stalk fields along Brush creek, Pottawatomie county, were driven some eight miles to a stalk field on the river bottom just south of the Kansas river. The second day they commenced to die, and 33 head died before noon. Dr. Brady, a qualified veterinarian who was called, reports that the first symptoms noticed, the animal would be standing apart by itself with head elevated and licking its nose or a tree, often for one-half hour at a time. They were easily frightened at imaginary objects. One especially ran about 300 yards into the stalk field, turned suddenly and ran directly back, fell down an incline, became comatose (unconscious), and died in a short time.

Some of the sick steers were caught and treated by giving active purgatives, but no action from the purgatives was obtained. Autopsies revealed congestion of brain and moderate impaction of

omasum. These cattle were taken off the stalks and fed straw for two days, and given a mixture of two parts of salt, sulphur and hyposulphite of soda, which they licked greedily. They were then turned back into the field. No more loss occurred.

Dr. J. B. Minturn, of Colwich, Sedgwick county, Kansas, who is a well qualified physician and a stockman of much experience, writes as follows regarding cornstalk disease:

One year I lost three fine steers in the feed lot, on corn and hay, with identically the same symptoms and pathological conditions as the so-called "cornstalk disease," caused by an indiscreet man putting them on full feed too soon. One of the steers in his insanity came nearly killing me. I was in the feed lot looking over he cattle, as is my custom. I had noticed nothing wrong with the steer, when he made a dash at me. I caught a glimpse of his action and jumped one side. The steer fell and never tried to rise. I studied his dying struggles and finally took a knife and bled him to death. I made a *post mortem* examination, and his condition will describe the condition of every one that I have examined that have died from "cornstalk disease," except that one found down and dying recently was badly bloated.

There was intense inflammation of the stomachs. The mucous membrane in places would strip off by a slight effort. The liver was congested to a marked degree and the spleen and brain congested to a like extent. Contents of stomachs natural and moist.

My diagnosis in these cases is acute gastritis, inflammation of the stomach from feeding dry indigestible corn stover, with metastasis to the brain through reflex nervous action. All die with brain symptoms.

The first that is noticed is that the animals are down and dying, or dead. I have made inquiry of many of my neighbors since I received your letter and it is the same story with all. The loss is about 10 per cent. There is no recovery as the disease is not recognized in time.

We have the same disease and the same brain complications in the human. You have the same brain complications in puerperal apoplexy in animals and man. You may have it in any acute inflammation of any large organ of the body. It is easy for me to understand and believe that 10 per cent. of cattle may have weak digestion, and when turned into a stalk field, the sudden change of feed, the amount of corn they may find, want of water, etc., may account for the loss. The remedy is to cut the corn in due time and run it through a husker and shredder, as I shall do in the future.

Mr. Edward O'Connor, of Stafford, Stafford county, writes, under date of November 21, 1895, that he had lost 17 out of 90 head that were running on stalk fields:

Cattle were first driven on Thursday forenoon, remained in two hours. Friday afternoon 1½ hours, Saturday forenoon three hours, and Saturday afternoon 1½ hours. The first one died about 6 o'clock Saturday evening, and 16 more died within the next 12 hours.

Symptoms: Stiffness and inclination to stagger as if affected by blind staggers: eyes rather wild and crazy looking. Died in a few hours after symptoms appeared and did not seem to suffer much.

The paunch seemed natural except in one case when the lining of the stomach was destroyed. The manifold was very full, hard, and dry. The inner

membrane parted from the outer coat in removing the contents. None recovered that were affected.

In response to my request, Mr. O'Connor kindly forwarded a small box of wormy and smutty nubbins of corn. He reported little wormy corn, but abundance of smut.

Mr. D. M. Adams, of Rome, Sumner county, on November 23, writes as follows:

There have been many deaths among horses and cattle in Sumner county this fall from pasturing in cornstalks. I lost two cows and a bull calf six months old.

The corn made only about six bushels per acre. On many stalks there were ears that did not fill out. There was only a cob and husks with a large quantity of worm dust inside. I think the worm dust was the cause of the animals' dying.

It had been very dry until we turned the cattle in. We pulled off every bit of smut that we found and threw it on the ground. We turned the cattle in on Monday forenoon and let them run about three hours, then took them out until afternoon and put them in again for three hours. On Wednesday there was a drizzling rain all day and we let them stay in the stalk field all day. They had free access to water all the time. On Saturday night, when we went for them, one of the Holstein cows jumped up as if scared and went with other cattle to the creek but did not drink. When we attempted to milk her she trembled and would not give down her milk. The bull calf laid down when the cattle came to the yard but showed no other signs of sickness. The Jersey cow appeared perfectly well and gave the usual quantity of milk. This was at sundown. On Sunday morning the Holstein cow and bull calf were dead, the Jersey cow was down and swollen so tight she could not touch her upper feet to the ground. She suffered greatly. A little froth ran from her mouth. She died in about an hour. I opened them and the stomachs appeared moist as usual, but as I have had but little experience I could not tell how a healthy stomach should look. Both cows were well along with calf. The Holstein cow's gall was bursted and the bull's bladder was so full that it looked like the bladder of a large beef blown up. There was a 3-year-old bull, a cow and a six-months-old heifer with these cattle that were not affected in any way.

In response to my request, Mr. Adams sent me a quantity of the wormy and mouldy ears of corn. He says regarding the corn, December, 1895:

The ears that are husked here have been husked about six weeks and in a dry granary. The husks were gathered in the field, I was husking yesterday. You will find on opening that many of the husks have worm dust in them as in the field that killed the cattle. The latter field was upland and husked in October.

This corn received from Mr. Adams and that received from Mr. O'Connor was turned over to the chemical department of this Station, and Professor Failver, the chemist, reports as follows.

Fourteen hundred thirteen grams of wormy and mouldy corn (including grain and affected portions of cob and husk, not a large proportion, however,

being real worm dust), well ground and extracted repeatedly with alcohol. The extract was concentrated at low temperature under diminished pressure until all the alcohol was driven off. The watery syrup remaining was further concentrated by standing over sulphuric acid until it had the consistency of thick molasses. Fifty-three and two-tenths grams of this thick syrup were obtained. Sixteen grams were used in testing its effects upon guinea-pigs. Thirty-seven grams were treated after Dragindorff's method for active substances. No well-defined bodies were found in the portion operated upon. But this is considered only a preliminary trial. The work will be continued. It is hoped to be able to isolate the poisonous or active principle if one exists.

A guinea-pig weighing 350 grams was given 11 grams of this extract as prepared by Professor Failyer. The first dose was given at 9:40 a. m., and between this time and 4 p. m. the pig was given in all 11 grams, with no apparent ill effect except a slight appearance of nausea. About 4 o'clock the pig appeared sluggish, hair erect, and pig huddled down in corner of box. The pig died during the night. Autopsy.—Pig was found stretched out lying on belly. There was considerable moisture about the pig's mouth, probably from ensalivation. *Rigor mortis* well marked. The only abnormal condition observed internally was congestion of the stomach and small intestines, and a congested spot in abdominal wall directly beneath the stomach. There was a distinct odor of the corn extract apparent when the abdominal cavity was opened.

On April 8, 1896, another pig weighing 350 grams was given two grams of wormy corn extract at 10 a. m. At 11 a. m. pig appeared sluggish, hair erect, lying stretched out on its belly. The pig appeared to improve for the next two hours and at 1 o'clock p. m. was given three grams more. No marked effects were noticed. Pig did not appear well for the next two days, eating but little and remaining quietly in the corner of the pen. On the 11th, while out on a grass plot in an open pen, it was exposed to a shower of rain and when brought in at evening was found to be in a dying condition. It died during the night. The control pig which had been in the same pen appeared perfectly well. Autopsy revealed only congestion of stomach and intestines.

A bacteriological examination was made of blood and tissues from both of these pigs but no organisms were found.

This experiment is not at all conclusive, although both guinea-pigs died to which the extract was given. All indications point to worm-eaten and mouldy corn as the cause of cerebritis in horses, whether they eat it in the feed or whether it is fed separately as grain. I am not satisfied that there is any connection between corn-stalk disease in cattle and cerebritis in horses. A great many horses have died in southwestern Kansas this year from this disease. This

disease was investigated in 1890 and reported in Bulletin 24. Experiments have since been made and, in connection with the chemical department, will be carried on to determine what the active injurious principle is in the wormy corn and whether it has any relation to the cornstalk disease. It is doubtful if this mouldy corn has any direct bearing upon the cornstalk disease in cattle, as no case has ever been reported where mouldy or wormy corn alone, fed to cattle, has had any ill effects, while at certain seasons of the year it is very fatal to horses.

In Bulletin 49 of this Station an account is given of cattle being poisoned by eating cornfodder containing large quantities of salt peter (nitrate of potash). While the presence of salt peter in cornstalks suggested a possible cause of cornstalk disease, it was not advanced in that bulletin because of the absence of any evidence pointing to the presence of salt peter in cornstalk disease. Yet, if the cattle that died near Winfield, which an account is given in Bulletin 49, had been pasturing in stalk fields, there is little doubt but that they would probably have been reported as dying from cornstalk disease.

Unfortunately I had no opportunity to examine a corn-field where cattle had died from cornstalk disease except the one at Garnett, where I found no potash in the small area examined. In the Newton *Republican* of December 6, 1895. Mr. A. E. S. Danner, a prominent and well known farmer of Harvey county, writes as follows:

Last Friday evening I noticed a short sketch about saltpeter poisoning and reference to Kansas Agriculture Bulletin No. 49 in the last issue of "The Homestead." An idea struck me at once and I rooted No. 49 out of my bulletin drawer and read it from Alpha to Omega. The first part of it is an investigation and experiment of cattle poisoning by potassium nitrate, nitre saltpeter, or by whatever name you happen to know it, by N. S. Mayo, professor of physiology and veterinary science.

The bulletin gives results of examinations of cattle that have died in large numbers in proportion to the number fed on corn fodder that was heavily charged with saltpeter. It said nothing about cornstalks standing in the field, so I was disappointed, but I couldn't see why If corn fodder has saltpeter in it in dangerous quantities, why not cornstalks too? and why isn't that an explanation for cornstalk disease? The opportunity for investigation is all around us at present, so I spent a good deal of Saturday examining and experimenting, and I don't know but I thought a great deal about it Sunday, and Monday I went off on horseback to examine several of our neighbors' fields where cattle have recently died, with the following results:

S. T. Danner, one field of 18 acres, pastured off well with about 25 head of horses, no signs of sickness, showed some saltpeter in one corner of the field next to the hog pen. The same stock in another field of nine acres, no sickness, showed four stalks with considerable of saltpeter out of about 20 tested. We have a third field of nine acres that shows much saltpeter. We haven't turned into it and are afraid of it.

Frank Ewart, one field of 20 acres, pastured off clean with about 60 head of cattle, no sickness, 45 samples of these stalks showed seven well-charged with saltpeter and two slightly. The same cattle after eating this field were turned into another field of 100 acres a few days ago and by Monday morning four of them were dead. Mr. Ewart only let them run in this field in the morning at first and to millet and straw in the afternoon, but as they began to die he shortened up on the time until now he lets them run only three hours a day. Out of 43 samples of this field there were 19 strong with saltpeter, on some of them it could be seen with the naked eye in little crystals on the outside of the stalk after peeling off the leaf sheath.

P. Nickle has pastured about 18 acres, some with about 20 head of cattle, very carefully without any bad results. Out of 21 samples six showed considerable salt and four slightly.

Peter Schroeder pastured seven acres with 12 head of cattle before the rain and sleet storm very carefully and lost two, then after the damp weather he finished up pasturing carefully without any more loss. There was about one acre of this that was highly manured, thinking this would be the place where I would find the nitre I took nearly half of my samples from this acre, but they were nearly all good. The test was 26 samples from the field, seven strong and two slightly salt.

In all the tests I tried to get as honest a test as possible, taking a stalk here and there in all parts of the field, cutting about two joints, from six inches to a foot above the ground. You will generally find it there if anywhere, though sometmies one joint will have it and the next will not. Sometimes one stalk in a hill will have it and the others will not. Sometimes there will be quite a patch where they nearly all have it, then there will be a patch, apparently the same to look at, where there is absolutely none. Why these peculiarities I cannot say unless it be the difference of growth and condition of the stalk at that particular season of the year when the climatic conditions are favorable for the storing up of the drug, viz., a hot, dry spell, while the stalk is, or ought to be, growing. I believe the leaves and all have the drug in them, but at this time of the year it is more or less washed out of the lighter exposed parts. My laboratory is all out of doors, my furnishings in this case is a lucifer match. Take the pith of the stalk and light it; if there is much saltpeter in it you will have no trouble in getting it to burn. It will sizz like a fire-cracker fuse and sometimes almost explode as if it contained powder. If you are interested get bulletin No. 49 and read it.

Mr. Danner kindly sent me by mail specimens of some of the stalks he had found to be heavily charged with salt peter. As there was but a small amount of stalks sent no analysis was made, but judging by previous analysis. I should estimate the amount at 12 to 15 per cent. I have examined the stalks in feilds in the vicinity of Manhattan and have found in every field some stalks which contained unusual quantities of salt peter, but have not found it so prevalent as did Mr. Danner. I examined no fields where cattle had died from cornstalk disease. The examinations I have made indicate that the potash in cornstalks is liable to be in patches, and even in these patches only a portion of the stalks contain the potash in an unusual amount.

IMPACTION.

Probably the most common theory held by farmers and stockmen regarding cornstalk disease is that it is caused by impaction of the omasum (manifolds) with dry indigestible cornstalks. There are many facts to support this theory, assuming that by impaction is meant indigestion, because impaction of the manifolds is but one symptom of indigestion. The disease occurs when cattle are turned into the stalk fields which must contain a large proportion of indigestible food. There is probably no food so likely to cause indigestion as stalks left standing in the field. A large portion of the digestible part is lost. The leaves have blown away, and, exposed to the sun, rain and wind, the most that remains is the coarse, dry, weather-beaten and indigestible cornstalks.

The history of the disease also indicates indigestion as the disease usually occurs when cattle first go into the stalk fields. It is we1 known that a sudden and violent change of food is apt to cause indigestion, and, having an indigestible food, a sudden change is likely to aggravate the trouble.

The symptoms, too, indicate indigestion with reflex nervous symptoms. There is constipation, often bloating symptoms of abdominal pain and disinclination to eat. The *post mortem* conditions usually indicate indigestion, as shown by impaction of the omasum, and some irritation of the stomach. It must be considered, however, that the *post mortem* condition of the stomachs rarely indicates sufficient disturbance to cause death. In this connection it is important to call attention to some conditions of the stomachs of cattle that are apt to mislead one that is not familiar with them. First, the naturally hard and dry condition of the omasum or manifolds. It is naturally firm and quite hard, probably harder in consistency than a bag of putty, and the contents are quite dry. This naturally hard and dry condition is decidedly increased in cornstalk disease as it is in some other diseases. I have noticed that impaction of the omasum is a very prominent condition in rabies (hydrophobia), and is not the result of a high temperature, for, in both rabies and cornstalk disease, there is very little rise of temperature (fever), while in cases of Texas fever I have examined cattle whose temperatures registered 110 deg F., which was as high as my thermometer registered, and yet in such cases there is no impaction of the omasum, showing that impaction is not caused by high temperatures. From the symptoms shown by cattle sick with cornstalk disease, there would seem to be paralysis of the stomachs, and this view is further strengthened by failures to get any action from purgative medicines, even the most violent purgatives. As indiges-

tion or impaction does not seem sufficient to cause death in many cases, we must look for the presence of some injurious or poisonous substance probably in the cornstalks. Corn, wormy and mouldy, especially, will also be studied and examined, but it seems doubtful if any substance will be found in this especially injurious to *cattle,* as no reports of ill effects on cattle where corn has been fed alone or in connection with other foods.

In cornstalks then, the only poisonous substance so far found is salt peter (potassium nitrate). An unusual amount of potassium nitrate in cornstalks can be readily detected by lighting a dry piece of the stalk with a match. If salt peter is present the stalk burns with little flashes, or "sizzing" as if gun powder was scattered though the stalk. Sometimes the potash can be seen on the outside of the cornstalk, under the leaf sheath or "boot," where it occurs in fine white crystals and looks like mould, but is readily distinguished by touching with the tongue, the taste of the potash is characteristic.

There is a possibility of other poisons being found in cornstalks, such as might result from decomposition, but the conditions are against this. Decomposition of standing cornstalks in the winter season is *extremely slow* in this state. As the cornstalks are not especially rich in proteids, it is not probable that anything in the nature of nitrogenous bases or ptomaines would be found.

PREVENTION.

Of course the certain method of preventing cornstalk disease is not to pasture the stalk fields, but as many stockmen claim that it pays to pasture stalk fields, taking into account the losses that result.

The following precautions have given the best results when pasturing in the stalk fields:

1. Cattle should never be started into the stalk fields hungry. They should be well fed and watered, care should be taken that bosses do not keep younger cattle away from water, before starting into the stalk fields. If cattle are well fed they will take more pains in picking and chewing their food and are not so apt to engorge themselves and bring on indigestion. Or, if the disease is caused by some poisonous substance, the action of the poison is much less violent and less apt to be fatal when taken on a full stomach. Hence, whether the disease results from indigestion or a poison, feeding and watering cattle well before turning into the stalk field will tend to reduce the losses.

Salt and plenty of good water are essential to the proper nutri-

tion of all cattle, and especially if there is a tendency to indigestion. Cattle should have all the good water they care to drink, often. They should have free access to salt, or given small quantities *frequently*. Large quantities of salt at irregular intervals are probably worse than no salt at all. Cattle crave salt and if given considerable at one time will eat sufficient to irritate the stomachs and bowels, and thus cause indigestion.

Cattle in stalk fields should have plenty of good water frequently, and salt as described. Salt and water are only preventives in so far as they tend to assist digestion and assimilation, and thus tend to keep the animal's digestive system in good condition.

2. Cattle should be started into the stalk fields gradually, not allowing them to remain on but a short time—one-half hour—the first day, and gradually increase the time. Such a plan, gradually accustoming the cattle to a change of feed, is less apt to cause indigestion. Some stockmen think it a good plan to feed cattle corn fodder for a week or 10 days before turning into the stalk field, thus getting the cattle used to digesting cornstalks before putting on the stalk fields. Whether cornstalk disease results from indigestion or a poison, the above plan will tend to reduce losses. It is well known that with many poisons a gradual increase in the quantity taken produces a kind of tolerance to its action, until large doses can be taken without serious results, that under ordinary conditions would be fatal. The practice of keeping cattle in a corral or on scant pasture until they are hungry and then turning them into the stalk field for a short time is bad practice. Cattle if hungry will eat enough in a very short time to cause serious results. Better to feed and water well and turn them in the stalk field and let them go.

If cattle can have some laxative food, such as millet hay or alfalfa hay or green feed, while running in the stalk fields, it seems to give excellent results towards preventing cornstalk disease.

CONCLUSIONS.

From experiments and observations made, here and elsewhere, it seems conclusively demonstrated that corn smut *(Ustilago maidis)* and the bacterial disease which sometimes attacks cornstalks, known as the Burrill bacterial corn disease, have no influence in the production of what is known as cornstalk disease in cattle.

Observations seem to indicate that there may be more than one disease or condition which are liable to be confused under the term cornstalk disease.

There are indications that what may be called typical cornstalk disease in cattle is a combination of indigestion and some toxic substance in the cornstalks, possibly salt peter (potassium nitrate).

FUTURE INVESTIGATIONS.

While the real cause and nature of cornstalk disease cannot be definitely stated, future work will be free from the incubus of the corn smut and bacterial cornstalk disease theories.

The investigation of this disease will of necessity be what may be called field work, an examination of the disease under the conditions and circumstances where it occurs.

Some of the important subjects that need further investigation as to determine by a close observation of symptoms and *post mortem* examinations whether there is more than one disease or condition that is likely to be confused under the common term "cornstalk disease." In connection with *post mortem* conditions the following points must be considered: The natural comparatively hard and dry condition of the omasum or manifolds. Also that very soon after death decomposition sets in and the lining membranes of the stomachs slip off quite easily. The digestive juices probably play an important part in the separation of these membranes.

It is hoped that more information can be collected to show what relation, if any, storms bear to the production of cornstalk disease.

The presence of poisons in cornstalks and especially to determine the presence of salt peter in cornstalks and what relation it bears to cornstalk disease.

To determine whether mouldy or wormy corn has any influence in the production of cornstalk disease of cattle, also whether there is any relation between this disease of cattle and cerebritis or staggers in horses caused by eating wormy or mouldy corn.

Another great obstacle to the proper investigation of this disease is the difficulty of getting outbreaks to investigate. There were plenty of outbreaks but it was difficult to hear of them in time to investigate them properly.

It is hoped that with the co-operation of farmers and stockmen of the state, the true cause and nature of this peculiar disease can be determined and means found for its prevention.