Twelfth and thirteenth annual reports of the Bureau of Animal Industry for the fiscal years 1895 and 1896.

U. S. DEPARTMENT OF AGRICULTURE.

BUREAU OF ANIMAL INDUSTRY.

TWELFTH AND THIRTEENTH ANNUAL REPORTS

OF THE

BUREAU OF ANIMAL INDUSTRY

FOR THE

FISCAL YEARS 1895 AND 1896.

WASHINGTON:
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1897.

[PUBLIC—No. 15.]

AN ACT providing for the public printing and binding and the distribution of public documents.

SEC. 73. Extra copies of documents and reports shall be printed promptly when the same shall be ready for publication, and shall be bound in paper or cloth, as directed by the Joint Committee on Printing, and shall be of the number following, in addition to the usual number:

* * * * * * *

Of the Report of the Bureau of Animal Industry, thirty thousand copies, of which seven thousand shall be for the Senate, fourteen thousand for the House, and nine thousand for distribution by the Agricultural Department.

LETTER OF TRANSMITTAL.

U. S. DEPARTMENT OF AGRICULTURE,
BUREAU OF ANIMAL INDUSTRY,
Washington, D. C., October 20, 1896.

SIR: I have the honor to transmit herewith the twelfth and thirteenth annual reports of the Bureau of Animal Industry, prepared in accordance with section 1 of the act of Congress approved May 29, 1884, and the printing of which is authorized in section 73 of "An act providing for the public printing and binding and the distribution of public documents," approved January 12, 1895.

As in past years, this report contains an account of the more important operations of the Bureau of Animal Industry for the years mentioned, although the results of many investigations have been published in special bulletins and are not included. An interesting article has been inserted on the "Contagious diseases of animals in Great Britain," which gives important information concerning the history of these diseases and the methods adopted for their control. This was compiled from English authorities. The article by Dr. Theobald Smith on "Investigations of diseases of domesticated animals" completes his series of reports on investigations made by him while Chief of the Division of Animal Pathology. His studies of sporadic pneumonia and the points of difference between sporadic broncho-pneumonia and contagious pleuro-pneumonia are of great interest and value, as they illustrate the cause for the difference of opinion which has existed between American and English inspectors as to the nature of the disease in American cattle landed in Great Britain, which the inspectors of that country assumed to be contagious pleuro-pneumonia.

The articles on infectious leukæmia in fowls, tuberculosis in swine, leeches and rabies, represent heretofore unpublished work of the pathological laboratory, which is of considerable importance from economical and sanitary points of view.

The compilation of the laws in the various States and Territories for the control of contagious animal diseases is a continuation of similar compilations which have appeared in the previous reports of this Bureau, and is very useful to State authorities and officers of this Bureau whose duty it is to cooperate in their official work.

I recommend that the report outlined above be forwarded to the Public Printer for publication.

Very respectfully,

D. E. SALMON,
Chief of Bureau of Animal Industry.

Hon. J. STERLING MORTON,
Secretary of Agriculture.

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MEAT INSPECTION.

The inspection of meat has been during the whole fiscal year the most extensive and urgent work engaged in by the Bureau. Every effort has been made to inspect all the animals slaughtered for the interstate and foreign trade at the cities where the inspection has been inaugurated. Sheep and calves have been included to a greater extent than in former years.

The number of animals inspected before slaughter for the abattoirs in the cities where the inspection was made was 18,783,000, consisting of 3,752,111 cattle, 1,344,031 sheep, 109,941 calves, and 13,576,917 hogs. During the preceding year the total number inspected was 12,944,056. The increase has consequently been 5,838,944, or about 45 per cent. In addition to this there were 5,102,721 ante-mortem inspections made in the stock yards on animals intended for shipment to other cities or purchased by miscellaneous buyers—1,083,013 on cattle, 648,358 on sheep, 10,708 on calves, and 3,360,642 on hogs.

The number of post-mortem inspections made was 18,883,275—on 3,722,042 cattle, 1,428,601 sheep, 116,093 calves, and 13,616,539 hogs. There were tagged 10,393,991 quarters and 9,641 pieces of beef, 322,670 carcasses of hogs and 94,405 sacks of pork, 1,380,267 carcasses of sheep, and 112,615 carcasses of calves. There were 3,840,406 packages of beef and 4,865,297 packages of hog products stamped.

The cost of this inspection was $262,731.34, or an average of 1.1 cents for each ante-mortem inspection; this covers all the subsequent work of making post-mortem inspections, tagging of carcasses, stamping of packages, and issuance of certificates of inspection for exported products. The cost of the inspection has steadily decreased; in 1893 it was $4\frac{3}{4}$ cents per head, and in 1894 it was $1\frac{3}{4}$ cents.

The inspection was maintained at 55 abattoirs, situated in 19 different cities. During the preceding year the inspection was conducted at 46 abattoirs in 17 cities. The inspection was more complete and thorough at all points than it has been during any previous year.

The force of inspectors was placed in the classified service by order of the President at the beginning of the fiscal year. This force has

been increased as rapidly as was warranted by the list of eligibles obtained by the Civil Service Commission through its examinations. The inspectors and assistant inspectors appointed from this source have been in nearly all cases intelligent, competent, and faithful men.

MICROSCOPIC INSPECTION OF PORK.

There were 45,094,598 pounds of microscopically examined pork exported during the year, as compared with 35,437,937 pounds in 1894 and 20,677,410 pounds in 1893. There were 905,050 carcasses and 1,005,365 pieces examined, making a total of 1,910,415 specimens for the microscopical force to inspect. The cost of this branch of the inspection was $93,451.10, and the average cost per specimen examined was 4.9 cents. In 1893 the cost per specimen was 8¾ cents, and in 1894 it was 6⅝ cents. There was, consequently, a reduction of 25 per cent in the cost of inspection in 1894 as compared with 1893, and a further reduction of 25 per cent in 1895 as compared with 1894. The cost of inspection per pound of inspected meat exported was reduced from 0.248 cent in 1894 to 0.2 cent in 1895.

INSPECTION OF EXPORT ANIMALS.

The number of cattle inspected for the export trade was 657,756, as against 725,243 during the previous year. The number actually exported was 324,299. The number of cattle exported in 1894 was 363,535, and there was, consequently, a falling off of 39,236 head. Of the cattle inspected 1,060 were rejected for exportation, as compared with 184 in the preceding year.

The number of sheep inspected for the export trade was 704,044, the number exported being 350,808. The exports of sheep in 1894 were 85,809. The increase has therefore been 264,999, or over 300 per cent.

It is seen from the above statement that 1,361,800 head of animals were inspected for the export trade, and that a total of 675,107 animals were exported. All of the cattle were tagged with numbers, and these were registered, in order that individual animals might be identified, if necessary.

It has been found somewhat embarrassing to certify to the healthfulness of sheep under present conditions, as, although healthy when inspected, they sometimes become affected with scab before they are landed. The crowding of large numbers of these animals together on board ship, together with the atmosphere by which they are surrounded, favors the rapid development of scab, and in case any of the parasites of this disease are present there is an extraordinary development of the symptoms during the voyage. Flocks which are carefully examined and found free from any symptoms of disease at the time of loading were discovered to be badly affected when they were landed in European countries.

Long and careful consideration has been given to the different measures that might possibly be adopted to prevent infection with this disease. No doubt some of these sheep are infected in cars which had previously carried diseased animals, others are infected in stock yards through which they pass, and still others may be infected from the ships. It is evident that to guard against all these sources of infection comprehensive regulations are required that will secure disinfection of ships, cars, and stock yards, and, most important of all, that will prevent the shipment of diseased sheep to market.

VESSEL INSPECTION.

The vessels carrying the exported cattle and sheep were all inspected by the officers of this Bureau, in accordance with the act of Congress approved March 3, 1891. New and revised regulations were issued embodying the amendments which had been suggested by practical experience during the time the law has been in operation.

The losses during the year have been unusually heavy. The record, which had previously shown smaller and smaller losses during each succeeding year that the Department regulations were in force, has been so completely changed that an investigation has been commenced to determine whether any part of these losses was due to noncompliance with the regulations.

Of the cattle exported to Great Britain, 294,331 were inspected at the time of landing, and the loss in transit was found to have been 1,836, or 0.62 per cent, as compared with 0.37 per cent in 1894. The number of sheep inspected after landing was 311,038, and there had been lost out of these shipments 8,631, or 2.7 per cent, as compared with 1.29 per cent in 1894. The losses were due to a variety of accidents, some of which apparently could not have been avoided. Others appear to have resulted from insecure fittings. There are some vessels which do not regularly carry animals, but occasionally take a consignment when good freight rates can be obtained or when other cargo is not available. With these the fittings must necessarily be of a temporary character, and can not be given the security which is obtained with the permanent fittings of the regular cattle boats. If it appears after careful investigation that the losses are due to temporary or otherwise insecure fittings, it is evident that a more rigid inspection must be enforced and that vessels which are unsafe must be denied the privilege of carrying live animals.

STOCK-YARDS INSPECTION.

The stock-yards-inspection service is maintained to prevent the spread of contagious diseases through the channels of interstate commerce. At present Texas or Southern cattle fever is the only disease controlled by this inspection. With the further development of the force engaged in this work it will probably be found advisable to include other diseases, such as sheep scab, hog cholera, and tuberculosis. It is probable that the ravages of the first two of these diseases may be very materially reduced by guarding against the contamination of animals in transit; and that, after our people have experienced the benefits to be derived from such measures, they will be no more willing to dispense with them than they now are to go back to the methods under which the stock yards and cars were continually infected with Texas fever.

During the quarantine season, from February 15 to December 1, 1894, there were received from the infected district and inspected at the quarantine pens 43,271 carloads of cattle, containing 1,197,997 animals. There were 13,545 carloads inspected in transit, and 41,485 cars cleaned and disinfected under supervision of the inspectors. There were also inspected 156,660 cattle from the noninfected section of Texas which were shipped or driven to Northern States for grazing and feeding purposes. It was necessary to identify the brands of these cattle to determine if they could be safely used for this purpose.

The cost of the Texas fever and export inspection was $104,492.46. Assuming that half of this should be charged against the inspection of export animals, the cost of inspecting the 675,107 cattle and sheep exported would be $52,246.23, or 7.74 cents per head. The average cost during the preceding year, computed in the same manner, was 10.75 cents. The number of individual inspections made on these animals was 1,361,800 in this country, and 604,469 in Great Britain, a total of 1,966,269. This gives an average cost of 2.66 cents for one inspection of each individual animal.

INSPECTION AND QUARANTINE OF IMPORTED ANIMALS.

The number of animals imported and quarantined during the year was as follows: At the Garfield station, 142 cattle, 146 sheep, 23 swine, 3 moose, and 9 India cattle. At Littleton, 12 sheep. At Buffalo, 366 cattle. At Port Huron, 1 head of cattle. Altogether 702 imported animals were held in quarantine for the prescribed period. There were inspected 293,594 animals imported from Canada, but not subject to quarantine, as follows: Sheep, 292,613; swine, 908; cattle, 48; moose, 5. There were also inspected 63,629 Mexican cattle imported into the United States from January 1 to June 30, 1895.

Table showing number of Mexican cattle inspected at ports of entry by Bureau of Animal Industry, January 1, 1895, to June 30, 1895.

Month.	San Diego.	El Paso.	Eagle Pass.	Laredo.	Las Palomas, etc.	Marfa.	Yuma.	Don Louis.	Fort Hancock.	Presidio.	Bisbee, etc.	Nogales.	Lochiel.	Buena Vista.	Buenos Ayres.	Aravaca.	All ports.
January	----	1,160	------	------	4,354	954	47	373	----	----	5,140	----	----	----	----	----	12,028
February	----	951	------	------	338	----	----	----	810	----	3,594	794	----	----	550	----	7,037
March	----	3,518	------	------	4,775	997	----	----	----	719	7,872	5,306	558	857	550	28	25,180
April	37	720	1,637	1,600	------	----	----	----	----	----	------	----	----	----	----	----	3,994
May	472	1,123	4,027	2,219	------	----	----	----	----	----	------	----	----	----	----	----	7,841
June	454	1,381	2,989	2,725	------	----	----	----	----	----	------	----	----	----	----	----	7,549
Total	963	8,853	8,653	6,544	9,467	1,951	47	373	810	719	16,606	6,100	558	857	1,100	28	63,629

Animals imported into the United States from foreign countries for the fiscal year ended June 30, 1895.

PORT OF NEW YORK, N. Y.

[Quarantine station located at Garfield, N. J.]

Date of arrival.	Name and address of importer.	Port of shipment.	Breed and kind.	Number.
1894.				
July 5	P. T. Norton, Somerville, N. J.	London, England	Berkshire swine	3
26	Dr. G. H. Davison, Millbrook, N. Y.	Liverpool, England	Shropshire sheep	22
26	----do	----do	Hampshire ram	1
31	August Belmont, Newport, R. I.	Bombay, India	India cows	9
31	----do	----do	Moose deer	3
Aug. 7	Metcalf Bros., Elma, N. Y.	Liverpool, England	Berkshire swine	10
7	E. O. Craft, Heistersburg, Pa.	----do	Dorsethorn sheep	9
7	----do	----do	Hampshire sheep	10
7	Wm. Rockefeller, Tarrytown, N. Y.	----do	Southdown sheep	10
7	----do	----do	Shropshire sheep	2
22	Levi P. Morton, Rhinecliff, N. Y.	----do	Guernsey cattle	24
22	J. Milton, Marshall, Mich.	----do	Hampshire sheep	4
Oct. 1	H. K. McTwombly, Madison, N. J.	----do	Guernsey cattle	17
1	----do	----do	Southdown sheep	17
1	----do	----do	Jersey bull	1
3	C. B. Dustin, Pittsfield, Ill.	----do	Shorthorn cattle	3

PORT OF NEW YORK, N. Y.—Continued.

[Quarantine station located at Garfield, N. J.]

Date of arrival.	Name and address of importer.	Port of shipment.	Breed and kind.	Number.
1894.				
Oct. 11	Foote Bros., Medina, Ohio	Liverpool, England	Sheffield pig	1
31	T. S. Cooper, Coopersburg, Pa	do	Dorsethorn sheep	55
Dec. 6	C. S. Hacker, Bakersfield, Cal	London, England	Jersey bull	1
19	Chas. Green, Rye, N. Y	Hull, England	Southdown sheep	6
1895.				
Feb. 2	M. C. Campbell, Spring Hill, Tenn	Liverpool, England	Berkshire swine	7
6	J. B. Guillot, Mahwah, N. J	Antwerp, Belgium	Norman cattle	12
Apr. 6	F. S. Peer, Mount Morris, N. Y	London, England	Jersey cattle	8
6	do	do	Guernsey cattle	43
24	do	do	Ayrshire cattle	2
19	Ferd. Piper, Casselton, N. Dak	Bremen, Germany	Merino sheep	10
May 2	J. D. Wing, Millbrook, N. Y	Liverpool, England	Shorthorn cattle	17
27	T. A. Havemeyer, Mahwah, N. J	Antwerp, Belgium	Simmenthal cattle	14

PORT OF BOSTON, MASS.

[Quarantine station located at Littleton, Mass.]

1894.				
Nov. 15	J. H. Warren, Hoosac Falls, N. Y	Liverpool, England	Southdown sheep	6
Dec. 5	M. B. I. Goddard, East Greenwich, R. I.	do	do	6

PORT OF BUFFALO, N. Y.

1894.				
July 5	J. D. E. Hay, Derby, N. Y	Thorndale, Canada	Holstein cattle	24
Nov. 23	D. P. Norton, Council Grove, Kans.	Bridgen, Canada	Shorthorn cattle	1
25	B. C. Rumsey, Buffalo, N. Y	Cayuga, Canada	do	1
26	A. P. Grout, Winchester, Ill	Montreal, Canada	Aberdeen Angus cattle.	15
1895.				
Mar. 16	C. E. Colburn, Portlandville, N. Y.	Compton Station, Canada.	French Canadian cattle.	8
Apr. 11	J. W. Wadsworth, Avon, N. Y	Lucknow, Canada	Canadian cattle	154
May 2	do	do	do	118
23	Edw. Green, Springfield, Ill	Seaforth, Canada	Shorthorn cattle	6
23	J. B. Gerlack, Dayton, Ohio	Toronto, Canada	do	8
June 3	M. E. Griffith, Manor Station, Pa.	Orillia, Canada	Jersey cattle	1

SCIENTIFIC WORK.

Important scientific investigations have been in progress which have yielded valuable results, while with others the objective point has not yet been reached.

A subject of investigation which promised well was the application of mixtures to Southern cattle with the idea of destroying the ticks upon them and thus avoiding the danger of disseminating Texas fever. A considerable number of insecticides have been experimented with and marked progress has been made, but a thoroughly reliable mixture for this purpose has not yet been discovered. It is probably only a question of time and research when such a discovery will be made. The mixtures so far used which kill all of the ticks are too irritating to the skin of the cattle, while on the other hand those which are not irritating to the cattle do not kill all of the ticks.

Investigations concerning the nature of various animal diseases, particularly of a hitherto undescribed but very destructive disease of turkeys, will be published in special bulletins.

During the fiscal year ending June 30, 1894, the laboratory has sent out on application of the proper State authorities tuberculin sufficient to test 35,000 cows, and mallein sufficient to test 1,200 horses.

ESTABLISHMENT OF DAIRY DIVISION.

Action has been taken for the establishment of a dairy division to be organized July 1, 1895, with a chief, an assistant, and two clerks. The work of this division for a considerable time in the future will be for the most part confined to the collection and dissemination of information concerning the dairy industry as it exists. Original scientific investigations bearing on this subject must be postponed until a foundation has been laid in other directions, and special facilities for research have been acquired. There is, however, a vast amount of information of the greatest value to the dairyman which may be secured by observation and correspondence. This relates to the condition of the industry, statistics of production and trade, markets, and improvement in the manner of producing and handling dairy products. The present is an era of rapid changes, and the dairyman on this account needs a reliable source from which to obtain a knowledge of the latest modifications in the trade and the most desirable improvements which have been suggested.

The great dairy interest has been so long neglected by the United States Department of Agriculture that a special effort should now be made to press forward the work outlined above and to establish intimate relations with the dairy organizations of the country.

PUBLICATIONS.

There have been published during the fiscal year the following reports, bulletins, and circulars:

Report of the Chief of Bureau of Animal Industry for 1893. (Reprint.)
Bulletin No. 7. Investigations Concerning Bovine Tuberculosis.
Circular of Information No. 1. Directions for the Sterilization of Milk.
Circular of Information No. 2. Wheat as a Food for Growing and Fattening Animals.
Farmers' Bulletin No. 24. Hog Cholera and Swine Plague.

APPROPRIATION AND EXPENDITURES.

The appropriation for the year was $800,000, and expenditures so far have not exceeded $533,000. When all accounts are finally closed the unexpended balance to be turned into the Treasury will certainly exceed $250,000.

REMARKS AND RECOMMENDATIONS.

The work of the Bureau of Animal Industry is more comprehensive and affects interests of a greater magnitude and to a larger degree than is generally appreciated. It is now conducted in accordance with legislation which experience has shown to be defective in many respects. This legislation imposes the duty and responsibility of preventing the introduction and spread of the contagious, infectious, and communicable diseases of animals; of inspecting animals about to be slaughtered, and certifying to the healthfulness of their products; of inspecting animals about to be exported, preventing the exportation of those diseased, and certifying to the healthfulness of those shipped; of inspecting vessels carrying such animals, and requiring proper space, fittings, and care; of investigating diseases not thoroughly understood

and discovering how to control them; of investigating the different branches of the animal industry and supplying information by which they may be more profitably conducted.

The greater part of this work is of an executive nature, and to be effective the regulations must be in many cases arbitrary, inflexible, and thorough. A service to prevent the spread of disease among animals which fails to accomplish its purpose, or an inspection of meats which gives no protection to the consumer, is an injury to the country rather than a benefit; that is, any attempt to accomplish such results is accompanied by the expenditure of money, and necessarily interferes with trade and commerce, damaging some people and benefiting others, and should only be tolerated because it brings great good to the community, or will do so within a reasonable time. This being admitted, it is apparent that the laws and regulations under which the executive work of this Bureau is performed should be so perfected that the objects of the work may be accomplished as completely and with as little delay as the nature of the subject will permit.

THE SHIPMENT OF ANIMALS AFFECTED WITH CONTAGIOUS DISEASES SHOULD BE PROHIBITED.

In the appropriation act for the last two years tuberculosis in all animals and scab in sheep have been mentioned as diseases the control of which is specially authorized. This being the case, I would recommend that regulations for preventing the spread of contagious diseases, under the acts of May 29, 1884, and March 2, 1895, be issued, and that these be made to prohibit the shipment from one State into another of any animal affected with any contagious, infectious, or communicable disease, and particularly with tuberculosis, sheep scab, hog cholera, and swine plague. These diseases are disseminated by, and are to a large extent due to, contagion carried through the channels of interstate commerce. They can never be controlled or their ravages greatly diminished until these interstate channels of commerce are thoroughly supervised and purified, and this purification must include all of these channels, the stock yards in which the animals are unloaded, watered, and fed, as well as the railroad cars and boats which transport them.

NECESSITY FOR DISINFECTION OF STOCK YARDS AND STOCK CARS.

Recently a large export trade in live sheep has been established, and this trade is menaced by the discovery of scab in many lots when they are landed in foreign countries. Although these sheep are very carefully inspected before they leave American ports and all affected lots are rejected, the disease continues to appear during the voyage. This is due to exposure in the stock yards and cars or to infection on the vessel. The vessels are thoroughly cleaned and whitewashed each trip, so that the stock yards and stock cars are for the most part responsible. The unrestrained shipment of scabby sheep in this country has undoubtedly thoroughly infected the channels of commerce, and we can not expect to prevent or eradicate the disease until these channels are freed from the contagion and protected from its further distribution. It is not sufficient to guard against the shipment of affected animals, because, the yards and cars being infected, the animals which pass through them will continue to contract the disease.

The companies which own and operate railroads and steamboats carrying stock in transit from one State into another, and stock yards forming a part of the channels of interstate commerce, should be required by law to disinfect them whenever this is directed, under regulations of the Secretary of Agriculture. This is absolutely necessary to prevent the spread of contagious diseases from one State into another. It is useless to provide penalties for the interstate shipment of diseased animals so long as the sound animals must be exposed and become infected during transit. An amendment to the law by which proper disinfection may be effected is extremely important for the protection of both the export and the domestic trade.

THE CHANNELS OF INTERSTATE COMMERCE SHOULD BE GUARDED FROM INFECTION.

To properly guard the interstate trade, it is also necessary to extend the provisions of the act of May 29, 1884, so as to prohibit the shipment of diseased animals or infected stock upon railroads (1) that form a part of a line used for transporting stock from one State into another; or (2) which use cars that are allowed to go into other States; or (3) which transport animals to stock yards that are in the channels of interstate commerce. The law as it stands gives its whole attention to diseased or infected animals, but infected cars and infected stock yards are not given consideration. If, for example, a railroad company knowingly ships animals affected with a contagious disease from any part of the State of Illinois to the Union Stock Yards at Chicago, although they deliberately infect the channels of interstate commerce and endanger the stock interests of the whole country, there is apparently nothing in the Federal law under which they may be punished or restrained.

A case of the kind just mentioned has actually occurred during the past summer. Cattle known to be infected with Texas fever were shipped from a point in Illinois to the Chicago stock yards, and when the railroad company was requested to disinfect the cars in which these animals were shipped they refused, asking the Department to point to the provision of the statutes which compelled them to do so, as they had not shipped the animals from one State into another. So long as these fatal defects exist in the law, how can we guarantee our export cattle as free from Texas fever infection, or our export sheep as free from scab? And yet the very existence of this export trade depends upon the complete exclusion of contagion in all forms.

The losses from contagious diseases of animals in this country due to the dissemination of the contagion through stock yards and cars is not fully appreciated, and is certainly enormous. Most of the outbreaks of hog cholera originate from hogs purchased in the markets and shipped for feeding. Most of the cases of foot rot, scab, and Texas fever are also accounted for in this way. There is, consequently, so much depending upon the purification of our channels of commerce that I unhesitatingly invite your early attention to this subject.

IMPORTED AND EXPORTED HORSES SHOULD BE INSPECTED.

Horses brought from foreign countries are liable to be infected with various contagious diseases, such as glanders, maladie du coït, and foot-and-mouth disease. There is, consequently, the same necessity for their inspection at the port of entry that exists with ruminants and

swine. Unfortunately there is no authority in the law for such inspection, and horses are allowed to enter the country without any sanitary supervision. This trade, as at present conducted, is therefore a menace to the animal industry, and I renew the recommendation of my last report that the subject be brought to the attention of Congress, with the view of securing additional legislation. If such imported animals, on inspection, are found affected with a dangerous contagious disease they should be refused entrance and either slaughtered or returned to the country whence they came.

Horses for export should also be inspected and certified the same as are other species of animals. Our exports of horses are becoming quite heavy, and the lack of inspection should not be allowed to stand as a reason for other countries to prohibit the trade, particularly as the inspection may be conducted by the force already in the field and with no appreciable increase in the expenditures. The question of contagion has already been raised by certain foreign countries in connection with exported horses, and it can not be properly met by this Government until authority is given by Congress to inspect and certify healthy horses and reject those affected with contagious disease.

EXPENSES OF QUARANTINING IMPORTED ANIMALS.

The act of August 30, 1890, provides "That the Secretary of Agriculture be, and is hereby, authorized, at the expense of the owner, to place and retain in quarantine all neat cattle, sheep, and other ruminants, and all swine, imported into the United States." There is, however, no means provided in the law by which the expenses of quarantine may be collected in case the owner refuses to pay. It has been assumed that the expenditures necessary for the feed and care are a lien upon the animals, and that, in case of the owner's refusal to pay, the animals may be sold and the proceeds used to defray such expenses. The experience of the past year has shown, however, that in the absence of specific legislation authorizing the sale of the animals embarrassing complications may arise. It is desirable, therefore, that the law should be amended so as to give a lien upon imported animals for the quarantine expenses, and authorizing their sale at the expiration of the quarantine period in case such expenses are not paid. This provision should also apply to animals smuggled across the boundary and afterwards captured and quarantined, as well as to those which have in any other way evaded the requirement for inspection and quarantine at the port of entry.

MEAT INSPECTION—SOME SUGGESTIONS FOR THE IMPROVEMENT OF THE SERVICE.

It has been shown above that during the last fiscal year there were inspected more than 18,000,000 animals at the time of slaughter. This demonstrates the rapid extension of the meat-inspection service, and indicates the near approach of the time when the provisions of sections 2 and 3 of the act of March 3, 1891, as amended in the act of March 2, 1895, must be strictly enforced. Briefly, these sections provide that no beef shall be exported unless the cattle from which it is produced are inspected before slaughter, and a certificate of inspection accompanies the beef; also that all cattle, sheep, and hogs must be inspected prior to their slaughter in case the meat made from them is to be shipped from one State into any other State.

These provisions are mandatory; they apply to the whole country, and as the trade and other interests involved are enormous there should by all means be made an effort to perfect the law before they are put into effect. The amendments which I would suggest to perfect the meat-inspection law (act of March 3, 1891) are as follows: Section 2 should be amended to read—

That the Secretary of Agriculture *may* also cause to be made a careful inspection of all live *domesticated animals* the meat of which is intended for exportation.

The object of withdrawing the mandatory language and making this inspection discretionary with the Secretary of Agriculture is to avoid a demoralization and possible ruin of a part of the export trade which can not comply with the requirements of this section. Inspected beef is purchased in the carcass by retailers who sell at retail the portions suitable for their trade and dispose of what remains to packers, who cure it for export. This meat, although inspected at the time of slaughter, loses its identity before it reaches the packer, and unless some satisfactory means of marking the individual cuts can be devised it will be impossible to certify it, and consequently it can not be exported.

There is also a large quantity of beef prepared in small slaughter-houses where there is no Federal inspection, and where from the small business done by each individual plant this inspection can not be established. Particular cuts of this meat which are not salable in the local trade are packed for export. The law as it stands would entirely prevent this trade.

There is also a market for uninspected meat in certain countries where the people are willing and desirous of purchasing it upon the reputation of the packers. There is no apparent reason why this trade in uninspected meat should be prohibited by the United States when the countries consuming it are satisfied with it. To do this will reduce our export trade without securing any benefit in return. It is forcing inspected meat upon the inhabitants of other countries where inspection is not desired, at great expense to ourselves, and at the same time forcing the undesirable cuts and uninspected meat upon our local markets to be consumed by our own citizens.

The foreign trade should, however, be given ample protection. There should be an inspection maintained to include the meat of all species of animals, in order that any country which desires only inspected products might provide in its laws for the prohibition of the uninspected. Exporters should also be required to plainly mark all packages of meat in such a way as to indicate the species of animal from which it was derived, and a penalty should be provided for failing in this or for incorrectly labeling it. This, it appears to me, is as far as we are called upon to go in protecting the citizens of other countries. There should be the same authority for inspecting and certifying other kinds of meat for export that is given for beef. The trade is just as important and there are the same reasons for requiring certification.

The last clause of section 2, which prohibits the issuance of a clearance to any vessel having on board beef not certified, should, if these suggestions are accepted, be stricken out.

Section 3 should include all domesticated animals, as well as cattle, sheep, and hogs. The shipment of uninspected meat should be prohibited, except under such regulations as may be made by the Secretary of Agriculture, and a penalty fixed for any violation of this provision.

Section 4 should give authority to treat condemned meat in such a way as to absolutely prevent its being sold and used for human food. There is very little gained by condemning meat unless it is at the same time saturated with coal oil or carbolic acid, or rendered in the fertilizer tank. The inspector may require a condemned carcass to be removed from an abattoir, but how can he prevent its being returned by an unguarded door, or during the night, and used in the interstate trade? It would be impossible in most cases to identify such condemned meat, and if it were shipped to another State in violation of the law the shipper could not be convicted.

The only way to make the inspection entirely satisfactory is to provide that whenever and wherever unwholesome or diseased meat is found by the inspectors it shall be made inedible, and the community in that way protected from its subsequent sale as a food product. The theory of the law as it was enacted, no doubt, was that the penalty provided was sufficient to prevent interstate and export shipment of condemned meat and that the local authorities should prevent its sale within the city and State where the slaughtering is conducted. Experience in the endeavor to enforce this inspection shows that the provisions of the law are not adequate to prevent the interstate and foreign shipment of condemned meat, and the Department has been compelled to instruct its inspectors to see that it is tanked, or in case of refusal to do this the firm conducting the business is required to make a statement showing what disposition is made of it. In this way a partial remedy has been found. On the other hand, it has been made plain that in most cases the local health authorities can not be relied upon to take charge of the condemned meat and cause its destruction.

The best equipped municipal boards are those of the large cities, but even these seldom have more than three or four men available for the meat-inspection service, and they are all needed at establishments which slaughter for local consumption. Too often, as experience has shown, the municipal inspectors know little if anything concerning the diseases of animals. They appear to be selected because of their skill and activity in other lines of effort. They belong to the class of men who delight to magnify their office. When an attempt is made to cooperate, they take issue with the Federal inspectors as to their decisions, insisting upon passing meat which has been condemned and condemning meat which has been passed. Such an attitude at once causes friction, brings discredit upon the inspection, makes effective cooperation between the Department and the health boards impossible, and leads to a much less satisfactory condition than where no cooperation is attempted.

If the local health boards would accept the decisions of the Federal inspectors as final, and instruct their inspectors to see that all condemned meat was properly disposed of, thus avoiding conflicts of opinion and authority, much good would come from cooperation, and the great difficulty as to what should be done with condemned meat would be largely overcome. The writer has endeavored to secure such harmonious cooperation, and at the recent meeting of the American Public Health Association presented a report, as chairman of the committee on animal diseases and animal foods, urging the value and necessity of cooperation to prevent the sale of meat from diseased animals. As nearly all the local health boards are represented in the association named, there is reason to hope that some of them will give this matter more attention in the future than they have in the past.

The city inspectors are generally embarrassed in very much the same way as are those of the Bureau of Animal Industry. The latter are instructed that if an abattoir company insists upon selling condemned meat within the State where slaughtered it has a right to do it, subject, of course, to local regulations; while the city inspectors are instructed that meat which is not offered for sale within the city does not come under their jurisdiction. The superintendent of the abattoir may, consequently, say to the Federal inspector, "I shall sell this condemned carcass within the State," and to the city inspector he may say, "This meat is for shipment to a distant part of the State." As the State boards are seldom, if ever, represented by an inspector, the condemned meat goes upon the market in spite of Federal and municipal cooperation. Worse than all, when the carcass passes beyond the sight of the inspectors the condemned tags are removed, it is sold to innocent parties, and may then be shipped to another State, without anyone being legally responsible for violating the act of March 3, 1891.

Again, there are large abattoirs which are situated beyond the jurisdiction of the city boards of health, in sparsely settled districts, where there are no local health officers, and where the community can not afford to maintain an inspection service. Such communities may not be interested in destroying the condemned meat of the abattoirs. These establishments are operated primarily to prepare meat for the interstate and export trade. The condemned meat, which is excluded from interstate trade, may be shipped to the nearest city within the State and there sold as sound and wholesome. The municipality where the abattoir is operated is not injured; on the other hand, a majority of its citizens are directly interested in the success of the plant, and any local regulations are more likely to be enforced for the protection of the abattoir than for guarding the health of other sections of the State or country. Here also, unless the Federal inspectors are authorized to require a proper disposition of unwholesome meat, it may find its way to market.

Each State might provide an efficient method of cooperation by enacting legislation making it illegal to sell for human consumption within the State any meat condemned by the Federal inspectors, or any meat prepared from animals condemned by such inspectors, unless the animals are held a sufficient time before slaughter to enable them to recover and become fit for the production of food products. This would obviate the necessity for applying a State meat-inspection service to the abattoirs which have Federal inspection, and, by leaving the enforcement of the law in the hands of local authorities already constituted, the full benefit of the inspection could be secured at very slight expense.

The only reason which occurs to me why this plan of cooperation may not be adopted is the uncertainty of its being accepted by all of the States or by any considerable number of them. No States have yet shown a disposition to take such action—possibly because it has not been suggested from the proper sources.

The problem, however, is a very urgent one, and the meat-inspection service will not be in a satisfactory condition until it is solved. State legislation authorizing cooperation would require considerable time for its consummation, even if taken up at once and pressed forward as rapidly as possible.

Under the condition of affairs as we find them to-day, and we can not expect any great change in the near future except by Federal

legislation, the simple exclusion of condemned meat from interstate shipments at the abattoir means that it will be sold locally; it will go into the hands of innocent parties, lose its identity, and may possibly be shipped to other States. The only result that would be reached by the Federal inspection, under such regulations, would be to take the diseased meat out of the export and interstate trade at the abattoirs and throw it upon the local market, to be finally sold for consumption within or without the State, as the inclination of the dealers may decide. Is it fair and just that the Federal Government should conduct an inspection which takes the diseased meat from the trade of one portion of its citizens and allows it to be sold to other citizens? Is it desirable to maintain a great meat-inspection service which simply takes the unwholesome meat from certain channels of trade and throws it into other channels? A service conducted in that way must soon become a byword and a reproach to our citizens and a reflection upon our intelligence as a nation.

The Department has in the past prevented the sale, even for the local trade, of the condemned carcasses of animals slaughtered in abattoirs where inspection existed. It has done this by regulations not specifically authorized by the law and by threatening to use its power to expose those firms which would deal in that class of meat. As the inspection is extended, however, firms with less reputation and those more unscrupulous are involved, and it is becoming more and more difficult to protect the consumers.

NEED OF ADDITIONAL LEGISLATION.

The facts above referred to indicate conclusively that more power is needed for the proper administration of the meat-inspection service. It is not a question of destroying property. The condemned meat need not be destroyed, but the owners should be compelled to use it in a legitimate manner, viz, in the manufacture of fertilizers and grease. It is not proper for use as human food and it can not be sold for that purpose unless this is done fraudulently. To saturate such meat with a nauseous compound like carbolic acid, which would enable anyone to detect it at once, would not detract from its intrinsic value, but would prevent its fraudulent sale. The same may be said of action requiring it to be immediately rendered into fertilizer or other similar products. There is in my opinion no good reason for allowing compensation in such cases, as the owner obtains all that the carcass is actually worth. In addition to authorization for the Department to require the proper disposition of condemned meat there should be a penalty for failure to dispose of it according to the Department regulations.

I would, in addition, suggest that a section be added to the law prohibiting, in the absence of the inspector, the operation of abattoirs where inspection is maintained, and also providing that such abattoirs should be closed on Sunday. These requirements are necessary to prevent uninspected meat from becoming mixed with that which has been inspected. There are some abattoirs which have shown a disposition to kill animals at night, or very early in the morning, or on Sunday, without notifying the inspector. It is hardly necessary to add that any animals which it is desired for any reason to prevent the inspector from seeing at the time of slaughter may be disposed of when the plant is operated at such irregular hours.

The desirability of allowing the members of the inspection force to

have their Sundays free from the duties and cares of this responsible and arduous service, except possibly in cases of great emergency, is certainly too apparent to need argument.

Finally, I would urge the addition of a section to this law prohibiting the importation of the meat or meat products of any of the domesticated animals for consumption in the United States unless the animals from which it originated have been inspected by the government of the country in which the slaughtering was conducted, and unless the meat is accompanied by a certificate of inspection showing that the animals were free from disease and the meat sound and wholesome. Such a provision is required to protect the health of the people of the United States. We have now a great meat-inspection service to protect our citizens from diseased and unwholesome meat prepared from animals slaughtered in this country, but an abattoir in Canada may ship to our markets the meat of all their diseased animals, and we have no means of protection.

In the same way trichinous hams may be shipped here from Germany, or bologna sausage from Belgium or other countries, where it is asserted they are made from the flesh of diseased horses and other refuse meat.

It can hardly be considered as otherwise than absurd for this Government to maintain such a rigid system of inspection for its domestic meats and allow the meat products of the whole world to enter our markets freely without any inspection.

The country continues free from contagious pleuro-pneumonia. The efficiency of the stock-yards inspection and the large number of cattle examined at time of slaughter permits this statement to be made in the most emphatic manner.

The following tables are appended to show the rapid development of the meat-inspection service and the exportation of microscopically inspected pork:

Table showing the number of animals inspected for abattoirs, by fiscal years.

	1891.	1892.	1893.	1894.	1895.
Beef cattle	83,891	3,167,009	3,922,174	3,862,111	3,752,111
Calves		57,089	92,947	96,331	109,941
Sheep		583,361	870,512	1,020,764	1,344,031
Hogs				7,964,850	13,576,917
Total	83,891	3,809,459	4,885,633	12,944,056	18,783,000

Table showing exports of microscopically inspected pork, by fiscal years.

	1892.	1893.	1894.	1895.
	Pounds.	*Pounds.*	*Pounds.*	*Pounds.*
To countries requiring inspection	22,025,698	8,059,758	18,845,119	39,355,230
To countries not requiring inspection	16,127,176	12,617,652	16,592,818	5,739,368
Total	38,152,874	20,677,410	35,437,937	45,094,598

TRANSACTIONS OF THE BUREAU FOR THE FISCAL YEAR ENDED JUNE 30, 1896.

WORK OF THE INSPECTION DIVISION.

MEAT INSPECTION.

The inspection of all the animals slaughtered in the United States for human food the meat of which is to be shipped in the channels of interstate or foreign commerce is a task of such magnitude and requires a service of such proportions that it requires years to build up and train a sufficient force and extend its operations over our entire territory, as contemplated by the law. The work of this division has, therefore, been progressive, and the meat inspection at the abattoirs, which in 1892 included but 3,800,000 head of animals and in 1893 but 4,885,000, has in 1896 been applied to the enormous number of 23,275,000. In addition to this abattoir inspection, there has been an inspection in the stock yards of animals going to abattoirs in other cities, or which have been purchased by various buyers not having other inspection, to the number of 12,641,000. The inspection force of the Bureau has, consequently, made an ante-mortem inspection during the year of a total of 35,917,000 meat-producing animals. This has been an increase of more than 50 per cent over the preceding year. The number of abattoirs and cities at which inspection is conducted has nearly doubled within the year.

There must be a still further increase of the inspection service, for, although by far the greater part of the meat shipped from State to State or to foreign countries is inspected, there is still a considerable quantity which is not reached. The consumer of our meats in other countries may be easily protected from that which is uninspected by a regulation of his government requiring all imported meats to bear inspection marks, but our own citizens do not yet receive such an absolute protection, as the inspection is not sufficiently extended to warrant a prohibition of the interstate shipment of all meat that has not been inspected. American consumers may, however, demand of the retailers of meats that only the inspected article be sold to them, and they can satisfy themselves that this is done by examining the tags and stamps which are put upon it for identification.

The meat-inspection force is now a very efficient one. A large proportion of its members are able and experienced men, and the additions made by appointment from the eligible list upon certification by the Civil Service Commission have been notably superior to the men obtained before this force was brought within the classified service. The discipline has also greatly improved since the merit system was adopted.

With this efficient force at our command it has been possible to greatly extend the system of ante-mortem inspections in the stock yards and to embrace all of the important centers of the live-stock trade. The animals entering these yards are inspected, and those found to be diseased or in a condition unfitting them for the production of wholesome meat are marked with a condemnation tag, showing that they have failed to pass the inspection. Such animals are held for future disposition, and as a large proportion of them have been rejected on account of pregnancy they and their young are allowed to be shipped to the country in order that they may be fed until in a suitable condition for slaughter. Of the condemned animals which are not returned to the country, some die in the pens, others are released after they have been held until their condition has improved, but the greater part is slaughtered, and if the post-mortem examination confirms the diagnosis that the animals are in a diseased or unwholesome condition the carcasses are disposed of in such a manner that they can not be utilized for human food.

Below is a statement of the ante-mortem work at the abattoirs and stock yards. The figures in the first column approximate the actual number of animals inspected for abattoirs having Government inspection, and includes those inspected in the yards for such local abattoirs and those inspected at the abattoirs in cities where there is no yard inspection. The second column gives the additional number of inspections in the yards on animals not purchased for the official abattoirs in those cities, and does not represent the actual number inspected, for the reason that, as the inspection is made at the scales and the animals may change hands several times, being weighed on each occasion, the same animal may pass the inspector more than once. The number of animals rejected as unfit for food at the time of the ante-mortem inspection is given under the heading "Animals condemned."

While the exact number of individual live animals inspected can not be told, the number finally condemned as unfit for food may be ascertained by adding the number condemned at the abattoirs, both ante-mortem and post-mortem, and the number condemned post-mortem in the stock yards inspection.

Ante-mortem inspection.

	Number of inspections.			Animals condemned.		
	For official abattoirs in cities where the inspection was made.	For abattoirs in other cities and miscellaneous buyers.	Total.	At abattoirs.	In stock yards.	Total.
Cattle	4,050,011	3,479,512	7,529,523	233	22,123	22,356
Sheep	4,710,190	1,608,094	6,318,284	692	12,533	13,225
Calves	213,575	101,271	314,846	47	2,790	2,837
Hogs	14,301,963	7,452,863	21,754,826	11,889	39,092	50,981
Total	23,275,739	12,641,740	35,917,479	12,861	76,538	89,399

Last year the number of animals inspected for abattoirs having official inspection was 18,783,000, and the total number of ante-mortem inspections made was 23,885,721. There has been an increase, therefore, in the number of animals inspected for abattoirs where inspection was maintained of 4,492,739, or nearly 24 per cent, which is due principally to the extension of the inspection of sheep where it had not been possible to do so before. The increase in the total number of inspections is 12,031,758, or over 50 per cent.

Following is a table showing the number of animals inspected at time of slaughter and number of carcasses and parts condemned:

Post-mortem inspection.

	Number of inspections.			Carcasses condemned.			Parts of carcasses condemned at abattoirs.
	At abattoirs.	On animals condemned in stock yards.	Total.	At abattoirs.	Stock yards inspection.	Total.	
Cattle	3,985,484	9,977	3,995,461	4,886	3,871	8,757	6,798
Sheep	4,629,796	3,546	4,633,342	2,794	1,541	4,335	242
Calves	256,905	931	257,836	276	761	1,037	33
Hogs	14,250,191	28,028	14,278,219	31,178	15,011	46,189	33,930
Total	23,122,376	42,482	23,164,858	39,134	21,184	60,318	41,003

Last year the number of post-mortem inspections reported was 18,883,275.

There were 13,289,680 quarters and pieces of beef, 328,589 carcasses of hogs, 151,959 sacks of pork, 3,516,896 carcasses of sheep, and 183,685 carcasses of calves tagged or otherwise marked as inspected meat. Of these there were exported 1,030,334 quarters and 16,818 smaller pieces of beef (equivalent to nearly 260,000 cattle), 349 carcasses of sheep, and 3,281 carcasses of hogs.

The meat inspection stamp was affixed to 3,697,701 packages of beef and 6,034,165 packages of hog products; 63,313 of the latter contained microscopically examined pork. There were issued 15,211 certificates of inspection for meat products, of which 3,481 were for microscopically examined pork.

There were sealed 11,855 cars containing inspected meat in bulk for shipment to establishments having Government inspection and to other places.

The cost of this work was $341,456.24, or 0.95 cent for each ante-mortem inspection, and covers the expense of all the subsequent work of post-mortem inspection, tagging, stamping, and issuance of certificates of inspection. In 1895 it was 1.1 cents, in 1894 it was $1\frac{3}{4}$ cents, and in 1893 it was $4\frac{3}{4}$ cents.

Table showing number of abattoirs and cities where inspection was maintained during the fiscal years given.

Fiscal year.	Number of abattoirs.	Number of cities.
1892	28	12
1893	37	16
1894	46	17
1895	55	19
1896	102	26

MICROSCOPIC INSPECTION OF PORK.

The following table shows the exports of microscopically inspected pork, 1892–1896:

Fiscal year.	To countries requiring inspection.	To countries not requiring inspection.	Total.
	Pounds.	*Pounds.*	*Pounds.*
1892	22,025,698	16,127,176	38,152,874
1893	8,059,758	12,617,652	20,677,410
1894	18,845,119	16,592,818	35,437,937
1895	39,355,230	5,739,368	45,094,598
1896	21,497,321	1,403,559	22,900,880

The great decrease in the exports this year as compared with last year is probably due to less favorable conditions. The exports for 1895 were unusually heavy, but if we compare with other years it will be seen that the shipments to countries requiring the inspection were greater than in 1893 and 1894, and not materially different from 1892. The shipment of microscopically inspected pork to countries not requiring this inspection has been intentionally discouraged, as the expense in such case is not warranted.

There were 469,025 carcasses and 510,355 pieces examined, making a total of 979,380 specimens inspected by the microscopical force; 11,100 samples contained trichinæ. The cost of this inspection was $60,485.93, an average cost per specimen of 6.18 cents. Last year the number of specimens examined was 1,910,415 (almost double the number this year), and consequently the average cost was less, being 4.9 cents; in 1894 it was 6⅝ cents, and in 1893 it was 8¾ cents.

The cost of the microscopical inspection per pound of inspected meat exported was 0.264 cent; in 1895 it was 0.2 cent, and in 1894, 0.248 cent.

[NOTE.—The cost per pound, as given above, was obtained, as heretofore, by dividing the cost of the work during the year by the number of pounds exported. This method is objectionable, because the true average cost per pound can not be found by it, for the reason that the meat examined during one month may not be exported for several months. To illustrate this point: During the first six months the cost was $19,848.92; pounds exported, 10,492,180; last six months, cost $40,637.01; pounds exported, 12,408,700, making an average of 0.19 cent for the first period and 0.33 cent for the last. From this it would seem that the meat examined during the latter part of the fiscal year was intended for shipment during the next year.]

INSPECTION OF VESSELS AND EXPORT ANIMALS.

There were 819 clearances of vessels carrying cattle and sheep. All of these vessels were carefully inspected as to fittings, space, and other accommodations for live stock before a clearance would be authorized. The number of certificates of inspection of export animals issued was 1,393.

Following is a statement showing the inspection of domestic cattle and sheep for export and the number exported for 1896 and previous years:

Fiscal year.	Cattle.				Sheep.		
	Number of inspections.	Number rejected.	Number tagged.	Number exported.	Number of inspections.	Number rejected.	Number exported.
1896	815,882	1,303	377,639	365,345	733,657	893	422,603
1895	657,756	1,060	324,339	324,299	704,044	179	350,808
1894	725,243	184	360,580	363,535	135,780	.....	85,809
1893	611,542	292	280,570	289,240	.....	.....	.....

During the year the number of Canadian cattle exported from American ports was 1,482; number of Canadian sheep, 10,512. Last year there were 1,834 cattle and 38,873 sheep from Canada.

The percentage of loss in the shipments of cattle and sheep to London, Liverpool, and Glasgow, where inspectors of this Department are stationed, is about half that of last year. The number of cattle inspected after landing was 348,833; the number lost in transit was 1,107, or 0.32 per cent, against 0.62 per cent last year and 0.37 per cent in 1894. The number of sheep inspected was 389,534, and 4,587 were lost on the voyage, a percentage of 1.16, compared to 2.7 in 1895 and 1.29 in 1894.

The cost of the export inspection and the Texas fever work, which includes the inspection of live stock imported from Mexico, was $107,273.07. Taking half of this sum as the amount chargeable against the inspection of animals for export, the cost of inspecting the 787,948 cattle and sheep exported would be $53,636.54, or 6.8 cents per head. Last year the average was 7.74 cents, and in 1894 it was 10.75 cents per head. The number of individual inspections made on these animals was 1,549,539 in this country and 738,367 in Great Britain, a total of 2,287,906. This gives an average cost of 2.34 cents for each inspection, against 2.66 last year.

SOUTHERN CATTLE INSPECTION.

During the quarantine season, from February 15 to December 1, 1895, 47,082 cars, containing 1,224,715 cattle, from the infected district were received and inspected at the quarantine pens in the various stock yards, and 45,390 cars were cleaned and disinfected under supervision of the inspectors.

Orders issued by the Secretary of Agriculture modifying the regulations governing the importation of live stock admitted cattle from Mexico, after inspection, for immediate slaughter or for grazing below the quarantine line, subject to the regulations applying to the native cattle of the infected district. Under these orders there were 219,814 Mexican cattle imported and inspected during the year.

INSPECTION AND QUARANTINE OF IMPORTED ANIMALS.

The number of animals imported and quarantined during the year was as follows:

Quarantine station.	Cattle.	Sheep.	Swine.
St. Denis, Md		45	
Garfield, N. J	54	265	22
Littleton, Mass		7	8
Vanceboro, Me	1		
Newport, Vt	10		
Buffalo, N. Y	380		
Port Huron, Mich	10		
Total	455	317	30

There were also at the Garfield Station 12 camels, 1 goat, and 1 deer, making a total of 816 imported animals held in quarantine for the prescribed period.

The number of animals imported from Canada and inspected, not subject to quarantine, was 317,038 sheep, 216 swine, 151 cattle, and 2

deer. There were also inspected 2,168 sheep, 42 hogs, and 3 goats imported from Mexico.

Table showing number of Mexican cattle inspected at ports of entry by Bureau of Animal Industry, fiscal year ending June 30, 1896.

Months.	San Diego.	El Paso.	Laredo.	Nogales.	Brownsville.	Eagle Pass.	All ports.
July	384	123	7,184	.....	726	3,159	11,576
August	259	102	3,456	.....	2,241	5,160	11,218
September	346	152	1,384	.....	3,126	4,209	9,217
October	162	1,009	498	2,782	2,314	3,319	10,084
November	254	19,939	4,898	18,231	1,287	2,785	47,394
December	68	29,039	4,149	9,620	776	3,941	47,593
January	104	14,304	2,159	5,263	487	3,256	25,573
February	218	6,394	.....	7,424	1,333	3,388	18,757
March	22	2,188	3,035	448	3,748	351	9,792
April	298	4,718	2,281	1,223	3,614	2,771	14,905
May	162	1,600	787	7,173	264	596	10,492
June	136	108	.....	2,495	474	.....	3,213
	2,413	79,676	29,831	54,659	20,390	32,845	219,814

For the purpose of comparison the following tables are given:

Table showing the number of animals inspected for abattoirs having inspection.

Fiscal year.	Cattle.	Calves.	Sheep.	Hogs.	Total.
1891	83,891	.....	.....	.....	83,891
1892	3,167,009	59,089	583,361	.....	3,809,459
1893	3,922,174	92,947	870,512	.....	4,885,633
1894	3,862,111	96,331	1,020,764	7,964,850	12,944,056
1895	3,752,111	109,941	1,344,031	13,576,917	18,783,000
1896	4,050,011	213,575	4,710,190	14,301,963	23,275,739

Table showing total number of employees engaged in meat inspection (only) on June 30 of each year and the number of these who were appointed upon certification by the Civil Service Commission.

Year.	Inspectors and assistant inspectors.		Stock examiners and taggers.		Clerks.		Microscopists.	Assistant microscopists.	Laborers.	Total.
	Total.	Civil service appointments.	Total.	Civil service appointments.	Total.	Civil service appointments.				
1892	33	.....	151	.....	11	.....	7	143	30	375
1893	32	.....	163	.....	6	.....	9	177	31	418
1894	40	.....	199	.....	6	.....	7	171	16	439
1895	51	14	232	.....	12	.....	5	195	32	527
1896	77	46	287	76	17	4	4	183	11	579

The effect of placing the force of this Bureau within the classified service has been very marked in increasing its efficiency and improving its discipline. This is particularly apparent with the employees stationed at other cities than Washington. The decreased expense of the inspection work is largely due to this improvement in the force. Every person feels now that his standing, retention in the service, and chance of promotion depends upon the interest which he shows and the care and fidelity with which his duties are performed.

WORK OF THE PATHOLOGICAL DIVISION.

The most important work of this division during the year has been an experimental study of Texas fever, with a view to discover a method of making animals which are shipped to the infected district immune from the effects of the disease. A number of animals have been treated by placing ticks upon them at a season of the year when the disease develops in a mild form, and others have been inoculated with blood from affected animals. The cattle so treated were shipped to the infected district early in the summer, together with other animals that were not made immune, and they have shown a very marked power of resisting the contagion. It is hoped that by treating animals according to this plan it will be possible to take them to the infected district for the improvement of the herds, and that the serious losses which have occurred in the past under such circumstances will be avoided.

Experiments have been continued during the year in the hope of securing a mixture that could be used as a dip for the destruction of the ticks which disseminate the contagion of this disease. No mixture has been found which gave complete satisfaction, but there is reason to believe that by continuing the investigations a plan will be developed by which the cattle can be freed from these parasites before they are shipped from the infected district. A successful method for accomplishing this would be of such great importance to cattle raisers in the infected district that the experiments should be continued until definite results are reached.

Investigations have also been made of the disease known as rabies, particularly as it is discovered in the District of Columbia and vicinity. These investigations show the disease to be more prevalent than is usually supposed, and indicate the necessity of giving information which will enable those interested to identify the disease when it exists and adopt proper preventive measures.

An investigation has also been made of the peculiar disease of Florida and adjacent sections, popularly known as "leeches," and interesting results have been obtained. Full information concerning the investigations of this division will be found in special articles accompanying this report and in the bulletins issued from time to time.

WORK OF THE DAIRY DIVISION.

The dairy division was established July 1, 1895. Its work has consisted during the year in making a survey of the dairy industry of the United States and collecting such information as was needed for forming an intelligent idea of the magnitude, condition, and needs of the dairy industry. A number of bulletins have been issued, for which there has been a great demand, and much satisfaction has been expressed by those interested in dairy matters that the Department of Agriculture has established this division.

There is undoubtedly a great opportunity to assist those engaged in dairying by the collection and dissemination of information relating to this important industry. During recent years there has been a great development in dairy matters, many valuable discoveries have been made, and methods radically changed. This development is still in progress, and it is essential to the prosperity of the dairy industry in this country that those engaged in it should have early and reliable information of the devices for reducing the cost of production which have been successfully adopted here or elsewhere. It is the purpose

of the dairy division to collect such valuable information, to bring it properly to the attention of our dairymen, and to bring those engaged in dairying in closer and more helpful relations with each other.

UNITED STATES ANIMAL QUARANTINE.

The superintendents of the various animal quarantine stations report the names of the importers and the number and breed of each shipment imported during the fiscal year ending June 30, 1896, as follows:

Animals imported into the United States from foreign countries, and quarantined, for the fiscal year ending June 30, 1896.

PORT OF NEW YORK, N. Y.

[Quarantine station located at Garfield, N. J.]

Date of arrival.	Name and address of importer.	Port of shipment.	Breed and kind.	Number.
1895.				
July 24	Metcalf Bros., East Elma, N. Y	Liverpool, England	Berkshire swine	12
24	do	do	Dorsethorn sheep	5
24	do	do	Shropshire sheep	14
24	Dr. G. H. Davison, Millbrook, N. Y	do	Hampshire sheep	3
24	do	do	Lonks sheep	4
24	do	do	Jersey cattle	2
Sept. 4	A. H. Morris, 68 Broad street, New York.	do	Deer	1
4	do	do	Goat	1
4	do	do	Shropshire sheep	10
5	W. A. Perrin, Rochester, N. Y	do	do	2
5	Robert Davies, East Toronto, Canada.	do	Berkshire swine	4
5	J. T. Breckenridge, Austin, Tex	do	Ayrshire cattle	8
11	G. H. McFadden, 121 Chestnut street, Philadelphia.	do	Shropshire sheep	106
Oct. 17	John Mahoney, Rawlins, Wyo	do	Southdown sheep	10
Nov. 6	Wm. Rockefeller, Tarrytown, N. Y	do	Berkshire swine	2
14	W. A. Seward, Budd Lake, N. J	London, England	Dorsethorn sheep	100
14	T. S. Cooper, Coopersburg, Pa	Liverpool, England	Berkshire swine	2
14	H. D. Nichol, Nashville, Tenn	do	Berkshire boar	1
Dec. 28	J. T. Breckenridge, Austin, Tex	do	Norman cattle	8
1896.				
Jan. 7	J. B. Guillott, 1 Broadway, New York.	Hamburg, Germany	Camels	12
Apr. 8	Victor Rodile, Coney Island	Liverpool, England	Berkshire swine	1
16	H. D. Nichol & Son, Nashville, Tenn	do	Guernsey cattle	9
22	F. S. Peer, Mount Morris, N. Y	London, England	Jersey cattle	12
22	do	do	Ayrshire cattle	3
22	do	do	Kerry cattle	12
June 17	Whitelaw Reid, New York City	Liverpool, England	Hampshire sheep	11
17	do	do		

PORT OF BOSTON, MASS.

[Quarantine station located at Littleton, Mass.]

1895.				
Aug. 14	M. B. I. Goddard, East Greenwich, R. I.	Liverpool, England	Southdown sheep	1
31	C. I. Hood, Lowell, Mass	do	Berkshire swine	8
Nov. 5	M. B. I. Goddard, East Greenwich, R. I.	do	Southdown sheep	6

PORT OF BALTIMORE, MD.

[Quarantine station located at St. Denis, Md.]

1896.				
Apr. 12	Robert Taylor, Casper, Wyo	Liverpool, England	Hampshire Down sheep.	45

Animals imported into the United States from foreign countries, and quarantined, for the fiscal year ending June 30, 1896—Continued.

PORT OF BUFFALO, N. Y.

Date of arrival.	Name and address of importer.	Port of shipment.	Breed and kind.	Number.
1895.				
Oct. 26	Arthur E. Spahn, Corpus Christi, Tex.	Guelph, Ontario	Durham cattle	5
26	do	do	Devon cattle	29
Nov. 28	J. W. Wadsworth, Avon, N. Y.	Owen Sound, Ontario.	Canadian cattle	48
1896.				
Jan. 19	Miller & Sibley, Franklin, Pa	Toronto, Canada	Jersey cattle	5
Feb. 8	do	do	do	8
11	do	do	do	3
Apr. 17	J. W. Wadsworth, Avon, N. Y.	Owen Sound, Ontario.	Canadian cattle	130
30	do	do	do	126
May 8	do	do	do	26

PORT OF NEWPORT, VT.

1895.				
Nov. 27	W. H. Page, Lisbon, N. H	Danville, Quebec	Ayrshire cattle	7
1896.				
Feb. 26	John Lodge, Manchester, N. H	do	do	3

PORT OF PORT HURON, MICH.

1896.				
May 6	C. F. Reynolds, Denver, Colo	Guelph, Ontario	Hereford cattle	10

PORT OF VANCEBORO, ME.

1895.				
Dec. 4	W. R. Finson, Vanceboro, Me	St. Croix, New Brunswick.	Native cow	1

SHEEP AND SWINE IMPORTED WITHOUT QUARANTINE.

In the regulations for the inspection of imported animals, prescribed under the authority of the act of Congress, approved August 30, 1890, it is provided that sheep, other ruminants, and swine from North America, when accompanied by certain health certificates and other papers, may, after passing inspection, be admitted without quarantine. These animals are imported for immediate slaughter and also for breeding purposes. Those imported for the latter purpose, together with the name and address of the importer and place of origin, are reported by the inspectors of this Bureau along the Canadian border as follows.

Sheep and swine imported into the United States for breeding purposes through ports along the Canadian border, and not quarantined.

PORT OF PORT HURON, MICH.

Date of inspection.	Name and address of importer.	Breed and kind.	Port of shipment.	Number.
1895.				
July 12	J. W. McCrabe, Glasston, N. Dak	Yorkshire swine	Burford, Ontario	2
		Oxford sheep	Guelph, Ontario	15
23	Uriah Prevett, Greensburg, Ind	Shropshire sheep	do	6
		Southdown sheep	do	1
25	John A. Irion, Gallipolis, Ohio	Lincoln sheep	Lucan, Ontario	1
29	L. Austin, Norris City, Ill	do	St. Marys, Ontario	1
29	William Shier, Marlette, Mich	do	do	8
Aug. 6	And. Decker, Deckerville, Mich	Berkshire swine	Chesterfield, Ontario	1
		Oxford sheep	Guelph, Ontario	5
8	G. J. Campbell, Pittsfield, Ohio	Southdown sheep	do	6
8	J. E. Coffland, Richland Center, Wis	Shropshire sheep	Paris, Ontario	12
13	F. C. Babbit, Boyd, Wis	Southdown sheep	Hamilton, Ontario	2
13	Chris. Harvey, Carsonville, Mich	Yorkshire swine	Kincardin, Ontario	1
14	Wesley Neeley, Brooklyn, Mich	Oxford sheep	Guelph, Ontario	9
		Southdown sheep	Quebec	5
15	G. Allen & Son, Allerton, Ill	Shropshire sheep	Quarantine	31
		Oxford sheep	Quebec	9
		Cotswold sheep	do	6
16	George McKerrow, Sussex, Wis	Southdown sheep	Quarantine	6
		do	Pickering, Brampton, and Guelph, Ontario.	6
		Oxford sheep		20
16	F. W. Harding, Waukesha, Wis	Cotswold sheep		26
		Shropshire sheep		4
		do	Guelph, Ontario	28
		Lincoln sheep	do	27
17	Henry Struck, Park City, Mont	Oxford sheep	do	51
19	J. J. Hillen, Orchard Lake, Mich	Lincoln sheep	Ilderton, Ontario	2
21	J. J. Farquhar, Modoc, Ind	Shropshire sheep	Woodville, Ontario	1
21	C. Cook, Stanton, Mich	Oxford Down sheep	Guelph, Ontario	1
24	George Allen, Greenfield, Ind	Shropshire sheep	Pickering, Ontario	2
29	John Ross, Bucyrus, Ohio	do	do	38
Sept. 4	John Monell & Co., Ottumwa, Iowa	Yorkshire swine	London, Ontario	2
10	N. D. McNeil, Grand Blanc, Mich	Shropshire sheep	Mildmay, Ontario	5
14	N. A. Gentry, Indianapolis, Ind	Berkshire swine	Brampton, Ontario	14
18	A. O. Fox, Oregon, Wis	Shropshire sheep	Burford, Ontario	51
20	H. Arkell, Fort Fred Steele, Wyo	Oxford sheep	Guelph, Ontario	63
21	William Oliver, Springfield, Ill	Lincoln sheep	St. Mary's, Ontario	22
		Leicester sheep	Hamburg, Ontario	12
23	Carey & Whisler, Wakarusa, Ind	Shropshire sheep	do	53
25	C. H. Hudson, Shepardsville, Mich	Lincoln sheep	Ilderton, Ontario	1
27	D. K. Hitchcock, Kendallville, Ind	Shropshire sheep	Lindsay, Ontario	1
30	H. A. Rohr, Napoleon, Ohio	Lincoln sheep	Ailsa Craig, Ontario	1
Oct. 1	C. C. Rice, Racine, Wis	do	Ilderton, Ontario	52
1	S. F. Cole, Miles City, Mont	do	do	3
1	S. T. Rignall, Flowerfield, Mich	do	do	1
2	S. W. Collins, Cynthiana, Ky	do	do	2
4	J. F. Weaver, Sussex, Wis	Shropshire sheep	Woodville, Ontario	1
4	F. W. Harding, Waukesha, Wis	Cotswold sheep	Uxbridge and Brampton, Ontario.	121
4	A. Buckman, Buchanan, Mich	do	Brampton, Ontario	1
5	F. L. Evans, Plano, Ill	Yorkshire swine	Newcastle, Ontario	4
9	Gradner & Kammerer, Brodhead, Wis.	Shropshire sheep	Pickering, Ontario	1
9	M. Levering, La Fayette, Ind	do	do	1
9	S. W. Hayes, Oconomowoc, Wis	do	Mildmay, Ontario	10
9	D. Campbell, Carson City, Mich	do	Watford, Ontario	1
13	J. G. Massey, Rawlins, Wyo	Hampshire Down sheep.	Quebec Quarantine	232
22	T. Frizzel, Brooklyn, Iowa	Lincoln sheep	Ilderton, Ontario	13
22	J. A. Irion, Gallipolis, Ohio	do	Lucan, Ontario	1
22	W. M. Bennington, Volga, Iowa	Leicester sheep	Forrest, Ontario	1
23	J. McKenzie, Emmett, Mich	do	do	12
		do	Guelph, Ontario	15
		Leicester (grade) sheep.	do	60
24	C. W. Barney, Aurora, Ill	Cotswold sheep	do	44
		Oxford sheep	do	50
		Berkshire swine	Ontario	1
25	F. W. Harding, Waukesha, Wis	Cotswold sheep	do	216
26	H. F. Schwartz, Box Elder, Mont	do	Brampton, Ontario	70
29	do	do	do	70
29	S. F. Cole, Miles City, Mont	Lincoln sheep	Ilderton, Ontario	3
29	William Shiar, Marlette, Mich	do	London, Ontario	1
30	M. Levering, La Fayette, Ind	Shropshire sheep	do	1

Sheep and swine imported into the United States for breeding purposes through ports along the Canadian border, and not quarantined—Continued.

PORT OF PORT HURON, MICH.—Continued.

Date of inspection.	Name and address of importer.	Breed and kind.	Port of shipment.	Number.
1895.				
Oct. 30	C. F. Curtis, Ames, Iowa	Shropshire sheep	Guelph and London, Ontario.	8
		Lincoln sheep		13
		Cotswold sheep		10
		Dorset sheep		11
		Oxford sheep		10
		Leicester sheep		10
		Southdown sheep		10
		Suffolk sheep		11
		Tamworth swine		3
		Yorkshire swine		3
30	R. Sangster, Mount Pleasant, Mich	Cotswold sheep	Stouffville, Ontario	1
Nov. 2	W. C. Nichols & Son, Cresco, Iowa	Shropshire sheep	Pickering, Ontario	1
2	A. A. Livingston, Marlette, Mich	Berkshire swine	Stafford, Ontario	3
6	A. I. Fox, Oregon, Wis	Shropshire sheep	Burford, Ontario	64
8	F. W. Harding, Waukesha, Wis	Cotswold sheep	Ontario	165
		Oxford sheep	do	4
9	W. H. Dennis, Deckerville, Mich	Berkshire swine	Woodstock, Ontario	1
11	William Grimes, Yale, Mich	do	Ailsa Craig, Ontario	1
12	W. F. Harding, Waukesha, Wis	Cotswold sheep	Forrest, Ontario	69
12	A. E. Green, Orchard Lake, Mich	Shropshire sheep	Owen Sound, Ontario	14
14	H. E. Hudson, Shephardsville, Mich	Lincoln sheep	Ilderton, Ontario	10
16	John Large, Brown City, Mich	Cotswold sheep	Kenwood, Ontario	1
16	W. J. Boynton, Rochester, Minn	Shropshire sheep	London, Ontario	61
16	do	do	Pickering, Ontario	54
19	C. & W. Frazer, Green Valley, Ill	do	Woodville, Ontario	6
19	Gardner & Kammerer, Brodhead, Wis.	do	Pickering, Ontario	24
29	F. W. Harding, Waukesha, Wis	Cotswold sheep	Brampton, Ontario	75
Dec. 2	John Ross, Bucyrus, Ohio	Shropshire sheep	Pickering, Ontario	36
3	W. B. Green, Indianola, Ill	Berkshire swine	Chesterfield, Ontario	4
4	T. M. Crocker, Mount Clemens, Mich.	Yorkshire (imp.) swine.	Burford, Ontario	1
5	J. V. Coltin, Casper, Wyo	Shropshire sheep	Clinton, Ontario	120
7	G. Harding & Son, Waukesha, Wis	Cotswold sheep	Brampton, Ontario	25
11	F. G. Woodruff, Climax, Mich	Lincoln sheep	Ilderton, Ontario	23
12	A. McKay, Edina, Mo	Shropshire sheep	Mildmay, Ontario	2
13	William Newton, Pontiac, Mich	do	Guelph, Ontario	3
		Leicester sheep	do	3
13	Alex. Reed, Port Huron, Mich	Tamworth swine	Sarnia, Ontario	1
		Chester white swine	do	2
17	F. L. Evans, Plano, Ill	Yorkshire swine	Kerby, Ontario	1
19	John Marshall, Imlay City, Mich	Leicester sheep	Guelph, Ontario	8
19	S. Bennington, Volga, Iowa	Cotswold sheep	Forrest, Ontario	2
1896.				
Jan. 8	Graham & Kemmerer, Brodhead, Wis.	Shropshire sheep	Pickering, Ontario	57
14	Alex. Fesler, Grindstone City, Mich	Cotswold sheep	Forrest, Ontario	1
17	Post & Bigford, Ovid, Mich	Lincoln sheep	Ilderton, Ontario	22
31	Margaret Milen, Croswell, Mich	Leicester sheep	Shelburne, Ontario	4
Feb. 4	Ed. Drayer, Ithaca, Mich	Cotswold sheep	London, Ontario	6
5	W. L. Carson, Ramsey, Ill	Lincoln sheep	Ilderton, Ontario	1
24	G. R. Sheats, Cromwell, Iowa	Shropshire sheep	Hespler, Ontario	1
26	Henry Holbrook, Laotto, Ind	Oxford sheep	Guelph, Ontario	5
27	F. W. Harding, Waukesha, Wis	Cotswold sheep	Brampton, Ontario	103
Mar. 3	H. A. Daniels, Clio, Mich	Lincoln sheep	Lucan, Ontario	15
4	W. J. Carson, Ramsey, Ill	do	Ilderton, Ontario	4
10	G. & G. M. Boothly, Goose Lake, Iowa.	Tamworth swine	Petersburg, Ontario	3
26	E. P. Oliver, Flint, Mich	Berkshire swine	Toronto, Ontario	26
27	Jas. Hunter, Carrington, N. Dak	Shropshire sheep	Alma, Ontario	7
27	Henry L. Holbrook, Laotto, Ind	Oxford sheep	Guelph, Ontario	4
Apr. 2	George Harding, Waukesha, Wis	Cotswold sheep	London, Ontario	32
3	Thomas Price, Oblong, Ill	Berkshire swine	Park Hill, Ontario	3
8	J. O. Ducker, Albion, Mich	Oxford swine	Lucan, Ontario	9
		Lincoln swine	do	8
8	J. H. Thompson, Huntley, Ill	Tamworth swine	Petersburg, Ontario	2
8	J. Ellard, Jeffersonville, Ohio	do	do	1
9	H. B. Willet, Lansing, Mich	Lincoln sheep	Lucan, Ontario	1
15	J. E. Coffland, Richland Center, Wis.	Shropshire sheep	Paris, Ontario	66
		Cotswold sheep	do	5
24	Henry Parr, Alpena, Mich	Tamworth swine	Sarnia, Ontario	1
28	J. L. Littlefield, Farwell, Mich	Yorkshire swine	London, Ontario	2
30	William Farr, Richmond, Mich	Tamworth swine	Sarnia, Ontario	1
May 13	A. Campbell, Reed City, Mich	Yorkshire swine	London, Ontario	1
14	W. F. Wilcox, Hugo, Minn	do	do	3
June 11	P. D. Fuller, Sutherland, Iowa	Tamworth swine	Brucefield, Ontario	2

Sheep and swine imported into the United States for breeding purposes through ports along the Canadian border, and not quarantined—Continued.

PORT OF DETROIT, MICH.

Date of inspection.	Name and address of importer.	Breed and kind.	Number.
1895.			
July 5	C. C. Ramer, Brecksville, Ohio	Oxford Down sheep	1
17	J. S. Taylor, Henderson, Tenn	Berkshire swine	2
17	W. W. Wilson, Muncie, Ind	Cotswold sheep	1
23	do	do	1
26	E. G. McComas, Sturgeon, Mo	Berkshire swine	2
Aug. 1	H. Ruzey, Fairmount, Ill	Cotswold sheep	1
10	W. W. Wilson, Muncie, Ind	do	1
13	V. Meredith, Cambridge City, Ind	Southdown sheep	1
13	D. E. Lowell, Rising Sun, Ind	do	1
13	G. Richardson, Benedict, Nebr	Leicester sheep	1
14	C. J. Kellog, Reading, Mich	Southdown sheep	1
16	M. Williams, Muncie, Ind	Shropshire sheep	6
		Cotswold sheep	1
21	Ed. Morris, Glasgow, Ky	Berkshire swine	2
Sept. 3	William Newton, Detroit, Mich	Leicester sheep	4
6	Robinson & Hagerty, Hanover, Ohio	Southdown sheep	1
11	John Rife, Clyde, Ohio	Cotswold sheep	1
14	D. G. Hammer, Cambridgeboro, Pa	Shropshire sheep	1
16	Coffland & Leybrand, Milwaukee, Wis	do	1
18	G. W. Shetler, Beach City, Ohio	Lincoln sheep	3
24	E. Morris, Glasgow, Ky	Berkshire swine	4
24	C. E. Emmons, Freeport, Mich	do	1
26	Robt. Lowrie, Greensburg, Pa	Shropshire sheep	1
27	E. Campbell, Oberlin, Ohio	Oxford Down sheep	1
Oct. 5	H. Phillips, Detroit, Mich	Shropshire sheep	1
14	Valentine Bros., Dexter, Mich	Lincoln sheep	2
15	Mr. Bewick, Detroit, Mich	Shropshire sheep	1
23	John McCallum, Cass City, Mich	Leicester sheep	2
25	George Bradford, Ovid, Mich	Lincoln sheep	15
29	John Hudgins, Chillicothe, Ohio	Cotswold sheep	2
30	I. J. Hiller, Four Towns, Mich	Lincolnshire sheep	2
Nov. 1	H. P. Gager, St. Johns, Mich	Lincoln sheep	1
5	Mrs. N. W. Culver, Hamilton, Ohio	Berkshire swine	2
7	J. R. Thompson, Earlham, Iowa	Oxford Down sheep	1
15	George M. Hendrie, Detroit, Mich	Shropshire sheep	1
15	do	Jersey Red swine	3
18	J. W. Arnold, Washington, Ga	do	2
29	W. C. Fraizer, Atlantic, Iowa	Southdown sheep	1
Dec. 5	J. Hoffman, Dunkirk, Ohio	Lincoln sheep	7
6	R. H. Lidd, Livingston, Ala	Berkshire swine	2
10	T. Whitaker, Stone Bank, Wis	do	1
10	J. A. McGiven, Rudyard, Mich	do	2
11	H. Reaves, Hartsville, Tenn	do	2
27	R. H. Hoskins, Louisville, Ky	do	4
		Yorkshire swine	1
		Jersey Red swine	1
1896.			
Jan. 14	L. L. Byrne, Morganfield, Ky	Berkshire swine	2
15	William Messner, Cass City, Mich	Jersey Red swine	2
21	J. P. Foster, Beaver Dam, Ky	Berkshire swine	2
22	W. J. Samuels, Bardstown, Ky	do	18
24	L. E. Healey, Butler, Ind	Oxford Down sheep	1
Feb. 5	W. H. Irvine, Anchorage, Ky	Berkshire swine	2
8	D. J. Wild, Charlotte, Mich	Lincoln sheep	55
8	W. H. Rey, Roswell, N. Mex	Leicester sheep	2
14	D. Bleam, Elm Dale, Mich	Yorkshire swine	1
27	L. W. Preston, Glasgow, Ky	Berkshire swine	1
Mar. 17	W. H. Irving, Anchorage, Ky	do	1
17	Henry Dirksour, Binger, Ind	Chester White swine	1
19	N. B. S. Roke, Gladwin, Mich	do	1
26	N. B. Rushmore, Linesville, Pa	Berkshire swine	3
Apr. 14	A. G. Wilcox, Hugo, Minn	Chester White swine	1
15	Edson K. Care, Jonesville, Mich	Lincoln sheep	1
16	Agricultural College, Clines, Iowa	Jersey Red swine	3
22	E. E. Stine, Cuyahoga Falls, Ohio	Berkshire swine	1
May 19	Charles Friend, Madisonville, Ky	do	1
June 3	E. E. Stine, Cuyahoga Falls, Ohio	do	1

Sheep and swine imported into the United States for breeding purposes through ports along the Canadian border, and not quarantined—Continued.

PORT OF BUFFALO, N. Y.

Date of inspection.	Name and address of importer.	Breed and kind.	Port of shipment.	Number.
1895.				
Aug. 19	W. N. Noble, Brecksville, Ohio	Southdown sheep	Paris, Canada	1
21	L. J. Bashford, Hudson, N. Y	do	Abingdon, Canada	1
21	L. M. Reed, Burton, Ohio	do	Edmonton, Canada	2
22	W. N. Noble, Brecksville, Ohio	do	Caledonia, Canada	4
27	F. Schaffer, Newcastle, Pa	Shropshire sheep	Delaware, Canada	1
Sept. 11	C. H. Lyons, Kingsville, Ohio	do	Maple Lodge, Canada	4
14	J. Blodgett, Olmsted Falls, Ohio	do	Woodville, Canada	1
21	J. G. Dam, Warren Tavern, Pa	Southdown sheep	Abingdon, Canada	1
22	G. W. Chapman, Winters, Cal	Lincoln sheep	London, Canada	1
23	Wm. Ringwalt, Newcastle, Ohio	Shropshire sheep	Burford, Canada	3
Oct. 15	L. M. Crothers, Taylorstown, Pa	Southdown sheep	Paris, Canada	2
17	do	do	Toronto, Canada	1
23	Shook Bros., Rising Springs, Ohio	Berkshire hog	Edmonton, Canada	1
30	Sidney Sprague	Shropshire sheep	...	1
31	S. G. Millard	Oxford Down sheep	...	1
Nov. 5	John D. Hass	Southdown sheep	...	4
6	W. J. Dale	{Cotswold sheep	...	3
		{Leicester sheep	...	1
23	D. Shaffer, Newcastle, Pa	Oxford Down sheep	London, Canada	1
Dec. 10	W. N. Wakeman, Dalton, N. Y	Lincoln sheep	do	4
28	A. P. Wagner, Elizabeth, Pa	Yorkshire hog	Burford, Canada	1
1896.				
Jan. 1	J. Mortimer, Hempstead, N. Y	Yorkshire boar	Delaware, Canada	1
6	D. W. White, Hornellsville, N. Y	Leicester sheep	Durwick, Canada	4
24	M. J. Kuhen, Buffalo, N. Y	Yorkshire swine	Wellington, Canada	7
31	James Plumstead, Buffalo, N. Y	Berkshire swine	Paris, Canada	2
Feb. 13	H. M. White, Hornellsville, N. Y	Cotswold sheep	Caledonia, Canada	3
13	W. W. Burns, Salem, Ohio	Berkshire swine	Snelgrove, Canada	1
Apr. 7	A. A. Bates, Irwin, Ohio	Shropshire sheep	St. Marys, Canada	93
18	Metcalf Bros., East Elma, N. Y	Yorkshire swine	Woodstock, Canada	1
21	do	Berkshire swine	Stratford, Canada	1
May 8	John Anderson, Coudersport, Pa	Yorkshire swine	Whitby, Canada	3
19	S. V. McDowell, Fredonia, Pa	Cotswold sheep	Uxbridge, Canada	1
June 27	A. F. White, Hornellsville, N. Y	Shropshire sheep	Clinton, Canada	9

PORT OF SUSPENSION BRIDGE, N. Y.

1895.				
July 23	A. Bardwell, Corfu, N. Y	Oxford Down sheep	Guelph, Canada	4
Aug. 3	W. H. Chaffee, Brecksville, Ohio	Shropshire sheep	Woodsville, Canada	1
3	J. G. Hanen, Middletown, Md	do	London, Canada	71
5	A. Cummings, Cuylerville, N. Y	do	Brooklyn, Canada	2
15	H. W. Keyes, North Haverhill, N. H	do	Pickering, Canada	4
27	J. Wyler, Fredericksburg, Ohio	do	Woodville, Canada	1
Sept. 10	W. S. Wells, Little Genesee, N. Y	do	Claremont Station, Canada.	1
25	J. G. Hanen, Middletown, Md	do	London, Canada	69
27	G. J. Campbell, Trenton, N. J	American Oxford Down sheep.	Gourock, Canada	1
Oct. 2	J. H. Todd, Wakeman, Ohio	Shropshire sheep	Woodville, Canada	1
10	W. S. Hawkshaw, Hagerstown, Md	do	Glenwood, Canada	59
15	S. D. Hewlett, Pleasant Valley, N. Y	Lincoln sheep	Abingdon, Canada	1
15	C. Gordon, Bellefonte, Pa	American Oxford Down sheep.	Inderman, Canada	1
Nov. 13	W. C. Woods, Knoxville, Pa	Oxford Down sheep	Culvers, Canada	1
15	Massachusetts Agricultural School, Amherst.	Berkshire hogs	Amber, Canada	2
Dec. 11	J. G. Hanen, Middletown, Md	Shropshire sheep	Glenwood, Canada	50
12	Daniel Lappan, Arlington, Mass	Yorkshire hog	Streetsville, Canada	1
1896.				
Jan. 18	J. G. Hanen, Frederick, Md	Shropshire sheep	Glenwood, Canada	119
24	Elliott Warren, Winston, N. C	Berkshire hog	Dearborn, Canada	1
Mar. 18	J. A. Andrews, Charlottesville, Va	Yorkshire boar	Delaware, Canada	1
Apr. 15	William Wilkinson, Lewiston, N. Y	Chester White hog	do	1
16	J. G. Hanen, Middletown, Md	Shropshire sheep	Glenwood, Canada	69
May 12	H. W. Chaffee, Brecksville, Ohio	do	Woodville, Canada	2
22	Mrs. J. A. Wilson, Pittsfield, Ohio	Berkshire hog	Belleville, Canada	1
29	Valentine Schenck, Southampton, N. Y.	Yorkshire swine	St. Jacobs, Canada	2
June 10	Aaron Bardwell, Fargo, N. Y	Oxford Down sheep	Guelph, Canada	2

Sheep and swine imported into the United States for breeding purposes through ports along the Canadian border, and not quarantined—Continued.

PORT OF OGDENSBURG, N. Y.

Date of inspection.	Name and address of importer.	Breed and kind.	Number.
1895.			
Aug. 2	State Asylum, Utica, N. Y	Yorkshire swine	1
Oct. 23	Curtis Bristol	Shropshire sheep	1
Nov. 21	George Bennett, Dekalb, N. Y	Berkshire swine	2
1896.			
Mar. 27	Guy I. Colman, Watertown, N. Y	Oxford sheep	1
May 26	F. A. Folger	Jersey swine	2
June 17	F. A. Boshart, Lowville, N. Y	Berkshire swine	1

PORT OF MORRISTOWN, N. Y.

1895.			
Sept. 30	William Lanyer	Leicester sheep	1
Oct. 23	Curtis Bristol	Shropshire sheep	1
1896.			
Mar. 12	R. B. McGregor, North Hammond, N. Y	Yorkshire swine	2
May 22	Henry Brenaugh, Redwood, N. Y	Berkshire swine	1

PORT OF ROUSE POINT, N. Y.

1895.			
Sept. 24	Jas. Dwyer	Leicester sheep	1
26	P. B. Brackin	do	5
Oct. 23	S. W. Webb	Cotswold and Shropshire sheep.	275

PORT OF ST. ALBANS, VT.

1895.			
Oct. 10	Phillips Bros	Leicester sheep	1
Nov. 3	C. H. Marshall	Lincoln sheep	14
1896.			
Jan. 18	Joseph Ballard, Georgia, Vt	Shropshire sheep	49

PORT OF NEWPORT, VT.

1895.			
Sept. 3	Moses Lovelett	Southdown sheep	6
Dec. 23	Isaac Parnell	Leicester sheep	1
1896.			
Jan. 21	B. B. Morrill, Bryants Pond, Me	Oxford sheep	1
Feb. 17	E. A. Baldwin, Stanstead, Quebec	Southdown sheep	3
25	John Lodge, Manchester, N. H	Yorkshire swine	2
		Berkshire swine	1
Mar. 17	B. B. Morrill, Bryants Pond, Me	Oxford sheep	3
May 20	J. Turner Routledge, Georgeville, Quebec	Yorkshire swine	2
June 27	Edward Gallagher, St. Marie, Quebec	White Chester swine	3

PORT OF ISLAND POND, VT.

1895.			
July 9	Leon Gague	Leicester sheep	3
Sept. 7	A. A. La Ferrier	do	1
Oct. 23	J. A. Cochrane	Shropshire sheep	2
Nov. 12	W. S. Hawkshaw, Hagerstown, Md	do	1
1896.			
Apr. 21	Oscar Shirley, Houlton, Me	Yorkshire swine	2

PORT OF BEECHER FALLS, VT.

1895.			
July 16	Mark Goddard	White Chester swine	2
Aug. 3	J. Y. Keazer	Leicester sheep	1

Sheep and swine imported into the United States for breeding purposes through ports along the Canadian border, and not quarantined—Continued.

PORT OF HOULTON, ME.

Date of inspection.	Name and address of importer.	Breed and kind.	Number.
1895.			
July 16	Wilfred Hardy	Chester White swine	4
Aug. 20	J. A. Collins	Cotswold sheep	2
21	H. N. Piper	Common stock	13
Sept. 14	J. M. Clark	Berkshire swine	1
20	O. Shirley	Shropshire sheep	1
Oct. 12	F. Theriault	Chester White swine	3
Nov. 2	C. Ackerson	Grade Leicester sheep	7
6	F. Theriault	Chester White swine	1
12	J. M. Clark	do	2
1896.			
June 3	J. A. Britton, Monticello, Me	Cotswold sheep	24

PORT OF VANCEBORO, ME.

Date of inspection.	Name and address of importer.	Kind.	Port of shipment.	Number.
1895.				
Oct. 4	George Moffit, Oldtown, Me	Swine	York County, New Brunswick.	8
1896.				
May 5	Jane McDonald, Boston, Mass	do	Bel River, New Brunswick.	2

PUBLICATIONS.

There have been published during the fiscal year the following reports, bulletins, pamphlets, circulars, and map:

Tenth and Eleventh Annual Reports of Bureau of Animal Industry, 1893–94.
Report of the Chief of the Bureau of Animal Industry for 1895. (Reprint.)
The Federal Meat Inspection. (Reprint from Yearbook.)
The Pasteurization and Sterilization of Milk. (Reprint from Yearbook.)
Some Practical Suggestions for the Suppression and Prevention of Bovine Tuberculosis. (Reprint from Yearbook.)
The Dairy Herd, its Formation and Management. (Reprint from Yearbook.)
Bulletin No. 8. Investigations Concerning Infectious Diseases Among Poultry.
Bulletin No. 9. Rules and Regulations Governing the Operations of the Bureau of Animal Industry.
Bulletin No. 10. Corn-Stalk Disease and Rabies in Cattle.
Bulletin No. 11. Statistics of the Dairy.
Circular No. 3. A Nodular Tæniasis in Fowls.
Circular No. 4. Crossing Improved Breeds of Swine with the Common Hogs of Florida.
Circular No. 5. The Direct Transmission of Infectious Entero-Hepatitis in Turkeys.
Circular No. 6. Black Quarter. (Reprint.)
Circular No. 7. Actinomycosis, or Lumpy Jaw. (Reprint.)
Circular No. 8. Injuries to Cattle from Swallowing Pointed Objects. (Reprint.)
Circular No. 9. Check List of the Animal Parasites of Chickens.
Circular No. 10. List of the State Dairy Commissioners and Associations of Dairymen in the United States and Canada for 1896.
Circular No. 11. How to Select Good Cheese.
Circular No. 12. Check List of the Animal Parasites of Turkeys.
Circular No. 13. Check List of the Animal Parasites of Ducks.
Circular No. 14. Check List of the Animal Parasites of Geese.
Butter Substitutes. (Reprint from Yearbook.)
Inefficiency of Milk Separators in Removing Bacteria. (Reprint from Yearbook.)
The Manufacture and Consumption of Cheese. (Reprint from Yearbook.)
Map Showing the Boundary Line of the District Infected with Splenetic, or Southern Fever of Cattle.

CONTAGIOUS DISEASES OF ANIMALS IN GREAT BRITAIN.

In recent years animal diseases have become subjects of universal popular interest. This is due largely to the fact that the modern practice of eradicating contagious disease or of preventing its spread by legislative process has incited an apparent conflict between the aims of veterinary science and the interests of commerce. For, when any one great stock-trading country of the world adopts a system of legislating contagious animal diseases out of existence, immediately the commerce in stock, not only of that country, but of other parts of the world, may be indirectly and for the time being injuriously affected. The causes of this are obvious. The fundamental scientific principle for the extinction of all contagious disease by legislation is the isolation both of diseased animals and of all that have been exposed to infection. Manifestly the first step toward the success of such isolation is the imposition of rigid restrictions upon commerce in animals throughout all the regular channels of foreign and domestic trade by which the contagion might be introduced. A nation which seeks to free itself from a contagious animal disease first imposes restrictions upon the importation of all susceptible animals from infected nations. Likewise a community, however small, which has the same object in view, primarily regulates or prohibits traffic in susceptible stock from infected communities. Commercial interests are thus made subservient to the interests of the community. Temporary inconvenience and loss are imposed upon the owner, the dealer, the transporter, and all others interested in stock traffic for the single purpose of insuring to a country the permanent and inestimable benefits to be derived from the total extinction of a specified disease.

A popular interest in this subject even more universal than that which is associated with commercial operations has lately arisen from the discovery that some animal diseases, notably tuberculosis and anthrax, are communicable to man. It is, also, an indisputable fact that some diseases of the human race may arise from food derived from animals and thus the operations of veterinary science, particularly in their relations to the abattoir and the dairy, are matters of vital interest to every household.

From the prominence of Great Britain, both as an importing and as an exporting nation, the subject of animal diseases with reference to that country is one of exceptional interest to the whole civilized world. In food-producing animals, being the greatest importing country, the rigid system of laws which that Government has adopted to prevent the introduction of animal diseases into its native herds from foreign lands has indirectly had a far-reaching effect upon the animal industry

of all other exporting nations. From some countries infected with communicable diseases the English Government prohibits the importation of certain species of animals entirely. From others, the importation of certain species is permissible only for slaughter at the port of debarkation. Within the Kingdom severe restrictions have been from time to time imposed upon the movement and transportation of animals from one part of the country to another, with a view to checking the spread of disease. These restrictive operations have been productive of many indirect results. Commerce in animals has been temporarily restricted in order to insure future permanent freedom in the traffic. The sanitary conditions, not only of home-bred animals, but of those of exporting nations, have been greatly improved. The humane as well as economical care of stock during transportation has also been a natural sequence of this sanitary legislation. Moreover, what is of more general importance, the prices and quality of animals and of the food derived from them have been greatly affected both in England and in countries tributary to her trade.

The importance of Great Britain as an exporting country is aptly epitomized in the expression that "England is the great stud farm of the world." Resorted to by all the great stock-producing countries for finely bred animals with which to cross and improve their native breeds, England is ever a center of interest to the stock-breeders of all nations both in respect to the purity of its breeds and also in respect to the health of its stock. It is recognized that not only is an obscure contagious disease, which already exists there, liable, through the medium of a highly prized animal, to be transported across the seas and introduced into a finely bred herd of another country, but experience teaches that in the British Isles danger always exists of sudden and often inexplicable outbreaks of diseases which can not be wholly warded off even by the absolute prohibition of the importation of living animals, but may be carried into and spread rapidly throughout the country by the importation of hides or other substances capable of carrying the contagion. The system which Great Britain has adopted for the suppression and prevention of animal diseases within her own borders reduces danger to the stock of other countries through the medium of importation to the lowest possible minimum, but the prevention of the importation of disease from any one country to another must always be dependent for success principally upon excessive caution and the rigidity of the enforcement of the preventive laws of the importing nation.

The animal diseases now prevalent in Great Britain which are of the greatest importance are those pertaining to cattle and swine. Of late years swine fever has attracted the greatest part of the attention of the veterinary department both because it affects more animals than any other disease and because of its stubborn resistance to the vigorous efforts that have been made for its extinction. Moreover, it is the only swine disease that has been made a subject of legislation. The contagious diseases of cattle have always been of more importance from an economic point of view than other animal diseases chiefly because of the greater susceptibility of cattle to the more destructive maladies, and because of the greater individual and aggregate value of this species. The contagious diseases which now prevail among the cattle of the United Kingdom and (excepting tuberculosis) are legislated against by the contagious diseases [animals] acts are pleuro-pneumonia, foot-and-mouth disease, tuberculosis, and anthrax; and,

though the first two are almost extinct, their remarkably infectious nature makes the existence of only a few centers of these diseases of more importance than would be a much wider distribution of less contagious maladies.

Appended will be found a brief history, compiled from recent English authorities, of the origin and dissemination of swine fever and of the contagious diseases of cattle in Great Britain, with special reference to the success or failure of the extinctive efforts of legislation.

SWINE FEVER.

The disease specifically known in England as swine fever is identical, so far as scientific investigations have determined, with hog cholera in the United States. Under the former title the malady is recognized in the veterinary legislation of Great Britain as the one contagious disease peculiar to swine with which that country is afflicted; and if other contagious swine diseases, such as swine plague, rouget, etc., prevail, they have not been differentiated in the laws for the suppression of animal diseases, but have been dealt with under the generic title "swine fever." The English authorities have maintained that such a differentiation is of little importance in this case, where a disease or diseases are to be exterminated by one process of law, because a classification neither modifies the urgent necessity of exterminating the plague nor alters the means to be used in its suppression. Moreover, it has been authoritatively proved that the prevalence of other contagious diseases peculiar to swine in Great Britain is of comparatively little importance. In the visceral examinations made by the qualified veterinary authorities, the intestinal lesions show that in a great majority of the cases where a contagious disorder is found to have affected the animals, the disease was specifically swine fever; and the lesions in such cases were usually identical with those characteristic of hog cholera in the United States. It is therefore said that for all practical purposes the difference between the two diseases is one of nomenclature. The value of this conclusion is that a system of extermination, successfully applied to one, is made equally effective in the extinction of the other.

The most conspicuous fact in the history of swine fever in Great Britain is that eighteen years of exterminative efforts have failed to check the prevalence of the disease to any satisfactory extent. More dreaded maladies—cattle plague, foot-and-mouth disease, and pleuro-pneumonia—have in that period yielded almost entirely to the rigid system of restrictions; but swine fever alone, once the least regarded of all these afflictions, continues to elude all measures for its extinction. For the past two years the suppressive action has been of extraordinary vigor, but the result has been ineffectual. The latest official utterance regarding the scourge is that it "combines the fatality of cattle plague with the obscureness of pleuro-pneumonia." This failure in extermination has been attributed to many diverse causes, ranging at first from an apathetic disregard of the disease in the minds of the people to a later claim of inefficient administration of the laws.

How long swine fever may have prevailed in England, or whence it came, are subjects of obscurity. It is only known that it received its first recognition as a specific disease in 1862. Very meager, too, is all knowledge of the earlier history of the malady. During the first sixteen years of its recognized existence no official reports were made upon the contagion, and what is known is of that vague and unreliable

character that attaches to all unsystematized and disconnected reports from individual sources.

The first authentic and comprehensive knowledge of the prevalence of swine fever in Great Britain dates from 1878. In that year it was for the first time included in the list of contagious diseases sought to be eradicated by the diseases of animals acts and the orders of the privy council issued thereunder. Annual official reports were made necessary by the terms of the acts, and the first of these, issued for the year 1879, indicated the extent of prevalence that the disease had gained; fifty-three counties were returned as infected and over 79,000 swine had been attacked by the fever.

The effort thus begun in 1878 to free the country from this plague was continued under the same general system of extermination, contracted or expanded to meet the exigencies of occasions, until November, 1893. And the fact that after this prolonged trial the system was pronounced a failure and was then subjected to radical modifications makes a brief review of the alleged causes of the failure a subject of instructive interest.

The futility of this fifteen years' struggle is ascribed principally to a lack of uniformity in action among the multiplicity of authorities intrusted with the execution of the laws for suppression. That this was a prominent factor in the failure is easily apparent. Great Britain, for the purposes of the diseases of animals acts, is divided into upward of 300 different county and borough districts. Each of these districts is controlled by a separate committee, or perhaps several subcommittees, each having a separate executive staff. These committees are denominated local authorities, and they have supervision over police and sanitary regulations, etc. It should be noted that each authority is entirely independent of the others, and the power of each, absolute within its individual district, ceases immediately when the confines of that district are reached. Thus the power of a county authority does not extend within the boundaries of a borough. A semblance of unity and cohesion, however, was given to these separate districts by the institution of a central anthority, first the privy council and afterwards the board of agriculture; but its functions, which will be described later, were of an advisory rather than of an executive nature, and therefore modified only to a slight degree the independence and individual responsibility of each district for the enforcement of sanitary laws within its confines. It may therefore be said that the diseases of animals acts, during the period from 1878 to 1893, intrusted the local authorities with almost the entire execution and administration of the laws for suppressing animal diseases within their respective districts. The detection of the disease—that first and most important step in a stamping-out system—depended solely upon the local authorities, although in this responsibility they were aided by an enactment that made the declaration to such authorities of the existence of a contagious disease compulsory upon the owners of the stock among which it occurred. The next important step, the determination of the true nature of the contagion, likewise devolved upon the skill of the veterinary surgeons of local authorities. And the existence of a contagious disease having been thus definitely ascertained, almost all the subsequent machinery of extirpation was managed by the local authorities; in them was vested the power to declare a place infected and to impose all the consequent restrictions upon the movements of animals in, into, and out of such localities; upon them, too, rested the responsibility of slaughtering

all diseased and in-contact animals within two days after the disease was discovered, and of compensating for them out of the local rates. Moreover, the supervision of the operations of disinfection, the declarations that infected places were free from infection, and the prevention of the entrance of swine from infected districts into their respective territories, were all functions of the local administrations. The important prerogative of prohibiting the exposure of all swine at fairs, shows, markets, and auctions within their respective districts was also exercised by the local authorities by and with the consent of the privy council. Great Britain was thus divided into over 300 independent districts, and, while all were presumably pervaded by a unanimous desire for the extirpation of animal diseases and guided to that end by a common law, each was more or less free to exercise stringency or laxity in the execution of that law, and all were influenced by such diverse local interests as made combined and determined action impossible of attainment.

The chief defect of this local administrative system was that neither the electors nor the elective authorities were usually possessed of any adequate appreciation of their responsibilities or of any comprehensive knowledge of the far-reaching measures and inflexible precautions necessary to cope successfully with so infectious and insidious a disease as swine fever. In some instances the inutility of the system as administered was manifest at the very starting point—the detection of the disease. The indifference of farmers and stock owners, especially in districts where the aggregate value of the swine was small and where the animals were in the hands of persons of small means—and such were not usually alert to the importance of the suppressive measures—made a thorough detection impossible. The result invariably was that from these hidden or undetected centers the affection was often spread to other districts where the existence of swine in large numbers had stimulated the interested persons to the most vigorous efforts for the extinction of the scourge. The result of a successful war of extermination in one district was thus often neutralized by a reintroduction of the infection from others. In other instances it was found that, even after the centers of infection had been discovered, the decision of the veterinary officials as to the true nature of the contagion was sometimes faulty, liability to error, in a disease often so obscure in its symptoms as swine fever, being increased by the fact that solely antemortem appearances frequently had to be relied upon, since there was no power to slaughter for purposes of diagnosis. It often happened, therefore, that a failure through accident or ignorance to recognize the disease in suspected cases left a center of contagion that eventually proved disastrous to herds of the surrounding country. The most fruitful source, however, of the continuance of this most fatal of all contagious diseases of swine was doubtless the perfunctory manner in which many authorities exercised the power of slaughtering diseased and in-contact swine. It mattered not that this function was at times compulsory, at times discretionary, with the local administrations; the results in either case differed in no important respects, so far as the general extinction of the plague was concerned. The action of these elective bodies naturally conformed each to a local public sentiment; and therefore a stubborn distrust in some districts in the eventual efficacy of a stamping-out system, an aversion in others to burdening the local rates with the charges of compensation and like diverse local prejudices, had the effect of either clandestinely or carelessly keeping up infected centers from which the disease was

constantly escaping into districts where vigorous restrictive measures had given grounds for faith in its extermination. Experience had taught that the power of prohibiting the exposure of swine at any public sale, show, fair, etc., within a district, excepting by license of a local authority, and then only for slaughter within a specified time, was, wherever exercised, exceptionally effective in checking the local spread of the disease; but, in the face of the laxity in the execution of the law in some districts and the apathy in public sentiment regarding the contagion in others, as detailed above, the beneficial results of such action was not often of general permanent value.

The best and clearest exemplification of the extent and manner of the evasion of the law was furnished regularly by the yearly official reports upon the plague. The acknowledged intent of the acts was the immediate slaughter of diseased animals upon discovery, but the large proportion of affected swine returned each year as having died of the disease was inconsistent with a very strict construction of what the law intended. For the first few years the slaughter of diseased swine was compulsory, but later, when the local authorities were allowed discretion in the use of this power, large numbers of swine were invariably returned as having recovered, indicating a popular disinclination to a rigid enforcement of the slaughter clause. Each year, too, swine in greater or less numbers were returned as being affected, but still remaining alive. Thus, by divers lapses in the administration of the laws, infected centers were constantly kept up throughout the Kingdom, and, though the plague varied at different times in intensity and extent, usually owing to the expansion or contraction of local restrictions upon the movements of swine, no headway was permanently gained toward the extirpation of the disease. The following table shows the progress of swine fever in Great Britain during the period from 1879 to November, 1893—the complete period of the local administrative system.

Swine fever in Great Britain from 1879 to 1893.

Year.	Number of counties reported from.	Number of fresh outbreaks.	Number of swine attacked.	Diseased swine.			
				Killed.	Died.	Recovered.	Remaining.
1879	53	2,765	17,074	13,643	3,416	124	99
1880	50	1,936	9,865	7,961	1,940	23	40
1881	56	1,717	7,994	6,217	1,781	24	12
1882	56	2,983	14,763	11,903	2,799	18	55
1883	53	2,400	11,225	8,950	2,257	32	41
1884	54	1,877	8,939	6,364	2,372	153	91
1885	70	7,926	38,798	27,478	9,919	1,375	117
1886	63	6,813	35,029	26,745	7,438	745	218
1887	71	7,238	41,973	24,831	14,502	2,332	526
1888	69	6,449	32,241	16,111	13,288	2,856	512
1889	61	4,841	25,885	13,188	10,782	2,160	267
1890	64	5,076	29,002	12,844	12,785	2,999	731
1891	66	5,595	32,349	15,229	14,112	3,389	340
1892	66	2,745	13,957	7,124	5,789	1,146	238
1893	65	2,377	11,729	5,405	5,020	1,542	.....

These fifteen years' experience with swine fever, though productive of such disappointing results, had nevertheless given rise to a widespread belief that the stamping-out system, if it were properly enforced, would be entirely effective in mastering the plague. Some modifications of the system, as originally devised, had been evolved from this long experience and from a study of the conditions that had

surrounded some cases of temporarily successful local extermination; and it was felt that the theory that had thus been built up needed only to be placed in the hands of an authority capable of putting it into universal execution throughout the Kingdom in order soon to place swine fever in the category of extinct animal diseases. Perfect confidence was now felt that the total extermination of the disease was dependent only on certain processes—first, the early detection of the disease in every existing center; then, the immediate and perfect isolation of all diseased and in-contact swine, and the prompt slaughter of all swine that had been attacked or even exposed to the infection; this to be followed by a thorough cleansing and disinfection of infected premises, and such restrictions upon the movements of all susceptible animals over sufficiently large areas, without regard to district boundaries, as would forestall all possibility of an introduction of the disease into a free locality, or the escape of the infection, either through mediate or direct contagion from infected ones. The consensus of opinion was that these processes could be brought to a successful issue only by the intervention of the central authority; and, on November 1, 1893, the work of exterminating swine fever in Great Britain was intrusted to the board of agriculture, with the recommendation that the disease should be dealt with in the same way that pleuro-pneumonia had been.

The significance of this change in executive policy in relation to the extirpation of infectious and contagious diseases of animals may be better understood by reverting briefly to the limited powers of the central authority under the local administrative system. Then its executive functions were directed principally to two ends: first, to the prevention of the importation of disease from foreign countries, and second, to a general supervision over only such animals within the Kingdom as were not upon the premises or wholly under control of their owners. In relation to the latter class, the jurisdiction was over such animals as were in transit, embracing regulations for and the supervision of the cleansing and disinfection of loading pens, railway cars, etc. It also included the important power of restricting the movements of susceptible animals by prohibiting the holding of fairs and public sales over large areas comprising several or, in cases of extreme prevalence, all the districts of the Kingdom. But such sweeping restrictions upon movement were so irksome to farmers and stock owners, and avowedly so hurtful to trade and individual interests, that this function had been seldom exercised for prolonged periods. It may, therefore, be said that the duties of the central authority in the suppression of swine fever among home animals had during the local administrative system been principally confined to the issuance of orders and to advising the local authorities directly intrusted with the administration and execution of the laws and to making regulations as to their procedure and action generally.

In the adoption of a central administrative system the principal aim was to overcome the lack of uniformity of action that had prevailed among a complexity of independent districts by establishing a single source of administration. Instead of a confederation of over 300 separate areas, each striving to suppress the disease as local prejudices or interests suggested, Great Britain now became a single area subject to one executive authority. And beyond a few preliminary steps in the detection of the disease the responsibility of the entire work of exterminating swine fever was, by the act of 1893, vested in the board of agriculture. A radically changed system of procedure

was therefore adopted. Immediately upon notice of the supposed existence of the affection the inspectors of local authorities are required to place the suspected premises under restrictions, with a view to prevent the disease, if it exists, from escaping.

Such precautionary restrictions had doubtless always been the intent of suppressive legislation, but under the old system many local authorities, either through carelessness or a deference to the wishes of the owners, had failed to impose them until after the true nature of the disease had been determined by a veterinary surgeon, thus often leaving a dangerous center of contagion unguarded for days.

As soon as notice of the suspected case reaches the board a telegram is sent to the local veterinary surgeon nearest the infected premises, instructing him to make immediate examination of the suspected swine. If his investigations justify the opinion that the suspicions of swine fever are unfounded, the restrictions are at once removed. If external appearances indicate the existence of the contagion, he is required to slaughter a diseased animal, as a test case, and forward the whole of the viscera, including the lungs and heart, to the laboratory of the board in London, together with a statement of such pertinent facts as bear upon the history of the animal and the condition of the rest of the swine on the premises.

It is in the examination of these viscera by the board that the cause of one of the greatest difficulties in exterminating swine fever becomes apparent. A perplexity arises in the search for intestinal lesions in determining the existence of this contagious disease, and the English authorities illustrate this difficulty by a comparison between the certain diagnostic reliance placed upon the special lesions of pleuro-pneumonia and the doubtful results often obtained in a search for the visceral lesions of swine fever. In pleuro-pneumonia the existence or nonexistence of the disease is positively determined by an examination of the lungs alone regardless of ante-mortem symptoms in the animal. If the disease exists, the characteristic lesions are always present and only in those organs; if it does not exist, the absence of the special lesions in the lungs is regarded as proof absolute of the animal's freedom from the infection. In swine fever such certain deductions can not always be made from the visceral examinations. In the first place, the difficulty of examination is increased by the fact that the special lesions may be found in any part of the intestinal track between the stomach and the terminal portions of the large intestines, and, though most frequently present in the cæcum and large intestines, there are cases in which they exist only in the stomach or small intestines. And, what is more confusing, in some cases where the living animal has shown the most marked symptoms of swine fever post-mortem examinations have failed to reveal any definite lesions of the intestines, thereby necessitating the slaughter of a second and sometimes a third animal before any satisfactory evidence of the existence of the infection could be detected in a herd. Until 1893 there had also existed a popular, and sometimes a scientific, belief that swine fever attacked both the intestines and the lungs of animals, and in the earlier work of the board this idea was constantly kept in view and increased greatly the difficulty of arriving at a definite decision. But later the conclusion was reached that the only lesions to be relied upon as indicative of the disease are those which are found in the intestinal track, lesions in the lungs not having been discovered to be at all constant. It is evident how much depends upon extreme accuracy on the part of the board in this often perplexing

labor, not only a responsibility for the decision that the disease is of an infectious nature and not due to preventable causes, but also for the imposition of restrictions upon movements of animals, always irksome to the community, and for the serious national outlay of compensation for slaughter.

After the existence of the disease is verified by these visceral examinations, a traveling inspector of the board is sent to the infected premises to arrange for the slaughter of all animals that in the opinion of the board may be desirable for stamping out the infection. Restrictions upon the movements of animals are also imposed over areas discretionary in extent with the judgment of the board. At first, in compliance with a generally expressed wish, these restrictions were confined to the actual places where the disease was discovered and the immediate neighborhood; but, finding that the number of outbreaks increased under this system, the plan was adopted of extending the restrictions over large areas, and in districts most seriously afflicted the congregation of large numbers of store swine in markets, fairs, and sale yards was, as far as possible, prevented, and for a time there was an absolute prohibition of the holding of all store stock sales of these animals throughout the Kingdom. Owing to a general evasion of the restrictions, even this procedure resulted in small success.

To the surprise of those who had been ardent advocates of the board of agriculture undertaking the task of extirpation, the action of that board has been as unproductive of permanent results as that of the local authorities, and many theories have been advanced as to the cause of the failure. The peculiarly obscure character of the disease, which enables it to remain for months undetected in a herd and to baffle all attempts at diagnosis; the uncleanly environments of swine, which furnish conditions favorable for the nurture of the contagion; the opposition or indifference of owners and breeders to the restrictions upon movement, which seem absolutely essential to the eradication of the infection; the usual ownership of this species of animal by persons of small means who evade restrictions which entail upon them not only inconvenience but ill-endurable loss—these and other causes are ascribed for the unabated prevalence of the disease. But in this connection considerable significance may be attached to the fact that, so far as is known, no country in the world where the disease has once obtained a firm hold has been successful in its extermination.

The statistical history of the disease in Great Britain for the last two months of 1893 and the whole of 1894 is as follows:

Year.	Number of counties reported from.	Number of fresh outbreaks.	Swine slaughtered—		Died of swine fever.
			Suspected, but found free from swine fever.	Diseased or having been exposed to infection.	
1893	67	536	93	6,045	1,147
1894	73	5,682	1,320	56,293	7,231

The apparent inefficiency of the stamping-out system and other causes aroused the authorities in the summer of 1894 to a consideration of the advisability of modifying the method of procedure. It was observed that the policy of slaughtering all swine in any way exposed

to the infection as well as those actually diseased had had no appreciable effect in checking the plague; and, plainly, a persistence in this plan foreboded a heavy drain, continuous and undiminishing, upon the national fund for compensation. It was therefore decided that slaughter should be confined only to diseased animals and those more closely exposed to the infection, and that those more remotely associated with the contagion should be subjected to a rigid and prolonged system of isolation. It may at least be said of the trial of this plan that it was not attended by any appreciable increase in the disease; and the number of isolated animals that failed to contract the disease or, having contracted it, recovered indicates the financial advantage both to owners and to the nation of this system of procedure, provided only that experience teaches that it does not keep up infected centers or increase the spread of the disease. The following table shows the number of swine isolated and the number that died during the period of isolation covering the months that the system was in operation in 1894:

Month.	Number of pigs isolated, having been exposed to infection.	Number of pigs died during the month.	Percentage.
August	5,580	114	2.04
September	7,820	108	1.3
October	7,509	46	.61
November	3,973	14	.35
December	3,438	12	.34
Total	28,320	294	.....
Average	5,664	58	1.03

PLEURO-PNEUMONIA.

Because of its fatal and often obscure character and its continued prevalence and stubborn resistance to all methods of extermination, contagious pleuro-pneumonia was naturally one of the first diseases in Great Britain to the extinction of which the stamping-out system was applied. From that time up to the present the two have been constantly and antagonistically associated. And since the system in its incipiency was not a perfect creation, but has rather been a thing of inconstant growth, depending for its perfection upon various changes of operation suggested by experience, the varying prevalence of the disease throughout this period is a perfect indicator of the success that has attended each modification of the slaughtering plan.

Almost thirty years' experience was had with contagious pleuro-pneumonia in the British Isles before the adoption or even conception of the principles of the exterminative system now in force. During the greater part of this long period the disease was popularly regarded as noncontagious; the unaccountable appearance of the affection repeatedly in noninfected districts, where none but apparently healthy cattle had been introduced, long diverted attention from an appreciation of the true infective properties of the disease,and gave rise to easily credible theories that it was spontaneous in its origin or indigenous to the soil.

The first appearance of pleuro-pneumonia in the United Kingdom was in the neighborhood of Cork, Ireland, in 1840. Its origin was obscured in mystery. The disease had existed at various places on

the continent of Europe for about a century, and it was then prevalent in the Netherlands. But the preponderant weight of authority now is that the infection is communicable mainly by immediate contagion; that is, that healthy animals can contract the disease only by direct contact with diseased ones. The circumstance, therefore, which makes the Irish outbreak mystifying is that the importation of all animals into the British Isles was at that time entirely prohibited; and the only possible explanation of the appearance of the disease on that side of the English Channel is the unauthenticated report that some Dutch cows had been introduced into Ireland by the collusion of an Irish dairyman with the consular authorities. Once established, the contagion spread rapidly. In 1842 it was discovered in some London dairies. In 1843 it had reached Scotland; and after that time it continued its ravages among the herds of Great Britain without any period of intermission.

The infectious character of pleuro-pneumonia first attracted national attention in 1865 through the accidental circumstance that the contagion was noticeably checked by the widespread restrictions placed upon the movements of animals on account of cattle plague and foot-and-mouth disease; but the mysterious character of many outbreaks of pleuro-pneumonia had been apparently so irreconcilable with the theory of contagion that the new idea was by no means universally accepted, and an attempt to legislate for the disease on this basis failed.

The mystery which perplexed observers as to the contagious or non-contagious properties of the infection arose, as science afterwards determined, from symptomatic peculiarities. But even after the question of contagion was settled the occult and mysterious still continued to be so striking a characteristic, at least until a recent date, of the ravages of the disease that some knowledge of its symptoms is an essential precedent to a clear understanding of the history of the disease, particularly in respect of its stubborn resistance to methods of extermination. In the acute form of the affection the external signs are so well defined as not to be easily misunderstood, and hence need not be considered in this connection. But the malady in the course of its development often runs into a chronic form which has been a source of great perplexity, and has been responsible in many instances for all that was mysterious in the spread and continuation of the plague. In the early stage an animal may either show no suspicious symptoms until a considerable portion of the affected lung has become implicated; or, in the advanced stage, the disease may run its full course, only affecting that organ and the animal's general condition to such a limited extent that the presence of the infection altogether escapes attention. In these cases the latent period may vary from several days to a few months, and animals which have shown symptoms of illness for only a short time before slaughter are often found upon post-mortem examination to have a large portion of the lung consolidated, an indisputable proof that the affection had existed for some time before it was detected. Moreover, thousands of apparently healthy cattle in infected herds, after having withstood every known test for the infection, when slaughtered solely on account of having been in contact with diseased animals, have shown in the lungs the unmistakable lesions of pleuro-pneumonia. When it is considered that cattle affected in this chronic form are fully as capable of communicating the disease to the healthy of their kind as are those which show the most marked symptoms, it will be readily seen that the apparently healthy cattle, which might live to subserve all the

purposes for which they were bred and reared, are often fraught with more disaster to the aggregate cattle life of a country than are those suffering from the disease in its worst form. The latter are of necessity limited in their spread of the contagion to a narrow area; their sickly appearance debars them from the ordinary course of trade; they are usually isolated from their kind; they generally die or are slaughtered and their power of infection ceases. But the apparently healthy animals which are infected move about, through the hands of unsuspecting purchasers, in all the channels of traffic; they carry the infection into the crowded lairs of the markets and sales yards; they breathe it about them in the loading pens and cars of railways; and, through the medium of all of their kind with which they come in contact, disseminate the dreaded plague through the farms and dairies of a country in a manner mysterious and often untraceable. Nor is this the only obscure and insidious form of the infection. In the later stages of the disease—this occurs in the mild form always, and in the aggravated, also, when the animal withstands the attack and recovers—the diseased portion of the lung becomes separated from the surrounding tissues, and a cyst wall gradually forms around it. The growth of this cyst occupies a considerable period of time, after which an animal, even if affected with an acute attack, may regain all the outward appearances of perfect health. The exact period at which the power to communicate the contagion is lost is usually distant and always undeterminable, and thus so-called recovered animals are often more dangerous factors in herds or in the marts of trade than those plainly suffering from the infection. These symptomatic peculiarities explain many circumstances and events that have seemed obscure and perplexing throughout the entire history of the disease.

In 1869 the English Parliament passed the first contagious diseases of animals act, and pleuro-pneumonia, the insidiously infective nature of which was then better understood, was one of the few uncontrolled plagues specifically mentioned as subject to its provisions. In respect to the extinction of the plague, the act of 1869 was of little avail, for during the nine years' operation of this law the contagion received no material check. But the act is important in that it established in Great Britain the fundamental principle of the eradication of animal diseases by legislative process and led to the organization of a sanitary and police system for the control of contagious affections. By this legislation the number of ports at which foreign animals could be landed was reduced and expressly defined, and veterinary inspectors stationed at each formed a cordon of police around the island to ward off the invasion of disease from abroad. Within the Kingdom a system of internal inspection was formulated with a view to the discovery and isolation of the disease in every existing center, and, aided by penal enactments in the statute, to prevent the exposure of actually diseased cattle in all the operations of business, to prohibit their movement, either by land or water, and to supervise the cleansing and disinfection of infected premises, both public and private. Statistical reports, issued annually, gave detailed results of the varied operations of the system, indicating the futility of some repressive measures and suggesting the wisdom of the extension of others. So little, however, was the obscure and insidious nature of pleuro-pneumonia at that time appreciated that slaughter, the all-important feature of any successful system of extermination, was for the first three years simply a permissive function of the local authorities; and healthy animals which

had been rendered liable to infection through actual contact with diseased ones were allowed perfect freedom of movement through all the channels of trade if they showed no symptoms of disease after twenty-eight days' surveillance. The futility of the first three years' operations of the act, however, led to an order of council in 1873, making the slaughter of diseased animals compulsory, but beyond diminishing the number of recovered animals the order had no marked effect. The power to move exposed animals out of infected premises after twenty-eight days' quarantine was unwittingly a mysteriously active and insidious factor in perpetuating and disseminating the infection, and public aversion in some districts to the hardships imposed by slaughter and by restrictions upon the cattle trade resulted in the concealment of many cases of the contagion and in often disastrous nonenforcement by officers of the local authorities of many requirements of the law. The following tabulated statement gives the history of the disease in its relation to the operations of repressive legislation from the year 1869 to 1878:

Year.	Number of infected counties.	Number of fresh outbreaks.	Number of cattle attacked.	Diseased cattle—		Healthy cattle in contact slaughtered.
				Killed.	Died.	
1870	68	1,508	4,602	1,755	1,276	2,035
1871	68	1,669	5,869	2,207	1,339	1,836
1872	71	2,474	7,983	3,871	1,979	3,245
1873	72	2,711	6,787	5,061	1,028	2,030
1874	71	3,262	7,740	7,434	289	1,485
1875	71	2,492	5,806	5,584	190	1,417
1876	66	2,178	5,253	5,131	114	1,288
1877	70	2,007	5,330	5,223	107	1,353
1878	67	1,721	4,593	4,488	114	1,357

From the above statement it is seen that so far as direct results were concerned the act of 1869 was a failure. Indirectly, however, it was productive of considerable benefit. The stubborn resistance of the plague to repressive measures had had the effect of turning to it more studious and discriminating attention of scientists and acute observers. This resulted in the discovery of some of the peculiar phases of the disease.

The importation of animals from abroad was recognized as a constant and potent factor in keeping up the infection, notwithstanding the rigid inspection of cattle at the ports of landing. Moreover, the continual introduction of convalescent home animals, or of so-called recovered cases that had reached the chronic stage, into the intricacies and complications of the English cattle trade, where cattle are sold and resold and often pass untraceably from the premises of one dealer to those of another several times in the course of a few days, was seen to be an effective barrier to the efficacy of any system of extinction and to make the compensation for the slaughter of actually diseased animals a fruitless expenditure of public funds. In many instances the reckless introduction of fresh cattle into infected premises had plainly added abundant fuel to the flames of infection, and the purchase of cattle from unknown sources was frequently discovered to be the cause of outbreaks.

From these primitive investigations into the insidious nature of the disease it seemed that nothing less than a prolonged blockade of the entire cattle traffic of the country could give relief from the ravages

of the plague. A lack of popular faith, however, in the efficacy of suppressive measures and a consequent feeling of irritation over the losses and inconvenience already imposed by restrictions upon the movement of cattle made far-reaching legislation impossible of attainment. New legislation, therefore, was made only on lines of greater stringency and broader comprehension.

The second contagious diseases of animals act was passed in 1878, and suppressive operations were conducted under its provisions until September 1, 1890. The predominating repressive principle embodied in this act and in the earlier orders of the privy council issued under it was that of isolation; interference with the regular cattle trade of the Kingdom was kept at a minimum. Slaughter, though given a wider scope than in the act of 1869, was still a secondary idea, and the power to exercise it was granted only as an isolated instrument. The peculiar character of pleuro-pneumonia plainly made the isolation of Great Britain from all the rest of the world a matter of prime importance. This was effectually accomplished by a provision of the act vesting the privy council with the power and responsibility of prohibiting the importation of all cattle from countries infected with the disease, except for slaughter at the port of landing. The effectiveness of this measure is apparent; pleuro-pneumonia, being probably communicable by immediate contagion, is the only plague under the provisions of the diseases of animals acts from the importation of which slaughter at the port of landing affords absolute security.

The isolation of the Kingdom was, however, attended by the unfortunate circumstance that the disease was raging within its confines, and the task of isolating each separate case, and thus preventing the infection from spreading to the healthy animals of the country, was undertaken with great vigor. Accurate knowledge of the prevalence of the disease with regard to each existing center was the first requisite to successful operations. To attain this there was put in action a combined system of persuasion and compulsion, in which compensation for slaughtered cattle was an inducement to owners to reveal the existence of the disease, and a fine was the penalty of neglect. When a suspected case had been verified, and certified to by the local authorities, public warning was given by handbills posted conspicuously around the premises. The actually diseased cattle were required to be slaughtered within two days by the officers of the local authorities, and discretionary power was also given by the act to slaughter all cattle which had been exposed to the disease. The introduction of fresh cattle into infected premises was absolutely prohibited, and the removal of those that had been exposed to the infection was not permissible except by license of the local authorities, and then only for slaughter. The period after which cattle could be removed for purposes other than slaughter was made discretionary with the authorities, provided only that it could not take place less than fifty-six days after the last exposure to the infection; and rules for cleansing and disinfecting infected premises were rigidly enforced. Diseased animals which were not on the premises or under direct control of the owner, such as those in transit or those exposed in the operations of public trade, were subject to seizure and slaughter by officers of the privy council. Under this burdensome system of restrictions the cattle industry of England labored for upward of ten years. And though there were many remonstrances against the inconvenience entailed by a rigid enforcement of the measures in some districts, and loud complaints against the laxity and inefficiency of local

officials in others, though murmurs of discontent were often heard against the heavy drain upon the local rates for payment of compensation, nevertheless a popular opinion widely prevailed that the ultimate extinction of the plague could not fail to result from the operations of the system. Costly experience alone taught that regulations much more rigid and burdensome even than these would be ineffectual to eradicate the plague under the political system by which they were administered.

From 1878 to 1885, as will be seen from the table below, the number of infected counties and fresh outbreaks steadily decreased. But it will be observed that no significant decline occurred excepting between the years 1880 and 1884; and the futility of the pleuro-pneumonia restrictions is illustrated by the fact that this decline was due not so much to them as to the extensive restrictions that were, during that period, imposed upon the movements of cattle on account of the existence of foot-and-mouth disease. In 1885 those restrictions were removed, when pleuro-pneumonia steadily increased until 1887, from which time it remained somewhat constant until the passage of the act of 1890.

The perfunctory enforcement by many local authorities of their power to slaughter in-contact cattle which had every appearance of health was always a proceeding which, though often grateful to the individual immediately interested, was loudly deprecated by those who looked to the total extinction of the plague throughout the entire realm, and in 1888 an order was passed making this discretionary power compulsory. From that date not only diseased cattle, but all that had been in any way exposed to the infection, were subject to slaughter. The operations of this order were attended by some surprising results. Healthy cattle were slaughtered, as compared with diseased ones, in a ratio of about 5 to 1, and the consequent expenditures for compensation reached enormous proportions. But, notwithstanding the clearly manifest fact that a contagion communicable only by immediate contact can be extinguished by the destruction of all infected and exposed subjects, this plague in Great Britain for the next two years showed no signs of abatement, but rather the contrary. This unexpected turn of affairs, after twenty years' costly, burdensome, and futile experiments, first called attention to the fact that the causes of failure were not so much to be found in the suppressive measures or the persistent character of the disease as in the local administrative system under which the extinctive regulations were executed..

More than any other contagious disease of animals, pleuro-pneumonia is dependent for its extinction upon a uniform and hence national administration of the suppressive laws of the infected country. Its persistent infectiousness through a long period of incubation and during its obscure and often undeterminable stages frequently makes the discovery of a single case of the disease a subject of national importance, necessitating the tracing of the movements of the affected animal for weeks previous to detection, possibly into many counties and districts, through markets, dairies, and premises of dealers, with the double purpose of finding the source of the infection and stamping out the trail of contagion that has been spread. The local administrative system, under which the acts and orders had up to this time been administered, was not conducive to concerted extinctive action. By it the power of suppression was distributed among a federation of petty and independent local authorities, between whom a diversity of interests and opinions made effective action impossible. To each

was delegated the task of stamping out only so much of the infection as was shut up within the proper boundaries of each separate district, county, or borough, but participation in the extinction of the disease beyond their respective limits was impossible. Cattle known to have been exposed to the disease could be surreptitiously moved from an infected territory where rigid suppressive measures were in force, and thereafter were free from the interference of the local authorities of the district in which the contact occurred. Knowledge might be had by one authority of the exact location of infected cattle in the district of another, and yet, beyond a friendly word of warning, such knowledge might be of no avail. The results of energetic action in some localities was thus constantly neutralized by the inertness displayed in others, and the practical enforcement of the principle of extinguishing a contagion by the destruction of every animal that was infected or had been exposed to the infection failed, not from any fallacy of the principle itself, but from the defects of the system by which it was administered. The following table gives the yearly progress of pleuro-pneumonia in England from 1878 to 1890, the year in which the local administrative system was abandoned:

Year.	Number of infected counties.	Number of fresh outbreaks.	Number of cattle attacked.	Diseased cattle—		Healthy cattle in contact slaughtered.
				Killed.	Died.	
1879	63	1,549	4,414	4,296	119	2,042
1880	51	1,052	2,765	2,681	88	1,389
1881	45	729	1,875	1,797	78	914
1882	46	494	1,200	1,161	39	962
1883	40	349	931	897	35	981
1884	33	312	1,096	1,074	20	751
1885	41	404	1,511	1,469	42	1,167
1886	48	553	2,471	2,409	63	2,446
1887	47	618	2,437	2,384	52	3,817
1888	39	513	1,843	1,786	59	8,722
1889	41	474	1,646	1,603	42	7,297

On the 1st of September, 1890, there was put in operation in Great Britain a system of administering laws for the extirpation of pleuro-pneumonia that was designed to insure absolute uniformity of execution throughout the Kingdom. The power to slaughter diseased and exposed cattle was transferred from the many independent local authorities to a single central authority, and the cost of the execution of the laws and of compensation for slaughtered animals was made defrayable out of the imperial funds instead of from the local rates, as heretofore. And, although the local authorities in all other respects retained their prerogatives in dealing with the disease, the transfer of the power of slaughter and of compensation above referred to made necessary the adoption of a new and central system of administration.

Under the new system the preliminary operations for the detection of the disease in every existing center were carried on as before; but, an outbreak having been discovered, the entire work of extinction now devolved upon the central authority, i. e., board of agriculture. The officers of the local authorities, having been apprised of the existence or suspected existence of the infection, were required to declare the place infected and forthwith report the case to the board of agriculture. Immediately on receipt of this information the board, by telegram, authorized a veterinary surgeon near the infected premises to slaughter the suspected animal and send the lungs to the Royal

Veterinary College for inspection by officers of the board. If the examination revealed the animal to be suffering from disease other than pleuro-pneumonia, instructions were at once forwarded to remove all restrictions that had been imposed on account of the suspected existence of the infection; but if the lungs manifested the characteristic lesions of the contagion, a traveling inspector was sent at once to take charge of the premises. The prescribed duties of this official were, first, to arrange, in concert with the veterinary surgeon, for the slaughter of all the diseased cattle, and second, for the slaughter of all others to which attached any reasonable suspicion of infection, whether on the same or on near or remote premises. The latter duty involved the most searching inquiry as to the source of the outbreak and as to the distribution of all cattle which had been removed from the premises for a long period before the infection was discovered. In many instances the origin of the disease was untraceable or was lost in the assertions of previous dealers that they had no knowledge whence the infected cattle had come into their possession. But, the most remote origin possible once having been attained, all cattle which had since been exposed to the infection through the agencies of transportation, markets, or sales were traced to their destinations and slaughtered, and if any one of them was found to be diseased, the whole herd into which it had been introduced and all cattle with which it had been in contact since the date of probable infection were killed without discrimination. Thus the discovery of a single case of pleuro-pneumonia often led to a wholesale slaughter of cattle, not only on different farms, but on those separated from each other by long distances and situated in different counties.

Other measures of prevention similar to those enforced under the act of 1878 were put into universal and uniform operation throughout the Kingdom, and the work of extirpation was carried on with assiduous zeal. The results are said to have exceeded the most extravagant hopes of the promoters of the central administrative system. The following table illustrates, so far as statistics can do, the operations of the pleuro-pneumonia act of 1890, and discloses some of the causes that have led to an unprecedented decline in the prevalence of the disease:

Year.	Number of infected counties.	Number of fresh outbreaks.	Number of cattle attacked.	Diseased cattle—		Healthy cattle in contact slaughtered.	Cattle slaughtered as suspected, but found free from pleuro-pneumonia.
				Killed.	Died.		
1890	36	465	2,057	2,022	37	11,301	.....
1891	27	192	778	778	.....	9,491	232
1892	10	35	134	134	.....	3,477	188
1893	4	9	30	30	.....	1,157	86
1894	2	2	15	15	.....	391	41

Two facts should be kept in mind in a consideration of the above figures—first, that during 1890 the local administrative system was in force, excepting during the last sixteen weeks of the year, so that for all practical purposes a comparison between the figures for that year and for 1891 is a comparsion between the results of the local and the central administrative system; secondly, that the term "outbreak" was subjected to a change in signification on September 1, 1890; before this it was used to describe the prevalence of the disease upon every

separate farm or set of premises where it appeared, but afterwards it signified all cases known to be of a common origin, though distributed over several farms.

From the above table the efficacy of a uniform enforcement of the stamping-out system is apparent. Under such operations pleuropneumonia decreased from an alarming prevalence in 1890 to a practical nullity in 1894; and though the existence of a few cases of this insidious disease can not be regarded with complacency, yet under a stringent and watchful repressive system there is little danger of its getting beyond control. Millions of dollars have been expended in its extirpation by the nation, and an endurance little short of heroism has been shown by the people in their patient submission to irredeemable losses consequent upon the rigid restrictions placed upon the cattle trade. A costly experience alone has taught that the most economical system of dealing with contagious animal diseases is the one which will most quickly lead to their effectual eradication.

TUBERCULOSIS.

Tuberculosis occupies an anomalous position in the history of contagious animal diseases in Great Britain; it is the only infectious malady of widespread prevalence against which no extinctive measures have been directed by legislation. In view of the fact that it has been known for ages to be extensively disseminated, that it is an unusually constant and unintermitting force in the destruction of life, and that it possesses the quality, uncommon to most diseases, of being intercommunicable among man and the domesticated animals, the causes of the failure to concentrate upon this contagion the exterminative forces embodied in the diseases of animals acts becomes a subject about which are clustered many suggestive facts. Of late years exceptionally intense interest has been attracted to this contagion on account of astonishing discoveries as to its causes and the agencies of its dissemination; but the futility of all efforts to include it in the list to be eradicated by legislation has left us without accurate and valuable statistical knowledge of its prevalence and depredations; and the fact that it is communicable to man has in recent years detracted attention from possible methods of minimizing its prevalence—the usual and natural economic course in the treatment of animal diseases—to the more philanthropic study of the effect of food derived from tuberculous animals upon the health of the human race. Interest in tuberculosis in Great Britain, as manifested by governmental action, has, therefore, been confined principally to two lines of investigation—first, to the possibility of eradicating the disease by the usual legislative processes, and that having been deemed impracticable, next, to the effect of tuberculous food upon human health.

The earlier history of tuberculosis in the United Kingdom contains little that, in the light of later knowledge, is of scientific or historic importance. The disease has prevailed extensively for centuries, and, until within thirty-odd years, the scientific and the popular world had generally accepted the belief, though not without much discussion and contention, that it was an incurable and unavoidable affection, due to hereditary and hygienic or unknowable causes. A complete revolution of sentiment concerning the etiology of the disease was precipitated in 1864, when Villemin announced his discovery that tuberculosis was infectious, and furnished definite proof that inoculation

with tuberculous material produces the disease in the animal experimented upon. Eighteen years later this valuable service to medical science was supplemented by the famed discovery by Koch of the tubercle bacillus, the specific cause of the disease. Upon these two discoveries is based all modern investigations into the pathology of the disease, and from them dates a world-wide awakening of popular interest in scientific investigations as to the causes and possible cures as well as the dangers of tuberculosis that is almost unprecedented in the history of other medical discoveries. In some countries, as the successive results of experiments and investigations were announced, almost a feeling of panic prevailed and fears were aroused that beef and milk were scarcely safe articles of food. The English people, however, resolutely declined to be unnecessarily alarmed, and the history of the disease in that country is free from features of sensationalism.

It was not until 1888 that an organized effort was made to include tuberculosis in the list of contagions scheduled in the diseases of animals acts. The stamping-out system as applied to pleuro-pneumonia was then being vigorously enforced, and a number of the aggrieved dairymen and agriculturists had entered a protest with the privy council against the hardships and losses imposed upon them by the enforcement of the new regulations for the slaughter of in-contact cattle. Their grievances were referred to a departmental committee, and this body was incidentally instructed "to inquire into and report upon the disease known as tuberculosis and advise as to the best means of prevention."

The committee examined a considerable number of witnesses in reference to tuberculosis among the lower animals, and a general opinion was found to prevail that steps should be taken to guard against the consumption of meat and milk of tuberculous animals. The question of curative treatment was summarily disposed of, as it is well known that no cure or antidote is known for this disease. Legislation alone was looked to for a remedy, and the conclusion was reached that legislation directed to the protection of cattle from tuberculosis should also include measures that would prevent its communication to man.

In its final report the committee recommended that legislation for the protection of animals should be made on the lines of prevention and extirpation. It was suggested that preventive operations should be carried on in improvements of the hygienic surroundings of animals, especially in the direction of proper ventilation, pure water supply, adequate disinfection of stalls wherein tuberculous animals have been kept, together with isolation of all suspected cases, precautions against the feeding of the flesh or milk of tuberculous animals to healthy ones, and the careful handling of all substances likely to be sources of mediate contagion. Warning was also given against the breeding from tuberculous stock. As an extirpative measure it was recommended that, in order to insure the gradual extirpation of tuberculosis, it should be included in the contagious diseases (animals) acts for the purpose of certain sections of those acts, so as to provide for the slaughter of and compensation for diseased animals found on the owners' premises; for the seizure and slaughter of diseased animals exposed in fairs, markets, etc., and during transit, and for the seizure and slaughter of diseased foreign animals and those in the same cargo at the port of debarkation.

It is not to be inferred from the apparent incompleteness of this proposed system of extirpation that tuberculosis was believed to be a

disease that would yield readily to extinctive measures. On the contrary, it was recognized that in its eradication human ingenuity was confronted with a problem more difficult of solution than any that had been propounded in the history of veterinary legislation. With this malady it was plain that the established system of eradicating animal diseases, complete and elaborate as it was, must be modified and would be more or less ineffective.

This attempt to apply the stamping-out system to the extinction of tuberculosis was confronted at almost every successive step by insurmountable obstacles. In the first place, it was manifest that the existing provisions for the prevention of the introduction of diseases from abroad were not sufficiently comprehensive; and the almost universal prevalence of this contagion would at least necessitate, as a preliminary preventive step, the enactment of a law to slaughter at the port of debarkation all imported cattle from every country on the face of the earth. As is well known, such an act of Parliament would be by no means distasteful to a large proportion of the English people; but, with that end attained, the application of the system to the extinction of the disease as it existed among home-bred animals seemed a far more hopeless undertaking. Tuberculosis is a disease not only widely prevalent in England, but it is a disease that was then in many cases exceptionally difficult of detection; and many an animal whose fine external appearance had been such as to attract the careful eye of the butcher was found after slaughter to have its organs severely affected with tubercles. Hence to impose upon stockowners the usual penalty for not declaring the existence of a disease, a marked feature of which was that its presence was often undetectable, was plainly so unjust and futile an act that the majority of the committee reported against compulsory declaration, the very corner stone of the stamping-out system. Effective regulations for a rigid plan of detection thus being abandoned, it is manifest that all other suppressive measures of the stamping-out system would be of little avail. Slaughter could be and was recommended of only such animals as showed outward symptoms of this obscure and widely disseminated infection; this might alleviate but it would not extinguish the contagion; and the recommendations for slaughter to this limited extent were based upon the hope of nothing more than an annual reduction of the disease, with a possible chance of permanency through the fact that tuberculosis is less contagious than the other diseases included under the diseases of animals acts. Effective restrictions upon the movements of animals diseased and of those which had been exposed to the disease—measures that had proved remarkably potent in the extinction of other contagious diseases—were of necessity unenforcible in an exterminative system that made slight provision for the detection of the disease. And compensation for slaughtered animals on the customary basis of valuation before infection was provocative of much difference of opinion because of the often obscure lingering nature and eventually emaciating effects of the contagion. Tuberculosis, it is true, bears in the obscurity of its symptoms a striking analogy to pleuro-pneumonia, and viewed from this point its extinction would not seem impossible. But, unlike the latter disease, this infection does not present the single phase of being confined to one species of animals, but prevails to a greater or less extent among all domesticated species; and a still more insurmountable obstacle to its extinction by legislative process is that it is disseminated widely among mankind. Success in the complex work of eradicating the disease

from the entire animal kingdom would therefore leave still dangerous and ineradicable sources of infection from man. A careful study and realization of these characteristics of the disease afterwards turned the tide of scientific investigations in England from possible schemes of eradication to less comprehensive consideration of the effect of food derived from tuberculous animals upon human health; and bovine tuberculosis thereafter became naturally the subject of especial attention. The failure of this attempt to include tuberculosis in the list of diseases to be eradicated by legislation has, however, continued to be a subject of considerable current interest, because the objections then interposed against the adoption of such a course have ever since remained an insurmountable barrier to arguments in favor of suppressive legislation.

The failure to schedule tuberculosis in the diseases of animals acts left the veterinary authorities helpless to deal with the infection. However, the power to prevent traffic in manifestly tuberculous animals and to seize such beasts for slaughter had been granted by the public health act of 1885 to medical officers of health and inspectors of nuisances. In the hands of these latter authorities, it should be noted, the exercise of this power had no reference to the eradication of the disease, but was directed solely to the abatement of nuisances and the protection of human health; and though for several years the provisions of the law were seldom enforced, public anxiety in regard to the effect of tuberculous food upon human life finally reached such a pitch that a rigid exercise of these functions of the sanitary authorities became necessary. As no power to compensate owners for the slaughter of diseased animals or for the seizure of condemned carcasses was granted by the public health act, its strict enforcement entailed heavy losses upon persons in whose possession tuberculous animals or carcasses were found. Carcasses of cattle which had been bought in the open market as healthy and in fine condition were often found on post-mortem examination to be affected with tuberculosis; these were confiscated without compensation to the purchaser, though he had acted throughout in good faith, as had also the seller and all other persons concerned in the transaction. The actions of the sanitary authorities, therefore, soon proved so burdensome that in April, 1890, a deputation of butchers and cattle traders waited upon the board of agriculture and demanded redress for their grievances, avowing that they were powerless under the circumstances to protect themselves and had a fair claim to compensation for losses which were rendered unavoidable for the protection of the public; and maintaining, moreover, that there was no ground for the seizure and destruction of meat which was perfectly healthy to the eye of the meat inspector and presented all the appearance of meat of good quality, merely because some of the internal organs of the animal from which it was taken were affected with tubercles. These and other representations made to the board resulted in the next and, up to the present time, the last governmental action concerning this infection, i. e., the appointment of a royal commission on tuberculosis. The position to which this infection had been relegated in public sentiment in England was indicated by the fact that the instructions to this commission were simply "to inquire and report what is the effect, if any, of food derived from tuberculous animals on human health; and, if prejudicial, what are the circumstances and conditions with regard to the tuberculosis in the animal which produce that effect upon man." No reference was made in the

instructions to possible methods of eradication or prevention, or even to administrative procedures available for reducing the amount of tuberculous material in the food supplied by animals to man. Nor was it the province of the commission to consider any other means by which man can become tuberculous than through the effects of eating meat and drinking milk of tuberculous origin.

The commission met for the first time in July, 1890; and, after a series of preliminary consultations, and of examinations of distinguished pathologists and physicians, but with no pertinent results as to what information was possessed by the medical profession as to manifestations of tuberculous disease, having an origin in food, among the human community, it was decided to institute a series of experimental researches, viz: (1) as to the means of recognizing tuberculosis in animals during life, to be conducted by Prof. J. McFadyean; (2) as to the influence upon lower animals of food of tuberculous origin, by Dr. Sydney Martin; and (3) as to the effects of cooking processes upon food from tuberculous animals, by Dr. Sims Woodhead. Provision was also made for a certain amount of consultation among the three inquirers. Investigations on these lines were prolonged far beyond anticipations, and the final report of the commission was not made until April 3, 1895.

The investigations of the commission as to the means of recognizing tuberculosis in animals during life confirmed in a general way the prevalent belief that the disease may exist extensively in animals that appear, to all tests, to be in perfectly good health. It happened, however, in 1891—almost coincident with the beginning of the work of the commission—that the substance known as "tuberculin" was discovered by Dr. Koch of Berlin. It was commonly known as "Koch's lymph," and attracted world-wide attention as a cure for consumption in man. But, as is well known, its value as an antidote was overestimated and was succeeded by its acceptation, to a greater or less extent in different countries, as a diagnostic for detecting tuberculosis in cattle. In England experiments as to the diagnostic value of this substance have been carried on far less extensively and systematically than in many other lands, and the few isolated tests there made have been chiefly of a somewhat crude description, consisting simply in the application of the test and the slaughter or isolation of reacting animals. This may partially account for the fact that the promising results obtained elsewhere from the use of tuberculin are not confirmed by the experimental trials made by the English Government. Professor McFadyean, in his investigations for the commission, applied the test to a total of 75 animals and records that he received wrong indications from tuberculin, about the presence or absence of tubercle, in 24 of them. Nevertheless he sums up his report by the statement: "I have no hesitation in saying that, taking full account of its imperfections, tuberculin is the most valuable means of diagnosis in tuberculosis that we possess." Force is added to this assertion in the final report of the commission by the statement: "We understand that since the time of Professor McFadyean's experiments, the method of using tuberculin as a test has been much improved, and that it is now regarded as affording more trustworthy indications for diagnosis." Beyond this reference to tuberculin, the researches and inquiries of the commission resulted in no new knowledge as to the means of recognizing tuberculosis in its obscure stages in animals during life.

The primary object of the commission was to learn "the effect of

food derived from tuberculous animals upon human health," and, as this knowledge could obviously not be attained by direct experiments upon human beings, it was evident that the only method of attaining it would be by experimenting upon the lower animals and applying, by analogy, the knowledge thus obtained to the case of man. In this connection the inquiry as to "the influence upon lower animals of food of tuberculous origin" becomes of paramount importance.

Preliminary to a broader exposition, by analogy, of the danger of tuberculous milk and meat to human health, Dr. Martin instituted two series of experiments upon a variety of animals which differed in their usual food material—pigs, guinea pigs, and rabbits. In both series the animals were fed with their usual food; but in the first there was added to this food meat and milk, always uncooked, derived from tuberculous animals—the distinctive feature of the experiment being that no particular examination for actual tubercle was made of the material added except to avoid obvious masses of tubercle. The second series of experiments were carried on under like circumstances and conditions with the distinctive feature that the meat and milk added to the customary food contained tuberculous matter plainly recognized as such and purposely added. It is noteworthy that in the latter experiments the tuberculous matter was sometimes taken from a human source, sometimes from a bovine one, and that each animal received but one dose. The results of both series of experiments were that some of the animals experimented upon became tuberculous; but it was significant that in the second series the proportion that became infected was much the larger, comprising in all but a few cases the entire number experimented upon—thereby not only indicating the element that was dangerous to the healthy animals that had been fed, but also, by comparison with the first experiments, suggesting (and this is the feature of economic importance in the whole investigation) that possibly some meat and milk derived from a manifestly tuberculous animal may be harmless, from the fact that all parts of a diseased animal may not be actually invaded by the tubercle. For present purposes, however, the important fact derived from both these series of experiments is the general one that danger arises to a healthy animal from the introduction into its food of material taken from a tuberculous animal.

The commission did not hesitate to apply to the case of the human subject the evidence thus obtained from a variety of animals that differ widely in their habits of feeding—herbivora, carnivora, omnivora—and to state that, as regards man, any person who takes into his system matter which is capable of giving rise to tuberculosis in the lower animals incurs some risk of acquiring tubercular disease. The fact of prime importance now is that this matter may be found in parts of animals affected by the disease, and that it is known to the naked eye by some well marked though various characters, and microscopically by the discovery of the tubercle bacilli. The disease, it is confidently asserted, is the same in man and in the food animals, and the bacilli of tubercle are transmissible from man to animals and animals to animals, and all but identical in all subjects, are the specific cause of the disease.

The establishment of the fact that "the effect of food from tuberculous animals upon human health" is "prejudicial," and that, too, through the agency of the above-described matter, opens up the practical phase of the commission's investigations, i. e., what parts of a tuberculous animal are liable to contain this tuberculous matter. In

other words, What are "the circumstances determining danger to man from meat and milk from tuberculous animals?" Investigations as to the respective risks to human health from the use of these two food substances, when derived from tuberculous sources, were carried out and reported upon separately; and, from the fact that it is always difficult to make sure of the absence of tuberculous matter from any part of a carcass that shows ocular evidence of tubercle elsewhere, conclusions were arrived at, as in the former experiments, chiefly by analogy. The first part of the commission's report upon this query deals with the "danger from meat."

As a rule tuberculous matter is found principally in those organs of the diseased animal that are removed by the butcher in "dressing" the carcass, most abundantly in the lungs, lymphatic glands, serous membranes, often in the liver, spleen, kidneys, intestines, and other organs; and to the practiced eye it is in these organs easy of detection. But in the tissues that go to form meat, as it is known in a commercial sense—except perhaps occasionally in bone or in some small lymphatic gland embedded in intermuscular fat—tubercle bacilli are seldom found. The recognized possibility, however, of the occasional invasion of tuberculous matter into this common food substance led to a series of experiments to determine, if possible, the circumstances and conditions under which meat from a manifestly tuberculous animal may be an absolutely safe article of human food.

In these experiments the beef of 21 tuberculous cows was used separately; of these 8 were affected with mild tuberculosis; 8 with moderate tuberculosis, and 5 with tuberculosis in the advanced or generalized form. The beef of all these cows failed, under the microscope, to show the presence of tubercle bacilli, excepting in two cases, and in those the quantity of tuberculous matter was small. The beef was then submitted to the more delicate tests of both feeding and inoculating healthy animals with it. The results were, viz, by feeding, no animals became tuberculous from the beef of the cows affected with mild tuberculosis, none from the beef of those moderately tuberculous; but by inoculation one animal contracted the disease from the beef of a mildly tuberculous cow, and three became tuberculous from the beef of the cows affected in the moderate form. The beef of 4 out of the 5 cows affected with generalized tuberculosis produced the disease, either by inoculation or by feeding, in the animals experimented upon, 1 only out of the 4 appearing to answer to both tests. It was a peculiar fact that the beef of the cows affected with generalized tuberculosis had, under the microscope, shown no traces of tubercle bacilli; but the beef of the 2 cows that had given affirmative results to the microscopical test came from the group of cows affected with moderate tuberculosis and had given no result in the feeding test; but beef from 1 of the 2 had produced the disease by inoculation.

Manifestly these were unexpected results. It was not expected that tuberculous disease would be repeatedly produced by the use of material that had shown no evidence of tubercle under the microscope. It was expected, on the other hand, that the disease would more uniformly originate from the use of beef that had shown visible evidence of the characteristic bacilli, though lack of uniformity might be accounted for in the present experiments by the small amount of tubercle discovered in the beef of the two cows that had given affirmative results to the microscopical test; and more unexpected still were the "egregious irregularities" observed in the feeding and inoculation experiments.

Even the hypothesis that some minute tubercles might have been overlooked in the small portions of meat that had been used in the feeding and inoculation tests would not account for all the irregularities or all the observed facts; and though this hypothesis could not be wholly disregarded as being a possible cause of some of the unexpected affirmative results obtained by the feeding and inoculation experiments, it was of the utmost importance for practical purposes that some more obvious and probable cause should be discovered for the irreconcilable results.

Attention was now directed to features of the present method of experimenting that had heretofore not entered into consideration, namely, the operations of the slaughterer and butcher, and it was readily seen that a real and considerable danger had arisen to the beef from the probability of its contamination from the actual tuberculous lesions, present in other parts of the carcasses, and conveyed from thence to the proper meat substances by the hands, knives, and clothes of the butcher during the process of flaying and dressing. Accepting this as the method of contamination, the irregularity of the results of the above experiments are easily explained. From this point of view the danger of contamination is obviously dependent upon the quantity of tuberculous matter in the general carcass. In cases of mild tuberculosis, where the quantity of such matter is small, there is little danger to the meat of contamination in the course of the butchering operations; in cases of moderate tuberculosis, where chiefly the lungs and the lymphatic glands in the thorax are affected and may be easily removed without the incision of a tuberculous lesion, the danger, though comparatively greater, is still small; but in cases of generalized tuberculosis, where many parts of the body are affected with the disease, the danger of contamination would proportionately increase. Apparently this theory explained the irregularity of the results of the feeding and inoculation experiments, and Dr. Martin was driven to the conclusion that "when meat is infective it commonly acquires its properties by being accidentally contaminated with tuberculous material during its removal from the carcass." The commission accepted this view to the extent of stating in the final summary of the results of its investigations that "there is reason to believe that tuberculous matter, when present in meat sold to the public, is more commonly due to the contamination of the surface of the meat with material derived from other diseased parts than to the disease of the meat itself."

Investigations into the circumstances determining danger to man from the use of milk from tuberculous cows were of a less complex nature, and the conclusions arrived at were consequently more accurate and reliable. Without going into the details of the careful and thorough experiments made by the commission, it will be sufficient to outline the practical results obtained. To quote from the report of the commission—

> According to our experience the condition required for insuring to the milk of tuberculous cows the ability to produce tuberculosis in the consumers of their milk is tuberculous disease of the cow affecting the udder. This affection of the udder is not peculiar to tuberculosis in an advanced stage, but may be found also in mild cases. * * * The milk of cows with tuberculosis of the udder possesses a virulence which can only be described as extraordinary. All the animals inoculated showed tuberculosis in its most rapid form. * * * The withdrawal from the dairies of every cow that had any disease whatever of her udder would form some approach to security against the serious danger incurred by man from the use of tuberculous milk, but it would not be an adequate security. * * * It is of

supreme importance to the consumers of milk that the existence of any tuberculous disease of the udder should be ascertained without delay. Now, there is no difficulty whatever about recognizing the presence of some abnormal condition in a cow's udder, and the presence of such a condition—whatever it be—demands that the judgment of a responsible expert should forthwith be obtained about its nature. * * * If the expert finds tubercle bacilli in the milk, the cow has dangerous tuberculosis of the udder. If he does not find them, he may apply the further test of inoculating some susceptible animals with the milk and thereby learn the nature of the udder disease. By this test he will rarely be misled. Obviously the cow must be in seclusion, and every particle of her milk must be treated as highly dangerous during any delay that can be permitted for diagnostic purposes, and until the disease has been proved not to be tuberculosis.

The third and last inquiry of the commission was "as to the effects of cooking processes upon food from tuberculous animals." As to meat, investigations were conducted by subjecting pieces into which had been injected tuberculous matter or upon which such matter had been smeared, to various degrees of heat for various lengths of time and then testing its power of infection by processes of feeding and inoculation. The ordinary methods of cooking, such as boiling and roasting, were used in the experiments and the conclusion arrived at was that "ordinary processes of cooking applied to meat which has got contaminated on its surface are probably sufficient to destroy the harmful quality. They would not avail to render wholesome any piece of meat that contained tuberculous matter in its deeper parts."

The effect of cooking upon tuberculous milk was considered an inquiry of special importance, not solely because it is a food extensively used in cookery, but more especially because in a raw state it is habitually drunk by the English people, and the central idea of Dr. Woodhead's report is "to advocate the boiling of all cow's milk that can by any chance contain tuberculous material." Being a liquid substance, it is more readily affected by heat than is the solid tissue of meat; and Dr. Woodhead says:

> The most deadly tubercular material can be rendered absolutely innocuous, so far as any spreading infective disease is concerned, by the action of a temperature at which water boils. * * * A lower temperature than this is sufficient to bring about the same results when allowed to act for a longer time, but for the present it is sufficient to state that boiling, for an instant even, renders the tubercle bacillus absolutely innocuous.

Owing to the objectionable flavor imparted to milk by boiling, investigations were made as to methods of "sterilization" that might be effective and yet be free from this objection; but nothing was found that would take the place of the "simple expedient of putting every suspected milk over the fire and taking it off when it boils."

FOOT-AND-MOUTH DISEASE.

The most notable event in the history of animal diseases in Great Britain in the past few years was the unexpected appearance of foot-and-mouth disease in February, 1892. Particular interest attaches to this outbreak, because England, after having suffered inestimable losses from this malady for almost half a century, had been officially predeclared free from the affection since 1886, and the most careful precautions had subsequently been taken to prevent its reintroduction. These went so far that no vessel was allowed to land susceptible animals in Great Britain which on its voyage had even touched at any port of a country suspected of being infected with foot-and-mouth complaint.

A characteristic feature of this disease, however, is its insidious

infectiousness; the source of its origin and the means by which it is so rapidly spread over the country are often impossible to determine. It seems to be beyond question that the disease first appeared in England in 1839, and at that period—not from sanitary measures but from a principle of agricultural protection—the landing of all foreign animals from any source whatsoever was absolutely prohibited. Under these conditions the introduction of the disease from other countries seemed even more impossible than under the less sweeping restrictions of present laws. Yet it seems to be fairly well established that the outbreak of 1839 was of foreign origin. The fact that foot-and-mouth disease was then prevalent on the neighboring coasts of France and in the Netherlands led to a vague belief that the infection had in some way been conveyed from one of those countries. Though no direct means of contagion was ever discovered, veterinary science has since added some confirmation to this belief by the discovery that, though the life of the virus of foot-and-mouth disease is under ordinary circumstances a short one, yet the infective matter can, under favorable conditions, be carried considerable distances by either animals, persons, or substances which have been in contact with animals suffering from the complaint. Mediate, as well as direct, contagion is a characteristic of the disease.

A clearer idea of the dread which was caused by the reappearance of the malady in England in 1892 may be had by a glance at that nation's past experience with the malady. In a brief review it may be of interest to note that the history of the disease naturally is divided into two eras, the line of demarcation being the contagious diseases (animals) act of 1878. The first era extends from the first appearance of the disease down to the outbreak of 1880, during which time the natural course of the disease was virtually uninterrupted by restrictive measures; the second era dates from 1880 to the present time, and shows the course of the disease as modified and finally contrblled by the sanitary and restrictive legislation resulting from the experiments and investigations of veterinary science.

Immediately after the unaccountable appearance of the disease in the Smithfield market in September, 1839, above referred to, there occurred a rapid succession of outbreaks, almost incredible even in a disease of so short a period of incubation as this, at various points in England, Ireland, and Scotland. Little was then known of the potency either of legislative action or of concerted individual effort in checking contagious diseases; and for a period of three years the contagion ran an uninterrupted course. Though seldom fatal, the disease was of extreme virulence. Comparatively few animals within its range escaped infection, and the losses inflicted upon the cattle, sheep, and swine industry of the United Kingdom were incalculable. It was no uncommon thing for large numbers of animals to lose their hoofs while standing in the markets, and it is a matter of record that basketfuls of the hoofs of sheep and swine—the feet of this species of animals being usually most affected—were swept up daily at Smithfield after the market was over. The disease, no means of checking which were in those days thought of, declined, apparently of its own accord, in 1842, and little was heard of it for the next two years. In 1845, however, with an apparent spontaneity little less surprising or unaccountable than in the first outbreak, it appeared again, and, spreading with like rapidity, ran an unchecked course of about three years, after which it gradually declined and remained in a quiescent state during 1848 and a part of 1849. In succeeding years like

outbreaks, but of varying virulence, occurred at regular intervals; and some idea of the anxiety with which each recurrence of the malady came to be expected may be formed from the mere statement that in the outbreak which began in 1869 it was estimated that not far short of 3,000,000 animals suffered from the affection.

As early as 1849 it was recognized that the plague was probably periodic in character, and this belief was fully confirmed by the next thirty years' experience with the disease. From that date down to the outbreak of 1880 there were seven clearly defined periods of prevalence, each followed by a period of dormancy or subsidence. It should be kept in mind that during the outbreak of 1880 and subsequently the natural course of the disease was diverted by repressive legislation, particularly by the contagious diseases (animals) act of 1878. But the natural law of the infection, when uninterrupted by restrictions, seems to have been that each outbreak or period of excessive prevalence had an average duration of something over two years and the period of quiescence has usually continued about a year and a half. Thus every outbreak has been disconnected from the succeeding one by an interval of rest, during which the affection has remained in a dormant state, never ceasing entirely, but attracting little attention. Notwithstanding this recognized endemic character of the disease, it became the custom of the English people in later years, when the laws prohibiting the importation of foreign cattle had been somewhat modified, to attribute each fresh outbreak to the reintroduction of the infection from abroad; but it is evident to the impartial observer that the regular periodic nature of the disease can not be accounted for on a theory founded on the importation of diseased animals at irregular intervals from foreign countries. A more reasonable explanation of its periodic appearance is that during periods of prevalence, when the disease was allowed to run its natural course, it attacked all susceptible animals within its range, and, having then exhausted its virulence, it remained in a dormant state until a new generation was produced susceptible to infection. Regular periodicity was a marked peculiarity of the disease so long as its natural course was undisturbed by legislative and sanitary restrictions.

The outbreak of 1880 was the first that showed any marked variation from this natural law. During the first nine months of the year Great Britain, for the first time in forty years, was declared free from foot-and-mouth disease. Why this may not have been merely a period of subsidence is not entirely apparent. The malady had made its periodic appearance in 1877, prevailed through 1878, and was declining in 1879; and, reasoning from the past history of the disease, the quiescent period was now due. There were circumstances, however, that made a declaration of freedom from the disease at this date bear more weight than a like statement would have borne at any time since 1839. The contagious diseases (animals) act, by which animals afflicted with foot-and-mouth disease were for the first time placed under severe repressive measures, had made it compulsory upon stock owners to declare the existence of the disease, and had put in operation a system of rigid governmental inspection. Thus the existence of the disease was not so likely as heretofore to be concealed or to escape attention. And, excepting that this was the time for the natural periodic recurrence of the disease, other circumstances indicate that this outbreak was of foreign origin.

The disease was first discovered in the tongues of some French cattle that had been killed in the foreign cattle market at Deptford. No

symptoms of the disease had been noticed in the animals when alive, but the post-mortem examination left no room for doubt as to the character of the affliction. Soon afterwards the inspection of a cargo of cattle from Havre on the second day after their landing at Deptford revealed the infection among some of them. Thus the disease was presumably introduced into the Deptford market.

Taking into consideration the peculiarly infective nature of this disease, no conditions could have been found more favorable to its spread than those of the foreign cattle market at Deptford. The original design in planning this market had been to have it of such ample proportions that the whole continental import trade could, if desired, be converged to this one point. The magnitude which this trade would assume had, however, been underestimated and American imports had not even been calculated upon. Now when the disease was communicated to this market the lairs were overcrowded with cattle from America as well as Europe, and the infection was rapidly carried over the whole area, not only by cattle passing day and night from the lairs to the slaughterhouses, but also by employees in the course of their duties carrying the infection from one point to another. The overcrowded state of the market also prevented effectual disinfection of the places known to have been infected by diseased animals, and consequently perfectly healthy ones became infected soon after entering the lairs. All the powers vested in the veterinary department were brought into play to prevent the spread of the disease beyond the limits of the market, and it is certain that the subsequent widespread contagion was not due to any animal having been allowed to pass from the place.

Within a fortnight after the discovery of the disease at Deptford outbreaks occurred in London, in Luton, Bedfordshire, and in Charlton, Greenwich, and Woolwich in rapid succession. No connection could ever be traced between any one of these outbreaks and the presumedly direct center of the disease at Deptford, excepting that a possibility of mediate contagion was found in the visit to the city dairy of London of a butcher who had premises in the infected Deptford market. From these newly established centers, moreover, the disease spread rapidly with its characteristic insidiousness; and between September 20, when it was first discovered at Deptford, and the end of the following December, there were 1,461 outbreaks in England, and 20,918 cattle, 9,572 sheep, and 1,886 swine were attacked. It was manifest that the provisions of the contagious diseases act of 1878, as thus far enforced, were ineffectual to control the disease.

The act of 1878 is of particular importance in the history of animal diseases in Great Britain because upon it, and the subsequent orders of the privy council under it, is founded the intricate system of police and sanitary regulations governing contagious diseases that has been developed there from that date up to the present time. The single object of the act is the prevention of contagious diseases among animals in the United Kingdom, and the design was to effect this in two ways—first, by preventing the importation of diseased animals from foreign countries, and, second, by the prevention of contagious diseases of animals within the Kingdom. To effect the first, the landing of foreign cattle is restricted to foreign animal wharves (now seventeen in number), and all imported stock is required to be slaughtered upon landing, excepting that there is delegated to the privy council the power of exempting from the latter provision animals from any country

where the laws relating to the importation and exportation of animals and to the prevention and introduction or spreading of disease and the general sanitary condition of the animals therein are such as to afford reasonable security against the importation therefrom of diseased animals. There is also granted to the privy council the power of prohibiting even the landing of animals from countries where certain diseases are known to exist. This latter provision was originally intended only to prevent the importation of cattle plague; but foot-and-mouth disease, on account of its singular infectiousness, was in later years included in the prohibition.

The system for the prevention of contagious diseases among home animals, as created by this act, was an intricate one, requiring for complete success a conformity of action between the stock owners, the local authorities, and the privy council that experience taught to be difficult to attain. The act imposed upon the stock owner compulsory declaration of the disease. When this requirement had been complied with, or the existence of the disease had been otherwise detected by officers appointed for that purpose, it devolved upon the local authorities, aided by a qualified veterinary inspector, to make an examination, and, if the reported existence of a contagious disease under the act was verified, to declare the place infected and to prescribe regulations for the movement of animals in, into, and out of the place. This provision of the act was so plain as to leave no doubt that it was intended that nearly all action necessary for arresting the spreading of a disease should be taken by the local authority in whose jurisdiction an outbreak occurred. The power of the local authorities, however, was limited to their own territories, and a declaration of an infected place could not extend into the territory of a neighboring local authority without the written consent of the latter. The consequent danger of a lack of cohesion on the part of the local authorities in the exercise of the powers granted them was sought to be guarded against by a grant of supplementary powers to the privy council. After the declaration of an infected place it was required that the local authorities report the facts in the case to the privy council with a recommendation, if they deemed it advisable, that the council declare an infected area of specified limits around the infected place wherein the usual restrictions upon the movements of animals should be enforced. Jurisdiction was also given to the privy council over all animals not on the premises of their owners, such as those at fairs, sales, exhibitions, and in transit, with a general supervision over the cleansing and disinfection of market places, sales yards, and of the loading pens and trucks of railway companies. The permanent idea of the system, however, was the detection of the disease on the farms and premises where animals were fed and bred and the concentration of restrictive measures on these centers of disease, leaving the movements of animals outside the limits of infected places as free from interference as circumstances would permit.

The inefficiency of this system for controlling so infectious a malady as foot-and-mouth disease seemed apparent in its application to the outbreaks of 1880, and was very discouraging. Consequently during the following year the devising of means by which the provisions of the act could be so enforced as to eradicate the prevailing malady occupied much of the attention of the veterinary department. It was evident that the weakness of the system was due to the failure of the local authorities to realize their responsibilities. The signal success

achieved in particular districts by the energetic action of some authorities proved the efficacy, theoretically, of the system, but the negligence and laxity of other authorities neutralized all practical benefits as far as they related to the general good. It was soon manifest that the policy of concentrating restrictions on the centers of disease, as thus far tried, was a failure; and the privy council, whose powers were originally intended to be only supplementary to those of the local authorities, now took the initiative in an attempt to suppress the infection. From the council's action is learned the principal object lesson of the year in the control of the disease. An order was issued prohibiting the holding of fairs and markets throughout England during a period beginning January 17 and ending March 31, 1881. The efficacy of such an additional restriction upon the movement of animals was at once apparent in a marked subsidence of the infection; from 240 reported outbreaks the week before the order went into effect the number dwindled to 25 in the week, when it ceased. But the remonstrances of farmers and stock owners against such an avowed infringement upon their rights and privileges made it impossible to continue the order in effect beyond the first month of spring, and the value of the restrictions immediately became apparent in an increase of the disease. In April the number of outbreaks increased to 212, only to be followed by an increase of 513 in May. During the remainder of the year the disease continued to spread with a rapidity characteristic of former accessions, but undoubtedly restricted in respect of the number of animals attacked by the enforcement of the suppressive measures of the contagious diseases act; 4,883 outbreaks were reported during the year and 183,046 animals were afflicted.

In compliance with the natural law of periodicity observed in foot-and-mouth disease in England during the past forty years, it was expected that, regardless of the effects of repressive measures, the malady would reach its highest state of prevalence and a period of subsidence begin about the middle of 1882. It was therefore a source of surprise and perplexity to find that the disease, after decreasing satisfactorily in 1882, suddenly increased and attained its highest maximum in 1883. In seeking the cause of this unexpected accession it was common to attribute it to the importation of the disease from foreign countries. But it is more than likely that the restrictions which had been enforced during the previous three years had prevented large numbers of animals from showing their liability to the disease at once, and that these now yielded to the contagion either through a laxity of restrictions or unusually favorable conditions for the spread of the disease. This fresh accession of the disease led to renewed restrictive efforts—perfect isolation and effectual disinfection—and after running a further virulent course of two years the malady ceased to exist altogether in 1886. The beneficial effects of the repressive measures used is attested by the decrease as compared with former outbreaks in the number of animals attacked, the whole number infected between 1880 and 1884 being less than three-quarters of a million. After almost half a century of prevalence there followed six years of freedom from the disease—an interim during which there arose in England a feeling of security against further attacks. The most sedulous care, moreover, was taken to prevent a reintroduction of the infection from abroad, and, on account of the wide prevalence of the disease on the Continent, an embargo was placed upon the shipment of animals from almost every country

in Europe. An incident occurred in 1892, however, that gave rise to an opinion in England that nothing short of an absolute prohibition of the importation of all foreign animals would insure nonliability to the recurrence of the disease.

Denmark was one of the few countries of Europe in favor of which the privy council had exercised its prerogative of exempting animals from slaughter at the port of landing. On January 27, 1892, sixty-eight cattle from North Jutland were shipped to England from the port Esbjerg, arriving at Harwich on January 30. After the usual period of detention these animals were sent to the Metropolitan market at Islington and exposed for sale on February 1. All but eleven were sold, and, as the next market was not held till the following Thursday, these were returned to the lairs to await that sale. Before the opening of the market on Thursday foot-and-mouth disease was discovered among these unsold animals, and they were immediately removed to the adjoining slaughterhouses and killed.

A careful inquiry was at once instituted into the movements of the entire 68 head from the time they left North Jutland until they reached the metropolitan market, as well as into the subsequent movements of those that were sold, the object being both to discover the source of the disease and to adopt preventive measures against its spread throughout England.

As to the origin of the disease, it was found to be enveloped in obscurity. Denmark was proved to be free from the affection, and the theory that the cattle were infected by mediate contagion just before being shipped from Esbjerg was incapable of direct proof. Even the circumstantial evidence in support of that theory was very limited. At Hamburg, about 80 miles distant from Esbjerg, foot-and-mouth disease was rife at the time, and dealers from the former place were in the habit of attending the Esbjerg market, for communication existed between the two points, and, though German cattle were excluded from Denmark, Danish cattle were shipped to Germany, and the railway trucks used for that purpose were returned in the ordinary course of trade. These were the only links in the chain of evidence in favor of mediate contagion. In fact, no positive proof existed that the Danish cattle were at all responsible for the introduction of the infection; but the disease was then extinct in England, and the fact that its spread in the Kingdom was afterwards plainly traced to cattle sold in the metropolitan market on February 1 makes it probable that the infected Danish cattle were suffering from the disease at that date and were the source of the contagion.

The most stringent measures were at once taken to prevent the spread of the infection. It happened that on account of regulations aimed at pleuro-pneumonia no cattle could be moved out of the county of London at that time without a license, and this circumstance greatly aided the authorities in tracing animals from the metropolitan market that were likely to spread the disease. Affected animals were almost immediately traced into the county of Kent and slaughtered; but, before the end of February, outbreaks had occurred in rapid succession at various places, the range of infection extending as far as Glasgow and Edinburgh in Scotland. During the following few months the calamity of a general spread of the scourge was fearfully anticipated, but the repressive measures were so effective that by the middle of June the course of the disease was stayed. Isolated outbreaks have since occurred, the last one being in 1894, but no danger has been felt from these that the disease would again get

beyond control. During the prevalence of the disease in 1892, 75 outbreaks occurred in England and 20 in Scotland. The total number of animals attacked amounted to 5,267, of which 4,530 recovered, 586 were slaughtered, and 151 died.

A feeling of disquietude naturally prevails among stock owners so long as a single case of this remarkably contagious and insidious disease remains in Great Britain. The peculiarly rapid disseminative power of the virus, conveyable from one place to another through the medium of almost any substance that it touches, makes the presence of a single case no trifling menace to the entire stock interests of the Kingdom. Unlike most other contagions scheduled in the diseases of animals acts, the slaughter of diseased and in-contact animals can not be relied upon to exterminate the infection, since the virus of foot-and-mouth disease is communicable through the clothing and shoes of cattle attendants, the litter of stables, and all substances with which the diseased beast has been in contact. Isolation of infected animals until death or, as usually happens, until recovery, with subsequent disinfection or destruction of all material liable to contain the virus, has, as a general rule, been relied upon for the suppression of the malady. In the case of this disease the avenues of dissemination are so manifold that an outbreak in any locality naturally arouses grave fears as to the possibility of its having been spread over the entire neighborhood through the medium of the common everyday operations of life around the seat of the infection. It was therefore considered a matter of more than ordinary importance when, during the year 1893, two outbreaks of the disease gave evidence that the virus of foot-and-mouth disease still prevailed on British soil. One of these outbreaks occurred in London in such dangerous proximity to the metropolitan cattle market that the risk of the disease being carried thither by mediate contagion seemed alarmingly imminent, and the whole of the cattle on the premises were slaughtered without delay, the litter and fodder on the premises destroyed, and the shed in which the cattle had been confined subjected to thorough cleansing and disinfection. No trace of the origin was found, but as no fresh cattle had been introduced into the premises it was certain that the disease was the result of mediate contagion. The preventive measures were so effective that no other cases of the disease appeared in London during that year.

The second outbreak in 1893 occurred in East Sussex. The surroundings of the infected animals were such as to render isolation easy. The disease ran through the whole of the cattle on the farm, but all finally recovered. No origin could be found for this outbreak other than the suppositive one of mediate contagion.

Evidence that the suppressive measures had not yet been wholly effective was again abundant during the year 1894. Thirteen cases of the existence or supposed existence of the disease were reported to the department in that year, but, as many diseases are easily mistaken by inexperienced persons for foot-and-mouth disease, only three of the thirteen reported outbreaks were found upon investigation to be true cases of the infection. Of these, one outbreak was in a herd of cattle, the other two among flocks of sheep. In the case of the cattle and one flock of the sheep isolation was practiced and followed by success. But, as the third outbreak was in two flocks of sheep in the immediate district where the disease had given so much trouble in 1892, and as the disease was of a particularly aggravated nature, affecting both the feet and mouths of the animals, the slaughter of the entire flocks was

ordered. Careful inspection of stock on the neighboring farms for a fortnight after the slaughter failed to reveal any new centers of the disease.

After over half a century's experience with foot-and-mouth disease Great Britain may be said, though not entirely free from the infection, to have it under a control far more complete than that of any country that has been so extensively afflicted. With an admirable system of laws for the prevention of the importation of the disease from foreign lands and for the suppression of the disease within the boundaries of the Kingdom, it would seem that the nation is not likely to be again subjected to serious ravages from this disease.

ANTHRAX.

Anthrax is an incurable and extremely fatal disease, of unknown origin, that has undoubtedly prevailed in Great Britain from a very remote period; but until the comparatively recent discovery of the specific cause of the malady it did not attract national attention, interest being usually confined to the scattered and separate localities where losses occurred. Recognizedly sporadic in its nature, it was never productive of the widespread fear of general dissemination that was always provoked by an outbreak of cattle plague or pleuropneumonia. Usually there was no premonition of the existence of the disease; the first indication of its presence was sudden death. Sometimes only a single animal of a herd was attacked, and sometimes a number of them were stricken down as by some invisible power in an incredibly short space of time. The remainder of the herd would, as a rule, altogether escape the deadly stroke of the infection, and the disease would seem to disappear as suddenly as it came. Months, perhaps years after, other animals grazing in the fields where deaths had occurred would now and then, singly or in groups, be stricken down in a like unaccountable manner, and thus give evidence of the permanent local existence of the infection. In earlier times, before science had thrown light upon the pathology of the disease, it was but natural that many supernatural causes should have been ascribed to it and many superstitious beliefs associated with it. These naturally had a tendency to deflect the attention of even the more acute observers from the less marked characteristics of the malady; hence the occasional premonitory symptoms of its attacks, as well as the fact that it is not in every case fatal, did not attract the attention that such symptoms might otherwise have done. The disease was popularly regarded as of sudden and fatal effect, and the name that was formerly applied to it is suggestive of this characteristic—murrain, a derivative from the Latin word meaning death.

Before the specific cause of anthrax was discovered there had likewise long prevailed in Great Britain a sporadic disease of man then known as "the wool-sorters' disease." For many years occasional cases of sudden death among healthy men employed in woolen factories, particularly in carding and sorting wool, had attracted attention. Investigations revealed that in some cases death appeared to be the result of direct inoculation of some poisonous material into the body, for a form of malignant pustule was observed under the skin, and the disorder was looked upon as a form of malignant carbuncle. But in other cases there were no external manifestations, and symptoms of

blood poisoning, often proving rapidly fatal, suggested other channels for the introduction of the disease.

The discovery of the specific cause of anthrax and the subsequent investigations which resulted in the establishment of the identity of this animal disease with the human ailment, "wool-sorters' disease," form a chapter of inexhaustible interest in medical science.

In 1855 Pollender, in his investigations into anthrax, was the first to discover minute organisms in the blood tissue of deceased animals—peculiar rod-like bodies which are now familiar to the scientific world by the name bacilli, and this particular variety as the bacillus anthracis. And, though neither Pollender nor Brauell, who followed him in the investigations, attached to these organisms the significance which they now possess as active agents in the cause of the disease, yet the mere discovery of these micro-organisms is a notable event in scientific research, since it is the foundation of the science of bacteriology and led to a complete revolution in the theory and practice of medicine.

The discovery of the bacillus anthracis was followed by exhaustive investigations into the disease both in Great Britain and on the Continent, and during the next thirty years a large amount of valuable and interesting information was accumulated. The fact of primary importance established is that the bacillus anthracis is the cause of the disease, and when introduced into the blood of an animal produces changes which ultimately result in sudden death. The disease is, therefore, infectious, and because of its remarkable fatality would, excepting for certain conditions upon which depend the vitality of the bacilli and their transmission from one animal to another, be the most destructive of all animal diseases to life. The bacillus anthracis is a microscopical organism which lives and grows only in the presence of oxygen and requires a temperature of not less than 70° F. Under these conditions, which prevail over a large part of the earth's surface, these micro-organisms are capable of indefinite multiplication in two ways. First, in the blood of the living animal, where the bacilli are supplied with oxygen by the animal's respiration, they increase rapidly by becoming elongated and then dividing in two, each organism continuing this operation indefinitely until death occurs. When the animal dies, if the carcass remains intact and no blood escapes therefrom, the bacilli, deprived of oxygen, soon lose their vitality and become incapable of spreading the infection. As decomposition goes on the putrefactive bacteria, too, the natural enemy of the bacilli, aid in their rapid destruction. Second, when in the convulsive throes of death, blood, as is usually the case, escapes from the animal, or soon after death this fluid is exposed to the air in attempts to make some salvage from the hide or flesh of the dead beasts, the bacilli, then under conditions unfavorable to increase by multiplication, increase in a different way. Spores form within the bacilli, and these bodies, which may be compared to the seeds of higher plants, are remarkably resistant to the influence of heat and drying and retain their vitality for years. The soil and substances of any kind whatever upon which the blood has fallen, therefore, become contaminated, and henceforth permanent sources of danger to any animal whose system presents favorable receptive conditions.

The means by which either the bacillus anthracis or its spores can gain access to the animal system are peculiar. An animal can contract the disease only by the introduction of these organisms into its

blood, and, unlike the more highly infectious diseases, the only channel through which conveyance can take place is a wound or an abraded surface of the skin, or of the mucous membranes of the mouth, nostrils, throat, or intestines. A diseased animal, therefore, can communicate the infection directly to a healthy one only through the medium of blood, which may have escaped from it before or immediately after death, coming in contact with some abraded surface of the healthy animal's body. Numerous experiments have proved that the organism is harmless to healthy animals so long as the skin and mucous membranes are free from the minutest wounds, and that infection can not be produced by smearing the skin with anthrax blood or by taking it into the digestive system, so long as the surface in both cases is unabraded. Evidently insusceptibility to infection, therefore, depends upon the absolute freedom of an animal from all abrasions, a condition which, unfortunately, is by no means common; and since all the animals of the farm are liable to the disease, the danger of infection is greatly increased by the fact that everything that comes onto the farm, whether of home or foreign origin, water, food, litter, manure, etc., is capable of bringing the spores of the infection. Moreover, the not uncommon habit of ignorantly feeding carcasses of animals dead from anthrax to living ones, especially to swine, or of carelessly disposing of them without due regard to the danger of infecting articles which come in contact with the virus has long been known a potent factor in perpetuating the disease.

In 1880 there occurred an extensive outbreak of "wool-sorters' disease" among wool sorters in the midland counties of England. A scientific investigation was made which resulted in the discovery of the bacillus anthracis in blood taken from the affected men, and established beyond a doubt that the disease theretofore known as "wool-sorters' disease" was true anthrax. It was in these cases the result of inoculation, through the medium of abrasions, from handling wool cut from sheep which had died of the disease. The demonstration that anthrax was communicable to man attached to it additional and intense interest, and plainly revealed the imminent danger to mankind, not only from handling infected wool, but from all the operations attendant upon flaying, destroying, or making post-mortem examinations of the carcasses of all species of animals that had died of the disease. An important part of the labors of the English veterinary authorities in reference to this incurable and uneradicable disease has been a wide dissemination of published warnings against the careless handling of anthrax-infected carcasses.

It is evident that anthrax is a disease the eradication or even restriction of which is not likely to be effected by the application to it of the suppressive principles embodied in the contagious diseases of animals acts. In the first place, the prevention of the importation of the infection from foreign countries could be attained only by placing an embargo upon the entire import trade, since wool, hides, grain, hay, and all substances associated in any way with animal industry are capable of carrying the germs of the infection. And if within the Kingdom the system of compulsory declaration of disease could be so perfectly enforced that every single case could be detected, the usual plan of isolating the outbreaks by the declaration of infected places would be impracticable and unendurable to farmers and stock owners, because the consequent restrictions upon the movements of animals and interference with stock traffic would likely be regarded as needlessly

oppressive and an unwarrantable interference with individual rights in the case of a disease like this, which usually attacks only a few animals in a locality and then often disappears. Nor is the slaughter of diseased and exposed animals likely to tend to the eradication of the malady, because sudden death usually anticipates this action in the case of diseased animals; and in the case of animals merely exposed to the infection it seems a wasteful course to pursue, because usually the great majority of the latter do not contract the disease. The course pursued by the British Government in reference to this affection, therefore, has been more of an educational than a suppressive nature, with a view to teaching farmers and stock owners, through the issuance of leaflets, etc., as to the means by which this extremely fatal disease is spread, and as to the precautions necessary to prevent its extension upon infected premises, earnestly urging the immediate burial of the carcasses of diseased animals intact, because of the dissemination of the disease by the blood which escapes from a carcass upon which any attempt at salvage is made.

The statistical knowledge to be had of anthrax in Great Britain dates from 1886. In that year some of the local authorities urgently represented to the privy council that great risk was constantly being incurred from the removal of carcasses of animals, without precautions, which had died of anthrax. As a consequence the council passed an order in September of that year making anthrax a disease for certain purposes of the diseases of animals act of 1878, but, for reasons given above, the powers of the local authorities, as applied to this malady, were limited simply to making regulations for the movement of animals, carcasses, fodder, litter, etc., and for the cleansing and disinfection of infected premises. These regulations are now of little interest, because, either through a lack of their proper enforcement or observance or through some insidious power of resistance in the disease, they have proved ineffective as preventive measures. But the annual statistical reports, published under the provisions of the order, have shown that the disease was more widely distributed in Great Britain than was at first supposed, and tend to dissipate the idea, which formerly prevailed, that the prevalence of the disease is dependent upon certain conditions of soil and climate, for the returns indicate that it prevails at all seasons of the year and on all lands—either wet or dry—without distinguishable difference.

After legislation against anthrax was once undertaken by the British Government, many local authorities became desirous of a grant of power to slaughter and pay compensation. The ground upon which they based their requests to the council was that on some premises where only a few animals were kept the disease might be stamped out with less inconvenience than attends the usual process of suppression by isolation. On account of a marked increase in the disease an order was passed, and came into operation at the beginning of 1893, including horses, asses, and mules as animals susceptible to the disease as well as cattle, sheep, and swine, and giving the local authorities power to slaughter such animals as were affected, or suspected of being affected, but allowing the owner to appeal to the board of agriculture, if for any reason he should deem slaughter undesirable. Notwithstanding the adoption of these more stringent measures, the number of outbreaks and the number of animals attacked increased in a remarkable degree during the year 1893, and almost doubled those of any year since statistics have been obtained. In 1894 a decrease

occurred, but not of a magnitude sufficient for valuable deductions. The following table gives the history of the prevalence and fatality of anthrax in Great Britain from 1886 to 1894:

Year.	England.			Wales.			Scotland.		
	Infected counties.	Fresh outbreaks.	Animals attacked.	Infected counties.	Fresh outbreaks.	Animals attacked.	Infected counties.	Fresh outbreaks.	Animals attacked.
1887	38	215	594	1	1	6	12	22	49
1888	37	158	310	1	1	6	11	26	91
1889	33	145	264	.....	.....	.....	11	23	51
1890	37	139	510	.....	.....	.....	11	18	32
1891	39	200	416	.....	.....	.....	12	34	68
1892	44	243	557	1	1	3	15	50	104
1893	45	413	1,005	5	29	77	18	121	218
1894	45	368	791	4	29	37	15	112	172

It is evident that anthrax derives its chief importance in Great Britain from its remarkable fatality both to men and animals rather than to the financial losses it imposes upon stock owners. Individual losses, usually due to ignorance or neglect of proper precautionary measures, are in some instances unquestionably serious; but to the stock-raising industry as a whole the losses are comparatively unimportant, the average number of deaths since statistics have been collected being about four to each outbreak.

Since the disease has increased so rapidly in England of recent years, notwithstanding the restrictive legislation that has been directed against it, considerable attention has been attracted to other proposed methods of prevention. For obvious reasons interest has centered around the safest method for the disposal of carcasses. The conclusion arrived at by the English authorities is that "the burial of a carcass as near as possible to the place where it has been found lying is the safest method of dealing with it. Contamination of the soil undoubtedly happens very frequently, not, however, from the buried carcass, but from the blood which may have escaped from it before burial, during its removal to a convenient spot for the purpose, or during the post-mortem examination. In order to avert the consequences arising from the contamination, every part of the surface of the ground on which blood or excretions from the animal have been spilt should be covered well with quicklime, or, still better, taken off to the depth of some 6 inches and burned." Burning the carcass is advised against because "it involves cutting, to the great danger to the operators and the certainty of contaminating their clothes and the ground on which the operation is conducted." Post-mortem examination is unnecessary and should never be made, the bacillus being easily detectable in the blood of the ear, which may be cut off for the purposes of investigation.

In some countries, especially in France, vaccination as a preventive of anthrax has been practiced with great success. The subject was first brought to the attention of the medical and veterinary professions in England by M. Pasteur, the discoverer of the protective virus, in an address before the medical congress held in London in 1881. The announcement of the discovery did not attract general attention in England at the time, because anthrax had not then been legislated against, and its prevalence was not believed to be sufficiently widespread to warrant fear of any serious losses from the disease excepting in somewhat rare instances. Interest in the subject,

however, from a scientific point of view led to a test of the efficacy of the vaccine material under governmental direction, with results in direct conflict with the assertions of M. Pasteur, as all the sheep experimented upon either died as a result of the injection of the vaccine material or afterwards succumbed to anthrax when inoculated with the germs of that disease. The recent increased prevalence of the disease in Great Britain, however, and the apparent success that has attended vaccination against anthrax in some other countries have of late years attracted the attention of the Government to this means of rendering animals immune to the disease, but many objections seem to be found to the general adoption of this preventive operation.

The exact method of producing the vaccine material is a secret. A monopoly of its manufacture and sale was formerly held by M. Pasteur. A company was afterwards organized in Paris, with a capital of $200,000, which now holds the right to manufacture. Large quantities are used in France, where the vaccination is not carried on under Government supervision. A considerable export trade is also done with other countries in Europe and South America.

LIVE-STOCK SHIPMENTS FROM CANADA, ARGENTINA, AND AUSTRALIA TO ENGLAND.

For more than a year past experiments have been in progress to test the practicability or shipping live stock to England, both from Argentina and from Australia. These ventures are of especial interest to the United States, as the successful issue of either of them would place a new and formidable competitor to the sale of United States cattle in the English markets. The experiments, too, furnish an interesting illustration of the wonderful progress that has of late years been made in the transportation of live stock over long and tedious distances by sea. A few years ago the skill and expense required in fitting up a vessel for the safe and careful carrying of 1,000 head of live stock through many climates over a voyage of from sixty to seventy-five days, and of furnishing room for the immense stock of feed and water necessary for the sustenance of the animals, would have been thought an effective bar to profitable trade.

The shipments from Argentina have proved profitable, and the live-stock trade between that country and England seems permanently established. From the initial shipments in 1890 of 653 cattle and 22,075 sheep, the trade has grown steadily and without interruption until in 1895 the estimated shipments were respectively 39,000 and 317,000 head.

The trade with Australia, however, is still a tentative one, and a brief sketch of its progress and growth may be interesting. The first shipment arrived at Deptford, England, on September 24, 1894, and consisted of 19 fat and store bullocks. It was designed by this venture simply to reveal two conditions precedent to a successful future trade—first, to ascertain how the cattle would fare through the long voyage, and second, how they would thrive after arrival in England. The result was in the latter respect a failure. Only one animal died on the voyage, but the remainder arrived in bad condition.

The experiments, however, were not abandoned, and in July, 1895, another shipment of 71 bullocks and 20 sheep arrived at Deptford. The cattle consisted of Herefords and Hereford crosses, Devons and Devon crosses, and a few Shorthorn crosses. Though their appearance was bad, the hair being lost in patches, the cattle were in excellent condition. After slaughter the carcasses had a very marketable appearance, thick in chine and without wasteful fat. The discouraging feature of this experiment was the heavy death rate, 9 bullocks and 20 sheep having been lost en route.

The success of these, and also of some other small ventures from New Zealand, seems to have at least been sufficient to warrant the broadening of them into a large and essentially commercial venture in which Australia manifested great interest.

On the 9th of last July the steamship *Southern Cross* left Sydney with a cargo of 550 bullocks, 488 sheep, and 29 horses. A good proportion of the cattle were full or three parts bred Shorthorns, with some Hereford-Shorthorn crosses, and a few Herefords, all fairly well bred. They were described as a grand lot of bullocks, from 4 to 7 years old, and very wild—the latter quality likely to be conducive to a great loss of flesh during the voyage. The sheep were a rough lot of Merino and Merino crosses.

After being out for sixty-two days this cargo was landed at Deptford. The damaging effects of the voyage were very apparent; the cattle had lost flesh considerably, the hair was off in patches, probably caused by the heat in the ship, and they presented a very ragged appearance. About one-third of them would make good, useful beef, but when it came to the inferior portion of the cargo it was very poor and of low consuming value.

The losses during this voyage comprised 51 bullocks, 80 sheep, and 1 horse. A partial cause of this heavy mortality was revealed after reaching port by the discovery that 6 bullocks were afflicted with pleuro-pneumonia, and the total number found affected by this disease was doubtless increased when the animals were slaughtered. It is prevalent in Australia, and, if the animal is affected before starting, the rapid development of the disease during the long and trying voyage is almost certain. Another obstacle was thus found in the way of a live-stock trade from the Antipodes. There is little chance of a liberal English market for Australian cattle until pleuro-pneumonia is exterminated from its herds.

Notwithstanding the discouraging features of these ventures, great confidence is still felt in Australia that a successful and profitable trade will be established; and a second important consignment of Australian cattle arrived at Deptford, via Buenos Ayres, in October, 1895. Out of 230 bullocks shipped at Sydney, 210 were landed alive. Though too big and rough and having too much age to suit the English trade, they were landed in a decidedly better condition than those which came by the *Southern Cross*. Like the former cargoes they were reported as having lost their hair in patches and as looking very ragged, but as having some very useful Shorthorns and Herefords among them.

The result of the last two shipments from Australia would, taken all in all, indicate that the obstacles in the way of establishing a profitable trade may not be unsurmountable. The cure of two evils at least would seem essential to its success—first, the eradication of pleuro-pneumonia; second, improvements in ship appliances for handling cattle from embarkation to debarkation, such as would insure all possible immunity against loss of weight, against the "ragged" appearance complained of, and against loss at sea. The history of the export cattle trade of the United States plainly illustrates the possibility of a remedy for both these evils. Here pleuro-pneumonia has been completely eradicated, and our facilities for transporting cattle across the Atlantic have steadily improved since the inception of the trade. It will be remembered that the first attempt to supplement the meat supplies of Great Britain by the transatlantic transportation of live cattle from the United States was made in 1875. The venture at first met with many of the difficulties that now confront the Australian experiments. Bad ventilation and inefficient facilities aboard ship for the care of stock, particularly during tempestuous weather, and other causes, resulted in heavy losses, and in

some cases in the destruction of the entire cargo. Improvement, however, followed close in the wake of experience, and the percentage of loss decreased as the volume of trade grew. From the first the business met with comparative success, and as early as 1877 amounted to 20,000 head of cattle and a somewhat larger number of sheep. In 1889 the trade in cattle alone had increased to 294,424 head, but the losses then amounted to 21 of every thousand that were embarked. In 1891 occurred an event which marks the beginning of an era of assured and permanent success, and from that time the losses at sea rapidly dwindled to a minimum. In that year the United States Department of Agriculture issued regulations prohibiting the embarkation of live stock suffering in any way from disease, or even from the liability of contagion, and requiring the fitting up of vessels intended for the export trade with a strict regard for their cleanliness and disinfection, ample room, reliable care, and proper food and water for the stock. The beneficial results of the regulations were immediately apparent. In 1893 the mortality among animals in transit had been reduced to a minimum loss of three in every thousand head. The degree of perfection that has now been attained in United States vessels in the handling of export cattle is perhaps best attested by good English authority. Mr. George T. Turner writes in the Live Stock Journal (London), under date of September 30, 1895:

> With regard to the United States cattle boats, they are now fitted to perfection, as a rule, and the quality and condition of the great bulk of the cattle landed here would be a surprise to many. They are put upon the Deptford market in far and away better condition than that in which the Irish cattle come to any of our markets; and, more than that, they have a much less traveled appearance than the ordinary run of cattle exposed in our markets after from twelve to twenty-four hours trucking by rail. It is only those who have actually seen these beasts many times who can realize the truth of this statement. As stated on a former occasion, the States beasts are nearly all polled, not from sawing off the horns of adult cattle, but from the application of caustic potash to that portion of the crown where the horns would appear, after cutting away the hair, before the calf is 3 days old. This is a course which breeders of store stock in this country (and especially in Ireland) might adopt with great advantage.

The lack of any such facilities for handling stock aboard the Australian vessels is perhaps best illustrated by the fact that it took fifteen hours to land 499 cattle and the few sheep from the *Southern Cross*, whereas 769 United States cattle were landed from one boat in fifty-two minutes and 655 from another in forty minutes, the total loss on the two latter cargoes being only 1 beast.

Existing conditions, however, seem to favor, if not to necessitate, the ultimate establishment of broader trade in some form between Great Britain and her distant colony. In Australia the supply of live stock is excessive; a sparse population limits the home markets within narrow bounds and makes a foreign trade a public neccessity. In England the people rely more and more each year upon the colonial and foreign supply of meats with a racial predilection in favor of the colonies. Australia has long discussed diverse schemes to attain the greatest advantage from these relative conditions. The first and apparently the most feasible plan for relieving the glut in her live stock supply was by transporting meats in a frozen state to the needful English markets, but this has not been productive of wholly satisfactory results. The experiment was begun in 1880 with a shipment of 400 carcasses of mutton, and has since assumed mammoth proportions. Experience in the business resulted in a gradual lowering of the cost of handling, freezing, and freights, and in 1895 it was estimated that 933,401 carcasses of frozen mutton and 449,000 hundred-

weight of frozen beef were transported from Australia to London; a tendency to reach out for a broader market was also manifested by a consignment of 19,500 carcasses of sheep to Liverpool and 16,800 to Manchester, the latter the initial shipment of a regular monthly steam service to the north of England markets. In fact, it may be said that Australia now holds a monopoly of the frozen-beef markets of England, her only competitors in 1895 being New Zealand, with estimated consignments of 15,000 hundredweight, and the River Plate, with a little less than double that amount. The frozen-mutton trade, however, was more equally distributed. New Zealand, though she did not enter the English markets until two years later than Australia, sent 2,409,577 carcasses to London in 1895, and the River Plate, which consigns most of her supplies to Liverpool and thence virtually monopolizes the northern trade of England, furnished 1,615,242 carcasses to the markets of the United Kingdom.

This frozen-meat trade is not so satisfactory, either to consignor or consumer, as its volume might purport. The quality of the product is impaired by freezing, and its sale is limited to the class of buyers who are attracted only by the cheapness of price. The supply, on account of the long distance between the source of the product and the place of sale, coupled with a lack of proper organization in the trade, is not equally distributed throughout the year; a glut in the market often alternates with scarcity of supplies, resulting in dissatisfaction to all parties interested, because of the uncertainty and wide variations of price. The sometimes inferior quality of the Australian product often results in an all-round lowering of prices in the British markets on all kinds of imported meats. The dissatisfaction which arises from these and other features of the trade naturally turns the attention of the Australians to additional possible means of handling their surplus that may prove more profitable and satisfactory, i. e., both to a live-stock trade and to a different method of handling dead meats.

Almost simultaneously with the endeavor to organize a live-stock trade experiments were also undertaken to test the possibility of shipping chilled instead of frozen beef to England. It will be readily recognized that success on these lines would confront the United States with almost as serious a competition as would the establishment of an Australian live-stock trade. The United States has for many years held almost a monopoly of the chilled-beef trade in the British market, furnishing fully three-fourths of the total supply—the amount that was estimated as shipped by this Republic in the year 1895 was 1,652,000 hundredweight. As regards the possibility of Australian success in this field, however, the experiments are as yet inconclusive. The first trial shipment was made in 1894. Three more shipments followed in 1895, the first and second from New Zealand, with the Shiel's thermostat as an essential feature; the third from Queensland, with no special means of regulating the temperature. Of these last three experiments the first was the most satisfactory, due largely to the fact that it reached an exceptionally brisk market for American chilled beef, and, though slightly frozen, the beef was described as being in a nice, bright condition. The quality of the second shipment, though less impaired by freezing, was more or less affected by mold, and entailed a loss upon those engaged in the experiment. The third shipment, after being forty-nine days aboard ship, had to be frozen, and, after a seventy days' voyage, arrived in London in a condition not distinguishable from ordinary frozen beef. But, unpromising of success as these ventures seem to be, the inventive genius of man may

doubtless be relied upon to find a way to success. It is claimed even now that there has been invented in the United States a process of keeping meats during transportation without ice, by the use of sterilized air, and that this process has already been sold to an Australian meat transportation company. There is little doubt that in some way Australia will become in time a still more serious competitor of the United States in the meat markets of England.

The following report, under date of May 27, 1895, by Dr. W. H. Wray, United States inspector at London, furnishes important data concerning the live-stock trade from Argentina and Australia to England:

Hon. J. STERLING MORTON,
Secretary of Agriculture.

SIR: In reply to your request of the 30th ultimo, I take pleasure in herewith sending you some particulars regarding the live-cattle trade from Argentina and Australia to England.

During the year 1894 there were shipped from Argentina to Deptford, England, 5,018 head of cattle and 37,235 head of sheep, with a loss at sea of 45 head of cattle and 674 head of sheep.

Between the 1st of January and the 16th of May, 1895, there were shipped from Argentina to Deptford 7,659 head of cattle and 89,924 head of sheep, with a total loss at sea of 361 head of cattle and 2,276 head of sheep. There have also been shipped 3,136 head of cattle and 34,607 head of sheep from Argentina to the port of Liverpool between January 1 and May 16, 1895, with a loss of 247 head of cattle and 1,170 head of sheep.

The shipments of cattle and sheep from Argentina will undoubtedly increase, as such cattle and sheep are paying the exporters a fair profit.

The cattle that are shipped from Argentina to England weigh about 1,100 to 1,200 pounds each, are rough, coarse, and unfinished, are very wild, and sell there at $25 to $30 per head.

The ocean freight from Argentina to England is from $22.50 to $25 per head, according to the season of the year and the space occupied on board the ship; the feed and attendance will cost about $15 per head. The voyage from Buenos Ayres to Deptford usually occupies from twenty-eight to thirty days.

The average price realized from these cattle at Deptford is $70 to $80 per head. The cattle from Argentina are mostly fed upon alfalfa, which causes the meat to be soft and flabby and very difficult to "set" after slaughter. The meat is not such a bright or good color as that from the United States cattle. Many butchers will not purchase these cattle on that account.

The trade in live cattle from Argentina to England began in 1891 with a shipment of 22 head, and has since grown to its present large proportions.

As a rule, the cattle from Argentina are landed in England in very fair condition and with a reasonably small percentage of loss considering the class of steamers and the primitive mode of erecting the stalls, etc. The space for cattle is invariably on the top deck or in the wells. The cattle fittings are erected in order to comply with the British regulations, but are very poorly erected, are only temporary, and are erected so that two tiers of sheep pens can be built on top of them.

Only a small number of cattle are shipped on each steamer on account of the large space required for fodder. The fodder for 220 head of cattle from Argentina to England would require from 12,500 to 15,000 cubic feet of space.

The live cattle and sheep from Argentina are not allowed to go inland, and are all slaughtered within ten days after landing at the foreign cattle markets.

There are no companies or combinations being formed in England for the purpose of carrying on the South American live-cattle trade; in fact, it would be impossible to form such because the laws, which are now enforced at all the foreign cattle markets throughout England, compel the animals to be slaughtered in many different places, a small number at a time. There is not room or conveniences in any of the slaughterhouses at the foreign cattle markets to slaughter more than 300 head of cattle per week.

If a company could be formed for the purpose of handling the South American cattle it would be compelled to have a special act of Parliament passed giving it the privilege to erect an abattoir and to land its cattle at such a place.

There have been a few cargoes of live cattle received at Deptford from Australia as follows: In 1894, two cargoes of 19 and 17 head, the original shipments being 20 and 18 head, respectively, each steamer having lost 1 bullock during the voyage.

With the last consignment were 48 head of sheep, 42 of which were landed in a very poor condition.

To date during 1895 there have been five cargoes of live cattle received at Deptford from New Zealand, one cargo of 34 head, lost 4; one of 33 head lost 4; one of 18 head, lost none; one of 80 head, lost 6, and one of 102 head, lost 9 during the voyage. The number lost at sea should be added to the number received in order to get the total number that was shipped.

The cattle from New Zealand that have been landed at Deptford have been of very good breeding, mostly Herefords and Durhams, were in good flesh, weighing between 1,500 and 1,800 pounds; in fact, some of them were too fat for the London market. These cattle realized as good a price as cattle of like quality and weight from the United States.

There is very little, if any, prospect of the Australian or New Zealand live-cattle trade increasing to any extent, owing to the many obstacles that would interfere with any profit being made therefrom.

The main obstacles to shipping live cattle from Australia to London are the long voyage (between sixty and seventy-five days); the many different degrees of latitude through which a steamer is compelled to pass, necessitating great care on the part of the attendants in order to prevent the catte from becoming sick, owing to the many changes in the climate; the high freight charges ($40 to $50 per head), and the large space on board ship necessary for fodder.

At the present time there is no port between Australia and London where a cattle steamer can put in for fodder on account of the regulations imposed by the British board of agriculture, which declare all ports en route to be infected with foot-and-mouth or some other disease.

In two instances steamers were forced to put in port for coal, and on arrival at Deptford the cattle were not allowed to be landed alive but were slaughtered on board steamer and hauled ashore.

No cattle have been shipped from Australia to any port in Great Britain except London. The live cattle and sheep from Australia are landed at the foreign cattle markets, and are there subject to the same rules and regulations as the live cattle from the United States.

I will send you a special report about the cattle on steamships *Queensland* and *Helenes* as soon as they are landed and sold.

It may interest you to know that a large company is being formed, to be composed of American and English capitalists, for the purpose of shipping live cattle from Galveston, Tex., by steamer direct to Manchester, England. I will let you know more about this company in the near future.

Very respectfully,

W. H. WRAY.

LONDON, *May 27, 1895.*

Dr. Wray also submits the following report of the number of cattle received at the foreign cattle markets, Deptford, from Argentina between January 1, 1894, and January 1, 1895:

Name of steamer.	Number of cattle.		Number of sheep.	
	Landed.	Lost.	Landed.	Lost.
	69	1		
Lomas	80		185	4
Do	127		633	7
Ariosto	123	3	562	12
Do	78			
Petarch			643	10
Do	137	3		
Ovingdean Grange	149	1		
Do	124		693	18
Bellova	80		568	23
Do	91	5	287	2
Hesperides	69			
Burton	140		735	5
Bellagio	142		783	17
Do	143		789	
Do	135	5	492	3
Port Adelaide	35		280	2
Acanthus	132		370	2
Italian Prince	158			
Hornby Grange	119			
Do	163		194	6
Do	163	2	282	15
Do			1,272	9
Buenos Ayres	140		500	
Mascotte	152		949	11
Queensland	150		979	21
Do				

Number of cattle received at the foreign cattle markets, Deptford, etc—Cont'd.

Name of steamer.	Number of cattle.		Number of sheep.	
	Landed.	Lost.	Landed.	Lost.
Tasso	110	.....	196	4
Bellanock	119	1	497	2
Rosarian	132	.....	984	14
Asturian Prince	104	1	425	5
Do	59	.....	598	5
Franklin	.....	.....	298	1
Zeo	.....	.....	794	6
Zeno	.....	.....	1,550	18
Do	.....	.....	1,580	13
Dante	.....	.....	586	.....
Bellarden	75	1	398	2
Eskdale	55	3	1,187	7
Hawkhurst	161	.....	.....	.....
Eiffel Tower	136	.....	610	1
Nutfield	.....	.....	1,389	20
Cynthiana	128	1	390	1
Melbourne	154	.....	669	3
Florence	119	1	.....	.....
Hounslow	99	1	876	23
Siddons	.....	.....	592	8
Avona	183	.....	.....	.....
Zita	.....	.....	1,296	26
Do	.....	.....	1,236	171
Zodeac	100	.....	707	7
Bishopsgate	.....	.....	1,579	24
Valor	104	.....	673	12
Emin	.....	.....	1,194	14
Dryden	122	3	593	7
Baron Glamis	114	13	592	36
Spenser	.....	.....	1,540	48
Mercedes	.....	.....	632	8
Endeavour	.....	.....	1,644	21
Total	4,973	45	33,561	674

Number of cattle received at the foreign cattle market, Deptford, from the Dominion of Canada between January 1, 1894, and January 1, 1895.

Name of steamer.	Number of cattle.		Name of steamer.	Number of cattle.	
	Landed.	Lost.		Landed.	Lost.
Hamburg-American Line:			Allan Line:		
Wandrahm	971	.....	Austrian	1,757	86
Pickhuben	698	.....	Brazilian	1,609	1
Steinhoft	211	.....	Monte Videan	1,946	4
Stubbenhuk	420	.....	Rosarian	1,660	1
Baumwall	582	1	Total	6,972	92
Italia	193	.....			
Total	3,075	1	Elder, Dempster Line:		
			Mariposa	228	.....
Thomson Line:			Memnon	127	.....
Gerona	2,064	1	Merrimac	375	.....
Dracona	452	1	Etolia	455	.....
Hurona	1,666	1	Total	1,185	.....
Escalona	700	.....			
Fremona	1,317	4	Tramp steamers:		
Avlona	338	4	Anvers	181	.....
Iona	2,247	4	State of Georgia	114	26
Total	8,784	15	Total	295	26
Ross Line:					
Storm King	168	.....			

RECAPITULATION.

Name of line.	Number of cattle.		Name of line.	Number of cattle.	
	Landed.	Lost.		Landed.	Lost.
Allan	6,972	92	Ross	168	.....
Thomson	8,784	12	Tramps	295	26
Hamburg-American	3,075	1			
Elder, Dempster	1,185	.....	Total	20,479	131

TEXAS FEVER IN AUSTRALIA.

Australia is suffering great uneasiness from the ravages of a cattle disease. It is impossible to confirm a diagnosis of an animal disease with entire certainty without an opportunity to observe the symptoms and pathological appearances; but, presuming that the written and published descriptions are accurate and complete, little doubt is left that the disease from which Australian cattle are suffering is Texas fever. Another link is thus added to the chain of evidence in proof of the belief often maintained by this Department that Texas fever, which was in the first instance asserted to be limited to the southern portion of the United States, does exist in other countries where the conditions, climatic and otherwise, are favorable to its development.

The Australian outbreak is one of especial virulence and wide and swift dissemination—a state of affairs largely due to climatic influences and the conditions of life on that continent. The infected district, which is very hot and has a supply of water at certain seasons of the year scant and unhealthy, is devoted to cattle raising on a large scale. When, in the course of trade, the immense herds are moved from one part of the country to another, they are usually driven on foot, and, as was the case in the United States when this method of moving cattle from the Southern States was common, the traveling herds infect with ticks the stock routes or trails along which they pass and the fields where they are fed and pass the nights. Bordering the stock routes in Australia are immense tracts of uncultivated land, where numerous herds, known as "bush cattle," run wild. These cattle, crossing the routes, become infected with ticks, and, roaming at will over a wide expanse of country, in turn infect large areas. The ox teams, too, which in the primitive development of the country are still an important factor in the transportation business of the continent, undoubtedly aid much in infecting the highways over which they laggardly draw their heavy loads. It is not strange, in the midst of conditions so favorable to the dissemination of this peculiar disease, that in one year it has been carried 200 miles inland from the extreme limits where it had formerly appeared.

The localities which are or have been infected are scattered over a broad expanse of territory that borders the northern coast of the continent along the Gulf of Carpentaria and extend as far south as the twenty-first degree of south latitude. On the east and west the infected regions are comprised between the one hundred and thirtieth and the one hundred and forty-fourth degrees of east longitude. The infected district thus covers a portion of two colonies, viz, Queensland and the northern territory of South Australia.

The length of time during which the disease has prevailed in Australia is not exactly determined. The first official inquiry into its

nature and cause was made in Queensland in 1895, and was induced by the uneasiness that existed in the minds of the cattle owners, financiers, and others who had large interests in the infected districts. But there is little doubt that the disease was in existence in the northern territory of South Australia ten years before, and thence was introduced into Queensland. It was first noticed among droves of stock and ox teams traveling between the MacArthur River and Port Darwin; and in 1885 it was known to be working destruction among cattle along the Roper River in the former colony. The extent to which the disease was disseminated over the country from these sources was not realized until a few years ago when the meat works for boiling down purposes were erected on the Albert and Norman rivers in the colony of Queensland. Droves of cattle for these works, starting from or crossing an infected district, acquired the disease, and not only died in great numbers but infested the roads over which they passed, whence the disease was carried by bush cattle over the entire country. Out of 18,000 head of cattle forwarded in different droves to the meat works on the Norman River between June and December, 1894, fully 8,000 came from healthy districts and along stock routes where no infected cattle had passed and consequently arrived in fair condition; but of the remaining 10,000, which either came from or traveled over infected country, 1,000 died from the disease. Great alarm ensued over this situation. The colonial government of Queensland instituted an investigation and the greater part of the colony found infected was placed under quarantine for a period of six months.

Under the authorization of the colonial secretary, Mr. C. J. Pound, director of the Queensland Stock Institute, Brisbane, proceeded to the infected district, and, after two months of minute observations and experiments, made a lengthy and detailed report under the caption Redwater Disease in Cattle in the Gulf District. Excerpts from this report, given below, point plainly to the identity of this disease with Texas fever. The well-known outward symptoms of Texas fever—the blood-red color of the urine, the manifestations of the disease in two forms, acute and mild, the immunity of animals from a second acute attack, and the insusceptibility of animals other than cattle—are not only found in the disease treated of in the report, but the scientifically observed symptoms—the period of incubation, the thermometric tests, the increase of cardiac and of respiratory action, and above all the causation of the disease by the introduction of a microorganism into the blood through the agency of a tick—all coincide in minute detail with the studies of Texas fever made in the United States.

Director Pound says:

> The disease is characterized by the affected animals passing urine of a blood-red color, but which varies in degree according to the severity of the disease.
>
> In some instances the disease appeared in an acute form, often killing numbers of animals, while in others it was of a mild or nonfatal type.
>
> Last year on some of the stations or stock farms when the disease made its appearance for the first time it assumed a most virulent form, and although numbers of undisturbed cattle died in consequence, the remainder of the herds completely recovered.
>
> This year, however, on the same farms the disease again made its appearance, but was of such a mild type as to be scarcely noticeable to the ordinary observer. In fact, in the majority of cases it was found practically impossible to diagnose the disease in the living animal, and, providing it was deemed advisable to kill the animal, the post-mortem examination, as evidenced by the appearance of the internal organs, would only reveal the fact that the animal had suffered from an extremely mild attack.

I attribute this to the fact that, like several other animal diseases of microbic origin and a similar character, a primary nonfatal attack always more or less affords protection or immunity against subsequent attacks.

To the stock owners residing in these infected districts this subject of acquired immunity in connection with this disease is of extreme interest and importance, and no opportunity should be lost in collecting notes and making observations on the nature of the disease from its first appearance, as such information will very materially assist in bringing about some effectual means of mitigating the disease.

By some stock owners this disease is often mistaken for and in some instances believed to be true anthrax or Cumberland disease, which is easily accounted for, as the symptoms in the affected animals are very similar. Moreover, on post-mortem examination we find an enormously enlarged spleen with dark-colored tarry-looking contents, which is also a peculiar feature in anthrax. It has frequently been asserted that this condition of the spleen is not only peculiar to but sufficiently diagnostic of anthrax, which is altogether erroneous and very misleading to the ordinary observer, for although the naked-eye appearances of the internal organs materially assist us in diagnosing certain diseases, it is only a careful microscopical examination of the spleen blood with a suitable instrument and the necessary reagents that we are able to prove whether or not we are dealing with anthrax. However, in our disease we find post-mortem appearances of some of the other internal organs which are entirely absent in anthrax.

Further, we have something still more remarkable which differentiates this disease from anthrax, namely, that it is peculiar to cattle only; horses, sheep, pigs, goats, and other domesticated animals enjoy perfect immunity, i, e., they are naturally insusceptible and never acquire the disease under any circumstances, even although they may feed and mix with cattle in every stage of the disease.

This is substantiated by the following practical observations:

(*a*) At Donor's Hill Station, after the disease made its first appearance, numbers of cattle were very severely attacked and some could be seen dead and dying alongside of the different water holes, while the sheep which were associated with them were in a perfectly healthy condition; moreover, they were entirely free from the same kind of tick which infested the cattle. Now, had this disease been anthrax, the mortality among the sheep, which are very susceptible animals, would have been considerable.

(*b*) A flock of sheep, numbering between 6,000 and 7,000, were driven from Avon Downs, northern territory, to the Normanton Meat Works. The greater part of the journey was along the main stock routes and often in the same tracks of mobs of cattle badly affected with the disease, while at night they would camp at the same lagoons where dead cattle would be lying all around, yet not a single sheep exhibited any symptoms of sickness whatever. Further, I had an opportunity of seeing more than half of them slaughtered, and examined the internal organs, but could not find any of the characteristic post-mortem appearances which are indicative of the disease in cattle.

(*c*) Pack and saddle horses which accompany traveling mobs of cattle in which the sickness prevails have never been known to show signs of the disease under any circumstances. The same holds good for station horses that are associated with sick cattle in the same paddock.

(*d*) Owing to the scarcity of grass and the stunted vegetation in an around Normanton, some of the inhabitants keep small herds of goats, which mix up with infected cattle. Strange to say, the ticks apparently do not live on these animals, and they are always in a healthy condition.

(*e*) At the Dalgonally Meat Works, on the Norman River, several hundred pigs are kept for the purpose of consuming the offal. Fully 50 per cent of this comes from animals that have been slaughtered in various stages of the disease, yet none of the pigs exhibited any signs of the disease, either before or after slaughter; but had this material come from anthrax animals it would have proved quite different, for, although pigs are not very susceptible to anthrax in the ordinary state of affairs, under these circumstances a good many, especially the young ones, would certainly have contracted the disease and died.

The period of incubation, i. e., the time which elapses from the date of infection to the first appearance of the disease, varies considerably in different animals, according to their environments. As a rule it is from five to twelve days; afterwards the course of the disease is so rapid and acute in some animals that they readily succumb in a few days, whereas others remain sick for several weeks, or even months, and become so emaciated that they are generally spoken of as wasters.

Traveling stock are greatly influenced by the surrounding conditions, namely, scarcity of good grass and water, the latter, especially in the water holes on traveling stock routes, often being in such a filthy state that even thirsty cattle are loath to drink it, and, worse than all, driving cattle at a fast rate across open plains

during the hottest part of the day, which, in my opinion, tends to increase the mortality considerably.

As already indicated, we have two types of this disease, one very acute, of short duration, with high fever, and frequently fatal; the other a very mild form, more prolonged, fever less pronounced, and very rarely fatal.

There can be no doubt that climatic influences and other conditions are in some degree accountable for the differences in the virulence of this disease, for whereas the mild, less fatal, form exists to a certain extent all the year round, the acute and more fatal type is practically present during the summer months only.

A very characteristic symptom in the more acute form is the extraordinary high temperature of the body, which is indicative of very serious changes in the constituents of the blood. By means of the clinical thermometer I was able to ascertain the temperature of over 200 animals representing nearly every stage of the disease. These were controlled by taking a series of temperatures of healthy cattle. The normal temperature varies from 100° to 102.4° F., while in the diseased animals the minimum temperature was as low as 100.8° F., and the maximum reached as high as 109.2° F. In 63 per cent of the cases examined the thermometer never registered below 107.6° F., in 28 per cent it ranged between 104.2° and 107.4° F., while of the remaining 9 per cent the temperature never rose above 104° F.

The majority of these temperatures were taken at the meat works. Directly the animal was pithed in the killing pen the thermometer was passed into the anus and the temperature taken per rectum; some of these were open to fallacies from the fact that the animals had been overheated through driving from one yard to another. In the bush, however, the sick animals are picked out from the rest and without any excitement shot on the spot; immediately afterwards an incision or pocket is made into the cellular tissue just behind the shoulder; into this pocket the thermometer is inserted; the objection in not taking the temperature per rectum is that during the struggles of animals the thermometer is liable to be broken.

The exceptionally high temperature in the acute form usually remains fairly constant until the end of the fever, when, if the animal should recover, the temperature gradually falls to the normal, but if the disease should terminate fatally the temperature will fall as low as 97.4° F. an hour or so before death.

Among traveling stock the fact must not be overlooked that this high temperature is in some degree influenced by exposure to the sun's rays, for it has been demonstrated repeatedly that the temperature of an animal in perfect health will often rise 2 and 3 degrees when exposed to the action of the sun's rays for a short time only.

The fever in the acute form of disease may be readily detected by any ordinary stockman who is accustomed to the handling of cattle, as the whole of the skin feels very hot and dry to the touch, more especially under the shoulders or between the hind legs; in fact, any part of the animal's body where the skin is thin and there is a scarcity of hair.

As the disease advances the heart's action becomes more rapid, until we find the number of pulsations increase to between 100 to 120 per minute, nearly double that of the healthy animal, which varies from 60 to 70 per minute.

In the acute form of disease the respirations range from 70 to 120 per minute, whereas in healthy animals the number varies from 25 to 35 per minute.

Although the sense of vision is somewhat impaired, the animal has a full, bright eye, which is entirely different from some other diseases, notably pleuro-pneumonia, in which the animal has a dull, sleepy look about the eyes. In the more advanced stages of the acute type the animal becomes somewhat unconscious, and has a staggering mode of walking; the nose and lips become very pale colored, hot, and dry; the animal refuses to eat, and rumination or chewing the cud ceases entirely; the fæces or dung, owing to constipation, appears as small round hard balls, often coated with blood-stained mucus.

Another still more important and characteristic symptom from which the disease takes its name, "redwater," is the color of the urine, which varies from a deep yellow to a dark red, almost black, according to the stage of the disease. This condition of the urine is known as hæmo-globinuria, which means that the red color of the urine is due to the presence of hæmo-globin, or coloring matter from the blood, and is brought about in the following manner: A micro-parasite, evidently the actual cause of the disease, is introduced through the agency of a species of tick into the animal's body, and gains access to the circulation. Here it finds everything suitable for its requirements. Under these conditions it takes up nourishment, develops, and reproduces its species. It is highly probable and quite possible that these organisms or their products are the means of disintegrating the red blood cells and setting free the hæmo-globin, which is carried by the blood stream to the kidneys, from whence it flows with the urine into the bladder.

Still more convincing evidence of the identity of the Australian disease with Texas fever is found in Director Pound's description of the post-mortem appearances and the pathological changes in the organs, tissues, and constituents of the blood. He writes:

On opening up the abdomen and exposing the internal organs the first striking feature is the enormous enlargement of the spleen which may weigh as much as 7 or 8 pounds, while a healthy spleen will only weigh about 2 or 3 pounds. There is considerable thickening of the outer cellular tissue which sometimes contains a quantity of œdematous fluid. On removing this serous membrane the capsule is seen to be mapped out very decidedly with injected blood vessels. On cutting through the substance of the spleen each surface presents a dark red, almost black, tarry appearance, closely resembling black currant jam, while the structure is so disintegrated and friable that the malpighian corpuscles (the little white specks in a healthy spleen) are hidden from view altogether. In some cases, if the spleen after removal from the animal be held up by one end, the internal structure is so completely broken up that the pulp gravitates quite freely toward the other end.

A microscopical examination will at once reveal that this enlargement and dark-red color of the spleen is brought about by an engorgement of both normal and disintegrated red blood corpuscles.

The liver is always more or less affected, being considerably enlarged, and of a light, yellowish-brown color. It is so extremely friable that unless it is handled with great care the lobes will break or crack in all directions. The bile ducts and gall bladder are very much distended, with dark-greenish contents, often very thick in consistency and somewhat granular.

The kidneys are slightly enlarged, very light in color, and may in some instances show distinct hemorrhages on the outer surfaces. In very advanced cases of the acute type the connective tissue which covers the kidney is found to be in an œdematous condition.

The lungs as a rule are apparently in quite a normal condition; sometimes, however, there may be slight isolated patches of red hepatization, and occasionally emphysema of one or more of the lobes, but chiefly confined to the apices.

On the external surface of the heart little masses of petechiæ may be seen, which are really minute extravasations of blood from the capillaries or smaller blood vessels, while the capillaries of the muscles of the heart are more often injected with red blood cells; the cavities of the heart are usually distended with blood, which is very thin and watery, and has but a very slight tendency to coagulate.

The rumen and recticulum, i. e., the first and second stomachs, respectively, do not exhibit anything peculiar more than that the lining membrane is slightly discolored or pigmented; but the omasum, or third stomach, commonly known by the term manyplies or bible, is frequently very much impacted, i. e., fully distended and very tight and hard, owing to the dry and compressed nature of its contents, while the tendency for the epithelium or lining membrane to peel off is not at all uncommon.

The abomasum, i. e., the fourth or true stomach, has a very decided congested appearance, the mucous membrane being covered with patches of petechiæ; in some instances these petechiæ undergo a kind of granulation.

The small and large intestines are usually in a more or less congested state, with small hemorrhagic patches and pigmentary deposits on the mucous membrane. In very acute cases there may be occasional sloughing of parts of the mucous membrane from the colon and rectum or lower bowel, which come away with the dung or fæces as gelatinous-looking casts.

The bladder, which may have slight petechiæ of the mucous membrane, usually contains urine varying in color from a bright orange to a deep port wine or almost black, and in addition to small quantities of mucus and broken-down blood corpuscles may contain variable quantities of albumen.

In the early stages of the disease the muscular tissue throughout the body is firm, of a bright red color, and has quite a normal looking appearance, but in the advanced stages of the acute type we have a totally different condition, the muscles are very soft and clammy, and of a light pink color, due to the anæmic condition of the animal before death, while the subcutaneous connective and adipose tissues in different parts of the body are intermingled with œdematous-like material and occasionally small extravasations of red blood corpuscles.

In very exceptional cases the fat and connective tissue is of a deep yellow color, which is indicative of jaundice.

As the disease advances the blood becomes very thin and watery, owing principally to the disintegration of the red blood corpuscles and their rapid absorption by the spleen. As this destruction proceeds, the hæmoglobin with the broken-down

cells is carried by the circulation and deposited in the spleen, which gives that organ the characteristic enlarged and dark-colored appearance.

As far as my observations go, this destruction of the red blood corpuscles appears to be due to the action of a micro-organism, analogous in many respects to the organisms found associated with the red blood corpuscles in malarial disease in man, and therefore belongs to the lowest division of the animal kingdom, namely, the Protozoa.

If a small drop of blood is taken from a diseased animal in the acute stage and examined under the microscope with a one-twelfth homogeneous immersion objective and a No. 4 compensating ocular, we find that the red blood cells are quite normal in size and shape, but in the interior of a certain number there are one, two, or several minute clear bodies with a refractive index slightly higher than the surrounding protoplasm of the blood cell, and at first sight might easily be mistaken for bacteria.

In addition to the organism in the blood cells we find a number floating about free in the blood serum, also the remains or fragments of broken-down red blood cells, and in the blood from the spleen irregular-shaped masses of concentrated blood pigment.

The best and most suitable method for studying the morphology or shape of these organisms is to prepare a series of cover-glass specimens and stain them with Löffler's alkaline methylene blue, or still better, Kuhne's carbolized methylene blue, then wash thoroughly in distilled water, dry slowly, and mount permanently in Canada balsam dissolved in xylol. By careful microscopical examination of these specimens the organisms referred to are seen to vary in form in different blood cells; more frequently they appear as minute oval-shaped bodies, usually in pairs, and sometimes singly; in the former condition they resemble diplo-bacteria; others again are club-shaped and appear like the isolated rays of the actinomyces fungus, while a few have a distinct amœboid outline.

The smallest of these organisms, namely, the oval-shaped bodies, scarcely exceeds 0.5 of a micro-millimeter in length, which is about the one fifty-thousandth of an inch, while the largest of the amœboid forms are about 2.5 micro-millimeters, or the one ten-thousandth of an inch.

I have made numerous attempts to isolate this micro-organism from the blood of animals in various stages of the disease, also to cultivate it artificially on nutrient gelatine, agar-agar, beef broth, and other kinds of nutriment media outside the animal body, in order to more accurately study its mode of development and the different phases in its life history, but owing to the peculiar nature and character of the organisms, these cultivation experiments were attended with negative results.

Nevertheless, the results of this investigation appear to show very conclusively that the microorganism is the actual cause of the redwater disease in cattle; moreover, it is readily transmissible from infected to healthy animals through the agency of the cattle tick (*Ixodes bovis*).

The description of the cattle tick found in Australia and its life history would in nearly every detail answer for a description and history of the parasite found on cattle in the southern portion of the United States. It is as follows:

The mature female tick, as seen on an animal's skin, has an oblong, oval-shaped body, resembling the seed of the castor-oil plant, and is of a dull leaden color, due to the blood-red contents of the body showing through the distended semitransparent cuticle. It rarely exceeds 12 millimeters (half an inch) in length and 7 millimeters (a little less than five-sixteenths of an inch) in breadth, and possesses four pairs of legs, situated on the anterior lateral portion of the body. It attaches itself to its host by means of peculiarly constructed mouth organs, collectively known as the rostrum, in the center of which is a barbed dart furnished on either side with several rows of teeth set obliquely, which enables the creature to adhere to the skin more firmly.

The male tick is usually found attached to the skin of its host, immediately underneath the anterior part of the female; its body is of a dark brown color and somewhat triangular in shape, and when full grown is only about one-fiftieth part of the size of the fully developed female, and, never being in an engorged condition, is very much more active and also stronger compared to the female.

As the female tick engorges herself with blood she becomes fecundated by the male, and, having arrived at maturity, releases her hold from the skin by withdrawing the barbed mouth organs and falls to the ground. On recovering herself she walks away to some secluded spot and there lays an enormous number of eggs, which are agglomerated in a dense mass. As oviposition or the process of

egg laying proceeds she gradually gets smaller or shrivels up and ultimately dies beside what will be subsequently her own progeny.

What becomes of the male tick after the female's departure I am not in a position to say. Possibly he wanders through the hairs of the animal's skin until he mates with another female, but it is more than probable that he only fertilizes one female during his lifetime.

During my stay at the Normanton Meat Works I was able to make some observations and study part of the life history of the tick, which is so intimately associated with the redwater disease. I procured a number of fully developed mature female ticks, which had recently fallen off from an infected animal, and placed them separately in a series of small Petrie's glass capsules with vertical sides; the inside of each cover contained a small circle of filter paper, which was kept moistened with water every few days. This I found by experience was necessary in order to keep the atmosphere within the vessels in a state of humidity, so essential for the well being of the tick.

Under these conditions the tick invariably remains in a quiescent state for about eight or ten days, during which time the contents of the abdomen are undergoing important changes prior to oviposition. The period of oviposition generally takes from seven to fourteen days, and the number of eggs laid varies according to the size of the tick. As a rule each fully matured tick is capable of laying from 1,600 to just over 2,000 eggs. A noticeable feature during this process is the appearance on the back of the tick of a bright yellow spot, very irregular in outline, which gradually gets larger as oviposition proceeds. This yellow spot is caused by the lessening in size or shrinking of the ovaries, which leaves a kind of vacuole or space between them and the cuticle, thus giving the creature an entirely different aspect, and which sometimes results in its being mistaken for one of a totally different species.

The ovipositor, namely, the little tube down which the egg passes from the oviduct after leaving the ovaries, is situated close to the mouth, midway between the first pair of legs, which at this stage are turned in an upward direction and kept continually moving about in the air like a pair of antennæ or feelers, which the creature does not possess.

As each egg leaves the ovipositor it is taken by the palpi or outer parts of the mouth and covered with some kind of secretion of an albuminous nature which cements the eggs together in a dense mass. The eggs are bluntly oval in shape and of a light brownish red color, and may vary in size according to the age of the tick; for instance, several ticks were removed from the skin of an animal before they were half grown, yet capable of laying eggs, which were pale in color and mostly very small compared with normal eggs; moreover, with few exceptions, instead of hatching they collapsed and eventually withered away, from which I concluded the female tick had been imperfectly fecundated. The period of incubation in a temperature ranging between 90° and 110° F. was from fourteen to twenty-six days after the last egg was laid, but when the temperature was lower the incubation period was correspondingly longer.

The various changes which the egg undergoes during incubation can be watched under the microscope with a 1-inch objective, the outer membrane or shell remaining almost transparent till the young tick is fully developed, when, within four to six days before hatching, the shell gradually becomes opaque, looking like a small pearl. When the young tick emerges from the shell it has only six legs, and is of a light brown color, but gradually assumes a much darker shade. In this state the young ticks are extremely active in their movements, continually walking and running up and down the sides of the glass vessel, and leaving behind them little white amorphous deposits of excreta.

Eventually they settle down and collect in large numbers immediately under the glass cover, but should by any chance the cover be knocked or removed they commence to scatter in all directions.

They are extremely tenacious of life when kept in confinement during this larval stage, for I have at the present time in my possession a number kept confined in well-stopped bottles that were hatched over seventeen weeks ago and now still active, although neither food, water, nor fresh air has been supplied to them. Moreover, I have not observed any further alteration in their anatomical structure. Probably no further development takes place until they gain access and adhere to some susceptible animal, when, by taking sufficient nourishment for their requirements, they undergo another change or molt, and finally each one appears with its extra pair of legs as a perfectly formed male or female, which, by gradual and continual sucking the blood or juices from the body of its hosts, ultimately arrives at maturity, thus completing its metamorphosis or life history.

As a rule, nearly every animal affected with redwater is more or less infested with ticks. In some instances they are present in extraordinary numbers, being

either distributed equally over the whole surface of the animal's body or restricted to certain parts. Sometimes only a very few are present, while in very rare and exceptional cases they are absent altogether, although they were present originally.

Generally speaking, they prefer to live on those parts of the body where the skin is very soft and thin, as, for instance, along the belly, on and around the udder in the cow or scrotum in the bull or bullock, and on either side of the neck, which is inaccessible to the tongue, limbs, or tail of their host.

Both male and female ticks live on the same animal. The female tick gradually increases in size by continually sucking the blood, which, as we have already seen, contains, both in and outside the red corpuscles, the specific micro-organisms.

Having arrived at maturity, after being fecundated by the male tick she leaves the animal and deposits her eggs upon the ground. In course of time the eggs hatch and the young ticks go roaming about in all directions and are ever ready to attach themselves to the skin of any suitable animal that may happen to be standing or lying down in the vicinity.

It seems almost impossible to think that, although the adult female tick may have taken the micro-organisms into her system with the blood, she should transmit them through the egg to her progeny, but is nevertheless a substantiated fact that these young ticks are capable of communicating the disease under favorable conditions to healthy cattle, consequently it appears that the micro-organism which is the cause of redwater must be constantly associated with the tick throughout its life history from the mature female to the egg, thence to the young tick, which in turn conveys it to the healthy susceptible animal, but it must be in an entirely different form from that which we see in the blood of diseased animals; for I have repeatedly prepared coverglass specimens of the contents of the bodies of ticks in various stages of development, also the contents of the ova or eggs, and exercised special care in staining them with various anilin dyes and mounting them in different media; but when such preparations were submitted to a careful and crucial microscopical examination I failed to find any bodies that could be identified with the micro-organisms present in the blood of cattle suffering from redwater disease.

Below is appended Director Pound's account of some of his observations of the disease in the course of his investigations and his recommendations for its control:

The Dalgonally Meat Works are situated on one side of the Norman River, and the township of Normanton, about 2 miles away on the opposite side, and all traveling mobs of cattle for the meat works have to cross the river about 14 miles above Normanton. This leaves a tract of country free fully 12 miles square for some of the residents of the township to keep small herds of cattle, and, owing to the winding river on one side and a stretch of fairly thick scrub on the other, there is but very little chance under ordinary conditions of the animals becoming infected with redwater disease from outside this boundary.

However, shortly before I left Normanton, Mr. David Downes, a dairyman residing just outside Normanton and owning about 200 head of cattle, informed me that certain unscrupulous persons had been circulating information to the effect that his cows were infested with ticks and suffering from redwater, which statement, he assured me, was quite erroneous, and that unless this could be contradicted by some competent person giving him a certificate which stated that his cattle were perfectly healthy, which he again assured me they were, his milk trade would be very seriously injured.

I may say that previous to this I had on several occasions seen these cattle, and as far as I was able to judge from naked-eye appearances they were perfectly healthy and free from ticks.

I therefore made arrangements with Mr. Downes that I should accompany the mayor of Normanton to inspect the cattle. On arrival at the place where the animals were feeding I noticed a number of them were in an emaciated condition and infested with ticks, and while Mr. Downes was rounding up the mob for my inspection I observed that some of the animals, on being agitated, passed urine of a very high color, and in one instance it presented a bright-red appearance; moreover, they had great difficulty in defæcating, or passing their dung, which appeared as hard, round, and almost black-colored balls. Some of the animals had also a very decided staggering gait.

These various symptoms clearly indicated that the redwater disease was unmistakably present in the herd, although up to this time no fatal cases had been recorded.

I inquired of Mr. Downes if any fresh animals had been introduced from an

infected herd, and he said decidedly not, but informed me that he had recently purchased two or three healthy young bulls in order to improve his stock. I next inquired from whom they were purchased, and how much was paid for them. He said he bought them from a local commission agent, and they were very cheap, only costing a one-pound note.

On making further inquiries in Normanton I found that these young bulls had been traveling for some considerable distance with a mob of cattle from Fort Constantine which were infested with ticks, and nearly every one badly affected with redwater; in fact, out of about 650 head of cattle which left the station nearly 200 died on the road within 80 miles of the meat works.

Previous to purchasing the bulls Mr. Downes was warned by several people not to do so, as they were unhealthy looking and infested with ticks. Nevertheless he refused to accept any advice in the matter, with the result that these bulls, which were evidently suffering from redwater and infested with ticks, were the means of introducing the disease into a healthy herd and communicated from one animal to another through the agency of ticks.

On my way to Normanton I had an opportunity of interviewing the Hon. John Douglas, Government resident at Thursday Island, who informed me that up till within a short time ago such things as ticks were practically unheard of on the island; moreover, the few cows kept for milking purposes had never shown any signs of sickness until after the ticks made their appearance on the animals. These ticks were introduced from Normanton with a consignment of bullocks which were affected with redwater, several of them having died during the voyage. The remainder of the affected animals, which were to be killed for beef, were kept in a yard till required, and therefore were never actually allowed to come in contact with the milking cows. Nevertheless the latter became infested with ticks, and eventually every animal developed symptoms of the disease and subsequently died. Further, I am informed that now the disease has become established and is of such a virulent and fatal type that all ideas of keeping milking cows on the island have been abandoned.

It will thus be seen from the foregoing remarks that the evidence is strongly in favor of the tick being the communicating agent or medium for conveying the disease from infected to healthy animals. This is further substantiated by the fact that if healthy susceptible cattle be brought into contact or even kept with animals suffering from redwater, but which are entirely free from ticks, they never acquire the disease, although it seems quite possible for the disease to be communicated by agencies other than ticks, namely, the various bush flies and mosquitoes; but according to our present state of knowledge this does not appear to be the case.

Redwater being a disease peculiar to cattle only, there is, practically speaking, no possibility whatever of its being communicated to man; but at the same time, although the milk or meat of animals suffering from the disease in the early stage may be quite harmless, the fact must not be overlooked that such milk and meat from animals in the more advanced and acute form of the disease is, from a hygienic or health point of view, decidedly unwholesome, and therefore should on no account be used as food for human consumption.

Owing to the peculiar nature and character of the country the quarantining of the affected area can only be conducted in a more or less imperfect manner, for, although traveling cattle are not supposed to pass out of the district unless they are healthy, we occasionally hear reports that traveling mobs of cattle are suffering from redwater outside the boundary. Probably these animals had crossed the boundary just before the affected area had been quarantined. However, in the future I would especially recommend that all traveling stock be carefully inspected in a systematic manner in order to see that the animals are healthy and free from ticks. The latter should be especially sought after, otherwise, if this is not carried into effect, we may expect to hear of serious outbreaks of the disease among station cattle in the central districts.

We have seen in the foregoing remarks, based upon practical observations, that the ticks are the communicating agents or medium for conveying the disease from infected to healthy animals, which means, in other words, that when the ticks are absent the disease, although it may not disappear entirely, has a less tendency to spread, which is a very great consideration. Therefore, every stock owner within these infected districts should divert his attention in every possible direction in endeavoring to exterminate this tick pest.

Considering the wild nature of the cattle on these large northern stations, it would be out of the question to recommend washing all the animals infested with ticks with any of the well-known sheep dips, but at the same time the method would be applicable among pedigree or stud cattle.

In my opinion, the most satisfactory and effectual way of dealing with the ticks

is to burn off the grass on the whole of the run, which would not only eliminate numbers of ticks, but destroy other parasites which spend part of their life history on the ground.

In resorting to the last method it is needless to say that only parts of the run should be fired at one time.

When the disease makes its appearance among either station or traveling stock the sick animals should on no account be overdriven or excited, for the condition of the circulation of the blood is such that any effort may bring about rupture of the blood vessels and lead to a speedy death.

The exposure of the cattle to the sun's rays during the middle of the day in summer time is liable to increase the already abnormally high temperature and thus favor the development and progress of the disease; therefore, in order to obviate this, it is recommended that the cattle be allowed to camp in some shady scrub during the hottest part of the day.

One very great difficulty in coping with this disease among traveling stock is the inability to destroy dead animals by fire owing to the scarcity of timber in the locality; but when cattle die on traveling-stock routes where there is a fair supply of wood it becomes the duty of every drover to at once destroy by fire all dead animals, and station owners who have stock routes through their runs should, whenever practicable, see that this is carried into effect.

Later reports from Australia intimate that a difficulty which has heretofore baffled scientists in their investigations into the cause of Texas fever has been solved. The perplexing question has been, "Whence comes the microorganism that is invariably found in the blood of tick-infested cattle?" The natural supposition was that it was introduced into the blood by the tick itself. If that were true, a deduction equally natural was that this same microorganism should in some stage of development be found in or upon the body of the young tick just previously to or simultaneously with its attaching itself to the host. But in the American investigations careful microscopical examinations failed to reveal either in or upon the body of the tick protozoa which could be identified with those found in the blood of infected cattle. This failure was perplexing, and it was difficult to explain the manner in which the young tick carried the infection. This difficulty would be largely overcome if an Australian scientist has, as he claims, found a microorganism in the body of the unattached tick identical with that found in the blood of infected cattle.

After the issuance of Director Pound's report further investigations were carried on at Hughenden under the direction of Dr. Hunt. On the 28th of November, 1895, he made a claim of the discovery referred to above in a report to the colonial secretary of Queensland. The fact of especial interest in it is set forth as follows:

> In your letter of the 30th of September (page 3) you said: "One thing strikes me as of importance, and yet absent, in the American experiments. There seems to be no evidence to connect the microparasite in the blood of diseased cattle with a similar organism in the tick." This point seems to me to have been hitherto rather a stumbling block, so I am sure you will be interested to know that I have been able to find in young ticks hatched in the laboratory the identical amœboid bodies I have so often in my letters referred to as being present in the blood of cattle affected with ticks. The method of demonstrating these bodies is so simple that anyone accustomed to use the microscope can do it in about three minutes, thus: Onto a clean slide place a drop of boiled or distilled water containing one-half per cent common salt. In this droplet place a young tick. Lower a very thin cover glass onto it. Squash the young tick by pressing down the cover glass. Brush round the edges of the cover glass with melted hard paraffin (which will prevent evaporation and render the specimen fit for examination for many hours). Examine with one-twelfth oil immersion lens. The amœboid bodies will be seen moving in the saline solution in just the same way as they are in the blood of tick-affected cattle. As I have before mentioned, they vary from one-tenth or less to one-third or more the size of a bovine red-blood corpuscle. Though I have called their movements "amœboid," that is not exactly the right description. They have

the kind of appearance that a bright 3-penny piece has when it is sinking in deep, clear water. It is better to squash two or three young ticks at a time, as they appear to be more abundant in some than in others. In some of the little bodies a dark central spot can be made out, as is also the case when they occur in the blood of affected cattle. In and around the young tick, squashed in the saline fluid as above described, may be seen much larger cells—as large, or in some cases larger, than bovine red-blood corpuscles. They are round, have often a central spot and are generally motionless except for a slight movement of their edges, sometimes seen in the smaller of them. They appear also to have a kind of concentric structure, i. e., as if they had concentric laminæ, but I am not quite sure of this. In young ticks hatched in laboratory these large cells are of a faint straw tint; in ticks gorged with the blood of cattle they are very dark, reddish brown. My idea is that this dark coloration is due to hæmo-globin, appropriated by these cells from the blood of the beast to which the tick has adhered. These are very possibly a further stage of the amœboid bodies referred to. The discovery of these bodies in young ticks seems to me to almost complete the chain of evidence connecting the ticks with the disease in cattle unfortunately and absurdly called "redwater."

1. The disease is associated with the presence of ticks (and never unassociated?).
2. In diseased animals' blood there is found a peculiar motile parasite.
3. In young ticks that have never been in contact with a beast the same peculiar motile parasite is present.*
4. It is reasonable to suppose the parasite is the cause of the disease.

I have found the parasites in question in young ticks whose parents were hatched in the laboratory and planted on a young calf and collected at maturity, and whose grandparents were originally taken from cattle among which the "tick fever" was present. I am now trying whether by injecting a solution containing the amœboid parasites into a healthy beast I can produce symptoms of "tick fever." Unfortunately for diagnostic purposes, fever seems, in the mild forms, to be often absent or very slight. The only certain diagnostic sign is the discovery of the amœboid parasites in the beasts' blood.

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*Owing to the small size, the lack of structural peculiarities, and the inconstant form, the identification of this parasite elsewhere than in the blood corpuscles must always be uncertain. There is no doubt that it causes the disease, and from our present knowledge we should expect to find it in the egg and young tick. The experts of this Bureau have not identified it, however, in these situations by microscopical investigation, which is not remarkable considering the practical impossibility of identification in that manner; but, what is more significant, they have failed in a preliminary experiment to produce the disease by inoculating animals with a suspension of the young ticks ground up in a mortar and injected hypodermically.—D. E. S.

THE CURING OF MEATS.

An inquiry concerning the famous Smithfield hams was recently received by the Department of Agriculture, and the information desired was deemed of sufficient importance to warrant the sending of a special inspector to make an investigation as to the especial merits of this product, the process of manufacture, and the causes from which its excellence is derived. The report of the inspector is given in full below.

The celebrated "Smithfield ham" gets its name from the little town of Smithfield, which is located on Pagan Creek, about 30 miles from Norfolk, Va. About one hundred years ago a gentleman by the name of Todd began to cure the meat, i. e., hams and bacon, from the almost wild hogs that were allowed to run semi-wild in the extensive forests surrounding Smithfield.

The fame of the hams thus produced spread; consequently the business of producing hams extended. The business has descended from one generation to another, and has been perfected until the only rival to the Smithfield ham is the celebrated Westphalia ham, which is considered by many to be inferior to this product.

There are numerous producers of the genuine Smithfield hams and they have many imitators. The principal packer of these hams is Mr. E. M. Todd. He puts up about 12,000 hams annually. Mr. J. O. Thomas is probably the next largest packer.

Unlike Mr. Todd, Mr. Thomas raises a good many of the hogs he uses. He has a farm of about 2,500 acres. A large part of this is heavy woodland, and in these woods his hogs run almost wild. All the farmers raise hogs to some extent. Most of them sell their hams "green," that is, uncured, to the "packers," who in most cases are farmers upon a larger scale. The hams thus bought are very carefully selected, all those not coming up to a certain required standard being rejected. The standard price paid last fall for green hams was 14 cents per pound, delivered at the smokehouses.

There are several things necessary in order to produce a Smithfield ham.

(1) *The kind of hog used.*—It is impossible to make a good ham from a Western hog.

By that I mean a ham with the peculiar qualities of the Smithfield. The demand for these hams has grown to such an extent that some of the packers have been tempted to try hams from other parts of the country. Mr. Todd, and also Mr. Thomas, purchased some of the Western hams, and, by curing them with care, tried to make them like those grown near Smithfield, but they failed to give satisfaction. They even tried to purchase the live hogs and kill them themselves, but then they were not like the home-grown animal. The hog from which the ham is produced is the unimproved, half-wild "razorback" hog that is peculiar to the mountainous portions of Virginia, Kentucky, and Tennessee. I asked this question of numerous farmers and packers:

"Why do you not use the improved breeds of hogs at least for crossing with your "razorbacks" and get an animal with less nose, shaped more like a hog and less like a race horse?" The unvarying answer was, "We have tried it and nearly spoiled our hams. The grain is too coarse and the shape of the ham is not the same. We want the long-nosed, slab-sided, long-legged rooter." These hogs appear to be nondescript in breeding; they have extremely long noses, are very thin-sided, deep-chested, with small flanks and extremely long, sloping hams.

They show the attempts that have been made to improve the breed by the variety of colors and marks seen among them. Many of them are black with no white. Some are white. The majority are spotted black and white, while not a few show black, white, and red mixed irregularly, and many are a peculiar iron gray in color.

(2) *The manner in which the hogs are fed.*—I visited a number of farmers who are successful producers of these hams and from them I find a singular similarity in methods. The sows run at large in the woods and pig about the first to the middle of April. From the manner in which the hogs are kept, however, the time of pigging is not easily controlled. The pigs run during the summer months in the woods, living upon the nuts and roots which they can secure. They are not fed anything to make them fatten. Most farmers give about one ear of corn night and morning to each hog in order to "keep them to the call." In the fall when the corn crop has been gathered the hogs are turned into the cornfields. In these fields every other row has been planted to "black-eyed" peas, and the hogs are allowed to gather these and the small corn that has been left in the field. When turned into these fields they are very thin. The feed they get there causes them to begin to fatten rapidly. As the potatoes are gathered, the hogs are allowed to follow in these fields and get those that are left. In that district which produces the most Smithfield hams there are a great many sweet potatoes and peanuts raised, and the hogs are allowed free access to these fields as soon as the crops are gathered.

This method of feeding fattens very rapidly. The potatoes, and particularly the peanuts, add fat with astonishing rapidity, but the fat is very soft. Peanut fat in particular has a translucent, oily character, which, from its tendency to drip when the hams are hung up, causes a great shrinkage in the weight.

After the fields have been cleaned up in this manner and before the hogs can "go back" any in their fattening, they are taken out of the fields and put into close pens and fed on corn and clean water. The pens are made dry with good shelters on the northern exposures and a chance for plenty of sunlight. The hogs are plentifully bedded with pine straw gathered from the woods and fed all the corn they will eat and given all the clear water they want. When the hog is penned his days for wallowing are over. From this time until slaughtered he lives a live of enforced cleanliness. The hogs are kept in this feeding pen until just the right condition is produced. They must not be too fat nor too lean. Many of the larger hog raisers inform me that they frequently have several killings from the same fattening pen, some getting into condition sooner than others, and as they do, being culled out and slaughtered, the rest remaining for a longer time until just the right condition has been attained. All were positive in the assertion that "swill feed" spoils the hams, and that corn was a necessary article to finish the product in order to produce the best hams, while all were equally positive that it was impossible for them to buy hogs from the West—by that meaning anywhere away from their immediate vicinity—and produce the same quality of meat, even if they kept them long enough to fatten them themselves. These hogs when fat weigh about 125 to 190 pounds as the extreme limits; larger hogs are not considered desirable.

(3) After fattening, the hogs are carefully slaughtered. In this, as in all the other processes, great care is used. The animals are carefully handled to prevent bruising, and are carefully bled. The most particular part of the killing process is cutting up the meat. The hams are the first consideration. They are very carefully cut out, care being taken to trim them so as to leave the tissues in the ham proper intact. The "hock" joint is left long. The hams are not cut close, care being taken to leave all the connective tissues possible at the "upper" end to prevent as much as possible the hams from dripping when hung in the smokehouse.

(4) The next important process is curing. Mr. Todd, who cures more than any other one man in Virginia, kindly gave me his whole process. His method is the same as used generally in the South, which is noted for its production of superior pork. In an article on "Something about hams" in Leslie's Illustrated Weekly, August 16, 1894, Philip Poindexter gives Mr. Todd's method in full and Mr. Todd assures me it is correct. This method is the one employed by all the producers of Smithfield hams. It is as follows:

1. The hams are placed in a large tray of fine Liverpool salt. Then the flesh surface is sprinkled with finely ground crude saltpeter until the hams are as white as though covered by a moderate frost; or, say, use 3 to 4 pounds of the powdered saltpeter to 1,000 pounds of hams.

2. After applying the saltpeter immediately salt with the Liverpool fine salt, covering well the entire surface. Now pack the hams in bulk, but not in piles more than 3 feet high. In ordinary weather the hams should remain thus for three days.

3. Then break bulk, and resalt with the fine salt. The hams thus salted and resalted should now remain in salt in bulk one day for each and every pound each ham weighs; that is, a 2-pound ham should remain two days, and in such proportion of time for larger and smaller sizes.

4. Next, you wash with tepid water until the hams are thoroughly cleaned, and after partially drying rub the entire surface with finely ground black pepper.

5. Now the hams should be hung in the smokehouse and this important operation be begun. The smoking should be very gradually and slowly done, lasting thirty to forty days (most packers using green hickory or red oak to smoke with).

6. After the hams are cured and smoked they should be repeppered to guard against vermin and then bagged. These hams improve with age, and may be considered perfect at about one year old.

The conclusion naturally to be drawn from these facts is that any ham treated as these packers treat theirs would be better than the average. The Smithfield ham, however, owes its popularity to its peculiar flavor, and this flavor is not due to the manner in which it is cured, else any ham cured in the same manner would rival it. It does not owe its flavor to the manner of feeding altogether, else hogs from other parts of the country could be brought here and perfected. The necessary things in the opinion of the producers may then be summed up as follows:

(1) A slow-growing, peculiar-shaped hog.

(2) A peculiar game flavor produced by the wild life in the woods and the nuts, berries, etc., upon which it lives.

(3) Rapid production of flesh when the fattening process begins. The fat formed of corn and pure water.

(4) The method of curing and smoking.

All these points seem necessary to the production of these hams.

It would be of interest to know if the hogs produced in southern Kentucky and parts of Tennessee would not produce these hams. The hogs in many parts of these States are raised in the same manner, except in the process of fattening, and to all appearances are the same hog.

Undoubtedly many hams are sold as Smithfield hams which are produced in other parts of the country, and which are inferior in many respects to the genuine article. The supply is limited and the demand is great. Very few of those produced are sold to the trade. Mr. Todd is the principal packer. He buys his hams whenever he can get those that are suitable. He assures me that he can not get hams outside of four counties in Virginia that will answer his purpose at all. Isle of Wight, Surry, Southampton, and a portion of Nansemond counties, Virginia, produce all the hams that are the genuine Smithfield.

Mr. Todd	about	12,000
Mr. J. O. Thomas	do	2,500
Mr. M. F. Loughron	do	2,000
Capt. O. G. Delk	do	1,000
Mr. Chapman	do	1,500
Other local dealers	do	1,000
A total of about		20,000

sold from Smithfield and vicinity.

There are probably one-third more sold by various farmers who cure them in a small way, making a total of about 30,000 hams which are produced annually, and which are the genuine Smithfield hams.

These hams average when cured about 10 pounds in weight; some are a little heavier, while perhaps the greater number weigh rather less. At 10 pounds the total output would be 300,000 pounds.

They sell in the smokehouses at about an average price of 20 cents per pound, some packers, like Mr. Thomas, getting an average of 22 cents for their whole product. This makes an annual income of about $60,000 from hams alone to be distributed among the farmers of Isle of Wight, Surry, Southampton, and Nansemond counties. This territory could undoubtedly raise many more hogs.

The output could be doubled many times were the farmers not afraid of losses from hog cholera. As it is, most of the farmers raise a limited number of hogs, knowing that they have a ready sale for their hams either cured or green, and have their other crops "to fall back on" should hog cholera break out. I found the largest number raised was about 200 head, while almost every farmer has a few. Mr. Todd sells his hams on orders received before the crop is cured. He supplies a limited trade in the principal cities of this country and sends some to England and France.

Mr. Thomas sells his whole output on private orders, selling none to the trade. His principal market is family trade in Boston, New York, Philadelphia, Washington, D. C., San Francisco, and he receives orders from private parties in Europe and the Sandwich Islands.

Many of the smaller producers sell their hams in this manner, while some others trade theirs at the stores for groceries, or sell in small lots to commission houses in various near-by cities.

The rest of the meat, viz, the side pork and shoulders, is in most instances cured in exactly the same manner as the hams, and used by the farmers to feed their field hands, or to furnish breakfast bacon for the local trade. The sides make very desirable bacon that is much sought after among the negroes and in the small towns. The farmers who sell their hams use most of the side meat to feed the families of their farm hands, although some of it is sold in the markets. The breakfast bacon from these hogs is peculiarly sweet and is much sought after by many. It is impossible to say how many parties are engaged in the production of these hams without taking a census of the farmers in the counties referred to, but the principal packers are the ones named.

[In the above report, full details have been given, including the names of the packers, in view of the limited and comparatively local trade affected and of the value of presenting for the information of pork breeders generally some of the nice points involved in the production of superior and high-priced pork.]

Other inquiries in regard to the processes in use in the United States of curing meats, particularly hams, suggested the utility of extending like inquiries to other places. Letters were addressed to inspectors of the Bureau of Animal Industry at the principal packing houses of the country, asking them to obtain for comparison the formula used by the packers in preparing such products for the market.

In a comprehensive report concerning the curing of hog products in a Western packing plant the feature of especial interest embodied is a description of the process of pickling by injection.

The curing of the meat commences when fresh-slaughtered swine are placed in a chill room, the temperature of which is 40° F. at the start, gradually falling to 28° F. thirty-six hours later.

After remaining in this condition forty-eight hours, the carcasses are removed to an adjoining room, where they are cut into such sizes and shapes as the quality of the meat or the demands of the market require. From the cutting bench the meat passes to the curing rooms below, where it is then exposed to the action of the following-described pickle:

Formula for curing hams, shoulders, bacon, dry salt meat, and mess pork.

	Parts.
Chloride of sodium	78
Saccharine	28
Nitrate of potash	20

Use of pure water a sufficient quantity to make a 78 per cent solution.

Hams and shoulders are first injected with this solution. The process is as follows: A hollow needle, 10 inches long, to which is attached a rubber tube connecting with a small vat containing the pickle, is inserted three or four times in different parts of the ham. At the same time a lever is lowered which forces the liquid through the needle into the depths of the ham. The ham is then placed in the pickle, where it remains for ten days. It is then removed and the injecting process repeated.

Large hams weighing 20 pounds or more may require a third injection. Two injections and sixty-four days in large vats, submerged in the above pickle, completes the cure with the exception of smoking.

This consists in placing the meat in a close, dark room, where it is suspended from the ceiling. It is then subjected to a dense smoke

caused by the destruction of hickory or ash wood by heat for twenty-four to forty-eight hours, the former being considered a mild cure.

Bacon is first injected with the pickle in the same manner as the hams, after which it is placed in large vats and entirely submerged in the pickle. At the expiration of ten days the injecting process is repeated, when the meat is again returned to the vats. In about sixty days the curing is completed, with the exception of the smoking, which is conducted in the same manner as with the hams.

Dry salt pork is first injected with the pickle in the same manner as the other-mentioned products. After receiving the injection it is merely dipped in the solution. While still damp it is thoroughly hand rubbed with salt. It is then laid on the floors of the cellar, where it lies for ten days, at the end of which time the injection is repeated, as well as the dipping and hand rubbing with salt. In heavy meat the third operation is necessary to thoroughly cure the meat, which is accomplished in ninety days.

Mess pork is cut into convenient sizes and pieces and packed firmly in barrels made for the purpose. The pickle used in the other kinds of meat above described is then poured into the barrel until it is filled, when it is headed.

In sixty to ninety days the curing is completed. The difference in time required is due to the thickness of the meat.

The following formula is the one in use by a large New England packing house:

Hogs to be well cooled off before cutting. Hang forty-eight hours in 35° temperature. For a tierce, 300 pounds of hams, 18 pounds of salt, 1 pound saltpeter. 7 pounds sugar. Fill the tierce with water. Keep in a temperature of 38° to 40°, Overhaul twice a week, or oftener, if case requires, for four weeks. Will be ready to smoke in sixty to eighty days, as to size.

Hams from well-cooled hogs, use 8 pounds salt, one-fourth of a pound saltpeter. 2 pounds sugar for 100 pounds meat.

Medium-sized hams will be cured enough to smoke in forty-five days if kept in a temperature of 38° to 40°.

Incidental to a description of the method of curing meats both by the "dry-salt" and the "pickle" cure, another report explains the process of the manufacture of "California," "boneless," and "cottage" hams:

The hogs after being slaughtered are left in the cooling room about half an hour. The carcasses are then put into a room whose temperature is about 44° and left there for about six hours or until the animal heat has left them. They are next transferred to another room, the temperature of which is 32°, and left there for twenty-four hours. From here they are taken to the cutting room and cut into special pieces, the quality of the hog and supposed condition of future trade (when cured) governing the kind of cuts made out of the carcasses. For instance, three kinds of so-called hams are made from shoulders, viz, California, cottage, and boneless hams. They are made as follows: The shoulder is cut in two and the upper part is cut round to resemble a ham. This is called a California ham. The lower part, with the leg cut close off and part of the shoulder left in, is called a cottage ham, and when the shoulder blade is taken out it is called a boneless ham.

Then we have the genuine hams made out of the hind quarters. They are called long-cut hams when the shank is left pretty long, and short-cut when left short. They are divided into three grades—light, medium, and heavy: They are cured by two methods, viz, first, dry-salt cure; second, pickle. It appears that by the latter method the packer gets more weight out of his product.

Dry-salt cure.—About half an ounce of saltpeter (nitrate of potash), half an ounce of sugar, and 1½ pounds of common salt to each ham, rubbed in. They are

then put in the cellar and left from five to seven days, when they are turned over and left for about twenty-eight days. They are then cured. Heavy hams and summer weather will take a few days longer.

Cured in pickle.—The strongest pickle registers 24° by the pickle tester. You can not make it any stronger, as the water will not dissolve any more of the ingredients. What is called mild cure registers about 16°. It is made as follows: Eight ounces of saltpeter, 3½ pounds of sugar, 21 pounds of salt, and 12 gallons of water. This is sufficient for 1 tierce. The tierces contain 280 pounds of ham. They are rolled around every day for fifteen days to keep the pickle from settling, and the hams are cured in from thirty to thirty-five days. If not called for before ninety days, the pickle is drawn off, the hams are taken out of the barrels, piled in cold storage, and a sprinkling of borax thrown on them. If they are to be shipped, fresh pickle is put on them, and it is generally of 20° strength.

The other cuts, such as bellies, sides, backs, and shoulders, are cured in the same way, excepting that the sugar is generally left out of the formula. Saltpeter is left out in curing what is called a French back. Dry-salt-cured meats when shipped are sometimes packed in borax and sometimes in salt.

Smoked meats.—After being cured they are put in a vat and washed. Then they are put in a smokehouse and smoked with hickory wood from thirty-six to forty hours. Sometimes they are shipped in bulk, sometimes packed in boxes. Sometimes they are sewed up in a covering with a gaudy brand on it and sold to the trade.

The report from another abattoir in the same locality shows only a slight difference in the treatment of the hog products:

The hogs are left about six hours in the cooling room, as there is no intermediate room. The chill room is always down to 32°, so the animal heat must be allowed to leave the carcasses before putting them into so cold a place, or they would freeze on the outside and spoil in the center. When any meats begin to get old, borax is used very liberally, and when dry-salt-cured meats are shipped they are very often packed in borax.

A statement of the methods which have been adopted by another packing company in curing various kinds of meats is furnished in the following:

Barreled beef.—Chilled at 40°; carried in brine pickle 70° strength, using 2½ ounces saltpeter to 100 pounds beef. This is used for all such things as mess beef, plate beef, etc.

Beef hams.—Chilling temperature 40°. After chilling carry in 70° strength brine pickle; use 3½ ounces saltpeter to 100 pounds beef; 2 pints of sirup to 220 pounds beef.

Beef, hog, and sheep tongues.—Use 70° strength brine pickle; 3 ounces saltpeter to 100 pounds tongues; cure in temperature of 40° to 45°.

Pork hams—To 100 pounds hams, 2 pounds 11 ounces best granulated sugar, 3 ounces saltpeter, 4 ounces borax, sufficient salt to raise 5½ gallons of water to 75° strength. Salt, sugar, etc., are well dissolved before mixing, and filtered into the pickle.

When chilling hogs, hold coolers at 32° to 34° temperature; leave hogs in coolers forty to seventy-two hours; after cutting, rack the hams in a room at 32° to 34° for twenty-four hours, after which pack in above pickle. At the age of 60 days the pickle shows a strength of 53° to 54°.

Dry-salt meats.—Hogs are chilled as stated under the heading of "Hams." After being cut the salt meats are piled up in a dry salt with a very light sprinkle of saltpeter. They are overhauled at the end of five, fifteen, and thirty days. During curing they are kept in a temperature of 40°.

Special reference to a difference in treatment of meats designed for foreign or domestic trade is made in the system of curing given below:

After slaughter the carcasses are allowed to hang in a cooling room for about forty-eight hours before being cut up, and as each piece is trimmed it is immediately consigned to the cellar. Those pieces destined for the dry-salt cure are well rubbed with salt and packed on the floor in layers six to twelve high. In about ten days more they receive another rubbing of salt and are packed as before, and again in fifteen days the process is repeated, after which they are allowed to stand in bulk until they are cured, which takes about forty-five days.

To English meat a little saltpeter is added.

For pickling hams the following is the formula, the quantities named being sufficient to cure 280 pounds of meat, and its strength as indicated by the salimeter is 80°:

	Pounds.
English salt	21
Saltpeter	1
Sugar	4

Water sufficient to fill the tierce in which the meat is placed.

The tierce is then closed, and for the first three or four days it is rolled around daily three or four times to insure an equal distribution of the pickle. Every three or four days thereafter the process is repeated, the time required for curing varying, according to the size of the hams, from forty-five to eighty days.

At this stage the meat can be supplied to the trade, but for certain kinds of trade, especially the domestic, it has to undergo the process of smoking for from twenty-four to thirty-six hours. Breakfast bacon and other pickled meats are treated in a similar manner and require from twenty to forty days in curing.

Two processes in use at one packing house, denominated the "dry-salt cure" and the "sweet-pickle cure," are thus outlined:

There are two methods of curing, one called the "dry-salt cure" and the other the "pickle cure." In the former, after the meat has been sufficiently cooled so as to get out the animal heat, which generally takes from twenty-four to forty-eight hours, according to the season of the year, the meats are well rubbed in salt and bulked on the floor, usually from six to ten high, and resalted again when they are about from three to five days old and left lie until they are about twenty days old, when they are resalted once more, being bulked higher each salting, so as to not take up too much room, and left in the bulk until it is fully cured, which usually takes about from forty-five to fifty-five days, according to the size of the meat. The extremely heavy meat would of course take sixty days in order to get it fully cured. We use nothing but Kansas salt in the dry-salt cure, except a small amount of saltpeter for the shoulders.

For the sweet-pickle cure the meat is handled in the same manner, except that instead of being cured in bulk it is cured either in large vats or tierces. It takes about the same length of time to cure in pickle that it does in dry salt. The sweet pickle is a brine which is made from Kansas salt, with a small portion of saltpeter and borax added and a sufficient quantity of sirup to give it a sweet flavor.

A brief sketch of the operations and curing process of a great packing house reads:

After the hogs are killed they are run on rails into a chill room, usually at a temperature of 38° to 40°, for about twenty-four hours. Next day the temperature of the chill room is reduced to 32° and the hogs are left there for twenty-four hours more; that is, forty-eight hours before being cut up into the various cuts as the trade demands. After the hams are chopped or sawed off the side of hogs they are trimmed and dropped down a chute into the curing room, where they are sized and sorted according to quality of six different grades. They are then trucked out into a spreading room, 32° temperature, where hams or shoulders are each separately spread on latticed racks or hung on hooks for twenty-four hours at least. They are then taken and weighed into oak hogsheads containing 1,300 to 1,500 pounds of meat, or into tierces of 300 pounds each, and pickled with brine 60 to 80 per cent strength, to which solution of salt and water is added 2 pounds of pure cane sugar and 4 ounces of saltpeter to the 100 pounds of meat, and then tightly pressed down underneath the pickle to keep air-tight. It is then overhauled or taken from one hogshead and placed into another four different times so that the pickle may reach all parts of the pieces of hams or shoulders; also that it can be evenly and uniformly cured, which takes from sixty to ninety days, according to the weight and size of the pieces of meat.

A process dissimilar in some details from the preceding was outlined by another correspondent, viz:

The hogs after being dressed are hung up in the chill room, cooled by artificial cold, the temperature being about 34° to 38°, not less than forty-eight hours, and when thoroughly cooled are made into the various cuts. The regular American

cut ham for sweet pickling is then put into vats or tierces and to every tierce of 300 pounds, or its equivalent in the vat, we use three-quarters of a pound of East India saltpeter, half a gallon of sugarhouse sirup of high grade, and 27 pounds of salt. This is partly made into a pickle and partly put into the tierce dry; when tierces are used, the tierce is moved after being packed, say six or seven days, for the purpose of giving the hams a more uniform cure or preventing salt from settling and remaining at bottom of tierce. If cured in vats, the hams are removed from one vat to another and brine poured over again, this for the purpose of mixing the brine and keeping it uniform throughout the vat; the hams being kept in cold storage in temperature of 34° to 38° for a period of forty-five to sixty days. Sweet-pickle meats—that is, shoulders and bellies—are treated the same way. The meats for dry salting, being chilled as aforesaid, are put into cold storage in bulk, and same amount of saltpeter is used with them as in the sweet pickling, one-quarter of a pound to 100 pounds, and then they are banked in salt. The amount of salt used depends on the length of time they are kept in bulk before shipping. These are resalted from time to time, they being restacked at intervals, until they are ready for shipping. It is difficult to tell the exact amount of salt, they having to be entirely covered when in process of curing, the salt having to be swept off before shipping and packed with fresh salt. The sides remain in salt not less than thirty days, and if heavy a longer period; the hams and shoulders forty-five days and upward, according to the size of pieces. These must remain in this condition until sold and shipped, sometimes remaining some months.

A description of the methods of curing hams, making barrel beef, shipping sweet-pickle meats, and preparing fresh canned meats is embodied in the following:

After the hogs are killed they are hung in the cooler forty-eight hours or more, until the temperature of the meat in the center is 40° F., or cooler. The hogs are then cut up and the hams or shoulders are placed in a cooler at about 30° F. for forty-eight hours or longer. Three hundred pounds of hams are usually placed in a tierce; for one tierce of hams 24 pounds of Saginaw salt, 12 ounces nitrate of potash, and 5 pounds Louisiana sugar. The tierce is filled with water. For pickling in vats the same proportion of ingredients is used. Sixteen-pound hams cure in sixty days at a temperature of 40°. After packing, the barrels must be rolled once every two weeks in order to detect leaks and equalize the pickle. In some cases the pickle is injected into the hams in order to hurry the process. By this method a pickle can be effected in about thirty-five days. This, however, is not the usual practice. I find that the proportion of ingredients varies some according to whether they are making a mild or strong cure. For meats going to hot countries strong pickle in usually used, and in smoking the same is the case.

In making barrel beef they usually put 200 pounds of beef in a tierce. One peck of coarse salt is put in the bottom of the barrel. The pieces are rubbed with fine salt and packed. Eight ounces of nitrate of potash is added, together with 1 peck of coarse salt on the top of the barrel. The barrel is then filled with a solution of salt 100 degrees strong. The barrel is rolled once a week and is considered cured at the end of four weeks. It is kept in cold storage about 35° or 40° F.

In shipping sweet-pickle meats, it is usually the custom after they have been removed from the pickle and dried, to rub them with finely powdered borax. They are then packed in boxes and shipped in that condition, although they are also shipped in salt and in pickle.

In curing dry-salted meats the meat is thoroughly rubbed with salt and packed in piles in cold storage of about 40°. It is handled about once every two weeks, when it is unpiled and rubbed again with salt and repiled, that on the top being placed at the bottom and vice versa. The moisture of the meat furnishes the water for the pickle.

Fresh canned meat is first cocked in boiling water for from one-half to three-fourths of an hour. It is then placed in cans and is cooked again in steam retorts 240° F. from one to three hours according to the size of the can. It is then taken out of the retort and the can opened to allow the escape of air, water, etc. It is then resealed and cooked a second time at the same temperature and for about the same length of time. The cans which seem to contain air after the second cooking are again opened and sealed. Salt canned meat is also cooked in this manner. A secret process is used for preserving hog livers. Livers cured in this manner are shipped to Germany and are said to present an almost fresh appearance upon their arrival. The shippers, who are Germans, preserve the livers themselves, but will not reveal the secret of the pickle.

A formula submitted to the Department for sugar-curing hams is as follows:

Sugar pickle.

Salt	pounds	200
Crude saltpeter	do	5
Sal soda	do	7
Sirup or New Orleans molasses	gallons	9
Water sufficient to make	do	120

First, have your hogsheads, to contain 120 gallons of pickle, in good order and well cleaned. Then have a few hogsheads separate from the others in which you make and prepare your pickle. Weigh off the 200 pounds of salt and place it in the hogshead and fill up with water until the same is two-thirds full, constantly stirring until the salt is entirely dissolved. For each 5 pounds of saltpeter and 7 of sal soda, pounded up and placed in an iron kettle, add 2 gallons of water, and with a slow fire simmer until the ingredients are all dissolved. Take 3 gallons of this solution and put into every hogshead of brine you have thus far prepared, and should there be any remaining divide it up pro rata among the hogsheads, keeping up a constant agitation until the whole is thoroughly incorporated. Lastly, take 9 gallons of molasses and add it to each hogshead of pickle, constantly stirring until the saccharine matter is perfectly mixed, and should the cask not be entirely full add water sufficient, and the brine will be ready for use.

Then take the hams as cut at the block and sort or divide them into three different sizes, viz, small, medium, and large. As they are delivered at your salt box, take each ham separately and plunge it shank downward into the salt, flesh side up, over which you sprinkle about one-half an ounce of fine ground saltpeter. With the palm of your hands rub the saltpeter thoroughly into the meat, and in like manner afterwards the salt. You then pack into the cask, shanks to shanks and butts to butts, until the same is full, placing the butts of hams downward first, and so on alternately until the hogshead contains about 850 pounds. The hams, or whatever kind of meat you are packing for cure, are to be braced down, so that they will not float or rise to the surface; have each size packed in hogsheads separate, then pour the pickle over the meat until the cask is full and the meat entirely submerged.

In this condition you let the hams remain five or six days, then overhaul or change from one cask to another, packing them as already described, and fill up the cask with the same pickle. Continue this overhauling for three consecutive times, at intervals of five or six days, and then let them remain undisturbed until perfectly cured. Allow small hams to remain in pickle thirty days, medium forty-five, and large sixty days.

On removing the cured hams from pickle, dip or wash them in hot water, allow to partially dry, and finally smoke them for eight or ten days. Green hickory has been practically demonstrated to be best for smoking.

Shoulders, breakfast bacon, and all other pork products are treated in the same manner.

The following process differs in no important respects from the general principles of several of the preceding formulas:

Hogs, when killed, are first placed in the dry room, and after remaining there from three and one-half to four hours they are put into the chill room, at a temperature of 28° to 30° F., for from forty-eight to sixty hours. They are next cut up into sides, hams, shoulders, etc., ready to be cured.

Dry-salted meats.—Sides are packed in dry salt, and after fifteen days are turned and resalted. The complete curing process requires from forty to fifty days.

Long hams are packed in dry salt with 4 ounces of saltpeter to the hundredweight of meat. These are not resalted, and are cured in about thirty days at a temperature of from 36° to 38° F.

Meats for export are packed in borax.

Pickled meats.—Hams and shoulders when taken from the cutting room are replaced in the chill room; they remain there from twenty-four to forty-eight hours, so as to be thoroughly chilled. They are then packed in an 80 per cent salt solution with 4 ounces of saltpeter and 1½ pounds of sugar to the hundredweight of meat. After eight days the meat is turned in the barrels in which it is packed, so that the salt will be thoroughly distributed. It then remains undisturbed until cured.

Picnic hams and shoulders require from sixty to sixty-five days and hams from eighty to eighty-five days to cure.

Mess pork is cured in a 100 per cent salt solution and from 18 to 20 pounds of rock salt to the hundredweight of meat.

Another packing company submits its process of curing meats in the following terms:

Beef hams cut into regular sets of three pieces, viz, the outside, inside, and knuckle pieces. After being thoroughly chilled, weigh 220 pounds to the barrel, add 70 degree salimeter-test pickle, 10 ounces saltpeter, and 3 pints of sirup. After forty days' cure it is ready to be smoked.

Plate beef.—The plates cut in three pieces, brisket, navel, and rib; pack 200 pounds to the barrel; use 30 pounds coarse capping salt, 100 degree pickle, 10 ounces saltpeter; plain cure.

Extra mess beef.—Same cure as plates; meat used, chucks, plates, flanks, and rumps; 200 pounds to the barrel.

Canned beef.—Plain cure; 75 degree pickle, 6 ounces saltpeter per hundred; after twenty-five days' cure, ready to can.

Pork hams, sugar cured.—After being well chilled they are rubbed with fine salt and left standing up in the cooler over night. Pack them 300 pounds to the tierce, 68 degree strong pickle, 5 pounds granulated sugar, or 3 quarts of Washington butcher sirup. Time for curing, fifty days.

Shoulder and bacon.—Same cure as hams.

Short ribs.—Cured in dry salt, forty days, ready for shipment; fifty days ready for smoking.

The following is a formula generally in use for curing standard meats at another abattoir:

Twenty-three pounds salt, three-quarters of a pound saltpeter, 3½ pounds sugar to 300 pounds green meats.

The hams are then kept in a temperature of about 36° to 40° for a period varying from seventy to ninety days, when they are ready for smoking. They can be smoked in about from two to three days with hickory wood and sawdust used as a fire.

Dry-salted meats are cured with dry salt and about the same proportions as stated above of saltpeter and sugar. They are kept in process of curing, varying for the time of year, from thirty to forty days, when they can be smoked and put on the market. The usual time for smoking dry-salted meat is from two to three days with hickory wood and sawdust.

The above formulas would cover all meats which are put in pickle of such as hams, shoulders, etc.

Mess pork is cured with full strength brine and topped off with clear rock salt.

Another correspondent writes:

There are so many varieties and different ways of curing the different cuts of meat that it is almost impossible for us to know where to begin to give you the information you desire.

The time of curing hams depends upon the size of the piece of meat. It takes from forty-five to ninety days to cure a 12-pound to a 20-pound ham. The hogs, immediately after slaughtering, are run into a chill room, where the temperature is about 32° twelve hours after killing, which temperature is run down to 25° to 27° thirty-six hours after killing. When the hogs are cut up the hams are spread on shelves in a temperature of 25° to 28°. They are allowed to remain in this temperature twenty-four hours, when they are taken out and put in pickle made of salt, sirup or sugar and water, and allowed to remain in this pickle from forty-five to ninety days, depending upon the weight of the meat. These hams are taken out of pickle after being cured and are either shipped to foreign countries rubbed in borax and packed in boxes or smoked and sold to the domestic trade.

Especial importance is attached in the succeeding formula to the "cutting" of hams with reference both to shape and weight.

Hogs when dressed hang from forty-five to forty-eight hours in an average temperature of about 35°. They are then cut up and the hams shelved for about twenty-four hours in a temperature of about 35°, when they are placed in pickle and cured for about sixty days before being smoked.

Standard sweet-pickled hams should be cut short and well rounded at the butt, properly faced, shank cut off enough above the hock joint to expose the marrow, to be reasonably uniform in size, and to average in lots not to exceed 16 pounds, with no ham to weigh less than 12 pounds and none to weigh over 20 pounds.

Three hundred pounds block weight shall be placed in each tierce, with either 22 pounds salt, 3 quarts of good sirup, 12 ounces of saltpeter and tierces filled with water, or tierce filled with sweet pickle made according to above standard.

The treatment of the hog and its product from the time of delivery to the packing company until ready for market is briefly outlined in the subjoined:

Hogs used for packing purposes are well fed and watered from three to five days after delivery, then slaughtered and hung in cooler for forty-eight hours. They are then cut into different parts, which are also hung in cooler for forty-eight hours more, placed in tubs in cooler with brine composed of water, salt, saltpeter, and sirup, and left there from sixty to eighty days. They are then taken out, smoked, and sold to the trade.

Reference to the importance of allowing the animal to become thoroughly cooled before slaughtering prefaces the process of curing in use by another packing company:

Allow all hogs to rest for twenty-four hours in a pen, in order to have them thoroughly cooled out before killing. After hogs are killed and thoroughly chilled (it takes at least forty-eight hours), they are then cut up into the different cuts of either hams, shoulders, bellies, or sides.

The hams, shoulders, and bellies are cured in sweet pickle, which is composed of sugar, salt, saltpeter, and water, thereby making a brine. The meats are packed in a tierce, covered with this brine, and are then allowed to remain in cure from forty-five to sixty days, the same depending upon the weight before being smoked.

After these meats are smoked they are ready for the trade. The dry salt meats are packed in cellars in piles, and between each layer of meat is thoroughly sprinkled a large quantity of salt. They are then allowed to cure from twenty to forty days, depending on size of meat, and after being thoroughly cured in this manner they are ready for the market.

Another response to the inquiries of the Department, different in few particulars from the above, is:

All hogs are allowed to rest in pen from four days to one week before being slaughtered. After they are slaughtered they go in chill room for forty-eight hours, then are cut in different parts, and these are put in chill room for forty-eight hours more. Hams, shoulders, and bacon are then packed in tierces and a pickle composed of salt, saltpeter, and sirup is put on them. They are left in this pickle from sixty to eighty days, then taken out, smoked, and sold to the trade.

This formula, used by a Western packing company, has been submitted:

For sweet pickled meats take a pickle of 90° Baumé test and add thereto the usual amounts of saltpeter and granulated sugar. The joints placed in this pickle are shifted two or three times during the process of cure and are kept in the pickle from fifty to ninety days, in accordance with the weight of the product; or, in other words, for picnic hams about fifty days, for New York shoulders about sixty days, and for hams from seventy-five to ninety days, in accordance with their average weight.

For white-brine cure the process is similar, excepting that a clear pickle of 100° Baumé test is used on the meat with the addition of a little saltpeter without the sugar.

A receipt for curing meats both in pickle and dry salt is as follows:

To 1,000 pounds hams thoroughly chilled make a pickle of the best Liverpool ground salt of 85° strength, to which add 15 pounds granulated sugar and 10 ounces saltpeter. For dry salt meats use the same proportion salt, saltpeter, and sugar, rubbed carefully into the ham after being thoroughly chilled and dried.

FORMULA FOR CURING PORK, HAMS, MESS BEEF, MESS PORK, ETC.

Pork hams.

Temperature, 35° to 40°.
Pickle, 80 to 100 per cent salt.
Saltpeter, 6 ounces per 100 pounds meat.
Sugar, 3 pounds per 100 pounds meat.
Overhaul fifth, fifteenth, and thirtieth day.
Cure seventy to one hundred days.

Plate, extra mess beef, mess pork, etc.

Use pickle 100 per cent salt.
Saltpeter, 6 ounces per 100 pounds meat.
Capping additional, 10 to 25 pounds per barrel.
Overhaul five days, and every twenty-five or thirty days thereafter.

Dry-salt cure—sides, shoulders, backs, etc.

Rub in dry salt, sprinkle lightly saltpeter.
Overhaul fifth and twentieth day.
Cure forty to sixty days, according to size.

Meats for potted ham.

Pickle, 80 per cent.
Saltpeter, three-fourths of an ounce per 100 pounds meat.

A process similar to many others received by the Department is as follows:

Twenty-five pounds of salt, 5 pounds granulated sugar, three-fourths of a pound nitrate of potash. This quantity is used for 300 pounds of hams.

The products are overhauled three or four times and remain in pickle, according to size, viz: 10-pound ham, forty days; 15-pound ham, sixty days; 20-pound ham, seventy-two days. All other products are treated in like manner.

Another process is as follows:

The hogs hang in chill room for three days before being cut up. The hams are then put in pickle of 70 per cent in winter and 76 per cent in summer. To every 300 pounds of hams are added three-fourths of a pound of saltpeter and 4 pounds of sugar. They are allowed to stay in this from sixty to ninety days, according to size, and are then washed, hung in the smokehouse for twenty-four hours, and are ready for market.

Another formula:

The best fine salt, 21 pounds; saltpeter, 8 ounces; sugar, 5 pounds to 290 pounds of green hams; mild cure.

Meat should be properly cooled and handled, say three, five, and ten days, and remain in pickle sixty days. This depends on the size of the ham or piece.

DIPPING CATTLE FOR DESTRUCTION OF TICKS.

(Compiled from a report by VICTOR A. NÖRGAARD, inspector.)

The discovery that the Southern cattle tick was the cause of Texas fever and was the sole agency by means of which the disease was transmitted from Southern to Northern cattle, when the two were brought into contact, called attention at once to the vital importance of the total extermination of these parasites from Southern herds. Previously the destruction of the tick was almost a subject of indifference, because the Southern cattle themselves did not sicken from the infestation. But the restrictions that have been placed upon the transportation and marketing of Southern cattle have had such a blighting effect upon the cattle-raising industry south of the Texas-fever line that the invention of some practical method by which all cattle, or even those intended for commercial purposes, might be freed from the pests would be second in importance only to the discovery of the agency itself.

During the past year a series of experiments with this end in view has been conducted by Dr. Victor A. Nörgaard, inspector, of this Bureau, on the Santa Gertrudes ranch in Nueces County, Tex. A detailed account of the plan followed in these investigations and the results attained are herewith summarized.

The bulletin issued by the Bureau of Animal Industry in 1893 concerning Texas fever proved that Southern cattle, when completely freed from cattle ticks, could be brought into contact with susceptible Northern cattle without danger of communicating Texas fever. It also suggested that, could some method be devised whereby Southern cattle could be entirely freed from ticks, their transportation into the uninfected district might be carried on without restriction during the entire year, whereas this is at present possible only from November 15 to February 15.

As the question affects thousands of cattle, the greater number of which are very wild and have never been handled, it is of course impossible to apply any remedy to them by hand, and the only means which hitherto has proved practicable is to run them through a disinfecting bath or, as it is termed, to "dip" them. In order to accomplish this it is necessary to construct a dipping-vat which is sufficiently large to allow the complete immersion of a full-grown animal. Such a vat, which will be fully described below, has been designed and built by Mr. R. J. Kleberg, manager of the Santa Gertrudes ranch in Nueces County, Tex., who kindly placed it at the disposal of this Bureau, and also offered to furnish the cattle necessary to carry on experiments for the purpose of finding a disinfectant which, when applied to the animals in the form of a bath, would destroy the ticks attached to them. A series of experiments has been conducted at this place during the past year, the results of which are herewith given.

In conducting such experiments there are several points to be taken into consideration. If the animals are intended for immediate transportation into the uninfected territory, it is desirable that all the ticks on them should be dead by the time the point of destination is reached on the other other side of the quarantine line; this, on the supposition that they were subjected to the disinfection at the shipping point, and directly placed in clean, disinfected cars. In instances, however, where the transportation would take only a few hours, the time would be insufficient for the full-grown ticks attached to the cattle to be destroyed without using a solution of such strength as also to seriously injure the animals. Such extreme strength, however, is not absolutely necessary. It is sufficient if the solution possesses such properties that the immersion of the ticks in it will destroy them, even though several days elapse before death occurs. If convinced that our solution will accomplish this, it is of no importance that, upon arrival at their destination, live ticks may still be found on the animals; they will perish in due time. The ticks must, however, show visible signs of being affected by the dip, such as having shrunk, and changed their color to either black or yellow-mottled. Whether a tick in this condition is able to rally has not been ascertained; but it is hardly probable, as no instance of revival has been noted, though special attention has been given to this matter.

The next point to be considered is that the disinfectant should not injure the cattle, even though the injury be slight and of a passing nature. This has been the only barrier to the success of many experiments. The greatest difficulty has been encountered in determining a line of distinction between the power of resistance of the host and that of the parasite against the caustic effect of the various disinfectants used. This line of distinction is always very fine and does not even exist in the case of the greater number of disinfectants hitherto tested, all of which revealed that a composition which does not injure the host also leaves the parasite intact, and that which destroys the tick will also more or less injure the animal. Many disinfectants, when applied in a sufficiently strong solution, will destroy the parasite in the time desired, but they will also leave the animals in a suffering condition and may render them for a time unfit for transportation or for market, and may even in extreme cases necessitate a protracted after treatment.

Considering the remarkable tenacity of life which the tick possesses and the strength of dip consequently requisite for its destruction, the complete immersion into such solution must necessarily prove severe upon the animal.

Even granting that such a composition has been found as will ordinarily destroy the ticks and leave the animal uninjured, still it must be kept in mind that ticks frequently attach themselves to those parts of the body which, during the process of dipping, are only momentarily immersed—such as the head, the ears, and the upper part of the neck—whence the fluid recedes so quickly that, while it may be depended upon to destroy the younger and weaker ticks, it will often leave the full-grown ones unaffected.

It is a well-known fact that when a tick has become fully gorged or ripe it remains attached to the host for only a few days, and experiments have proved that until the tick reaches this state it readily succumbs to the action of many solutions so weak as to have no ill effect upon the animal. When ticky cattle are dipped in such solutions only the ripe ticks are left undestroyed, and when at the

expiration of a few days these have dropped off, provided no new infection has taken place in the meantime, the animals are free from ticks, and no longer able to communicate Texas fever.

When, however, it is desired to dip a large number of cattle preparatory to shipping them into the uninfected territory, it would be difficult or impossible to prevent a reinfection from taking place while awaiting the disappearance of the ripe ticks; but this new invasion of young ticks might be easily disposed of by redipping the cattle in a proper solution immediately before placing them in the disinfected cars.

Considering that the survival of a very few ticks might lead to serious consequences, this method of double dipping will always prove the safer, for, however reliable the disinfectant may be, the single dipping of a herd of half-wild Southern cattle which rush uncontrollably through the chutes and jump on top of one another into the vat can never be so thoroughly effected as to warrant a safe result.

Should the method of double dipping be adopted, dipping-vats might be constructed in connection with quarantine pens at convenient places near the Texas-fever line where it intersects with the trunk lines running north. Cattle intended for transportation into the uninfected territory might be dipped here immediately upon their arrival, and quarantined for about a week to allow all the ripe ticks, which survived the dipping, to drop off; then redipped and shipped under such precautions as to exclude the possibility of a reinfection.

As the South, and especially the Southwest, is largely a cattle-raising country, and as the production of grain in the Northern and Central States is far in excess of their demand, it is essential that the two, in order to realize the greatest profit, should cooperate—the South by raising the cattle, the North by feeding them. When this occurs it will largely be young, grass-fed animals which will be shipped North, and these could, without any great expense, be kept in quarantine for a week; while those animals which have been fattened on Southern grain or cotton seed could be shipped for immediate slaughter, as heretofore, without any quarantine restrictions.

Laboratory tests.—In order to determine whether a disinfectant possesses tick-destroying properties, and at the same time to obtain some idea of the minimum strength required, some preliminary experiments are necessary. These have generally been made by applying the disinfectant in solutions of various degrees of strength to a few detached ticks. These are allowed to remain in the solution for the same length of time as is required for an animal to be plunged into the vat, to swim through it, and to emerge at the other end. This is from ten to fifteen seconds. The liquid is then poured off and the ticks, with the adhering fluid, are placed on a piece of paper and covered with a glass. (It has been found that when the ticks are placed on blotting paper the liquid is absorbed more quickly from them than when they are partly covered by the hair on an animal which has been dipped.)

When a disinfectant is to be tested, five different solutions are made of it, ranging in strength from 0.1 to 5 per cent, taking as a starting point the maximum strength which it is supposed an animal might endure when dipped in it. One solution is made a certain degree stronger (for instance, 0.5 per cent), and three more, successively, the same degree weaker. The same number of ticks must be used in testing each solution, and, while it is desirable that the individual ticks in each bunch range in size from small to full-grown and

ripe ones, care must be taken to have the different bunches as nearly alike as possible. Each bunch of ticks is then placed in a separate glass, one after another the solutions are poured over the ticks, allowed to remain for fifteen seconds, then drained off, and the glass is inverted over the ticks and placed on a piece of white paper sufficiently large to allow notes to be made thereon, stating date, name, and strength of the solution, time of exposure, and number of ticks.

By daily observations it is easily determined which of these solutions, if any, will kill the ticks. If on the following day the ticks under any of the glasses have changed their original color, shrunk considerably in size, and become hard and brittle, then the solution applied to them may be deemed effectual. A solution which will produce this effect on the ticks in the laboratory will also be sure to destroy them when it is applied to tick-bearing cattle as a bath; but it will also, in most cases, be strong enough to burn the cattle more or less severely. Only one exception to this rule has been found, which was when glycerin was applied. This common commercial article will, when applied to the ticks in the laboratory, dry them up as hard as chips in twenty-four hours, even when it is diluted with three to four times its volume of water. It may with equal success be applied to ticky cattle without any disagreeable effect on the animal. But the expense involved in filling a 2,500-gallon vat with a 25 per cent solution of glycerin reaches such figures as to exclude it from practical use.

In observing the ticks subjected to the solution 1 degree weaker it may be noted that three or four days are required to produce changes similar to those produced in twenty-four hours by the stronger solution. When the solution is 2 degrees weaker no such pronounced changes may be noted, for several days at least, in the ripe ticks; these may even lay a small number of eggs before perishing, while in the meantime all the smaller ones have died. The next weaker solution has not prevented any of the ripe ticks from laying the normal amount of eggs; even those not gorged may have laid a small number. If all these eggs are left alone, and the weather be warm, they will hatch within a month; and it would be natural to conclude that this solution was altogether too weak, if practical results did not prove that in this case, as in many others, it is absolutely unsafe to depend wholly upon negative results. Such a solution, which in the preliminary test did not kill the ticks or prevent them from laying eggs, or the eggs from hatching, may, when applied practically to the ticky animal as a bath, destroy all the ticks in a short time. This recently discovered fact is of great importance, as it makes it necessary to repeat a large number of experiments which have been made during the past year, and to give a more thorough trial to a number of disinfectants which were considered unfit for the destruction of ticks simply because they failed to kill in the preliminary experiments. No composition should be condemned until it has been subjected to a practical test, viz, until animals carrying live ticks have been dipped in a solution of it and the ticks have not been destroyed. The expense involved in testing a disinfectant this way, in which 2,500 gallons of the solution is required, naturally precludes the possibility of adopting this method in general. It would be natural to infer that a few gallons of a solution to be tested applied liberally as a wash to a ticky animal would be equally efficacious. But experience teaches us that the results of dipping and washing with the same solution are by no means alike. The positive result, the killing of the tick, is

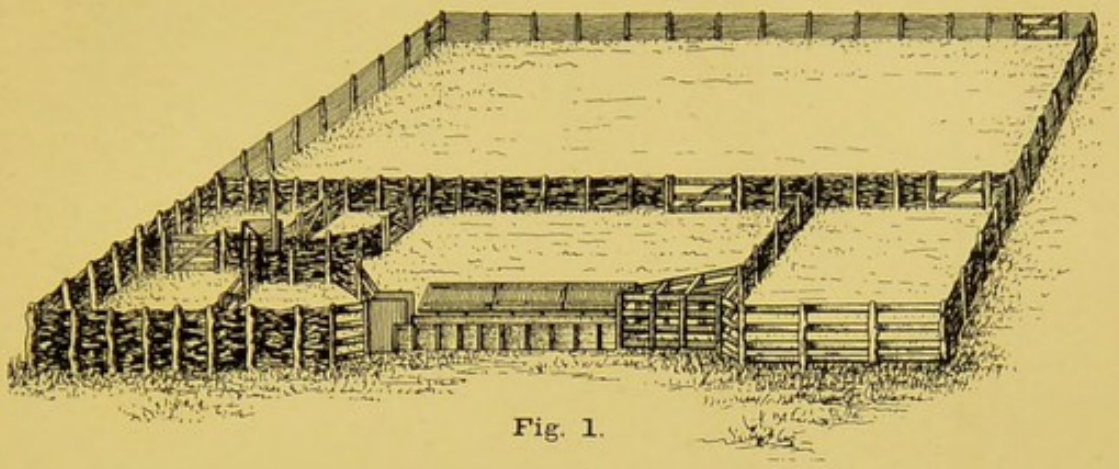


Fig. 1.

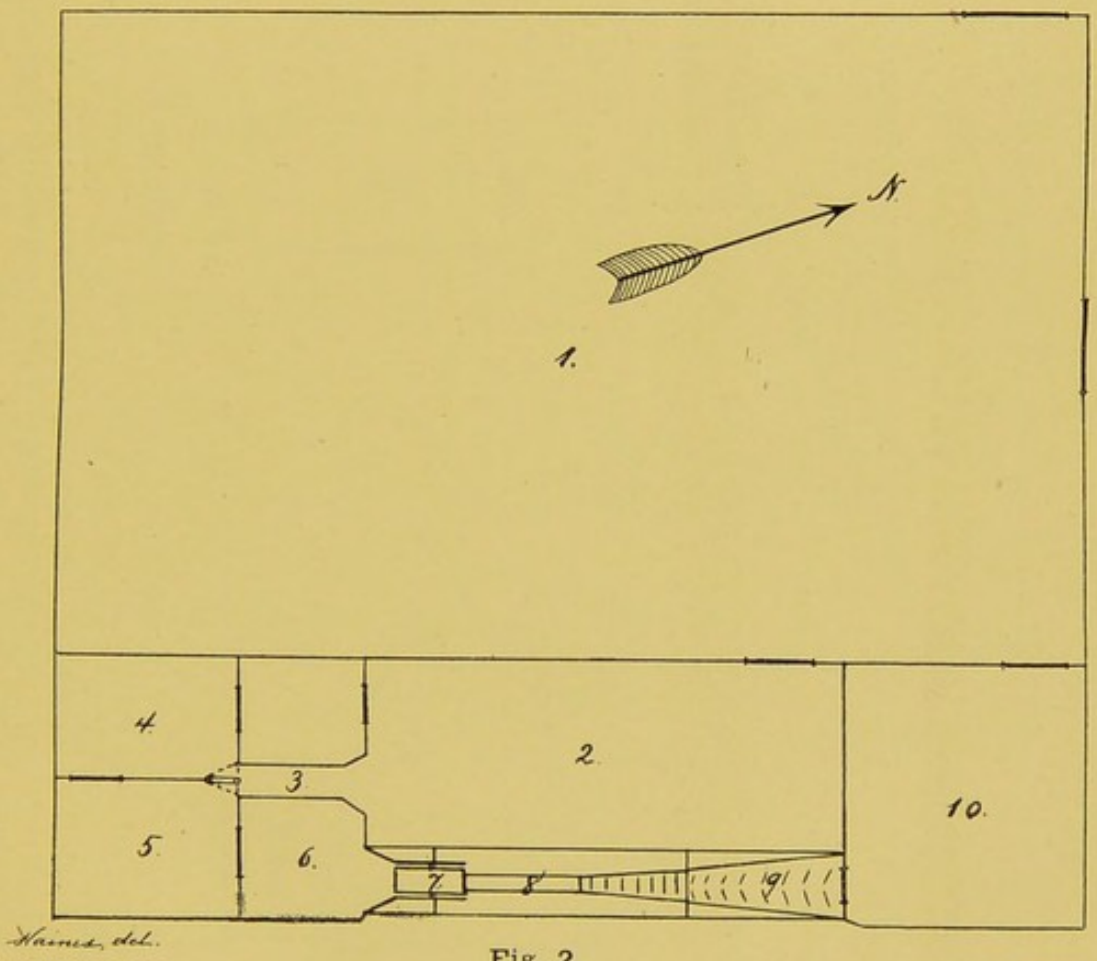


Fig. 2.

THE DIPPING STATION AT SANTA GERTRUDE'S RANCH NUECES COUNTY TEX.

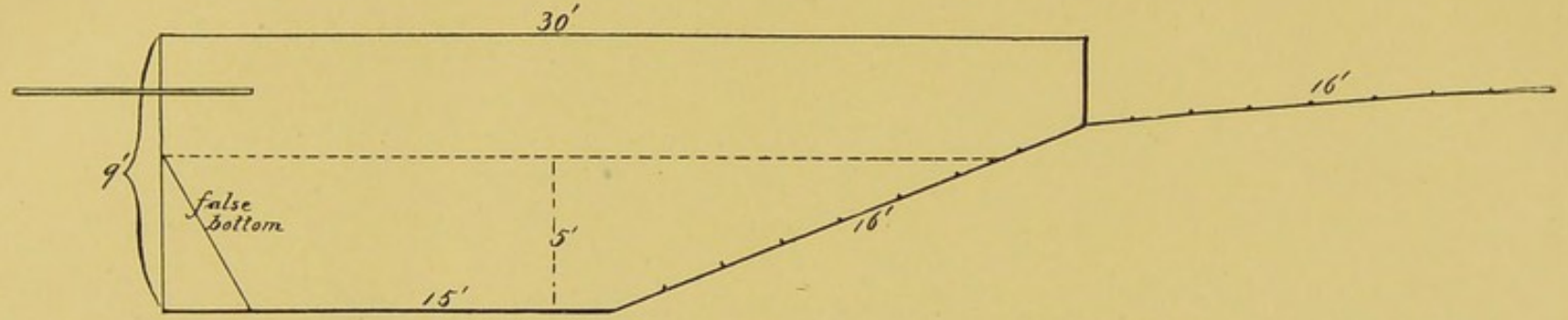


Fig. 1.

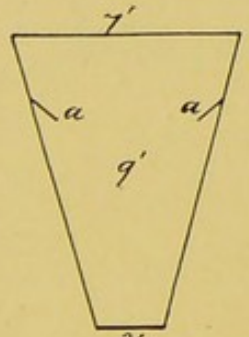


Fig. 2.

Haines del *Vat.*

Fig. 3.

Trap-door.

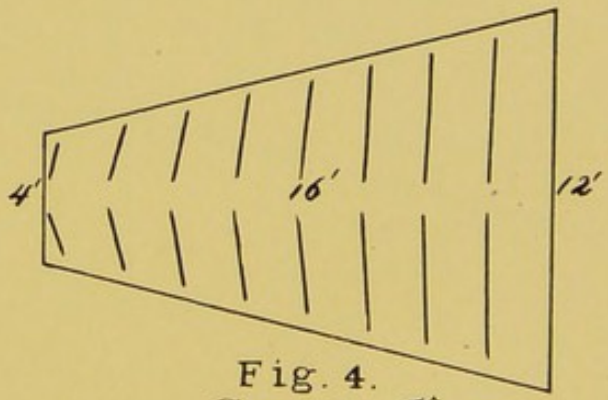
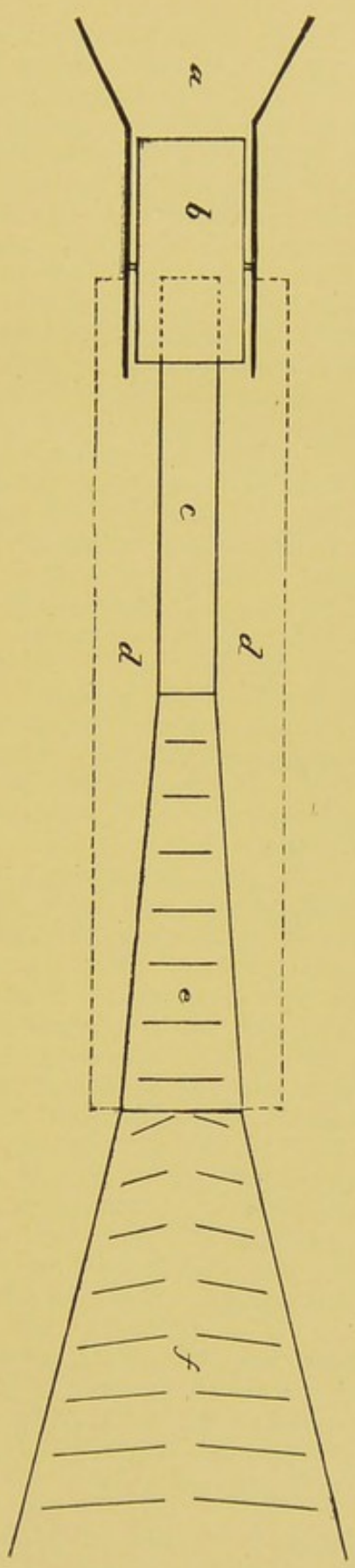


Fig. 4.

Dripping Vat.

SECTIONAL VIEWS OF DIPPING VAT AT SANTA GERTRUDE'S RANCH.

AN. REPT. CHIEF B. A. I., 1895-96.



Sectional View of Chute and Vat at Santa Gertrudis Ranch.

also in this instance conclusive. The solution which will destroy the parasite when applied to the host as a wash will also prove itself efficient when the animal is dipped in it. But the negative result, viz, that the wash has no disagreeable effect on the animal, does not prove that this will be the case when applied as a bath. In fact the difference is very great. A 2 per cent solution of Marchaelis cattle wash may be applied very liberally to an animal as a wash. It will then destroy the parasites without injuring the host. But if this solution be applied as a dip, the animals which are immersed in it will barely escape with their lives. It seems that the sudden exposure of the whole body, as well as the strong pressure of the solution against the body suspended in it, increased by the violent movements of the animal in swimming through it, cause a deeper penetration into the skin than when the solution is simply poured over a quiet or well-secured animal. Hence, a conclusive test of a disinfectant is only obtained by a complete immersion of an animal in exactly the same manner as would be pursued in practice.

The dipping station.—It consists, as is shown in Pl. I, of a number of pens so arranged as to facilitate the handling and assorting of large numbers of cattle in order that those which are not to be dipped may be "cut out"—for instance, young calves and pregnant cows. All these pens, except the largest (Pl. I, 1), are built very strongly of mesquite timber. The largest pen is built of mesquite posts connected by ten smooth wires. The pens all connect with one another, the dipping vat being constructed between the last two. The vat itself (Pl. II) is 30 feet long at the top and 15 feet at the bottom, is built of 4 by 4 inch upright timbers 3 feet apart, planked inside with 2-inch cypress boards, and is well calked. It is 2 feet wide at the bottom and 7 feet at the top. The vat is 9 feet deep, but is never filled to a greater depth than 5 feet, at which point it holds about 2,500 gallons. A short narrow chute (Pl. III) leads to the entrance of the vat, and in this is located a trapdoor, the heavier end of which rests on the floor in the chute, while the other end protrudes over the vat between the extended sides of the chute. The trapdoor, which is 4 by 8 feet, turns on a bolt, is located 7 feet from the bottom of the vat and 2 feet from the surface of the dip when this amounts to 2,500 gallons. This insures a complete immersion of the animals. A false bottom directly underneath the trapdoor compels the animals to swim forward and make room for those following, as the vat is narrow enough to prevent an animal from turning around in it.

An incline leads from the end of the bottom to the top of the farther end of the vat, where it is joined to the dripping floor. This is a platform 16 feet long, 4 feet wide where it connects with the incline in the vat, and 12 feet wide at the farther end. It is fenced in with boards, slopes slightly toward the vat, and, like the incline, is heavily cleated to prevent the animals from slipping.

The vat is provided with a tongued and grooved cover which, for convenience of handling, is divided into three sections and is attached to one side of the vat; that side, for the purpose of drainage, is built slightly higher than the other. The cover protects the dip from sun, rain, and dust, and should always be kept closed when the vat is not in use.

When a number of cattle are to be dipped they are driven into the large pen, where men on horseback "cut out" those not to be dipped. The animals are then turned into pen No. 2. From this leads a narrow chute, which allows one animal only to pass at a time. At the

end of the chute is a swinging gate manipulated by a man standing on a triangular platform above it. By means of this contrivance all the calves are separated from the cows, and any animal which may have escaped the first "cutting out" in the large pen is here separated from the herd. Those to be dipped are driven into pen No. 5, the others into No. 4. A few of those in No. 5, usually eight or ten head—as many as the dripping pan will hold—are then let into pen No. 6, where a couple of men drive them on the trapdoor, which springs and throws them one by one into the vat. The animals then swim quickly toward the other end, walk up the incline, and are detained on the dripping floor for a few minutes to allow the dip to drain off and flow back into the vat. The gate is then opened and they pass into pen No. 10. One or two animals are always retained in the dripping pen because the following bunch will enter the chute more readily when the leader sees cattle on the other side of the vat.

A thousand head of range cattle may be dipped in a few hours, for these wild cattle need no urging. Seeing that their only escape is through the vat, they jump boldly in and follow each other in quick succession. Gentle cattle, however, are very much harder to handle. They must be driven to the trapdoor and often pushed on it by force. Therefore, as compared with range cattle, scarcely one-half the number of gentle cattle can be dipped in the same length of time. This process of dipping is absolutely safe. Of more than 20,000 animals dipped in this vat not a single one has met with accident. When dipping calves, however, it is necessary to place a man at one side of the vat to right any that may be turned around, as the trapdoor chute is not sufficiently narrow to prevent two calves from passing in at the same time.

When a very large number of cattle are to be dipped it may be necessary to replenish the vat, as each animal carries away about one-half of a gallon of the dip. The contents of the vat should not be allowed to diminish to less than 2,000 gallons.

Experimental dipping.—When the preliminary tests have proved that a disinfectant, in a solution not stronger than 2 per cent, is able to destroy detached ticks, and that it is equally effective when applied to a ticky animal as a wash, leaving no traces of injury upon the beast, the next step is to ascertain whether the solution will be too strong when an animal is dipped in it. The reason why none stronger than 2 per cent are to be considered is that in a vat holding 2,500 gallons a common 50-gallon barrel of the disinfectant will make a 2 per cent solution. The additional expense for freight and transportation, if more than 1 barrel is used, would render an otherwise cheap disinfectant disproportionately more expensive.

The experimental dipping may be done in two ways. The shorter is to fill a vat with a solution which is known to be strong enough to destroy the ticks. One animal, generally a common bred 2-year-old, is driven through the dip. After it emerges at the other end, it is detained for a few minutes in the dripping pen and then let into the adjoining pen for observation. If at the end of half an hour the animal has shown no symptoms of being uneasy, drowsy, or otherwise affected, a few more animals are compelled to go through the dip. If these remain equally unaffected they are all let into a small adjoining pasture. Here they are to be closely observed, and should be examined at least once every day to watch the effect of the dip.

If at the end of a few days all the ticks are dead, the next question is whether the animals will become affected or not. If these pre-

sented no abnormal symptoms immediately after the dipping, it will be about five days before the secondary symptoms appear—a slight stiffness may be noted, the skin on the more tender parts folds in wrinkles, and the epidermis peels off. These symptoms are caused by the caustic effect of the dip on the skin, and generally disappear in a few days. They should, however, be avoided, and to that end the strength of the dip must be reduced by adding more water and the experiment be repeated upon other animals. If then the dip ceases to destroy the ticks before it ceases to burn the animals it is of no value.

Another method of practically testing a dip is to commence with a solution which is certain not to injure the animals and gradually to increase its strength toward the point necessary to destroy the ticks. If this be not reached before the dip commences to burn the cattle, further experiments on this line are unnecessary. This method, which has the advantage of not exposing the test animals to any serious injury, requires, however, too much time, as the strength of the dip can only be increased with one-eighth of 1 per cent at a time, and at least five days must elapse between each test. Consequently the first method is generally used, as with this one or two dippings are sufficient to demonstrate the utility of a disinfectant.

If a satisfactory result be obtained by these preliminary dippings it is necessary to submit the solution to a practical test by dipping a considerable number of animals—for instance, from 100 to 500. Only such results as are obtained in this way may be regarded as conclusive. Experience in a great number of experiments has shown that a solution which proved efficient in the preliminary tests was frequently found to be inefficient when employed practically.

It may be well to give here a short description of the symptoms which follow immersion in too strong a solution of any disinfectant possessing caustic properties. If the animal be a heifer or a cow it will urinate immediately upon reaching the dripping pen. This is caused by the irritation of the vulva and of the mucous membrane of the vagina. A bull will kick the scrotum, on which an excessively strong dip seems to have a very irritating effect. The dung is passed constantly until none is left to pass, but the animal continues to strain as if it had the colic, is very uneasy, lies down and jumps up again, while saliva and mucus flow from the mouth. This state of irritation may last from ten to fifteen minutes, after which the animal either becomes quiet again and seeks for food or else passes into a drowsy state. In this state it stands with hanging head and drooping ears and pays little attention to its surroundings. Its movements are slow and uncertain and often it lies down with head upon the ground. This state of drowsiness is also of short duration; the animal soon recovers and begins to walk.

Only in cases where the solution has been entirely too strong does the animal stagger or become "drunk" and, in some instances, fall to the ground, unable to arise. When this happens it is best to thoroughly wash the animal with water, help it to rise, and lead it around for a while; it will then soon recover. These extreme symptoms are seldom seen except when very weak or thin-skinned animals—especially those of a light color—or young calves, either by mistake or carelesssness, are allowed to jump in the dip.

If the animal is at all restless after leaving the dip we may be sure that from three to five days later it will become stiff. How many days will elapse before the secondary symptoms appear is dependent

upon the strength of the dip which was used, the age and condition of the animal, whether thick or thin skinned, and also upon the weather, if the animal is exposed to it after dipping. Coarse-skinned, common-bred animals may be exposed to an excessively strong dip without showing any symptoms immediately after the dipping, but will nevertheless become stiff in due time. This stiffness, which is caused by the caustic effect of the dip on the skin, which becomes hard and dry with brushy-looking hair, is in lighter cases of little consequence and will disappear in a few days, being followed only by a slight peeling off of the epidermis on the more tender parts of the skin. These parts chiefly consist of the regions between the hind legs, those behind the elbow, on the lower part of the chest, the sides of the neck, and the dewlap. In severer cases the skin in these places will fold in numerous wrinkles and subsequently crack in deep fissures, out of which the blood oozes, forming crusts with the desquamating epidermis. The animal moves with great pain and may be unable to lower its head to the ground to feed. Such extreme cases necessitate a radical treatment in the form of a generous application of glycerin or oil, or anything at hand which will soften the skin and relieve the pain. Meanwhile the animal must be fed and watered, and should be sheltered against sun and rain.

Of the numerous disinfectants and disinfecting preparations which have been tested during the past year the best results have been obtained from two coal-tar preparations. Of these one called Chloro-Naphtholeum bids fair to prove entirely satisfactory, though experiments with it were so recently begun that further tests will be necessary before reaching a final decision as to its merits. The other preparation, the Lone Star Cattle and Sheep Wash, having been believed the best obtainable, received for many months almost exclusive attention, and thousands of cattle were subjected to its action, but the results were never altogether satisfactory. The few experiments which so far have been made with Chloro-Naphtholeum gave such superior results as to exclude the Lone Star Wash entirely from experimentation. A few of the experiments made with this are, however, worthy of reference, as the results obtained with it have frequently been so encouraging as to warrant repeated tests. But these have revealed a number of disadvantages, the most serious of which is that the line of distinction between the destroying of the ticks and the burning of the cattle is so fine as to leave no margin for individual differences of resisting power in a herd of cattle. When, through preliminary tests and dippings, a certain solution had been decided upon and a herd of cattle had been subjected to it, the result was invariably that when the dip was strong enough to destroy the ticks it also burned a number of the animals more or less severely. Furthermore, the variableness of the strength of this disinfectant, of which two successive barrels were hardly ever found to be alike, necessitated the repetition of preliminary tests for every new barrel procured. It was also found that when dissolved in the vat the resulting dip did not retain its tick-destroying properties for any length of time, a noticeable difference in its efficacy becoming evident even within a week. Of less importance is the difficulty with which this disinfectant dissolves in water. This is especially pronounced where well water must be employed, as is the case on this ranch. The disinfectant is a black fluid of a consistency like that of oil, and when poured in water only a portion of it will dissolve, while the greater part of it, like crude carbolic acid, either floats on top or sinks to the bottom. When boiled in equal

parts of water the greater part dissolves, but to render it perfectly soluble it is necessary to add soap. To dissolve 35 gallons of this disinfectant in 2,500 gallons of well water 100 pounds of soap are required. This makes the preparation of a vat full both tedious and expensive. If the solution is not perfect, a black scum forms on top. As this consists chiefly of crude carbolic acid, and as an animal in leaving the vat becomes covered with this, it will always be badly burned. To alleviate the caustic effect of this dip by administering a preventive simultaneously with dipping the cattle, experiments were made by pouring a barrel of oil over the surface of the dip, so that each animal when leaving the vat might receive a coating of oil. The preliminary dippings were comparatively satisfactory, but when the dip was tested practically with a large number of range cattle it was found that less than one-half of them retained any oil. The violent stirring caused the oil to become emulsified, especially when soap had been employed in dissolving the disinfectant, and the rapid succession of the animals prevented the oil from accumulating on the surface. Similar methods were given a trial, as, for instance, the one suggested by Dr. Francis, who mixed the disinfectant with oil and applied this to the cattle by dipping them in a vat full of water, on the surface of which the medicated oil floated, but these experiments only tended to prove that to be absolutely reliable a dip must be applied in a perfectly homogeneous solution.

To make such a solution of the same ingredients, an attempt was made to dissolve the disinfectant in a permanent oil emulsion which would contain sufficient oil to leave a coating on the animals when the water in which it was emulsified had evaporated. This was done by boiling disinfectant, oil, and soap together in an equal amount of water, which formed a perfect emulsion. The result with the test animals dipped the first day was satisfactory. But by the time the strength of the dip had been regulated the oil had begun to accumulate on the surface, and its very dark color indicated that it contained undiluted disinfectant. More and more appeared each day, and being crude cotton-seed oil, it soon commenced to grow thick and pasty, and finally sank to the bottom.

It might have been better to use an animal oil instead of crude cotton-seed oil, but that would have made the dip too expensive.

Experiments with oil were therefore discontinued. It may be well to mention here a preparation recommended by Dr. Francis for destroying ticks on a smaller scale. It consists of 15 parts of crude carbolic acid dissolved in 80 parts of crude cotton-seed oil, to which is added under slight heating 5 parts of pine tar. This is applied with a brush to gentle ticky animals, for instance, milch cows. It will also protect the animal from the horn fly for several days.

Chloro-Naphtholeum is a common nonpoisonous disinfectant. A 50-gallon barrel had been sent to this ranch to be tested as a tick destroyer. The preliminary experiments with it were decidedly discouraging. A 6.6 per cent solution applied for one minute to detached ticks did not prevent them from laying eggs. Exposed to a 2.2 per cent solution for the same length of time, all the ticks laid eggs within a week, and most of these hatched in due time. Consequently this disinfectant was not expected to destroy the ticks on cattle in a solution of 2 per cent or less, and for many months the experiments with Lone Star Wash were continued. Only the fact that the supply of this gave out before a new consignment had arrived prompted the testing of Chloro-Naphtholeum in the dipping vat. This disinfectant is easily

soluble in rain water, forming a milk-white solution of a creosotic, carbolic odor. In well water it is necessary to add a little soap to make it dissolve. The vat was filled with a 2 per cent solution, for which was used 50 gallons of Chloro-Naphtholeum and 40 pounds of soap. A couple of 2-year-old heifers were dipped to test the effect of the solution upon the animals. As these did not become affected in any way, 40 head of Kentucky cows, all very ticky, were dipped. The result was a surprise. Twenty-four hours after the dipping all the smaller ticks were dead, and likewise a great number of the fully gorged ones. The following day all ticks still alive had become soft, wrinkled, and of a yellow, mottled appearance. When picked off they were, however, still able to move. A number of them were gathered and placed under a glass cover in a warm room, but no eggs appeared, and after two days they were all dead. The same was true of those remaining on the cattle. They all turned black and died. When bloated ticks shrink, they seem to be unable to leave the cattle, even though they were ripe and ready to drop off immediately before the dipping. When they die they remain attached to the animal until the mouth parts are expelled, probably by suppuration. There is, however a possibility that a tick which remains alive for several days after the dipping, even though it be shrunk and discolored, may revive, but such a case has not been observed. During the following three weeks more than 300 head of cattle were dipped in the same solution. None of them became stiff, and only a slight peeling off of the epidermis between the hind legs and on the sides of the neck indicated that they had been dipped. Many of these animals were calves less than half a year old, and they seemed to endure the dipping as well as the grown animals. The ticks were all destroyed, though some of them remained alive four or five days after the dipping.

Experiments with this disinfectant will be continued, and it is to be hoped that it will fulfill its promise of efficacy. It seems to retain its tick-destroying properties much longer than the Lone Star Wash does, and animals of different ages and condition may be exposed to it with little discrimination.

For ranch purposes both of these disinfectants may be used to advantage when dissolved in solutions of such strength as will have no ill effect on the animals. Especially may they be used as a treatment and preventive against mange in cattle. This fact is of great importance to all ranch owners, especially in the western part of Texas and in the Panhandle, where the cattle suffer extensively from psoroptic mange during the colder season of the year. If the cattle on all the larger ranches were regularly subjected to such disinfecting baths, it would necessarily be beneficial to their general health, and by freeing them from lice, ticks, and mange would make them develop faster and fatten quicker. This is carried on to a great extent on this ranch, and the benefit derived from it can not be overestimated, especially as far as the thoroughbred and improved stock is concerned. Durham cattle are almost invariably infested with ticks and are also very susceptible to mange, but by means of a couple of dippings during the winter they may be kept entirely free from both.

INVESTIGATIONS OF DISEASES OF DOMESTICATED ANIMALS.

By THEOBALD SMITH, Ph. B., M. D.

NOTES ON SPORADIC PNEUMONIA IN CATTLE; ITS CAUSATION AND DIFFERENTIATION FROM CONTAGIOUS PLEURO-PNEUMONIA.

Though contagious pleuro-pneumonia, the great scourge of the bovine species, has been successfully rooted out from the United States, it has been of not infrequent occurrence since to have animals shipped from this country condemned in England as affected with this disease. Differences of opinion concerning the character of certain cases of lung disease in cattle have been frequently offered, not only in England but also in this country, and it must be truly said that no unanimity exists as to what is and what is not contagious pleuro-pneumonia. In view of this state of affairs it was deemed desirable to publish some observations made during the past eight years in the hope that perhaps a little light may be shed on the subject, or at least a stimulus given for a more exhaustive study of the different forms of lung disease in cattle and their causation, and for the development of a more rational view concerning the etiology of the lung disease in cattle leaving our ports. In the absence of any positive knowledge concerning the cause of contagious pleuro-pneumonia certain forms of lung disease will continue to remain obscure and a source of doubt wherever the contagion is known to exist; but there are other forms which it appears to the writer do not come under this category, but may at once be regarded as nonspecific forms of disease.

The following studies were made entirely upon lungs received from different parts of the country. Some were obtained from cattle shipped to England and there condemned as affected with contagious pleuro-pneumonia. In many cases only portions of lungs were received. In others the entire lungs were accessible, and from a few of such complete specimens much suggestive information was obtained. The investigation consisted in a careful inspection and dissection of the lungs, in the microscopic examination of teased preparations and secretions of fresh lung tissue, of sections of tissue hardened in various ways, of bacteriological examinations, and of inoculations of isolated bacteria and portions of tissue into animals to determine pathogenic activity.

The kinds of lung disease which have been encountered may for convenience be grouped under the following heads:

1. Simple broncho-pneumonia.
2. Broncho-pneumonia with interlobular emphysema.
3. Broncho-pneumonia with interlobular exudate.

4. Traumatic pneumonia.
5. Contagious pleuro-pneumonia.[1]

To these might be added cases of actinomycosis of the lungs, which do not come within the scope of the present discussion.[2]

SOME CHARACTERS OF SPORADIC PNEUMONIA.

Simple broncho-pneumonia, or, indeed, any diseased condition of the lungs, is not so common an occurrence in cattle as might be supposed. Of 200 animals killed and examined for tuberculosis after the tuberculin test, I found but 1 animal with a single lobe affected.[3]

In introducing this rare though well-known lung affection here my object is to trace the evolution of the peculiar, often striking, interlobular changes associated with broncho-pneumonia which give rise to a regular marbling of the cut surface of the lung tissue, and which are without doubt frequently referred to contagious pleuro-pneumonia as the cause. In using the word marbling I restrict it for the moment to the presence of a network of whitish interlobular bands. The variegated color of the cut surface of lungs affected with acute pleuro-pneumonia to which the term marbling is sometimes applied does not appear in any other type of lung affection.

Simple broncho-pneumonia may be limited at the outset to the small ventral lobes on one or both sides. As the disease progresses it tends to extend to the cephalic lobes, and lastly to the large caudal lobes. This extension forward as well as backward (in the standing position of the animal) is almost invariably symmetrical on both sides, as shown by the shaded area in Pl. IV. The invaded portions of the lungs are, moreover, the lowest or most dependent portion of the lungs. This can easily be seen by holding the lungs in the position they occupy in the standing animal. The extension of the disease forward and backward is always accompanied by an extension upward toward the back of the animal, where a strip of air-containing lung tissue may be found after death as the only portion still performing its function to the last.[4]

In the early stages of this lung disease the appearance of the diseased lobe is usually as follows: The affected portions are slightly, if any, larger than the healthy lobes in the collapsed state after the thorax has been opened. The color is dark red or grayish red, often mottled quite regularly with minute hazy, grayish dots. The lung tissue feels firm to the touch, often nodular, because of the presence of minute hard bodies embedded in it. These are groups of air vesicles greatly distended with catarrhal (cellular) masses. The diseased, red portion of the lobe is irregularly marked off from the air-containing portion, and not infrequently pervious lobules are found embedded in the hepatized lobes. (See Pl. XXXI, p. 388, of Special Report on Diseases of Cattle.) These lobules are then very much distended and emphysematous. When the air tubes of such a lobe are slit open they are, as a rule, found covered with whitish mucus mixed with pus. The mucous

[1] It is perhaps needless to add here that cases of pleuro-pneumonia have not come under observation since the close of the work of suppression.

[2] Special Report on Diseases of Cattle, p. 411, Pl. XL.

[3] This estimate excludes pneumonia due to tuberculosis and injury.

[4] In infectious pneumonia of swine (swine plague) the lungs are similarly affected. In fact, I have encountered only a few cases of lung disease in swine, not due to injury, in which the dependent portions of the lungs were not diseased first, and the dorsal or upper portion pervious to the last. (See Report on Swine Plague, 1891, p. 98, Pl. I–VI.)

membrane is of a dusky hue, often dotted with fresh hemorrhages. These remarks apply to the small ventral lobes in which the disease usually begins, and in which progress appears to be quite slow. When the large caudal lobes become affected the course of the disease is more rapid, for the lesions have a fresher, more uniform appearance throughout the attacked portion. In this advance to other lobes a new feature appears which is of considerable interest. Owing to the filling up of such large territories of lung tissue breathing becomes more difficult, and under any strain the labored respiration causes rupture of the air vesicles and the filling up of the interlobular connective tissue with air (emphysema). The distension of the connective tissue can also be traced in some cases along the perivascular sheath of the large intrapulmonary veins. The appearance of air bubbles under the pleura is also a common occurrence.

A further stage in the evolution of interlobular lesions is the filling up of the distended lymph spaces, wholly or partially, with a whitish coagulated lymph. The lymph spaces may be completely filled with whitish plugs, easily lifted out of the containing mold. Such plugs are very firm, and are torn to pieces with great difficulty. They consist almost wholly of fibrin. In other cases the cavities formed by the air are too large to be filled up entirely by the coagulated lymph. A thick coating may then form on the walls of the cavity and leave a central hole resembling the cavities in certain kinds of cheese. These conditions are well brought out in the drawings from alcoholic specimens in Pl. V. The interlobular bands are more or less honeycombed with cavities of various dimensions. We may thus have some cavities unfilled, some completely and some only partly filled with the firm coagula. The impossibility of a reduction of the distended spaces which have been formed in the connective tissue is due to this solidification of the lymph, which gives the appearance of a congealed condition. The lung tissue in these affected lobes becomes rigid. Normal collapse is no longer possible, even though the lung tissue itself remain unaffected. This condition is shown on Pl. V. The lung tissue here represented is entirely pervious.

It is evident from what has been stated that these interlobular plugs and bands may arise in the absence of any preexisting pneumonia when the emphysema has been extensive and the flow of lymph is retarded. The cause of the coagulation is probably the presence of air, together with bacteria, and possibly catarrhal secretions from ruptured air vesicles. When broncho-pneumonia is present these elements combined seem to be more than sufficient to account for the coagulation and other changes following the coagulation to be referred to below; when broncho-pneumonia is not present a temporary delay of the lymph flow may account for the coagulation of the stagnating lymph.

The distention of the interlobular tissue with air and the subsequent filling up or thrombosis of the spaces is nearly always limited to the still air-containing or freshly hepatized lung tissue. Rarely are plugs found among the lobules of the small ventral lobes in which the hepatization is of long standing. This is to be accounted for by the fact that the emphysema naturally occurs where respiration is most active, i. e., in those lobes where the parenchyma is not yet filled up, and where the greatest expansion takes place during inspiration. The marbling is thus most frequently found in the large caudal lobes where hepatization is still absent or newly formed. A few illustrations of the conditions here described are given below. In the following the

interlobular changes are mainly emphysematous, with but little plugging or exudation:

Lung A, January 2, 1891.—Both lungs of a cow received from New Jersey this morning. In both the hepatization was quite extensive and restricted to the anterior (cephalic) half. In the right lung the larger division of the cephalic lobe has the dependent half hepatized, while only a small region of the other division is solid. In this lung the ventral, median or azygos, and the adjacent fourth of the large caudal lobe are also hepatized.

In the left lung a small part of the cephalic, the entire ventral, and the adjacent fourth of caudal lobe hepatized. There was no pleuritis.

Throughout both lungs, but especially marked in the nonhepatized portions of the caudal lobes, the interlobular tissue is greatly distended with air, so that the individual lobules are in part separated from one another by imprisoned air and readily dissected out. The lungs are thereby greatly enlarged in appearance. These spaces contain not infrequently firm whitish coagula (lymph thrombi). The hepatized tissue is of a bright red color mottled with faint grayish or yellowish dots. It is quite firm and nodular to the touch, the nodules evidently corresponding to the grayish or yellowish dots, which in turn represent the subpleural, ultimate air spaces distended with exudate. This was composed chiefly of large granular cells, mixed with blood corpuscles. In preparations stained with alkaline methylene blue no bacteria were detected. The description given applies to all of the hepatization, excepting about one-quarter of the median or azygos lobe, which is smooth, firmer, and yellowish in color, and in which the exudate consists entirely of pus cells (polynuclear leucocytes). This evidently represents a more advanced stage of the disease.

Trachea and bronchi have the mucosa coated with a thin layer of fine white foam.

Portions of both the red and the yellow hepatized regions were hardened in alcohol and sections prepared. In those from the red hepatization perhaps the most obvious fact observed was the occlusion of the minute bronchioles with cell-massed and the peri-bronchial cell infiltration, causing a considerable thickening of the walls of the smaller air tubes. The alveoli around these bronchioles, as well as other groups of alveoli not traceable to air tubes in the sections examined, were fully distended with cells, while outside of these a zone of alveoli were free from exudate, but more or less collapsed.

In sections from tissue having a more uniformly yellowish color the lesions were somewhat different, in that there were small foci in which the alveoli were very densely packed with cells, while outside of these, and sharply separated from them, were areas in which the alveoli contained fibrin and very few cells.

The interlobular tissue is broadened and composed of a very delicate network of fibrin inclosing isolated cells in its meshes. These cells are not massed at any place, but indifferently scattered throughout.

The following case is one of broncho-pneumonia, showing the usual symmetrical distribution of lesions, upon which are superimposed exudative and suppurative changes in the interlobular tissue. (See Pl. VI, figs. 1, 2 and 3.)

Lung No. 30, March 12, 1892.—Lungs received to-day in good condition, free from any odor of decomposition.

The pneumonia is more or less generalized; pleuritis slight. In the right lung the dependent free portion of the cephalic lobe, the entire ventral lobe, and about one-third of the caudal lobe (latero-cephalic region) involved.

In the left lung the distribution of the disease is the same, but the amount of disease in each lobe is less than in the right lung. In the caudal lobe there are, in addition, several isolated pneumonic foci. The median or azygos lobe is likewise involved. There is more or less subpleural emphysema over hepatized regions. The lesions in this lung are both parenchymatous and interlobular. The parenchymatous changes are due in part to a broncho-pneumonia, in part secondarily to the rigid condition of the lung tissue following the formation of an interlobular exudate. To the naked eye the parenchyma is pale red and mottled with closely set grayish or pale yellow dots. In sections of hardened tissue these appearances are interpreted as minute broncho-pneumonic foci. The small bronchi are plugged with cell masses, the peribronchial tissue is infiltrated, and there is more or less filling up of the neighboring alveoli with cell masses. The mucosa of the larger air tubes is coated with more or less brownish-red, viscid mucus. When this is removed the subjacent mucous membrane is reddened in points and arborescent lines.

The interlobular disease is disseminated over the greater portion of the lung tissue, but is most prominent in the large caudal lobes. It may or may not be associated with hepatization of the parenchyma. It appears as mere interlobular emphysema, with a slight thickening or opacity of the connective tissue, and as an exudate filling out the cavities formed by the emphysema. The exudate appears as grayish, somewhat translucent bands about 1 to 2 millimeters thick. In the course of these bands enlargements occur. The bands inclose removable plugs, the outer zone of which is yellow, the inner pinkish-gray, and of a soft, gelatinous texture. When removed the plug is found composed of a very firm, elastic mass, coated with a thin layer of leucocytes. The mass of leucocytes (or pus) contains many very minute bacteria. In sections of hardened tissue these suppurative lymph thrombi are easily studied. The grayish bands which inclose them are made up of immature connective tissue. The line suppuration is indicated as a very dense layer of leucocytes, in part disintegrated. Within this the fibrinous nature of the plug is still recognizable. The firm connective tissue character of the bands is further demonstrated by the fact that the parenchyma can be scraped away from it, leaving the whitish neoplastic partition behind.

Though most prominent in the caudal lobes, the interlobular exudate is present in the other lobes as well. In the left caudal lobe one of the interlobular spaces is nearly half an inch wide, and contains in a length of 2 or 3 inches three suppurative foci. (Pl. VI, fig. 2.)

The following cases are of special interest. The lesions in the lungs of one very closely simulate the gross appearances of contagious pleuro-pneumonia so far as the interlobular disease is concerned. In the other, interlobular disease was absent. Both were primarily affected with broncho-pneumonia. Both animals had been subjected to the same conditions. On March 20, 1893, they were taken from the Union Stock Yards at New York to a stable in Secaucus, N. J., 3 or 4 miles distant. Probably this journey gave rise to the emphysema and the subsequent interlobular changes.

Lung No. 46.—Lungs received at the laboratory March 31, 1893, in a perfectly fresh condition. They were extensively diseased, the distribution of the diseased regions being that of the ordinary broncho-pneumonia. The dependent portions of the small ventral and cephalic lobes of both lungs and the portion of both caudal lobes adjacent to these were involved. The lesions vary in different parts of the lungs. The broncho-pneumonia is most advanced in the dependent ventral lobes and interlobular disease is absent, if we except some subpleural emphysema. In the cephalic lobes the broncho-pneumonia is more recent, and there is associated with it the distention of the interlobular tissue with firm, removable, fibrinous plugs. In the large caudal lobes the process is still more recent. The broncho-pneumonia is just beginning to appear, while the interlobular disease is extensive and well advanced toward suppuration.

If we take the ventral lobes as a starting point of our description, as they are undoubtedly the focus whence the disease has spread into the other lobes, the subject may be presented in the clearest manner.

The ventral lobes of both sides are in a state of pale-red hepatization, only slightly enlarged beyond the normal collapsed stage. The pleura is not affected. There is some subpleural emphysema. From the surface the parenchyma has a mottled appearance and is somewhat granular to the touch. On section it appears red, mottled uniformly with grayish dots which coalesce in some places into larger grayish masses. Some of these appear to be undergoing necrosis. The interlobular tissue is very slightly œdematous and contains occasional air bubbles. The larger bronchi are in general free from any exudation or secretions.

In sections from one of these lobes hardened in alcohol the minute air tubes are seen plugged with cell masses consisting largely of leucocytes intermixed with desquamated epithelium. The submucous tissue of the air tube is hyperplastic and the alveoli in the immediate neighborhood filled with leucocytes and desquamated epithelium or with fibrin.

The parenchyma outside of these broncho-pneumonic foci is in certain regions partly, in others wholly, collapsed. In the former the alveolar walls are distended, the alveoli containing either a finely granular material or else a few large cells from the alveolar walls. In the collapsed regions the walls of the alveoli are much broadened. The epithelium is prominent, desquamating, and the capillaries distended.

The cephalic lobes appear to be involved in a more recent disease. The subpleural lymphatics are plugged with fibrin. The lung tissue is pale red and very

œdematous. The interlobular tissue is distended with large firm masses of fibrin, of a yellowish white color and very firm. Some readily fall out of the incised interlobular cavities, which do not collapse, since their walls are thickened by fibrinous deposits. Others are more firmly lodged. The size of the cavities, and consequently the width of the interlobular bands, varies considerably, some being as much as one-half of an inch wide. In sections from one of these lobes the parenchyma shows fewer changes than those observed in the ventral lobes. The broncho-pneumonic foci are present, but the parenchyma is still nearly free from exudative changes. However, there is a tendency toward collapse with considerable broadening of the alveolar walls. The alveoli contain a finely granular substance.

The interlobular exudate, in the sections examined, consists of a homogeneous network of fibrin, in which cellular elements are sparsely but more or less uniformly distributed.

The caudal lobes presented some further changes. In the first place, the diseased region, limited to the cephalic third of the lobes, was distinctly enlarged and uniformly firm to the touch, in these respects resembling contagious pleuropneumonia. The surface has a distinctly mottled appearance, owing to the marked distension of the subpleural lymphatics with fibrinous plugs. This injection increases in prominence in the more dependent portions of the lobe. Subpleural emphysema appears over the still healthy adjacent regions of the lobes.

On section, the interlobular tissue at once arouses attention (Pl. VI, fig. 4). It appears as intersecting whitish bands of varying width, the largest being about one-fourth of an inch in diameter. The appearance varies from band to band. The smallest have a homogeneous grayish appearance. The larger ones are characterized by zones of different color and consistency. Thus the center is represented by a homogeneous, semitranslucent mass, outside of which is a pale yellowish border about one-half of a millimeter in diameter. Adjacent to the parenchyma is a thin, grayish, partly translucent border.

The parenchyma is more or less uniformly red, œdematous, and faintly mottled with grayish dots. In the main bronchus is a rather firm, grayish mass containing fibrin, blood, and many leucocytes.

In sections of hardened tissue from this region minute broncho-pneumonic foci are visible to the naked eye. Under a high power the alveoli are seen in many places packed with leucocytes. These pneumonic regions are as a rule associated with a bronchus plugged with cell masses. The remainder of the parenchyma contains alveoli, either empty or filled with fibrin. The interlobular exudate is composed of several zones. The most prominent one, readily visible to the naked eye as a deeply stained band, is a dense zone of leucocytes. This surrounds a central mass of fibrin containing some scattering cells, and is in turn bounded externally by another zone of fibrin. Inclosing this whole mass is a narrow zone of embryonal connective tissue attached to the walls of the contiguous alveoli. In the smaller lymph plugs the mass of leucocytes may be situated centrally.

Lungs No. 47, received with No. 46.—They appear very large. The distribution of the pneumonia is the usual one of broncho-pneumonia. In the right lung the free half of the cephalic and of the ventral lobe and the latero-cephalic fourth of the caudal lobe is hepatized. In the left lung the affection is equally extensive. In the caudal lobes beyond the pneumonic region there are scattered hepatized lobules surrounded by air-containing tissue. One-half of the median lobe is hepatized. Over the lateral aspect of the left lung there is a marked fibrinous deposit fully one-eighth of an inch thick, gluing the various lobes together. The pneumonia is of the usual mottled type, i. e., a red flesh ground in which pale yellowish dots are crowded. Contents of main bronchi as in No. 46.

The large size of the lungs is due to a very extensive interlobular emphysema of the large caudal lobes and to a certain extent of the air-containing portions of the other lobes. Exudation into the cavities thus formed and subsequent suppuration thereof, as in No. 46, has not yet taken place in these lungs.

Sections of tissue from three different regions of this lung hardened in alcohol were carefully examined.

(1) In the left caudal lobe, near the diaphragmatic surface, the small air tubes up to 0.4 mm. in diameter are plugged with dense cell masses, consisting of a mixture of leucocytes and epithelial elements, the former greatly predominating. The epithelium is still present. Among and below the epithelial cells are many polymorphous leucocytes. The peribronchial tissue is in a state of active hyperplasia. The parenchyma, viewed with the naked eye, has a mottled appearance due to the broncho-pneumonic foci just described. Around the plugged bronchi there is a zone of alveoli distended with leucocyte elements, and intermingled with

these a number distended with fibrin only. Outside of these foci the alveoli are largely collapsed and the walls broadened, owing to capillary distension. Only occasionally a partly collapsed air cell contains a group of leucocytes.

(2) In an isolated hepatized focus in the right caudal lobe, the broncho-pneumonic foci, which consist of cellular plugs in the air tube with the surrounding zone of alveoli filled with cell masses or with fibrin, are the same as in the sections from the left caudal lobe. The alveoli beyond these are not as a rule collapsed and the walls nearly normal. They are either empty or partly filled with finely granular material or contain a few desquamated epithelial cells.

(3) In sections from more advanced disease in the median lobe we observe the same broncho-pneumonic foci. They are not so demarcated, however, from the rest of the parenchyma, because the latter are quite generally collapsed and the alveolar walls much broadened. The peribronchial infiltration is likewise more extensive than in the more recent disease of the caudal lobes.

The foregoing cases show sufficiently well the different stages of lung affections from simple broncho-pneumonia to broncho-pneumonia associated with emphysema and finally interlobular thrombosis, inflammation, and suppuration. How far the cases with interlobular disease could be mistaken for contagious pleuro-pneumonia would depend upon the training, skill, and bias of the person upon whom the diagnosis depends.

The interlobular conditions described may appear in any form of infectious lung disease, as, for instance, in contagious pleuro-pneumonia, in addition to the very characteristic interlobular and perivascular lesions of this disease. If my theory of their causation is correct, any disturbance which throws all the work of respiration upon a portion of the entire lung tissue might give rise to these interlobular changes. It is therefore likely to happen that these interlobular affections are regarded as the result of contagious pleuro-pneumonia, because they may be present in this disease. It should be borne in mind that they are nonspecific lesions, the result of mechanical causes and secondary bacterial infections mainly.

SOME CAUSES OF SPORADIC PNEUMONIA.

The primary causes of the sporadic pneumonia (leaving aside all those which are manifestly infectious, and which usually attack many animals at the same time) are of several different kinds. Among these, exposure to cold, by which congestion of the mucous membrane of the air tubes may be produced, figures conspicuously in treatises on animal diseases. I have, in view of such possible causation, examined carefully the air tubes, as far as was possible with the naked eye, of all animals slaughtered after the tuberculin test, especially those killed in winter. It was a surprise to me to find very little inflammation of the bronchial tubes. Occasionally a little mucus was found in the terminal portion of the bronchi of the large caudal lobes where broncho-pneumonia does not begin, and to which it rarely extends when begun. Only when tuberculous changes had partly or wholly occluded a bronchial tube, catarrhal secretions were usually present in that tube only. Extensive tuberculosis of the lungs is not frequently associated with broncho-pneumonia, but in cases of this kind the latter is more obviously secondary to the former. We must look, therefore, for other causes than those of exposure, and among these the escape of foreign substances into the air tubes is probably the most frequent. In the administration of medicines, fluid or otherwise, there is much danger of an escape into the air tubes. The fluid will naturally gravitate to the most dependent portions and set up disease

there first. These dependent portions are, first of all, the small ventral lobes, next, portions of the cephalic and caudal lobes, as shown in Pl. IV. The amount and nature of the disease produced will depend on the quantity and character of the fluid which escapes into the lungs. If it is acrid or corrosive, a severe bronchitis with broncho-pneumonic changes will take place. If containing suspended matters, the fluid will probably produce occlusion of the smaller air tubes followed by broncho-pneumonia. The writer some years ago saw portions of a cow's lung in which the minute air tubes were completely occluded with a greenish, powdery substance. The affected lobes were collapsed and of a red flesh color. The persistence of the disease started by accidents appears to be due to the pathogenic bacteria of the throat washed down with fluids or other substances into the air tubes, as will be pointed out further on. The subsequent extension of the disease is most likely due to the aspiration of catarrhal secretions mingled with these pathogenic bacteria from the diseased into healthy air tubes. The distribution of the disease shows this mode of extension to be highly probable, for it is always governed by gravity, and the dorsal or uppermost portions of all lobes remain pervious to the last.

Other mechanical causes of lung disease in cattle by no means uncommon may here be mentioned for the sake of greater completeness.

It is a well-known fact that a local thickening or increase of the interlobular tissue into a network of white bands may occur during all inflammatory affections due to foreign bodies which penetrate into the thorax from the second stomach and pierce one of the lobes of the lungs. The resulting abscess is always surrounded by a zone of dense hepatized lung tissue permeated by bands of firm, whitish tissue. Thrombi or cavities in the interlobular lymph spaces I have not seen in such cases.[1]

Another form of injury which is likely to give rise to the diagnosis of contagious pleuro-pneumonia are blows upon the chest. In the transportation of cattle in vessels, the occurrence of such injuries during stormy weather would appear to be hardly avoidable. A severe blow upon the thorax might give rise to inflammation of the pleura, with laceration of tissue and hemorrhage into the lung tissue, which would lead to interlobular exudation and lymph thrombosis. The accidental aspiration of any pneumonia bacteria would add to the lesion by causing suppuration of the interlobular tissue. The existence of an evidently croupous inflammation of the lungs and interlobular exudate, and the absence of an initial broncho-pneumonia, might mislead an inspector into the diagnosis of contagious pleuro-pneumonia, especially if the personal bias were in that direction. These lesions should be thoroughly studied where exported cattle are being slaughtered, and the resemblances and differences between them and contagious pleuro-pneumonia definitely determined and published for the use of the inspectors. Such studies would sooner or later be accepted by foreign authorities and redound to the protection of American interests.

[1] E. Semmer (Deutsche Zeitschr. f. Thiermedicin XIII (1887) S. 187) observed a case of interstitial pneumonia in a steer resulting from castration. The animal died six days after the operation. At the autopsy Semmer found the scrotum inflamed and the inflammation extending along the subcutis of abdomen and thorax. The peritoneal, pleural, and pericardial sacs contained much yellow fluid. The lungs were dark red but not hepatized, and the connective tissue inflamed and distended with a yellowish exudate. Semmer regards the interstitial pneumonia as the direct result of the infection caused by the operation.

THE BACTERIOLOGY OF SPORADIC PNEUMONIA.

It has already been stated that the causation of isolated or sporadic cases of lung disease in cattle is probably in all cases some mechanical or chemical agent to which certain pathogenic bacteria are superadded, which modify and prolong the disease. Their relation to these agents is probably similar to that existing between the tubercle bacillus as the primary agent of tuberculosis in the human lung and the streptococci as secondary invaders. The latter are the cause of more rapid destruction of tissue and of general constitutional disturbances, such as fever. In this section the bacteriological studies of sporadic pneumonia will be given mainly to elucidate the lesions simulating contagious pleuro-pneumonia. The relation of the bacteria found in sporadic pneumonia to epizootics of infectious pneumonia will be briefly reviewed in the following section.

The bacteria present in diseased lungs are not infrequently present in the mouth and upper air passages of healthy cattle. Experiments demonstrating this fact have been published by Dr. V. A. Moore in the writer's report on Swine Plague (p. 152) as far back as 1891. This point should be borne in mind, as it accounts for the presence of these bacteria in the air passages, lung tissue, and interlobular spaces of diseased lungs. The source of the pathogenic bacteria in sporadic pneumonias is thus an unfailing one, and always accessible. To facilitate the reader's comprehension of the following pages I might anticipate a little by stating that the pathogenic bacteria associated with these pneumonias are closely related and probably identical with the bacteria of swine plague (infectious pneumonia in swine).

When, as explained above, any severe strain or accidents have occurred which cause labored efforts at breathing, the pathogenic bacteria living in the mouth and pharynx may be drawn into the air tubes and thence into the ultimate air cells. From these they may be aspirated into the lymph spaces of the interlobular tissue together with air and fluid through ruptures in the lung tissue. In this way, emphysema, followed by the coagulation of lymph, which becomes mixed with pathogenic bacteria, takes place. The presence of the pathogenic organisms in the interlobular exudate is demonstrated in the following two cases, which at that time were regarded as affected with contagious pleuro-pneumonia, although there were equally good reasons for considering the region from which they had come as free from this disease.

Case 1, December 22, 1887.—About one-third of one lung accessible to examination. The lesions in this portion were limited to the pleura and interlobular tissue. The pleura was nearly one-eighth of an inch thick, opaque, whitish; the interlobular tissue was thickened from one-quarter to one-half of an inch in width on section, whitish, firm, forming long trabeculæ, the lateral branches of which were but slightly infiltrated. The most striking feature of these interlobular bands was their honeycombed condition. They inclosed cavities about the size of a pea or bean, communicating with one another. They were so numerous as to comprise perhaps two-thirds of the space occupied by the newly formed interlobular bands. Most of the cavities were empty. The remainder contained a rather viscid, translucent mass, or were filled out by molds of solid, whitish material. The parenchyma of the portions examined was not affected.

December 23. From the lung tissue kept on ice since yesterday cultivations were made by thoroughly scorching the surface of the lung along the interlobular bands with a red-hot platinum spatula. Along this scorched area an incision was made with flamed scalpels and the contents of the honeycombed bands carefully removed with platinum loop and placed in six tubes, three containing peptonized

bouillon, and three blood serum. Several roll cultures, made at the same time, melted during the night.

December 24. In all the tubes a faint growth had appeared made up of very minute cocci, not over 6 μ long, usually single, rarely in pairs. The peptone bouillon was faintly clouded, the growth on the blood serum very feeble. In the former a capsule could be seen surrounding the cocci when examined in a hanging drop. By focusing on the edge of the drop where the cocci were crowded together and where the layer of liquid was very thin, the capsule could be distinctly seen. The cocci themselves were separated by the width of the capsules. Stained, the capsules disappeared, and the cocci resembled very closely the swine plague and rabbit septicæmia organisms.

The pathogenic action of this germ was tested by inoculating a calf December 24 in the lungs through the chest-wall with 5 cubic centimeters of a peptone bouillon culture. The calf was dead the next morning. The only lesions observable were scattered hemorrhages in the thorax and abdomen under the serosa of the ribs, pericardium, and diaphragm. The cocci were detected in cultures from heart's blood.

From the blood of the calf a rabbit was inoculated by pricking the skin with a lancet. It died in five days. At the point of inoculation a little purulent infiltration and a few hemorrhagic points in the subcutis. Spleen very large, blackish and soft. In cover-glass preparations from blood and spleen numerous polar-stained cocci were found. Cultures confirmatory.

Case 2.—A lung from another case was brought to the laboratory late December 24. Owing to other work it was kept in the refrigerator until December 27 before cultures could be made therefrom. The lesions were the same in character as those observed in the first case, i. e., there was a considerable broadening of the interlobular and the subpleural tissue, but in this case the thickening appeared in spots rather than in bands, and, moreover, in the depths of the lung tissue. The infiltrated regions were rather firm and honeycombed, thus resembling certain kinds of cheese, both in texture and appearance. The largest cavities were nearly three-quarters of an inch in diameter, filled with a yellowish serum or with a gelatinous grayish substance. The septa branching from those which were infiltrated were in general not more than one-sixteenth inch thick, firm, cartilaginous in appearance. The parenchyma in the immediate neighborhood of the involved septa was hyperæmic. No hepatization in the portion of lung examined. A considerable number of culture tubes were inoculated in a manner similar to that described for the first case. The difficulty, however, was increased owing to the fact that it was necessary in some instances to cut through lung tissue before reaching the interstitial infiltration. Cultures were made from the contents of nine cavities, two or three tubes being used for each cavity. Blood serum, gelatine and peptonized bouillon were used as culture media. On the following day the same coccus found in the first case appeared in some of the cultures. Only one of the tubes remained pure, however, for after two or three days other germs appeared, among them streptococci and large and small bacilli. The coccus appeared in cultures from six out of the nine cavities, no other germ appearing in more than one or two of the cultures. The capsule of the coccus, taken together with its minute size and want of motility, made the determination of its presence under the microscope a matter of but little difficulty.

Cultures from this case were equally fatal to rabbits, as the following inoculations show:

January 4, 1888, 0.25 cc. of a peptone-bouillon culture was injected beneath the skin of the thigh of a rabbit of medium size. Dead January 9. At the point of inoculation a rather pasty infiltration of slight extent in the subcutis. In the abdomen the coils of intestines are lightly glued to each other and to chest wall by a fibrino-cellular exudate, which likewise covers the spleen and liver with a tenacious grayish membrane. Spleen large, dark, friable. Kidney diffusely reddened; urine contains much albumen. Few bacteria in the various organs; very many in the peritoneal exudate.

At the same time another rabbit was inoculated with the same culture by injecting one-eighth of a cubic centimeter into an ear vein. Dead in twenty-four hours. Head drawn back, rigor well marked. Slight fibrinous exudate on intestines in the forms of threads and fibrils, due probably to the large number of cysticerci free in the cavity and in the omentum. Numerous cocci in spleen and liver with distinct polar stain. Cultures from the spleen, liver, and blood of these rabbits contained only the cocci injected.

On December 29 a calf 6 months old was inoculated into the right shoulder by injecting subcutaneously 5 cc. of a peptone-bouillon culture. For several days following the inoculation there was a slight swelling at the place of inoculation,

with stiffness of the shoulder. The temperature rose 2° F. on the second day, but gradually subsided. On January 10 the swelling was the size of a hen's egg. The animal showed no other signs of disease, and remained well for months after.

January 7, 1888, a black calf was inoculated by injecting 15 cc. of a bouillon-peptone culture into the trachea. On the second day the temperature had risen 2½° F. No other signs of disease appeared, though the calf was watched for several months after. (These inoculations were made by Dr. F. L. Kilborne.)

As these cultures were made several years before I had any knowledge of the existence of certain pathogenic bacteria in the upper air passage of healthy cattle, and as these two lungs from which this bacterium was obtained were regarded as affected with contagious pleuro-pneumonia, I naturally enough fell into the temporary error of regarding these bacteria as the cause of this infectious disease, as had been done by Poels and Nolen. The examination of a few lungs affected with undoubted pleuro-pneumonia soon after promptly dispelled this hypothesis.

In the following notes it will be observed that the specific bacteria found in the cases just quoted may be absent from lungs affected with unquestioned pleuro-pneumonia, although their presence might be anticipated wherever marked dyspnoea occurred before death. They were not detected in Nos. 3, 4, and 5, but were found in case No. 6.

Case 3, December 31, 1887.—Cultures on various substrata were made from a completely solidified lung showing the lesions of typical acute pleuro-pneumonia. Two gelatin roll cultures made from a particle of the hepatized parenchyma remained sterile. Of three peptone-bouillon cultures from the interstitial tissue two remained sterile and one contained three organisms, a wavy bacillus, a streptococcus, and a butyric bacillus. Three blood-serum cultures from the interstitial tissue remained free from growth. After keeping the lung on ice until January 3, six peptone-bouillon tubes were inoculated each with a particle of partly organized pleural exudate. All but one remained clear. This contained a diplo-coccus. From the parenchyma minute bits of tissue were placed in three bouillon-peptone tubes. All became turbid and contained large bacilli, some motile, others not.

Case 4, January 6, 1888.—Lung of cow affected with contagious pleuro-pneumonia. The caudal lobe of the left lung was used for cultures. Of this all but the dorsal portion hepatized, of a dark red color and granular on section. The interlobular tissue is partly distended with serum, partly thickened and fibrous in appearance. The dorsal portion is very œdematous, the interlobular and subpleural lymph spaces distended with a pale yellowish serum. Tube and gelatin roll cultures were made from the œdematous and hepatized parenchyma, as well as from the interlobular tissue. Of the 14 cultures all but 1 remained sterile. This contained a diplo-coccus different from the coccus obtained from cases 1 and 2.

Case 5.—Portions of the lungs taken January 13, 1888, from a cow affected with contagious pleuro-pneumonia and kept near or below the freezing point until January 16. Pleura about 2 to 3 mm. thick, containing small cavities, empty or filled up with a gelatinous exudate. The interlobular infiltration is 4 to 5 mm. wide. When the lobules are torn apart, which is done with more or less facility, it is found that the interlobular thickening or exudate is represented by a deposit on the contiguous faces of the lobules, firm, whitish. Between these layers is a middle layer of exudate, which may be pulled out in shreds and which is rather tough, yellowish white. In other words, the interlobular exudate is more or less lamellar in its arrangement. In some portions of the lungs the interlobular tissue is similarly thickened and almost cartilaginous in appearance, containing empty spaces as large as split peas. The parenchyma more or less œdematous throughout the greater part of the lungs. In some places that part of the lobule touching the interlobular exudate is hemorrhagic, in others one-quarter or one-half of the lobule is hemorrhagic. Rarely a few contiguous lobules are completely hemorrhagic. In a few places the lobules are hepatized, of a pale red aspect on section. The interlobular tissue very slightly thickened and distended with serum. Judging from the portions brought to the laboratory, hepatization was of slight extent. Series of cultures were made from the interlobular exudate, from the pleura, and from the œdematous parenchyma. All but one remain free from any growth. This contained a large vibrio.

Case 6.—An acute case of contagious pleuro-pneumonia which died during the night of February 1, 1888. The entire left lung consolidated. Three tubes of

bouillon inoculated from bits of tissue taken from the pleura and subjacent tissue all contained the same coccus on the following day. This has no capsule, but is evidently identical with the coccus from cases 1 and 2, as the following inoculation shows: A rabbit inoculated subcutaneously from one of these cultures died within forty-eight hours. There was a slight amount of purulent infiltration at the place of inoculation. Plastic exudate covering spleen, liver, and coils of intestines of a very tenacious character and containing immense numbers of cocci. In blood they were few in number. In cultures from this rabbit the capsule appeared, though not so distinctly as in former cases.

In connection with these facts pointing to the occasional presence of these pathogenic bacteria in lungs affected with contagious pleuro-pneumonia, it is of interest to note that in 1884 Poels and Nolen, of Rotterdam, Holland, published a preliminary communication of investigations made by them of contagious pleuro-pneumonia, and in 1886 a more detailed report appeared.[1]

The authors describe a nonmotile coccus which they found in lungs of 60 head of cattle slaughtered because affected with the disease. The coccus was found in abundance in the exudate into the alveoli, in the serum of the lymph spaces before coagulation, and in the pleural effusion. It was not detected in the lungs of healthy cattle. The cocci averaged about 0.9 μ in diameter, and were provided with capsules. They grew quite well on gelatin and agar-agar, and also multiplied to some extent on potato. On gelatin the growth slightly resembled Friedländer's bacillus. On potato the growth had a moist, feebly yellowish appearance.

Rabbits, guinea pigs, dogs, and cattle were inoculated into the lungs directly through the chest wall. A certain number of the rabbits and guinea pigs were killed for examination and the remainder survived. In those killed slight pneumonic infiltrations were observed. Of the two cattle inoculated considerable lung disease with presence of the injected cocci was observed in one case.

Several dogs received injections of culture liquid into the trachea. They were killed about forty hours afterwards and numerous dark-red pneumonic infiltrations observed. It should be noted that in these various experiments with smaller animals control injections were made with harmless fluids, such as sterilized water, and no changes were subsequently observed.

Mice were also exposed to the spray of culture liquid. These became very ill and were killed in three days. The lungs of all mice were more or less affected with pneumonia.

Finally, 100 head of cattle inoculated with cultures of this coccus resisted lung plague in infected localities.

In summarizing the results of their work the authors are disposed to conclude that this coccus represents the cause of contagious pleuro-pneumonia.

It is evident that the frequency of bacteria in lungs affected with this disease should make us careful not to interpret all the various appearances of interlobular disease occurring in contagious pleuro-pneumonia as due to its specific cause alone until more information has been gathered.

The presence of these bacteria usually in pure culture in broncho-pneumonia with or without interlobular disease is demonstrated by the following bacteriological studies upon lungs which have been already briefly described in foregoing pages:

Lungs of Case A (see page 122).—From tissue involved in recent red hepatization two agar plates (A, B) were prepared by inoculating A with a small particle of

[1] Fortschritte d. Med. (1886), IV, S. 217.

lung tissue. This was obtained by thoroughly scorching the pleura and removing a particle from within and beneath the scorched area with flamed forceps and scissors.[1]

On plate A only ten colonies appeared; all alike. The surface colonies after three to four days were 4 to 5 mm. in diameter, round, convex, grayish, glistening. They were made up of very minute bacteria, identical with the pneumonia bacteria. They showed distinctly a capsule in the hanging drop.

January 28. A rabbit was inoculated by placing a particle of agar growth derived from a colony on this plate under the skin of the ear.

January 30. Rabbit very quiet; ear drooping.

January 31. Quite sick; head resting on floor of cage.

February 2. Dead this morning. On the ear a spot about 1 inch in diameter in which the skin is infiltrated with pus and dotted with ecchymoses. Very slight peritoneal exudate in the form of fibrils of coagulated lymph across coils of intestine. Spleen large, dark red, and still firm. Kidneys hyperæmic. Lower colon hemorrhagic. Lungs very œdematous, with dark-red foci. In both blood and spleen a very large number of bacteria, showing very well the polar stain with alkaline methylene blue. Agar tube cultures from blood and spleen both contained on the following day a rich, shining, grayish growth of the inoculated bacteria.

At the same time that the agar plates were prepared two peptone bouillon and one agar tube were inoculated directly from the lung tissue, either by adding some serum or some tissue obtained as described above. In these three tubes only the pneumonia bacteria subsequently appeared. A rabbit inoculated subcutaneously with a particle of lung tissue died in three days. The lesions were nearly the same as those described for the rabbit inoculated with the pure culture, slight peritoneal exudate in the form of fibrils of pale coagulum, enlarged, dark spleen, and œdematous, hyperæmic lungs. At the point of inoculation slight suppurative infiltration of the skin and subcutis. In stained preparations of blood and spleen pulp numerous minute oval bacteria, showing the polar stain distinctly. An agar and a bouillon tube inoculated with blood from the heart and two similar tubes inoculated with spleen pulp contained subsequently only the pneumonia bacteria. From the area of yellowish hepatization in the median lobe plate cultures (A, B) were prepared. Only one colony composed of streptococci appeared on A.

An agar tube inoculated directly from the same region contained one colony of motile bacilli. A bouillon culture inoculated in the same way contained only the pneumonia bacteria.

From the emphysematous interlobular tissue several tubes were inoculated as follows:

The still intact pleural covering was scorched away with platinum spatula, and from the little serum in the cavities thus exposed two agar and two bouillon tubes were inoculated with platinum loop. One agar and one bouillon tube remained permanently free from growth. One agar tube contained subsequently a viscid orange-colored growth in condensation water made up of micrococci. One bouillon tube contained motile bacilli.

In order to test the pathogenic power of the pneumonia bacteria on cattle the following experiment was made:

On March 16, from the culture derived from a colony of the lung plate and passed through a rabbit a flask of peptone bouillon was inoculated and placed in the thermostat. On the day following the bouillon, now uniformly clouded, was used to inoculate two young animals. Calf No. 84, about 11 months old, received 7 c. c. of the culture liquid into the lungs through the right chest wall. Cow No. 154 received the same quantity into the trachea in the region of the neck. No. 154 did not become sick. No. 84 was found dead early on the morning following the injection, that is, in about sixteen hours. At the autopsy the following pathological changes were observed; nostrils filled with reddish foam, and on moving the animal a considerable quantity of blood flows from them. Trachea and bronchi contain a frothy blood-stained liquid, and the mucosa is intensely reddened. The lungs partly collapsed when the thoracic cavity was opened. Both more or less hyperæmic. The various lobes of the right lung show occasional subpleural petechiæ. The two small cephalic (or anterior) lobes are very much congested, and from the cut surface a considerable quantity of frothy liquid escapes. In the

[1] The method used largely by German pathologists and bacteriologists of cutting into the lung tissue successively in different planes with different sterilized knives seems to the writer less safe, because the farther we go toward the root of the lung the more liable we are to cut large air tubes, and thus increase the chances of dragging bacteria from these over the cut surface.

caudal lobe about 2 inches from the cephalic edge and 1½ inches from the lateral edge is a firm, dark-red hepatized hemorrhagic mass involving several lobules, and extending from convex to diaphragmatic surface. This is evidently the point where the needle entered the lung tissue. In the left lung near the caudal border of the principal lobe a group of lobules in condition of bright-red hepatization surrounding the terminal portion of the left bronchus. Bronchial glands in part hemorrhagic. Extensive ecchymosis of the pericardium, slight extravasation on left auricle and ventricle. Large patches of extravasation on pulmonary artery and aorta. Heart cavities empty, the still liquid blood having escaped through the previously cut vessels. Spleen congested, dark red in color; under capsule a few hemorrhages. Other organs not markedly affected.

Lung No. 30 (see p. 122).—Cultures were made on inclined agar from the interlobular exudate as follows:

Nos. 1 and 2 from interlobular exudate of left caudal lobe, two different regions.

Nos. 3 and 4 from interlobular tissue of left ventral lobe.

No. 5 from interlobular exudate of right caudal lobe.

In all these tubes an abundant grayish growth appeared on the inclined agar the next day. In all but No. 2 only pneumonia bacteria could be detected. In this latter tube a motile bacillus was likewise present.

At the same time two agar plates were prepared from a bit of interlobular exudate from right caudal lobe. On the first plate only four colonies appeared, which were all found to be made up of pneumonia bacteria. On the second plate two surface colonies appeared made up of large cocci and tetracocci, respectively. Both were probably aerial in origin.

Two agar plates were also prepared from a bit of the hepatized parenchyma of the right ventral lobe. On the first plate only two colonies appeared, one made up of pneumonia bacteria, the other (yellowish) of micrococci. On the second plate several colonies, probably of aerial origin, appeared.

With a bit of tissue from the left caudal lobe a rabbit was inoculated under the skin of abdomen. It died in twelve days. There was locally quite extensive suppuration and suppurative pericarditis and pleuritis with grayish-red hepatization of the small cephalic lobes of the lungs. Cultures from this exudate remained sterile.

Another rabbit inoculated subcutaneously with a loop of an agar culture derived from a colony on the first set of agar plates died in four days. At the place of inoculation the skin and subcutis was infiltrated with pus. There was exudative peritonitis indicated by a marked ecchymosis of and a viscid pseudo-membrane on the large intestines, and by an exudate on liver and spleen. The exudate composed of fibrin, leucocytes, and immense numbers of bacteria which in agar cultures prove to be the ones inoculated.

Lung No. 46 (see p. 123).—Sections stained with aniline water, methyl violet, and decolorized in one-half of 1 per cent acetic acid were negative as regards the presence of bacteria. This is not surprising, because the demonstration of bacteria among the dense mass of leucocytes is well-nigh impossible without decolorization, and this is apt to decolorize the bacteria as well. Moreover, the presence of fibrin is associated with an abundance of granular precipitate. Again, the small number of bacteria which appeared in the cultures shows that living individuals were very scarce in these lesions. The cultures described in the text were from the following sources:

1. A piece of interlobular exudate from right principal lobe on inclined agar.

2. A piece of the parenchyma from the same region. On the following day an abundant surface growth and turbid condensation water containing only pneumonia bacteria on microscopic examination.

3. A piece of tissue including both parenchyma and interlobular tissue from another region on the same lobe on inclined agar.

4. From the same locality a small particle of the parenchyma on agar.

On both tubes 3 and 4 a few isolated colonies of pneumonia bacteria appeared on the following day.

5. A piece of interlobular exudate from the ventral aspect of the left principal lobe on inclined agar.

5a. A loop of the condensation water from tube 5 rubbed on inclined agar.

In tube 5 about 12 colonies appeared on the following day; in tube 5a only 1 appeared.

6. A piece of interlobular exudate with some parenchyma adhering, from the right ventral lobe, ventral aspect.

6a. Dilution of 6 as 5a of 5.

On tube 6 a considerable number of colonies appeared on the inclined surface. Tube 6a remained sterile. In these various cultures only pneumonia bacteria were

found. Two rabbits were inoculated subcutaneously with bits of tissue from the left caudal lobe. In both the temperature had risen to 104.2° and 104.8° F. on the fourth day. They slowly recovered and were chloroformed on the sixteenth day. Marked local subcutaneous suppuration was found at the place of inoculation, but the internal organs were free from microscopic changes.

Lung No. 47 (see p. 124).—Cultures all made on inclined agar in tubes.

Tube 1. Inoculated with a small piece of the parenchyma from left ventral lobe.

Tube 2. Inoculated from preceding with loop.

Tube 3. Inoculated with a small piece of parenchyma of right ventral lobe.

Tube 4. Inoculated from No. 3.

In tube 1 a confluent growth appeared, while in tube 2, inoculated from this, about six isolated colonies of pneumonia bacteria appeared. In tube 3 a similar confluent growth of apparently only this organism appeared, while tube 4, a dilution of No. 3, remained sterile.

The following case is one of interest; although unlike the three reported above, there must remain some doubt as to the contagious character of the disease, especially since the entire lungs were not accessible to inspection:

Case 8, March 28, 1889.—Portions of the lungs of a cow sent from New York. The cow is supposed to have calved several weeks ago. Was killed March 25. It was stated that three others in the herd showed the same condition of the lungs. The lesions are limited chiefly to the pleura and interlobular tissue. On the pleura are patches of an elastic membrane, peeled off with some difficulty, one-sixteenth of an inch thick. The interlobular tissue is thickened into bands one-sixteenth to one-fourth of an inch wide. The thickening is due to exudative layers on the contiguous faces of the lobules. Between these layers and forcing them apart are round and elongated masses of a firm yellowish-white exudate, which may be lifted out without difficulty, leaving the bands in a honeycombed condition. The lumps thus removed are so firm that they can scarcely be crushed with forceps. When rubbed on cover glasses, dried, and stained with aniline colors, a large number of leucocytes appear, among which are very many exceedingly small cocci, some showing the polar stain distinctly. These plugs vary in size from a pin's head to a large pea. They are not always opaque, yellowish-white, for in some places, on sections, the thickened, whitish sides of two consecutive lobules inclose between them a translucent, rather soft, gelatinous exudate easily crushed with the forceps. The interlobular tissue of all the lung tissue brought to the laboratory was in this condition. A portion of the lung tissue is also affected with broncho-pneumonia.

Portions hardened in alcohol were examined under the microscope. The interlobular exudate consisted of two distinct masses. The external layer was made up of interlacing fibrils of fibrin and very few cells. The inner mass or nucleus consisted almost exclusively of densely packed clumps of leucocytes. The sections when stained in alkaline methylene blue showed this condition very well. The nucleus of deeply stained cell masses is surrounded by a very clearly defined zone of unstained material representing the fibrin. Outside of this zone are the boundaries of the lobules. In other words, the lymph spaces have become the seat of multiple abscesses.

Cultures were made from the interlobular exudate as before. The media used were agar and peptone-bouillon. Owing to the very feeble precarious growth on gelatin of the cocci found in cases 1 and 2 and subsequently, gelatine was not employed. In the cultures the pneumonia bacteria, a streptococcus, and a motile bacillus appeared. At the same time two rabbits were inoculated by placing bits of the exudate from the lungs under the skin. Both rabbits died from the effects of the inoculation, one in less than twenty hours and the other in forty-eight hours. The local lesions were slight. There was no peritonitis. In the spleen of both animals are numerous cocci identical with those found in sections of the cow's lung and in cultures therefrom. From spleen and blood of both pure cultures of the cocci were obtained.

In order to test the pathogenic nature of this germ more fully, some other animals were inoculated. Two pigeons received each (on January 5) one-half of a cubic centimeter of a bouillon peptone culture, one subcutaneously, the other superficially, into the pectoral muscle. Both remained well. Two mice, on the same day, were inoculated subcutaneously, each with one-twelfth of a cubic centimeter of the same culture. One mouse died on the following day, with no bacteria demonstrable in spleen or blood under the microscope. The other remained well.

From agar cultures the growth was removed and suspended in sterile water

until a turbid, whitish liquid was formed. Of this liquid 2 c. c. was injected into the lungs of a heifer through the chest wall. Within half an hour the breathing became labored, and continued so until late at night, the injection having been made at 5.30 p. m. It was found dead and still warm early next morning. There were found on post-mortem examination extensive subpleural extravasations on the ribs of both sides, on the epicardium, endocardium, and diaphragm. Lungs very hyperæmic, bronchial glands with deeply reddened cortex. Extravasations also found on omentum and peritoneal covering of the abdominal organs.

In the two following cases, evidently not contagious pleuro-pneumonia, only portions of the lungs were accessible. The same bacteria were isolated from them.

February 25, 1890. Portion of a cow's lung was received from Baltimore, Md., in good condition. Two distinct stages of the disease are represented, a dark red, firmly hepatized, and a pale red œdematous region, distinct and sharply defined from one another. The pleura over the œdematous region shows interlacing grayish bands, which are subpleural whitish thrombi in the lymph spaces. By tearing away the pleura these thrombi are easily lifted out. Over the hepatized portion the pleura is thickened, but the subpleural mottling is the same.

On cutting into the œdematous portion much serum collects in the incision and speedily coagulates. The interlobular tissue is broadened and contains whitish lymph thrombi, easily removed. Some are as large as split peas. When crushed and examined in salt solution, as well as after staining, many leucocyte elements observed. The minute bronchi contain pale coagula, which can be pulled away as long elastic, sometimes forked, cylinders.

The hepatized portion is firm, the lobules dark red, surrounded by the interlobular spaces distended with whitish plugs. The condition of the interlobular tissue and of the minute bronchi is the same as in the œdematous portion. Fresh sections of this tissue show the redness to be due largely to the alveolar capillaries overdistended with red corpuscles. The aveoli are filled with cellular elements containing fat granules. Red corpuscles are few in number. The larger bronchi contain some serum. The mucosa faintly discolored.

In sections from material hardened in alcohol and stained in various ways the following features were observed: In that portion of the lungs which, in the fresh condition, appeared to be in a state of inflammatory œdema, the walls of the alveoli are somewhat broadened and more approximated than in normal lung tissue compared with it. Occasionally cell masses are seen in a single alveolus, or perhaps two or three together, and in the smallest air tubes. But these cell masses are small and do not fill the lumen of the tube or cavity containing them. The interstitial tissue is to a moderate extent distended with a finely granular material mingled with cells.

In the hepatized portion the alveoli are distended with fibrin and cell masses. Those containing cells chiefly appear in the sections as foci of variable dimensions, some of which stand in direct relation to bronchi which are densely packed with cells. Such bronchi are, in the sections, about 0.15 mm. in diameter. Surrounding these foci the alveoli are filled chiefly with fibrin containing a few cells. The peribronchial tissue is not infiltrated, and it seems as if the bronchioles were filled by cellular exudate coming from the alveoli.

The interlobular tissue is fully distended and the various lymph spaces filled with a meshwork of very delicate fibrils. In this meshwork cellular elements are scattered throughout in small numbers, not localized anywhere. There is as yet no formation of connective tissue visible.

From the hepatized portion cultures were made in the following manner:

The pleura was thoroughly scorched and in this scorched area a piece of lung tissue cut out with flamed scissors. From this hole another piece was cut out and stirred up in melted agar. In transferring to a second tube of melted agar the bit of lung tissue clung to the wire and was transferred. The two plates thus prepared showed on the following day large numbers of identical colonies, which were made up of minute coccus-like forms showing a distinct capsule in the hanging drop. From one of these plates a peptone bouillon and an agar tube were inoculated. The bouillon was faintly clouded next day with minute, nonmotile bacteria. In the agar tube only the condensation water contained any growth which was composed of the same bacteria.

While the agar plates were being prepared, a bouillon and an agar tube were inoculated directly with a loop of serum from the depths of the hole cut into the lung tissue. The bouillon became clouded on the following day with the same bacteria. On the agar surface a rather abundant grayish growth of the same organism appeared. In neither tube could other bacteria be detected.

From the œdematous portion plate cultures were prepared in the same manner; on Plate A, about forty colonies, about half of these covered by a surface of viscid consistency and made up of plump bacilli in chains not exhibiting any movements. The deep colonies are composed of plump, short, motile bacilli. Second plate similarly overgrown. The motile bacillus was subsequently found to be spore-bearing and to liquefy gelatine. While making the plate cultures, a peptone bouillon and an agar tube were inoculated directly with a loop of serum. Both remained sterile.

From the same portion kept in the refrigerator for twenty-four hours another set of agar plates were prepared. Plate A was completely overgrown on the following day with the same bacillus. Beneath this growth about thirty deep colonies are visible. Two of these, examined by carefully scraping away the surface growth, were found to consist of the minute bacteria showing capsules. Plate B remained sterile.

At the same time that cultures were being prepared two rabbits were inoculated subcutaneously with particles of hepatized lung tissue. Both were found dead within forty hours. In one animal there was some hyperæmia and ecchymosis in the subcutis at the place of inoculation with slight purulent infiltration; spleen engorged; invagination of lower portion of colon. In cover-glass preparations from spleen, liver, and blood bacteria with polar stain and closely resembling swine-plague bacteria present. Cultures in agar, gelatine, and in bouillon developed within twenty-four hours, all containing the same oval, nonmotile bacteria. In the other animal the post-mortem appearances were the same, but there was no invagination. Cultures also were the same.

One rabbit which received subcutaneously one-fourth of a cubic centimeter of a bouillon peptone culture from preceding rabbit died within twenty hours; considerable extravasation at point of inoculation in subcutis. From an agar culture of the foregoing rabbit a fresh rabbit was inoculated with a loop inserted under skin of ear. This rabbit succumbed on the fifth day; at the point of inoculation a small abscess; fibrinous and cellular exudate on peritoneum and pleura containing large numbers of the oval bacteria; cultures on agar and in bouillon confirmatory.

February 20, 1891. Portions of diseased lung tissue from a cow were received to-day from New York. The portions sent are evidently the smaller ventral or cephalic lobes and slightly enlarged beyond the normal. The firm hepatized tissue is pale red and along the borders of the lobes there is faint yellowish mottling. The tissue gives a slight nodular sensation to the touch indicative of considerable distension of the ultimate air spaces with firm masses. They are shown to be chiefly leucocytes under the microscope; the same pale red appearance present on the cut surface, together with the localized mottling referred to. The section rapidly fills up with serum. Certain areas of the hepatized lobes also show subpleural thrombosis of the lymph spaces with grayish-white coagula, giving the particular area involved a mottled appearance, differing from the mottling above described. These lymph thrombi are also found in the interlobular tissue and are easily pulled out. The mucosa of the bronchi contained within the diseased lobes is intensely hyperæmic, in some places hemorrhagic.

The bacteriological examination was restricted to some agar plates, tube cultures, and inoculation of rabbits from the hepatized lung tissue. A tube of agar was inoculated with a minute particle of lung tissue for Plate A. From this several loops were transferred to a second agar tube for Plate B and to a gelatine tube for Roll B. Both the latter remained sterile, while a large number of colonies appeared on Plate A within twenty-four hours. These were of two kinds, chiefly small colonies of capsule bacteria and a few large colonies not examined. From the same bit of lung tissue two particles were placed under the skin of two rabbits. One died in sixty hours. At point of inoculation the vessels of the under surface of skin were distended and tortuous, with extensive punctiform ecchymosis around them. In abdomen a portion of the cæcum and upper colon ecchymosed, slight fibrino-purulent exudate on coils of intestines and on liver; fatty condition of liver and kidneys; lungs normal.

In peritoneal exudate very many minute oval bacteria; in blood and spleen a smaller number. Those of the blood and spleen show the polar stain. In agar and bouillon cultures, from blood and spleen, the polar-stained capsule bacteria, and these only, subsequently appeared.

The second rabbit was found dead on the fourth day. The lesions were the same as those just described, with the exception that the peritonitis was further advanced and the various abdominal organs covered with a fibrinous and cellular membrane. The cæcum and colon were extensively ecchymosed. The bacteriological examination and the cultures gave precisely the same results as in preceding case.

DESCRIPTION OF THE BACTERIA ASSOCIATED WITH SPORADIC PNEUMONIA.

With rare exceptions only a certain kind of bacteria was encountered in the diseased lung tissue. These are closely related to the swine-plague bacteria and hence to the entire group of pathogenic bacteria denominated by Hüppe *Septicæmia hemorrhagica*. The description which has been given for the swine-plague bacteria in the bulletin on this disease will apply to the bacteria now under consideration.

Morphologically there is no difference, excepting, perhaps, the almost constant presence of a capsule around the individual bacteria. This capsule consists of a transparent envelope around the bacillus, having the outline of the latter. I have been unable to stain it with the ordinary anilin dyes, and hence it is not brought out in stained preparations. It is not, in fact, as distinctive a part of the organism as is the capsule of the diplococcus of human pneumonia, for example. The capsule of the latter appears in dried and stained coverglass preparations of the blood and organs of inoculated rabbits; for example, as a characteristic, unstained zone around the bacillus. I have never seen the capsule of the bacilli under consideration under the same conditions. They are, however, readily demonstrated in young cultures soon after they have been derived from the animal body. In the hanging drop, on its border, the bacteria do not touch one another, but are separated by a space of uniform width. Not infrequently the outline of some of the capsules themselves is distinctly seen. In such cases each capsule was observed to inclose a single organism, or two when in pairs. The capsule of individuals was about 1.2 μ long, of pairs about 1.6 μ. The production of this capsular substance is likewise demonstrated in unstained material from agar cultures in which the bacteria are still massed together, but in such a way as to be separated from one another by a certain uniform space.[1]

Nearly all cultures of the pneumonia bacteria become quite viscid with age. This likewise indicates the presence of capsules. Prolonged cultivation on alkaline substrata seems to increase the solubility of the capsular substance and make it less distinct.

The characters revealed by the cultivation of pneumonia bacteria on various substrata are more or less negative. Their growth on gelatin is feeble or else it remains entirely invisible. On agar the growth, both in tubes and on plates, in the form of colonies, is not characteristic. The colonies on the surface expand into grayish, partly translucent roundish disks of variable size, depending on the amount of moisture present. They may attain a diameter of 4 or 5 mm. In tubes, on the inclined agar surface, the growth consists of a grayish, glistening, rather fleshy layer. The deposit in the condensation water becomes dense and quite viscid after a time.

On potato no development is manifest to the eye. Milk remains unchanged even after weeks. Their resistance to destructive agents is feeble. They are to all appearances not adapted to live outside of the animal body, for they are speedily destroyed by drying, and even

[1] The existence of capsules around bacteria is a matter of more frequent occurrence than appears from bacteriological literature. There is evidently a great variation in the consistency of the capsular substance, in virtue of which it is readily dissolved in some species and quite persistent in others. As a diagnostic character it should not be lost sight of, for its presence seems to be linked to certain groups of bacteria.

in cultures their life is quite short. A dried-out culture contains no longer living bacteria, and even in those not dry the majority of the bacteria are no longer alive after a few days.

Biochemical characters.—Like the swine-plague bacteria, they produce phenol. A copious precipitate of tribrom phenol may be obtained by adding bromine water to the distillate from peptone-bouillon cultures several weeks old. The indol reaction is variable, usually not obtainable. Toward sugar, this group of bacteria, including the swine-plague bacteria, have a peculiar action which should always be investigated when any doubt exists as to the nature of the culture under observation. In bouillon this group acts upon dextrose and saccharose, with the appearance of acids in the fluid, but it has no effect upon lactose. With the hog-cholera group only dextrose is attacked, while both saccharose and lactose remain untouched.

Pathogenic action.—This is precisely like that of swine-plague bacteria. The virulence of the many cultures obtained did not, however, rise up to the level of the virulence manifested by some races of swine-plague bacteria. Taking the rabbit as the animal most susceptible to this group of bacteria, a small quantity of bouillon (0.1 to 0.2 c. c.) proves fatal in from twenty-four hours to seven or eight days. The rapidly fatal disease is characterized by no marked changes in the animal and the blood is full of the inoculated bacteria. As the diminished virulence lengthens the period of disease, exudates appear locally in the peritoneal, the pleural, and the pericardial cavity in a certain sequence fully discussed in another publication.[1]

The lungs are sometimes attacked from the pleura secondarily. In these cases the bacteria are few in the blood and very numerous in the exudates, which consist at first mainly of fibrin, later of leucocytes.

The action of the pneumonia bacteria on calves has been shown in the preceding pages to be not infrequently followed by speedy death when the culture fluid is injected into the thoracic cavity. Injections intratracheal and subcutaneous did not produce any recognizable effect.

The great similarity between these bacteria and those associated with pneumonia in swine (swine plague) led to the following experiment:

June 5, 1889, the growth on four agar cultures, about 4 days old, was removed and shaken up in distilled water. The milky, turbid liquid thus produced was used to inoculate three pigs. One and one-half cubic centimeters was injected through the walls of the thorax into the lungs of one pig, the same quantity into the abdominal cavity of a second, and beneath the skin of the third pig. There was no decided effect from any of these inoculations. November 16, nearly five and one-half months later, the pig which had been inoculated in the lungs died from the effects of another experiment. Both lungs were everywhere adherent to the walls of the thorax, the diaphragm, and the pericardium by means of very fine fibrous bands. The pericardium was firmly adherent to the heart. On the lateral border of the right principal lobe a tumor as large as a hen's egg was inseparably adherent to the lung tissue. The wall was one-eighth of an inch thick, inclosing a dry, cheesy mass of pus.

Owing to the great prevalence of this pathogenic species of bacteria and its importance as a disease producer in domestic animals, a synopsis of the most prominent characters are reproduced, with some additions, from the author's report on swine plague:

(1) Bacteria about 1 μ long and 0.5 to 0.6 μ wide. Size somewhat variable in different culture media.

[1] The variability of infectious diseases as illustrated by hog cholera and swine plague, by Theobald Smith and Veranus A. Moore. Bulletin No. 6 (1894) of the Bureau of Animal Industry.

(2) They show the so-called polar stain in coverglass preparations from tissue, rarely in preparations from cultures.
(3) Nonmotile. (This nonmotility is an important character frequently misinterpreted as motility, because in the hanging drop a peculiar jerky or dancing motion is noticeable.)
(4) Growth in bouillon feeble. Deposit nearly always viscid in older cultures.
(5) Growth in gelatin very feeble or absent.
(6) Growth on potato fails.
(7) Does not produce gas in presence of carbohydrates. Produces acids in presence of dextrose and saccharose, but not in presence of lactose.
(8) Produces phenol; presence of indol variable.
(9) Very vulnerable. Rapidly destroyed in water, soil, and when dried.
(10) Multiplies diffusely in blood (virulent) or on serous membranes of rabbits (attenuated), with production of exudates.

EPIZOOTICS OF INFECTIOUS PNEUMONIA.

In the foregoing pages only sporadic pneumonia has been considered. Outbreaks of pneumonia which spread to other animals in a herd, or from one herd to another, have not been studied by the writer. Such outbreaks are, however, very rare, and deserve a brief notice here, as they also are likely to be mistaken for contagious pleuro-pneumonia by inexperienced persons. In dealing with sporadic pneumonia, it was maintained that the associated bacteria were secondary causes, and that the primary causes were injuries of one kind and another. When pneumonia spreads from animal to animal, bacteria are undoubtedly the primary cause, and in accordance with accumulated knowledge we must regard such bacteria as endowed with a relatively high degree of virulence.

In 1886 J. Poels described an infectious disease among calves which he denominates septic pleuro-pneumonia. The disease was fatal in fifteen to twenty hours. In a few cases recovery ensued. At the autopsy the lungs were diseased and in appearance resembled those of pleuro-pneumonia. The costal pleura was ecchymosed and covered with fibrinous exudate. The pleural cavity contained a considerable quantity of straw-colored coagulable lymph, in one case clouded, purulent. The lungs were involved in lobar pneumonia. In three cases the anterior lobes (ventral and cephalic), in a fourth the posterior (principal) lobe, were hepatized. Abundant lymph flowed from the surface, and fibrinous coagula were found in the smaller bronchioles. The author adds that there was nothing to differentiate these cases from contagious pleuro-pneumonia. There was also present a fibrinous deposit on the pericardium and serous exudate within it. In three cases killed when the disease was at its height, the thoracic organs were scarcely at all affected. However, the spleen was enlarged, the liver friable, the peritoneum clouded. In those which died the blood vessels of the skin were distended with fluid blood. In those which were killed the blood coagulated normally.

In the lungs, the pleural and pericardial exudate, bronchial glands, liver, spleen, kidneys, and blood a large number of very small, ovoid, rod-like bacteria were detected, which stained easily. There were always a few distinctly rod-like bacteria present, which the author states are similar to those of mouse septicæmia. They were cultivated without difficulty in the thermostat on agar and in bouillon.

Rabbits and guinea pigs died fifteen to sixty hours after inoculation of culture liquid into the lungs, with exudate pleuritis, pericarditis, and more rarely pneumonia. Subcutaneous inoculation killed a rabbit in thirty hours. A calf 13 days old died twenty hours after

intrathoracic inoculation, with pneumonia and exudate pleuritis. A calf 11 months old died sixty-six hours after intrathoracic inoculation of culture liquid from extensive pleuro-pneumonia. Similarly a calf 7 weeks old died in fifty-four hours after intratracheal injection of the culture liquid and with the same pathological changes.

It is highly probable that Poels had before him the bacteria I have described above. The lesions which he describes could not have been produced by the mouse septicæmia bacilli, as they do not produce serous exudates at any time. The lesions described correspond closely to those produced by the pneumonia bacteria or the swine-plague group.

C. O. Jensen[1] describes an epizootic among calves in Jutland which is of interest in this connection. In a herd of 200 head of cattle about 16 calves died twelve to twenty-four hours after the first symptoms of disease. These were chiefly high fever and diarrhea. At the autopsies a few calves were found affected with a recent, severe fibrinous pleuritis and pericarditis and a gastroenteritis. In one case a rather firm, œdematous swelling appeared on the under surface of the neck. In the spleen of one of the calves minute ovoid bacteria were found, which stained only at the poles. They killed rabbits and mice, after subcutaneous inoculation, in twelve to thirty-six hours.

In another herd the same rapid mortality among calves appeared. The lesions were more of a septicæmic character and associated with the same bacteria in the organs as described above.

In a third outbreak the lesions in the calves were, in addition to enlargement of the spleen and changes of the blood, gelatinous infiltration around pharynx and larynx, and more rarely a phlegmonous inflammation of the posterior regions of the mouth. The same bacteria were found in this outbreak. These bacteria Jensen identifies as belonging to the swine-plague group, both in morphological and pathological properties.

The author made additional inoculations into animals, which are of interest. In a dog, a colt, and two pigs 3 to 4 months old subcutaneous injection of cultures of these bacteria caused more or less local inflammation. In the dog and colt it disappeared. In the pigs it terminated in abscesses. A guinea pig died eight days after subcutaneous inoculation, with extensive phlegmonous inflammation. Two pigeons resisted subcutaneous inoculation. Of seven fowls inoculated, only one died in eleven days. Rabbits were most susceptible, for they died in from ten to twenty-four hours after inoculation; gray and white mice in twenty-four to forty-eight hours.

The relation of these bacteria to the disease was tested on two calves. One 4 to 5 weeks old was inoculated subcutaneously with a few drops of rabbit's blood containing the bacteria diluted in water. The calf became very sick within fourteen hours. It showed great prostration with high temperature and feeble pulse. A considerable swelling appeared at the place of inoculation. It was found dead thirty-six hours after inoculation. The chief lesions were those produced locally in the neck by the inoculation, fibrinous pleuritis, and hyperæmia of the intestines with occasional hemorrhages.

A second calf inoculated in the same manner from an agar culture showed the same symptoms, but finally recovered. An extensive inflammatory swelling on the neck, due to the inoculation, turned

[1] Ueber eine der Rinderseuche ähnliche Kälberkrankheit. Monatshefte für praktische Thierheilkunde (1890), II, p. 1.

into an abscess. This same calf, previously fed with the blood of the preceding calf, and a pig fed with the viscera of several inoculated rabbits did not show any disease subsequently.

These observations of Jensen's are very important in demonstrating the existence of races of the swine-plague group which may produce epizootics of a serious and fatal character. The abstract given leaves no doubt as to the nature of the bacteria found. It is of interest to note in some of the affected calves inflammations of the back of the mouth and around the pharynx and larynx, as if the bacteria entered the mouth first and thence penetrated into the connective tissue. It will be remembered that attenuated varieties of this bacterium are present in the mouths of cattle.

Perroncito[1] has described an infectious pneumonia among calves and young pigs. The pneumonia is of the more chronic catarrhal type. The youngest are most susceptible, and with them the disease is most fatal. At the autopsy the lesions are limited chiefly to the small anterior (ventral and cephalic) lobes and represent a true broncho-pneumonia. Subsequently Perroncito described as the cause of this disease a micrococcus (*M. embratus*) which stains gelatin and agar cultures amber yellow.

Imminger[2] gives a brief account of an infectious pneumonia among calves which runs a more chronic course, with fever, great emaciation, cough, and fetid discharge from the nostrils. The disease was introduced by calves brought from another place by dealers and sold singly or in pairs.

Septic pleuro-pneumonia in calves has also been observed by E. Lienaux[3] in Belgium, who describes the lesions found in one case and the bacteria associated therewith. In this case the anterior lobes of the lungs were hepatized and of a dark red, brown, or violet hue. The interlobular tissue was also affected. "Thick connective-tissue bands separated the different diseased lobules. The bands were œdematous, of a white or yellowish color, and from them flowed a clear serum. Distended lymphatics were noticed in them, full of lymph and fibrinous plugs." The further description of the lung lesions gives rise to the suspicion that it may have been a case of contagious pleuro-pneumonia grafted upon a slight broncho-pneumonia.

The bacteria isolated from these lungs and there present in large numbers are evidently identical with the pneumonia bacteria. They are described as motile; yet this mistake or misinterpretation has been made so often that this apparent difference may be safely disregarded. The inoculation experiments correspond closely to those of Nocard's and ours. Calves succumbed to intrathoracic inoculation in twenty to sixty-six hours and "on examination presented the lesions of septic pleuro-pneumonia."

Preusse[4] reported an outbreak of some infectious disease among calves which he regarded as possibly transmitted from swine which had been dying from swine erysipelas or *Rothlauf*. The evidence that the swine disease was swine erysipelas and not *Schweineseuche* (or swine plague) is not presented, and judging from the brevity with

[1] Il Medico veterinario, 1884. From a review in the Journal de Médecine vétérinaire, April, 1885, p. 210.

[2] Ueber eine infektiöse Kälberpneumonie. Wochenschrift f. Thierheilkunde u. Viehzucht, 1891, S. 213–217.

[3] The Veterinary Journal, XXXV (1892), p. 415.

[4] Rothlaufartige Infectionskrankheit bei jungen Rindern. Zeitschrift f. wiss. u. prakt. Thierheilkunde, XIII (1887), S. 450.

which the bacteriological facts are disposed of with regard to the cattle disease we must, to say the least, refuse to accept the positive statements of the author.

The disease attacked chiefly young animals 6 to 12 months old and was very rapid in its course. Four days after its appearance it had again disappeared. Nine animals became diseased and only one recovered. The disease began with dullness and loss of appetite. The calves lay down much of the time and soon stopped ruminating. There was considerable fever (104° to 106° F.), rapid, feeble pulse, constipation, and in some cases tympany. The disease usually terminated fatally twenty-four hours after the appearance of the first symptoms. In a certain number of the affected calves an œdematous swelling appeared around the pharynx and larynx, which was associated with foaming at the mouth and difficult breathing, finally terminating in death by suffocation. The autopsy gave evidences of an acute infectious disease, and in the blood and local swelling the author found bacilli resembling those of rouget. In one case there was found a severe peritonitis.

The evidence in this paper is incomplete, and the facts could apply to the pneumonia bacteria as well as to rouget bacteria. The latter have not thus far been accused of attacking the bovine species, excepting by Poels, quoted above, whose statements are equally ambiguous. Other outbreaks of disease probably due to this same group of bacteria, which have been briefly abstracted in the bulletin on swine plague, page 141, are those of wild seuche and a buffalo plague.

Some years ago F. S. Billings described a disease among cattle which he denominated cornstalk disease, and in which lung disease was supposed to play a prominent part. As to what the cornstalk disease really is has been investigated by Dr. V. A. Moore for the Bureau of Animal Industry (Bulletin No. 10, 1896), and I therefore shall only refer to it here in so far as the descriptions of Billings have made pneumonia one of the prominent features.

This terminology had even penetrated into Europe and had been accepted in good faith by those who were unable to investigate the subject themselves, so that when a few head of cattle imported from America into France were found affected with pneumonia the disease was promptly described as the cornstalk disease, i. e., it was stamped as something peculiar to this country, when, as the preceding pages clearly show, the same agent causing lung disease in cattle had been described not only as existing in various parts of the Continent, but as producing general outbreaks, especially among the younger animals. The circumstances connected with the so-called American cornstalk disease in France are given by E. Nocard in brief as follows:[1]

> On November 15 and 16, 1890, 4 head of cattle imported among 400 head from Indiana and Illinois into France were found affected with lung disease. One died and three were slaughtered. At first there was suspicion of contagious pleuro-pneumonia, but this was dispelled by a careful examination. The disease during life was marked by a very high temperature (108° F.), depression, weak and abortive cough. A careful examination of the lungs of the slaughtered cases yielded the following description: At first sight, a section of the hepatized tissue presented the appearance of a recent pleuro-pneumonic lesion. The tissue is dense, firm, compact, friable, varying in color from a light red to a deep brown, or nearly black. The lobules are separated from one another by broad connective tissue bands, infiltrated with a considerable quantity of yellow limpid serum. An attentive examination sufficed to reveal notable differences between these lesions and those of pleuro-pneumonia.

[1] Recueil de Méd. Vét. (7 ser.), IX, 1891, pp. 424–429.

The infiltration of the connective tissue is less abundant, the serum less albuminous, less limpid. Here and there pressure forced from the overdistended lymphatics small white fibrinous concretions, smooth, of firm consistency. The lobule imprisoned in this inclosure of connective tissue, so unusually thickened, has not the uniformity of tint or of consistency which characterizes the pleuro-pneumonia lesion. It is firm and more manifestly hepatized in the center than in the periphery. The lesion proceeds from the bronchus and not from the perilobular tissue. In pleuro-pneumonia it is the opposite. There one finds, where the lesion is very recent, lobules in which the tissue is very dense and blackish at the periphery, while at the center it is still rosy and permeable.

Another important differential sign: Pressure causes a notable quantity of thick, viscid, yellowish-white muco-pus, quite like that observed in certain forms of verminous bronchitis, to exude from the air tubes. A preliminary microscopic examination permits us to eliminate this hypothesis. The muco-pus of the bronchi, the scrapings of the hepatized tissue, contains neither ova, nor embryo, nor worms of any kind.

An incision into the bronchi shows that the mucosa is inflamed, thickened, the epithelium more or less desquamated. The submucous tissue is always infiltrated with serum, acquiring sometimes considerable thickness. On the other hand, this muco-pus of the bronchi contains in abundance a short, ovoid, motile bacterium, which seems to be present to the exclusion of all other microbes. This bacterium is also found in veritable pure culture in the hepatized tissue, and above all in the limpid serosity which distends the perilobular lymph sacs.

This single character suffices to demonstrate that the lesion in question is not pleuro-pneumonic in nature. It is known that the pleuro-pneumonia serum is extremely poor in bacteria, and that if one collects it with precaution against contamination from the infiltrated connective tissue septa it ordinarily is sterile.

I have quoted from this communication of Nocard's, since it aroused considerable unnecessary attention at the time because of its supposed relation to a so-called specific cornstalk disease in this country, and because of its resemblance to contagious pleuro-pneumonia.

The cursory examination by Nocard showed it to be an aggravated form of broncho-pneumonia associated with the bacteria I have described in these pages. Its existence in four animals may be accounted for perhaps by an increased virulence of the bacteria associated with a similar exposure of these animals on the journey. That the virulence was quite high is shown by Nocard's report of his inoculation experiments. Mice, rabbits, guinea pigs, and even pigeons succumbed in less than forty-eight hours to small subcutaneous doses of the serum. A calf 8 months old and a ram of 2 years, which received an intrathoracic injection of a little peritoneal exudate from a guinea pig, died within forty-eight hours with fibrinous pleuritis and exudative broncho-pneumonia. It will be noted that Nocard describes these bacteria as motile. This made it at first impossible to identify the bacteria found by him and the pneumonia bacteria. This difficulty was overcome by a study of one of Nocard's cultures, which demonstrated the identity of the organism as found in this country with that found by Nocard in France. The following notes show that the foreign culture had retained much of its original virulence:

Through the kindness of Professor Nocard cultures of the bacterium found by him were sent to the Department and handed to me by Dr. Salmon for examination on January 22, 1892. The bacteria were contained in pipettes sealed in the flame. Two contained bouillon, the third the blood of a guinea pig which had succumbed to inoculation into the abdomen in eighteen hours. Of these one bouillon culture and the blood were examined.

From the bouillon culture, agar and bouillon tubes were inoculated directly. The bouillon became faintly and uniformly clouded, and contained very minute oval bacteria which were nonmotile, but which showed very active Brownian (dancing) movements. In the hanging drop on its edge the accumulated bacteria show a distinctly polar arrangement of the protoplasm. In the agar tube a considerable number of isolated colonies appeared. They were round, slightly convex, and of a grayish, partly translucent, appearance. The condensation water

was quite turbid and became later on viscid. The bacteria in it appeared like very minute cocci.

A rabbit inoculated subcutaneously with a loop of the condensation water died in twenty-five hours. The various organs contained a large number of oval bacteria, many of which showed the polar stain characteristic of this group of bacteria.

On January 26 the pipette containing the blood was opened and three agar plates prepared from a loop of blood, a microscopic examination of which had revealed a large number of minute oval bacteria. The colonies which developed on the plates were all identical in appearance, the bacteria composing them evidently identical with those in the bouillon culture. From a colony an agar tube was inoculated January 27, and on the following day a rabbit inoculated subcutaneously on abdomen with a loop of the resulting growth: Temperature 105.7° F. on January 29, 4 p. m.; 104.2° F. on January 30, 10 a. m. The rabbit was found dead January 31. The local lesion and the exudative peritonitis were the same as those of the preceding rabbit. Similarly there were immense numbers of bacteria in this exudate. Cultures therefrom contained the injected bacteria only. A final test of the virulence on rabbits was made February 18. On this day a bouillon culture twenty-four hours old, inoculated from the same agar culture from which the preceding rabbit had been inoculated, was injected in doses of 0.1 cc. under the skin of two rabbits. Both succumbed in thirty-six and forty-eight hours, respectively. The former had, in addition to a slight local suppuration, an engorged spleen, dark liver, and œdematous and emphysematous lungs. Many polar-stained bacteria in blood and spleen. The lesions in the other rabbit differed in that peritonitis was already well marked and the slight exudate made up largely of bacteria. These were also present in the spleen.

SOME POINTS OF DIFFERENCE BETWEEN SPORADIC BRONCHO-PNEUMONIA AND CONTAGIOUS PLEURO-PNEUMONIA.

Though contagious pleuro-pneumonia has been the object of investigation for many years, it is surprising that so little information of permanent value concerning it has been placed on record. This largely accounts for the differences of opinion entertained by those who have had most experience with this disease when it comes to make a decision as to the existence or nonexistence of this disease in special cases. The most unfortunate thing, to be sure, is our ignorance of the nature of the cause, but it would seem that the gross and microscopic characters of the disease, all taken together, furnish enough evidence upon which to base a fairly reliable decision.

One of the means of enriching our knowledge on this subject would be a thorough study of bovine pneumonia in all its various phases as it occurs in our own country. Its freedom from the contagious lung disease would put the facts thus accumulated on a permanent basis. In other countries the prevalence of contagious pleuro-pneumonia makes a sharp distinction between it and sporadic pneumonia a difficult thing, to be undertaken only by a skilled pathologist. The coexistence of contagious pleuro-pneumonia and broncho-pneumonia may be expected, and the lesions produced by each would in such cases be indistinguishable.

It is not my object here to fill up any gap in the diagnosis of pleuro-pneumonia, but simply to point out for practical use some of the differences between it and sporadic, i. e., broncho-pneumonia, in the main, since an accurate diagnosis of any case of lung disease will depend upon the number of facts which can be brought together on the one side or the other.

The regions of the lungs affected serve as an important differential character. It has already been stated that broncho-pneumonia begins in the small ventral lobes and spreads thence by the aspiration of bronchial secretions to the cephalic and the caudal lobes. More rarely, at the outset, the right cephalic lobe is affected, together with the ventrals.

Again, the disease usually affects both lungs simultaneously in the more advanced stages. In contagious pleuro-pneumonia the distribution is quite different. In many cases only one lobe is affected, usually only one lung. So far as the writer's observations go the large caudal lobes are most frequently diseased, and the smaller lobes when at all diseased are usually affected by contiguity or through the agency of extensive pleuritic exudates. Rarely does it appear that the disease began in them. This condition suggests that the inspired air is the main vehicle of the contagium and not any fluid secretions escaping into the trachea. In a former publication[1] the writer pointed out that the tubercle bacilli lodge in the majority of cases in the large caudal lobes of cattle, since in these most primary foci are found. There is thus a close similarity between the distribution of the lesions in tuberculosis and in contagious pleuro-pneumonia. Both have a predilection for the main (caudal) lobes; both are unsymmetrical.

The difference between the hepatized portions in broncho-pneumonia and pleuro-pneumonia are numerous and have been dwelt upon by most writers. They pertain to the respiring portion or parenchyma as well as to the interlobular and perivascular connective tissue. The affected lobes are very large and heavy in pleuro-pneumonia; in broncho-pneumonia usually smaller than the normal collapsed lung. The cut surface in acute pleuro-pneumonia presents a bewildering variety of appearances, while that in broncho-pneumonia has a more or less uniform appearance. In the latter the solidified parenchyma is usually of a dark or light red color, mottled quite uniformly with pale grayish or yellowish dots which represent the cellular infiltration within the minute air tubes and around them in the connective tissue and the adjoining zone of alveoli. Only in certain cases upon which I have already dwelt at length are interlobular changes present which might lead to confusion. Contagious pleuro-pneumonia seems to affect primarily the connective tissue framework of the lungs, secondarily the parenchyma itself. There is present in all cases an increase or thickening of the connective tissue surrounding the air tubes and the blood vessels which extends a variable distance into the connective tissue between the lobules. This thickening gives rise to bands of a firm texture and pearly whiteness. In severe cases the large vessels, especially near their root, are sheathed with a zone of similar texture and consistency. Not infrequently the dorsal mediastinum becomes involved and the fatty tissue and the lymph glands become firm owing to abundant infiltration of the tissue with leucocytes as well as to exudation of fibrin. These lesions, which seem to be characteristic of contagious pleuro-pneumonia, must be distinguished from certain interlobular changes which probably occur later and are very much like those I have described as occurring in some cases of broncho-pneumonia. We may find cavities in the interlobular tissue wholly or partly filled with fibrinous or gelatinous translucent plugs, precisely as in broncho-pneumonia and probably due to the same causes—emphysema and stagnation of the lymph current and occasional infection with the pneumonia bacteria from the mouth and throat.

Another feature of the changes going on in the coats of the vessels, first pointed out by Klebs in 1841, is the plugging of the veins, due to an inflammation of the inner coat. The disease process extends from around the vein through the walls of the latter to the inner coat. A

[1] Investigations Concerning Bovine Tuberculosis (Bulletin of the Bureau of Animal Industry, No. 7), p. 90.

deposit forms on the diseased spots within the vein which partially or wholly occludes the vessel. It is a curious fact, first noticed by Klebs, that the thrombi occur only in the veins, although the connective tissue around the arteries is equally involved. The greater thickness of the arterial coat may perhaps have something to do with this fact. I have in vain sought for thrombi in the arteries accessible to ordinary dissection. These may, and usually are, filled with dark clots after death in acute cases, but they are easily distinguished from the firmly attached thrombi.

It is this thrombosis or plugging of the pulmonary veins which accounts for many of the changes which the parenchyma itself undergoes. We observe, for example, on making sections through different regions of an affected lobe at least three varieties of lesions: (1) the very firm, granular, dark-red, and yellowish hepatization; (2) the blackish hepatization or infarction, and (3) an œdema or filling up of the lung tissue with serum. The infarction is probably due wholly to the plugging of veins. In all cases examined I have found a definite relation between the size of the infarct or blackish tissue and the size of the occluded vein leading to it. The granular hepatization and the œdema are perhaps traceable in part to the interference with the venous circulation, in part to the plugging and effacement of the interlobular and perivascular lymphatics, and consequent rigidity of the framework of the lungs, in part to the specific activity of the unknown cause. The specific action of this agent upon the connective tissue, the occasional entire absence of hepatization, leads me to assume that the changes of the lung tissue proper, the hepatization, œdema, and hemorrhagic infarction are all secondary changes due to the disease of the veins and lymphatics which cause partial or complete occlusion of these channels. If this be borne in mind the variegated cut surface of pleuro-pneumonia lungs will be much better understood, and the differences between contagious pleuro-pneumonia and sporadic pneumonia, or even infectious pneumonia, due to other causes more easily ascertained, especially as thrombosis of pulmonary veins, has not been found, except as an accidental complication, in other lung diseases.

Another quite characteristic feature of contagious pleuro-pneumonia is the appearance of dead masses of lung tissue, of variable size, which finally separate and many become surrounded with a connective tissue capsule. These sequestra are recognizable as such if traces of the dead lung tissue can be found. If the mass is broken down into an amorphous fatty detritus, the absence of foreign bodies and of other lung affections will usually decide in favor of contagious pleuro-pneumonia as the original cause.

The characters of contagious pleuro-pneumonia as revealed by bacteriological and microscopical methods are in the present stage of our information concerning the causation of this disease of little use, excepting in the hands of the skilled pathologist, whose final decision as to the nature of any bovine lung disease will be made from all available characters, macroscopical as well as microscopical. There are, however, several features in the diseased pleuro-pneumonic lung which are readily detected after attention has been once called to them.

One of the most conspicuous features in a microscopic section from a lung affected with acute pleuro-pneumonia is the presence of intensely stained foci and lines. These lines, to which Prof. W. H. Welch called my attention some years ago, are visible to the naked

eye, and when viewed with a hand lens suggest by their peculiar curves the contour lines on a map. They are situated at the margin of and within the inflamed connective tissue which surrounds the large vessels and separates the lobules from one another. A closer examination of these lines indicates that they coincide with the boundaries of the lobules and of the individual lymph spaces of the interlobular tissue. Under a high power of the microscope they are resolved into dense masses of leucocytes in various stages of degeneration. These dense bands are presumably attracted to the connective tissue boundary of the lobules and to the walls of lymph spaces within the connective tissue by the unknown cause of the inflammation, presumably also the cause of the disease itself. The space between the lines is filled with fibrin, in which very few leucocytes are found.

It is these lines which enable the experienced observer to pick out from a number of slides those representing lungs affected with acute pleuro-pneumonia. The sporadic pneumonia associated with interlobular disease, which has been dealt with in the foregoing pages, also shows certain deeply stained lines occasionally within the interlobular tissue, but these are distinguishable in several ways. The band of leucocytes represents a closed wall within the interlobular tissue around a lymph plug, which has become the nucleus of an abscess. Outside of the leucocytic wall there is usually a zone of young connective tissue cells situated between it and the lobule proper. The leucocytic wall does not appear on the very edge of the lobule, as in pleuro-pneumonia. In broncho-pneumonia there is furthermore an entire absence of cell infiltration around the larger bronchi and blood vessels, whereas in pleuro-pneumonia the cell masses are as dense and as abundant around these as in the spaces between the lobules themselves. In fact, it is the penetration of these cell masses into the inner coats of the veins which accompanies the thrombosis so characteristic of and peculiar to this lung affection. The masses of exudate which appear in the dorsal mediastinal space, and involve the fat deposits and the lymph glands embedded in them, present the same cell masses found in the connective tissue framework of the lungs themselves.

A peculiar phenomenon which may be mentioned here is the rapid and wholesale degeneration of the alveolar epithelium of that portion of the lung tissue which is involved in œdema, probably because it has lost its elasticity by reason of the thickening and rigidity of the interlobular framework. In this tissue, infiltrated with serum, all or nearly all the epithelial cells from the alveoli are packed with roundish bodies, all of the same size in the same cell, but varying among different cells and in different lungs from 1.5 to 4 μ in diameter. They are visible only in fresh tissue as pale bluish gray, homogeneous bodies. Their striking uniformity, coupled with their abundance in the cell protoplasm, at first strongly suggested foreign parasitic organisms, but they were subsequently identified with the so-called Buhl's bodies, or myelin bodies, not uncommon in human sputum, where, however, they vary in size in the same cell and entirely fail to give one the impression of parasitic organisms. These bodies appear as disks, presenting either the face or the edge. The latter appears either as two dark bands with a light band between them, or the reverse, according to the focus. This appearance would indicate that each body is a double disk. Sometimes they suggest, when on edge, an open clam shell. They failed to retain any color when aniline dyes with or without mordants were used. They equally refused to be colored by iodine with and without sulphuric acid. In fresh, teased preparations they

remained for a time intact in a 10 per cent solution of caustic potash. Later on they assumed to some degree the bizarre outlines of the myelin bodies of human sputum. There was apparently a peeling off of the external layers of the bodies, or a partial uncoiling. These bodies have no diagnostic significance, for cells containing them may be found in the lungs of healthy cattle, though in exceedingly small numbers. By treating the fresh, healthy lung tissue with caustic potash to dissolve the cell protoplasm they can be occasionally detected. Most probably they are the result of a certain disturbance of the nutrition of the affected tissues. In broncho-pneumonia they may be detected at times, but this kind of lung disease does not appear to produce them in any abundance.

The hepatization of the parenchyma in broncho-pneumonia is readily distinguished in microscopic sections, even with a hand lens, from that of pleuro-pneumonia by its focal arrangement. Each minute bronchus is plugged with cell masses and around it is a zone of alveoli similarly occupied. In the hepatization of pleuro-pneumonia this focal arrangement is absent, and the alveoli are uniformly filled with fibrin or a variable mixture of fibrin and cells, depending on the stage of the disease.

In making a diagnosis between contagious pleuro-pneumonia and sporadic pneumonia, based on the lung lesions alone, it should always be borne in mind that the entire lungs should be subjected to examination. Attention should be given to signs indicating injury to the chest wall, to the possible presence of foreign bodies, and to severe inflammation of the air passages which may result from the aspiration of irritant or corrosive fluids. Where these agents may be excluded special attention should be paid to the condition of the pleura and to the distribution of the disease, careful notes or sketches being made of the lobes involved. Next in order come the peculiar perivascular and interlobular thickening and the thrombosis of veins.[1] This leads to local necrosis of greater or less extent (infarcts), and finally to encapsuled sequestra if the animal should recover. The different features of the hepatized and œdematous portions of the lung tissue should be carefully noted. Lastly, the peculiar exudation and infiltration in the connective and fatty tissue of the dorsal mediastinum and of the embedded glands should not be overlooked. With the microscope the peculiar dense cell masses of the diseased connective tissue should be looked for and the nature of necrotic tissue determined in case macroscopic appearances are no longer reliable owing to hardening processes.

It should finally be borne in mind that the lesions of broncho-pneumonia and the interlobular changes which may follow it may coexist with contagious pleuro-pneumonia, and that both kinds of lesions may be encountered in the same lungs. Hence, great caution must be exercised in expressing an opinion when only a small portion of the lungs are presented for examination, because only that portion which is affected with broncho-pneumonia may have been submitted.

As regards the value of bacteriological examinations, it is safe to concede that if the pneumonia bacteria are generally disseminated throughout the diseased lung tissue, and more particularly in the interlobular tissue, the disease is not contagious pleuro-pneumonia, because in the latter the interlobular tissue becomes affected first, and

[1] Care should be taken not to confound mere clots with adhesive thrombi. In acute pleuro-pneumonia, after death, the arteries are usually distended with clots.

is therefore closed to the entry of bacteria, while in broncho-pneumonia they are aspirated into the still normal interlobular tissue when new portions of the lung tissue become involved by reason of some great strain, producing interlobular emphysema. In general, the bacteriological outcome must be taken as one of the important factors in a diagnosis until the true cause of contagious pleuro-pneumonia shall have been definitely recognized.[1]

GENERAL CONCLUSIONS.

(1) Sporadic pneumonia of cattle usually appears as a broncho-pneumonia, to which may be superadded emphysema and interlobular disease, the latter simulating contagious pleuro-pneumonia.

(2) Broncho-pneumonia in cattle is a comparatively rare affection. It is probably in many cases caused by entry of fluids into the air tubes during drenching. When secondary to tuberculosis, foreign bodies, etc., these causes are easily recognized at the autopsy.

(3) Of the nature of traumatic pneumonia, sometimes called transit pneumonia, due to blows upon the thorax and injuries received on board of ocean steamers, nothing definite is known.

(4) The bacteria of the swine-plague group found in the upper air passages of healthy cattle are present, usually in pure cultures in broncho-pneumonia, and are, to all appearances, responsible for the interlobular disease and for the persistence and extension of the lung disease.

(5) In lungs affected with contagious pleuro-pneumonia most of the cultures made from the parenchyma and the interlobular fluids and exudates remain sterile. In some cases, however, as might be anticipated, the bacteria of the swine-plague group are present.

(6) The bacteria found in bovine pneumonia are more frequently provided with a capsule than those found in swine. Other differences are not constant.

(7) Outbreaks of septicæmia and infectious pneumonia in calves are probably in most cases due to highly virulent races of the same group of bacteria.

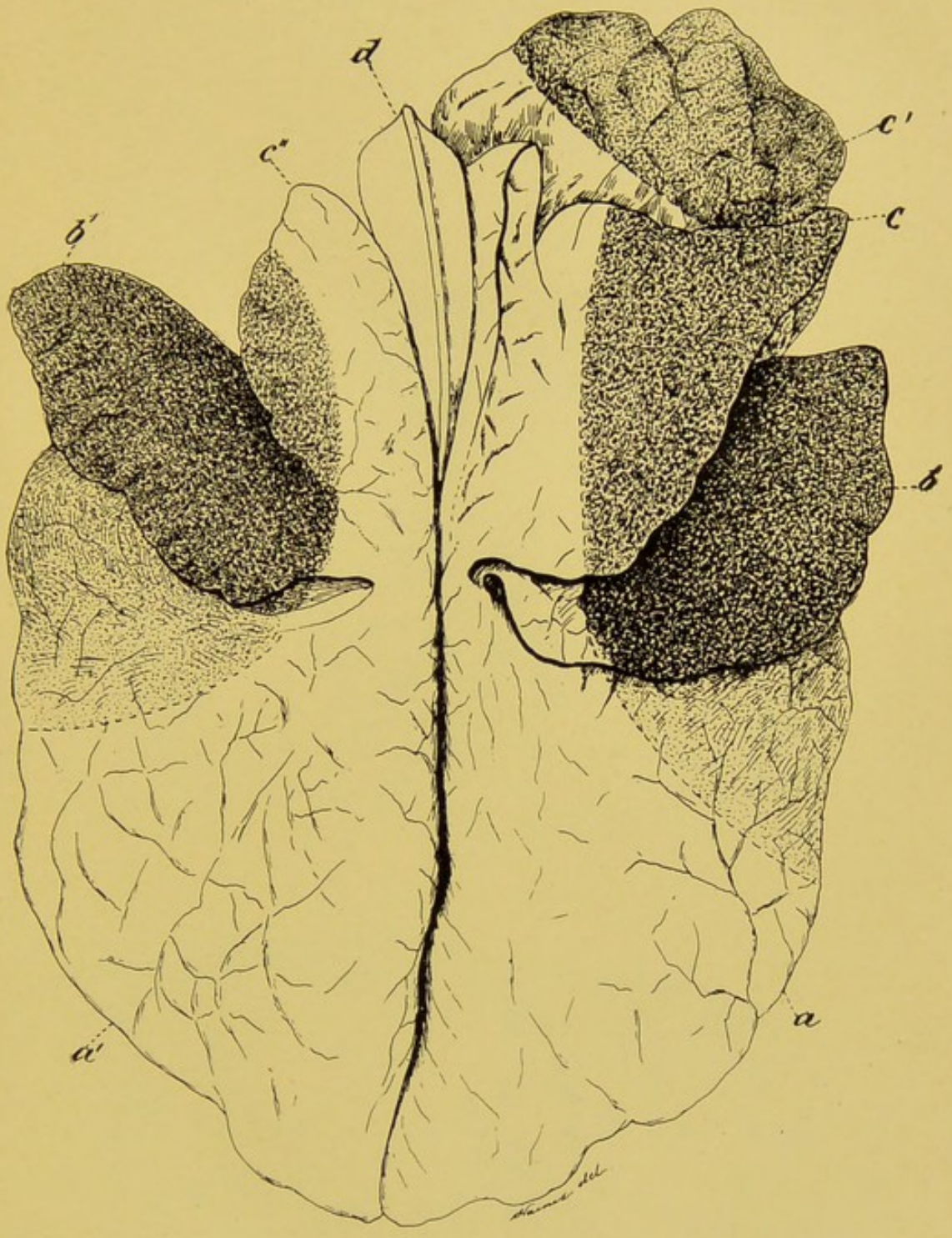
DESCRIPTION OF PLATES.

Plate IV.—Dorsal or upper surface of the lungs of a cow, reduced to one-sixth of the natural size a, a', the main or caudal lobes; b, b', the middle, or ventral, lobes; c, c', c'', the anterior, or cephalic, lobes; d, the trachea. The shaded regions are those affected in broncho-pneumonia. The disease usually begins in the lowest, or ventral, lobes (b, b') first; more rarely in the right cephalic, c, c', which consists of two partly divided lobes and is provided with a special air tube nearest the head. The disease next attacks the cephalic lobes (c, c', c'') as indicated by the shading, and then the shaded portion of the main or caudal lobes (a, a'), and may extend from there over the greater part of these lobes. In contagious pleuro-pneumonia the affection is frequently restricted to one side, and one of the main lobes (a, a') is the most frequent seat of the disease.

Fig. 1: Alveolar epithelium from the œdematous region of the lungs in acute pleuro-pneumonia (see page 146). The cells are partly filled with myelin (Buhl's) bodies, some of which present the disk, others the edge. (× 1000.)

Plate V.—Fig. 2: Alveolar epithelium from another case, illustrating the large number of myelin bodies found in a single cell, and the uniform size of these bodies.

[1] Arloing claims to have discovered the cause of contagious pleuro-pneumonia in a bacillus (*B. liquefaciens bovis*). As this discovery seems to be discredited in his own country by Nocard, we must await confirmatory investigations, especially as the statements appear improbable.



Portions of the Lungs usually affected in Broncho-pneumonia.

Fig. 1.

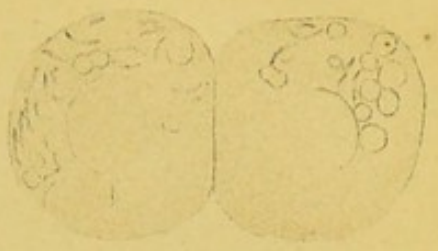


Fig. 2.

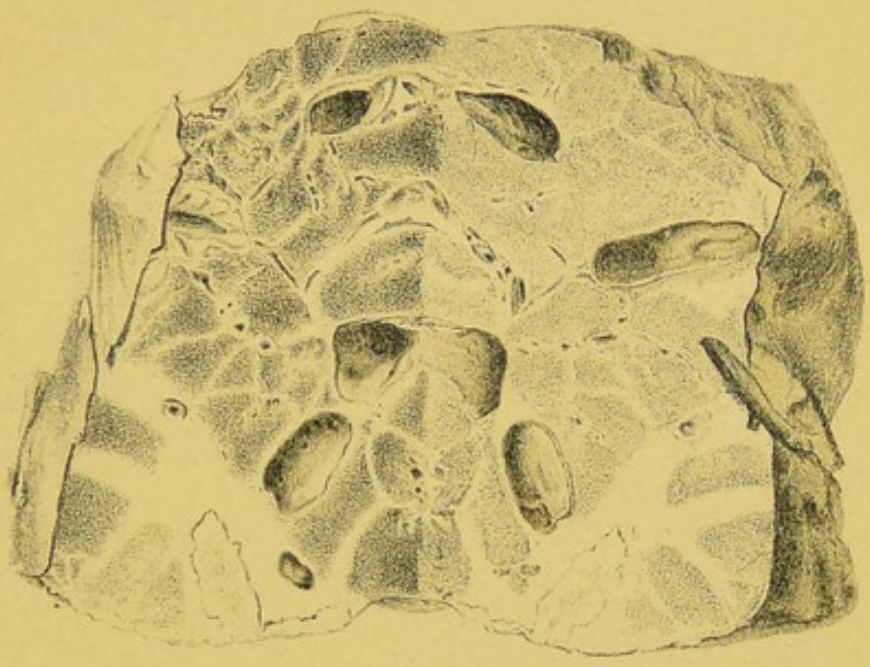


Fig. 3.

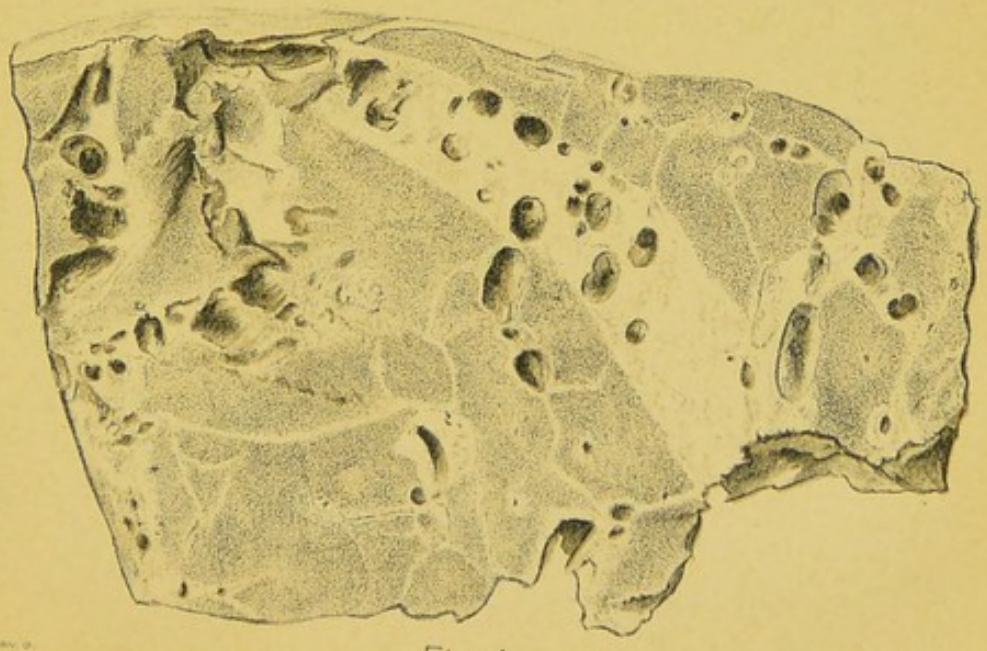


Fig. 4.

Haines, del.

INTERLOBULAR DISEASE MYELIN BODIES.

Fig. 1.

Fig. 2.

Fig. 3.

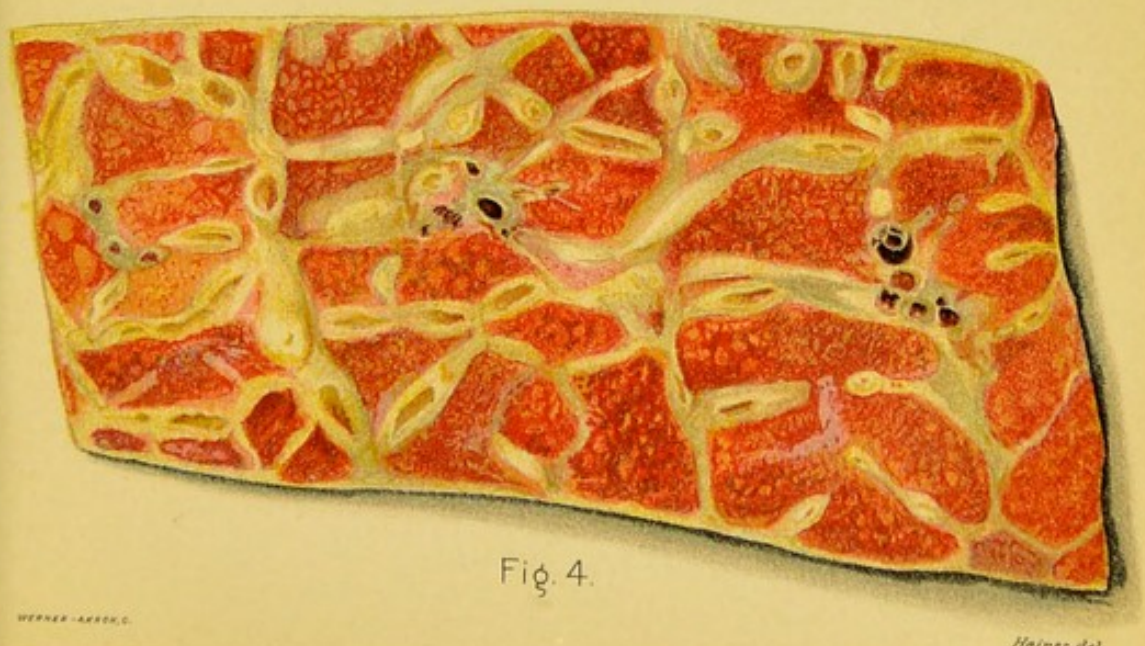


Fig. 4.

INTERLOBULAR DISEASE IN BRONCHO-PNEUMONIA.

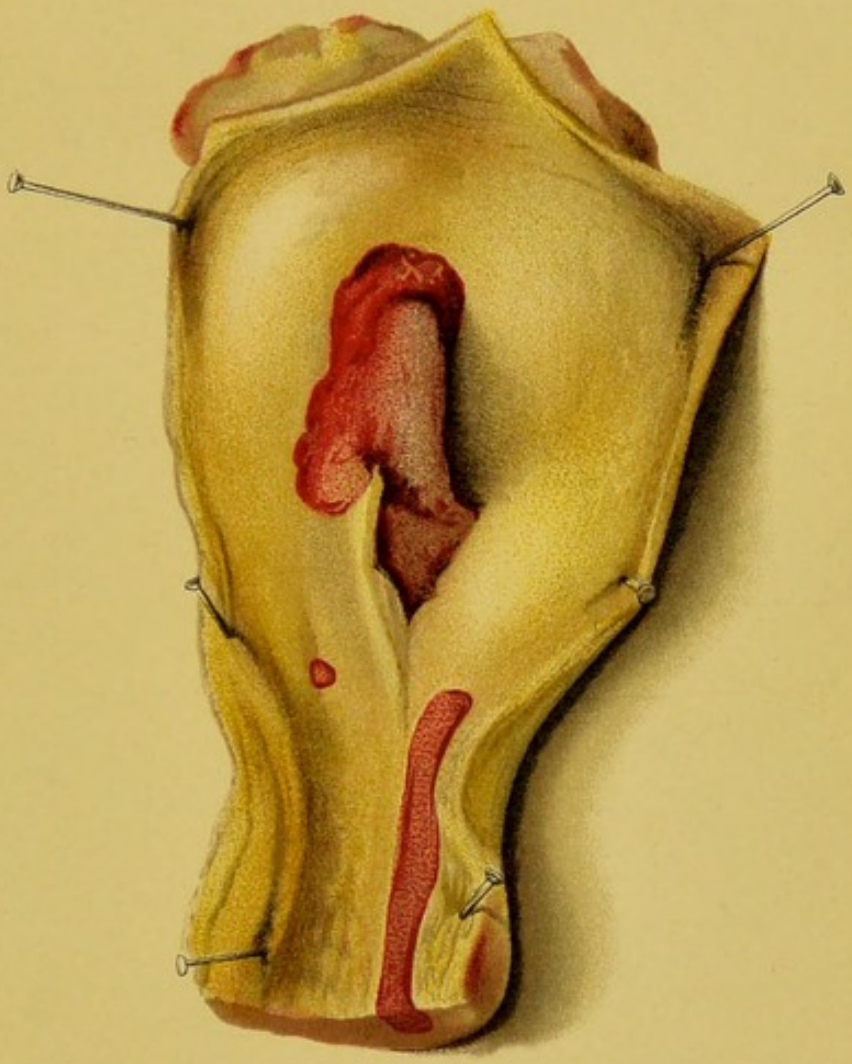


Fig. I.

Haines, del.

VENOUS THROMBOSIES IN CONTAGIOUS PLEURO-PNEUMONIA.

Fig. 3: Drawing of the cut surface of a lung affected with interlobular disease (alcoholic preparation of case 1, page 127). The dense whitish bands, honeycombed with cavities, are well brought out. The lung tissue itself was not hepatized. (Natural size.)

Fig. 4: Drawing of the cut surfaces of a similar case (case 2, page 128). The cavities in the interlobular exudate are quite large. Near the lower margin the outlines of a lymph plug are visible.

Plate VI.—Fig. 1: Interlobular inflammation and exudation with formation of abscesses from a case of broncho-pneumonia (lung No. 30, pages 122 and 132). The cut surface is shown in the figure. The lymph plugs which appeared in the interlobular tissue are now surrounded by a yellowish layer of pus corpuscles. Outside of the yellowish zone, the grayish band adjacent to the lung tissue proper represents newly formed connective tissue. (Natural size.)

Fig. 2: From the same case. The cut surface of the lung tissue shows a very wide, interlobular band, with cavities, and yellow, suppurative zones. Lung tissue proper œdematous, not hepatized.

Fig. 3: The surface of the same lung, showing the filling up of the network of lymph spaces under the pleura with coagulated lymph. The plugs formed are easily removed after the pleural covering has been torn away.

Fig. 4: The cut surface of the main or caudal lobe of the lungs from a case of broncho-pneumonia (lung No. 46, pages 123 and 132). The so-called "marbling" is quite conspicuous. The interlobular exudation causing this marbling shows, along its course, enlargements in which the lymph plugs are in process of suppuration. The absence of all thickening around the cut ends of the vessels should here be noted. In contagious pleuro-pneumonia the thickening would appear there first. Lung tissue proper hepatized. The fine mottling of the parenchyma due to broncho-pneumonic foci is quite distinct in some lobules.

Plate VII.—Thrombosis or plugging of the principal vein of one of the main or caudal lobes. From a case of acute pleuro-pneumonia. The vein is slit open at a point where it sends off a large branch, which is shown laid open below. The inner coat of this branch is discolored, mottled with yellowish spots, indicating the presence of dense masses of leucocytes. The thrombus or plug is shown partly adherent. The great thickness of the wall of this vein is shown on the cut edge below. Above, the large plug is shown completely occluding the main stem of the vein. The entire mass of this lobe was undergoing necrosis.

TWO VARIETIES OF THE TUBERCLE BACILLUS FROM MAMMALS.

The assumption that a pathogenic species, so called, does not contain within itself a greater or smaller number of varieties with which bacteriology tacitly started out about fifteen years ago has been fairly well demolished up to the present time. Varieties have been found among nearly all of those specific forms which have received a considerable amount of attention. Even the tubercle bacillus has been drawn into the circle of specific forms existing as varieties by the work of Maffucci on tuberculosis in fowls. The sharp distinctions between mammalian and avian tuberculosis which was thereupon drawn prevailed for a time, until recently matters have become more complicated. Koch's remarkable monograph stands on the ground of the unity of the tubercle bacilli which he obtained from human beings as well as from a variety of mammalia. Few investigations appear to have been made subsequently with cultures directly obtained from a case of spontaneous tuberculosis or from guinea pigs directly inoculated with tissue from such a case. In the study of tubercle bacilli which had been cultivated for a variable period of time and passed from hand to hand the element of saprophytic modification entered, which has some effect upon the virulence and the cultural and perhaps morphological characters of the tubercle bacillus.

The subject as it has been left by some recent writers seems to point to the existence of tubercle bacilli of varying virulence, which bridge over the original chasm between the avian and the mammalian cultures, and which tend to wipe out the specific difference between these two forms.

W. Kruse[1] gives the history of four cultures—one from Pansini (Naples), isolated from tuberculous sputum after having been passed through a guinea pig; one from Armanni, isolated in the same manner; one from Sanfelice, isolated from tubercles in the lungs of cattle, and one from Straus, of Paris, the origin of which seems to be not positively known. These four cultures failed to produce more than a local disease in guinea pigs. In two out of twelve rabbits miliary tuberculosis of the peritoneum was produced after intraabdominal injection. Fowls were infected quite regularly by intraperitoneal injections. These cultures are therefore regarded by Kruse as belonging to the avian variety.

Pansini[2] isolated tubercle bacilli having the character of the avian variety from a human being and from cattle after passing the tuberculous products through guinea pigs. Pansini further finds that avian bacilli may occasionally produce tuberculosis in guinea pigs, and finally concludes that transition forms do exist.

Fischel[3] is also inclined to look upon the avian and human tubercle bacilli as not sharply separated. His work does not, however, show that he studied cultures isolated by himself.

Johne and Frothingham[4] have reported what appeared to be a diffuse tuberculous infiltration of the mucous and submucous tissue of the intestines in a cow, quite different in appearance and histological characters from those of the ordinary bovine tuberculosis. Unfortunately, cultures did not succeed and subcutaneous inoculation of guinea pigs failed to produce even a local lesion. The authors are inclined to look upon the case as an infection with avian bacilli.

From these citations it becomes evident, I think, that more study of the tubercle bacillus is needed. I do not deny that the time spent in such study may be regarded as wasted by those who, as it has been recently epitomized, think that the fruitful discovery of Koch has exhausted itself in staining tubercle bacilli. However academic much of this work may appear from this standpoint, it is incumbent upon us to keep training and pruning the tree of knowledge without looking to the right or left. I therefore have considered it worth while to compare somewhat carefully two original cultures from two mammals which presented not only a different behavior on blood serum, but also in which the morphology and pathogenic characters showed certain well-defined divergences. The evidence presented is complete, because both cultures were compared from the time they were isolated and not after an indefinite period of growth on culture media. That such a comparative study may be at least fruitful in raising important questions the concluding portion of this paper will amply show.

I.

The source of one of the cultures was an animal of the bear tribe (*Nasua narica*). In this animal, which I saw the day after death (May 12, 1894), the tuberculosis was apparently of intestinal origin. A gland at the root of the mesentery as large as a goose egg was converted into a sac filled with a soft, viscid pus. The omentum was

[1] Ziegler's Beiträge, XII (1893), p. 544.
[2] Deutsche Med. Wochenschr, 1894, p. 694.
[3] Fortschritte der Medizin, X (1892), p. 908.
[4] Deutsche Zeitschrift f. Thiermedizin, XXI (1895), p. 438. The Veterinary Magazine, January, 1896.

extensively studded with small tubercles, but the spleen and kidneys were free from disease. The lungs were permeated uniformly with tubercles, evidently of hematogenic origin. Ulcers were reported as having appeared on the animal before it died. The owner of this animal, who had kept it as a pet, had succumbed to tuberculosis some time before. It is highly probable that this animal had contracted the disease from its master.[1] The culture was obtained as follows:

A particle of lung tissue was removed and kept in a sterile dish in the refrigerator until May 16, when an opportunity was found to inoculate with it two guinea pigs by placing small particles under the skin. The further history of these animals is given to illustrate the relative virulence:

May 16, 1894. Nos. 174 and 175. Inoculated.

July 26. No. 174 (original weight, 380 grams). Chloroformed two and one-third months after inoculation.

Enlargement and caseation of kneefold, axillary, and retroperitoneal glands of inoculated side. Other glands, including cervical, bronchial, and retrogastric glands, enlarged and firm. Spleen very large and containing multiple tuberculous foci. Liver large, shows a small number of necrotic patches and many grayish translucent foci. In lungs many grayish tubercles, some with necrotic center.

August 29. No. 175 dies to-day, after three and one-half months.

In this animal the liver was extensively necrosed; the spleen affection less marked. The lungs were dark red and contained a considerable number of rather large (3 to 4 mm.) tubercles, which are mainly grayish translucent in appearance.

These notes show clearly that the tissue inoculated was much less virulent than is the case with tuberculous matter from cattle. The latter is usually fatal in four to six weeks to guinea pigs weighing 300 to 400 grams, provided tissue be inoculated in which the disease process is still active.

Cultures were made on blood serum from tubercles in the lungs, spleen, and liver of No. 174. A month later none had shown signs of life. On September 17, fifty-three days after the inoculation of the tubes, a lung culture and a spleen culture showed distinct signs of growth in one or more places, and the bacilli were found to agree morphologically and tinctorially with tubercle bacilli.

The cultivation of this organism in two parallel series from spleen and lungs presented no special difficulties. On serum from cattle the growth after the second generation was fairly vigorous. It developed into a thin uniform expansion over the surface of the serum, which assumed the appearance of ground glass. When the bacilli were massed together with a loop the mass had a distinct straw color. It was neither dry nor very brittle. When it reached the condensation water it soon covered this with a uniform membrane resembling tissue paper, and even pushed itself some distance above the fluid along the walls of the culture tube. A period of two weeks was usually sufficient for the full development of a culture. When the fifth generation was transferred to serum from the dog a very rich whitish growth formed within two weeks.

In the earlier generations some tubes of serum would fail to favor the multiplication of this bacillus, while other parallel cultures were successful. In those tubes in which the growth remained restricted

[1] Dr. Frank Baker, superintendent of the National Zoological Park at Washington, D. C., who kindly brought the animal to the laboratory, thought that it could not have been infected from meat or milk. It was received by him September 27, 1893, and it died May 11, 1894. During this time several other animals of the same species were exposed to similar conditions, but this was the only one to die of tuberculosis. He also adds that it had been a special household pet, very familiar with its master.

a discoloration would appear by which the growth became brownish in part. This discoloration is an indication of the death of the bacilli.

Transferred upon distinctly alkaline glycerin agar, a rather feeble multiplication took place after the fourth or fifth generation. Multiplication on the surface of glycerin bouillon, at first very slow, became after a few generations quite vigorous. The membrane formed has a peculiar structure. It is composed of a number of disk-shaped whitish bodies about 1 to 1.5 mm. in diameter, each representing a center of growth. These disks loosely adhere to one another and leave spaces here and there. The entire membrane thus resembles heavy lace in a general way. The rapidity of growth on alkaline glycerin bouillon is shown by the following notes:

December 21, 1894. Flask inoculated by transferring a piece of membrane 4 to 5 mm. across.

January 3, 1895. Membrane now 10 mm. across.

January 16, 1895. Membrane now 35 mm. across.

Latterly this "lace work" on bouillon has attached to it very thin papery membranes or expansions from 1 to 2 cm. in diameter.

To test the pathogenic power of the pure culture, two guinea pigs were inoculated on the right side subcutaneously October 31 from a serum culture 29 days old (second generation of lung series). Each animal received a quantity of the bacilli which could be held with a small loop.

No. 235. Male, weight 570 grams, inoculated October 31.

November 16. A healed scar at seat of inoculation. Subcutaneous infiltration one-fourth inch thick over an area three-fourths of an inch in diameter. Neighboring kneefold glands as large as beans.

December 3. Pig evidently ill. Chloroformed.

Local ulcer with cheesy base and tubercles around it in subcutis. Right axillary kneefold and retroperitoneal glands of same side largely necrotic. Left glands with necrotic foci. Spleen enlarged. All Malpighian bodies prominent. Liver sprinkled with small necrotic spots. Lungs contain many grayish tubercles, some with necrotic center. Right cephalic lobe collapsed.

No. 236. Male, weight 580 grams, inoculated at the same time.

November 26. Found dead. Local cup-shaped ulcer with surrounding tubercles. Right kneefold, axillary and retroperitoneal glands partly necrotic. In the corresponding glands of opposite side minute necrotic foci. Spleen enlarged to twice normal size. Contains many whitish and yellowish specks. Liver sprinkled with necrotic foci involving usually a single lobule. Minute yellow foci in bronchial glands, but lungs not visibly affected. The death of this guinea pig was premature and probably due to some injury.

Two guinea pigs were inoculated subsequently with the seventh generation of the serum culture from the lung series.

March 15, 1895. Guinea pig No. 353, male, weighing 510 grams, receives into subcutis of one side a loop of the growth from a serum culture 15 days old.

March 28. A firm tumor about one-fourth of an inch in diameter at seat of inoculation. No enlargement of kneefold gland yet.

April 12. Local lesion now a small concave ulcer with thickened border and smooth dry base. Nearest kneefold gland the size of a bean.

May 30. Found dead to-day. (Notes by Dr. V. A. Moore.) Locally no ulcer, but subcutaneous infiltration with a series of caseous glands in kneefold and an eruption of tubercles on peritoneum near site of inoculation. Spleen, liver, and lungs affected; spleen very large.

March 15. Guinea pig No. 354, male, weighing 370 grams, is inoculated in precisely the same way as No. 353.

March 28. Local lesion as in 353. Slight enlargement of kneefold gland.

April 12. Local lesion represented by a large, quite superficial, scab with very little subjacent infiltration.

April 15. Found dead. Kneefold and axillary glands of inoculated side enlarged; contain necrotic foci. Small foci in glands of opposite side, in cervical and retroperitoneal glands. Spleen very large; contains necrotic masses. Liver permeated with yellow masses of dead tissue. Stained serum in thorax. Lungs congested. A few gray tubercles in them.

II.

The source of the other culture was a tuberculous bull. The disease was one of long standing. It started in the lungs, and secondary infection of the throat glands and generalized infection had already taken place.

Two guinea pigs were inoculated with small bits of the recently diseased hyperplastic retropharyngeal gland:

October 20. Nos. 232 and 233 inoculated.

November 28. 233 quite ill; chloroformed. On the inoculated side (right) a concave ulcer with caseous base. Nearest glands large, necrotic, firm. Corresponding glands of opposite side contain minute foci. Spleen very large; contains large areas of necrosis in addition to many isolated foci. Fully one-half of liver tissue necrosed. Lungs contain many gray tubercles; a few with necrosed center.

December 1. No. 232 chloroformed. Lesions less advanced. Evidence of a retardation of the disease. Bronchial glands quite large, but lungs free from visible tubercles.

The tuberculous tissue had thus the usual virulence when compared with tissues from a long series of other cattle tested in the same way during the past eight years. From No. 233 cultures were made on beef's serum. Repeated subsequent examination of the cultures showed that growth had not taken place. Finally, on January 19, 1895, one of the serum tubes showed evidence of growth. A considerable number of minute, grayish, cohesive bodies were found on the surface of the condensation water and in the bottom, none larger than a millimeter in diameter. These were crushed on coverslips and stained and found to be composed of tubercle bacilli.

The further cultivation of these organisms upon blood serum was attended with great difficulty. Only the feeblest increase of the transferred bacilli could be noticed on the surface of the serum. More favorable than this was the surface of the condensation water. In a number of tubes delicate patches of growth appeared on this, which responded to the specific stain for tubercle bacilli. These patches soon became brownish on the margin, an indication of disintegration.

After many failures to carry the culture beyond the second generation, which in itself was very feeble, I finally succeeded by returning to the original culture, removing some of the minute specks from the surface of the serum and rubbing them upon the surface of some fresh dog's serum. This original culture was now 4 months old. Within a month after the inoculation of the fresh tubes a very faint ground-glass-like appearance of the surface of the blood serum could be noticed, which was in fact made up of tubercle bacilli. Thereafter the continuance of the culture presented no special difficulties, although the growth remained quite feeble as compared with the *Nasua* culture.

The success in at last continuing the growth of the bovine tubercle bacillus enables me to compare its virulence with that of the *Nasua* (presumably human) bacillus directly on cattle.

The latter had already been tried, but with negative result:

November 19, 1894. A yearling bull (No. 296) was inoculated as follows: A feebly clouded suspension of tubercle bacilli from a blood serum culture of the *Nasua* bacilli 19 days old was made, and this injected into the thoracic cavity by Dr. Schroeder. The place chosen by him was the space between the sixth and seventh ribs on the left side. The temperature, taken twice daily, remained normal until February 20, 1895, when the animal was killed. No trace of tuberculosis found.

The experiment was now repeated by inoculating two heifers under as nearly the same conditions as possible, one with the bovine, the other

with the *Nasua* culture. At the same time four guinea pigs were inoculated, both to determine the relative effect of the two cultures upon them and to have them serve as controls upon the cultures used.

The cultures used had been prepared on April 22 by inoculating dog's serum. A fairly satisfactory growth had appeared by May 2, at which time suspensions were made in sterile bouillon and kept in the cold until May 4, when the inoculations upon cattle were made. The cultures injected were thus made under precisely the same conditions as to medium and age of growth. The only exception to the uniformity of conditions was that a slightly denser suspension of the *Nasua* bacilli was used for the injection, a condition which strengthens rather than weakens the inference deducible from the outcome:

Heifer No. 300, May 4, 1895. About 2½ years old. Common stock. In very good condition. To-day this animal received, with a hypodermic syringe, into the right thoracic cavity, between sixth and seventh ribs, 4 c. c. of a suspension of *Nasua* bacilli in sterile bouillon. The inoculation made by E. C. Schroeder, M. D. V.

No symptoms of disease were noticed in this animal, and it was killed June 27, fifty-four days after the inoculation. At the autopsy no lesions were found, after a careful search.

Heifer No. 284, May 4, 1895. About 2½ years old. Of common stock and in very good condition. This animal was inoculated precisely as the preceding, with the same quantity of a suspension of the bovine bacilli in bouillon. The suspension in this case contained probably fewer bacilli.

For the following notes I am indebted to Dr. Schroeder, as I left Washington early in May:

May 24. Heifer aborted. Fœtus about 7 months old.

May 27. Membranes had to be removed. Heifer has lost appetite and is growing thin and weak.

June 1. Animal extremely weak and emaciated. Expiration accompanied with a grunt. Back arched. Hind legs drawn forward under the body.

June 6. Heifer rapidly growing worse, but still on her feet.

June 8. Found dead this morning.

The autopsy notes of Dr. Schroeder show a case of acute miliary tuberculosis. All the lymphatic glands, including those of the limbs (shoulder and flank), were permeated with minute necrotic foci of a pale yellow color. Some glands, such as those in the thorax, were considerably enlarged owing to the tuberculous infiltration.

The severest lesions were found in the thorax, as might have been anticipated. The pulmonary pleura of the right side is uniformly, but loosely, adherent to the ribs and diaphragm, and covered with deposits of minute tubercles. At the seat of the inoculation the pleura is fully one-half of an inch thick. The right lung hyperæmic, permeated with small necrotic foci and does not collapse. The left lung, though not adherent, is in the same condition as the right.

The pericardium is completely adherent to the adjacent lung tissue. In the kidney tubercles are numerous in the cortex.

The ovaries and udder are apparently normal to the naked eye.

In the uterus there is nearly a liter of turbid yellowish fluid of offensive odor. The mucous membrane hyperæmic and streaked with hemorrhagic lines.

Tubercle bacilli were found by Dr. Schroeder in coverglass preparations of the crushed tissue of several lymph glands.

From portions of tissue reserved for me by Dr. Schroeder and hardened in alcohol sections were made, and the result of the microscopic examination is in brief as follows:

The sections of lung tissue showed when viewed with a loop a large number of tubercular foci quite uniformly scattered over the section.

They were about one-half of a millimeter in diameter and each presented a small number of darker points, the necrotic centers of the agglomerated tubercles. In each lobule there were twenty or more such agglomerations visible on the cut surface. These tubercles were made up of the usual embryonal connective tissue, with a decided tendency toward nuclear disintegration. Giant cells were very rare. Tubercle bacilli numerous.

Sections of one of the mediastinal glands, which was quite large, showed very many tubercles with central necrosis well under way. The tubercle bacilli were exceedingly numerous, but giant cells very rare.

Sections of liver tissue showed a few small tubercles provided with one or more giant cells. The bacilli were also very numerous.

In the kidneys there were numerous tubercular foci appearing in the main in the cortical portion, sheathing the minute arteries. Giant cells were very rare, and tubercle bacilli only occasionally detected.

In sections of the lower superficial cervical gland (shoulder gland) a number of agglomerations of tubercles were present, showing decided central necrosis. Giant cells were rare, but tubercle bacilli exceedingly numerous among the disintegrated nuclei.

The tubercle bacilli were thus most numerous in the lymphatic glands examined and in the lungs, the original place of deposit. They were fairly numerous in the sparse liver tubercles and quite rare in the foci in the kidney. The glands in this case, as in the natural disease, are evidently least able to resist the encroachments of the tubercle bacilli.

As regards the lung disease, I am unable to state whether the countless foci were due to the injection of the suspension into a small air tube and its dissemination into the alveoli, or whether the main distribution occurred through the blood vessels.

The case is of interest because miliary tuberculosis in a generalized acute form seems rare in cattle. I have never seen a case myself. It is probable that the number of bacilli injected are responsible for this form of the disease, inasmuch as they appear to have overpowered the animal before the mechanism of resistance and reaction could be brought into play. This may also account for the general scarcity of giant cells, which embody the reactive power of the individual and which are quite invariably present in the natural disease.

In view of this rather startling difference between the effect of the *Nasua* and the bovine culture upon cattle it may be claimed that possibly certain conditions may have favored the bovine bacillus—such as pregnancy. While I am not inclined to lay much stress upon this possibility, I hope that the inoculation experiment may be repeated soon before the prolonged cultivation shall have caused an attenuation or modification of the original virulence.

The results of the comparative inoculations upon guinea pigs simultaneously with the cattle are also in favor of the greater virulence of the bovine culture. It should be stated here that in testing the comparative pathogenic or invasive power of bacteria the most susceptible animals are of least value. It is only in the more refractory animals that differences begin to appear which give us a better insight into the degree of virulence or into any specific tendencies.

Nasua culture.—May 3: Guinea pig No. 382, weighing 312 grams, male, receives subcutaneously 0.5 c. c. of a bouillon suspension of tubercle bacilli. No. 383, weighing 340 grams, male, receives the same dose.

No. 382 was found dead on the forty-seventh day. The lesions did not present

any marked peculiarities. Besides the local ulcer, the lymph glands were large, cheesy, but still firm. The spleen is very much enlarged, infiltrated, and has necrotic patches. Liver almost entirely necrosed. In both lungs a moderate number of tubercles, some with necrotic center.

No. 383 was chloroformed on the fifty-fourth day. The lesions were much less severe, but more advanced, some glands having disintegrated foci. Liver with irregular surface; necrosis slight. Spleen extensively involved. Lungs contained a moderate number of tubercles, as in No. 382.

Bovine culture.—May 3: Guinea pig No. 384, weighing 312 grams, male, receives 0.5 c. c. of a bouillon suspension of tubercle bacilli subcutaneously. No. 385, weighing 340 grams, male, receives the same dose.

No. 384 died on the thirty-ninth day. The disease appears on the whole much more intense than in the preceding cases. The spleen, liver, and lungs are severely involved, the local ulcer large.

No. 385 died on the thirty-first day with nearly the same lesions, but less pronounced, probably because the disease had run a more rapid course.

Without insisting too much upon these inoculations as indicating a difference in degree of virulence, I still think that in these cases this difference was manifested both by the different life period after infection and the lesions. Thus:

No. 382 (*Nasua* culture) lived forty-seven days.
No. 383 (*Nasua* culture) chloroformed on fifty-fourth day.
No. 384 (bovine culture) lived thirty-nine days.
No. 385 (bovine culture) lived thirty-one days.

Nearly a year after these comparative tests the virulence of the two cultures was again tried upon guinea pigs and rabbits. The bovine and the *Nasua* bacilli had been cultivated on the same substrata, in the same thermostat, and the cultures simultaneously renewed every two or three weeks.

On March 20, 1896, cultures on dog's serum, inoculated March 4 and now presenting quite a vigorous growth, were used to make suspensions in sterile bouillon. The suspension of the bovine bacilli consisted of very minute particles, that of the *Nasua* culture of somewhat coarser clumps which it was impossible to reduce.

Rabbit No 2, weighing 1,800 grams, received 0.75 c. c. of the *Nasua* suspension into an ear vein; rabbit No. 3, weighing 2,278 grams, received 0.5 c. c. of the bovine suspension into an ear vein. The latter was found dead on the sixteenth day, although not manifestly ill beforehand. There was an exquisite embolic tuberculosis of every part of the lungs. They were densely studded with tuberculous foci, varying in size from mere specks to irregular patches 2 to 3 mm. in diameter. Generalized tuberculosis was manifested by the presence of small foci in the knee fold and axillary glands and in the kidneys. The spleen was four or five times the normal size, permeated with grayish specks, probably enlarged Malpighian bodies. The liver was large and pale, with a faint yellowish mottling indicative of fatty degeneration.

Rabbit No. 2 remained active until the fifty-fifth day, when it was chloroformed to close the experiment. The only traces of any tuberculous foci found after some search were a minute tubercle on the lateral edge of one of the left lobes, a second 1-mm. translucent tubercle, and a grayish hepatization of the extreme tip in a small lobe of the right lung. This focus was only 2 mm. in diameter. No traces of disease in other organs. Much adipose in abdomen.

The guinea pigs inoculated at the same time were adults weighing about 500 grams which, as a rule, show considerable resistance to tuberculosis as compared with younger animals. I shall not enlarge upon this part of the experiment, although the autopsy notes present suggestive features. All the guinea pigs had been used sometime before to test the strength of diptheria antitoxin.

Guinea pig No. 176 received 0.5 c. c. of the bovine suspension, subcutaneous.
Guinea pig No. 171 received 0.2 c. c. of the bovine suspension, intra-abdominal.
Guinea pig No. 102 received 0.75 c. c. of the *Nasua* suspension, subcutaneous.
Guinea pig No. 150 received 0.3 c. c. of the *Nasua* suspension, intra-abdominal.

No. 176 chloroformed on the fifty-fifth day. Very extensive invasion of the lymph glands, lungs, and spleen; liver large, mottled with gray.

No. 171 died on the thirty-third day. Extensive tuberculosis of peritoneum, liver, and lungs.

No. 102 died on the thirty-seventh day. Marked emaciation. Tuberculous changes of liver and spleen slight; lungs free.

No. 150 died on the forty-second day. Moderate tubercle eruption on peritoneum; spleen and liver only slightly affected. Lungs contain many minute tubercles.

The discrepancy between this and the preceding inoculations on guinea pigs is only apparent. In the *Nasua* animals the internal changes were comparatively slight when compared with those of the bovine cases. The early death of No. 102, associated with marked emaciation, may be due to a more poisonous action of the *Nasua* bacilli, or, perhaps, to causes unknown. Further observations must be made on this point.

This experiment reveals a striking difference in the pathogenic power of the bovine and the *Nasua* bacilli on rabbits. It shows as before a greater invasive power toward guinea pigs, coupled with more extensive necrosis of the invaded organs and tissues. The virulence does not appear to have been appreciably reduced by time in case of either culture.

Experiments to determine any difference in the thermal death point of these cultures have still to be made. They are important from a practical point of view, since there does not exist up to the present a strict unanimity upon the lowest thermal death point of tubercle bacilli in milk. The greater resistance of the bovine bacilli to the bleaching action of H_2SO_4 leads me to anticipate a greater vital tenacity of these bacilli toward heat.

MORPHOLOGY.

Nothing has thus far been stated concerning the morphology of these bacilli. This is discussed most easily by comparing them with each other.

Cover-glass preparations from parallel cultures were prepared by staining them for five minutes in carbol fuchsin and immersing them for about thirty to forty-five seconds in a 10 per cent (vol.) solution of sulphuric acid.

In serum cultures of the early generation about 2 weeks old the *Nasua* bacilli were quite uniform in size, about 2 μ long and slightly curved. In cultures 5 to 6 weeks old the bacilli had lost the regular outline to some extent. More deeply stained roundish bodies appeared within them, though not regularly so, and slightly distended them. They were also longer, fully 3 μ at this time.

On glycerin bouillon cultures 3 weeks old the bacilli were perhaps a trifle larger than those on serum. Some were irregular and contained occasional bodies more deeply stained.

The bovine culture presented some features quite different from those of the *Nasua* culture. In cultures about 2 weeks old the bacilli taken from the condensation water of serum tubes were nearly straight rods, not more than 1 to to 1.5 μ in length and about as thick as the *Nasua* bacilli. Many bacilli were swollen in the center or at one end and approached oval cocci in appearance. Degeneration was manifest. In the original culture from the guinea pig 2½ months old the bacilli appeared now 2 to 3 μ long. They were quite thick, not distinguishable readily from the *Nasua* bacilli.

In later parallel cultures the morphological differences were perhaps more defined and therefore more striking.

Cultures on dog's serum started October 12, 1895, were examined November 2. These cultures represented the eleventh of the bovine

and the fifteenth of the *Nasua* series, the date of the original *Nasua* culture being July 17, 1894; of the bovine, November 28, 1894.

Cover-glass preparations dried, heated, stained in carbol fuchsin, and decolorized in H_2SO_4 presented the following features:

Bovine bacilli, short, straight, deeply stained rods about 1 μ long. In size they were quite uniform, varying perhaps from 0.8 to 1.2 μ. With a magnification of 1,000 diameters, the rods were shown to be not very regular in outline. In some, one end tapered slightly; in others the central portion was slightly swollen. The bacilli were in masses or clumps; isolated ones not very abundant. Stain held after an exposure to H_2SO_4 for thirty and sixty seconds.

Nasua bacilli: Rods slender and more or less curved like the arc of a circle. Adhering to one another, side by side, in the usual well-known manner. In length they varied between 2 and 4 μ, the majority being about 3 μ. In outline they were not very regular, ends somewhat tapering. Staining well retained after an immersion of thirty seconds. Partial decoloration after an immersion of sixty seconds.

In this brief description it will be noted that the bacilli in these two cultures differ in form, length, and in the capacity to retain the stain in acids. These differences have thus maintained themselves and have perhaps become more accentuated within the period of a year. Anyone after one examination would be able to recognize the one or the other culture. It is not a difference of more or less, but one of kind.

Parallel cultures on dog's serum were again compared February 12, 1896, when 8 days old. In these the same differences were present. No change had taken place.

At this time the growths on the surface of the serum were easily distinguishable with the unaided eye. The *Nasua* culture had a decidedly whitish cast when the masses of bacilli had become enlarged into clumps by continued growth. They were not easily broken up, but when transferred to fresh serum persisted in cohering as little balls.

The bovine culture spread more evenly over the serum and formed an inconspicuous membrane, having the appearance of ground glass. The platinum loop passed over the surface brings with it a soft mass having little cohesion, and is easily spread out on a fresh surface.

These differences are of minor significance, yet they are of some value since all other factors likely to cause differences were eliminated by a strict uniformity of conditions.

It will thus be seen that the bovine bacilli were different from the *Nasua* bacilli in nearly every culture examined, in being shorter and less curved. This difference was also manifest in preparations of a bovine culture isolated in 1886, which were restained for this purpose. Furthermore, bacilli from a culture of human tuberculosis from Dr. Trudeau closely resembled the *Nasua* bacilli and differed from the bovine bacilli.

A number of important questions are raised by these observations. Of these only that one concerns us here which deals with the relation between human and bovine tuberculosis and the transmissibility of the latter to man. This question may be put in the following form:

Are cattle attacked by different races of tubercle bacilli, or is there but one race adapted to the bovine body?

Koch, in his deservedly renowned monograph, states that he cultivated through a number of generations tubercle bacilli from eleven head of cattle. Of these four were obtained directly and seven indi-

rectly through guinea pigs. He tacitly regards these as identical with those isolated from man and other mammalia, and we have no reason whatever to doubt his observation or judgment on this subject.

Kruse and Pansini, already quoted, describe what appear to be avian bacilli isolated from cattle.

Observers everywhere have experienced great difficulty in obtaining cultures of tubercle bacilli from man and animals. Only occasionally have they been successful. This may be ascribed either to want of skill in observing the precise conditions necessary in cultivations or else to an initial difference in the bacilli themselves, in virtue of which some are cultivable, others not. I am inclined to the second explanation, namely, that bacilli differ. The cultures which I have described demonstrate this in an unmistakable manner. Unless this were so it would be difficult to explain why, in bovine cultures made in 1886, I should have obtained cultures from two organs of the same guinea pig, and why this same thing should have happened in the *Nasua* cultures. And yet in spite of very numerous efforts I have obtained during the past eight years only these two cultures from guinea pigs which had been inoculated with tuberculous tissue from cattle. It would appear from the bacteriological standpoint that slightly different races of tubercle bacilli have been obtained from cattle. It is, however, not possible to determine definitely whether or not some of the easily cultivated and more or less attenuated bacilli were responsible for the disease process and not accidentally present. But any tubercle bacilli isolated from cattle which did not produce tuberculosis in guinea pigs should arouse our suspicion, since tuberculous tissue from cattle has always produced a generalized disease in guinea pigs.

It is difficult to assign any trustworthy value to differences manifested on culture media where the length of time during which the bacilli have been subjected to artificial cultivation is not known. Whatever value may be attached to differences manifested by absence of growth on one medium and vigorous growth on another must be derived from the earliest generations, owing to the peculiar adaptation which the tubercle bacillus undergoes. Thus the bacilli obtained from Dr. Trudeau failed in a number of trials to grow upon blood serum, while the *Nasua* bacilli in parallel cultures did just the reverse—grew well upon beef and dog's serum, but very feebly on glycerin agar. This difference might be considered sufficient to establish a variety, but I believe that it is due to the slow adaptation to certain culture media (agar) by which the power to grow on other media (blood serum) is forfeited.

This may perhaps account for the difference observed by Trudeau between two cultures.[1] One culture had been isolated by him from the lungs of a case of miliary tuberculosis and had passed through three generations only. The history of the other culture was unknown, but it was regarded as an old one. The first culture grew typically upon glycerin agar in the form of dry scales and abundantly upon potato. The growth of the second upon glycerin agar had the consistency of thick cream. Upon potato it failed to grow. This failure may have been the result of slow adaptation to glycerin agar.

If we were to take as guides the behavior of the earliest generations of tubercle bacilli toward different carefully and uniformly prepared substrata, some important facts might thereby be gathered on the different degrees of saprophytism manifested by the tubercle

[1] Proc. N. Y. Pathol. Society, 1890, p. 75.

bacillus. That such degrees exist is demonstrated by the two cultures I have described. It may also be read between the lines of the writings of others. Thus Weyl describes a successful culture on glycerin agar directly from the tuberculous spleen of a dog.[1] Kitasato obtained cultures on the same medium directly from sputum which grew at first atypically, but typically after four weeks.[2]

Passing now to differences in the manifestations of the human and the bovine disease we have at the outset the name *Perlsucht* to emphasize a difference which is perceived by the naked eye. The nodular affections of the serous membrane strongly reminds one of affections due to animal parasites. Next comes the predilection of the disease for lymph glands, as in tuberculosis of infants and children. The lung tissue is in many cases intact, so also the intestines, though the bacilli have evidently passed through them to the associated glands.

In the bovine disease,[3] the lung disease starts as a rule in the large caudal lobes, those in which we might suppose the tubercle bacilli to be deposited most easily. In the human adult the cephalic lobes in which we might suppose the fewest bacilli to be deposited are most commonly the seat of the disease. Any catarrh of the air tubes does not precede lung infections in cattle, although this is started up after the tubercles begin to bulge into and partly occlude the lumen of the affected air tube. The tubercle bacilli of cattle are furthermore not inclined to multiply in the catarrhal secretions of the air tubes or the bronchiectatic cavities found so often in lungs extensively involved. An examination of the mucopurulent contents of air tubes frequently fails to reveal tubercle bacilli, though there may be advanced disease.[4] This indicates a less saprophytic tendency than is manifested by the bacilli in human tuberculosis. In fact, it would seem that with the continued opportunity afforded by the human tubercle bacillus to multiply in the products of cavities and air tubes practically shielded from any adverse influence of the body cells, and the opportunity which such bacilli have of being transmitted and producing disease in others, varieties of bacilli might arise requiring special conditions, such as bronchial catarrh, for their successful location and further multiplication.

Differences between human and bovine tubercle bacilli might thus be predicated from the differences manifested by the disease process. Any claim which maintains that this difference is due to soil and not to the bacilli is one-sided, because it overlooks the biological fact that the effect is mutual and that change of soil or environment means in the end modification of the organism and the formation of varieties. The great sensitiveness of the tubercle bacillus to change of media—the difficulty in getting an artificial growth started—signifies that the transmission of certain bacilli from one species to another might encounter obstacles still greater than in the culture tube. Again, the tubercle bacillus is properly regarded as a parasitic organism, rather highly specialized toward an intracellular existence. It is a well-known law that the more specially fitted to certain environments the organism the less likely is it to survive or take root outside of those

[1] Centralblatt f. Bakteriologie, VI (1889), p. 689.

[2] Zeitschrift f. Hygiene, XI.

[3] Theobald Smith: Studies in bovine tuberculosis. Bulletin No. 8, Bureau of Animal Industry, U. S. Department of Agriculture (1894), pp. 88–126.

[4] In glancing over the recently published report of the royal commission on bovine tuberculosis, I noticed that McFadyean has made the same observation.

conditions. This is particularly true of protozoa. Thus the hæmatozoa of malaria fail to develop in lower animals. Those of Texas fever, though extremely virulent to cattle, are harmless to other animals. The same may be true to a less degree of the tubercle bacillus. Virulence toward one species in such organisms is no criterion of its invasive power toward other species. This must be determined experimentally or by the absolute identification of the causative organisms in the two allied diseases.

This subject has to-day assumed considerable importance, owing to the possible relation of bovine to human tuberculosis, especially among children. That much of the tuberculosis of infancy is due to infected milk is urged from many quarters, and much perturbation in the public mind has been caused by it. I have endeavored to trace in the recent literature of this subject some evidence for or against the claim. Kossel[1] reports upon 22 cases of fatal tuberculosis in which the lung infection predominated and in which an independent tuberculosis of the digestive tract appeared in but 1 case. In 14 other cases in which death was due to other causes caseous foci were found in the bronchial glands in 10, in the mesentric glands in 4 cases. Bearing in mind the habit of infants and children to put things of all sorts into their mouth, an infected environment would furnish as good an opportunity for food infection as milk. Kossel does not even touch upon the possibilities of infection through milk, but has merely collected evidence concerning the surroundings.

I have presented this subject, not as an advocate for or against the view that milk is the cause of tuberculosis, for a discussion of this subject would lead too far, but to call attention to the necessity for more information along the following lines:

(1) The existence of races of tubercle bacilli (both human and bovine).

(2) The identity or nonidentity of bovine and human tubercle bacilli.

(3) The effect of bovine tubercle bacilli on man. This problem could be attacked by cultivating bacilli from a case of tuberculosis (presumably contracted by drinking milk) and testing them on cattle, as I have done.

(4) More evidence on the subject of milk infection by tracing the source of the disease in infancy more carefully and by searching for primary foci of tubercular infection at all autopsies on children.

(5) Any existing differences between the disease produced in early life by human and that presumably due to bovine tubercle bacilli.

It seems to me that the time has come for the medical profession to probe this subject of milk infection more thoroughly, and for this purpose clinical, pathological, and bacteriological workers must unite in order to be successful.

NOTES ON THE EVOLUTION OF HOG-CHOLERA OUTBREAKS.

The restoration of the normal virulence of pathogenic bacteria, after these have been reduced by adverse agencies of one kind and another, is a very important problem in the study of infectious diseases. If it were known precisely by what means pathogenic bacteria regain or increase their disease-producing power, a long step in advance would be taken toward rational means of suppressing their activity.

[1] Ueber Tuberkulose im frühen Kindesalter. Zeitschrift f. Hygiene, XXI (1895), p. 59.

It is not necessary here to give proofs that pathogenic bacteria may and do lose some of their virulence and that they occur in nature as races possessing different degrees of pathogenic power. Former reports of this Bureau have insisted upon this fact and given copious illustrations, while many observers have commented upon the same subject by reason of observations of their own. The number of races of hog-cholera bacilli which I have had under observation since 1886 has impressed upon me the possibility of a continually fluctuating virulence and of a possible increase in virulence of the weaker races. How these changes may go on and what agencies favor them can not be answered by observation only. Experiment must give us clews to be applied as working theories to explain the occurrences going on at large in the animal world. The following pages are a contribution to this subject from the experimental standpoint.

The occasional and quite explosive outbreaks of swine diseases coming after certain long intervals are undoubtedly due in part to an increase in the pathogenic power of the provoking cause. Other agencies may also be at work, among them such as favor the transmission of bacteria, improper food, marked climatic and meteorological abnormalities which reduce the natural resistance of the body, but these, while they may produce an increase of the usual number of sporadic cases where the infectious disease is always present, can not produce epidemics excepting in so far as they favor an intensifying of the virulence of the specific disease germ in the manner to be suggested below.

It would be tempting to assume that the conditions which favor an increase of pathogenic power are the same for all bacteria. This is, however, likely to lead to false generalizations. In the following pages the facts brought out must be considered as referring to the hog-cholera group of bacteria only, or perhaps to closely related groups, such as the bacillus of typhoid fever in the human subject.

Methods of increasing the pathogenic power of bacteria experimentally may be classed under two heads:

(1) Passing the bacteria through the body of animals. A susceptible animal is inoculated with the given bacteria, and after its death either certain exudates or tissues of this animal containing the injected bacteria or pure cultures therefrom are inoculated into a second animal and the process continued.

(2) Pathogenic bacteria have been cultivated with others in the same culture media (mixed or impure cultures). Any permanent elevation of pathogenic power has not been brought about by this method, and it is probable that the temporary change was due to a favorable influence of the associated bacteria on the culture fluid.

The first method is therefore the only one deserving serious consideration as increasing more or less appreciably and permanently the pathogenic power of bacteria. Pasteur and his pupils, who have experimented with this method more than any other investigators, inject the bacteria into the most susceptible animals first (usually the very young) and use more resistant animals later on in the series. In nature this process of first weeding out the young probably goes on more or less and paves the way for a destruction of the older ones later on.

It occurred to the writer to determine whether there may not be a perpetuation of pathogenic bacteria with an increase in virulence even when infected animals do not die. In other words, may not certain causes be operative in preparing for outbreaks of infectious diseases without any suspicious manifestations? The experimental evidence of such a possibility was obtained in a simple way. The

suggestion came some years ago to the writer upon noticing the existence of hog-cholera bacilli in the body of rabbits that had recovered from the inoculation disease. The survival of pathogenic bacteria in the body of animals, as well as human beings, has also been noticed by a considerable number of observers within the past few years, and I pass over this subject.

An artificially attenuated culture of the hog-cholera bacillus isolated as far back as 1886 and kept growing on nutrient agar up to the date of the experiment was used.[1] At the beginning of the experiment it was not virulent enough to produce a fatal disease in rabbits when injected under the skin. Even small doses in the abdominal cavity were borne without leading to death. Injected into an ear vein it was still rapidly fatal.

This culture was made the starting point of a series of inoculations into rabbits. The surviving rabbit was chloroformed after recovery, i. e., after its temperature had become normal, and cultures made from one or more internal organs, usually the spleen and the liver. The culture thus obtained was injected into a second rabbit. This was killed after recovery and cultures made from its organs in a similar manner.

The following table gives the most important facts of the serial inoculations:

Table showing increase in virulence of hog-cholera bacilli in the body of rabbits.

Serial No.	Weight of rabbit.	Date of infection.	Date of examination.	Mode of infection.	Mode of death.	Dose of bouillon culture.	Age of culture.	Length of time bacteria in body.
	Pounds.							*Days.*
1	(?)	Mar. 3	Mar. 16	Intraabdominal	Killed	0.25 c. c.	24 hours.	13
2	4¼	Mar. 23	Apr. 9	do	do	0.3 c. c., from No. 1.	do	17
3	2¼	Apr. 14	Apr. 16	Intravenous	Died.	0.3 c. c., from No. 2.	do	2
4	2 13/16	Apr. 17	Apr. 25	Intraabdominal	Killed	0.25 c. c., from No. 3.	do	8
5	4¼	Apr. 27	May 12	do	do	0.25 c. c., from No. 4.	48 hours.	15
6	4¼	May 18	May 25	do	Died.	0.25 c. c., from No. 5.	24 hours.	7
7	5	May 26	June 1	do	do	0.25 c. c., from No. 6.	do	6
8	3 7/16	June 2	June 16	Subcutaneous	Killed	0.25 c. c., from No. 7.	do	14
9	3¼	June 20	July 14	do	do	0.25 c. c., from No. 8.	do	24
10	2 11/16	July 17	Sept. 6	Intraabdominal	do	0.25 c. c., from No. 9.	do	51
11	4¼	Sept. 10	Oct. 9	do	do	0.3 c. c., from No. 10.	do	29
11*a*	4	Dec. 11	Dec. 15	do	Died.	0.24 c. c., from No. 11	do	4¼
11*b*	3¼	Oct. 13	Oct. 26	Feeding	Killed	0.24 c. c., from No. 11		13
12	3¼	Oct. 12	Oct. 16	Intravenous	Died.	0.3 c. c., from No. 11.	24 hours.	4
13	4¼	Nov. 10	Nov. 27	Subcutaneous	do	0.25 c. c., from No. 12	3 days	17
14	3¼	Dec. 1	Dec. 7	Intraabdominal	do	0.25 c. c., from No. 13	24 hours.	6
15	4¼	Dec. 11	Dec. 23	Subcutaneous	do	0.25 c. c., from No. 14	do	12
16	2 5/16	Dec. 29	Jan. 8	do	do	0.5 c. c., from No. 15.	20 hours.	10
17	2¼	Mar. 22	Apr. 7	do	do	0.5 c. c., from No. 16.	24 hours.	15¼
18	2¼	Apr. 13	May 1	do	Killed	0.25 c. c., from No. 17	48 hours.	18

The fact presented in this table of most interest to us is the manifest increase in virulence toward the end of the series. Quantities of culture fluid which at the beginning of the series failed to prove fatal after intraabdominal injection proved fatal after subcutaneous inoculation later on. There appears to be a certain fluctuation and irregularity in the effects of the culture in the whole series, which, however, is explainable. Thus No. 3 died because the injection was made into an ear vein. No. 6 aborted during the inoculation disease, which probably was the cause of the fatal result. No. 7 was the first to die

[1] Theobald Smith. The hog-cholera group of bacteria. Bulletin No. 6 of the Bureau of Animal Industry (1894), p. 10.

with the usual characters of the disease (large spleen, necroses in liver, discoloration of heart muscle) as a result of the injection, pure and simple. In Nos. 8 and 9 the subcutaneous inoculation still failed to prove fatal. A slight decrease in virulence was again noted toward the end of the series, which was probably due to the interruption of work from January 8 to March 22. During this period of three and a half months the bacillus was grown on agar. Further experiments were interfered with by my removal to Boston.

The second point worthy of note is the persistence of the hog-cholera bacilli in the body of the recovered rabbits. Thus in the tenth of the series the bacilli were isolated fifty-one days after injection, in other cases twenty-four and twenty-nine days respectively. Inoculation of bits of tissue as large as peas was usually resorted to, and in nearly every case the culture proved fertile and pure. The chances of fertility were slowly diminished with the increase in the length of time after inoculation. In no case was any attempt made to determine the longest period of vitality of the hog-cholera bacilli in the tissues of the rabbit. Another interesting point brought out in this series is the penetrating power of the bacilli after they have been placed under the skin. In Nos. 8 and 9 the injection, though subcutaneous, was followed by the dissemination of the hog-cholera bacilli into spleen and liver, where they were found respectively fourteen and twenty-four days after inoculation. This penetration is still more striking in the case of a rabbit [No. 11*b*] fed with cultures from No. 11. The temperature of this case was not taken, but it appeared to be in good health when it was chloroformed, thirteen days after the first feeding. Three bouillon tubes inoculated with pieces of spleen and one with a piece of a kidney became clouded with the injected bacilli.[1]

The principle which it has been my object to apply in this investigation is to incite a modification of the hog-cholera bacillus by a prolonged though frequently broken sojourn in the tissues of the rabbit and in intimate relation with the cell and other forces operative against the intruding bacteria. Up to the twelfth of the series, where a marked increase of virulence was first observed, the bacilli had been in the body of rabbits for an aggregate period of one hundred and eighty-six days. The presumption was that this intimate contact between animal tissues and bacteria would strengthen in the latter that unknown element which we call pathogenic power, and this presumption has been sufficiently established by this series of inoculations.

Basing ourselves on the facts presented in the foregoing pages we may construct a theory as to the evolution of an epizootic from insignificant beginnings. In the case of the attenuated hog-cholera bacillus in rabbits all that would be necessary is a transference of bacteria from the body of one rabbit to another. This transference is possible because in the mild nonfatal disease in rabbits the hog-cholera bacillus localizes in the follicles of the appendix of the cæcum, less frequently in Peyer's patches. Thence a discharge from the occasionally ulcerated follicles may set some bacilli at liberty, which, gaining entrance into the body of another rabbit with the food, may continue the series toward a final epizootic explosion.[2]

[1] These were identified with the microscope and by the formation of a characteristic membrane not seen in cultures of other hog-cholera or of colon bacilli.

[2] A discharge of bacilli from the liver takes place regularly in the acute fatal form of the inoculation disease in rabbits. Whether the localization of the bacilli in the follicles of the intestine is dependent on this discharge of bacilli from the liver in mild cases I am not prepared to state.

It is not to be inferred that any explanation of the development of pathogenic from nonpathogenic bacteria is hereby offered. In what manner such an organism as the hog-cholera bacillus acquired its power of penetrating into and multiplying within the animal body and producing disease is as difficult of explanation as the appearance of species of higher plants and animals. The view here presented simply attempts to explain how certain pathogenic bacteria may maintain and even augment their disease-producing powers in insidious and unlooked-for ways. It attempts to explain how, with the removal of all manifestly diseased animals, the infection may still be present and show itself at some future time.

Another line of thought of considerable importance is the frequently urged possibility of so raising the strength of animals that they may successfully resist the attacks of disease-producing bacteria. If this were possible it would be no longer necessary to eradicate the disease germs, to suppress them wherever they may be supposed to exist, but simply to turn our attention to the animals. This theory is acted upon in all attempts at preventive inoculation where the animal body is artificially prepared for the attack of the disease germ. The results of such preventive treatment vary with each new disease germ. For hog cholera there is little hope of ever gaining any clear insight into the operations of vaccines owing to the frequent occurrence of two or more different disease germs in a single outbreak, and until more definite knowledge is obtained, nothing can be learned concerning the effects of vaccination on the offspring. It has also been expressed that the frequent appearance of infectious diseases would weed out the weaker animals and leave only the stronger to produce offspring. This may be true in a state of nature, but when we bear in mind that domesticated animals are bred in such a way as to produce a certain result, and are thereby made to a certain degree abnormal, it is evident that natural selection can not help us out, especially as the tendency toward making domesticated animals more and more dependent on mankind is growing rather than diminishing. The necessity of fighting the causes of infectious diseases is destined, therefore, to increase rather than diminish in the future.

The practical lessons to be derived are applicable, as stated at the outset, only to bacterial diseases whose causes are closely related to the hog cholera bacillus. Limiting ourselves to this disease as the greatest scourge of swine, we may make several deductions:

(1) The chief carriers of the infection are the swine themselves. This disease having its chief seat in the intestines, a discharge of bacilli from the ulcers of chronic cases, or of such as have survived an attack, may take place long after the subsidence of an outbreak or after they have changed hands. Infection may thus be carried over in the herd until a new susceptible generation of young swine appears to continue the losses. Outbreaks occurring without any traceable importation of infection from without are very probably due to latent infection in the herd itself.

(2) The custom prevailing in some parts of the country of not promptly removing dead hogs, or of allowing them to be gnawed at or even partly consumed by the living, is a potent cause for the perpetuation and strengthening of the infective agent. In such cases the bacilli consumed may cause mild, unrecognizable attacks, with discharge of bacilli from the bowels subsequently.

(3) Preventive inoculation with living cultures may disseminate and perpetuate the disease, because the attenuated vaccinal cultures may

regain their normal virulence in the body of swine after a certain lapse of time.

(4) The waves of epizootics which appear to sweep over the country at long intervals may be due to a sudden increase in virulence of the specific bacilli after they have been passed through the body of swine for some years.

(5) Pathogenic bacteria are always a menace, and no pains should be shunned to restrict their dissemination and multiplication in every possible way by quarantine, by disinfection, and by the destruction of the dead with fire, if possible.

SWINE ERYSIPELAS OR MOUSE-SEPTICÆMIA BACILLI FROM AN OUTBREAK OF SWINE DISEASE.

The prevalence of much disease among swine in Minnesota during the fall and winter of 1894 led Dr. C. N. Hewitt, secretary of the State Board of Health, to communicate with the Department concerning the nature of this presumably infectious disease. According to Dr. Hewitt, the lesions were obscure, resembling neither those of hog cholera nor those of swine plague, but manifesting a septic character.

According to a statement made by M. H. Reynolds, veterinarian of the experiment station, about 350 animals died in one township, the mortality ranging between 60 and 100 per cent.

Dr. Reynolds found the lungs "involved in every case examined, particularly the ventral and cephalic lobes, the diseased areas abrupt; frequently, but not invariably, pleuritis with fibrinous exudate. Bowels frequently show more or less enteritis, especially the large colon, but no typical button ulcers of hog cholera."

On November 3, 1894, there were received small pieces of tissue in test tubes taken from a pig which had succumbed to this swine disease. The pieces were sent October 29 by Dr. Reynolds, and received in evidently unchanged condition. Four rabbits were inoculated subcutaneously with bits of tissue on November 3, two with particles of spleen pulp, the third with a bit of lung tissue, and the fourth with a bit of what was labeled postpharyngeal lymph gland. Only the fourth rabbit became ill. The others were carefully watched, but the temperature remained normal. This animal was the source of a pathogenic bacillus, known as the mouse-septicæmia or swine-erysipelas bacillus.

Subsequently Dr. Hewitt sent a number of cultures made from different pigs. These were referred for examination to Dr. Moore, and he found in the cultures from one animal the true hog-cholera bacillus. That the swine-plague bacterium was also concerned in this outbreak I have no doubt in view of the description given by Dr. Reynolds of the cases examined by him. The outbreak was thus a mixed one in which several pathogenic species of bacteria were involved. Inasmuch as the relation of the mouse-septicæmia bacillus to swine disease in this country is not established, and since it is a serious scourge to swine in Europe, it was thought best to examine this organism more closely and to publish the results of such examination, for this species may at any time assume a more important part than it has hitherto played in the production of swine diseases.

Morphology.—This bacillus when mounted in balsam directly from the organs is from 1.5 to 2 μ long and 0.3 to 0.4 μ broad. It is straight, with ends squared off.

In the ordinary peptone bouillon it ranges in length from 1 to 2 μ. Sometimes filaments as long as 8 μ are found. These are usually

slightly wavy. In some cultures only the long wavy, almost spirillar, forms are found.

In a fermentation tube containing lactose bouillon all bacilli were both thicker and longer than in ordinary bouillon.

On the surface of inclined agar the growth, two days old, consisted of quite short bacilli 1.2 to 1.5 μ long.

The bacilli stain readily in alkaline methylene blue, and in carbol fuchsin; they also take the Gram stain. In sections of tissues they were brought out distinctly by hæmatoxylin. Motion has not been observed.

Biology.—The cultivation of this bacillus is best carried on in nutrient gelatin, which gives a distinctly alkaline reaction with litmus paper.

In plates and rolls made with such alkaline gelatin the colonies, when separated from one another by a distance of at least 2 to 3 cm., are visible under a low power of the microscope in forty-eight hours as round, sharply defined disks. As they become visible to the naked eye they appear at first like the beginnings of a fungous growth. In five or six days the surface and the deep colonies are easily distinguished. The former, about 3 mm. in diameter, appear as hazy spots situated in a concave shallow depression, the result of liquefaction. The deep colonies, now about 5 mm. in diameter, appear as circular spots of a uniformly delicate, cloudy, or hazy appearance and without liquefaction. In seven days these may be 8 to 10 mm. in diameter.

When these colonies are examined slightly magnified at different stages of growth, they appear at first as a central, compact nucleus surrounded by a wide zone made up of interlacing lines of variable length. Later minute zoogloœa appear in this zone, closely resembling in form those of *Proteus vulgaris*. In the largest deep colonies the nebulous zone consists of a feltwork of lines, zoogloœa, etc., running in various directions.

In gelatin tubes the stab culture grows in the characteristic manner described by all observers. From the track of the wire within the gelatin a delicate, fluffy growth radiates laterally in all directions and soon appears like a test-tube brush embedded in the gelatin. The lateral growth slowly extends until within two weeks it has reached the sides of the tube so that the entire gelatin has assumed a hazy appearance. In the meantime the liquefaction has progressed slowly down the needle track and is followed by the evaporation of the liquefied gelatin until a funnel-shaped hole is formed in the gelatin, which may extend to the very bottom of the tube in older cultures. A surface growth is uniformly absent.

The difference between neutral or feebly acid gelatin and that which is distinctly alkaline to litmus was brought out in a series of cultures on gelatin not properly neutralized. There was, first of all, no liquefaction noticed at any stage of growth. The track of the needle did not develop into the beautiful fluffy cylinder, but remained restricted, as in cultures of other bacteria. When the density of the acid gelatin was reduced by the addition of water in different quantities the same results were obtained. This difference of appearance in acid and alkaline gelatin it is well to bear in mind, especially in its bearing on the unnecessary creation of varieties or distinct species by different observers.

On the surface of the nutrient agar as usually prepared this bacillus multiplies feebly. In the thermostat the colonies appear after twenty-four to thirty-six hours as exceedingly minute, translucent dots, circular in outline and convex in form. These remain discrete

even when crowded together. When there are but few in the culture, each colony may become 1 to 1.5 mm. in diameter. The condensation water becomes clouded and a deposit forms in it.

The behavior of this bacillus varies toward the different sugars. In bouillon containing 1 per cent dextrose the growth is hardly better than in ordinary bouillon containing the usual quantity of muscle sugar. The reaction becomes distinctly acid and remains so.

In lactose bouillon the multiplication is more vigorous. The reaction becomes distinctly acid and remains so.

In saccharose bouillon there is either no clouding at all or only the faintest indication. The reaction is unchanged. The same is true of bouillon free from all sugars. The sugar thus appears necessary for its subsistence.

Anaërobiosis.—This bacillus while multiplying in the ordinary test tubes on agar and in bouillon has pronounced anaërobic tendencies. This may be easily witnessed in the fermentation tube. The closed branch becomes clouded first, and the bulb may remain permanently clear. When the multiplication is more active, the clouding extends into the open bulb. The multiplication of these bacilli fails, as has been noted above, when sugars (dextrose or lactose) are absent. This failure to multiply extends to the anaërobic as well as to the aërobic cultures. When sugars are present, the multiplication is at first anaërobic.

The amount of acid produced in bouillon containing 1 per cent of dextrose and lactose is nearly the same. Titrated with a one-twentieth normal solution of potassium hydrate and phenol phthalein as indicator, 5 c. c. of culture fluid required about 4 to 4.5 c. c. of the solution. In other words, 100 c. c. of the culture fluid require from 4 to 4.5 c. c. normal alkali.

In sterilized milk there is no change manifest, even after weeks in the thermostat. When, however, such milk is boiled, it may or may not coagulate.

November 30, 1894. A tube of milk inoculated from an agar culture.
December 12. Milk fluid, reaction acid. When boiled with a control tube of the same milk it becomes quite thick, while the latter remains fluid.
December 18. Milk inoculated from a bouillon culture. Reaction of milk acid.
December 24. Many bacilli in clumps in the milk. Reaction acid.
December 27. Milk becomes solid when boiled.
December 27. Milk inoculated from an agar culture.
January 3, 1895. Both it and control tube remain fluid after boiling.
February 9. Three tubes of milk inoculated and placed in the thermosat with a control. The initial reaction of the milk is acid.
February 15. The four tubes boiled for half an hour. No thickening noticed in any tube.

This bacillus produces acids in presence of lactose, as noted above, and this property explains its action upon milk.

In the usual peptone and salt solution (1 per cent peptone, one-half of 1 per cent sodium chloride) no multiplication took place either in the thermostat or in the laboratory. In one instance a little dextrose was added with equally negative result.

On potato no visible growth appeared.

Repeated inoculation of the surface of carefully coagulated beef's serum failed in every case.

In peptone bouillon faint uniform clouding is observed in one or two days after inoculation, according to the material inoculated. When such a tube is shaken the entire fluid appears as if suddenly

permeated with rolling clouds of smoke. This difference in the appearance of the fluid at rest and in motion is probably due to the pronouncedly rod-like shape of the bacilli, and is almost diagnostic of the purity of the culture. The cloudiness after some days becomes less marked as the bacilli settle to the bottom. Here a viscid, ropy mass may appear, made up of bacilli in a state of degeneration. A collection of bacilli on the surface of the culture fluid was not detected at any time.

Pathogenic action.—This bacillus corresponds closely in its effect upon animals with that of rouget (swine erysipelas) and mouse septicæmia, as described by former observers, and no new facts are brought to light in these studies. The detailed results of inoculations are given in subsequent pages, and a brief summary is all that is necessary here.

Two gray mice which received subcutaneously about 0.06 c. c. of a bouillon culture died in two and two and one-half days, respectively, after inoculation. Bacilli numerous in the organs, occasionally within cells in clumps.

Of three pigeons inoculated subcutaneously with small doses of culture fluid one died in fifty hours, a second in three to three and one-half days, and a third in three to three and one-half days. The lesions in these pigeons varied somewhat. In all, however, there was evident a marked phagocytosis in the blood and the liver.

Besides these quite susceptible animals the rabbit appears somewhat less so. One rabbit inoculated subcutaneously with a bit of infected tissue dies in seven days. A second animal inoculated subcutaneously with 0.18 c. c. of a fresh bouillon culture dies in six days.

A third animal which received the same dose into an ear vein dies in forty-eight hours. During the disease the temperature continues very high. The animal sits quietly in its cage and fails to arouse attention by any unusual symptoms.

The lesions found at the autopsy are characteristic of acute infectious diseases, for there is marked parenchymatous degeneration of the various organs. The bacilli, however, are so scarce that they are, as a rule, not found in cover-glass preparations. Cultures become fertile when bits of tissue are introduced. The number of colonies is always small.

Guinea pigs were found, as a rule, insusceptible. In one lesions traceable to the injected bacilli were found in the walls of the cæcum, but in this animal the greatly retarded disease was evidently favored by a peculiar hemorrhagic condition which results from a restricted diet.

The inoculation of swine-erysipelas bacilli into pigs by European investigators has failed in some cases to produce disease, while it has succeeded in others. In the following attempts it also failed to produce any disease:

November 14, 1894. Pig No. 154, male, grade Berkshire, about 6 weeks old, and weighing 15 pounds, receives subcutaneously 1 c. c. of a bouillon culture 24 hours old.

No. 155, of the same breed and age, receives 5 c. c. of the same culture.

In neither was any elevation of temperature noticed during the week following the injection.

Another pig (No. 156) of the same age and breed was penned with these for the purpose of noting any transmission of disease that might subsequently take place. On November 17 it was removed and fed with the viscera of one rabbit and several pigeons which had succumbed to inoculation with this bacillus. The feeding was without effect.

In the study of animals inoculated with this bacillus several interesting facts came to light. The notes on the various cases are therefore given in full:

The history of the rabbit inoculated with the particle of tissue sent from Minnesota is as follows:

Rabbit No. 216, male, inoculated November 3. Temperature three days later, 105.6° F. On the seventh day the rabbit was found lying on its side, breathing rapidly. Eyes no longer sensitive to the touch. Muscles of head twitching. The post-mortem examination showed at the point of inoculation slight whitish infiltration in the subcutis. The spleen is twice the normal size, dark, soft. Contains much pigment and many red corpuscles within cells. Liver rather dark, free from necroses. Heart in diastole; muscles slightly discolored. Granular condition of the fibers. Cortex of kidneys somewhat pale. Urine from bladder turbid, contains a slight amount of albumen. In the small intestine a reddish viscid fluid. Brain free from macroscopic changes. Bacteria were not detected microscopically in the blood, spleen, or local exudate. Cultures were made from the blood, spleen, liver, kidneys, and local lesion, particles of tissue being used because of the negative microscopic examination. Only agar and bouillon were used.

On the second day (Sunday intervening) all cultures had developed excepting that from the local lesion.

The bouillon tubes were feebly clouded, and on shaking them a peculiar smoky appearance was produced, or rather that of rolling clouds.[1] On the inclined agar surface only a small number of colonies (from 2 to 25) had appeared, the largest number in the liver culture. These colonies were very small, like translucent droplets, about 1 mm. in diameter.

The microscopic examination revealed in all tubes small, slender bacilli, which in the bouillon frequently appeared as long, slender filaments when examined unstained.

November 12. Two gray mice received each about 0.06 c. c. of a bouillon culture from the kidney of rabbit No. 216, now 48 hours old.

November 14. One mouse, very sick in the morning, dies at noon. Slight œdema of the subcutis at the seat of inoculation. Spleen rather large, liver pale. Bacilli in spleen and blood now and then in clumps within cells. Cultures were made from the spleen and the blood on agar and in bouillon and in a gelatin tube.

November 15. The agar culture, made by simply piercing the spleen, shows to-day an immense number of exceedingly minute discrete translucent colonies.

Bouillon culture from the blood characteristic, both to the naked eye and when examined microscopically. The gelatin stabs from spleen show a feeble indication of growth, that from the blood still undeveloped.

The second mouse was found dead this morning. Numerous fine bacilli in heart's blood, mainly in clumps.

November 16. Agar slant from the blood shows many minute translucent droplets.

November 17. Bouillon culture from the blood now clouded for the first time.

November 15. Two adult pigeons (Nos. 1 and 2) received subcutaneously 0.12 and 0.3 c. c., respectively, of a bouillon culture made from the blood of mouse No. 1, and now 24 hours old.

November 17. No. 1 very ill, resting on its keel on floor of cage. Respiration labored. No. 2, with feathers ruffled, stands quiet on floor of cage.

No. 1 dies at 4 p. m. Local lesion insignificant. Mucosa of mouth bluish. Pectoral muscles have a peculiar mahogany color. Liver shows a meshwork of distended vessels on the surface. Ecchymoses in pancreas. Duodenum injected. Peyer's patches appear from the serous surface as aggregations of pale dots. On mucous surface, swelling and reddening, bordering on hemorrhage. Probably a result of discharge of bacilli from the liver into the intestine. In the liver there are immense numbers of the slender bacilli, in the blood few. An agar slant inoculated with a delicate wire thrust into the liver shows on the second day a dense layer of the minute discrete colonies. They are so small as to be scarcely distinguishable without a hand lens. Bouillon from the blood feebly clouded.

Pigeon No. 2 was found dead November 19. The seat of inoculation was occupied by pale necrosed muscle tissue about 0.5 cm. deep and extending over an area 2 cm. square. Liver as in No. 1. No lesions of pancreas or Peyer's patches. Immense numbers of bacilli in the liver, few in the blood.

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[1] This peculiarity led me to suspect the swine erysipelas bacillus at once, although I had not given it any attention for a number of years.

Agar cultures from blood and liver show in two days a dense layer of the minute colonies.

December 18. A pigeon was inoculated subcutaneously with two loops of a bouillon culture derived from guinea pig No. 255, and 12 days old.

December 21. As the pigeon is apparently unaffected and there is some doubt as to the vitality of the culture used, this pigeon is inoculated subcutaneously with a small loop of a bouillon culture 20 hours old, derived from an agar culture of the liver of rabbit No. 216.

December 23. Found dead this morning. Liver with the network of injected vessels as in previous cases. Somewhat faded hemorrhagic spots in the pancreas. Pericardium distended with colorless serum. Clumps of bacilli in blood. From this animal were inoculated an agar tube from the liver, a gelatin tube from the spleen, and a lactose-bouillon fermentation tube from the blood.

On the second day growth in all tubes in the fermentation tube limited to the closed branch. This became markedly acid.

December 27, 2.30 p. m. A pigeon is inoculated subcutaneously with a loop rubbed over the agar culture from preceding case.

December 31. Found dead this morning. Lesions of liver and pancreas as in preceding case. Cultures positive.

The liver of pigeon No. 2, hardened in strong alcohol, was examined in sections stained in various ways.

The vessels, both the large interlobular and those composing the intralobular network, were very much distended and filled with red corpuscles. This condition had already been noted in the fresh liver in which the interlobular vessels formed an injected network. The parenchyma appeared extensively vacuolated as a result of fatty infiltration or degeneration. The nuclei are preserved. The largest vessels are bordered by zones of leucocyte infiltration which occupy the perivascular space. Within all the vessels, but more particularly the intralobular capillaries, there may be seen large deeply stained cells, which are especially striking when seen with a low power of the microscope. They form irregular groups or lines, not limited to any definite zone of the lobules.

Under a high power these cells, which are from 10 to 15 μ in diameter, are found filled with bacilli, to whose presence the intense staining is due. In some cells only a small number of such bacilli, mingled with some irregular chromatin particles, is seen; in others a dense mass is present, obscuring completely the cell itself. The character of these cells is not determinable, although it is probable that they correspond to the large circulating phagocytes which are detained in the capillaries of the liver. They are situated as a rule against the wall of the capillary, although this is not invariably true. Their situation here gives the impression of enormously swollen (phagocytic) endothelial cells projecting into the lumen of the tube. The bacilli are seen not only in these cells but also in linear groups along the walls of the capillaries. It is probable that there had been post-mortem multiplication both within and without the phagocytes, as the pigeon died in the night.

November 15. Two guinea pigs were inoculated at the same time with the pigeons, all with the same culture.

No. 254 (male, weight 560 grams) receives subcutaneously 0.25 c. c. of the bouillon culture.

No 255 (male, weight 530 grams) receives into the abdomen 0.25 c. c. of the same culture.

December 5. No. 255 found dead, although it had not been ill enough to be noticed before this.

The spleen is small, the liver uniformly fatty; lungs not collapsed. The heart contains dark clots. The most marked change is in the cæcum. This is hyperæmic. The mucosa is dark, hemorrhagic, with superficial necrosis in limited areas. The adjacent portion of colon also hyperæmic.

The long period between inoculation and death suggested that the cause of death might be something besides the inoculated bacilli. Hence cultures in bouillon and on agar were made only from bits of the liver. On the slant agar a large number of characteristic colonies of the injected bacilli appeared. The bouillon contained likewise only these.

Sections of the cæcum, after hardening in alcohol, showed that the lesion was due to the swine-erysipelas bacilli. The mucosa was necrosed in patches and the submucous tissue considerably increased and quite uniformly infiltrated with polynuclear leucocytes. This infiltration extended into the muscular coat. In one portion of the inflamed tissue the leucocytes had collected in a dense mass, inclosing thrombosed vessels. The blood vessels generally are very much distended and gorged with red corpuscles.

In sections stained according to Gram swine-erysipelas bacilli were found in abundance. They occupied the walls and surrounding tissue of thrombosed vessels in dense nests and were diffused generally through the diseased tissue. Nests were seen in the inflamed muscular tissue and in the infiltrated submucosa.

These sections, coupled with the cultures, indicate that the guinea pig probably succumbed to the injected bacilli, and that the lesions in the cæcum were directly due to them.

No. 254 died December 14, twenty-nine days after inoculation, after having shown signs of illness for a few days. There was no local lesion. On both hind limbs subcutaneous and deep patches of hemorrhagic discoloration. One small patch on mucosa of stomach. Cæcum hyperæmic. On mucosa scattering ecchymoses and small yellow necrotic spots. The ileocæcal valve encircled by a necrotic line. Cultures were made with bits of spleen, liver, and loops of blood. All but the liver tube remained sterile. In this (agar) a few colonies, not of the injected bacilli, appeared.

To explain the death of this guinea pig it will be necessary to record some facts which have been observed for some years in this laboratory. When guinea pigs are fed with cereals (bran and oats mixed), without any grass, clover, or succulent vegetables, such as cabbage, a peculiar disease, chiefly recognizable by subcutaneous extravasation of blood, carries them off in from four to eight weeks. The death of No. 254 was undoubtedly due to the absence of such food, as the attendant had neglected to provide it after the disappearance of grass in the fall of the year. Furthermore, No. 255 was weakened by the restricted diet and succumbed to an inoculation which otherwise might have had no visible effect. This is clearly demonstrated by the following test experiment:

December 29, 1894. Two guinea pigs (Nos. 298, 299) received into the abdomen 0.25 and 0.4 c. c., respectively, of a bouillon culture of this bacillus.

At the same time two others (Nos. 307, 308) received into the abdomen 0.25 and 0.65 c. c.[1] of the same culture. These two and a control guinea pig were put into the same cage and fed only oats and bran mixed, while the two first mentioned received daily rations of cabbage in addition.

The outcome of the experiment demonstrates the fatal result following the abstraction of green food. While those fed with cabbage survived, those not fed with it died.

January 22. No. 308 found dead to-day. Animal emaciated; blood tarry. Liver quite dark uniformly. On right lung several dark red patches. No other changes. Blood disks normal. Tubes inoculated with bits of liver and spleen and with loops of blood from the heart remain sterile.

January 23. The other inoculated guinea pig and the control pig are now quite ill. They are very quiet and their movements feeble. Coat staring.

January 26. Cabbage again fed to-day.

January 28. Both found dead. Cultures not made, as the writer was out of town.

These results show once again that the guinea pig is not susceptible to this bacillus excepting perhaps under aggravated conditions.

Since rabbits have been found not uniformly susceptible to this disease germ, and since the first one contained very few bacilli in its organs after death, two others were inoculated.

November 14. Rabbit No. 230 (male, weighing 3¼ pounds) received subcutaneously 0.18 c. c. of a bouillon culture of swine erysipelas bacilli 20 hours old. This culture was derived from a colony on an agar culture made from the spleen of rabbit No. 216. Culture very feebly clouded.

November 15. Temperature above 105° F. (record lost).

November 17. 106.6° F.

November 19. 105° F. Rabbit quite ill.

November 20. Dies at 4 p. m.

Local lesion insignificant. Blood in subcutaneous veins in heart and other organs thick, tarry, not coagulated. Spleen soft, enlarged. Liver shows distinct lobular mottling; interlobular vessels prominent. Kidneys very pale, urine turbid; gives after filtration an abundant albuminous precipitate. Lungs œdematous, not collapsed. Small intestine contains a translucent yellow, viscid fluid.

Bacilli were not detected in the slight abdominal fluid, nor in the liver, spleen, blood, and kidney.

The following cultures were made:

1. Bouillon from spleen; stab with delicate platinum needle.
2. Agar slant from liver; stab with delicate platinum needle.

[1] Owing to a slipping of the guard on the piston rod this larger dose was injected unintentionally.

3. Gelatin stab from kidney with delicate platinum needle.
4. Agar slant with loop of blood.
5. Bouillon with loop of blood.

On the following day only 4 and 5 show development.

On the second day the colonies in 4 are more distinct. They are from 1 to 3 mm. apart, scattered over the inclined surface. The other tubes remain sterile.

In sections of liver tissue hardened in corrosive sublimate no lesions were noticed. In sections of a kidney the only lesions were traceable to the glomeruli, many of which were somewhat retracted and the space between them and the capsule filled with an amorphous granular substance.

November 14. At the same time with the preceding rabbit No. 231 (male, weighing 3¼ pounds) received into an ear vein the same quantity from the same culture.

November 15. Temperature above 106° F. (record lost).

November 16. Rabbit lying on side quietly; dies at 3 p. m.

Spleen slightly enlarged and darker than normal. Liver uniformly dark. Much free pigment present. Both sides of heart distended, with pale clots, pale serum, and some dark coagula. Intestines empty.

Cultures from the liver, spleen, and blood were fertile on the second day.

GENERAL SUMMARY.

The bacillus described in the preceding pages has not been definitely proved to be pathogenic toward swine, because those inoculated and fed with the bacilli remained well. However, these negative results can not in themselves set aside this organism as harmless to swine, since the whole question is a complicated one and far from solution. This is made clear by the history of the investigations. In 1878 Koch described a mouse-septicæmia bacillus obtained from putrefying blood. Some years after a bacillus almost identical with it was found as the cause of an infectious swine disease (swine erysipelas, *rouget*, *Rothdauf*[1]). The two bacilli were naturally enough regarded as quite distinct, though related species at that time. Since then the discovery of varieties among other species has made this old position more or less untenable. The slight distinctions based on cultural characteristics by which the mouse-septicæmia and the swine-erysipelas bacilli have been distinguished are too unstable, too readily modified, and too easily influenced by the culture medium to be of much value. This is shown in the preceding pages, where a slight difference in the reaction of the nutrient gelatin is described as leading to a wholly different appearance of the growth. It is probable that both bacilli are one and the same, and that a difference in the degree of virulence of bacilli from different sources accounts for its capacity or incapacity to produce epizootics among swine.

Whether the bacillus described is the cause of much disease in this country is not known. Diseases described as swine erysipelas by European writers have not come directly to our notice, although they may have prevailed without being recognized at the time. This is the third time that this species has been encountered in the body of swine in this country. It was found in 1885, in 1888, and in 1894. The bacillus of 1888 has been described by Dr. V. A. Moore.[2] The present culture seems to be identical with it if we except a greater virulence toward rabbits. It is not improbable that this bacillus may gain enough virulence to produce epizootics, if such is not already the case, and that in endeavoring to trace the causes of swine diseases a search for the swine-erysipelas bacillus should not be neglected.

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[1] See also Bulletin No. 6, Bureau of Animal Industry, p. 91.

[2] The Journal of Compar. Medicine and Vet. Archives, 1892, p. 333.

In a recent communication Babes, Starcovici, and Cartiano[1] describe an outbreak of swine disease in Roumania in which they found the swine-erysipelas bacillus associated with another pathogenic bacterium called by them the bacillus of pneumo-enteritis. This latter organism is on their own experimental evidence—its morphology and its rapidly fatal action on guinea pigs—the swine-plague bacillus or one closely related to it. The bacillus of swine erysipelas presented characters the same as those usually ascribed to it, but it failed to produce any disease in swine after inoculation of the culture and after feeding the cadavers of infected animals. Their results thus agree with the negative ones reported above by the writer.

While they go so far as to doubt even the natural contagion of this malady on the strength of their negative results, I am inclined to consider this conclusion too far-reaching in the face of positive results obtained by former experimenters. It is highly probable that races of this bacillus exist which possess different degrees of virulence, and that the virulence determines the severity of the outbreak.

Among the interesting facts brought out in the foregoing pages is the effect of food upon the appearance of disease. A guinea pig inoculated with this bacillus died after several weeks with hemorrhagic lesions of the cæcum containing the inoculated bacilli in large numbers. While the immediate cause of death was the multiplication of these bacilli, yet the susceptibility not present in healthy pigs was produced by improper feeding.

The writer has several times in the past called attention to the importance to be attached to rational feeding as a preventive in regions permanently infected with certain disease germs. These disease germs gain access to the body, but fail to multiply until the condition of the animal is reduced. We may go further and maintain that in many instances pigs die from diseases brought on directly by improper feeding, and that any disease germs found in one or more of such animals by bacteriological methods may have no direct relation to the disease.

We may thus have on the one hand outbreaks due directly to pathogenic bacteria of a high grade of virulence; on the other, we may have outbreaks due to food unfit to nourish the animal or containing toxic substances. We may also have deaths due to a combination of these two causes, the one or the other predominating, as the case may be. It is probable from our experience that outbreaks of swine diseases due to the virulence of bacteria alone are rare, and that therefore much prevailing disease may be prevented by an attention to the physiological laws governing the body.

NOTES ON PECULIAR PARASITIC AFFECTIONS OF THE LIVER IN DOMESTICATED ANIMALS.

The liver of animals seems to be a frequent seat of certain peculiar circumscribed lesions which have no direct influence on the health of the affected animals, but which may be mistaken for more serious affections, and whose nature should therefore be determined if possible. For a number of years past the writer has given more or less attention to them, without, however, coming to any definite conclusion regarding their nature, excepting a provisional one that they are in some way due to animal parasites. These lesions have been found in the liver

[1] Recherches exp. sur le rouget et la pneumo-enterite du porç. Annal. d'l Institut de Pathol. et de Bacteriol. de Bucarest, V (1892–93), p. 454.

of cattle, horses, and swine, and may be of several different kinds, suggesting several different causes.

Those in the liver of swine are of two kinds. One of these has been briefly mentioned by writers heretofore (Welch, Dinwiddie), and will be discussed below. The other lesion I have encountered in swine from several different herds reared not far from the District of Columbia, and as it has not been hitherto referred to I shall describe it somewhat in detail.

On the surface of the liver there are present minute reddish-yellow points or dots situated within the lobule. Each lobule may contain from a few to a large number of such dots, which occupy no definite position in the lobule. These minute specks may be removed from the liver tissue, as they are firmer and more coherent than the latter. When crushed and examined under the microscope, only the peripheral portions are seen to be made up of cells. The central portion is homogeneous, translucent, and apparently structureless.

In sections of liver tissue hardened in alcohol and stained in various ways the liver tissue itself is no longer recognizable as such in these foci. It is replaced by compact groups of round cells in the smaller foci and in the larger by patches of amorphous matter which stains very feebly and which is surrounded by a zone of round cells. Distorted and fragmented nuclei, as well as free chromatic particles simulating cocci, are usually present within the necrotic region.

These foci were very numerous in some cases, and perhaps one-half of the parenchyma as seen in the section was destroyed by them. The capillaries of the remaining liver tissue were usually distended more or less. In other cases only a few foci were present, and these were situated either in the external or the middle zone of the lobule. The foci are uniformly distributed, and no portion of the liver appears to be free from them. Taking all facts into consideration, we must regard the lesions as the result of embolism. Minute particles carried in immense numbers in the blood of the portal vein into all parts of the liver are lodged in some one or more capillaries within the lobule. The vessel becomes plugged and the nutrition of the surrounding tissue interfered with. Necrosis of the liver tissue takes place, by which process a minute mass is converted into the firm, yellowish particle, barely visible to the unaided eye in the fresh liver. The slight amount of reaction which takes place and the restricted size of these minute foci make it probable that the particle which causes the disturbance is not infectious, i. e., not a living, multiplying organism. With this hypothesis in view I examined a large number of necrotic foci under the microscope, after teasing them out of the surrounding normal liver tissue and crushing them between slide and cover glass. In but one of the foci a foreign body was seen. It was the double-contoured ovum of some nematode, with a long diameter of 0.063 mm. and a short diameter of 0.04 mm. It contained a coiled-up, degenerated embryo full of large, fat globules. This body was, however, too large to get into any of the intralobular capillaries and produce disturbances there. It was more probable to assume the disintegration of some nematode accidentally within the portal system and the distribution of minute particles from the disintegrated body through the liver. This hypothesis was strengthened by the discovery of *Strongylus rubidus* (sp. nov., 1892) by A. Hassall and C. W. Stiles[1] in the stomachs of swine. This strongyle, the authors state, has

[1] Journ. Comp. Med. and Vet. Archives, April, 1892.

been found in some cases in such immense numbers that the mucus of the stomach seemed to be blood stained, while at other times but few were present. I found that the measurement of the ova within this worm (36 μ by 60 μ) corresponded closely with that of the ovum found in the liver. The latter was a trifle larger, but this may have been due to a slight pressure of the cover glass. The red color of the worm suggested that the worm might possibly draw blood directly from a small vessel, and that in this way opportunity might be given for it to enter a vein, die, and become disintegrated. In testing a few worms for the nature of the coloring matter I was able to obtain some hæmin crystals, thus proving that the coloring matter of the worm was derived from the blood of the host. The accidental entry of these round worms into some branch of the portal system from the stomach, their subsequent disintegration and transportation in the form of minute particles into the liver, where they finally lodge in the intralobular capillaries, appear to be the simplest and most consistent explanation of the lesions described.

The other lesion of the liver in swine which is very prevalent in different parts of the country has been referred to the presence of *Stephanurus dentatus* by several writers who have seen the worm in the diseased spots. It is not necessary, therefore, for me to discuss this disease. I might state, however, that before this discovery of the lard worm in the liver I had become interested in tracing the cause of the peculiar sclerosis resulting in the spots on the liver, but without any success. Many of them were examined, but no parasites found excepting in one case. In sections made with a razor through one of these spots I found round bodies 0.3 mm. in diameter. They were all alike in having a dense, fibrous capsule and in being filled with oval bodies measuring 6.3 μ by 9 μ. The contents of these were homogeneous, clear, and contained a very minute vesicular nucleus. Whether these bodies were cross sections of very young individuals of *Stephanurus dentatus* or of some other organism I am unable to state. Subsequently I found a nearly adult specimen of the lard worm in a large sclerotic focus. The precise relation which this worm bears to these multiple lesions in the liver does not appear to have been worked out. It seems probable that the female may gain access to some branch of the portal vein, die, and become disintegrated, and that the ova set free may continue their development wherever deposited in the liver up to a certain stage, depending on the subsisting conditions. This hypothesis would account for the great variation in the extent of the liver lesions, which range from a faint bluish spot under the capsule to a dense, whitish sclerosis or thickening of the connective tissue framework. It would also account for the absence of any traces of the invading parasite in most foci and the presence of large (adult) worms in a few.

In cattle the writer has noticed two kinds of focal lesions, one represented by small, firm, yellowish foci, the other by large dark spots.

The lesions of the first class might be mistaken for miliary tuberculosis by those not acquainted with their structure. They occur as isolated yellowish dots scattered throughout the entire organ in some cases. In others their number is much smaller. Their diameter ranges from one-third to one-half of a millimeter (one seventy-fifth to one-fiftieth of an inch).

They are made up of a rather dense mass of cells, which is not sharply demarcated from the surrounding normal tissue, but which sends lines or rows of cells situated within the intralobular capillaries

outward into the normal tissue. The cells are mainly those with a round nucleus; polyform leucocytes are not present in recognizable numbers. Toward the center of the cell mass there is much fragmentation of nuclei. These foci differ from tubercles of the same size in the dense cell infiltration and nuclear destruction and in the absence of giant cells. The true bacillar tubercle in cattle, however small, usually contains one or more giant cells, while the bulk of the new tissue in it is made up of cells with pale vesicular nuclei, representing proliferating connective tissue cells. Cell destruction occurs only in the later stages.

It is probable that these lesions are the result of the reaction produced by particles from disintegrated parasites within the portal system. In cattle there is abundant opportunity for such accidents, for we may have encysted parasites in the walls of the small intestine, in the mesenteric glands, and in the submucous membrane of the fourth stomach.

The second class of lesions is entirely different from the first. When the liver of rather old cows is sliced, dark-brownish, slightly depressed spots may be seen on the cut surface. These spots are roundish, from 1 to 1.5 cm. (two-fifths to three-fifths of an inch) in diameter. They may also be seen as dark-bluish or dark-slate colored slightly depressed spots on the surface of the uncut liver. These spots were seen at the abattoirs in Holland by Sluys and Korevaar. They were also noticed by Saake[1] and recently described by Kitt of Munich.[2] The description given by the last-named writer agrees in every respect with my observations, and I have nothing to add to his statements.

In the microscopic examination of sections through these dark-brownish patches it is found that the liver tissue is replaced by wide meshes filled with blood corpuscles. These spaces or lacunæ are distinctly visible to the naked eye when the mounted section is examined in a strong light. Their arrangement appears to be more or less radial around some central axis. They are separated from one another by single rows (or plates) of liver cells and are lined with endothelium, so that each cell plate has endothelium on both sides.

Kitt regards the lacunæ as unduly distended capillaries and considers the condition as a congenital one, produced by some interference in the normal development of the liver tissue. My own observations led me to regard these spots as a result of some parasitic invasion of the liver. After the publication of Kitt's view I again examined these spots in several herds of cattle with the view of testing his theory. I found, however, that, in harmony with my own view, these spots are not present in the liver of young cattle; that the older the cattle the larger the number of these spots. In other words, they are governed to a certain degree by the same laws governing other parasitic lesions—they increase in number with age. As to the nature of the parasite which might cause this peculiar lesion, I have no direct observations. It is, however, probable that *Linguatula tænioides* may be the cause. These parasites were first noticed by Dr. Schroeder[3] in the mesenteric glands of cattle reared in the District of Columbia. Since that time they have been seen at different

[1] Multiples dissem. Leberangiom des Rindes. Archiv. f. wiss. Thierheilkunde (1893), XIX, p. 193.
[2] Monatshefte f. prakt. Thierheilkunde, VI (1894), p. 157.
[3] Bulletin of the Bureau of Animal Industry, No. 7 (1894), p. 126.

seasons of the year. In January (1895) I found the mesenteric glands of a cow from Pennsylvania invaded by *Linguatula* to such an extent that every gland contained twenty or more individuals. In this case they were also seen on cut surfaces of the liver, but their exact location could not be made out owing to their rapid movements. The lesions in the mesenteric glands were such as to suggest the theory that the *Linguatula* found in them is also the cause of the brownish liver spots. They are found in small cavities of these glands embedded in a brownish soft mass which is composed of débris of the gland tissue. No inflammatory reaction appears to accompany these parasites. This may occur later, however, and in the bulletin referred to the writer has made these parasites responsible for the earthy and calcareous foci not uncommon in the mesenteric glands. Inasmuch as the intestinal walls contain similar foci caused by round worms (*Œsophagostoma*), some of these may perhaps find their way into the mesenteric glands to produce similar lesions there. If so, *Linguatula* is probably not responsible for any of these earthy foci, since the lesions produced by the same parasite are found to be fairly uniform in character and on this account distinguishable up to a certain point from the lesions produced by other parasites.

In the production of the peculiar lesions in the liver it would seem that the plugging of a small branch of the portal vein might lead to a reversal of the pressure from the hepatic vein under certain conditions, or at least to a decided disturbance of the equilibrium in a certain number of lobules. In old animals the disappearance of a portion of the liver cells and the enormous widening of certain capillaries might thus be accounted for. The greater soundness of the organs in young animals may prevent any such lesion during a temporary obstruction. These statements concerning the nature of these spots in the liver need confirmation. Should it be found that in those countries (Bavaria and Holland) where they have been found *Linguatula* can not be detected in the mesenteric glands, my theory naturally falls to the ground. The arrested development theory of Kitt can not, according to my observations, account for these spots.

Lesions of the liver in the horse, probably referable to parasites, appear to be not infrequently a stumbling-block in the diagnosis of glanders. These have been rather carefully described and figured by Kitt[1], who discusses their structure and the points of difference between them and nodules due to the glanders bacillus. Only one instance has come to my observation. A large portion of the liver of a horse was sent to the laboratory with the request that an examination be made to determine whether the animal had been affected with glanders or not.

Viewed from the surface the liver was densely speckled with grayish or grayish-yellow dots from one-fiftieth to one-fifth of an inch apart. The smallest dots, one-third of a millimeter (one seventy-fifth of an inch) in diameter, are barely visible as grayish, translucent spherical bodies having a central yellow nucleus. They occur chiefly in small groups or agglomerations. When the liver is incised these bodies are found equally numerous throughout its substance. When the finger is passed over them they impart a distinctly shot-like sensation to it. When crushed between glass slides all those examined were made up

[1] Die kalkig-fibrösen Knötchen der Leber und Lunge des Pferdes und deren Unterscheidung von Rotzknötchen. Monatshefte f. Thierheilkunde, II (1891), p. 433. See also Olt, Archiv. f. wiss. Thierheilkunde, XXI, p. 352.

of a dense outer wall or capsule of fibrous tissue and an inner translucent core in which is embedded a large angular body made up of inorganic salts.

The nature of these bodies is involved in obscurity. Kitt suggests as a possible explanation the transportation of infectious matter from the umbilical vein in case of an inflammation of this vein after birth (omphalo-phlebitis). I am inclined to refer these bodies to the transportation of fragments, possibly ova, of disintegrated parasites from the veins of the stomach or the intestines. The final solution of this problem will be made only when livers of horses are systematically examined without reference to any existing lesions. The cause may then be detected before it has been obscured by the reactive inflammation of the liver itself, which means in most instances the simultaneous absorption and disappearance of the offending body.

The facts presented in the preceding pages may be summarized under the following heads:

(1) The frequent existence of numerous minute focal lesions in the liver of some domestic animals, due probably in all cases to ova or particles of disintegrated parasites carried from the digestive tract through the portal vessels into the liver.

(2) The necessity of carefully examining such conditions so as not to confound them with tuberculosis, glanders, and other affections of importance.

TWO CASES OF CIRRHOSIS OF THE LIVER.

CIRRHOSIS OF THE LIVER IN A STEER.

This case came into my hands in 1893 through the courtesy of Dr. Cooper Curtice. A large herd of mostly young Devon cattle in the central portion of New York State had been tested by him with tuberculin and found extensively infected.[1] One of the animals, a steer 2 years old, was found affected with tuberculosis and a very marked diffuse cirrhosis of the liver. The general appearance of the animal did not suggest the double disease with which it was affected. At the autopsy both retropharyngeal glands were found two to three times larger than normal and completely infiltrated with tuberculous foci which are centrally necrosed. In the left bronchial gland were two foci, each one-half of an inch in diameter, made up of multiple cheesy nodules. In the azygos lobe of the right lung a tuberculous focus one-fourth of an inch in diameter, cheesy. Slight surrounding recent infiltration.

The liver was not much larger than in the normal condition. The entire surface was marked with a dense network of whitish bands varying considerably in thickness. No part of the organ was free. The same dense network was found throughout the parenchyma, involving the connective tissue around single lobules as well as groups. If it is borne in mind that in the normal bovine liver the interlobular connective tissue is so slight in amount as to be invisible to the unaided eye, and that even with the microscope the boundaries of the individual lobules are traced with difficulty, the amount of newly formed connective tissue in the liver of this animal will be better appreciated.

The examination of sections of hardened tissue confirmed the inference deduced from the naked eye appearances. The network of

[1] The extent of tuberculosis among the animals of this herd is indicated on page 141 of Bulletin No. 7 (Bovine tuberculosis).

whitish interlobular bands was made up of dense connective tissue containing but few nuclei, spindle-shaped in form. The density was in fact so great that sections were cut with great difficulty. Embedded in this new stroma were numerous quite narrow branched cellular structures resembling small bile ducts. In a thick section they present the appearance, under a low power, of artificially injected vessels. They are much more abundant in some places than in others.

The proliferation of connective tissue is not limited to the interlobular region, but invades the lobules themselves in circumscribed areas. Examined with a hand lens, the sections show great variation in the size of the lobules, some being very small, almost obliterated. The invasion into the lobules may be traced in several stages from a simple increase in the interlobular connective tissue nuclei with or without a rearrangement of the normal trabecular grouping of the hepatic cells to the complete substitution of dense connective tissue for the parenchyma.

In sections from one portion of liver tissue numerous intralobular cell foci were noticed. These were found associated in a few instances with typical tubercular giant cells.

Any changes in the hepatic cells and cell nuclei similar to those mentioned below were not detected.

CIRRHOSIS OF THE LIVER IN HORSES.

A disease known as "Bottom disease" appears to exist among horses in South Dakota. Some years ago E. C. Schroeder, M. D. V., investigated this disease on the spot, and his report will be found in the eighth and ninth annual reports of the Bureau of Animal Industry, page 371. All that seems to be known of the disease is that it occurs on the alluvial plains lying on either side of the Missouri River and that it has been ascribed to a weed called rattlesnake weed (*Crotalaria sagittalis*).

It is not my intention to discuss this disease, as it needs further study, but to describe certain lesions in the liver of one case which are distinctively cirrhotic in character. Dr. Schroeder brought with him tissues from two animals hardened in Müller's fluid and in alcohol. The liver of but one animal showed advanced cirrhotic changes associated with a certain peculiar modification of the cells and cell nuclei. This animal is stated to be a brown gelding 10 years old. The appearance of the liver is described by reference to a preceding case in which the organ had a mottled yellowish white and red appearance. Even in the alcoholic material brought to me the very marked change from the normal was evident at a glance. There was very little fluid in the serous cavities of this animal.

The histological changes as observed in sections of hardened tissue may for convenience of description be ranged under three heads:

(1) Cirrhosis; i. e., increase of the interlobular tissue.
(2) Hypertrophy of the liver cells and cell nuclei.
(3) Pigment deposits.

The hyperplasia of the interlobular tissue appears to be quite uniform throughout the section. The interlobular space is widened into dense bands between 0.15 and 0.3 mm. in thickness.[1] The newly

[1] In sections from a normal liver the interlobular tissue was recognizable only by the presence of the vessels and the epithelium of the bile ducts. The greatest diameter of the normal interlobular portal branches was but 0.1 mm.

formed connective tissue contains but few nuclei. The bile ducts have proliferated to only a moderate degree. In general the cirrhosis is not so extensive as that in the steer's liver above described.

A general enlargement of the individual liver cells is noticeable throughout the sections examined. It can be estimated only approximately, owing to the irregular outline of the cell itself. Taking the normal cell to be from 15 to 18 μ in diameter, the hypertrophied cells are from 30 to 40 μ in diameter; i. e., fully twice the normal dimensions. This general hypertrophy of the liver cells is associated with a marked enlargement of the nucleus, together with the appearance of peculiar vesicular bodies within the largest nuclei.

The size, appearance, and contents of the nuclei vary from cell to cell. The largest are about 30 μ in diameter, the smaller ones about 12 μ. Measurement of the nuclei of the normal liver in the horse shows them to be 7 to 8 μ in diameter. The enlarged nucleus appears like a vesicle with varied contents. The most frequently appearing intranuclear bodies are vesicular, vary much in size, and bear no definite relation to the size of the nucleus. Besides the vesicles, the nucleus may contain smaller spherules which assume, in sections stained in alum carmine, a uniformly pinkish color. It is probable that these are identical with the larger vesicles. The nucleus itself usually contains an indistinct network probably made up of substances precipitated by the hardening fluids. The nucleolus is occasionally present and frequently contains a small vacuole-like spot centrally situated.

The variations observed in the nuclei are best illustrated by a few descriptions of selected cells:

(1) Usual nucleus of hypertrophied cell, 12 μ in diameter, nucleolus 3 μ.
(2) Nucleus 24 μ in diameter; contains four vesicles from 6 to 10 μ in diameter. The unoccupied space contains feebly stained amorphous matter.
(3) Nucleus 30 μ long, in outline oval; contains a vesicle 14 μ in diameter and three stained spherules (nucleoli?) 4 μ in diameter. The remainder of nucleus is filled with an indistinct network.
(4) Nucleus containing a single vesicle 18 μ in diameter, which most fills it up. Small nucleolar body pressed to one side.
(5) Nucleus 22 μ in diameter, containing a considerable number of quite small vesicles and a large wrinkled one.
(6) Nucleus 30 μ in diameter, containing a vesicle 14 μ in diameter and one somewhat smaller, with a feebly stained body within it. By the side of these is a narrow elongated stained body (nucleolus?).
(7) Nucleus 32 μ in diameter, containing a dense but not very distinct network and three very small nucleolar bodies.

Throughout the parenchyma there is much deposit of yellow pigment. It appears in exceedingly minute particles grouped together, each liver cell being the seat of such a group. It is equally abundant within the epithelial cells of the interlobular bile ducts.

Among the hypertrophied tissue there are found small areas in which the liver cells appear of normal size and stain more deeply. The contrast between these patches and the surrounding tissue is very marked. The fresh appearance of these suggests the idea of regeneration.

Lewin[1] describes two modifications of the cell nuclei of the human liver in various affections which resemble to a certain degree those of this horse's liver. One is a simple vacuolar degeneration. The

[1] Contribution à la pathologie des cellules hepatiques. Archives des Sciences biologiques (1892), p. 353.

nucleus becomes somewhat enlarged and contains a vesicle varying much in size.

The other change consists in a great enlargement of the cell nucleus and the presence within it of one or more flat, round, colorless refrangent bodies, which fail to stain, or at most retain a feeble coloration after treatment with Ehrlich's hematoxylin or acid fuchsin. Cells affected in this way usually appear in groups of five or six. The author is inclined to regard these disk-shaped bodies with the enlarged vesicular nucleus as parasites.

Owing to the fact that but one case was examined by me I refrain from identifying the nuclear changes I have seen with those described by Lewin. There is, however, a certain likeness between them. Thus in fig. 1, on Pl. VIII, the contents of the cell nucleus *d* suggest the small intranuclear bodies regarded by Lewin as parasites.

The liver of the second case, of which pieces were submitted to me, was not affected with sclerosis, but showed entirely different lesions. This case is described by Dr. Schroeder as "a mare between 4 and 5 years old. She had been suffering for a long time and was expected to die almost any moment. In her case the affection of the liver was less extensive than in the other cases, there was less extravasation, and very little fluid in the peritoneal and pleural cavities." The liver of this animal presents in sections a very peculiar appearance. Around the central vein of almost every lobule the parenchyma, for a distance outward equal to one-third to one-half of the lobule, is largely replaced by lacunæ filled with red blood corpuscles. These lacunæ form a network made up of what is left of the tissue cells. Increase of connective tissue is noticed here and there along the course of the interlobular (sublobular) veins, along which the same rarefaction of the parenchyma is noticed. The intralobular stroma is not increased; in fact the capillary system is more or less obliterated by a moving together of the liver cells. These appear larger than in the normal liver, and there is also a well-marked hypertrophy of many nuclei, resulting in a considerable variation in size. Thus in one instance the nuclei of two contiguous cells measured, respectively, 10 μ and 20 μ in diameter. The large nuclei stain solidly, and no such appearances are detected which are described for case 2.

The notes presented by Dr. Schroeder do not mention any obstruction in or incapacity of the pulmonary circulation, which might in this case explain the intralobular distension of the capillaries into large lacunæ. The slight hypertrophy of the liver cells and nuclei may perhaps represent an effort to compensate for the tissue destroyed by vascular distension. Before Dr. Schroeder's investigations in South Dakota, Dr. McCapes, a local veterinarian, kindly sent to this Bureau portions of the liver of a horse which is supposed to have succumbed to the "bottom disease." The liver is described as "gray internally, bronze-gray externally; increased density, very hard and tough; external surface of the right lobe covered with white strips, three-fourths of an inch long, attached solidly to the surface. Size normal. Weight, 17 pounds."

I examined sections of liver tissue from this animal and found the general appearance quite different from that of the livers described above. There was no pronounced sclerosis, although in the intralobular spaces around the vessels there was a deposit which assumed a homogeneous pinkish-blue color in sections stained with hematoxylin, and may have been amyloid. The parenchyma itself was free from this substance but affected with a variety of lesions. The cells were

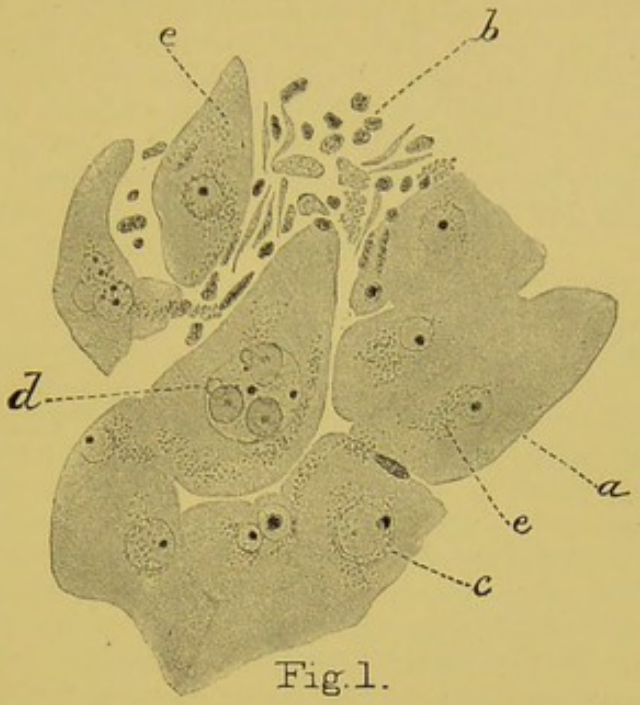

Fig. 1.

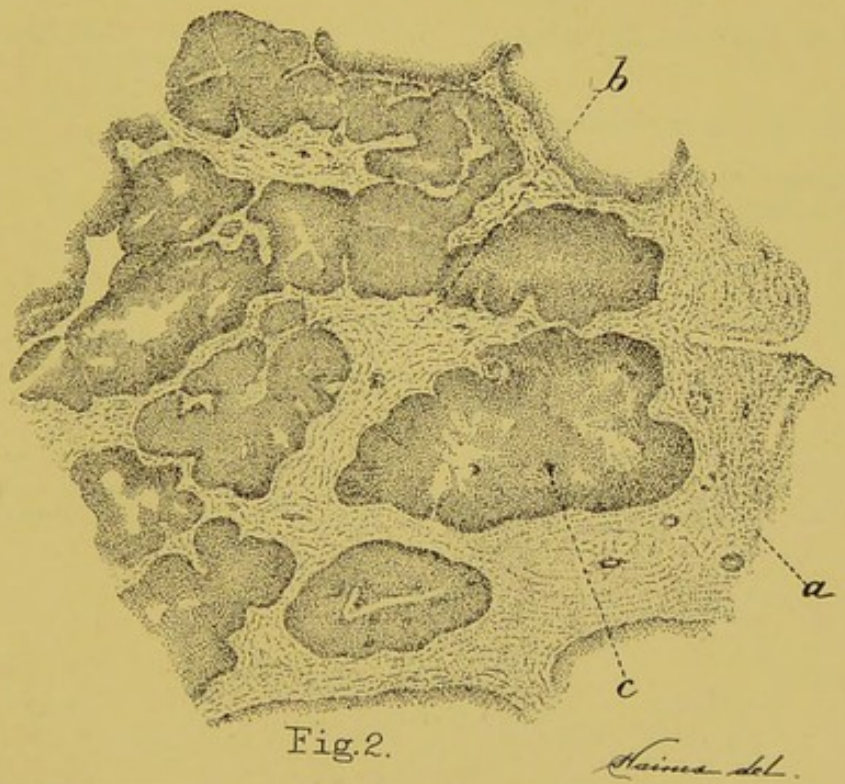

Fig. 2.

Haines del.

CIRRHOSIS OF THE LIVER.

more or less vacuolated from fatty changes, and contained groups of irregular pigment granules. The intralobular channels were considerably widened, and there was some rarefaction of the central zone of the lobule similar to that of the second case described. This case is mentioned simply to point out that the lesions of the liver are not constant and that further studies are necessary to determine precisely what constitute the so-called "bottom disease." Its probable relation to a peculiar cirrhosis of the horse's liver occurring in certain localities of Germany (Schweinberger Krankheit) has been mentioned by Dr. Schroeder, but needs confirmation.

On the other hand, it is probable that in a malady of a progressive character like that which terminates in cirrhosis various stages of the disease are encountered, differing anatomically among themselves. Only a large series of observations will enable us to trace the relationship among such probable stages. These facts are to some extent similar to those obtained by Wyatt Johnston in the study of a disease of cattle in Nova Scotia.[1] This disease is limited to two counties, where it has been known for thirty years. It is not contagious, but destroys all cattle living on certain farms within a space of three years. It also is ascribed to a weed—ragweed; but students of the disease have denied the existence of any such relationship between the weed and the disease. Johnston examined 35 cases and found the liver affected in all but one case. There is at first an increased secretion of bile. The parenchyma of the liver is affected with degenerative and fatty changes. In two or three months cirrhosis appears and the animals finally succumb to abdominal dropsy.

DESCRIPTION OF PLATE VIII.

Fig. 1. Cirrhosis of the horse's liver. (×340.) The section was stained in alum-carmine.
- (*a*) Enlarged hepatic cells.
- (*b*) Interlobular tissue, greatly increased in quantity, extending into the lobule itself.
- (*c*) Enlarged vesicular nucleus containing a large vesicle.
- (*d*) Greatly distended nucleus containing a number of spherical bodies, some feebly tinted.
- (*e*) Groups of granules of reddish-yellow pigment within the cells.

Fig. 2. Cirrhosis of the steer's liver.
- (*a*) Bands of connective tissue, varying in thickness and inclosing small groups of lobules.
- (*b*) Branched tube-like rows of cells (newly formed bile ducts?) indicated by delicate short lines in the drawing.
- (*c*) Tuberculous focus.

[1] Biliary cirrhosis of the liver in cattle (Pictou cattle disease). Proc. Thirtieth Annual Convention of the U. S. Vet. Med. Assoc. (1893), p. 120.

INFECTIOUS LEUKÆMIA IN FOWLS—A BACTERIAL DISEASE FREQUENTLY MISTAKEN FOR FOWL CHOLERA.

By VERANUS A. MOORE, B. S., M. D.,

Chief of Division of Animal Pathology.

It is well known that very destructive and apparently infectious diseases are quite common among poultry. On account of the limited knowledge of the nature of these affections, the outbreaks which they cause are usually attributed by the interested parties to fowl cholera, the diagnosis being based upon the high mortality rather than on the results of careful pathological or bacteriological examinations. In fact, the term fowl cholera has come to be generally employed to designate any serious affection of fowls not distinctly recognized as parasitic or diphtheritic in nature.

In the spring of 1894 and in the fall of 1895 a few outbreaks of a destructive disease among poultry came to my notice. A few of these were reported to be fowl cholera, while others were said by the owners to be unlike that disease. In the first instance this was the only disease which the parties had observed, while in the last the people had lost poultry from a different malady which they had called fowl cholera. The examination of the few fowls obtained showed conclusively that the disease from which they had died was distinct from that described as fowl cholera, although at first it was thought it might be a modified form of that disease. This opinion was entertained in 1895 when a brief description of the first three outbreaks was published,[1] but the subsequent study of the specific organism and the investigation of more recent outbreaks have shown that it is impossible to establish a near relationship between these affections. This necessitates the differentiation of the so-called fowl cholera into at least two distinct diseases. Already Lignières[2] has reported a septicæmia in fowls due to *Bacillus coli communis*. His somewhat meager description of the symptoms and lesions precludes their identification with those in the disease about to be described. The specific organism, however, belonged to another group. A further study of epizootics among poultry may reveal the existence of still other infectious or dietary diseases which are now masked under the ambiguous term of fowl cholera.

Notwithstanding the numerous reports of epizootics among fowls it has been found exceedingly difficult to obtain notice of their existence early in their course. There is a remarkable hesitancy among poultry raisers to report the existence of a disease in their flocks before its seriousness is demonstrated by the death of such a large number of individuals that it is usually too late for an investigation. Repeatedly places at which epizootics were reported have been visited by

[1] Bulletin No. 8, Bureau of Animal Industry, 1895, p. 63.
[2] Compt. Rend. Soc. de Biol., VI (1894), p. 135.

different inspectors of this Bureau, but invariably the conditions found precluded the determination of the nature of the disease, which had already run its course and disappeared. However, more recently certain knowledge of five outbreaks has been obtained. Unfortunately, very few cases from each were examined, and in four instances these were the last fowls to die. The differentiation of the disease causing these outbreaks from fowl cholera is based upon the facts obtained in these examinations and in the study of the disease artificially produced by means of the specific organism isolated from the organs of these fowls.

OUTBREAKS REPORTED AND FOWLS EXAMINED.

I.

April 7, 1894. A dead fowl was received from Mr. S., who resides near the city of Washington, D. C. He had lost during the preceding two weeks nearly all of his fowls (about fifty) from a disease which he supposed to be fowl cholera. He was requested to notify us if the disease continued in his flock and to send us all the fowls that were subsequently attacked. As no other fowls were received, it is presumable that the disease had disappeared and that the fowl examined was the last to be affected.

Fowl No. 1. This fowl was a female about 2 years old. She was in good flesh. The liver, spleen, kidneys, and lungs appeared to be normal. The crop contained a considerable quantity of dirt, kitchen scraps, and other food. In the abdominal cavity there were two large blood clots and a broken egg. The mucosa of the head were normal; the comb pale. Cover-glass preparations from the liver showed no bacteria. Tubes of agar and bouillon were inoculated from the liver and blood. On the following day these contained pure cultures of a nonmotile organism. It differed from the bacterium of fowl cholera, although it was fatal to fowls, pigeons, rabbits, guinea pigs, and mice.

II.

August 8, 1894. A dead fowl was received by express from Mr. S., of Tackett Mills, Va. A postal card from him bearing the date of August 8, addressed to the Department of Agriculture, read in part as follows: "I send you by Adams Express a hen with genuine cholera. I have lost about eighty hens and pullets in the last two months from fowl cholera." The autopsy showed that the fowl had died some hours before its arrival.

Fowl No. 2. Adult female. There was little loss of flesh. The organs were somewhat discolored from post-mortem changes. The liver was fatty, the kidneys yellowish in color, due to an excess of urates in the tubules. Heart muscle pale and sprinkled with grayish points. The contents of the intestines were greenish and semiliquid in consistency. Mucosa of the cloaca had a yellowish color. Cover-glass preparations from the blood and liver contained very few rod-shaped bacteria. Tubes of bouillon and agar were inoculated from the liver. The following day these contained pure cultures of a bacterium which upon subsequent study was found to be identical with the bacterium obtained in the fowl examined from the first outbreak.

III.

In August, 1894, while engaged in other investigations, Dr. Theobald Smith had the opportunity of examining a fowl which had died in a supposed outbreak of fowl cholera on Block Island, Rhode Island. The fowl died August 13. It was kept on ice until August 15, when it was carefully examined. It came from a flock of about seventy

fowls. The disease was reported to have begun in June, and at the time this fowl died only about fourteen or fifteen were still living.

Fowl No. 3. The autopsy notes made by Dr. Smith state that the organs appeared to be normal. Four cultures were made from the liver, spleen, and blood. On the second day these contained a moderately vigorous growth of a nonmotile organism.

August 31. Dr. Smith gave me these cultures for further study. They were found to be identical and pure. A series of inoculations and a careful study of the morphological and physiological properties of this organism showed that it was identical with the bacterium obtained from the two outbreaks previously studied.

IV.

September 18, 1895. Mr. C., who resides in Maryland, about 7 miles from Washington, brought two fowls to this laboratory. One of these was dead upon arrival, the other died late in the afternoon of the same day. Mr. C. made the following statement concerning the disease and the conditions under which it occurred:

The fowls were divided into two flocks. In one of these the disease had caused the death of about fifty old hens. The other flock remained free from its ravages. The conditions under which the two flocks were kept were, so far as he knew, practically the same. He stated that his neighbors had lost a good many fowls from apparently the same trouble. He characterized the disease as being very rapid in its course. It frequently happened that the fowls which appeared to be well at night were found dead the following morning. There was no intestinal trouble such as diarrhea, and Mr. C. was emphatic in his statement that the disease was not fowl cholera, as he had frequently seen fowls affected with that malady.

Fowl No. 4. A female about 2 years old. It was dead upon arrival, and the autopsy showed it to be in good flesh. The abdominal cavity contained considerable fat. The organs were changed in color, due to post-mortem effects. There was some reddening of the mucous membrane of the duodenum, which was explained by the fact that the fowl had been given a large dose of carbolic acid the day before. Tubes of culture media inoculated from the liver remained clear. Pure cultures were obtained from the blood.

Fowl No. 5. A female about 2 years old. Alive, but unable to walk. Mucosa about the head pale. When lying on the floor there were apparent chills. Temperature 109.5° F. Later in the day it seemed to be stronger and walked a little. A blood count showed 2,433,000 red corpuscles and 69,077 white ones per cubic millimeter. She grew weaker and died late in the evening and was put on ice. The autopsy on the following morning showed the body to be well nourished. The mucous membrane of the colon was slightly congested. The kidneys were unusually dark colored, due to blood injection. The other organs appeared to be normal. Heart contained very little blood. Lungs normal. Agar plate cultures were made from the congested mucosa. From these a bacterium very similar to the one found in the previously examined cases was isolated, also a bacillus belonging to the colon group of bacteria. Cover-glass preparations made from the liver and blood did not exhibit bacteria, and culture media inoculated from them remained clear. The viscera of this fowl was fed to two healthy ones with negative results.

It is curious that the specific organism was not found in the organs other than the colon. Again, the organism which resembled the one found in the previous cases, in its morphology and culture characters, was not pathogenic when large doses were inoculated into fowls and rabbits. The absence of diarrhea, the great exhaustion, and the diminution in the number of red corpuscles as observed in fowl No. 5 are conditions identical with those observed in fowls in which the disease was produced with the virus obtained from other outbreaks. Here, as in previous outbreaks, the fowls examined were among the last to die.

V.

December 10, 1895. Mrs. B., residing near Overlook Inn, near Washington, reported the loss of several fowls from a disease which she said was not cholera. The fowls usually died suddenly, but occasionally they were sick for a few days. It was the exception for them to have a diarrhea. She brought with her for examination a fowl that was found dead that morning. On the following day Dr. C. F. Dawson, of this laboratory, visited the infected locality. He reported that the poultry were kept under very unfavorable conditions respecting cleanliness and food. Several fowls had died during the preceding night, but he was unable to detect the disease in any of the living ones. He found that several other farmers in the neighborhood had lost fowls from apparently the same trouble. The dead fowl brought to the laboratory by the owner, and those obtained by Dr. Dawson, were carefully examined.

Fowl No. 6. Fowl brought to the laboratory by Mrs. B. December 10. It was reported to have been sick for several days. Mucosa about the head pale. Emaciated. Liver very much enlarged and mottled with grayish areas 0.5 to 2 mm. in diameter. The serous surface of the intestines appeared to be normal, but the mucosa of the duodenum and colon were hyperæmic. Kidneys pale, lungs normal. Heart muscle pale and the heart cavities contained very little blood. Cover-glass preparations of the blood showed many leucocytes. Cover-glass preparations from the liver, blood, and kidneys revealed the presence of very few elongated bacteria. Pure cultures of a short bacterium with oval ends were obtained from the blood and liver.

Fowl No. 7. Found dead December 11, kept on ice until December 12, when it was examined. This fowl was supposed to have been well until it was found dead. Body well nourished. Comb normal in color. Liver slightly enlarged, reddened, mottled with grayish or yellow areas. Intestines appeared to be perfectly normal. No evidence of diarrhea. Lungs normal. Heart pale and contained very little blood. In the blood there were many leucocytes, especially those containing spindle-shaped bodies. Many of the red corpuscles were broken and did not take the eosin stain. Cover-glass preparations from the liver, and blood exhibited a considerable number of bacteria singly, in pairs, and in clumps. They usually presented a light center with a deeply stained periphery. Pure cultures were obtained from the liver and heart blood.

Fowl No. 8. This fowl was found dead December 11, examined December 12. Body well nourished. Liver swollen, reddened, and mottled with punctiform hemorrhages. It contained very little blood. Condition of the blood the same as in No. 7. Intestines normal in appearance. Spleen abnormally dark. Kidneys swollen. Pure cultures of the bacterium were obtained from the liver.

Fowl No. 9. A large male. The body well nourished. There was no evidence of intestinal trouble. Liver was slightly swollen. Heart muscle much reddened, but sprinkled with grayish points. Blood pale and contained many leucocytes. Spleen dark colored. Intestines, kidneys, and lungs appeared to be perfectly normal. Pure cultures of the bacterium were obtained from the heart blood, and liver.

Later in the month four other fowls were brought to the laboratory from the same locality. Unfortunately, they had been frozen for about three days before they arrived. From one of these pure cultures of the bacterium were obtained. The absence of the organism, as determined by the inoculation of culture media, in the other three is not explained unless the continuous low temperature had killed it.

THE SPECIFIC MICROORGANISM.

Bacterium sanguinarium sp. nov.

This organism was partially described in 1895 in connection with a brief report of three outbreaks of the disease from which it was

obtained. It was then thought that upon further investigation it would be found to be closely related to, if not identical with, the bacterium of fowl cholera or possibly *Bacillus gallinarium* of Klein. The investigations of the last year have shown, however, that it belongs to an entirely different species. The organisms obtained from the different fowls, as stated in the post-mortem notes, were with one exception (No. 5) identical save slight differences in their virulence. In the exception the pathogenesis had disappeared. This description, written after a careful study more than a year later, differs in no essential point from that recorded in 1895 with the exception of the addition of several determinations of considerable importance. The genus *Bacterium* is in accordance with Migula's[1] classification of bacteria, and it is based on the absence of motility. As the most conspicuous lesion in the fowls affected with the disease in question was in the blood, the specific name *sanguinarium* was suggested.

CULTURE CHARACTERS.

Morphology.—A nonmotile, rod-shaped organism, varying somewhat in size according to the medium in which it has developed. In tissues of fowls or rabbits it is from 1.2 to 1.8 μ long and from 1 to 1.3 μ broad. The ends are tapering in cultures or rounded, at times in the short forms it could easily be mistaken for a micrococcus. In tissue it frequently appears in small clumps, but usually in pairs united end to end. (Plate IX.) Spores or vacuoles have not been discovered. Involution forms are common. In cultures on agar it is more slender than in tissues. When examined in a hanging drop preparation, especially at the edge, it frequently shows a marked polar arrangement of the cellular protoplasm. In these preparations there is observed a marked dancing motion of the organism. In old bouillon cultures short chains composed of this organism united end to end are sometimes observed. It stains with the aniline dyes ordinarily used, but retains the coloring matter very feebly, or not at all, when treated after the Gram method.

Agar.—On this medium, at 37° C., the growth is moderately vigorous. It has a grayish glistening appearance. Isolated colonies are from 1 to 2 mm. in diameter, convex, with sharply defined borders. Agar plates emit a peculiar penetrating odor, which differs decidedly from the pungent odor given off by *Bacterium septicæmia hemorrhagica.* The growth on this medium is not differential or characteristic, but resembles very closely that of the hog-cholera bacillus.

Gelatin.—In this medium the growth is less vigorous. In stick cultures it is more abundant along the line of inoculation than on the surface. Isolated colonies are about 0.25 mm. in diameter, appearing to the unaided eye as homogeneous bodies, but slightly granular under low magnification. On the surface of the gelatin the colonies are granular and slightly spreading. They are not characterized by any distinctive markings. It grows very feebly in acid gelatin. The most vigorous growth requires an alkaline medium. The color of the colonies on the surface of the gelatin is neutral gray. There is no liquefaction or softening of the medium.

Potato.—On the surface of potatoes a delicate grayish-yellow growth appears after forty-eight hours when kept at a temperature of 35° C. Frequently there is no development, owing, presumably, to the acids in the potato.

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[1] Die natürlichen Pflanzenfamilien, 129 Lieferung, 1896.

Bouillon.—In alkaline peptone bouillon at 36° C. the growth imparts a uniform cloudiness to the liquid within twenty-four hours. If the bouillon contains much sugar the reaction becomes acid, otherwise it remains alkaline. A grayish, friable sediment forms in the bottom of the tube. After several days' standing the growth settles, leaving a clear supernatant fluid. In a simple peptone solution containing one-half of 1 per cent sodium chloride the growth is less vigorous than in the one containing the meat juice. In meat extract bouillon the growth is likewise feeble. In acid[1] peptone bouillon there is a very faint cloudiness imparted to the liquid.

Milk.—This medium remains apparently unaffected for about four weeks. It then begins to change to a clear opalescent appearing fluid. In about six weeks it becomes translucent and strongly alkaline in reaction. The addition of a few drops of acetic acid precipitates the casein. A microscopic examination shows that the fat globules are destroyed. This condition is presumably due to the alkali produced by the growth of the bacteria. This appearance of the milk is not distinguishable from that produced by the bacilli of hog cholera and typhoid fever.

Fermentation of sugar.—Alkaline bouillon containing 1 per cent of dextrose in the fermentation tube becomes cloudy within twenty-four hours after inoculation. Strongly acid in reaction. Similar tubes of bouillon containing saccharose and lactose become clouded throughout, but they remain alkaline in reaction. The degree of alkalinity increases with age. Gas is not produced during the growth of the bacillus in bouillon containing sugars.

Indol and phenol.—Cultures in a peptone solution give a strong indol reaction when treated after Kitasato's method.[2] The phenol reaction could not be obtained when cultures were treated according to Weyl-Lewandowski,[3] but the indol reaction was constant.

Temperature requirements.—The range of temperature in which this bacterium will multiply in suitable media is exceedingly wide. The degree of cloudiness was practically the same in tubes of bouillon twenty-four hours after inoculation, allowed to stand at the room temperature and in incubators kept at 36° and 41° C., respectively. Tubes placed in a cool box (about 13° C.) were more faintly clouded. After forty-eight hours there was quite a vigorous growth in the bouillon kept at this low temperature.

Thermal death point.—This has been only approximately determined. Tubes of bouillon inoculated with several drops of a fresh bouillon culture and exposed in a water bath to a temperature of 58° C. for fifteen minutes or longer did not develop. Those removed in

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[1] Bouillon prepared from beef without neutralizing or rendering alkaline by a solution of soda or potash.

[2] This consists in cultivating the bacillus at 37° C. in a sterilized solution composed of 1 per cent peptone and 0.5 per cent sodium chloride in distilled water for from one to three days. To these cultures 1 c.c. of a 0.02 per cent solution of potassium nitrite, freshly prepared, and about 3 drops of chemically pure sulphuric acid are added. A violet pink color indicates the presence of indol.

[3] This consists in distilling the culture liquid, say 250 c.c. of ordinary peptone bouillon in which the bacteria have grown for ten days, with 50 c.c. of strong hydrochloric acid, and testing the first portions of the distillate for indol and phenol separately. For indol 2 c.c. of a 25 per cent solution of sulphuric acid is added to 5 c.c. of the distillate, and then 2 or 3 drops of a 0.1 per cent solution of sodium nitrate. A red coloration indicates the presence of indol. A fine crystalline precipitate, forming immediately or soon after the addition of the bromine water to the distillate, indicates the presence of phenol. (Deutsche med. Wochenschrift, 1890, S. 1186.)

from ten to fourteen minutes developed into pure cultures of the inoculated organism. An exposure at 54° C. for thirty minutes produced no deleterious effect, but tubes exposed for two hours remained clear. The growth was not retarded after an exposure of three hours at 50° C.

Effect of low temperature.—Bacteria dried on coverglasses were placed in sterile test tubes and exposed to a temperature of from zero to a few degrees above for seventeen hours. This exposure did not appreciably retard their subsequent multiplication in bouillon.

Effect of disinfectants.—A 1 per cent solution of carbolic acid was fatal in five minutes. A one-fourth of 1 per cent solution of commercial sulphuric acid destroyed life in ten minutes. Lime water was fatal in ten minutes, and a solution composed of 1 part lime water and 3 parts distilled water was equally as effective in one hour. Sulphur fumes were also fatal in a test applied by Dr. C. F. Dawson. This consisted in putting a drop of bouillon culture upon a sterile coverglass inside a large glass jar which had been especially prepared for the test. About one-half of a teaspoonful of sulphur was placed on a sheet of asbestus and ignited. In a short time the jar was filled with white fumes. In three hours' time the cover-glasses were carefully removed and dropped into tubes of sterile bouillon, which were placed in an incubator. They invariably remained clear.

The effect of drying.—This organism retains its vitality when dried on cover-glasses for from seven to fifteen days, according to the thickness of the residue left on the covers. This depends, of course, upon the quantity of the culture taken.

PATHOGENESIS.

This organism is fatal to fowls, pigeons, rabbits, guinea pigs, and mice. Other animals have not been tested. Excepting in intravenous injections, comparatively large quantities of a pure culture were required to produce fatal results. The effects of inoculations on the different species are appended.

Fowls.—Fowls inoculated in the wing vein with 0.3 c. c. of a fresh bouillon culture died in from three to thirteen days; usually on the fifth or sixth day. The temperature begins to rise on the second day after inoculation. It reaches 109° to 111° F. a few days before death occurs. In cases where the fowls live from five to six days they appear perfectly well for at least three days, when the feathers begin to have a slightly ruffled appearance. The exposed portion of the head ordinarily, but not invariably, becomes pale. Toward the last they sit in a crouched position, with the head drawn close to the body. They refuse food, but usually take more or less water. There is sometimes a slight diarrhea, but as a rule there are no observable intestinal disturbances.

The changes observed at post-mortem were not conspicuous. The liver was moderately enlarged, soft, and fatty. The spleen was rarely enlarged. The kidneys were marked with yellow lines, due to the injection of the tubules with urates. The intestinal mucosa was sometimes, although rarely, hyperæmic and sprinkled with punctiform hemorrhages, especially in the colon. The contents of the intestines were, as a rule, greenish in color and normal in consistency. The mucosa of the cloaca was frequently of a yellowish color, due to the deposition of urates. The heart muscle was pale and in most cases sprinkled with grayish points. In a few cases, where the fowls resist for an unusually long period, tubercle-like nodules were observed in the heart, and in one instance in the lungs.

The organs and blood of fowls dying from the inoculation disease contained comparatively few bacteria. This was shown in the cover-glass preparations and also in cultures. Frequently the inclined surface of agar rubbed with a large piece of the liver or spleen or a clot of blood contained on the following day isolated colonies. There were apparently more bacteria in the organs of the fowls which died from feeding upon cultures than in those inoculated intravenously.

A series of fowls inoculated intravenously with pure cultures.

Fowl No.	Date of inoculation.	Virus used.	Date of death.	Remarks.
	1894.			
18....	May 18	0.5 c. c. bouillon culture.	May 21, 3 days...	Liver fatty, engorged with blood; intestines pale.
19....	May 22	0.3 c. c. culture, fowl 18..	May 28, 6 days...	Liver fatty, engorged with blood; intestines normal.
20....	May 31	0.3 c. c. culture, fowl 19..	June 11, 11 days..	Very much emaciated; grayish nodules in heart; lungs contained grayish nodules, and acini filled with cells.
26....	June 12	0.3 c. c. culture, fowl 20..	June 18, 6 days...	Liver fatty.
27....	June 19	0.3 c. c. culture, fowl 26..	July 2, 13 days...	Very much emaciated; organs pale.
29....	July 3	0.3 c. c. culture, fowl 27..	July 9, 6 days....	Liver fatty; kidneys yellowish; intestines normal.
30....	July 10	0.3 c. c. culture, fowl 29..	July 14, 4 days...	Liver engorged with blood; blood thin; intestines normal.
31....	July 18	0.3 c. c. culture, fowl 30..	July 24, 6 days...	Do.
32....	July 25	0.3 c. c. culture, fowl 31..	July 30, 5 days...	Do.
33....	Aug. 2	0.3 c. c. culture, fowl 32..	Aug. 7, 5 days....	Liver engorged with blood; kidneys yellow; intestines normal.
34[1]...	Aug. 8	0.3 c. c. culture, fowl 33..	Aug. 18, 10 days..	Do.

[1] Intestines contained a large number of tapeworms.

In every case there was an elevation of temperature, a diminution in the number of red blood corpuscles and an increase in the number of white ones. In two instances (Nos. 20 and 27) there were important variations from the usual lesions.

In No. 20 the lesions were exceedingly interesting, for from the appearance of the heart and lungs it could readily have been mistaken for a case of tuberculosis. The disease in this fowl seemed to run the usual course for the first few days. On the third day after inoculation the temperature was 109.2° F., and it appeared to be very sick. On the following day the temperature was lower, and on the sixth day it was nearly if not quite normal. Death occurred very suddenly on the eleventh day, and the autopsy showed a most remarkable variation from the usual type of lesions. This is the only fowl in which the lungs were appreciably affected. The grayish nodules in the heart were well pronounced, being much exaggerated over the foci, ordinarily detected as grayish points on the surface of the heart muscle. (Pl. XII, fig. 1.)

In No. 27 the course of the disease was longer, and it manifested itself in excessive emaciation. These are two interesting cases, illustrating the variability of the disease, due, presumably, to the resistance or partial immunity of the fowls.

Several experiments were made to produce an outbreak of the disease by feeding pure cultures of the organism and viscera of affected fowls. In nearly every case a few of the fowls thus exposed died of the disease, but in every experiment where more than two fowls were fed upon the infected viscera or pure cultures one or more remained well. Healthy fowls placed in cages with the diseased ones did not in

a single instance contract the disease. The details of a single feeding experiment are appended:

March 13, 1896. Six healthy adult fowls (Nos. 504, 505, 506, 507, 508, and 509) were fed 400 c. c. of pure culture at the experiment station of the Bureau by Dr. E. C. Schroeder. The culture was mixed with the food, which was given after a fast of twenty-four hours. These fowls remained well for several days, when three of them (Nos. 504, 505, and 508) appeared to be sick, and on the morning of March 25 they were found dead. The others continued in a healthy condition. The autopsies of the dead fowls showed a slight variation in the lesions. In No. 504 the mucous membrane of the intestines was reddened. In the other two it was pale. In all three cases the blood was thin and contained an abnormal number of leucocytes. The blood spaces in the liver were engorged. Cover-glass preparations exhibited the specific organism, and pure cultures of the bacterium were obtained from each of the livers.

In but one instance did the disease continue through three consecutive feedings. Fowls fed with pure cultures or with the viscera of the birds dying of the disease usually perished in from seven to fifteen days. These inoculation and feeding experiments demonstrated a marked difference in the susceptibility of fowls to this organism. Several have survived the feeding of large quantities (300 to 500 c. c.) of pure bouillon culture. The subcutaneous injection of pure cultures gave uncertain results; in a few fowls fatal results were obtained, but usually they remained well after this treatment.

Pigeons.—Two pigeons were inoculated with 0.2 c. c. of a bouillon culture, one subcutaneously and the other in the wing vein. The pigeon inoculated in the wing vein died in four days, the other in eight days. The intravenous injection was repeated on a third pigeon with 0.1 c. c. of pure culture. The pigeon died on the sixth day. The lesions were not marked in either case.

Rabbits.—In rabbits the disease resembles very closely that produced by attenuated hog-cholera bacteria. Subcutaneous inoculations with 0.1 c. c. were rarely fatal; 0.2 c. c. destroyed rabbits in from three to five days. The lesions were characterized by slight local reactions, necrotic areas in the liver, enlarged and discolored spleen, and infiltration of cells into the follicles of the cæcum and the glands about the ileo-cæcal valve. (Plate XIV.) A larger quantity (0.3 c. c. to 0.8 c. c.) usually proved fatal in from five to eight days. An intravenous inoculation of 0.2 to 0.3 c. c. of a fresh bouillon culture produced death in from two to three days. In these cases the spleen was engorged with blood, the liver swollen, and the intestinal mucosa contained hemorrhagic areas. A larger dose produced death in from eighteen to twenty-four hours. An intravenous injection of a small quantity of culture produced lesions similar to those following the subcutaneous inoculation of large quantities. Cover-glass preparations made from the liver, spleen, and blood soon after death showed a few elongated bacteria. If a rabbit was allowed to lie for twenty-four hours after death, cover-glass preparations showed innumerable oval bacteria, some of which occasionally exhibited the polar stain.

Guinea pigs.—Guinea pigs inoculated in the abdominal cavity with from 0.2 to 0.3 c. c. of a bouillon culture died in from five to eight days. The autopsy showed usually a considerable quantity of a clear lemon-colored serum in the peritoneal cavity. The viscera were covered with a grayish membranous exudate. The pleural cavity contained a large

quantity of serum. Lungs collapsed. Subcutaneous inoculations of 0.5 c. c. proved fatal in from six to eight days. There was a purulent infiltration into the subcutis about the point of inoculation. Spleen enlarged, dark colored, and friable. Liver pale and usually fatty. No intestinal lesions. Comparatively few bacteria in the organs as shown in the cover-glass preparations.

Mice.—White and gray mice inoculated subcutaneously with 1 to 2 drops of bouillon culture died in from three to five days. The only pronounced macroscopic lesion observed was an enlarged and discolored spleen. Mice inoculated similarly with a much smaller quantity (a single small loop of culture) remained well.

CONSTANCY OF VIRULENCE.

The degree of virulence possessed by this organism when isolated from the different fowls seemed to be too low to account for the apparent rapidity of the disease. As the fowls from which it was obtained were among the last to die in their respective outbreaks, it was supposed that this fact would explain the attenuated condition of the organism. It was of importance, however, to determine whether this apparent attenuation was real or rapidly induced by undetermined conditions. Experiments in this laboratory have shown that certain pathogenic bacteria isolated from animals which die in outbreaks are not readily increased in virulence, but when attenuation was produced by a prolonged course of the disease, due to resistance on the part of the animal, the original virulence could be restored by the passage of the organism through a short series of animals. Selander[1] and Metchnikof[2] have reported experiments in which the virulence of the bacillus of hog cholera (swine plague), as isolated from outbreaks, was accelerated by passing it through a series of rabbits and pigeons.

After trying, without success, the effect of both slow and rapid cultivations and other methods which have been suggested for handling cultures for the purpose of accelerating their virulence, two series of experiments were made on animals for this purpose. The first was the inoculation of a series of rabbits intravenously and the other the feeding of fowls in a continuous series with the viscera of fowls dead from the disease. These were not continued to a great length, but sufficiently so to show that whatever changes were wrought upon the virulence of the organism must, if any, be very slow. The results of these experiments are given in the appended tables. The virus used was obtained from fowl No. 8, outbreak V.

Inoculation of rabbits.

Rabbit No.	Date of inoculation.	Virus used.	Method of inoculation.	Date of death.	Time after inoculation.
	1896.				
62	Feb. 14	0.4 c. c. bouillon culture	Intravenously	Feb. 17	3 days.
81	Feb. 18	0.2 c. c. culture, rabbit No. 62	do	Feb. 21	Do.
80	Feb. 24	0.2 c. c. culture, rabbit No. 81	do	Feb. 28	4 days.
60	Feb. 29	0.2 c. c. culture, rabbit No. 80	do	Mar. 3	Do.
85	Mar. 4	0.2 c. c. culture, rabbit No. 60	do	Mar. 8	Do.
86	Mar. 13	0.4 c. c. culture, rabbit No. 85	Subcutaneously	(a)	

a Remained well.

[1] Annales de l'Institut Pasteur, IV (1890), p. 543.
[2] Ibid., VI (1892), p. 289.

Feeding fowls.

Fowl No.	Virus fed.	Date of feeding.	Date of death.	Time after feeding.	Remarks.
500	Viscera and culture, fowl No. 506 *a*..	Mar. 26	Apr. 3	8 days..	Fowl 1 to 2 years old.
501	.....do.....	....do...	Apr. 4	9 days..	
507	Viscera and culture, fowls Nos. 500 and 501.	Apr. 4	Apr. 1	....do...	Do.
508	.....do.....	....do...	....do...	....do...	
511	Viscera and culture, fowls Nos. 507 and 508.	Apr. 15	Apr. 21	6 days..	Do.
512	.....do.....	....do...	Apr. 25	10 days.	
513	Viscera and culture, fowls Nos. 511 and 512.	Apr. 22	(*b*)	.....	
514	.....do.....	....do...	(*b*)	.....	Fowls about one-third grown.

a Fowl No. 506 died from the effect of feeding upon bouillon cultures. *b* Remained well.

It is evident from the outcome of these experiments that the virulence of this organism is not rapidly increased by these methods. In the latter case two fowls were taken for each generation of virus; this was necessary on account of the uncertainty of obtaining fatal results, as indicated in previous cases treated in a similar manner where a considerable number of the fowls were not affected, while others died at irregular intervals. The virus employed was from the same source (fowl No. 8). These experiments suggest that the organisms originally isolated were not temporarily attenuated through the influence of undetermined agencies. This conclusion is reached from analogy with the bacillus of hog cholera and the bacterium of swine plague, which I have found to retain, after passing through a long series of rabbits and pigeons, about the same degree of virulence they possessed when isolated. In a case with the swine-plague bacterium,[1] a slight attenuation, caused presumably by the prolonged course of the disease, was entirely overcome by passing it through a short series of rabbits.

DESCRIPTION OF THE DISEASE BASED UPON THE NATURAL AND ARTIFICIALLY PRODUCED CASES.

SYMPTOMATOLOGY.

From the statement of the owners of the fowls in the different outbreaks and from the appearance of those in which the disease was artificially produced, little can be positively recorded concerning the distinctive or characteristic symptoms. The only fowl examined antemortem from the natural outbreaks was first seen only a few hours before death, when it was unable to stand. If held in an upright position, the head hung down. There was a marked anæmic condition of the mucosa of the head. It had an elevation of nearly 3 degrees of temperature. An examination of the blood showed a marked diminution in the number of red corpuscles and an increase in the number of white ones. In the disease produced artificially by feeding cultures of the specific organism there was in most cases a marked drowsiness and general debility manifested from one to four days before death occurred. The period during which the prostration was complete varied from a few hours to two days. The mucous membranes and skin about the head became pale. There was an elevation of from 1

[1] Bulletin No. 6, Bureau of Animal Industry, U. S. Department of Agriculture, 1894, p. 102.

to 4 degrees of temperature. The fever was of a continuous type, as shown in the following temperature chart[1] of two fowls in which the disease was produced artificially:

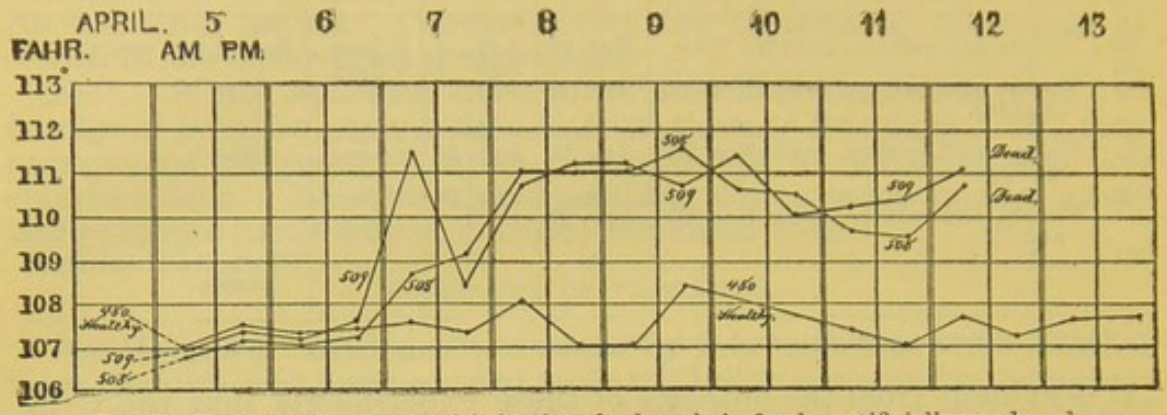


FIG. 7.—Temperature chart of infectious leukæmia in fowls, artificially produced.

Although the course of the disease in the different fowls was usually constant, there were many variations. In a few individuals the time required for fatal results was from two to three weeks, but ordinarily death occurred in about eight days after feeding the virus, the rise in temperature being detected about the third day and external symptoms about the fifth or sixth, occasionally not until a few hours before death. The symptoms observed in the cases produced by feeding correspond with those described by the owners of affected flocks.

As indicated in the inoculation experiments, the symptoms following the intravenous injection of the virus were, as would be expected, considerably modified from those in fowls which contracted the disease by the ingestion of cultures of the specific bacterium.

MORBID ANATOMY.

The only constant lesions found in the fowls which contracted the disease naturally, as well as in those fed upon its virus, were in the liver and blood. The liver was somewhat enlarged and dark colored, excepting in a few cases in which the disease was produced by intravenous injections. A close inspection showed the surface to be sprinkled with minute grayish areas. The microscopic examination showed the blood spaces to be distended. The hepatic cells were frequently changed, so that they stained very feebly, and not infrequently the cells were isolated and their outlines indistinct (Plate XI). Occasionally foci were observed in which the liver cells appeared to be dead and the intervening spaces infiltrated with round cells. The changes in the hepatic tissue are presumably secondary to the engorgement of the organ with blood.

The rareness with which the intestinal tract was affected in both the natural and artificially produced cases is exceedingly interesting from both the pathological and differential standpoints. There was in most cases a hyperæmia of the mucous membrane of the colon, but this condition is not uncommon in the healthy individual. The kidneys were generally pale, but streaked with reddish lines, due to the injection of blood vessels. In section the tubular epithelium appeared to be normal. The kidneys seemed, from the number of bacteria in the cover-glass preparations, to be especially favorable for the locali-

[1] Fowls Nos. 508 and 509 died from ingesting the virus of this disease. No. 480 was supposed to be healthy.

zation of the specific organism. The spleen was rarely discolored or engorged with blood. The lymphatic glands were not appreciably enlarged in any individual examined. The lungs were, with one exception, normal. The heart muscle was usually pale and sprinkled with grayish points, due to cell infiltration and the presence of bacteria (Plate XII). These lesions were so common that it seems safe to consider them one of the characteristic manifestations. Death usually occurred in systole, the auricles containing very little thin, unclotted blood. The brain and spinal cord were examined in several cases without the discovery of abnormal conditions.

As already stated, it is in the blood that the most important alterations are found. These consist in the gradual disappearance of the red corpuscles, as determined by blood counts made daily or every other day, from the time of inoculation or feeding the virus until the day of death. In Bulletin No. 8 mention was made of the blood changes in this disease when produced artificially by intravenous injections. The observations here recorded were made on fowls which contracted the disease by ingesting the specific organism as well as by inoculation, so that these alterations can not be attributed to changes brought about by chemical or mechanical agencies due to the injections being made directly into the circulation. In the fowl examined which contracted the disease from natural sources the blood changes were exceedingly well marked.

The diminution in the number of red corpuscles and the increase in the number of white ones[1] is illustrated in the two appended cases of artificially produced disease.

Fowl No. 82, inoculated in the wing vein February 6, 1895.

Date.	Temperature (° F.).	Number of red corpuscles.	Number of white corpuscles.	Remarks.
Feb. 6	107.4	3,744,444	21,222	Well.
7	109	3,417,391	26,087	Apparently well.
8	108.2	2,784,700	55,000	Do.
9	108.4	2,807,692	76,925	Do.
11	107.4	3,481,818	90,909	Feathers ruffled; refuses food.
13	110.2	2,133,333	100,000	Very quiet; comb pale.
14	108	2,530,000	140,000	Fowl died later in the day.

Fowl No. 501, fed culture March 26, 1896.

Date.	Temperature (° F.).	Number of red corpuscles.	Number of white corpuscles.	Remarks.
Mar. 26	106.2	3,534,000	18,940	Well.
28	110	2,430,000	70,000	Fowl eats very little.
Apr. 2	110.6	1,684,210	80,000	Blood very pale; fowl weak; refuses food.
3	106	1,745,000	245,432	Very weak; many red corpuscles attacked by leucocytes.
4	----------	----------	----------	Found dead.

[1] This condition of the blood suggested the name. The term leukæmia was applied by Virchow (Die Cellularpathologie, Berlin, 1859) to a disease characterized by a more or less notable and abiding increase of white corpuscles, accompanied by a decrease in the number of the red corpuscles. In this malady in the human species it is said that the number of white corpuscles may equal or exceed the number of red ones. As this disease seems to be dependent upon the presence of a bacterium, the name *infectious leukæmia* is proposed. It is not improbable that blood changes similar to those herein described may be found in other bacterial diseases of poultry. This is suggested by statements made by Dr. Salmon concerning the appearance of the blood in fowl cholera.

Additional cases showing the relative number of red and white blood corpuscles.

Fowl No.	Method of inoculation.	Time after inoculation.	Number of red blood corpuscles.	Number of white blood corpuscles.	Temperature of fowl (° F.).	Remarks.
80	Intravenously	Second day	2,059,000	11,636	108.7	
		Fifth day	1,384,000	56,000	109	Sick.
		Sixth day	1,546,153	115,384	109.5	Sick; refuses food; died during night.
83	do	Same day	3,786,341	14,474	106.5	Well.
		Third day	2,275,000	83,333	109.7	No symptoms.
		Sixth day	1,720,000	150,000	110.2	Sick; died on the following day.
500	Fed cultures	First day	4,560,000	26,666	106.1	Well.
		Fourth day	2,905,835	94,116	108.2	Quiet.
		Tenth day	3,610,000	42,000	107	Recovered.
507	do	Sixth day	1,758,333	132,333	111.6	
		Eighth day	1,838,461	138,000	110.4	Died during night.

In carefully heated cover-glass preparations of healthy fowl blood stained with methylene blue and eosin the nuclei are colored a deep blue, and the cellular protoplasm surrounding the nucleus is stained with the eosin. In similar preparations made from the blood of the affected fowls there are a greater or less number of corpuscles which do not take the eosin stain. In these the portion of the corpuscle surrounding the nucleus remains unstained or becomes slightly tinted with blue. It occasionally contains one or more vacuoles, and the margin is frequently broken. In some instances a considerable portion of the corpuscle has disappeared, leaving a few free nuclei. The apparently dissolving away of the red corpuscles has been frequently observed, and corpuscles showing the intermediate stages are readily detected in carefully prepared specimens. (Pl. X, fig. 4.)

The cause of the destruction of the red corpuscles is not yet satisfactorily explained. In his report on fowl cholera, Dr. Salmon[1] illustrates leucocytes surrounding the red corpuscles, but the marked diminution of the red cells was not determined, although he speaks of the pale color of the blood. In fresh preparations of the blood I have observed similar phenomena and have also seen portions of red cells within the granular leucocytes. The determination of the extent of this mode of destruction of the red corpuscles, however, necessitates further investigation.

In fresh preparations of the blood of affected fowls treated with Toison's fluid red corpuscles which took the violet stain throughout more or less intensely were frequently observed.

In the blood of poultry there are at least two distinct classes of white corpuscles. The first, which predominates in numbers, contains from one to four nuclei, and the cytoplasm is sprinkled with a variable number of round, elongated, or spindle-shaped bodies. In the fresh condition they are highly refractory. They stain with eosin, and if the preparations are heated sufficiently they will retain certain of the aniline dyes. The other class consists of round or nearly round cells which take the blue stain feebly. Usually it is difficult to detect the nucleus, although it is occasionally distinct. The leucocytes containing the spindle-shaped bodies appear to be the phagocytes, as they are the only ones I have observed attacking the red corpuscles. Bacteria have not been demonstrated in these cells, although their presence has, in several cases, been suspected. From the appearances observed in the red blood corpuscles it seems highly probable that phagocytosis plays a

[1] Reports of the Department of Agriculture, 1880–1882.

comparatively large part in their destruction. It is difficult to believe without further evidence that a toxin produced during the multiplication of the specific organism could have such an effect on the red corpuscles. The large number of blood counts which have been made shows beyond question that active and destructive changes are taking place in the blood. In the fresh preparations we can observe the phagocytes attacking the red cells and in the stained ones mutilated red corpuscles and free nuclei are present. The hypothesis is suggested that the leucocytes partially digest certain of the red corpuscles in their attack upon them. Whether these changes are entirely attributable to the phagocytes is still an open question.

It is an interesting and as yet unexplained fact that the increase in the white corpuscles is apparently restricted to those containing the spindle-shaped bodies. In the blood from healthy fowls it is comparatively rare to see one of the white corpuscles attacking a red one. As the disease progresses, however, this warfare becomes very conspicuous, owing perhaps to the increased number of the colorless cells. Up to the present the study of these corpuscles has not been extended beyond the observation of the general appearance of these structures, and no attempt is made to explain the apparently marvelous increase in the numbers of the leucocytes. In making these observations the fact has been realized that blood exposed to the air undergoes very rapid changes, and in all the preparations studied this danger has been carefully guarded against.

ETIOLOGY.

The investigations into the nature of this disease indicate that the exciting cause is a bacterium which combines in its properties certain of those found in *Bacillus coli communis*, *B. typhosus*, and *B. choleræ suis*. For instance, it gives a marked indol reaction, ferments glucose without forming gas, saponifies milk, and produces lesions in rabbits similar to those following the inoculation of a variety of the hog-cholera bacillus. It has not been found possessed of a high degree of virulence. It is presumable that its destructive effects in the flocks from which it has been obtained depended to a large degree upon accompanying conditions which, as yet, have not been determined. It is interesting to note, however, that the definite information concerning the conditions under which the fowls were kept in three of the outbreaks shows that the requirements of ordinary hygiene were not strictly complied with. It is apparent from the feeding experiments mentioned on a preceding page that the fowls kept under ordinarily favorable conditions do not readily contract the disease from an affected mate. Although the requirements necessary to prove the specific nature of a micro-organism have been fulfilled in this disease, the uncertainty of fatal results following inoculations suggests that bad sanitary conditions have much to do in determining the severity of the outbreaks. Fowls, like swine and cattle, are sensitive to certain unfavorable conditions respecting environment and food, and it is highly probable that these aggravate the disease in question. It seems very fitting to look upon this malady as a filth disease, and it is not unreasonable to suppose that the disgusting condition in which many farmyards are kept favor the perpetuation of the bacterium isolated and herein described. While it is useless to speculate upon the possibility of undetermined sanitary conditions it is safe to eliminate all suspicious ones which may give rise to the loss of property and the useless destruction of animal life.

DIFFERENTIAL DIAGNOSIS.

The fact that three of the five outbreaks from which fowls were obtained were reported to be due to fowl cholera suggests the possible difficulties which may be experienced in differentiating these two diseases which, in their clinical aspect, appear to resemble each other somewhat closely.

Notwithstanding the numerous reports of fowl cholera in this country, the disease has received very little attention. In Europe it has been more carefully investigated, and from the writings of Perroncito, Touissant, Kitt, Pasteur, and many others can be gathered descriptive accounts of fowl cholera, its pathology, and the properties of its specific organism. In order, therefore, to determine whether the disease in any epizootic among poultry is fowl cholera it is simply necessary to trace a similarity in the morbid anatomy and to discover and identify the specific organism. As the bacterium of fowl cholera has been described since the adoption of modern methods by writers among the foremost French and German bacteriologists, there should be little or no trouble in its identification. Furthermore, it is generally conceded by these writers that the bacterium of fowl cholera is identical with the bacterium of rabbit septicæmia, *Schweineseuche*, and American swine plague.

In 1879, Dr. Salmon investigated certain epizootics of fowl cholera in North Carolina, but since that time little or no advance has been made in our knowledge of the disease producing epizootics among fowls in this country. A careful perusal of Dr. Salmon's description of the disease and his inoculation experiments, and those of contemporary writers on fowl cholera in Europe, will show that the disease he investigated and fowl cholera can not be differentiated. His investigations, which are the only ones of importance reported in this country, established the existence of fowl cholera in the United States. This has afforded a slight excuse for the practice of many farmers and poultry raisers of attributing all destructive outbreaks among poultry to that disease.

From the tendency of the writings upon the etiology of infectious diseases it is evident that the specific organism must be the criterion for determining the nature of the affection. The variability in the manifestation of specific diseases is recognized so that if the specific organism is identified it determines to a large extent the character of the disease. Thus rouget in swine has been found to exist under several different forms, which were recognized as different diseases until Jansen[1] pointed out their identity by showing that their specific organisms were one and the same species. As the course of an infectious disease seems to be modified in accordance with the virulence of the specific organism or the resistance of the infected individuals, and as both of these conditions are subject to variations, the specific organism itself, considered from the standpoint of its morphological character and physiological properties, must be of the first importance in grouping infectious maladies. Applying this principle to the differentiation of poultry diseases, it becomes necessary to include under fowl cholera only such affections as can be demonstrated to be due to the recognizable fowl-cholera bacterium.

A comparison of the important changes in the morbid anatomy in fowl cholera as described by European writers and in the disease under

[1] Deutsche Zeitschrift f. Thier. med., XVIII (1892), p. 287.

consideration can be made from the appended columns, in which their more characteristic lesions are contrasted:

Fowl cholera.	*Infectious leukæmia.*
1. Duration of the disease from a few hours to several days.	1. Duration of the disease from a few hours to several days.
2. Elevation of temperature.	2. Elevation of temperature.
3. Diarrhea.	3. Diarrhea very rare.
4. Intestines deeply reddened.	4. Intestines pale.
5. Intestinal contents liquid, mucopurulent, or blood stained.	5. Intestinal contents normal in consistency.
6. Heart dotted with ecchymoses.	6. Heart usually pale and dotted with grayish points, due to cell infiltration.
7. Lungs affected, hyperæmic or pneumonic.	7. Lungs normal, excepting in modified cases.
8. Specific organisms appear in large numbers in the blood and organs.	8. Specific organisms comparatively few in the blood and organs.
9. Blood pale (cause not determined).	9. Blood pale, marked diminution in the number of red corpuscles.
10. Condition of leucocytes not determined.	10. Increase in the number of leucocytes.

Attention should be called to the fact that as yet there seems not to have been a careful study of the condition of the blood in fowl cholera. Dr. Salmon observed many changes in this fluid which may have been similar to or identical with those herein recorded.

The difference between the specific organism of these two diseases can be readily appreciated by a comparison of the more diagnostic properties of each arranged in parallel columns, as follows:

Bacterium of fowl cholera.	*Bacterium sanguinarium.*
1. Bacterium short, with oval ends.	1. Bacterium short, with ends oval or more pointed.
2. It usually appears singly in tissues.	2. It usually appears in pairs united end to end or in clumps in tissues.
3. Ordinarily it exhibits a polar stain. (From tissue.)	3. It gives a light center, with uniformly stained periphery. (From tissue.) Rarely a polar stain is observable.
4. Grows feebly or not at all on gelatin.	4. Decided growth on alkaline gelatin.
5. It does not change milk.	5. Saponifies milk.
6. Resists drying from one to three days.	6. Resists drying from eight to twelve days.
7. Kills rabbits inoculated subcutaneously in from eighteen to twenty-four hours.	7. Kills rabbits inoculated intravenously in from three to five days. Rabbits inoculated subcutaneously remain well or die in from six to ten days.
8. It kills fowls when injected subcutaneously in small quantities.	8. It does not kill fowls when injected subcutaneously in small quantities.

While there are many similarities in the symptomatology of these two diseases, there are pronounced differences in the morbid anatomy and in the specific micro-organisms. These facts will render the positive differentiation dependent upon a careful bacteriological and pathological examination. It appears, however, that in fowl cholera the disease is more rapid than in leukæmia.

There should be no difficulty in distinguishing between this disease and diphtheria (roup) in poultry. In the latter malady the lesions are usually restricted to one or more membranes of the head. The septicæmia of Lignières can be separated by its specific bacillus, *B. coli communis.*

PREVENTION AND TREATMENT.

From our present knowledge of this disease it is impossible to prescribe a medicinal remedy. A series of systematic treatments has not been tried on account of the failure to obtain outbreaks of the disease in time for such experiments. Its nature indicates, however, that, like the plague in China, cleanliness is the best remedy. Good food, pure water, and ventilation of poultry houses are indispensable. With these it has been found difficult to keep up the disease experimentally, and where these were provided by poultry raisers the disease has not been found to occur.

Several immunizing experiments have been made on fowls and rabbits, but the outcome has been very unsatisfactory. In this work sterilized cultures were used. Methods[1] which were found effectual in certain species with swine-plague and hog-cholera bacteria apparently produced immunity against the disease in but 50 per cent of the fowls treated. The fact that a large percentage of fowls do not die from this malady when fed upon or inoculated with the virus rendered these results of immunization against artificial infection difficult to interpret otherwise than in the negative. As with the hog-cholera bacillus, rabbits could not be immunized against the ordinary dose of this virus by means of sterilized cultures.

Several fowls which were not appreciably affected by the first feeding or inoculation succumbed when the operation was repeated with larger quantities of the virus. In view of these facts protective inoculations can not be recommended until the results obtainable by experimenting upon fowls exposed naturally to the disease, as in an outbreak, are obtained.

In handling a flock of poultry in which this disease has appeared, therefore, the only practical guide to be followed is that which characterizes the successful handling of all infectious diseases, namely, isolation and care. It need scarcely be suggested, therefore, that the sick and the well fowls should be separated. The unaffected should be placed on new ground and fed wholesome food, and the sick ones should be given every advantage of good food, water, and air. In addition to this, a stimulating medicinal treatment is indicated. Quinine in doses of 1 to 2 grains is said to have been used with good results in outbreaks of a disease resembling this in its general appearance. A solution of one-half to 1 per cent of sulphate of iron has also been suggested as a good prophylactic treatment.

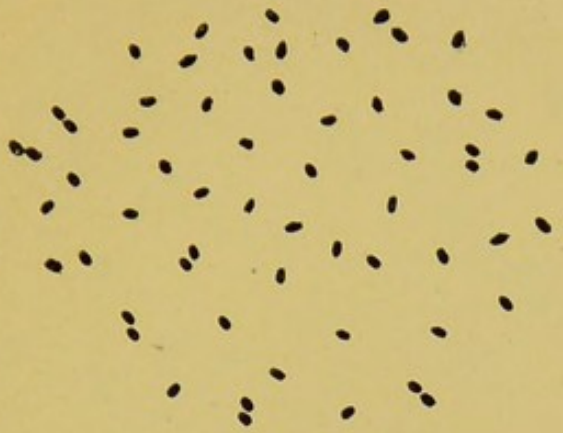
In purchasing poultry the absence of this disease from the flock should be determined before transferring any fowls. The immediate danger of carrying this disease, however, is much less than in fowl cholera, owing to the large quantities of the virus necessary to produce the disease.

The yards and houses in which the disease broke out or where sick fowls were kept should be thoroughly disinfected. For this purpose lime and whitewash, if thoroughly applied, would unquestionably prove effective. Another disinfectant which is quite as easily applied and also inexpensive is the following mixture:

Crude carbolic acid	½ gallon
Crude sulphuric acid	½ gallon

These two substances should be mixed in glass vessels. The sulphuric acid is very slowly added to the carbolic acid. During the mixing a large amount of heat is developed. The disinfecting power of the mixture is heightened if the amount

[1] For details see Bulletin No. 6 of this Bureau on additional investigations concerning infectious swine diseases. Issued 1894.



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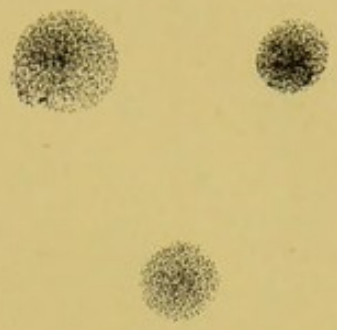

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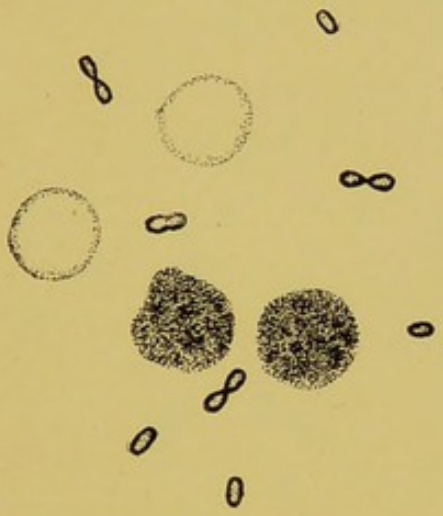

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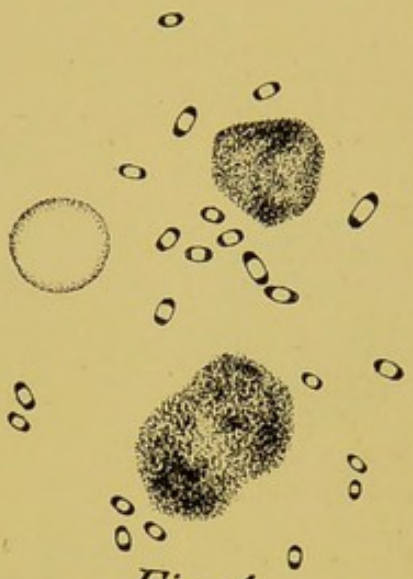

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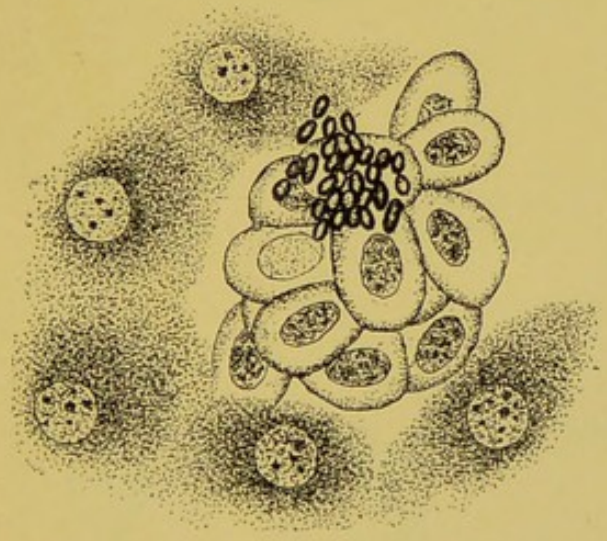


Haines, del. *Fig. 5.*

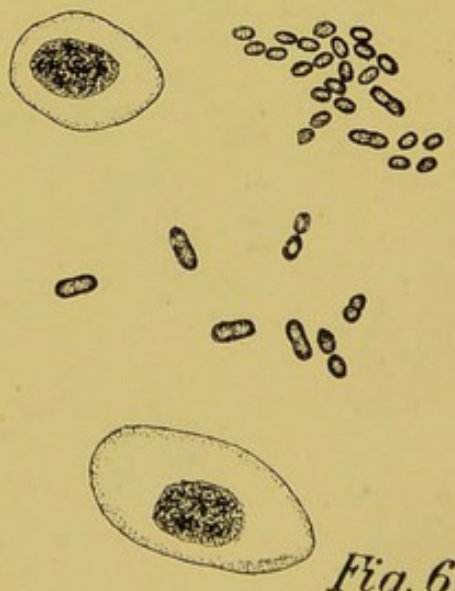
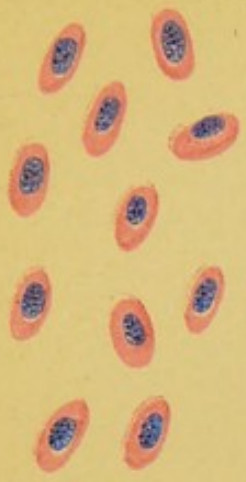


Fig. 6.

BACTERIUM SANGUINARIUM.



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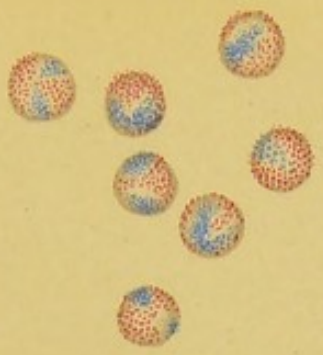

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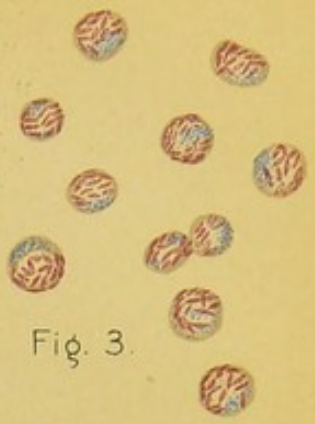

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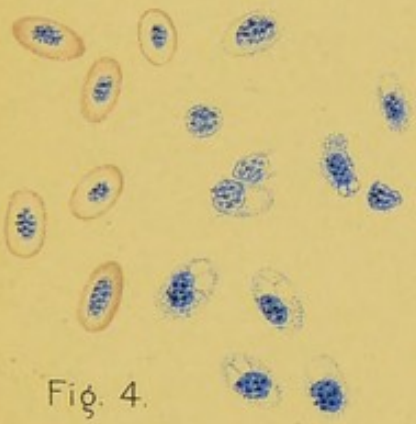

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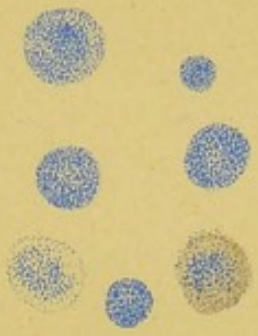
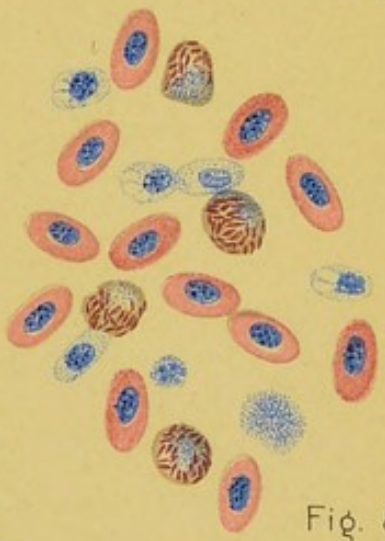


Fig. 5.

Fig. 6.



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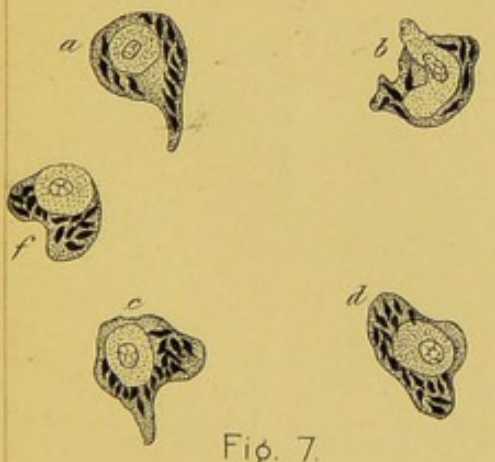
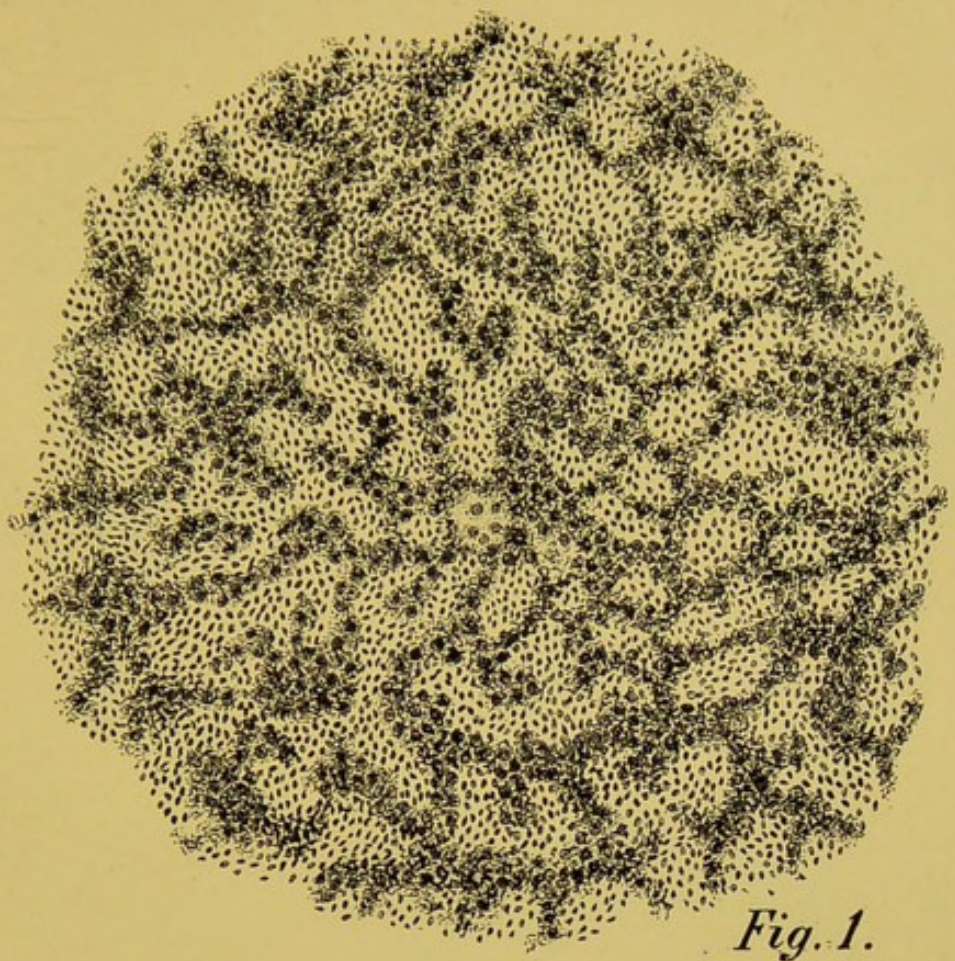


Fig. 7.

Haines, del.

NORMAL AND DISEASED BLOOD.



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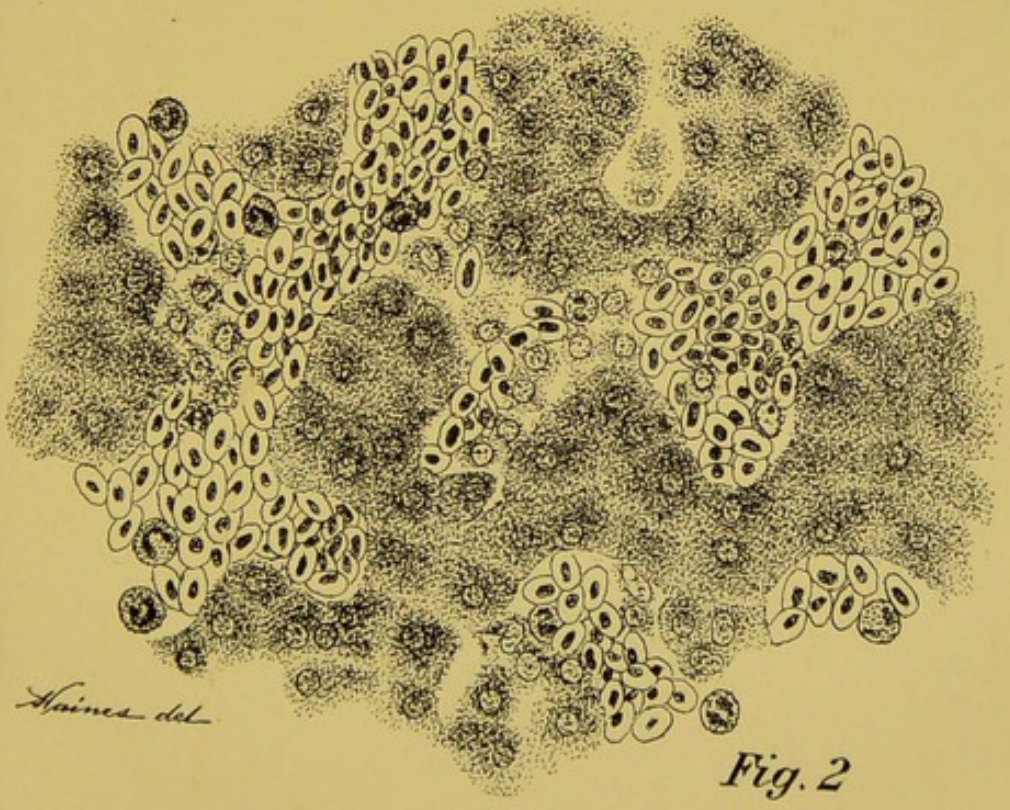


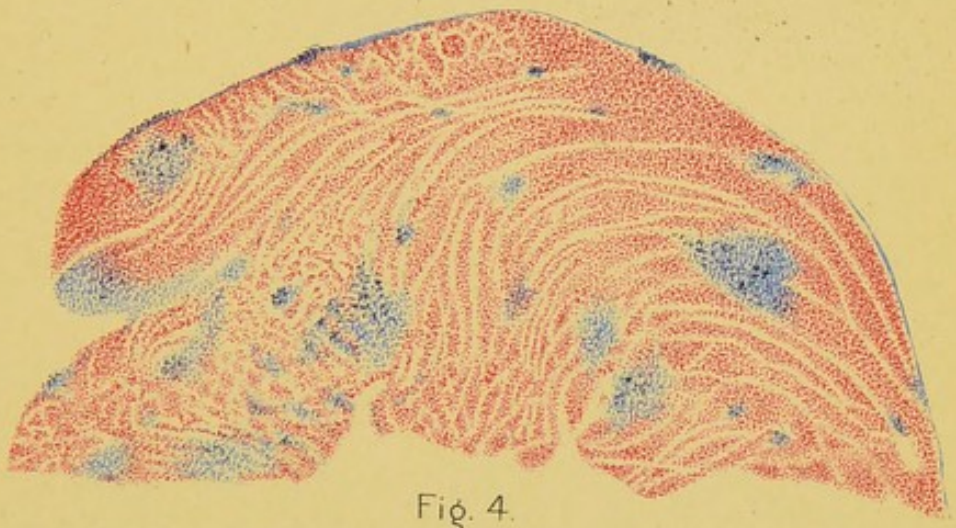
Fig. 2

LESIONS IN THE LIVER.

Fig. 1.

Fig. 2.

Fig. 3.



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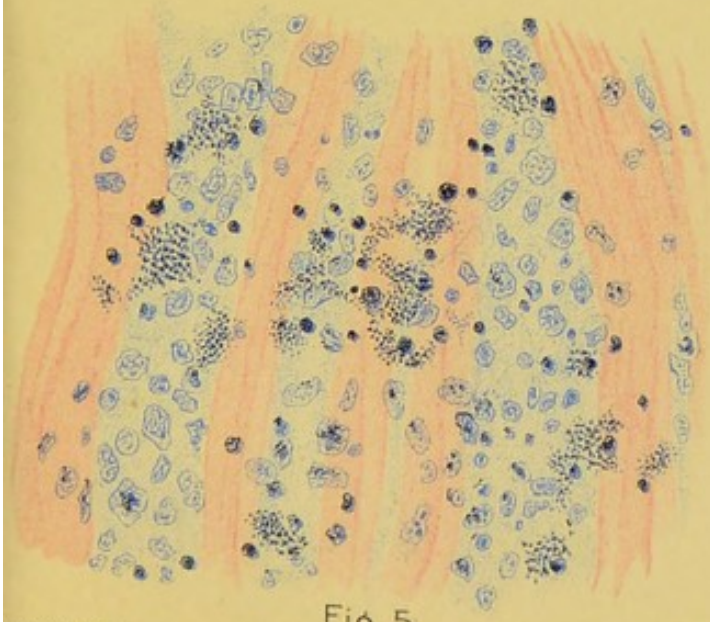

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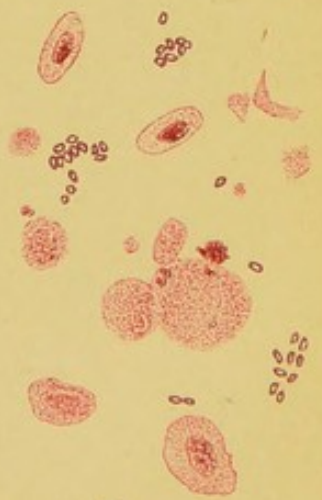
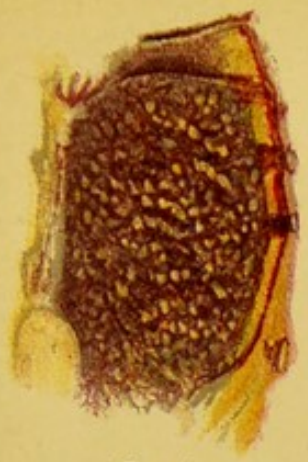


Fig. 6.

Haines, del.

CARDIAC LESIONS.



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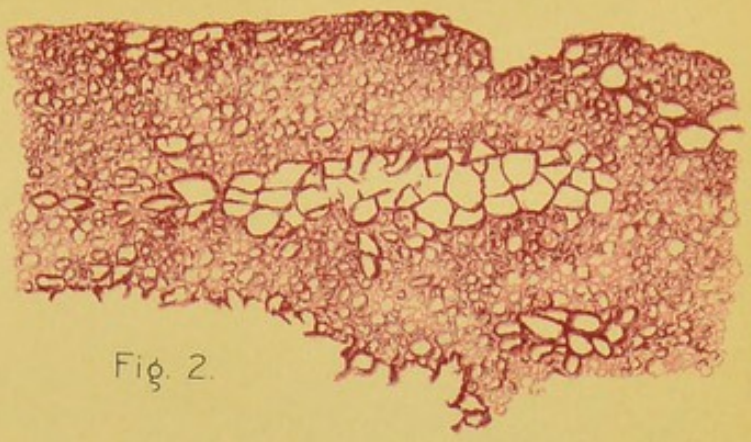

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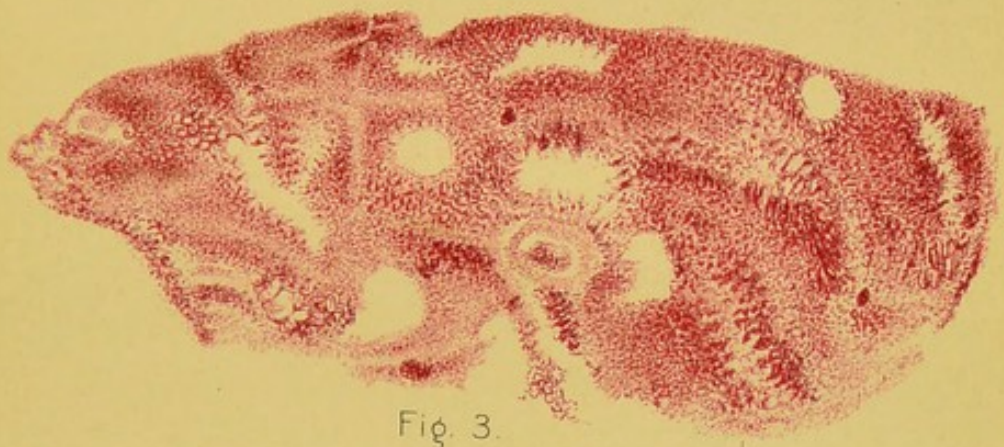

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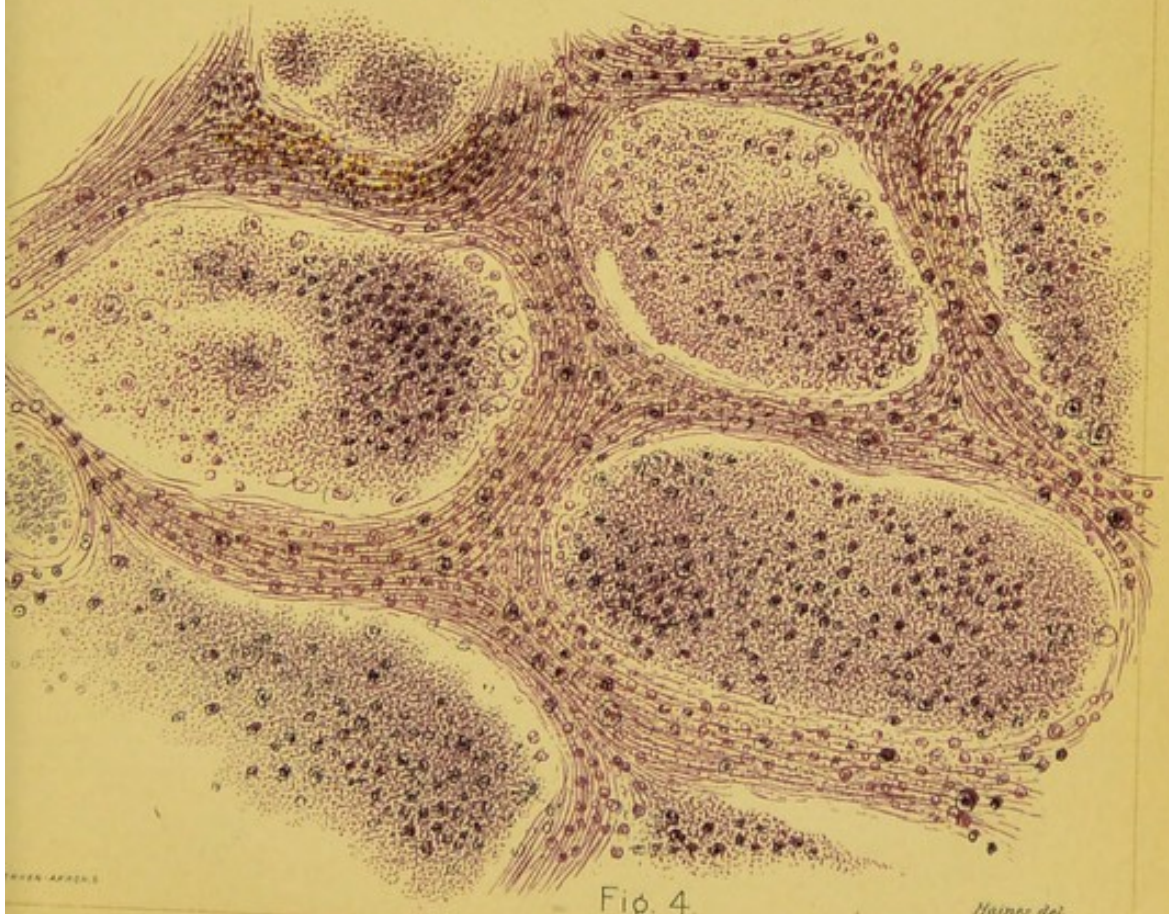


Fig. 4.

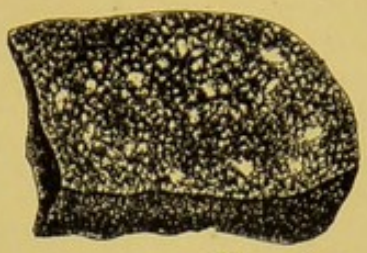
Haines, del.

LESIONS IN THE LUNGS.

Fig. 1.

Fig. 2

Fig. 3.



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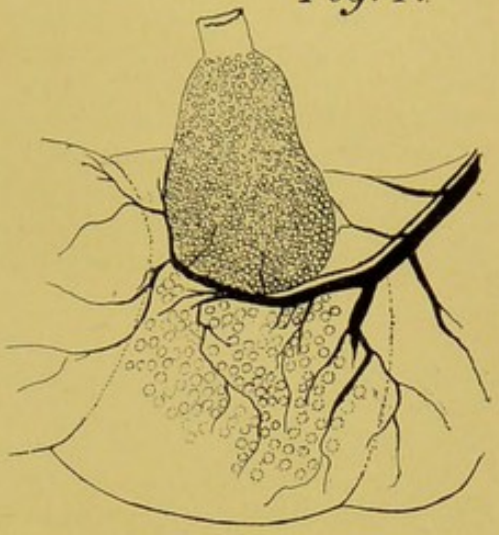

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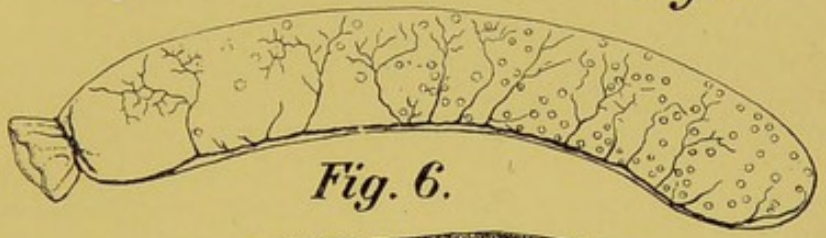


Fig. 6.

Fig. 7.

Haines, del

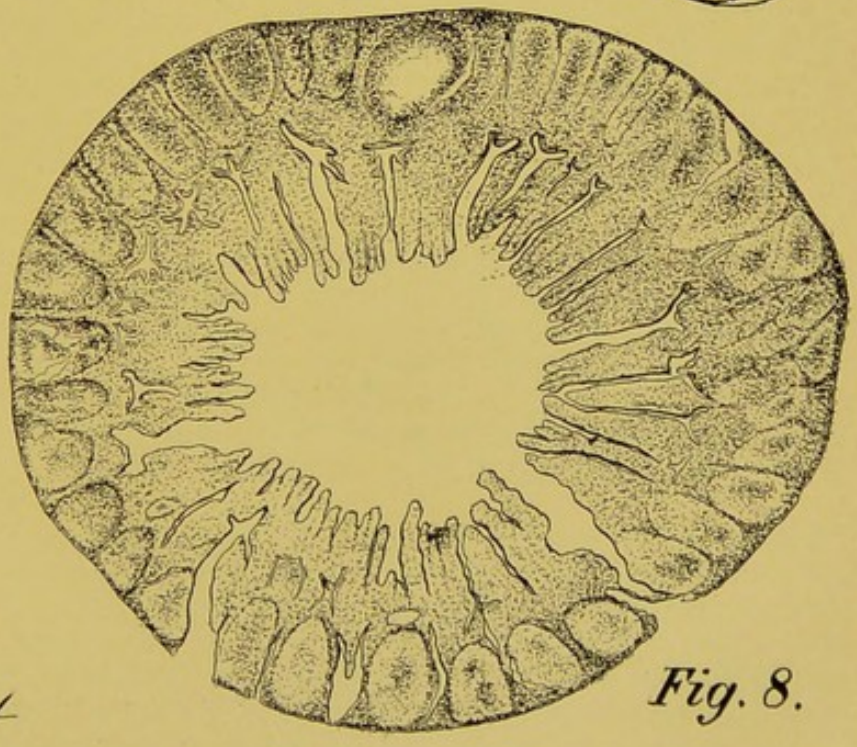


Fig. 8.

of heat is kept down by placing the tub or glass demijohn containing the carbolic acid in cold water while the sulphuric acid is being added. The resulting mixture is added to water in the ratio of 1 to 20. One gallon of mixed acids will thus furnish 20 gallons of a strong disinfecting solution having a slightly milky appearance. This can be applied to buildings with a broom and the surface of the ground in a hen yard can be saturated with it. It is not poisonous, but quite corrosive, and care should be taken to protect the eyes and hands from accidental splashing.

Too much can not be said respecting the sanitary condition of poultry houses and yards. The practice of many farmers in killing a sick fowl because it is the cheapest way to treat it may not be altogether wrong, but it is decidedly out of the realm of economy to neglect those conditions which require attention in order to prevent the occurrence of disease. The affections of fowls under consideration is unquestionably the outcome of bad management rather than the uncontrollable invasion of its specific organism. Just what all these conditions are can not at present be indicated. The experience of practical poultry raisers is necessary to establish the efficiency of the method suggested for preventing this disease.

CONCLUSIONS.

The investigations recorded on the preceding pages have led to certain new and definite results concerning the nature of the diseases which produce outbreaks among poultry. Although the facts elicited add but an increment to our knowledge of the pathology of poultry diseases, they have lowered the bars to an unexplored field in comparative medicine. The conclusions which the facts appear to justify may be summarized in the following brief statements:

(1) Certain of the outbreaks of the infectious disease among fowls popularly called fowl cholera are not due to fowl cholera, as determined by the specific organism, but to a disease resembling it in its fatality and in certain of its symptoms, but differing from it in its morbid anatomy and specific organism.

(2) Infectious leukæmia is characterized by certain well-marked changes in the blood, there being a diminution in the number of red corpuscles and an increase in the number of white ones. It is associated with and presumably primarily due to the presence of a new species of bacterium—*Bacterium sanguinarium*. This organism differs in all esssential differential properties from the bacterium of fowl cholera. It resembles somewhat closely in its physiological properties *Bacillus typhosus*.

(3) This disease of fowls has not been found in flocks where a good sanitary régime has been enforced. It is highly probable that it is a filth disease, being dependent upon unfavorable environments quite as much as the specific organism for the ability to run a rapidly fatal course and of spreading to the entire flock.

(4) Infectious leukæmia appears to be more prevalent than fowl cholera, for which it is often mistaken. Old fowls seem to be more susceptible than chickens.

(5) The economic importance of this disease can not be accurately estimated, but it unquestionably causes the death of thousands of fowls annually. Its elimination seems to depend upon the united efforts of farmers and poultry raisers to put their poultry yards and houses in as perfect a sanitary condition as possible and provide pure water and wholesome food for their fowls. This does not necessitate the expenditure of large sums of money or a great amount of labor. If the annual losses which are now sustained from infectious diseases among poultry are to be saved the causes which produce them must be removed.

DESCRIPTION OF PLATES.

PLATE IX.

The morphology of bacterium sanguinarium.

Fig. 1. Cover-glass preparation made from a bouillon culture 24 hours old. (× 2000.)
2. Colonies on gelatin plates 48 hours old. Drawn with Zeiss apochromatic 16 mm. and compensating ocular No. 4. Outlined with camera lucida.
3. Cover-glass preparation of the spleen of a rabbit showing the deeply stained periphery and light center of *Bacterium sanguinarium*. Also its appearance in pairs. Preparation stained with Loeffler's methylene blue.
4. Cover-glass preparation of the spleen of a rabbit dead from fowl cholera. It shows the marked polar stain of the bacteria; also the increase in numbers over those of a similar preparation (fig. 3) from the spleen of a rabbit dying from the effects of inoculation with *Bacterium sanguinarium*.
Fig. 5. A drawing from a section of a fowl's liver, showing capillary containing blood and a clump of the organisms. (× 2000.)
6. From a cover-glass preparation made from the liver of a fowl, showing the bacteria isolated and in pairs. A clump of the organisms is also observed. (× 2000.)

PLATE X.

Certain changes observed in the blood.

Fig. 1. Normal red blood corpuscles of a fowl stained with methylene blue and eosin. (× 1000.)
2. A form of leucocyte found in healthy fowls. Blood preparations stained with methylene blue and eosin. (× 1000.)
3. The same as fig. 2, but stained differently to bring out the spindle-shaped bodies more clearly. (× 1000.)
4. Red corpuscles found in preparations of blood from diseased fowl. Some of them take the eosin stain feebly, others not at all, and still others contain vacuoles. Many of these corpuscles are broken, the nuclei being either displaced or free. (× 1000.)
5. A form of non(?)nucleated and mononucleated leucocytes observed in fowl's blood. Drawing made from preparation stained with methylene blue and eosin. These forms have not been observed to retain the eosin stain. (× 1000.)
6. A very much enlarged leucocyte containing the spindle-shaped bodies. These bodies are highly refractive in the fresh condition.
7. A white blood corpuscle attacking a red one. Sketches made from a fresh preparation of blood from a diseased fowl. The changes illustrated in the different drawings occurred within a period of thirty-five minutes. The red corpuscle was not free from the leucocyte until the end, although it frequently became nearly so.
8. A drawing from a single field in a preparation of blood made from a fowl the day before death. (× 1000.)

PLATE XI.

The changes in the liver.

Fig. 1. Section of liver from fowl No. 508. It shows the blood spaces engorged. Drawing made with a Zeiss apochromatic 16 mm. objective and compensating ocular No. 4.
2. A higher magnification of a small area of the same section (fig. 1). The most marked changes are the engorgement of blood and the considerable number of liver cells which have a thin or ragged border. Many of these appear to be separated from the adjacent tissue and to lie wholly within the blood spaces.

PLATE XII.

The lesions in the heart.

Fig. 1. Drawing made from a heart of diseased fowl (No. 508) showing grayish areas or points on the surface. (Natural size.)
2. Section of heart (fig. 3) showing the grayish tubercles to extend through the heart muscle. (Natural size.)
3. The heart of fowl (No. 20) which lived eleven days after inoculation. The surface is covered with grayish tubercle-like projections. (Natural size.)
4. A section of heart muscle showing the grayish areas to be sprinkled throughout the muscle. Drawing made from a section stained with methylene blue and eosin. The grayish points consisting of round cell infiltration with bacteria and stained blue in the preparation. Traced with camera lucida. (× about 14.)
5. A drawing of a portion of one of the grayish points under high magnification with Zeiss apochromatic objective 2 mm., 1.30 N. A., and compensating ocular No. 4. Outlines traced with camera lucida. It shows cells and masses of the bacteria between and surrounding the muscle fibers.
6. A drawing from cover-glass preparation showing the bacteria. (× 1000.)

PLATE XIII.

[This plate illustrates the changes found in the lung of a fowl (No. 20) in which the course of the disease was unusually long.]

Fig. 1. A drawing of the lung in situ.
2. A section of a healthy fowl's lung. Magnified about 15 diameters. Outlines traced with camera lucida.
3. A drawing from a section of a diseased lung (fig. 1) under low magnification. Outlines traced with camera lucida.
4. A drawing from a section of the diseased lung, showing a hyperplasia of the intervalveolar tissue. The air spaces are filled with cells and a granular substance. (× 1000.)

PLATE XIV.

The lesions produced in rabbits with Bacterium sanguinarium.

Fig. 1. The enlarged spleen of a rabbit dying from the effect of inoculation with *Bacterium sanguinarium.* (Natural size.)
2. Cross section of spleen. (Natural size.)
3. The spleen of a healthy rabbit of same age and weight as the one inoculated. (Natural size.)
4. Liver of rabbit, showing grayish or necrosed areas of liver tissue. (Natural size.)
5. A drawing of infiltrated follicles on and adjoining the ileo-cæcal valve. (Natural size.)
6. A drawing of the infiltrated follicles in the cæcum in cases which would probably have recovered. (Natural size.)
7. From the cæcum of a rabbit which died as the result of inoculation with *Bacterium sanguinarium.* It shows a uniform cellular infiltration of the follicles. (Natural size.)
8. A drawing made under low magnification of a cross section of an infiltrated cæcum, showing every follicle in the section to be affected. In the section one of the follicles had become caseous in its center. Outlines traced with camera lucida.

TUBERCULOSIS IN SWINE; THE NATURE OF THE DISEASE, WITH A REPORT OF THREE CASES.

By VERANUS A. MOORE, B. S., M. D., and CHARLES F. DAWSON, M. D., D. V. S.

Tuberculosis has come to be recognized as one of the great destroyers of certain of the domesticated animals, as well as of the human species. Much has been written concerning the nature of this disease in cattle and the danger to mankind in using the milk and meat of tuberculous animals, but the literature on the extent of tuberculosis in swine, the peculiarities of the disease in this species, and the source of infection seem to have received little attention, especially in this country. The small number of cases reported may be accounted for in several ways, but the most rational explanation seems to be the fact that veterinarians have been slow in reporting the cases which have come under their observation; furthermore, it is highly probable that from the nature of things they are rarely called to treat this disease in these animals. At present, therefore, tuberculosis in swine is looked upon as an uncommon occurrence in this country, and abroad it is generally admitted to be rare. Statistics are wanting, however, whereby information may be given concerning the cause of death among swine in the dairying districts, where it is well known that it is not uncommon for farmers to occasionally lose pigs from an undetermined malady. If accurate information concerning the cause of these deaths were at hand it might increase very materially our knowledge as to the amount of tuberculosis in swine. If we turn to statistics on this subject, we find that in Europe tuberculosis is much less common in swine than in adult cattle, but that it is not so rare as it is in calves. This is explained by the fact that pigs are more susceptible than calves to tubercle bacilli. Dr. Nocard makes the following statements concerning the amount of tuberculosis in swine and other animals on the Continent:

In Saxony there were slaughtered in abattoirs under inspection in 1891 54,444 adult cattle, of which 9,476, or 17.4 per cent, were tuberculous; 230,808 pigs, of which 2,477, or 1.07 per cent, were tuberculous; 126,322 calves, of which 78, or 0.06 per cent, were tuberculous. In 1892 276,851 pigs, of which 3,804, or 1.37 per cent, were tuberculous. In 1893 309,200 pigs, of which 5,100, or 1.64 per cent, were tuberculous. (Jöhne.)

At Amsterdam in 1890 of 22,813 cattle slaughtered 755, or 3.3 per cent, were tuberculous, while of 30,406 pigs slaughtered 323, or little more than 1 per cent, were tuberculous. (Thomasset.)

On the other hand, at Rouen, M. Veyssière found only 15 tuberculous pigs out of a total of 38,164 slaughtered during four years, or less than 0.04 per cent.

In the duchy of Baden the percentage is still lower, for out of a total of about 8,000 pigs slaughtered each year from 1874 to 1882, only 22 on an average were found to be tuberculous, or a little less than 0.02 per cent. (Lydtin.)

The number of cases of tuberculosis reported from the examinations made by the United States meat inspectors is very small in comparison with the number reported from the abattoirs in Europe. Thus during the fiscal year 1894–95 there were 13,616,539 hogs examined post-mortem by these inspectors and only 579, or 0.00004 per cent, were condemned for tuberculosis. The fact should not be overlooked, however, that a large majority of these examinations were made in the abattoirs in the West, where the hogs slaughtered were obtained largely from the great corn-raising districts, so that a very small percentage of them had been subjected to the dangers attending the indiscriminate feeding of milk and its by-products in the manufacture of butter and cheese.

The infrequency with which tuberculosis is encountered renders it desirable to consider briefly the common source of infection and the manner in which the disease manifests itself. This is important from both the prophylactic and the diagnostic standpoints. As the number of cases which have come under our observation is very small, the description of the disease as we saw it will be preceded by a general discussion based largely upon the data obtained from the writings of European investigators.

SOURCE OF INFECTION.

Nocard[1] states that young animals belonging to the precocious breeds seem to be more liable than others to infection. In nine cases out of ten the animal is infected by ingestion. The pig easily becomes tuberculous when fed on material rich in tubercle bacilli. If pigs are fed on the refuse from dairies and cheese manufactories in districts where there is much tuberculosis in cattle or on tuberculous viscera they readily become infected. Infection through the respiratory tract, while it is certainly possible, he believes to be very rare. The piggeries where the refuse from butter and cheese manufactories is fed and those which adjoin the abattoirs or "knacker's" yards supply the majority of animals found on post-mortem to be tuberculous.

Ostertag[2] has called special attention to this disease as existing among swine in certain parts of northern Denmark and Germany, where there was much tuberculosis in cattle, and where swine were fed upon the slime from creamery separators. Experiments show the possibility of infection by means of the sputum of tuberculous people.

In the cases which have come to our notice there is very strong evidence that the swine were infected by being fed upon milk from tuberculous cows. In one of these cases the tuberculine test showed that a large number of the cows from which the milk was obtained were affected.[3]

SYMPTOMS.[4]

In most cases tuberculosis of the pig is said to be first recognized at the abattoir. Sometimes, however, it causes local and general

[1] The Animal Tuberculoses, 1896.

[2] Milch Zeitung, XXII, S. 672.

[3] We are indebted to Dr. F. L. Kilborne for information concerning this case. (See page 212.)

[4] In view of the fact that we have had little opportunity of observing the disease in living swine, we will quote somewhat extensively Dr. Nocard's description of the symptoms, diagnosis, and progress of the disease.

troubles, which vary according to the organ or system attacked. The following symptoms have been noted:

Its localization in the abdominal organs causes the arrest of fattening and the progressive wasting of the subject. The mucous membranes become pale; the hide becomes dirty; there is usually constipation or diarrhea. The animal is in low spirits and remains buried in its straw for entire days; the corkscrew of its tail is straightened; the abdomen is pendulous and the eye sunken; palpation of the abdomen is painful and may reveal more or less voluminous masses, which are hard and mammillated, due to the changes in the mesenteric glands. It is common to find glandular tumors in the submaxillary region at the level of or at the thoracic inlet. In this form the malady may last several months, but death supervenes rapidly if the lesions are generalized through the blood stream. Primary pulmonary tuberculosis is very rare, but sooner or later lung lesions complicate abdominal tuberculosis, and betray themselves at the outset by a short, dry, abortive cough and by difficult respiration. The cough soon becomes paroxysmal and painful and is often succeeded by vomiting; the respiration becomes hurried and gradually painful and more difficult; wasting is very rapid and death supervenes in a few weeks.

The scrofula of swine (glandular tuberculosis) usually shows itself by a puffing up of the face, which a careful examination shows to be lifted up by the subjacent glands, which are enlarged, indurated, still fairly mobile and free from heat or tenderness. The retro-pharyngeal, superior cervical, and sublingual glands usually take part in the lesion, forming a kind of necklace of unequal and knotty tumors, which extend from ear to ear, and become larger under the neck between the two rami of the lower jaw. Similar tumors may be developed at the thoracic inlet, behind the shoulder, or in the groin, which, as they increase in size, become harder and more adherent to the neighboring tissues. Sometimes, however, a slight fluctuation is perceptible; the tumor softens, then forms an abscess, and discharges a small quantity of thick and grumous pus; but the glandular tumor does not disappear, and the opening into the abscess remains for a long time as a fistula.

At the same time one may notice swellings of the bones, causing a true tuberculosis arthritis when the lesions happen to be situated at the level of an epiphysis. Persistent lameness, fistulous wounds suppurating indefinitely, necrosis, caries, etc., are the complications of these lesions of the bone, the evolution of which is always extremely slow.

DIAGNOSIS.

It is seldom that the veterinarian is consulted on the subject of a tuberculous pig. Most frequently it is after death that he is called to give an opinion on the nature of the lesions, in which case there is usually not any real difficulty. In case of doubt, however, he must not rely too much on the microscopic examination, for in the pig, much more than in the cow, the finding of the bacillus is difficult, and the negative result of the search does not justify the conclusion that tuberculosis is absent. To the examination of cover-glass smears prepared on the spot by scraping must be added the methodical study of sections, and, above all, inoculations. In the sections, even in the absence of bacilli, one may be certain of the nature of the lesion by the discovery of typical follicles almost always rich in giant cells.

Inoculation of guinea pigs into the peritoneal cavity, and into the

subcutaneous cellular tissue of the internal surface of the thigh, enables one to acquire quickly enough the certainty which is lacking. Indeed, although the guinea pig usually resists for a long time, especially when the inoculation product comes from a chronic lesion, either bone or gland, the gland of the fold of the flank becomes rapidly enlarged, then indurated and then obscurely fluctuating. These glands may be punctured or extirpated, and their contents examined for the bacilli. If the result is still negative these contents may be inoculated into a second guinea-pig, which in eight or ten days, the same procedure being made use of, will give the necessary data for a positive diagnosis.

It is sometimes possible to take for tuberculous lesions the changes caused by infectious pneumo-enteritis of the pig (hog cholera) when it assumes the chronic form; in fact, Rolf describes this disease as a scrofulous enteritis. Besides the well-known macroscopic characters which in the immense majority of cases permit one to make the diagnosis of "pneumo-enteritis," one will find, if it is necessary, in the microscopic examination, in the culture and inoculation of the caseous contents of the suspected glands, certain means of avery rapid diagnosis.

If consulted on the subject of tuberculosis in swine, it is quite certain that the clinical signs alone would rarely warrant the diagnosis. The injection of tuberculin would be the surest and most rapid way out of the difficulty, for in the tuberculous pig, as in the tuberculous cow, the reaction set up by the tuberculin is specific. The dose for injection varies from 10 to 25 centigrams, according to the weight of the animal.

The infrequency of tuberculosis in swine is said to be due principally to the fact that in this species the disease assumes the acute or galloping form, and as a result the affected animals succumb before they have been able to scatter the virus to any great extent. Although this may be the rule, there are numerous exceptions to it. During the last few years Nocard has collected more than thirty observations of scrofula or chronic tuberculosis in the pig. The disease always presents the same appearance—massive lesions, rather thinly scattered, rich in giant cells but very poor in bacilli, so poor in fact that it has been found necessary to carefully examine four or five sections before meeting a single bacillus, but the inoculation of the suspected material into the peritoneal cavity of guinea pigs reveals its tuberculous nature. The guinea pigs do not always succumb rapidly, but, on the contrary, frequently hold out for three or four months, and even longer. If those which seem well are killed five or six weeks after inoculation, the spleen, the liver, and sometimes the lungs will be found stuffed with tuberculous nodules in the center of which it is possible to find a few tubercle bacilli.

It is stated that there is a tendency to increase the rapidity of the disease by passing it through a series of animals by inoculation from one to another. The lesions become more minute, more confluent, and also richer in bacilli. When one has recourse to intravenous inoculations the experimental animals, after the fifth or sixth transmission, die in from twenty to twenty-five days from a veritable tuberculous septicæmia. The spleen, the medulla of the bones, and the liver, although they do not show nodules visible to the naked eye, are crowded with myriads of tuberculous follicles which are exceedingly rich in the specific organisms.

MORBID ANATOMY.

The manifestations of tuberculosis in swine, as suggested in the foregoing statement, are exceedingly interesting. Nocard finds the lesions to consist of miliary granulations which rapidly become caseous, as in cattle, but which more rarely contain calcareous salts. Generalization is common, in which case the viscera are thickly sprinkled with gray granulations which are translucent throughout, or opaque in their centers, and quite analogous to those found in tubercular lesions in other animals.

As the disease most often results from ingestion of the virus, the digestive apparatus and the corresponding lymphatic glands (submaxillary, parotid, pharyngeal, superior cervical, mesenteric, sublumbar, etc.) may be decidedly altered, while the other organs remain practically intact. Lesions of the small intestine and the cæcum are common, and take the form of ulcers of the mucous membrane, of miliary nodules, or of tuberculous infiltrations involving at once the mucous, the muscular, and subserous tissues. The lesions in the liver take the form either of miliary granulations, which are yellow and caseous and scattered in great numbers through the thickness of the organ, or else of rounded nodosities which are yellowish white in color, varying in size from that of a pea to a hazelnut, and of a tough consistency. On section they appear sometimes to be firm, homogeneous, and fibrous; sometimes softened in the center, but rarely infiltrated with calcareous salts. The peritoneum and the pleura are sometimes the seat of an eruption of fine granulations which remain in a state of miliary nodules. Lesions like those in the liver may exist in the lungs, but generally there is found in these organs an innumerable quantity of minute translucent, gray granulations, caused by generalization through the blood stream, in which case the liver, the spleen, the kidneys, the medulla of the bones, and the mammæ are usually infiltrated with similar growths.

It is common to find lesions localized in one or several lymphatic glands. The tonsils and the pharyngeal or submaxillary glands are the ones most often affected. They become voluminous, hard, and knotty, as they have undergone a true fibrous transformation and consequently, difficult to cut. This is shown by the tissues creaking under the cutting instrument. In section they have the appearance of old fibrous tissue; here and there small yellow foci are seen of a softer consistency, almost caseous; sometimes veritable purulent collections are found, either encysted or in communication with the exterior. If one submits the caseous or purulent matter to a bacteriological examination, tubercle bacilli are not usually found. The bacillus, however, is present and if this matter is inoculated into the peritoneal cavity or the cellular tissue of guinea pigs will produce tuberculosis.

These chronic glandular lesions, with their very slow progress, have long been looked upon as constituting the scrofula of swine, and to scrofula was also assigned the tuberculous lesions of bones (ribs, vertebræ, articular extremities, shoulder blades, hip bones, etc.) which are common in pigs, both young and old.

The older authors noted that the ancient scrofula was often accompanied by visceral tuberculosis, but they refused to admit the identity, and even the relationship, of the two affections.

The generalization of the disease especially in the muscular tissue

is reported by several observers. Moulé[1] calls attention to this peculiarity of the disease. Stockman[2] shows that while the disease is ordinarily generalized, muscular lesions may exist in swine in the absence of generalization. Zschokke[3] has called special attention to the localization of tubercular lesions in the head of swine, especially in the nares and brain.

The lesions in the animals which we have examined correspond very closely with those described by European writers.

DESCRIPTION OF THE THREE CASES.

The cases of tuberculosis in swine which have come under our observation are from two outbreaks only. They are, however, of much importance, as the source of the infection in both instances was presumably the consumption of milk from tuberculous cows. Although the data are few, the demonstration of the disease in two herds on dairy farms is of considerable interest.

Outbreak 1.

Pig No. 1.—In February, 1896, a piece of a diseased pharyngeal gland was received at this laboratory from Dr. F. L. Kilborne, of Kelloggsville, N. Y., from whose letters the following brief history of the disease was obtained.

He (Dr. Kilborne) had occasion to test a herd of milch cows with tuberculin, and found that a large percentage of them were tuberculous. The owner had from time to time lost swine, and Dr. Kilborne was requested to make an examination of a pig which had died at that time. The lesions indicated tuberculosis, and he sent a small piece of a diseased gland to this laboratory for further examination and diagnosis. Upon its reception the tissue was found to be somewhat decomposed from post-mortem causes. It was about the size of a walnut and consisted of a grayish, necrotic mass. Cover-glass preparations failed to reveal the presence of tubercle bacilli, and consequently a guinea pig was inoculated February 11 with a piece about the size of a small pea. It died March 5, with extensive tubercular lesions in the lymphatic glands, liver, spleen, and lungs. Properly stained cover-glass preparations from these lesions showed tubercle bacilli in considerable numbers.

A further and more detailed account of the outbreak in the herd from which this animal came has not yet been received. The important fact was obtained, however, that at least one pig in a herd fed upon the milk of known tuberculous cows was affected with tuberculosis.

Outbreak 2.

On May 2, 1896, Mr. D., who owns and operates a farm on the outskirts of the town of Rockville, Md., requested the Secretary of Agriculture to have a disease which existed in his herd of swine investigated. A few days later one of us (Dawson) visited his place and found his herd to consist of nine Berkshire pigs, which had been purchased in January at public sale from a dairyman residing near by. They all belonged to the same litter, but at the time of the visit

[1] Recueil de Médicine Vétérinaire (Annexe), Tome VI, Serie VII (1889), p. 455.
[2] The Veterinarian, May, 1895; ibid., March, 1896.
[3] Deutsche tierärztlich Wochenschrift, 1895, No. 36, S. 313.

they varied greatly in general appearance and size. Four of them appeared to be healthy, and would have weighed about 75 pounds each. The remaining five were emaciated, and would not have weighed more than 40 pounds. The owner reported that they were all affected with a cough when purchased.

The symptoms in the appreciably affected animals at the time of inspection were those indicating a disease of the respiratory tract. There were cough, hurried respiration, nasal discharge, thirst, loss of appetite, emaciation, and pallor of the mucous membranes. When undisturbed, the weak ones would lie down. If disturbed or made to run, they soon became exhausted.

Pig No. 2.—With the consent of the owner one of the smaller pigs was killed by a blow upon the head for the purpose of post-mortem examination. The carcass was fixed by tying its legs to pegs driven into the ground. An incision was made through the skin from the lower lip to the pubes along the median line, and the skin reflected. Of the superficial lymphatics thus exposed, those in the submaxillary region were the only ones showing the disease. They were much enlarged, and when sectioned imparted a grating sensation.

An incision through the abdominal median line exposed the viscera. The serous and mucous surfaces of the intestines were normal in appearance. The mesenteric lymphatic glands were affected generally. They approached an almond in size, and when sectioned were seen to be the seat of extensive tuberculous change. In some instances the glandular tissue was necrotic, and in others it had become calcareous. The necrotic material could be easily enucleated from its capsule.

The liver was dotted here and there with circumscribed, grayish areas about 2 mm. in diameter. On section this organ showed the disease to be largely near its surface.

The spleen was extensively involved, and its surfaces were literally covered with comparatively large projecting tumors having a yellow summit broadening gradually to the normal spleen tissue. The larger ones measured from 10 to 18 mm. in diameter. Besides the large ones, which numbered about 30, there were innumerable small nodules less elevated and varying in size from 1 to 5 mm. The spleen as a whole was considerably enlarged, probably one-third over its normal dimensions, and with the exceptions noted was normal in color.

The thoracic cavity was exposed by an incision on either side of the sternum through the ribs at the costo-chondral articulations. The lungs were extensively diseased. Palpation of their surfaces gave the impression of the existence of very numerous and closely set hard nodules beneath the pleura. Section of the whole of the right lung revealed the fact that it was extensively invaded by miliary tubercles situated mainly in the interlobular connective tissue, but in many cases the lobules themselves were entirely obliterated. When the lung was sectioned, an abundance of frothy mucus and pus welled up from its cut surfaces.

The entire cephalic lobe of the right lung was affected. About the center of the lobe there was an abscess about one-half inch in diameter containing pus, mucus, and necrotic tissue. This cavity was continuous with the main bronchus of the lobe, and probably discharged its contents into it. In the same lobe and near its base there was a large abscess containing a cheesy material and also considerable gelatinous mucus.

The left lung was equally involved with the right, the cephalic and ventral lobes being the seat of extensive abscesses containing fluid pus.

The posterior lobes of both lungs were adherent by strong, fibrous bands to the thoracic walls, to the pericardium, and to each other. The pericardium contained an abnormal amount of straw-colored serum.

The mediastinal glands were enormously enlarged, as were also the glands at the bifurcation of the trachea. Section of them showed their tissues to contain numerous pin-head tubercles.

GUINEA-PIG INOCULATIONS.

May 6 guinea-pig No. 299 was inoculated by placing under the skin of the right side a piece of the tuberculous lung tissue about the size of a pea:

June 15. The guinea pig was found dead this morning. Emaciation pronounced. At the point of inoculation there was an infiltration of a purulent substance into

the subcutaneous and subjacent muscular tissues, extending over an area about 1 inch in diameter.

The superficial lymphatics in the right inguinal and those in the right axillary and submaxillary regions were enlarged and indurated. On removing the sternum an enlarged lymphatic gland was seen situated upon the superior surface of the manubrium. It was about the size of a split pea. Both lungs were deeply reddened and thickly sprinkled with pin-head-sized tubercles.

The pleural cavity contained considerable sero-sanguinous liquid.

The tracheal and mediastinal lymphatics were much enlarged and cheesy. The liver was considerably enlarged, and its surfaces were almost entirely occupied by diseased areas of a bright greenish or yellowish color. The spleen was enormously enlarged, engorged with blood, presenting upon its surfaces numerous grayish tubercles. The lumbar and pelvic lymphatics were also much enlarged and cheesy upon section.

A second guinea pig (No. 298) was inoculated on the same day (May 6) by introducing into the subcutaneous tissue a small piece of a lymphatic gland:

June 18 this guinea pig was much emaciated, and exhibited other signs of tuberculosis. It was killed with chloroform for examination. The lesions resembled very closely those noted above in the guinea pig inoculated with the piece of lung tissue.

Cover-glass preparations stained after Ziehl-Neelsen's method from the liver tissue of both the guinea pigs exhibited tubercle bacilli.

May 15 a second visit to Mr. D.'s farm was made by one of us (Dawson) for the purpose of making further observations. During the intervening time one of the pigs had died. The owner kindly consented to the slaughter of another diseased pig which was carefully examined.

Pig No. 3.—This animal weighed about 40 pounds. The mode of procedure in the examination was identical with that described for pig No. 2.

Of the superficial lymphatic glands the left inguinal was enlarged, and upon section was seen to contain one small focus of the disease.

The submaxillary lymphatic glands on both sides were enlarged, the one on the right side enormously so.

The lymphatic glands in the smaller curvature of the stomach were enormously enlarged, and upon section their tissue was seen to be entirely changed to that of a tuberculous nature. One of the mesenteric lymphatic glands opposite the ileum was much enlarged, and upon section showed that its entire parenchyma had undergone tuberculous changes. It could be easily removed from the capsule.

The sublumbar lymphatics were much enlarged and contained foci of tuberculous disease.

The serous membrane between the coils of the large intestine was extensively infiltrated with miliary tubercles.

The liver was diseased throughout. Upon the surfaces of all the lobes there were many irregular yellowish areas. The interlobular connective tissue was hyperplastic, and in many instances the increase of this tissue had partly obliterated the individual lobules. In some cases the lobules were much atrophied. Section of the largest of these areas showed them to extend down into the liver parenchyma for variable distances, and that in them were situated small pinhead-sized granules of calcified matter. By scraping the cut surface of the organ the hepatic cells could easily be removed, leaving a surface resembling the cells of a honeycomb. The lymphatics in the portal fissure did not appear to be affected. The spleen was affected as in the first pig examined from this herd. The large yellow tuberculous tumors were very numerous, extending entirely through the substance of the organ. When cut through, the interior of the tumor mass was of a yellow color, resembling cheese. In the center were numerous calcified deposits.

The sublumbar and pelvic lymphatics were not noticeably affected in this case.

The lymphatics in the submaxillary regions were much enlarged and were palpable during life. When sectioned they showed the entire parenchyma infiltrated with tubercles.

Upon removal of the sternum and attached rib cartilages an enlarged lymphatic gland situated upon the superior surface of the manubrium was noted. It had

attained the size of a filbert nut, and when sectioned showed about half of its tissue infiltrated with small yellow tubercles.

One of the tracheal glands just anterior to the sternum was much enlarged and extensively diseased.

The mediastinal system of lymphatics was enlarged and extensively diseased.

The caudal lobe of the left lung was the seat of miliary tuberculosis throughout its mass, and at the anterior border there was a complete breaking down of the tissue into a muco-purulent substance, this area being directly continuous with the central bronchus of the lobe.

Fully one-half of the ventral lobe of this lung was similarly affected. One of the tips of the azygos lobe was completely consolidated. Section through this area showed it to be of the consistency of fresh cheese.

The right lung was affected similarly to the left lung. The posterior border of the right cephalic lobe was completely cheesy and was adherent to the anterior margin of the right caudal lobe.

The lymphatic gland to the left of the bifurcation of the trachea was much enlarged and imparted a grating sensation when cut through. Examination of the pharynx did not reveal a tuberculous affection of its lymphatic system.

Unfortunately the cranial and spinal meninges were not examined in either case, nor was any special examination made of the skeletal muscular system.

A letter from Mr. D., written early in June, stated that one of the remaining pigs had died. We were desirous of applying the tuberculin test on the apparently healthy animals in the herd, but the owner refused to allow any of them to be killed if they gave a reaction, and consequently the test was not made.

MICROSCOPIC EXAMINATION.

Pieces of the organs of these pigs were fixed in formalin and alcohol, sectioned, and stained, both for the study of the tissues and for tubercle bacilli. The study of these sections revealed some very interesting conditions, largely confirmatory, however, of the descriptions already written concerning the histology of this disease. They are, however, of sufficient interest to receive a brief consideration, in view of the fact that the minute morbid anatomy of swine tuberculosis is not generally understood. There seem to be a few variations in the histology of this disease in swine from that in other species, but as we have examined the lesions in but two animals these exceptions can not be considered of more importance than can be attributed to individual variations. However, it is only by the study of isolated cases and individual variations that a comprehensive knowledge of the pathology of this disease in swine can be obtained.

The disease process in the lymphatic glands was similar in its minute anatomy to that already described as existing in other animals. The sections of the infiltrated tissues adjacent to the necrotic foci contained very few giant cells. Tubercle bacilli were present in small numbers in the sections from the mesenteric glands of pig No. 3, but in those from pig No. 2 they were exceedingly abundant. (See Pl. XVII, fig. 2.)

The tubercles in the spleen were of special interest. (Pls. XV and XVIII, fig. 1.) Upon section they were firm, and presented, as a rule, a clear homogeneous appearance. In the larger ones foci of cell infiltration, which occasionally contained calcareous deposits, were found. Distinctive layers of round and epithelioid cells surrounding these foci were not detected. The greater part of the tubercle consisted of hyperplasia of the connective tissue sprinkled with round cells. Giant cells were abundant, especially in the outer third of the

nodule. Tubercle bacilli were not detected in any of the sections from pig No. 3, but in those from No. 2 there were a few.

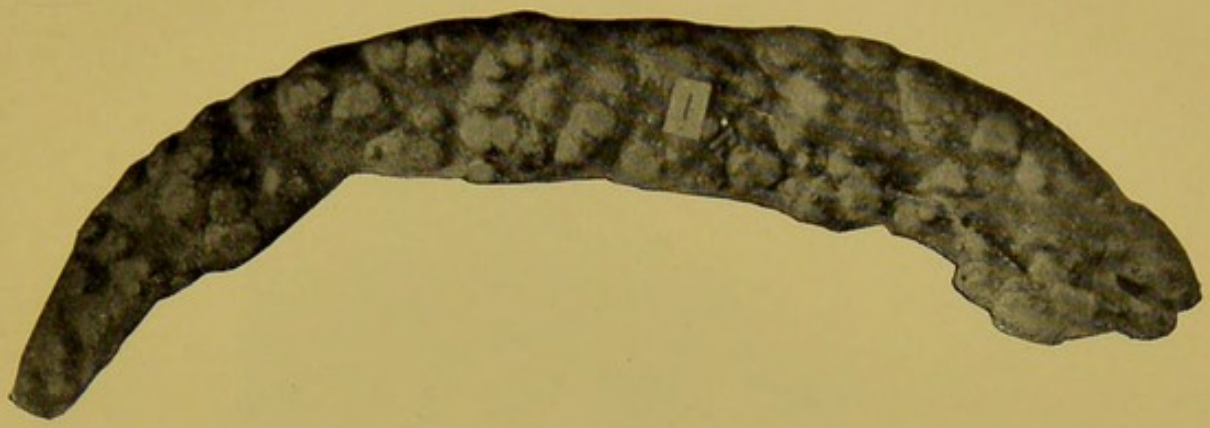
The lesions in the liver were also interesting. The cell infiltration seemed to be somewhat diffuse, following the interlobular tissue and gradually encroaching upon the lobules. Distinctive anatomical tubercles did not appear. In general the disease in the liver resembled that in the liver of guinea pigs. The grayish granules on the surface, or as they appear on section in the depth of the tissue, consist of areas of round cell infiltration sprinkled with a greater or less number of multinucleated or giant cells. The infiltrated tissue usually undergoes disintegration, giving it, when examined microscopically, a fine granular appearance. The line of demarcation between the diseased and unaffected tissue is very sharp. As the process advances the liver cells at the margin disintegrate. The nature of the disease in the liver is illustrated in Pls. XV and XVIII, fig. 2.

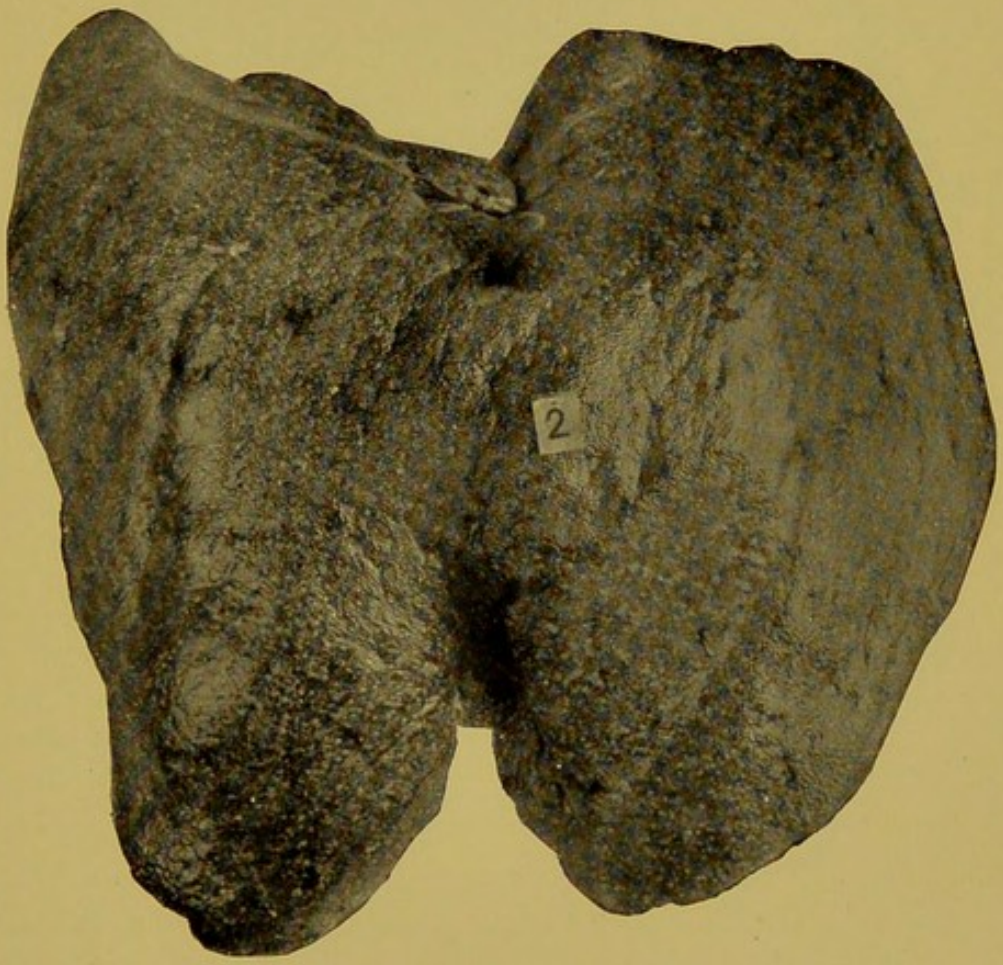
In the lungs the disease seems to follow somewhat the same course as it does in the liver, starting from the center of primary localization. In the cases examined the cephalic, ventral, azygos, and also a portion of the principal lobes were caseous. In histological sections of the recently invaded portions miliary tubercles were abundant (Pl. XVIII, fig. 4), separated by normal or slightly infiltrated interalveolar tissues. Frequently the centers of these tubercles were caseous, and in a few cases there were slight deposits of calcareous salts. While these tubercles were distinct, they did not exhibit any structure peculiar to this species. The central portion was usually densely infiltrated with round cells surrounded with a firm fibrous tissue also sprinkled with round cells. Giant cells were exceedingly rare, and tubercle bacilli were not detected in any of the lung tubercles.

The pleura was not appreciably affected in either animal. The muscular tissue was not examined microscopically, and unfortunately inoculation experiments were not made from the muscles or muscle juice. As tuberculosis is a disease affecting largely the lymphatic system, it is frequently impossible to detect the disease in any part or organ from a macroscopic examination. The marked generalization of the disease in the viscera and its tendency to follow the tissues rich in lymphatics renders it highly probable that the lymphatics of the muscular system might be involved without exhibiting anatomical changes by which such a condition could be readily detected.

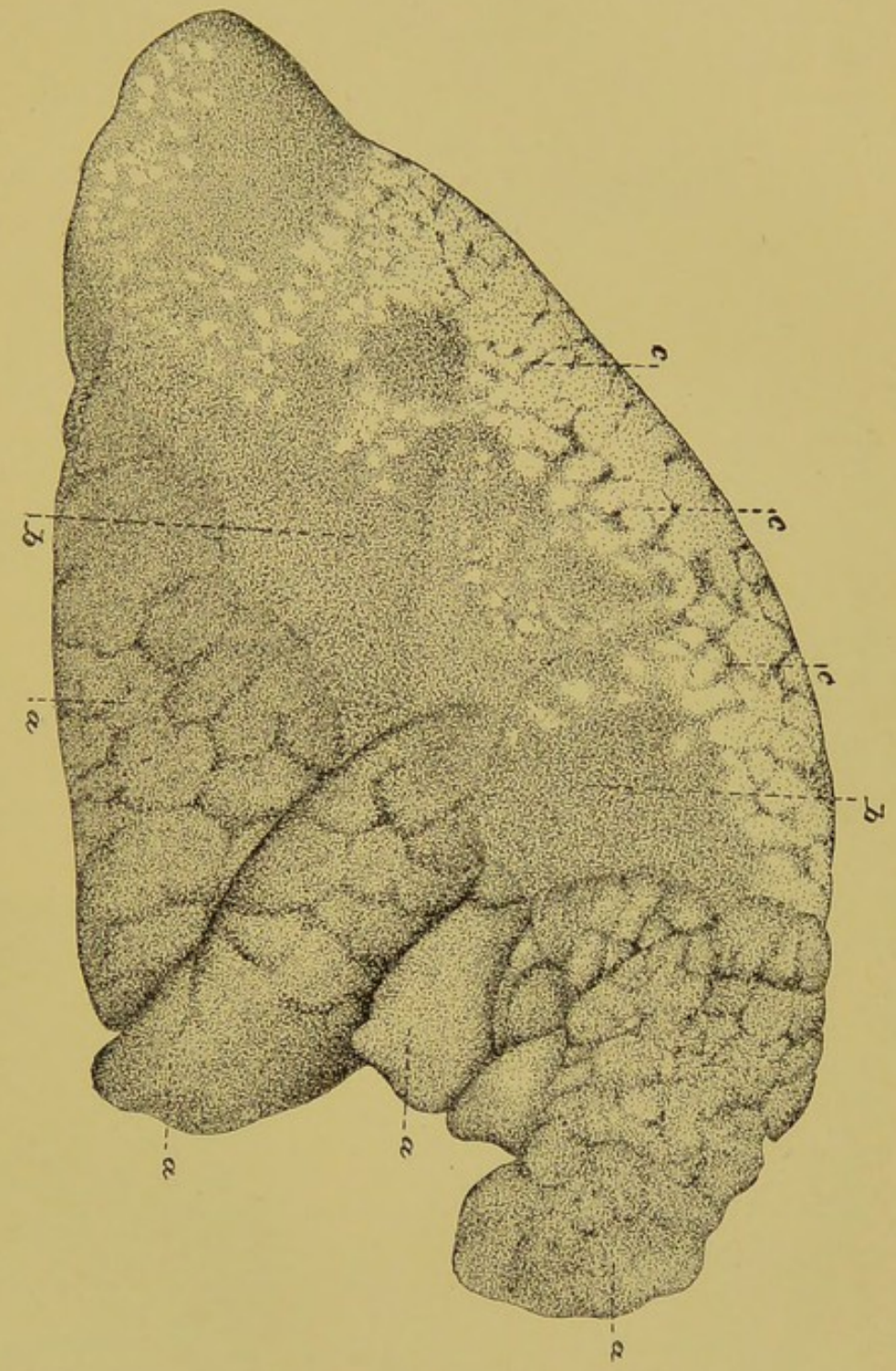
THE RELATION OF SWINE TUBERCULOSIS TO PUBLIC HEALTH.

In France the sanitary laws do not apply to tuberculosis in swine. The regulations of most of the abattoirs, however, require the seizure of tuberculous pigs. It is considered very fortunate that they do so, because in the pig the disease seems to become generalized much more readily than in other animals, and the result of experiments on this subject show that the muscle juice of tuberculous swine is often virulent. The fact that in France much of the pork butcher's meat is consumed without being previously cooked increases the danger in that country of the disease being transmitted to the human species. Although we have no positive knowledge that tuberculosis has ever been transmitted from swine to the human species, there is a very impressive lesson to be learned from the demonstration of the existence of this disease in the porcine tribe. To those who recognize the importance of tuberculosis in animals as a factor in public health the lesson is obvious, but to those who persist in questioning the transmission of this disease from animal to man or from one animal to





Tuberculous Spleen and Liver.



TUBERCULOUS LUNG.

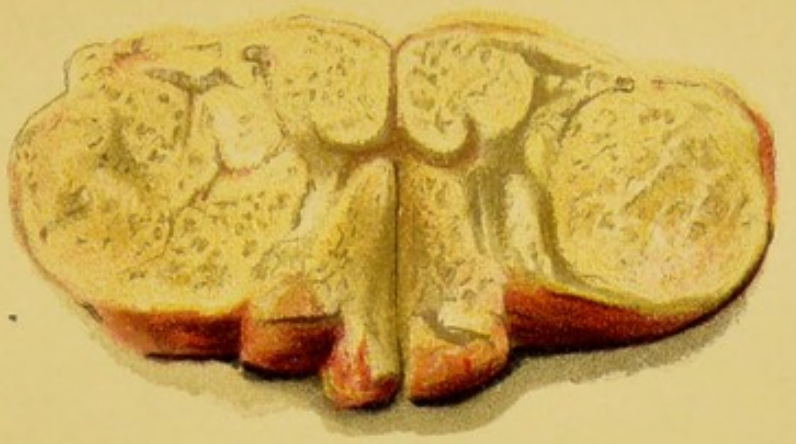


Fig. 1.

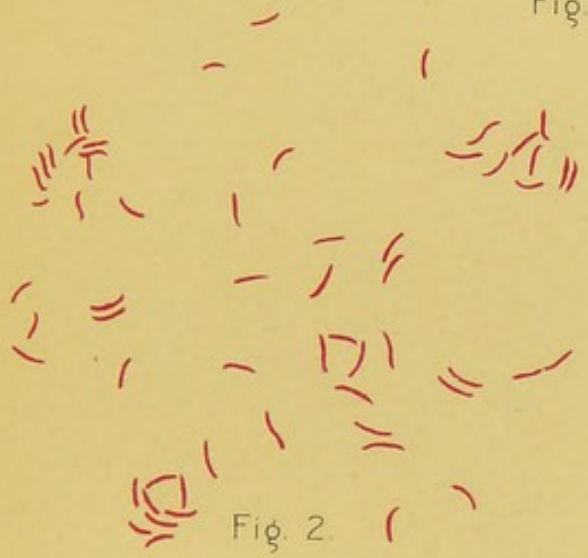


Fig. 2.

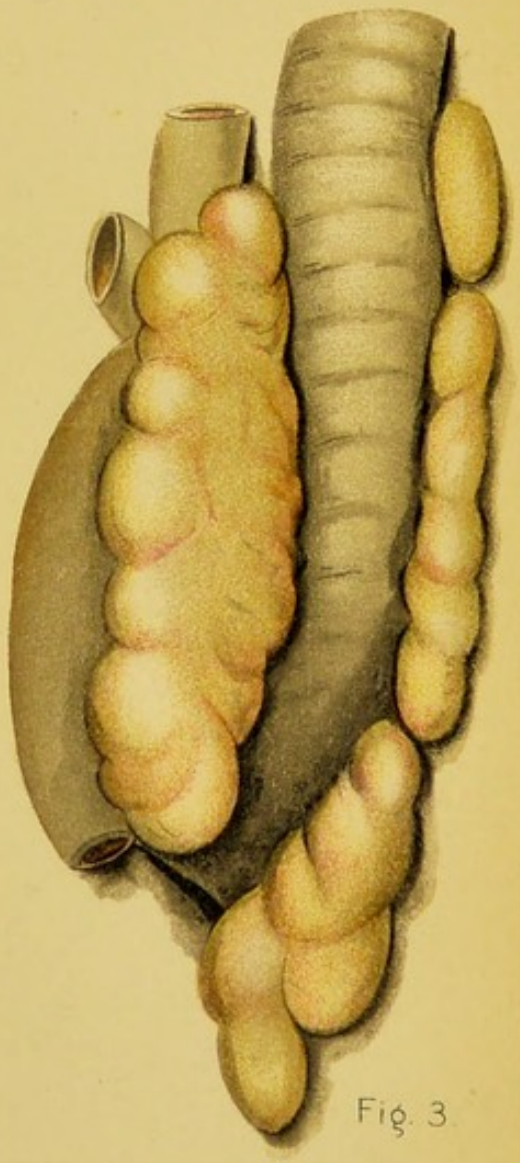


Fig. 3.

Fig. 4.

Haines, del.

TUBERCULAR GLANDS AND TUBERCLES.

Fig. 1

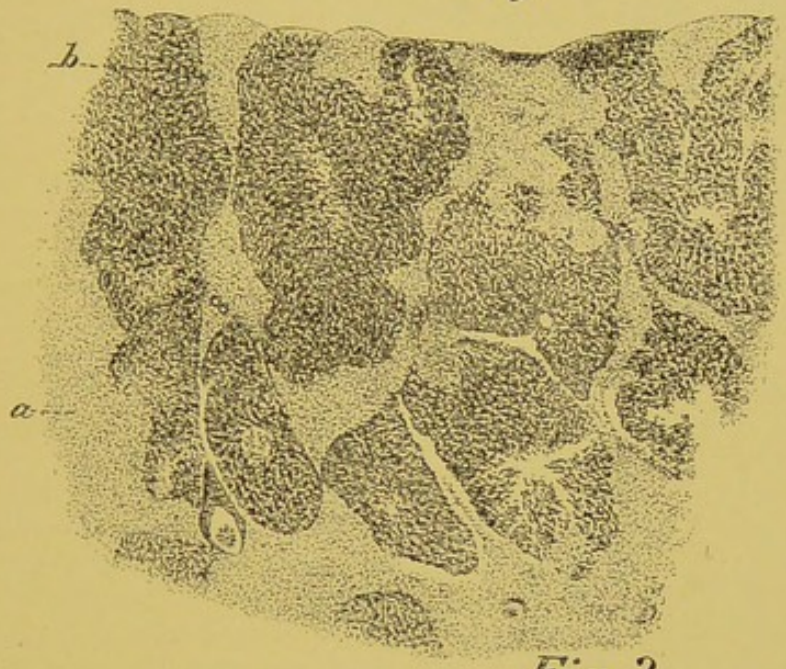


Fig. 2.

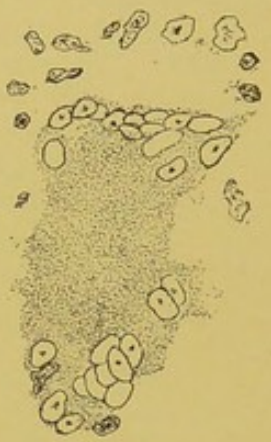


Fig. 3.

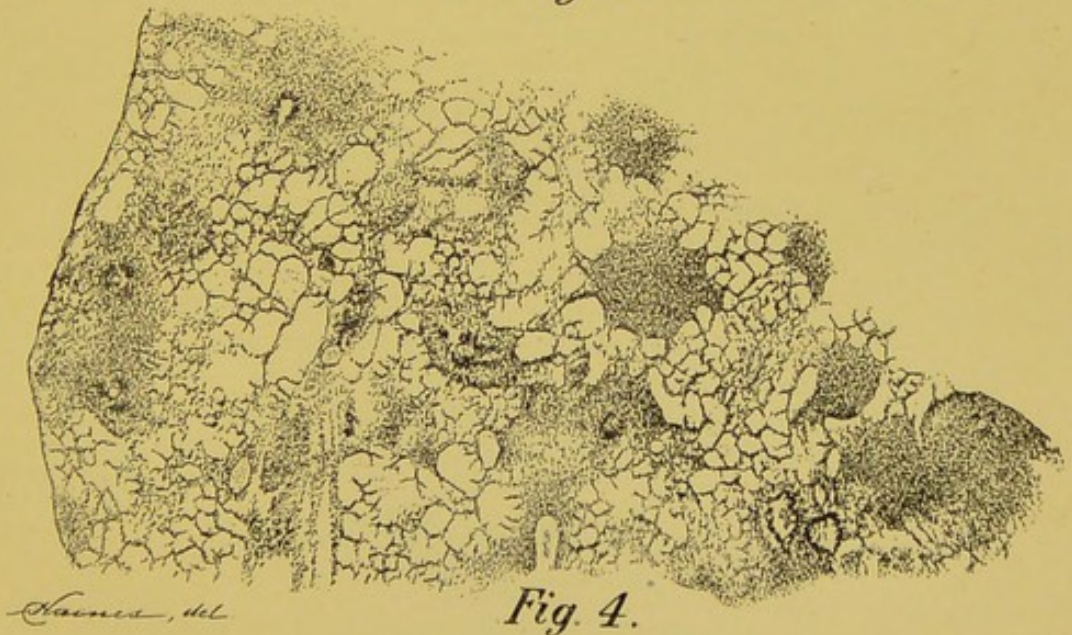


Fig. 4.

Tuberculosis in Swine.

another the demonstration of tuberculosis in milk-fed swine is deserving of careful consideration.

As pointed out by Nocard and others, and indicated in the history of the cases herein recorded, tuberculosis in swine is almost invariably found in animals fed on milk from tuberculous cows or the viscera of affected animals. Again, swine are rarely fed exclusively upon the milk of tuberculous cows or diseased offal, but upon a mixed diet of infected and wholesome food. There may be somewhat similar conditions in feeding children. They almost invariably, excepting when the practice of using milk from a single cow is followed, are fed the mixed milk from the dairy which may contain one or more tuberculous cows. According to statistics the number of swine affected with this disease in some localities is about 1.5 per cent, and owing to the allotted brevity of a pig's life they are at the longest fed upon infected food for a few months only, whereas human subjects are consumers of milk for many years. It is estimated further that one person out of every seven born dies of tuberculosis, and a considerable number of these die while children and while being fed on unsterilized mixed milk. This indicates that tuberculous cows should be excluded from the dairy and from the meat block excepting in the very earliest stages of the disease. In Germany the meat of animals slightly affected with tuberculosis is sold in the public market, but in a separate stall, at a reduced price. It is confidently believed that with this source of infection eliminated the amount of tuberculosis, not only in swine but also in the human species, would be greatly diminished.

The fact has already been established by repeated cultivations and inoculation experiments that whatever its origin, the bacillus of porcine tuberculosis has always been found to be the same and identical with the bacillus of the tuberculosis of other mammals. Different varieties or races of this organism may be found, but at the present time it is necessary to look upon them as belonging to the same species, whether found in swine, cattle, or man, and if transmitted under favorable conditions in food or otherwise to be capable of producing tuberculosis in any of these species and in still others. Knowing, as we do, the specific nature of the disease, it is evident to the most skeptical reader that if the cause—tubercle bacilli—is destroyed or removed altogether the disease will not appear. Another point worthy of consideration is the increased danger from the consumption of meat from tuberculous swine over that of tuberculous cattle. This is due to its greater tendency to become generalized in the porcine tribe. In localities or countries where uncooked pork is consumed the danger is of course correspondingly increased. Generally speaking, therefore, while the local meat inspector may be justified in being indulgent in the case of tuberculosis in cattle where the animals are in good condition and the lesions are restricted to one or two of the lymphatic glands, he should be very strict with the meat of swine affected with this disease. The facts elicited emphasize the necessity for the enforcement of more rigid laws for the prevention of the spread of this most common and dreaded disease.

DESCRIPTION OF PLATES.

PLATE XV.

Tuberculous spleen and liver.

Fig. 1. A photograph of the spleen of pig No. 3, showing the large number of tubercles.

2. A photograph of the liver of pig No. 3, showing the small tubercles on the surface. Photographs made by Dr. Pierre A. Fish.

PLATE XVI.

The right lung of Pig No. 3.

The dark parts (*a*) involving the cephalic, ventral, and a portion of the principal lobes were in a state of complete caseation. In the areas marked (*b*) the pleura and inter alveolar tissue was infiltrated. On the dorsal portion of the principal lobe there was a marked thickening of the interlobular (*c*) and to a limited extent of the interalveolar tissue. The light areas represent the apparently normal tissue.

(Three-fourths natural size.)

PLATE XVII.

Tubercular glands, tubercles, and tubercle bacilli.

Fig. 1. Section of a mesenteric gland infiltrated with tuberculous material. From pig No. 3. (Natural size.)
2. Tubercle bacilli in a single field from a cover-glass preparation from the cut surface of the mesenteric gland (fig. 1) stained after Zhiel-Neelson's method. (× 1000.)
3. A drawing of a section of the trachea near its bifurcation with much enlarged lymphatic glands. Pig No. 3. (Natural size.)
4. A short segment of a coil of the large intestine, showing numerous miliary tubercles on the mesentery covering it. (Natural size.)

PLATE XVIII.

Sections of tuberculous spleen, liver, and lung.

Fig. 1. Section of the spleen of pig No. 3, showing fibrous nature of tubercle with foci of cell infiltration and calcareous deposits. Traced with camera lucida. (× about 15.)
2. Sections of liver of pig No. 3, illustrating the normal and diseased areas (*a*) tubercular (*b*) normal. Traced with camera lucida. (× about 15.)
3. A giant cell from the liver of pig No. 3. Drawing made with Zeiss 2 mm. apochromatic objective and No. 6 ocular. (× 1000.)
4. A drawing of lung of pig No. 3, showing tubercles with foci of calcareous deposit. Traced with camera lucida. (× about 19.)

NOTE: Since this report went to press the remaining hogs in the herd of Mr. D. (Outbreak 2) were killed for pork. One of us (Dawson) was present. Although the animals appeared to be well, the post-mortem examination showed extensive tubercular lesions in every animal.

OUTBREAK OF A NONSPECIFIC DISEASE AMONG SWINE.

By VERANUS A. MOORE, B. S., M. D.,
Chief of Division of Animal Pathology.

The investigation of a disease among swine at Brookville, Md., in the spring of 1893 is here reported, as it affords a good illustration of a class of outbreaks popularly called hog cholera, and which can not be attributed to the effect of recognized pathogenic bacteria. In the annual reports of this Bureau several outbreaks of hog cholera and swine plague have been described, which show that these diseases exist in variously modified forms. The supposition was long since made by Dr. Theobald Smith[1] that epizootics of swine disease, falsely called hog cholera, and which are produced by certain unsanitary conditions, may also occur.

A considerable number of investigations into the nature of outbreaks among swine, popularly called hog cholera or swine plague, have been made, in which the presence of the specific bacteria of these diseases could not be detected. These have been more especially in the States of Iowa and Illinois. Unfortunately the greater number of these examinations were made in connection with other, at the time more urgent, inquiries, and consequently the results obtained were not based upon extended investigations. The bacteriological examinations, however, in several of these cases were sufficient in number and made under conditions that would have enabled a diagnosis of hog cholera or swine plague to be made had the specific bacteria of either of these diseases been present in the organs of the animals in the numbers in which they have almost invariably been found in genuine cases of these affections. In one of these outbreaks the disease was shown to be amenable to very simple hygienic treatment.[2]

The facts which have been collected point to the necessity for an early diagnosis of epizootics of swine disease in order that the proper methods of treatment may be applied or the necessary precautions taken to prevent the further spread of the disease should it prove to be of a specific bacterial origin. The facts at hand suggest the probability of the existence of a large number of destructive enzootics among swine which are not contagious, and, consequently, limited in

[1] Annual Report of the Bureau of Animal Industry, 1889.

[2] This outbreak occurred near Mazon, Ill., in August, 1893. When found it was assuming a serious nature, as many of the pigs (shoats) were dying daily. They were suffering from diarrhea, and upon post-mortem examination ulcers were found in the intestines. They were being fed abundantly on snapped corn, which was still soft (in the milk). The suggestion was made that the food be changed and the sanitary conditions generally improved. The suggestion was carried out with most excellent results in not only stopping the spread of the malady, but in restoring to health many of the sick animals. This is sufficient proof that the disease was produced by causes other than virulent hog-cholera bacteria.

their spread to the extent of the distribution of the causes which produce them. What these causes are can be determined only by careful investigations in the field, where all of the conditions surrounding the animals can be studied. It is a significant fact, however, that these outbreaks appear to occur more frequently in the West, where large herds of swine follow the corn-fed cattle, and where little or no attention is given to their food or shelter. In the East these animals are better cared for, and the losses from the so-called infectious diseases are much less.

The study of these apparently nonspecific outbreaks have not been sufficiently numerous or thorough to enable an enrollment of the various causes which may lead to their development. The study of the outbreak about to be described illustrates the difficulties attending the determination of the etiological factors in this class of cases. It also furnishes additional evidence of the existence of a false hog cholera produced by temporary and local causes.

OUTBREAK OF SWINE DISEASE AT BROOKVILLE, MD.

In the early part of May, 1893, a report of an outbreak of swine disease at Brookville, Md., was received and on May 8 the infected herd was visited in company with Dr. Theobald Smith. From the owner of the animals, Mr. H., we learned that he had lost about 13 pigs during the preceding four weeks from a disease which he designated as hog cholera. He had remaining 11 animals, consisting of an old sow, 2 shoats, and 8 pigs about 9 weeks old. Of these the sow and 4 of the pigs were suffering from the disease. The shoats had been quite sick, but they had nearly recovered.

The symptoms manifested by the sick animals were emaciation, swaying of the hind legs, slow movement, dullness, and a refusal of food. The owner reported that they had suffered from diarrhea. The sow had been sick for about three weeks and the pigs for about ten days.

It was thought by Mr. H. that the virus of the disease had been brought from a neighbor's farm. The facts to support this supposition were that a neighbor had lost a hog in March and dogs had brought parts of the dead animal into the field occupied by his pigs. The neighbor lost but the one animal. There had been hog cholera in the neighborhood during the fall and early winter, but Mr. H. stated that there had been no hog cholera or swine plague on his farm for several years prior to this. The pigs that had perished were said to have manifested symptoms similar to those which we saw in the living animals.

At the time of our visit the small pigs were housed in pens with board floors, but the older animals were running at will in the yards. The four sick pigs were in an apartment by themselves. They were fed on cooked ground rye and kitchen slop. They had no milk. The history and existing conditions indicated a somewhat mild outbreak of hog cholera, and to make sure of the diagnosis one of the sick pigs was killed for examination (see post-mortem notes, pig No. 140), and cultures were made from the various tissues and pieces of the organs were preserved for microscopical examination. It was our opinion that the animals were suffering from a somewhat mild or modified form of hog cholera. The cultures, however, excepting those that were made from a small area of hepatization in the right lung, remained clear. These contained saprophytic bacteria.

The failure to discover the pathogenic bacteria in the cultures from the first pig led to further examinations. May 15 the remaining three sick small pigs were brought to the laboratory by the owner. They appeared to be somewhat improved and the sow was reported as doing well. Since our previous visit and in accordance with Dr. Smith's suggestion the pigs had been allowed to run in an orchard. The three which were brought to the laboratory were taken to the experiment station and placed in a pen together.

May 17, pig No. 141 was found dead. The lesions indicated a mild case of chronic hog cholera. Tubes of culture media were inoculated from the various organs and exudates. Several of these remained clear, while others developed into cultures of different kinds of bacteria, mostly those belonging to the colon group. Hog-cholera bacteria could not be found. The condition of the other two pigs did not appear to change. They were killed for examination on May 24 and June 2, respectively.

A very careful bacteriological examination was made of the various organs, including the base of a few ulcers and the contents of a few closed infiltrated follicles in the intestines of pig No. 142, and a few of the infiltrated but closed follicles of pig No. 143. In all of these cultures and in rabbit inoculations with portions of the ulcers, hog-cholera bacteria were not detected. In fact, the cultures that were made from the organs which almost universally contain the specific bacteria in cases of true hog cholera remained clear. The great care with which these pigs were examined and the precautions that were taken in making the cultures seemed sufficient to insure the detection of the hog-cholera bacillus if present. The microscopical examination of the various tissues that were hardened in alcohol and Müller's fluid exhibited very few structural changes excepting the ulcers and these were not of diagnostic value. Although it may be possible not to detect hog-cholera bacteria in the tissues of pigs suffering with a chronic form of the disease, the organs from animals that died of the disease, or were killed within two weeks after they first showed signs of it, would be expected, in accordance with our present knowledge of hog cholera, to contain the specific organism.

In order that the nature of this disease may be more clearly understood and its resemblance and dissimilarities to hog cholera more fully indicated, the post mortem notes of the four animals examined are appended:

POST-MORTEM NOTES.

Pig No. 140.—May 8, 1893. Pig about 2 months old; weight, 20 pounds. Killed by a blow on the head and bleeding. The temperature was not taken. The limbs were mottled with indistinct rose-colored areas.

Lips and tongue normal, on the soft palate a yellowish, cheesy excrescence about 5 mm. in diameter, removable, leaving a depression. No hyperæmia or infiltration. The liver was very pale, bile thick and flaky. Spleen apparently normal. The mucosa of the stomach and intestines were very pale. In the colon a small number of petechiæ, contents markedly yellow, soft, excepting in the lower colon and rectum, where the feces were lumped into rather firm balls. Kidneys, the cortex sprinkled with ecchymoses varying from 1 to 1.5 mm. in diameter and separated by about 3 mm. The parenchyma very pale. A small quantity of pale, clear urine in the bladder.

Lungs: In one of the principal lobes a small region of collapse and beginning pneumonia. The associated air tubes were plugged with mucus. Lymphatic glands enlarged and pale; on section they were whitish and somewhat œdematous.

Cover-glass preparations of the blood from axillary veins, heated and stained, exhibited no change in the red blood corpuscles, and an increase in the numbers of

white cells could not be detected. A microscopical examination of sections of the liver and kidney cut from the fresh tissue showed changes in the parenchyma.

Bacteriological examination.—At the autopsy cover-glass preparations were made from the different organs and subsequently stained and examined for bacteria, but with negative results. Two agar tubes were inoculated with bits of the kidney and one with three loops of the heart blood at the time of examination. The organs were brought to the laboratory in anatomical jars and sterile bottles and placed in a cool box. The following day tubes of bouillon were inoculated with bits of the kidney, liver, and hepatized lung, and agar tubes were inoculated from the spleen, and agar plates were made from the diseased lung tissue. The cultures remained clear excepting those from the lung tissue. From those two species of bacteria were isolated. Upon cultivation they were found to be commonly distributed saprophytic bacteria.

Pig No. 141.—Found dead May 17, 1893. A small, black pig 2½ months old; weight, about 20 pounds. There were a few adult lice (hæmatopium suis) and many eggs of the same species on ventral surface of the body. A slight reddening of the skin around the feet and along the median line on the abdomen.

On the left side of the tongue about 2 inches from the tip there was a minute necrotic area surrounded by hemorrhages. In the abdominal cavity about 350 c. c. of a turbid, reddish-yellow fluid, which coagulated into a jelly-like mass within fifteen minutes after its removal. The large intestines were covered with a delicate exudate, greenish and viscid. It existed in larger quantities between the coils and shreds of an elastic character stretched from the intestines to the abdominal wall. The entire peritoneum was inflamed, over the large intestine it had a dull, opaque appearance, and was sparsely sprinkled with minute hemorrhages; likewise over the bladder and abdominal wall. The stomach contained a small quantity of bile-stained fluid and a dead ascaris. The mucosa in the cardiac portion was sprinkled with punctiform hemorrhages. In general the mucosa was slightly reddened. In the lower half of the small intestine there were a considerable number of subserous hemorrhagic points. In the ileum, 18 inches above the ileocæcal valve, the mucosa was necrosed entirely around the gut for a distance of about 5 cm. This appeared to be the oldest lesion in the intestine. There was one other old ulcer on a Peyer's patch. The mucosa was thickened over several small areas. In the large intestine there were dull, whitish areas, which showed through the serosa. The contents were firm and covered with mucus. Beginning necrosis and pigmentation of the gland at the ileocæcal valve. Trichocephalus in cæcum. For a distance of 2 feet above the cæcum the mucosa was more or less pigmented, glistening, and studded with a large number of flattish, yellow necroses, which were slightly elevated, the largest being about 5 mm. in diameter, and having a central depression which was of a darker color. The smallest ulcers were barely visible to the unaided eye. These ulcers were densely crowded. About 2 feet from the anus the mucosa contained but few necrotic areas. The mucous membrane had a glistening appearance. Within the intestine was a long band of gelatinous, cohesive, whitish fibrin, about 5 mm. in diameter.

There was a slight fibrinous exudate on the upper surface of the liver which was easily pulled off. Parenchyma pale. The entire surface was sprinkled with minute yellowish points. The gall bladder contained a thick bile, holding considerable flaky matter in suspension. The spleen was much enlarged, dark purplish color, pulp soft. Malpighian bodies not visible. Kidneys considerably enlarged, capsules easily removed. The ventral surfaces were dark and mottled, due to isolated and confluent hemorrhages. Cortex thickened, parenchyma pale, sprinkled with ecchymosis varying from mere points to 3 mm. in diameter. The pyramids were pale. A smaller number of ecchymoses on dorsal surfaces.

The submaxillary, parotid, inguinal, and kneefold glands were enlarged and presented a mottled reddish, bluish appearance from the outside. Some of the lobules were bluish red, others dark red, and still others were of a pinkish-red color. On section the cortex and interlobular tissue appeared as hemorrhagic lines. In some lobules a greater portion of the parenchyma was hemorrhagic. The cortex of the mesenteric glands was hemorrhagic. Mesocolic glands were mottled with punctiform hemorrhages. Posterior mediastinal glands hemorrhagic.

In the right pleural cavity there were about 75 c. c. of a turbid blood-stained fluid containing some small gelatinous flakes. In the left pleural cavity there was about 15 c. c. of a reddish-yellow fluid. The fluid coagulated into a jelly-like substance within fifteen minutes after its removal. The pulmonary and pericardial pleura were covered everywhere by a delicate exudate in the form of closely set grayish points. Near the sternum on the right costal pleura were hemorrhagic areas. The dependent half of both ventral and cephalic lobes, a small portion of the adjacent right principal lobe, and the cephalic half of the

median lobe were hepatized. There was a roughened membranous exudate over the diseased portion of the right lung and right side of the pericardium and a roughened condition of the remaining diseased portion of the lungs. The exudate appeared below a certain horizontal line on both lungs. The tip of the principal lobe was hepatized. On the thoracic aspect of the diaphragm were several blood extravasations. Much muco-pus in trachea and bronchi. Left principal lobe not hepatized. There was more or less interlobular œdema of a gelatinous character in the hepatized areas. On section the hepatized portions were of a uniform dull red color, with an occasional area showing paler mottling. The tip of the right lung appeared to be the most advanced. No lung worms were discovered. More or less interlobular œdema in the otherwise healthy portion of the lungs.

The auricles of the heart were hemorrhagic, more especially the right. Epicardium roughened.

Bacteriological examination.—At the autopsy cover-glass preparations were made from the different organs and exudates. These were properly heated, stained, and examined for bacteria. Those from the peritoneal exudate, liver, spleen, and kidneys exhibited no bacteria, but those from the pleural exudate and hepatized lung tissue showed a few small bacilli with ends rounded. Agar or bouillon tubes (usually both) were inoculated with the exudates and bits of the various organs. Agar plates were made from the lung tissue and a rabbit inoculated subcutaneously with a piece of the hepatized lung. The cultures that were made from the blood, spleen, and parotid glands remained clear. The others developed cultures of bacteria which resembled each other very closely, and which were found to belong to the colon group of bacteria. Roll cultures in gelatin were made from the original cultures to isolate suspected different forms, and rabbits were inoculated with certain of the pure cultures. These gave negative results.

Pig No. 142.—Killed by a blow on the head, May 24, 1893. It was of the same age and of about the same weight as pig No. 141. There were considerable numbers of lice and eggs present, as in No. 140. A slight reddening of the skin over the abdomen and extremities. The post-mortem examination showed the mouth and pharynx to be normal. Stomach contained a small quantity of fermented food; the mucosa was pale; over the fundus it was covered with a somewhat whitish layer of mucus and sprinkled with punctiform reddish areas. In the upper two-thirds of the small intestine the mucosa was pale and of a dull, opaque appearance. The mucous membrane of the large intestine was covered with a thin layer of a blackish, earthy-appearing substance. After washing the mucosa had a pale bluish appearance, but otherwise it was in good condition. In the cæcum there was one ulcer about 10 cm. in diameter with a raised, rounded border and a central blackish slough, which on section showed as a yellowish necrotic mass about 2 mm. in thickness, extending to the muscular coat. On the ileocæcal valve were one similar and two smallar ulcers. In the colon just below the valve were about five ulcers, from 3 to 10 mm. in diameter, with infiltrated margins. In the remainder of the large intestine the follicles were enlarged and appeared as small elevations on the mucosa. They were from 1 to 3 cm. apart and about 3 mm. in diameter. To the touch they felt like small grains in the wall of the intestine. Upon section they were found to contain a yellowish caseous substance.

Liver: The surface of the liver had a peculiar variegated appearance. The lobules were distinctly outlined, the peripheral zone was pale, but varied considerably in different places. The bile was moderately thick and held considerable flaky material in suspension. In the common bile duct and passing into the liver were four living and one dead ascaradies. Spleen normal. Kidneys not much enlarged, the capsule easily removed. On section the cortex did not present its usual markings. Malpighian bodies were not visible. The cortex throughout had a grayish, pinkish-yellow appearance. Lymphatics were of a mottled pale bluish-red color. Upon section the cortex of some of the lobules appeared as irregular reddish lines. The entire section had a dirty red appearance.

The lungs were normal. The heart muscle was pale, and large, dark clots were found in both sides.

Bacteriological examination.—Cover-glass preparations from the liver exhibited a very few rod-shaped bacteria which usually appeared in twos. They were slightly larger than the hog-cholera bacillus. Similar preparations from the other organs showed no bacteria. Tubes of agar or bouillon (usually both) were inoculated with the blood (heart) and bits of the liver, spleen, and kidneys, and agar plates were made from the base of the ulcer, and four fermentation tubes containing bouillon plus glucose were inoculated with the same. A culture was also made from the caseous contents of one of the infiltrated follicles. The tubes that were inoculated with the blood, spleen, and kidney remained clear. The tube inoculated with the liver developed a pure culture of *bacillus coli communis.*

The plate culture developed very few colonies of a motile bacillus which was considerably larger than the bacillus of hog cholera. The fermentation tubes inoculated from the ulcer were less satisfactory. The fermentation was slow, and they were not examined until it had ceased. The quantity of gas that was produced varied from 0.5 to 0.8 of the contents of the tube. Subcultures were negative excepting in two cases. These gave pure cultures of nonmotile slender bacilli. The tube inoculated from the infiltrated follicle developed a pure culture of a nonmotile bacillus which did not belong to the colon group of bacteria.

Rabbit No. 18 was inoculated subcutaneously on the side of the abdomen May 24 with a piece of the base of the ulcer from the ileo-cæcal valve. The necrosed slough was scraped away and the bottom of the ulcer washed thoroughly with sterilized water before a portion was introduced beneath the skin of the rabbit. The rabbit exhibited slight elevation of temperature, but no other symptoms of disease. It was killed May 31. There were no lesions excepting a small closed abscess at the point of inoculation. From this lesion agar plate cultures were made. These developed colonies of a nonmotile bacillus.

Rabbit No. 17 was inoculated May 24 with the base of the ulcer from the cæcum. The ulcer was prepared in the same manner as in the previously described case. There was no elevation of temperature. The rabbit was killed June 2. Agar plate cultures were made from the contents of a small closed abscess at the point of inoculation. These developed colonies of *bacillus coli communis*.

Pig No. 143.—June 2, 1893, this pig appeared to be somewhat improved. It was killed for examination by a blow on the head. There was no discoloration of the skin; a very small quantity of subcutaneous fat. On the base of the tongue over the entire soft palate were closely set yellowish dots which could be easily removed from the tongue, leaving small pits or depressions. On the soft palate they seemed to be associated with a peculiar opaque, pale, yellow necrotic condition of the entire palate. Stomach was nearly empty; the mucosa more or less bile stained. Around the cardiac orifice there was a thin, yellowish, easily removable layer composed entirely of mucus. When scraped away the mucosa underneath appeared to be healthy. The small intestine appeared to be normal. Contents of large intestine of blackish color; normal in consistency. In the cæcum were four small superficial ulcers of a yellowish color. Mucosa faintly mottled. Peyer's patch at the base of the ileo-cæcal valve occupied by two large blackish necrotic masses and a number of small, blackish, follicular ulcers. The neoplasm fully 4 mm. thick, yellowish and firm. In the lower half of the large intestine the mucous membrane was superficially necrosed, and in addition a small number of ulcers varying in size up to 1 cm. in diameter. Throughout the large intestine, excepting in cæcum and upper 50 cm. of colon, where they were absent, the follicles were enlarged and presented a yellowish appearance from the serous surface. In the lower colon the center of the follicles were eroded, and a small cheesy mass could be pressed out. The liver showed on its surface patches of faint bluish mottling, indicating a thickening of the capsule and interlobular tissue.

The bile was of a bright green color and held a small quantity of flakes in suspension. The common bile duct was very much distended by a dead ascaris, which was partially coiled up, one end of which extended into one of the liver ducts. Spleen nearly normal in size, of a pale red color, flabby. Malpighian bodies indistinct. Kidneys somewhat enlarged (9 cm. long and 4 cm. wide), weighing 68 and 69 grams, respectively. The tissue about the hilum and within the pelvis of the left kidney was somewhat œdematous. Externally the kidney exhibited a mottled, bluish-gray appearance, sparsely sprinkled with dark red points. Capsule easily removed. On section the cortex had a pale bluish-gray appearance, the normal markings were effaced, and distended veins were visible. Striæ of the medullary portion varied from a dark to a pale red. The parotid glands considerably enlarged and exhibited in some lobules groups of minute reddish points. Other glands normal in appearance.

The lungs showed small areas of reddish collapse in right ventral and principal lobes; trachea and bronchi were pale and contained a small quantity of mucus. Heart normal.

Bacteriological examination.—Cover-glass preparations made from the various organs showed no bacteria excepting in those from the intestine, where several species were observed. Tubes of agar and bouillon were inoculated with bits of the parotid gland, liver, spleen, kidney, and pus from closed follicle in lower colon. The one used from the follicle developed into a pure culture of *bacillus coli communis*. All of the other tubes remained clear.

Summary of the bacteriological examinations of the four pigs.

Pig No.	Blood.	Organs from which cultures were made.		
		Lungs.	Exudates.	
			Pleural.	Peritoneal.
140*a*	Clear	2 bacilli		Micrococcus.
141*b*	do	*B. coli communis*	*B. coli communis*	
142*a*	do			
143*a*				

Pig No.	Organs from which cultures were made.					
	Liver.	Spleen.	Kidney.	Lymphatic glands.	Intestines.	
					Ulcer.	Follicles (closed).
140*a*	Clear	Clear	Clear	Clear		
141*b*	*B. coli communis.*	do	*B. coli communis.*	do	*B. coli communis*	
142*a*	do	do	Clear	do	*B. coli communis;* also 4 other species of bacteria.*c*	2 species of bacteria (bacilli).
143*a*	Clear	do	do	do		*B. coli communis* and 1 other species.

a Killed.
b Died.
c Several of the species other than *B. coli communis* were obtained from the local lesions of rabbits inoculated with the diseased organs.

In considering the result of the bacteriological examination attention should be called to the interesting fact that all of the cultures made from the blood, spleen, and lymphatic glands (other than follicular) remained clear. This is of special significance in the case of pigs Nos. 140 and 141, as No. 140 was killed while sick, and about ten days, according to the statement of the owner, after it was first attacked, and No. 141 died after about two weeks' sickness.

The large number of colon bacteria isolated from the organs of pig No. 141 suggest a possible casual relation between them and the cause of death. The more probable explanation is that the weakened condition of the body allowed an extensive invasion of this organism into the internal organs from the different mucous membranes. However, the quite general infection indicates a possible secondary relation between the colon bacteria and the cause of death in this animal. It is somewhat curious that the peritoneal exudate did not contain this organism. The micrococcus which was isolated existed in considerable numbers, as indicated by the colonies, and is of interest on account of the frequency with which it has been found in diseased organs of this and other species of animals.[1]

The presence of the colon bacillus in cultures made from the liver and ulcer of pig No. 142 and the one from the closed follicle in No. 143 can not be considered as pointing to their etiological importance. The fact, however, must not be overlooked that while colon bacteria are not uncommon in the organs of pigs which have been sick for

[1] This micrococcus has appeared in pure cultures from the organs of cattle, swine, turkeys, and fowls. In several instances its etiological relation to the lesions with which it was associated seemed more than probable, but inoculation experiments have not shown it to possess any pathogenic properties. It has a peculiar porcelain-white appearance when grown on agar. It liquefies gelatine quite rapidly, forming a vigorous whitish growth on the surface of the liquefied medium.

some time, or when there are ulcers or abrasions of the mucous membranes, they are occasionally found under such conditions that their pathogenic significance is highly probable. The literature upon the disease-producing power of the colon group of bacteria is becoming quite voluminous. Only recently Lignière has discovered a septicæmia in fowls due to this organism. The other species of bacteria isolated from the various organs, but largely from the ulcers in the intestines of the different pigs, were recognized as species belonging to or at least common in the intestinal flora.

The probability of the presence of attenuated hog-cholera bacteria or other pathogenic organisms led to the inoculation of rabbits with pieces of certain of the deceased organs, especially from pig No. 141. As these did not die certain of them were subsequently chloroformed and cultures made from the local lesions. In these cases the local infiltrations were very slight.[1]

Inoculation of rabbits with the tissues and cultures from pigs Nos. 141 to 143.

Rabbit No.	Date of inoculation.	Method of inoculation.	Inoculated with—	Remarks.
	1893			
497	May 18	Subcutaneously	Hepatized lung, pig 141.	Rabbit died June 6 from injury received in handling.
14	May 19	Intravenously	0.5 c. c. bouillon culture (*B. coli communis*), lung, pig 141.	Chloroformed for examination June 5. Emaciated. No bacteria.
23	May 25	do	0.5 c. c. bouillon culture (*B. coli communis*), kidney, pig 141.	Chloroformed for examination June 22. Emaciated. No other lesion; no bacteria.
26	June 1	do	0.5 c. c. bouillon culture (*B. coli communis*), liver, pig 141.	Rabbit remained well.
18	May 24	Subcutaneously	Ulcer ileo-cæcal valve, pig 142.	Chloroformed May 31. Slight local lesion; otherwise normal. A nonmotile organism obtained in pure cultures.
17	do	do	Ulcer cæcum, pig 142.	Chloroformed June 1. Local abscess from which a small bacillus was isolated.
21	May 27	Intravenously	1.50 c. c. bouillon culture (*B. coli communis*), liver, pig 142.	Remained well.
32	June 8	Subcutaneously	Ulcer soft palate, pig 143.	Do.

The emaciation of rabbits Nos. 14 and 23 was undoubtedly due to the effect of the inoculation, although no bacteria were found in the stained cover-glass preparations from the organs or in cultures made from the same. Such results have been obtained in several instances where rabbits were inoculated intravenously with a moderately large quantity of cultures of bacteria which were not known to possess pathogenic properties.

Returning to the lesions found in the pigs, we find that anæmia, infiltrated follicles, and serous exudates in pig 141 were the most pronounced abnormal conditions. Although the history of the previous cases and certain of the lesions found in the animals examined

[1] The method of subcutaneous inoculation of rabbits or guinea pigs with pieces of the base of the ulcer or other tissues supposed to contain the disease-producing organism and killing them in from five to seven days afterwards, and making agar plate cultures from the pus in the lesion produced by the inoculation has been found to be very effectual in the isolation of hog-cholera and swine-plague bacteria when they existed in small numbers or in an attenuated condition. Saprophytic forms that are liable to be present are soon destroyed in the living tissue of the animal, but the more parasitic forms will survive and can be isolated by means of agar plate cultures from the local lesions.

indicated the presence of hog cholera, the failure to find the specific organism in the bacteriological examination of the four animals mentioned, together with the facts that the majority of the pigs in this outbreak which died were less than eight weeks old and that the shoats recovered, suggested the probability that the animals in this outbreak did not die from the effect of the hog-cholera bacillus. It appears to be an excellent example of an outbreak of false hog cholera. The importance of the investigation of this outbreak of swine disease lies in the fact that it has afforded an illustration of the existence of a disease unquestionably due to local conditions, presumably to some irregularity in the food or environments. The only reason for its being called hog cholera was the loss of several animals in rapid succession. A serious result of this popular error in diagnosis rests in the fact that when once convinced that an infectious disease exists in his flocks or herds the farmer usually accepts the condition much after the style of the fatalist, and thus neglects to look after the surrounding conditions which might be the cause of the trouble and which might be easily removed. Whenever disease exists every effort possible should be made to determine its cause and nature and to adopt such methods as are known to prevent its further spread. Although the loss of animals is the same whether they die from a specific bacterial disease or improper food and exposure, the number of animals which die may be materially influenced by looking after the sanitary conditions under which they are kept. The usual experience in the investigation of outbreaks among swine has been to find obscure diseases not positively traceable to any specific organism, or else very chronic or more acute cases of hog cholera or swine plague. Further investigation of the diseases of these animals is to be recommended. These inquiries should be broad in their scope, and should be made in different parts of the country and at different seasons of the year in order to bring together the numerous exciting and more remote causes which may lead to an unthrifty or diseased condition of swine.

LEECHES.

A Histological Investigation of Two Cases of an Equine Mycosis, with a Historical Account of a Supposed Similar Disease, Called Bursattee, Occurring in India.

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INTRODUCTION.

"Leeches," or "leeching," is the local name of a disease in Florida quite prevalent among the horse kind at certain seasons of the year. The same disease has been located in other parts of the United States, but not under the same name. The fluke disease of cattle caused by *Fasciola hepatica* (*Distoma hepatica*) is also known under the name of "leeches," but does not properly come within the scope of this paper.

It was the common though erroneous belief that the horse disease was due to the leeches which infest the numerous small lakes and ponds, into which the horses go to eat the grass, sometimes so deeply that their backs are submerged. According to some writers, the parts of the body which come in contact with the water seemed to be the most frequent seat of the lesions.

The disease has been but little investigated in this country, and some difference of opinion exists among those who have written upon it. One writer (Bitting) identifies it as cancer (round-celled sarcoma); another as a disease known as "bursattee," quite prevalent in India, and still others who do not classify it at all. It has been held by a few, among whom is Hart, followed by Burke, that the disease in India is like cancer. The cancer theory, however, does not seem to be generally accepted. It is commonly assumed that the form in the United States is the same as that in India.

The following list shows the variations of the names met with in the description of this disease: Barsati, barsáti, barsattee, barsatti, bausette, bursati, bursatie, bursatti, bursautee, bursautie, bursauttie, bursottee, burusatee, and burusauttee. Bursattee seems to be the term most used of late, and is therefore retained in this article with reference to the disease existing in India. These names have been derived from the Indian word burus or bursat, meaning rain or rain sore, it having been supposed that the malady was associated with the rainy season.

The disease in this country has not attracted very much attention, nor has it been considered of great economic importance. An explanation for this fact may be offered on account of its seemingly noncontagious character, and that in this country it has been thought to be

confined to comparatively limited areas, and that the animals, although infected, may yet be utilized for some purposes. On account of the chronic course of the disease the affected animals become such eyesores that from a sentiment of mercy they are often killed before the disease can terminate fatally.

Although this affection presents many points of similarity to the one found in India, the question of their complete identity ought to be held in abeyance until a more thorough investigation of the two can be made.

In the present article it is the aim to follow the disease historically by bringing together the available literature on the subject and to describe the lesions histologically with as much completeness as possible. Further research into the etiology, course, and treatment is desirable, and, it is hoped, may be the basis of a future communication. The name "leeches" is retained because the material was received from Florida, where the name is in common usage, and because the matter of the identity of this with the disease in India is not yet unquestionably established. As the cause and course of the disease becomes more thoroughly known a more accurate and scientific name would be indicated.

HISTORICAL.

In 1829 James Kerr, a retired veterinary surgeon of the First Bengal Light Cavalry, communicated to the Veterinarian[1] a short article on "The disease to which horses are subject in the East Indies, termed bausette." Small tumors were found at the angle of the lips, face, and scrotum which, if let alone, suppurated and became bausette ulcers. The conclusion was that the heavy rains must be the predisposing cause; but whether the disease was produced by the atmospheric change or alteration undergone by the herbage he was unable to say. He notes that animals once affected with the disease are almost certain to have it recur. In his opinion it was a disease of poverty. No satisfactory information was obtained as to the contagious or infectious nature of the malady. The natives, however, were of the opinion that it was frequently communicated from diseased horses to healthy ones through the medium of flies.

During the year 1831 Veterinary Surgeon John Tombs, of the Bengal Artillery, contributed three short articles to the Veterinarian[2] on "The Indian burusatte." According to this writer the name is derived from *burus*, signifying rain; *burnu*, to rain; *burusana*, rainy; *burusat*, weather, rains; *burusatee*, a disease in horses.

The statement is made that some people claim to have traced the origin of the disease to Arab blood, and that horses whose sires were Arabians, and whose dams were country bred were highly susceptible, and the conclusion is reached that the predisposing cause may be foreign blood, at least foreign to India. The exciting causes may be the vicissitudes of the temperature of the atmosphere.

It is stated that the superficial absorbents of the body are affected and sometimes the deeper-seated ones, and eventually the lungs, which may, though rarely, become tuberculous (nodular). "It appears to be a kind of spurious farcy."

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[1] Vol. II, pp. 419–420.
[2] Vol. IV, pp. 541–542, 623–625, 665–668.

Charles Jackson, veterinary surgeon of the Eighth Regiment of Light Cavalry, in 1842, takes issue in the Veterinarian[1] with Mr. Armstrong in his description[2] of bursattee. Mr. Jackson believes that the disease is not the same as that of burusautee in the south of India. Burusautee, he states, is in fact fungus hæmatodes, differing somewhat from that disease in the human subject, but still bearing a sufficient resemblance to warrant him in concluding that it is of the same nature.

J. T. Hodgson[3] (1853) diagnosed "bursautee," as the name implies, a cancerous ulcer which is prevalent during the *bursaut* or rainy season, and is considered to be of two kinds—simple and curable, and malignant, supposed to be incurable, or if healed up liable to ulcerate again.

James Western[4] (1853, Horse Artillery, Madras) differs from Mr. Hodgson in his treatment by recommending the use of the cautery. A system of treatment offered by Capt. W. W. Apperly is criticised on the ground that it is so dilatory and repetitious that it lasts the whole length of the rainy season, and after that the sores seem to improve of themselves. Mr. Western's opinion is that it is essentially a disease of debility and therefore should be treated by tonics.

In 1872 and 1873 Mr. Robert Spooner Hart,[5] of Calcutta, wrote at some length upon bursattee, giving detailed descriptions of the symptoms, course of the disease and treatment, and also comparing it with other maladies which seemed to present certain points of analogy, such as Delhi boil and lupus. He believes the disease to be peculiar to the Tropics and noninfectious. He classifies it with an order designated by dermatologists as *tubercula*, not signifying tuberculosis. "Included under this head are elephantiasis, keloid, frambœsia, cancer, epithelioma, rodent ulcer, and I think we might with propriety include bursattee." He conceives that in the true bursattee there are three distinct forms—(*a*) the papillated bursattee ulcer, (*b*) the medium bursattee ulcer, (*c*) the phagedænic bursattee ulcer. True gland implication is not found, although in bad cases the lymphatic glands do become acutely inflamed. In two cases the disease is said to have terminated in "farcy."

Mr. Hart's inoculation experiments are interesting. The length of time the animals were under observation is not stated.

In experiment No. 1 exudative matter from the surface of a bursatic ulcer was taken and inserted under the skin of the same subject from which it was taken. The part became scabbed and healed in a few days.

In experiment No. 2 bursattee matter was taken from one subject and implanted under the skin of another bursatteed subject.

In No. 3 exudative matter was taken from an old bursatteed subject and inserted under the skin of a horse not the subject of bursattee. This, like the preceding experiments, threw no light upon the matter. The bursattee exudative matter was also administered internally to other bursatteed subjects. Some were likewise drenched with the blood of bursatteed animals without effect. Mr. Hart concludes with the belief that "although the microscope fails to demonstrate anything

[1] Vol. XV, pp. 257–258.
[2] Proceedings of the Veterinary Medical Association for 1838, p. 233.
[3] "Diseases of horses endemic to India," Veterinarian, XXVI, 121–123.
[4] "On bursautee, a disease of the horses of India," Veterinarian, XXVI, 425–430.
[5] Veterinarian, XLV, 607–610, 711–715, 917–921; XLVI, 17–21, 97–102, 532–536.

like cancer structure, still it must be admitted that the disease is wonderfully allied to that fatal affection."

Mr. F. F. Collins[1] (Bombay Presidency, 1874), in "Observations on bursottee," states that the parts of the animal most subject to the attack are those to which water is oftenest applied, viz, the legs and face.

"In horses that once show a bursottee diathesis an ordinary wound is alone sufficient to establish the disease." He believes it to be of a parasitic origin, and conceives the parasite to be of vegetable organization. Hard tissues are always avoided, but the softer structures prove a favorable locality for germination.

Maj. Robert E. Cane[2] (1876), in "Some remarks on hippopathology in India," emphasizes the importance of proper hygienic conditions for healthy as well as diseased animals, and urges a thorough reform of the then existing abuses. He believes that the disease is, in its origin, due to the deficient power or action of the capillary cutaneous nerves.

"Max"[3] (1876) asks, "What is bursattee?" and answers the question by inferring "that the disease is nothing more nor less than an ordinary wound aggravated by climatic influences." The fly theory of infection is supported. The result of his experience had been that "no so-called bursattee sore will resist treatment if you preserve it from the flies."

In a subeditorial pointing out certain resemblances between Delhi boil and bursattee, in the same volume of the Journal, page 362, it is stated that after ulceration has disintegrated the surface, mycelium or other low forms of organism may be present; but this, according to Fayer, is not the essential cause, but rather an accidental introduction from without.

In a "Few remarks relative to bursauttie," Mr. Stephen Knott[4] (1877) states that in his practice in Burmah, as well as in India, he has noticed that any little abrasion during a monsoon has a tendency to run into bursattee; that it is a spreading sore of a climatic phagadænic character. He thoroughly agrees with "Max" in the supposition that flies do carry poison to wounds and abrasions by deposition of fæcal and decomposing matter from latrines and other sources which, when the wounds are influenced by a moist temperature, result in bursattee. He has met with chronic cases that continue all the year round, and which, when the rainy season has passed, improved but very little. Milder cases, however, get well when the rains cease, but usually reappear during the next monsoon.

"Ubique"[5] (1877) does not think bursattee is caused by an overabundance of acid in the blood or that it is analagous to syphilis or scrofula, as some veterinarians seem to think, but believes that it is a blood disease peculiar to the hot season, and renders the skin and mucosa, where it most approaches to the nature of the skin, unduly disposed to chancrous ulceration.

He believes that Dr. Fayer's theory of abnormal cell growth, with subsequent degeneration in "Delhi boil," to be the most probably correct one yet advanced for bursattee. In the matter of treatment "considerable success was obtained by administering iodide of potassium until the system becomes completely under its influence. * * * Without exception this abominable pest is the greatest nuisance to horses and their masters that possibly could be."

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[1] Veterinarian, XLVII, 791–792.
[2] Veterinarian, XLIX, 376–384.
[3] Vet. Journal, III, 338–344.
[4] Vet. Journal, IV, 182–184.
[5] Vet. Journal, V, 25–27.

Mr. J. J. Meyrick[1] (1878) almost invariably finds bursattee breaking out in the dry, hot weather, some weeks or even months before the rains commence. He thinks that there is no doubt but that previously healthy horses often become affected, owing to something deposited by flies upon wounds, mucosa, or parts where the skin is thin. Where the wounds were treated daily with a thick mixture of sulphur and oil no flies would settle, and he has never known a wound so treated to assume a bursattic character. "When bursauttee has existed for a considerable time, the poison appears to be carried to other parts of the body. Sores will come in places where the skin is thick and where there have been no previous wounds."

In a case of a high caste Arab, 15 years old (stud), pieces of kunkur (nodular growth) were found as large as a pea or bean deposited under the whole pleural surface of both lungs. Many were fully a quarter of an inch deep in the parenchyma. He also found two or three pieces of kunkur on the lungs of another old Arab which had never shown any signs of bursattee. In these two horses not the slightest trace of pus or even of irritation was found in the parts of the lungs surrounding the lumps of kunkur.

Mr. F. Smith[2] (1879) gives a very interesting and intelligent account of bursattee. He states that kunkur is applied to an extremely prevalent form of limestone used in India for road making and other purposes. "This is of a French-gray color and occurs in nodular masses about the size of an egg. It was supposed the bursattee concretions were in some way related to this soil, but no doubt the name originated from the stony hardness and nodular character of the concretions. Opinions have differed much as to the nature of these bodies. Some consider them bony-osseous spiculi (Western); others attribute them to aggregation of scrofula cells and subsequent calcareous degenerations of the small mass so produced (Hodgson); others consider them simply inspissated pus (Phillips); while Mr. Meyrick (loc. cit.) states that sometimes they contain crystals of uric acid and oxalate of lime. This must be, however, in those of very long standing, for my researches indicate that they have an organized character."

Mr. Smith's inoculation experiments, although mostly negative, were interesting, in that one or two of them suggested the possibility of conveying the disease from one animal to another.

Portions of a bursattee tumor were introduced, in two places, into a horse similarly affected. The material was introduced subcutaneously in the lips and in the side. No appreciable effect was noted at the mouth, but at the wound in the side there was considerable swelling and great pain the following day; this gradually subsided, leaving behind considerable induration around the original wound.

A horse which had never had bursattee was similarly inoculated with entirely negative results.

A subcutaneous inoculation was also made into the ears of a foal with negative results. In several other cases negative results were obtained.

Perhaps the most important of Mr. Smith's inoculations were those on the human subject. Before the patients would submit, however, he was obliged to inoculate himself repeatedly in their presence, each time with a negative result.

1. Ten syces (native grooms) were inoculated in the arm with cells and pus from a bursattee in the ulcerative stage. Each man was

[1] Vet. Journal, VII, 318-321. [2] Vet. Journal, IX, 300-307, 383-393.

kept standing in the sun until the matter placed on the exposed surface of the dermis dried. Negative results in each case.

2. A salootree (native farrier) was similarly inoculated, and on the third day, feeling some tingling about the wound and not willing to become a martyr to science, he surreptitiously cauterized the place and spoiled the experiment.

A dog was inoculated upon the nose and ears with bursattee material and the result was negative.

One inference may be drawn from these experiments, in that the disease, if inoculable at all, requires certain and peculiar conditions of the patient's system or that the material to be inoculated must have attained a certain stage of development in order to produce pathologic effects. Another point that might be considered is that the material thus inoculated may not produce immediate effects, but remain latent until a succeeding season with favorable conditions to call it into action.

Some of the well water used by the men and horses was subjected to examination. The water was clear, but contained particles just visible to the naked eye. Under a low power of the microscope it was found to contain fungi, epithelioid cells, animal organisms, fibers, and pigment granules (black and yellow). With a high power the fungi were found to be numerous, with a tendency to increase in size and combine in masses. The epithelioid cells were of a bright yellow color and showed nuclei and nucleoli, the largest containing other cells, some of which were elongated and nucleated.

A chemical analysis was also made of the water, the filtered, curiously enough, showing a greater proportion of deleterious substances than the unfiltered.

Reference was made to points of similarity in "Delhi boil," as well as to some important histologic conditions found, which may be referred to later.

Mr. George Oliphant[1] (1880), in his article on bursattee, confines his attention chiefly to the so-called kunkur, which he believes to be the exciting cause of the ulceration, although its exact nature is not definitely known.

The ulceration is an effort of nature to eliminate this matter, and if the deposit was confined to the skin alone the effort would probably be more frequently successful.

The kunkur appears to consist of animal matter, containing deposits of salts. When fresh it cuts like cartilage and when dried is distinctly gritty. In all these points it is very hard, like tubercle, but I have failed so far to trace in it one peculiarity of that diseased process, namely, softening.

A gentle squeezing is sufficient to remove the tubercle-like matter in portions of varying size. On removal of these, or on their natural ejection by the process of ulceration, ragged ulcerous cavities are left, which are speedily filled up by the rank granulations or fungoid growths. Other ulcers may form around the original sore, and, as the parts are very itchy, the skin becomes abraded by the biting or rubbing of the animals and takes on an unhealthy action. A high temperature seems to be necessary to the development of the disease, and combined with moisture it becomes very much aggravated.

In 1880 Mr. Richard W. Burke, then a student in the Dick Veterinary College, interested himself in bursattee and has written quite voluminously upon it since, including several controversial letters.

[1] Vet. Journal, XI, 16-21.

In the Journal of Comparative Medicine and Surgery, 1886,[1] he states: "It is not necessary to remark that my observations led me to state as early as 1880 (Veterinary Journal) that this disease was cancer of the horse; and I believe no other writer has preceded me in this conclusion, so far as my knowledge of its literature has shown."

Mr. Spooner Hart, in his article in 1872–73, to which Mr. Burke refers in a commendatory way, concludes that "although the microscope fails to demonstrate anything like cancer structure, still it must be admitted that the disease is wonderfully allied to this fatal affection."

Our space will not admit of an abstract of Mr. Burke's various articles. In nearly all of them he contends that the disease is of a cancerous nature and throws discredit upon the views advanced by many, that the real cause was due to a vegetable parasite.

Mr. John Steel, in his "Examinations of bursattee matter from the liver of a horse" (1881),[2] refers to a previous assertion by F. Smith, that a vegetable parasite is present in bursattee growths. In the present paper Mr. Steel observes "fungal development, from the specific brown cells of Smith."

Three elements are observed: (1) Crystals of various shapes; (2) brown débris and scale-like masses; (3) undoubted fungal elements in direct continuity with the brown elements and spores.

The latter element is considered the most important, and I give verbatim his provisional account of the "Life history of the brown bursattee fungus of Smith."

In its dormant stage it forms a mass (resembling to some extent the ergot stage of claviceps), hard externally, softer internally, owing its hardness to its density and to calcareous deposits, such as we see also in many of our common wounds and consisting of broken-down fungous elements with spores interspersed.

Under suitable conditions this ergot-like kunkur undergoes changes, resulting in the development of hyphal tubes from the spores whereby the mycelium of the fungus is formed. The characters of kunkur are such that it is thrown off from the body of the host, under ordinary circumstances, just when such a change is required by the fungus. On the soil the color and appearance of the kunkur perhaps serve to protect it from its enemies while it remains entire, but when it disintegrates the spores escape, and having been disseminated after the manner of the germinating elements of other fungi when they fall on suitable soil, such as a fresh wound, produce hyphal tubes on which young spheroidal brown cells appear, in their turn to lead to new spores, and then degenerate into the brown débris of kunkur. This life history, though at present requiring confirmation in many points, should lead to the following practical conclusions:

1. These bursattee sores, when taken early, should be dressed with such agents as destroy fungi—carbolic, salicylic, or sulphurous acid. Thus an absolute destruction to a bursattee center is brought about, for the case is taken before the spore-producing stage.

2. To prevent bursattee, the minute fungus spores diffused through the air should be excluded from wounds by antiseptic dressings, especially those of carbolic acid. The exposed tissues are thus to be rendered a bad soil for the growth of a crop of bursattee fungus.

3. In old bursattee sores in the kunkur-producing stage, these hard masses must be periodically removed and destroyed. By removal we assist nature in her efforts to free the part from these natural irritants. By destruction we thwart her efforts to propagate the vegetable parasite at this stage. Such dressings as caustic potash should disintegrate and destroy small masses of kunkur. Let it be remembered, also, that such kunkur as remains in the sore will be capable of recrudescence in that sore when, with recurrence of the climatic conditions, its season of activity again sets in; hence, the value of the heroic method of cutting out the whole of the diseased part, or destroying it with actual cautery.

Thus bursattee takes its place among parasitic disorders of fungal origin. It is hard to find a parallel to it. Closely resembling certain vegetable parasitic diseases of the skin, it somewhat approaches the cancer in its power of invading

[1] Vol, VII, 368-376. [2] Vet. Journal, XIII, 237-241.

internal organs as well as the surface of the body. When more is known of the true nature of cancer, it may be found not unlike bursattee; but a reference to current literature shows me that I must leave this matter in other hands. We urgently want full and recorded experiments on the communicability of the disease, and also its microscopic appearances in its various stages (the latter, of course, purely for confirmation of observations recorded by able workers who have before this devoted time and attention to the study).

Mr. J. Ferris[1] (1881), while not rejecting the possibility of bursattee being of a cancerous nature, or due to the presence of a parasite, believes that it is an affection of the blood, caused by a sudden change in the weather from wet to dry in certain localities, or by the necessary changes in the nutritive properties of the different grasses, the subject having a constitutional tendency to such a malady.

The reasons which strengthen him in the supposition that it is a blood disease are:

1. The abundant discharges from the sores.
2. A change of climate will frequently arrest its progress.
3. Horses seldom contract this disease in the hills.
4. The disease spreads rapidly immediately after the rainy season sets in.
5. When the rains are over, the malady generally yields to the mildest treatment.
6. The sores ordinarily appear in the less vascular parts.
7. The kunkur is evidently composed chiefly of the mineral substances of the blood, but undergoes certain changes when exposed to external influences.
8. In the early stages it is somewhat analogous to prickly heat in the human subject, which also sets in with the rainy season.

In Robertson's Practice of Equine Medicine[2] (1883) an extended description of the disease is given, compiled from various articles issued previous to that date. As nearly all of the important points alluded to in that work have been touched upon in the presentation of the history of the disease, it is not necessary to repeat them here. The clear, impartial manner, however, with which they are presented by Dr. Robertson is much to be commended.

Mr. F. Smith (1884), in a short but suggestive article on the "Pathology of bursattee,"[3] figures and briefly describes a "mould fungus" found by him in every fresh specimen of the sore examined, but did not find it after the sore had been hardened in chromic acid. "The filaments or hyphæ are branched and nonseptate, one twenty-thousandth of an inch wide and so long that they can not be followed as they pass in and out of the tissue. They converge and diverge, run parallel and transversely to each other, communicate freely by branched processes, and some filaments appeared to be curly. I have seen in a few cases the terminal extremity of a hypha end in a rounded, bright, refractive body very little, if any, larger than the filament itself. The nature of this is doubtful."

No spores were seen, although observations were made over and over again. Mr. Smith concludes with the statement that he had "lately produced typical bursattee ulcers by inoculation with portions of tumor," and that he was "engaged in working this out and endeavoring to produce the sore from the cultivated moulds."

I have been unable to find any later results of these experiments, which is a matter of regret, since they suggest an important line of inquiry.

No mention is made in the above description of any connection between the filaments of the fungus and the "brown cells" as noted by Mr. Steel.[4]

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[1] Vet. Journal, XIII, 333–335.
[2] Page 303 et seq.
[3] Vet. Journal, XIX, 16, 17.
[4] Loc. cit.

The first article that I have encountered stating that the disease existed in the United States is that of Mr. C. C. Lyford, who in 1886[1] gave a paper on bursattee before the Northwestern Veterinary Association at Minneapolis, Minn. He did not know that it had been recognized elsewhere in this country, nor in any other country except India.

"In the United States it is confined to a limited area in the neighborhood of Minneapolis and St. Paul with the exception of a few cases which migrate each year." He had not been able to ascertain how long the disease had been prevalent in that vicinity. Mr. Lyford first noticed it during the summer of 1880. It appeared early in June and lasted until late in the fall, some cases continuing into the latter part of November.

His experience has been that it makes little difference with the malady whether it is accompanied with a rainy season or not, although a comparison of one wet season with a dry one showed that there were more cases during the former.

Seven cases were reported, No. 1 being that of a pony, the entire right side of whose face from the eye to the nostril was at the beginning studded with a number of small bursattee ulcers, which later became confluent, the surface of which was beset with small, yellow fibroid bodies, some of which became caseous and even calcareous, varying in size from a pin's head to that of a filbert.

On the left hind leg there was an extensive ulcer from hock to fetlock externally. The animal would not only abrade and lacerate the surface during the day when the flies were bothersome, but whenever he could slip, break, or otherwise loosen himself from his halter he would bite the parts until they were a mass of raw flesh.

No. 2 was a large draft horse, one of fifty which were kept in the same stable. All of the others remained healthy. In this case a single ulcer appeared midway between the shoulder and the hips on the right side. At first it was not large, but caused the animal much uneasiness on account of the itching, so that he had to be restrained from biting and rubbing. Soon the ulcer became moist and discharged a characteristic albuminous or grumous material, which continued some three months. The sloughing continued until the ulcer became some 8 inches in diameter and the external surface of three or four ribs had been laid bare. This healed only as cold weather came on.

No. 3, a brown gelding, was affected chiefly in the urethra. The penis was amputated and some portions of it examined by a Dr. Hunter, who pronounced it of a cancerous nature.

No. 4 was also a brown gelding. There was one confluent ulcer from foot to knee, the bone being bare some 6 inches on the lower end of the large metacarpal bone, and the external lateral ligaments of the fetlock joints being lacerated allowed dislocation of the fetlock.

No. 5, a gray gelding, was affected in the tongue, which was badly swollen and protruding some 4 inches beyond the incisor teeth. There was an abundant flow of saliva and an offensive muco-purulent discharge from the mouth. The under surface of the tongue at the junction of the "frænum" showed indications of sloughing, being bulged and of a gangrenous nature around the edges of the swelling, the center of which was more of a yellow cast, and discharging an albuminous fluid. This case was examined in a logging camp on the 2d of January with the thermometer standing at 53° below zero.

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[1] Vet. Journal, XXII, 379-381.

No. 6 was a mule suffering from several bursattee ulcers. There was one on a hind and one on a fore leg which had been abraded by interfering; there was another ulcer on its shoulder and still another on the abdomen.

No. 7 was also a mule belonging to a street-car company. The tongue, as in case 5, was the part affected.

Mr. Sermon, who was present at this meeting, also recorded three cases of the affection. It should be noted that Mr. Lyford finds this disease occurring in mules as well as in horses, a matter deemed quite improbable in Robertson's Practice and by some veterinarians.

In the Fourth and Fifth Annual Reports of the Bureau of Animal Industry for the years 1887 and 1888, page 489, there is published a letter from Dr. J. C. Neal, Archer, Fla., under the date of July 13, 1887, in which he describes a disease in that locality as "leeching," to which horses and cattle are subject. He believes it to be peculiar to that section, where it is very common and very fatal to horses and mules. There are hundreds of ponds in the central portion of the State, around the margins of which there is usually a belt of grassy prairie, water grass, water lilies, etc. Into these grassy places the horses, mules, and cows often go during the summer and feed all day in the water. "After a varying exposure to the influence, or whatever it may be called, of the 'pond,' a slight lump or elevation of the skin may be found on some part of the body that has been submerged. To the touch it will feel as if a grain of shot were lodged beneath the skin. In eight or ten days the skin sloughs off centrally over this hard spot, leaving a bloody, bruised-like surface, exuding serum and blood, no pus. This rapidly grows in size till in a few weeks there is a raw surface from 4 inches to 1 foot square. This drops blood and serum, but no pus. An examination will show usually a mass of yellow gritty growth, coral like in shape, embedded in a mass of bruised, bloody tissue, dark in color and the edges roughened, elevated above the skin, and the skin decaying at the outside of the ulcer. The leech invades most any tissue, but seems most common on the legs, abdomen, and sides. Occasionally it is found in the head. The invaded tissues decay slowly, and apparently without pain. I have seen hoofs cut off, the abdomen opened, the eyes eaten out, the teeth destroyed, etc."

Some of the diseased tissue was sent to the Bureau along with his letter. Regarding this he says:

There was a small bunch at the angle of the jaw—outside—the skin not broken; also one in the face near the right eye. I cut to the bone and found the leech embedded in that, and also in the tongue, and pronounced the case incurable.

The treatment is to cut out all diseased tissues and sear with hot iron or nitric acid, followed by carbolic acid. Still, if the disease invades the abdomen, the hoof, or the mouth there is no known remedy. During the last twelve years I have devoted some time to the study of this unique trouble, and can give some facts regarding it:

It does not appear in cypress ponds. It happens that in ponds with cypress in one end only, there will be leeching only at the other end.

All ponds do not leech. Often of two ponds within half a mile of each other one only will leech stock.

All horses do not leech; only those of good blood. The Cuban and Texan ponies are, as a rule, exempt. It is not contagious. A sucking pony may be badly leeched and the dam will lick the ulcer and yet not be affected. Dogs, chickens, and cats will greedily eat the cut-out fragments of a leech and not take the disease, nor do those who cut out the spots ever have any trouble.

The mule is rarely affected, the cow still more rarely.

The period of incubation is from one to eight days. One attack does not protect against another. Some horses leech every year.

As described by Mr. Neal, there will be recognized may points of similarity in the symptoms of leeching and those of bursattee. It is perhaps a question as to whether the disease is developed by the influence of certain ponds or to an individual diathesis of the animals themselves in connection with some infective agent. Upon examination at the Bureau of the specimens sent by Dr. Neal, the sections showed considerable cell infiltration, which probably was due to inflammatory changes. There were also a few centers which seemed to be in a condition of necrosis. The examination failed to reveal the nature of the disease.

Mr. S. M. Smith[1] (1889), in his "Treatment of bursatti by sloughing," recommends the use of finely powdered white arsenic. This is applied to the ulcer and lightly pressed with the finger.

The sore becomes dry and the slough gradually becomes separated from the seat of the disease, leaving behind it a healthy granulating wound having a nice, bright-red color.

It is said that there is no danger of absorption of the arsenic or from the animal licking it off.

Mr. John R. Anderson[2] (1889), following Mr. Hart, finds three forms of bursattee ulcer: the papillated, medium, and phagedænic. The latter he states is at present (1889) prevalent in Kansas and the northern portion of Alabama.

"Those most subject to it are hard worked, poorly fed, coarsely bred animals surrounded by the most unfavorable hygienic conditions. It will probably be of interest to say that all the cases noted save two were in the mule, both sexes alike, although Robertson is inclined to credit this member of the equine family, as well as the jack, with enjoying immunity from this strange affection." Mr. Anderson states that it never occurs, as far as known, in any of the other domestic animals, nor in the well-bred horse kept under hygienic conditions, differing in this respect from Mr. Neal's account of leeching, in which not only high-bred horses but also cattle, though very rarely, are affected. "After the eradication of an ulcer on one part of the body others are found elsewhere regardless of season. Although its ravages are much modified under the influences of cold, the fact of its recurrence would point to its cancerous nature, but when we come to sum up its periodicity, course of treatment, etc., as already described, this theory is at once disproven."

In the Tenth and Eleventh Annual Reports of the Bureau of Animal Industry for the years 1893 and 1894 there are published two letters from persons in Florida bearing on the subject of the Florida horse leech. The material sent at that time was not in a good state of preservation when it reached the Bureau, and was not, therefore, examined. In his reply Dr. Theobald Smith states that a very cursory examination of some alcoholic material sent to him about two years previously gave him the impression that the disease was not due to leech, but to a fungus perhaps similar to actinomycosis or the "Madura foot" of India. The fungus spreads through the tissues, and the mycelium, becoming incrusted with lime salts, forms the leech. The correspondent states that the leeches have been known there for generations.

Mr. A. W. Bitting, B. S. (1894), in an article on "Leeches or leeching"[3] (bursattee), notes that in Florida there are "two diseases called

[1] Vet. Journal, XXIX, 325–326.
[2] Jour. Comp. Med. and Surgery, X, 73–77
[3] Florida Agr. Expt. Sta. Bull., No. 25.

leeches; one an external affection of horses and cattle, the other an affection of the livers of cattle produced by the invasion of a fluke (*Distoma hepatica*)."

It is stated that the disease is not peculiar to the State of Florida nor confined to the lake region, as many suppose, but is known all over the United States, except in the region lying east of the Alleghany Mountains and north of the Potomac River.

In Florida the disease seems to be less prevalent in the high than in the low lying countries. It occurs during the months of highest temperature, and particularly after the beginning of the rainy season. An animal seldom suffers its first attack before the middle of June, but may become affected at any time from that until the middle of October. There are the greatest number of cases during the months of July and August.

A rainy season is not necessary, but is a condition which increases the number of cases and adds to the virulence of an attack. The disease may develop in an animal on an upland pasture and even in a bare lot or stable. In a pond or prairie where an occasional horse will become affected a cow will go free, but in one in which an occasional cow becomes affected almost any horse will develop the disease.

Hogs, dogs, sheep, goats, and poultry seem to be exempt.

Leeches in the mouth or lips is most frequently a secondary affection and comes from gnawing the affected parts of the body. If an animal has leeches on one part and some other part comes in contact with it, as one leg pressing against the other while at rest, there is a strong probability of a secondary attack in the part which comes in contact with that already diseased. During the summer of 1892 three cases were brought to the experiment station which showed this feature in a striking manner. On one animal a secondary growth was produced after numerous experiments.

A thick-skinned horse is less liable to the affection than a thin-skinned one.

Among pathologists there are views held that the cause of the disease may be due to animal parasites, fungi, fly bites, bruises, and irritation.

"In six cases animals became affected that had access neither to water nor pasture, which would eliminate the possibility of its being due to water or to the water leech. Its occurrence in the sole of the foot and other parts of the body not accessible to flies would eliminate that factor. Animals become affected under divers forms of diet, so that poisoning from feed is eliminated as a cause. A microscopic examination of the tissue fails to reveal animal or vegetable parasites. We are not prepared to state what the cause may be. It certainly is not due to one thing, but the result of many."

Mr. Bitting examined microscopically tissues taken from eight different subjects in all stages of the development of the disease. He says:

> Different processes were used for fixing, hardening, and staining to determine the presence or absence of animal parasites and fungi as well as tissue changes. No parasites were found except as accidental invaders. The tissue changes that are found to be present are such as are present in cancer.
>
> The following characteristics are present: A stroma of compact fibrous tissue between whose elements are seen numerous small round cells. The fibers take an irregular direction so that a single section may present a longitudinal in one part of the field, a transverse in another, and the intermediate steps between the two. The cells between the fibers have no arrangement; they may be few or many squeezed together. The blood vessels are very numerous and irregularly disposed

and of varying capacity. The walls are composed of round or elongated cells, instead of those ordinarily found. The leech on section shows that it is a heterogeneous mass, and has been built up by additions to the outside. A very thin section will not hold together. These leeches or kunkur begin with a mass of round cells, and are the result of the squeezing of these cells together by the fibrous portions. In inflammation there is contraction of the tissue, and they are the result. They may contain crystals of inorganic matter and pigment cells. The cells are of an embryonic type. According to the pathological characteristics it belongs to the round-celled sarcoma. It is a cancer.

Mr. Bitting figures the lower jaw of a horse, showing an exostosis of considerable size, with a large area at its base presumably invaded by this disease, but does not discuss the matter in his text.

Mr. Joshua A. Nunn, principal of the Lahore Veterinary College, under the date of March 18, 1896, writing in the Veterinary Journal, page 346, on the "Influence of rainfall on bursattee," says he finds that during six months, from April to October, in 1895, there was only about half as much rain fell as during the corresponding months in 1894, and that concomitantly with the light rainfall only half as many cases of bursattee were received as during the wetter season of 1894.

HISTORICAL SUMMARY.

A summary of the history shows that the "fly theory" of the causation and dissemination of bursattee was entertained by the natives of India as early as 1820, and that among the old theories it was believed to be a blood disease, in many ways not unlike syphilis, scrofula, farcy, etc.

Jackson, in 1842, seemed to have been the first to put himself on record as believing that there was any connection between the disease and a fungus.

Hodgson, in 1853, refers to the sores as cancerous ulcers, and Hart, 1872, is strongly inclined to pronounce it cancer, although he can not get the microscope to confirm this view structurally.

It seems to be generally accepted that the disease is peculiar to the Tropics, but cases have been reported in Kansas and Minnesota in the United States, not only during the summer months, but when the thermometer registered below zero. We might also expect that the disease would exist in Mexico, and Central and South American countries, where the conditions of temperature and moisture are favorable.

Reports show that a high temperature is essential for the development of the disease, although exceptional cases are noted occurring during the cold season. Moisture does not seem to be necessary, since many cases develop when the season is dry. It is, however, an important factor. Statistics show that cases are more numerous and that the disease assumes a more aggravated character during the wet season.

In India native as well as foreign bred horses are susceptible, but, according to some writers, none of the other equine species take it.

In the United States not only the horses, but the mules and cattle are said to develop it, but not as readily as the horse. An outbreak in cattle is comparatively rare. Thin-skinned animals are more susceptible than thick-skinned ones.

Some discrepancy of opinion exists as to the kind of horses most likely to take the disease (assuming that bursattee and leeches are similar). Mr. Neal states that only horses of good blood leech, and the Cuban and Texan ponies are as a rule exempt.

Mr. Anderson states that it is the coarsely bred and hard-worked

horses that are the most susceptible. The well-bred ones, having the advantage of good hygienic surroundings, rarely take it.

Mr. Meyrick, in 1878, reported the finding of kunkur (inflammatory growths) in the lungs of an affected subject and also in a subject which during life showed no signs of the disease. A Mr. John Burke previous to this had found growths in the liver. As a rule, the lesions are at or near the surface of the body. The soft structures are the more favorable, the hard tissues always being avoided, according to the English writers. Mr. Neal, of Florida, writes that any tissue may be invaded, and Mr. Bitting figures the jawbone of a horse quite extensively affected with this malady.

After Mr. Jackson's suggestion, in 1842, that the disease might be related to fungus or to a vegetable parasite, and restated by Mr. Collins in 1874, Mr. F. Smith, in 1879 and 1884, seems to have been the first one to have worked along this line. He was able to find fungi in every fresh specimen of the sores that he examined. Mr. Steel, in 1881, also found fungal elements in these sores.

Experiments to determine the transmissibility of the disease from horse to horse were tried by Mr. Hart in 1872 and 1873, but without result. Mr. F. Smith's experiments in 1879 of inoculating horse from horse were interesting in that in one instance the wound in the inoculated horse did not heal as readily as usual, and also that one of the men who had been inoculated with the bursattee material showed signs of its taking after a few days, but spoiled the experiment by cauterizing the place.

Mr. Bitting's observations on this point are also interesting. He believes that the affection around the mouth or lips is most frequently a secondary one from rubbing and biting the diseased sores on the body; also that where the part of a sound limb is frequently brought in contact with a sore on the opposite limb there is a strong possibility that the former will develop sores secondarily.

On the basis that a fungus is concerned in the etiology of the disease, it is interesting to note that as early as 1877 an English veterinarian advocated the administration of potassium iodide until the patients became thoroughly under its influence, and claimed very beneficial results. Mr. Bitting has found that the use of this drug resulted in a large surface slough, which in two of his cases made an operation easy. The well-nigh specific action of this salt on the ray fungus (actinomycosis) of cattle gives to the above observation additional interest.

GEOGRAPHICAL DISTRIBUTION.

Bursattee has been reported from Burmah and Hindoostan. It is thought that the prevalence of the disease is associated with the principal river systems of India. In the hilly or rocky, and consequently drier, districts there is a very noticeable diminution or absence of it.

Outside of India there seem to have been no cases of this malady except in the United States, unless upon further investigation certain mycotic diseases, which have been described in Europe, should prove to be the same.

Lyford (1886) reported it in Minnesota, Anderson (1889) in Kansas and Alabama, and Neal (1887) and Bitting (1894) in Florida. The latter writer states, after correspondence with Dr. W. L. Williams, that it is "now known all over the United States except in that region lying east of the Alleghany Mountains and north of the Potomac River."

HISTOLOGY OF LEECHES.

In August, 1895, Dr. Shuford, of Gainesville, Fla., sent a specimen of leeches in alcohol to this Bureau for examination. The tissue was in very good condition, but in order to apply different tests with the hope of getting more extended results, a different fixing reagent was sent to Dr. Shuford, who, on October 3, 1895, kindly favored us with some more material, which reached us a few days later in a thorough state of preservation.

The specimen sent in August was cut from the leg, that in October from the lips.

METHODS.

In the first case the tissue, as already stated, was fixed in alcohol. In the second a mixture of picro-aceto sublimate was used in the following proportions:

Fifty per cent alcohol	1,000 cubic centimeters.
Glacial acetic acid	5 cubic centimeters.
Corrosive sublimate	5 grams.
Picric acid	1 gram.

After twenty-four to forty-eight hours fixation in this fluid the tissues were removed to 50 per cent alcohol for a day or two, and then immersed in 70 per cent alcohol, which was frequently renewed until the sublimate and picric acid were thoroughly washed out. The specimen was then dehydrated in 95 per cent alcohol, clarified in cedar-wood oil, and infiltrated with paraffin.

The sections were made to adhere to the slide by first coating it with a very thin film of glycerin. A drop or two of 35 per cent alcohol was placed on the slide and the section was laid upon the alcohol. The whole was then put in an incubator, the gentle heat causing the section to gradually unfold any wrinkles that might be present and the alcohol to evaporate very slowly. After a few hours the specimen becomes thoroughly dry, and if it then be gently heated over a flame until the paraffin begins to melt, and then allowed to cool, the section will adhere very firmly during the subsequent processes.

The collodion method was also employed, but upon the whole did not give as satisfactory results as the paraffin, especially when the anilin dyes were used.

Various stains were tried in addition to the ordinary hematoxylin and carmine methods. Those giving the most favorable results were the Biondi-Ehrlich mixture, methylen blue and eosin, toluidin blue, neutral red, and fuchsin. It was found to be an advantage when using the Biondi-Ehrlich mixture to immerse the sections for a few hours in Müller's fluid before staining. In other preparations it has been found that tissue fixed in Müller's fluid gives as good if not better results than when fixed in corrosive sublimate.

Portions of the tissue were also macerated for a few days in a weak solution of formalin in normal salt solution (formalin, 1 part; normal salt solution, 500 parts, Gage) with excellent results.

A piece of a nodule was boiled in a moderately strong solution of potassium hydrate, in which it was completely dissolved, only a few dirty particles remaining, which under the microscope showed no well-organized structure.

When the reaction was not permitted to become so violent, as, for example, when the tissue was left from twelve to twenty-four hours in

a 10 per cent cold solution of caustic potash, only the fleshy portions were dissolved and there was left a residue which had been more or less calcareous, and when examined under the microscope distinctly showed a fungoid structure. The potash solution changed to a deep-brown color, indicating the presence of a certain amount of pigment. The nodule while in situ showed a deeper color than the surrounding tissues. Another nodule was boiled for a number of minutes in commercial acetic acid; during the boiling and for a time afterwards numerous bubbles were given off from the mass. The substance of the tissue cleared up until it became quite translucent; it assumed an almost jelly-like consistency and was easily crushed, but normally retained its form intact. These nodules had been hardened in the picro-aceto-sublimate mixture.

A Zeiss microscope, with a 2-millimeter homogeneous immersion objective and a No. 4 compensation ocular, was most frequently used. Occasional combinations of higher and lower oculars and objectives were also found to be advantageous.

PATHOLOGIC ANATOMY.

Where the diseased portion has become well developed there is usually a more or less complete detachment of the central inflammatory growth from the surrounding tissue (Pl. XXI, fig. 1). This nodular or kunkur growth may vary in its density according to the stage of its development. During the earlier stages it is soft and easily cut; later it becomes firmer and ultimately assumes a hard or "gritty" character.

In cutting sections it is generally the exception to cut through the nodule or kunkur evenly and to have it retain its proper relation with the other parts. Even if successful in cutting, the nodule drops out during some of the later processes. In the specimens examined the lesions were confined entirely to the skin and subcutaneous tissue; no traces of muscular or glandular structure were observed. Around the central portion of the inflammatory growth there is a zone of leucocytes of the mononuclear and polynuclear varieties, the latter predominating. They are embedded in an abundant stroma of connective tissue which is in a greater or less stage of degeneration. The central portion of the zone is in some cases very closely packed with the leucocytes, while toward the periphery they are more loosely arranged and cause a marked irregularity of the margin from their uneven drifting into the tissue beyond. There is generally one and perhaps more points where this infiltration occurs quite extensively. In some of the preparations the wandering cells have been traced as far as the surface of the epidermis.

Occasionally there may be found a narrow area at the periphery of the nodule, as seen in cross section, which is lighter in color and less dense in texture than the central mass, evidently an extension of the growth.

In the specimens examined for the preparation of this report the parts where the lesions abounded were not characterized by a rich vascular supply. The few vessels that were encountered were not of a normal character; their walls were thickened, and the endothelium, instead of presenting the usual flattened appearance, was irregularly cylindrical (Pl. XXI, fig. 2). Although the condition was not observed, it is not impossible that the hyphæ of the fungus may develop to such an extent as to compress or actually penetrate the walls of the

vessels, causing inflammatory changes sufficient to permit, in the course of time, a disorganization or absorption of the portion of the vessel itself, and that ultimately it may become incorporated in the nodule.

THE NODULE.

The nodules are generally irregularly cone shaped and are of variable size. In section they reveal a very dense structure, the framework of which forms a close reticulum.

Within the meshes are what appear to be leucocytes in various stages of disintegration, and free nuclei. Among these, at places, there can be seen small bodies of nearly the same size as the nuclei, and taking the stains in the same way, but differing in form. At one portion of its circumference the substance of the body is seen to draw itself toward a point, and in favorable preparations that point has been followed some little distance as a delicate filament (Pl. XXIII, fig. 2). In most cases the filament remains unstained, or, as in one Gram-eosin preparation, the club end may stain blue and the filament red. Exceptionally one may find a clear area or vacuole in one of the clubs. From the fact that the filament is not usually traceable to its central connection a more or less flagellate appearance is given to the fungus, which represents a condition not believed to exist.

Not infrequently small spherical bodies were found not far from the clubs which take the stain readily and whose size is sufficiently small to admit of the possibility of their being spores, but no other filament or club was seen which actually contained these bodies. The free ends of many of the clubs point toward the periphery of the nodule, but this is not a constant feature.

The framework of the nodule stains very slightly or not at all and shows among the enmeshed corpuscles as a very irregular, distorted, and somewhat glistening network. It is this portion of the nodule that gives the hard, gritty feeling, and is probably due to a greater or less deposition of lime salts along the reticulum. Along the periphery of the nodule there has been seen, in addition to the leucocytes and pyriform bodies before mentioned, occasionally filaments of a fungus projecting from the mass (Pl. XXII, fig. 1). The filaments have also been seen ramifying to some extent in the adjacent tissue, but rarely.

It would therefore appear as if the framework of the nodule were composed of a myceliai net, which in the course of development had become more or less calcified. Running through the nodule in some of the preparations there were a few comparatively large cords, which present the same calcareous appearance as the network, and appear to be fibers of elastic tissue which have become somewhat calcified.

As a result of the treatment of the nodules with the 10 per cent cold solution of caustic potash a very profuse and intricately branched fungus became apparent. The branching is of an irregular order. In places there is seen in the filament a central axis, which takes the stain, and around this appears a transparent or hyaline sheath varying in size (Pl. XXV, fig. 6).

In some of the shorter and apparently younger branches of the fungus the mycelial sheath was of considerable size, while among the more closely woven filaments the sheath in places was scarcely if at all distinguishable.

In certain of the teased preparations (Biondi-Ehrlich stain) the wall of the filament, instead of being smooth and homogeneous, appeared roughened, as if covered with very minute but numerous spinous processes (Pl. XXIII, fig. 3).

In the sections of the tissue in which the fungus appeared the substance of the filament was not uniform. In places it was drawn together in an irregular manner, with intervening clear spaces of greater or less area (Pl. XXIII, fig. 4). One of the caustic potash preparations showed circular or oval clear areas in the filament resembling vacuoles. These varied in size from a mere point to the diameter of the filament (Pl. XXV, fig. 5).

In a Gram-eosin preparation of the fungus the substance of the filament is so much shrunken and distorted as to suggest its having entered into an involution stage (Pl. XXIV, fig. 1).

In some places the filaments show distinct septa, but the latter are not common. Some of the club-like endings, especially those that are elongated, show a septum at the union with the filament proper (Pl. XXIV, fig. 2). Scattered among and coiled around the ordinary filaments there have been observed much slenderer ones apparently devoid of any external sheath.

There have also been observed in some of the preparations numerous small circular bodies of inconstant size. They have been seen lying freely in the meshes of the mycelium and also closely applied to the filaments. These bodies are not spherical, but thin and flattened, and some of them present a curved appearance, convex on the outer side and concave on the inner side. They suggest the possibility of having been closely applied to the filaments and have something of a scale-like arrangement. They were also seen actually applied to the filaments, singly and in numbers. Occasionally when single it was not easy to determine whether they were in or outside of the sheath. The fact that they were of various sizes, their antipathy to stain, and their variable form would seem to indicate that they were not spores (Pl. XXV, fig. 4).

With possibly one exception, no trace of blood vessels was found in the nodules.

THE CIRCUMNODULAR TISSUE.

Pathologic conditions exist to a greater or less distance in the tissue adjacent to the nodule. Certain areas of this tissue are often necrosed to such an extent as to refuse to take the stain.

The more prominent appearance is the infiltration of the connective tissue with a great number of wandering cells. In some places there are well-defined nests of these cells in the stroma of the connective tissue (Pl. XXII, fig. 6), simulating, perhaps, a cancerous appearance. The character of the cells, which present a curiously vacuolated condition, would, however, tend to eliminate this view. The vacuoles vary in number and size, the average number being 10 to 12 in a cell.

In some of the preparations numerous leucocytes, of the mononuclear and polynuclear variety, had drifted away from the nodule. They were for the most part elongated, and in all the nucleus or nuclei appeared to be in a healthy condition. The cells contained numerous small bodies, which took a deep orange color with the Biondi-Ehrlich stain (Pl. XXII, fig. 4). In places adjacent to these leucocytes there were frequently noticed a number of these small bodies apparently lying free in the tissue. No definite cell wall was distinguishable in the leucocytes.

The vacuolated cells before mentioned were present in greater numbers than the heavily laden leucocytes. In the former nuclei were present and presented various phases of change (Pl. XXII, fig. 5).

In some there was a single nucleus, which may be circular, crescentic, or in the form of a dumb-bell; in others there may be two or more nuclei which in advanced cases appear only as remnants. In extreme cases no nuclei at all were visible. The wall of the wandering cell differs from that of the leucocytes proper in possessing an appreciable thickness. This thickened boundary apparently gives considerable rigidity to the cells, as nearly all of them are approximately circular in form. Their average diameter is about 8 microns. In the first specimen sent to us (taken from a horse's leg) there were encountered what appeared to be large giant cells, measuring from 12 to 18 microns and apparently possessing quite a distinct cell wall. Within each giant cell there was some appearance of vacuolated cells, each with a single nucleus and fairly well-defined cell boundary (Pl. XXII, fig. 3). As many as eight or ten of these nuclei have been counted in a single giant cell. There is the possibility that these apparent giant cells are simply some of the vacuolated cells fused together, but the nuclei are well defined and take the stain very intensely, which is not commonly the case in the ordinary vacuolated cells. In some places the giant cells had become detached from the tissue, and although freely floating around they nevertheless preserved their integrity.

In some of the earlier specimens examined vacuolated cells were encountered presenting no trace of a nucleus whatever (Pl. XXIII fig. 1), and the idea was entertained that they might be parasites of a protozoon nature or a mass of encysted spores, but the presence of a nucleus showing the conditions before noted and the possibility that in these sections (stained in methylen blue and eosin), in which the non-nucleated condition obtained, might be due to the washing out of the methylen blue from the nucleus by the eosin; and the further facts that they were found mixed with the other blood elements in a cross section of a blood vessel; that they were of the same size of a leucocyte, are mononuclear and polynuclear and are found wandering through the tissues in the same way, suggest the possibility of their being leucocytes modified by special pathologic conditions.

These cells, as well as the spore-laden leucocytes, have been encountered wandering freely through the dermal tissues as far as the surface of the skin and sometimes along the hair follicles. This appearance would suggest that the nodule was in process of coming into communication with the external surface and thus forming an open sore, apparently an effort to remove the irritant.

Constructing a theory from these facts, it would seem reasonable to assume that the small bodies found in the leucocytes were nothing less than spores which had been formed by the fungus in the nodule and seized upon by the surrounding leucocytes, and, if it be true that the vacuolated cells be modified leucocytes, that the phagocytic action has taken place between them and that each vacuole may represent the effect of a spore; that in this struggle many of the leucocytes likewise become disorganized, as evidenced by the degeneration of their nuclei, and finally drift off to the surface of the wound, where they may be shed with the serum. The leucocytes which do not succumb too readily wander to other parts, where under proper conditions new centers of the disease may develop.

The fact already mentioned that cells in the vacuolated condition were found in the blood vessels would militate against the phagocytic theory, unless it were demonstrated that the spores in some way entered the blood channels and were there acted upon by the leucocytes before, instead of after, diapedisis had occurred. Nothing in

the preparations, however, indicate any power of motility on the part of the spores. On the other hand, it might be conceived that a poison is formed by the fungus which could penetrate into the circulation or by some system of chemiotaxis act upon certain of the leucocytes while still in the vessels. There is no inherent impossibility against the existence of a vegetable and a protozoon parasite at the same time in the diseased organism; but this question would scarcely seem to demand serious consideration in the case at hand. The examination of the living or fresh blood from around the sores, the enumeration of the corpuscles, the study of some of the fresh tissues, and the attempt to cultivate the specific organism in artificial media and inoculation experiments might help to elucidate some of these doubtful points.

The connective-tissue cells surrounding the nodule show marked signs of degeneration, their cytoplasm in most cases being extremely vacuolated (Pl. XX, *c*, fig. 3). Among these connective-tissue cells, which for the most part are quite branching and elongated, is another class of cells which are in general of an oval or elliptical form. The noteworthy appearance of these cells is the presence of numerous dots in the cytoplasm which take the methylen blue and toluidin blue stains very deeply. The appearance is, indeed, very much as if the cells were filled with micrococci. These are the granule cells of Waldeyer, or still further differentiated as the plasma cells, in contradistinction to the "mastzellen" or "food cells," which indicate an exalted degree of nutrition. The nucleus of the plasma cell takes the stain very slightly, or not at all, and is almost entirely obscured by the numerous "granules" in the cytoplasm. These cells are well differentiated by the toluidin-blue stain, as they take a deep purple color, while the surrounding cells are blue (Pl. XX, *a*, fig. 3).

BACTERIA.

Numerous bacteria were found at certain places on the surface of the skin and in some of the diseased areas. Streptococci, as well as numerous bunches of staphylococci and bacilli, were present and easily demonstrated by the usual bacterial stains. These organisms were frequently met with along the surface of the nodule where it had become detached from the surrounding tissue. They were not found in the substance of the nodule nor in the adjacent tissue except where it might perhaps communicate with the exterior. It is believed from the above reason and from the fact that they were not found at all in one of the cases that they are of secondary importance and need not be regarded as having any particular etiological relation to this disease (Pl. XXIII, fig. 5).

SOME OTHER DISEASES OF MYCOTIC ORIGIN.

A NEW MYCOSIS OF THE HORSE.

In the Compt. rend. hebdom. Soc. de Biol., Tome III, No. 14, pages 425–428, May, 1896, there appears a "Note sur une mycose souscutanée innomée du cheval," by Drouin et Rénon, and again in the Recueil de Médecine Vétérinaire, Tome III, No. 11, pages 337–344, June, 1896, is an article "Sur une nouvelle mycose du cheval," by V. Drouin.

The material for the two articles was presumably furnished by the same animal. Toward the end of June, 1895, the horse developed a

series of cutaneous tumors which were at first miliary, but within two months acquired a considerable volume. They were located on the neck, superior border of the chest, at the point of the shoulder, and in the inguinal region. Some of the tumors became confluent. At the beginning an intense pruritus was one of the accompaniments.

A generalized sarcoma was excluded from the diagnosis on the ground that there was nothing in the consistence of the swelling resembling that of a sarcoma. They were manifestly fibrous, exhibited pruritus, and showed numerous vegetating products. Botryomycosis was also excluded because the parasite peculiar to that disorder was wanting and because the botryomycotic tumors generally do not become confluent. They vegetate slowly and are not pruritic.

In one case a tumor was removed weighing over 9 kilograms (about 20 pounds) and which necessitated an opening 75 centimeters long by 60 centimeters wide (about 30 by 26 inches), and in two months the animal was able to resume work.

In all cases where the extirpation of the parasite is not complete a return of the disease is certain. There is some hemorrhage, and a thickened condition of the walls of the blood vessels was noted.

The vegetative growth can be easily enucleated by pressing a little vigorously. It is an extremely anfractious mass, rugous on its surface, and presents considerable resistance to traction. Under the knife it gives the sensation of a dried-up hardened caseous substance. It may acquire the volume of the thumb, but more frequently it is that of the hazel nut. A ramified mycelium was found to exist which could not be classified with certainty in any of the known species.

A thin section was fixed to a glass slide with albumen and treated for some seconds with a weak solution of silver nitrate (1 per cent), then washed and immersed in a reducing bath. The latter was a diluted solution of a developer used in photography. In some instances the parasitic parts appeared intensely black, while the surrounding tissue assumed a light-gray tint. The specimen was again washed, dehydrated in alcohol, cleared in xylene, and mounted in balsam.

The tissue proper of the tumor was found to be exempt from the parasite.

In conclusion M. Drouin finds the neoplasm described by him differing from analogous products (1) by the facility of its subcutaneous generalization; (2) by the intense pruritus which accompanies it; (3) by the presence, in the tumors, of rugous, yellowish-green vegetating masses, which appear to be constituted of a felted mycelium; (4) by the rapidity with which a cure is effected if care is taken to remove all the pathological products; (5) finally, by the certainty of a return when the vegetable parasites are not completely extirpated.

In the article by Drouin et Rénon in the Compt. rend. Soc. de Biol. a description is given of their attempt to cultivate the parasite. Their results were not conclusive, nor were their inoculation experiments attended with positively satisfactory effects.

Many of the symptoms described by these gentlemen may readily be recognized as corresponding in a great measure to those found in leeches or bursattee. The climatic conditions, as, for instance, the time of the year, the latter part of July, corresponding with the time in this country when leeches are especially prevalent (July and August). It would be interesting to know whether the season was a wet or dry one, and if, as in leeches and bursattee, the disease improves of itself with the onset of cold weather. The intense pruritus accompanying the disease has been noted by Mr. Lyford in his Minnesota

cases and others. The apparent cure within a short time, if the diseased tissue is thoroughly removed, and the certain return of the malady if any of this tissue is left; the presence of a fungus with an irregular branching mycelium not yet classified, and the negative results of inoculation experiments are all characteristic of leeches, and, perhaps, bursattee. Further investigation may demonstrate that there is more than a mere coincidence in the similarity of the symptoms.

ACTINOMYCOSIS.

This disease, although predominating in cattle and man, is not infrequently described as existing in the horse. The parasite, curiously enough, it is said was discovered in man, although cattle are very much more subject to it.

In cattle the disease seems to prefer the region of the jaw and tongue, on which account the vernacular names "lumpy jaw" and "wooden tongue" have originated. It is caused by a fungus of the genus *Actinomyces*, which leads to the development of tumors about the neck as well as the head, and also to cause lesions in some of the viscera and to affect osseous tissue. The fungus is characterized by club-shaped bodies, which are arranged in a radiating manner so as to form a well-defined rosette, the clubs probably being in connection with the filaments which make up the central portion of the rosette.

When the fungus once obtains lodgment in the body there is evidence for believing that the spores and young fungi are disseminated through the body through the agency of the leucocytes and establish new centers of growth.

To Wolff and Israel belong the credit of having demonstrated the inoculability of the disease. They were also able to grow the fungus upon various media after having isolated it from abscesses in man. There appears to have been considerable difference of opinion as to the transmission of the malady from one animal to another by inoculation. There are apparently no satisfactory results when the discharges from the swellings are introduced into another animal; but if a portion of the diseased tissue be placed under the skin or into the abdominal cavity the disease is likely to develop.

Wolff and Israel succeeded in causing the disease in rabbits and guinea pigs by inoculating into the abdominal cavity a small quantity of a pure culture on agar or upon egg. The peritoneum developed small tumors and their contents showed the characteristic forms, including the clubs.

The fungus has been found in nature on various plants, and, it is believed, may be taken into the body through wounds in the skin or through the mouth by lodging in carious teeth and thus forming a nidus for the further extension of the fungus. Israel describes a case in man which was, apparently, directly acquired by inhalation.

Some of the symptoms as given for actinomycosis are not wholly unlike some of those described for leeches, the distinguishing difference being largely in the appearance of the fungus itself.

In leeches there is not the characteristic radiate or rosette arrangement of the clubs. Although the free ends of some of the filaments are taperingly enlarged distally, they do not show the marked development exhibited in actinomycosis.

Doubtless further differences will be encountered in their artificial growth and unlike reactions exhibited in their relation to the various culture media.

At present there seems to be a difference of opinion as to the inoculability of the two diseases, but we ought not to consider the non-inoculability of leeches as absolutely settled yet.

It is not impossible in the case of leeches that we may have a fungus which might be classed in the same genus as that of actinomycosis, but which present variations sufficient to place it in another species. Among some of the recent writers there is a tendency to reject the genus *Actinomyces* (Harz, 1877) and adopt the earlier one, *Oospora* (Wallroth, 1833). Sauvageau et Radais[1] (1892) stated that *Actinomyces* agrees perfectly with the genus *Streptothrix* (Cohn, 1873), and that both ought to give way to the earlier *Oospora*.

Gasperini[2] (1894), as a result of some new investigations, identifies the *Actinomyces* with the *Streptothrix*. He prefers to retain the former name, and includes in the genus *Actinomyces* 18 different parasitic organisms, the majority of which are still very incompletely known.

Blanchard[3] (1895) believes that while the *Actinomyces* have a great morphological resemblance to the common *Oospora*, they are nevertheless differentiated by their more delicate dimensions. He supports Trevisan, who in 1889 proposed the genus *Nocardia*, and in it includes among others the fungus *Actinomyces bovis* as *Nocardia bovis* and that of madura disease as *Nocardia madura*.

Lehmann and Neumann[4] (1896), following Sauvageau et Radais, adopt the genus *Oospora*. The former writers include 10 species, among which are the fungi of farcy in beef—Nocard, madura disease, streptothrix, cladothrix, etc.

MADURA DISEASE (MYCETOMA).

Although exclusively confined to the human subject, this disease in its symptoms and course bears some resemblance to certain pathological conditions found in animals. It appears to have been first known in India and was believed at one time to have been peculiar to certain districts there. Statistics show that it has been found in Italy, Algeria, and America, but not commonly.

The foot is the part generally affected, occasionally the hand, but, so far as is known, the trunk never. The malady is essentially local. At first there is a tenderness, then tumefaction. In time—a year, or perhaps two—the tumor suppurates and the pus leaves through one or several sinuses.

Two forms of the disease are known—the ochroid, or pale, and the melanoid, or dark.

A sero-purulent liquid containing small light-colored bodies or dark gunpowder-like grains is discharged through the sinus and the character of the granules determines the form of the disease.

It is very intractable and affects the bones. Cases have been known to run from eight to fourteen years. It is incurable; death may supervene from exhaustion or some complication.

In the early sixties Vandyke Carter believed a fungus to be the cause of the disease and succeeded in growing a pink mold; but this is not accepted as the true cause, on account of the faulty methods then in use. The parasitic theory was combated, and Carter seems not to have

[1] Ann. de l'Inst. Pasteur, VI, 242–273.
[2] Processi verbali della Soc. Toscina di Scien. naturali.
[3] Maladies parasitaires. Parasites animaux, Parasitaires végétaux, à l'exclusion des bactéries. Extrait du Traité de Pathologie générale. Tome II, pp. 649–926.
[4] Atlas und Grundriss der Bakteriologie, München, 1896.

insisted upon his pink mold, but nevertheless held that a fungus of some kind was the real cause. He suggested that it was a ray fungus similar to that found in actinomycosis. Omitting the various opinions advanced up to 1892, we notice a paper in that year by Kanthack,[1] whose purpose is to demonstrate the identity of mycetoma with actinomycosis—an attempt to confirm the suggestion of Carter.

In 1893 Boyce and Surveyor published an article on the existence of more than one fungus in madura disease[2].

Vincent[3] (1894) succeeded in isolating and growing the fungus upon artificial media. In the tissues he believes that various involution forms may be met with. He names the fungus *Streptothrix maduræ* and finds that it is not provided with a sheath or transverse septa.

A culture of this fungus was allowed to dry for nine months on blotting paper; another culture on potato was dried for twenty-one months, and both were found to be active at the end of those periods. This longevity is explained by the presence of spores. The presence of oxygen is not necessary for the fructification of the parasite; the spores flourish best in the air. They are small ovoid cells, very refrangent, and joined in twos and threes or forming quite voluminous masses. They are readily stained by the anilin dyes. The spores are killed at a temperature of 85° C., after an exposure of three minutes; they resist 75° for five minutes. Cultures without spores are killed at 60° in from three to five minutes.

Inoculation of the parasite granules and of the cultures were made into rabbits, guinea pigs, mice, and cats at different times, but without results. Nocard, with the same cultures, tried subcutaneous, intravenous, and intraperitoneal injections in the guinea pig, rabbit, pigeon, fowl, dog, and sheep with imperfect results, no trace of the parasite being found in the animals when examined a short or long time after inoculation. The fungus is not, therefore, parasitic in these animals.

Vincent rejects the view of the similarity of the fungus of actinomycosis to that of madura disease, basing his objections upon the fact that the former is inoculable and the latter is not; that actinomycosis is generalized, appearing in various parts of the body, preferably in the submaxillary region, and has a comparatively rapid course, while the madura disease is localized, affecting the foot, and exceptionally the hand, but not invading the other parts of the body, although its course is very slow. Patients have been known to carry the disease for more than twelve years. The treatment with iodide of potassium, which has such a beneficial effect in actinomycosis, is of no avail in madura disease.

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[1] Madura disease (mycetoma) and actinomycosis; Jour. Path., I, 140–162, 1892.

[2] Proced. Roy. Soc., LIII, 110–112.

[3] Etude sur le parasite du Piede de Madura. Ann. de L'Inst. Pasteur, VIII, 129–151.

Finally, the reaction of the two fungi to the same culture media is so interesting and different that the following table from Vincent is subjoined:

No.	Cultures.	Actinomycosis.	Streptothrix maduræ.
1	Bouillon	Culture abundant	Culture medium.
2	Sterilized infusion of hay or straw (15 grams per 1,000).	No development	Elective nutritive medium; culture precocious fourth day and abundant.
3	Ordinary peptone gelatin.	Liquefied	Does not liquefy.
4	Gelatin with hay infusion	Whitish culture, very feeble.	Development more rapid; the colony becomes rose or red at the surface.
5	Glycerinated gélose (agar)	Spots at first white, then grayish.	Colonies at first white, then rose or red, umbilicated.
6	Potato	Colonies dense, yellow and white circled with black; turns brown.	Cultures of rose, vivid red, or black red do not turn the substratum brown.
7	Cabbage, turnip, carrot	Does not cultivate	Does cultivate.
8	Serum	Develops	Does not develop.
9	Egg	do	Do.
10	Culture in vacuum	Anærobic facultative	Does not develop in vacuum.
11	Inoculations	Inoculable in the rabbit, calf, heifer, and guinea pig.	It is not inoculable in any animal.

PATHOGENIC BLASTOMYCETES.

Under this title Tokishige[1] (1896) describes a disease existing in horses and cattle in Japan. It is an infectious skin disease, and prevails in certain regions of that country.

According to the official disease list for the year 1891, 2,589 horses suffered from this malady (out of a total of about 1,500,000), of which 95 died and 84 were killed. The malady prevails more on the level than in the hilly districts and in the wet seasons rather than dry ones. The majority of the new cases appear during the cold portions of the year, while in the summer the cases are comparatively few. During five years (1887–1891) there were 9,361 cases, averaging a little more than 1,800 a year, out of which number more than 1,500 occurred between December and May on an average, leaving only about 300 for the remaining months of the year. Animals 3 to 4 years of age were especially susceptible. Tokishige recognized a fungus as the cause of the disorder as early as 1893.

As a result of inoculation experiments the disease does not spread from horse to horse, but, like miasma, seems to get in from without through small wounds in the skin. The primary changes appear mostly on those parts of the body which are most subject to injury, the lower portion of the extremities and parts subject to chafing by the harness. Infection may follow by means of earth or litter.

The primary nodes were always found in the cutis; only exceptionally were they found in the nasal mucosa. The front extremities were the parts mostly affected.

In the skin the affected parts appear as hard circumscribed nodes of the size of walnuts, and are slightly if at all painful. They may persist as hard nodes, but generally they soften and become abscesses or ulcers, the latter more frequently. This process seldom remains localized, but spreads superficially and deeply, and follows, by preference, the course of the lymph vessels. In inveterate cases changes occur in the perimysium, also the periosteum of bone and cartilage.

[1] Ueber pathogene Blastomycetes. Centralbl. f. Bakt. u. Parasitenk. u. infektionskrankheiten, XIX, No. 4–5, p. 105–113.

The abscess may discharge a thick glutinous or bloody pus, or a thin pus of a semitransparent nature mixed with flakes. When the respiratory tube is involved the affection is generally secondary, brought about by inhaling the infection. It may spread to the pharynx, larynx, and trachea. Very exceptionally are the lower bronchi affected.

The lymph glands are nearly always enlarged, especially those in the axilla, and those next affected are in the following order: the inguinal, pubic, and popliteal glands. The glands are mostly soft and movable; induration is infrequent.

The disease appears to have a predilection for the testicles. The process generally begins at the scrotum or prepuce and passes to the tunica vaginalis and ultimately to the parenchyma of the testicles, epididymis, and vas deferens. The focus in the testicle is always exactly circumscribed and appears to be of a tumor-like formation with a softened center.

Nodules in the lungs and localization in other organs are only exceptionally encountered. Symptoms of anæmia and cachexia are present.

In the lighter cases there are no general disturbances; the patients are active and have good appetites. Soon the disease extends over a greater part of the skin and becomes localized in the nasal mucosa, and the animals become so weak that they can no longer work. From the nose there is at first a mucous discharge, later a mucopurulent, and finally a bloody fluid; a characteristic and offensive odor is present.

The disease runs a very chronic course and may last many months. Acute cases are exceptional.

In the microscopical examinations, besides some bacteria, which were not found to be constant, there were present some peculiar cell-like bodies, which were easily found in the fluid and solid diseased products. These bodies are ovoid, 3.7 to 4 μ in length and 2.4 to 3.6 μ in width. They have a thick, doubly contoured membrane, with contents of a more or less homogeneous nature. The disease is caused by a fungus, which develops in its interior spores (ascospores), belonging to the Saccharomyces.

The fungus grows on the usual nutritive media; the colonies have an uncommonly slow development. In the animal body it exists and extends as a pure Saccharomyces, while pure cultures transmit forms similar to those in the thrush (Soorpilz). Inoculations with pure cultures and diseased tissue have, in spite of a great number of experiments, hitherto given no positive results.

According to the acceptation of some mycologists, the yeast fungi appear only as developmental forms of higher fungi, so that perhaps the natural infection occurs not directly at this but some later stage.

The disease as described in horses appears to be identical with the Italian and French malady "Lymphangitis epizootica, African or Neapolitan worm," the cause of which Rivolta has established to be ovoid bodies, which he calls *Cryptococcus farciminosus*. According to former deductions, *Saccharomyces farciminosus* is to be recommended as an exact designation for these bodies.

The disease may exist in cattle likewise, but apparently is not so common. The chief differences in the course of the disease as regards the cattle are:

1. The nodes are in the later stages more clearly isolated and never form bead-like cords.

2. They always remain circumscribed, grow very slowly, and do not develop into abscesses or ulcers.

3. The course in cattle is much more chronic than in horses.

Whether the disease described in the West Indian islands, especially Guadeloupe, under the name of "cattle worm" (Rinderwurm) is identical with the Japanese cattle disease is yet to be established.

The fact that this disorder flourishes best during the cold season is interesting in comparison with the apparent preference of most of the other fungal diseases for warm temperatures. Dampness in any case seems to be a factor in the development of the fungus.

The use of the word "worm" in connection with the malady would seem to indicate, as in the case of leeches, that the origin of the disease, in the popular mind, might be due to a similar cause.

ŒDEMA MYCOSIS (SOUTH AFRICAN HORSE SICKNESS).[1]

This disease is defined as a specific fever, characterized by intense congestion of the blood vessels, which pour out a large quantity of serum into the neighboring tissues. Whether or not it is peculiar to the horse has not yet been established.

Dr. Edington, Cape Colony, states that he found in the blood of affected horses a microbe (a fungus or mold) which he believes to be the cause of the disease, but he has not succeeded in cultivating it apart from the animal body and then reproducing the disease. The fungus growing in the blood acts as a foreign body, causing coagulation. The animal dies of thrombosis.

It is believed that the infection is brought into the system along with the fodder and perhaps the drinking water. Dr. Edington explains that the fungus remains in the passive condition of spores under the influence of dry air and is killed by prolonged drought, frost, and cold rain.

The disease is known under three forms: The lung form (paard ziekte), which is the most common variety and might be termed "specific equine pleuro-pneumonia;" thick head (dikkop), and blue tongue.

It can be conveyed by inoculation with the blood or with the yellow fluid which is given off from the nostrils, and by drenching with the diseased blood. The yellow fluid is not always virulent.

The period of incubation is about eight days, and the disease runs a fatal course usually in about four days. The internal temperature is high. The abundant serum is one of the characteristic features of the disease, and the animal has been described as being "drowned in its own blood serum." The animal may die very suddenly, frequently within an hour or two of the time it was first seen to be ill.

Horses are very susceptible to this disease, mules much less so, and zebras rarely contract it.

The disease seems to be present to some extent in some parts of the colony every year. It rarely assumes an epizootic form. At such a time the loss is very great. During the two years 1854–55, there were 64,850 horses killed by it, and in 1892, 13,979 horses and 149 mules died from this cause. The pulmonary form of the disease is almost invariably fatal.

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[1] M. H. Hayes, "South African horse sickness," Vet. Journal, XLII, No. 247, pp. 22–31, January, 1896.

GENERAL CONSIDERATIONS.

Fungal diseases of animals, although a comparatively new field for research, present some difficult problems for elucidation. The exact mode of infection is not yet clear in many cases, nor has the total dissimilarity in the growth of the parasite in artificial media as compared with that in the body been fully explained.

The distribution of this class of diseases is thoroughly cosmopolitan, the majority of them occurring during the summer season in warm or temperate climates, although, exceptionally, some forms thrive better during the cold weather.

The extent of the loss is not infrequently overlooked or unappreciated on account of the insidious course of some of these maladies. In many cases the affected animal is free from general systemic disturbances and may be used for all ordinary purposes. With the approach of the cold season the sores (leeches) tend to heal of themselves, only to recur with greater severity during the following heated season, unless completely extirpated. With the course of time new sores appear and the old ones become more aggravated, until ultimately the patient becomes so afflicted that he is slaughtered before death can terminate his agony.

Although some of the appearances may simulate cancer, the real question of identity hangs upon the cause. Excluding all other dissimilarities, it would yet remain to demonstrate the identity of the pathogenic organisms, and thus far this has not been proven. Actinomycosis was for a time diagnosed as cancer, and this diagnosis, upon the same general grounds, has been applied by some veterinarians to most of the other mycotic affections.

Statistics are meager, but they indicate that the loss is considerable. Except for actinomycosis, there seems to be no very satisfactory treatment. The suffering is considerable and prolonged in the chronic cases, and the consequent disability ought to be recognized from a statistical as well as a humanitarian point of view.

SUMMARY.

1. The investigation recorded in the preceding pages, so far as is known, has demonstrated for the first time the presence of a fungus in "leeched" tissues. The fungus is located in the inflammatory growth or nodule, and in rare instances may be seen ramifying for a short distance into the adjacent tissue.

2. The nodules are of various shapes and sizes. Their structure is very dense. The fungus often appears to have become more or less calcified, according to its stage of development. Inclosed within its meshes are a great many cells, apparently leucocytes, mingled with developing fungi.

3. The existence of spores was not conclusively demonstrated in the sections, although small spherical bodies of a suitable size were noticed in the nodule.

4. The margin of the nodule in sections is characterized by a narrow, lighter-appearing zone, which is found to be made up of the free, tapering, club-shaped ends (hyphæ) of the fungus, among which are scattered some leucocytes.

5. The fungus in the tissue appears in various forms—perhaps involution stages. Vacuolated and shrunken conditions were encountered. The branching mycelium is very irregular, and in places there

was observable a transparent, gelatinous (?) sheath around the mycelial axis. Around some of the younger branches this sheath has a distinctly swollen appearance. Quite rarely was there encountered distinct evidence of septa in the filaments.

6. Peculiar flattened or concavo-convex scale-like bodies were found, in certain places closely embracing the filaments and in others lying freely in the mycelial network. Their function was not determined.

7. The nodules are apparently devoid of normal blood vessels.

8. The tissue around the nodule is considerably infiltrated with wandering cells. They present in some places well-defined nests in the stroma of the connective tissue.

9. Two kinds of wandering cells were observed. The first variety contained numerous spore-like bodies. The second variety was characterized by the presence of numerous clear spaces or vacuoles. They also possess a marked cell boundary, which seems to give some rigidity to the periphery of these cells, since nearly all of those encountered were circular or oval in outline. Those of the second variety were much more numerous than the first.

10. The connective tissue cells in the circumnodular area are very much vacuolated and appear to be in process of disorganization.

11. Among the connective tissue cells there are found at intervals elliptical cells, filled, when stained, with minute dots. The nucleus colors very slightly or not at all. They are plasma cells.

12. Bacilli, streptococci, and staphylococci were found, but they are not believed to have any etiological relation to the disease.

13. Certain phenomena make it necessary to believe that spores must exist. Without them it would be difficult to explain the development of new disease centers in different parts of the body. The leucocytes, laden with so many small masses, are strongly confirmatory of this view.

14. How does the fungus or its spores gain entrance into the body? Two ways suggest themselves—through the mouth or some wound in the skin. If through the former, we might expect more or less distinct lesions in the viscera, which do not seem to have been reported. If through the skin, the wounds would tend to remain open sores. As a matter of fact, the first visible appearance of the disease in most cases is a small subdermal tumor which ultimately breaks through the skin and forms an ulcer. It is not impossible—indeed, some circumstances point to the fact—that either or both methods of infection may occur.

15. The discovery of vacuolated cells in the blood would lead us to expect more or less pronounced pathogenic changes in that fluid. A study of the blood in the fresh condition and the enumeration and determination of the relative proportion of the red and white corpuscles would be very important.

16. The disease seems to prefer the subcutaneous tissue in the less vascular parts of the body. The soft tissues are the ones usually affected, but there is evidence that the bones may likewise become involved.

17. The attempt to isolate the parasite under special antiseptic conditions, and to grow it and its spores (?) in various culture media, and to determine its vital activities and death point, if persevered in, might be crowned with success. Inoculation experiments are no less important, and it is a matter of great regret that this side of the work could not have been developed and at least partially incorporated in the present report.

18. No very satisfactory form of treatment has yet been devised beyond that of completely cutting out the diseased areas. Since certain indications point toward the presence of spores, it would therefore seem advisable to remove these growths early, before the spores had time to develop and be carried by the leucocytes to establish new centers of infection. The disease has been described as a "lingering agony," and its cure or prevention should be earnestly sought.

19. If it be true that bursattee in India does not attack mules nor cattle and develops only in the soft tissues, while in the United States (assuming bursattee and leeches to be similar) the disease attacks not only mules but cattle, and even invades the bones; then, according to this evidence, it would appear that the American form is the more virulent of the two.

As compared with actinomycosis it would appear that leeches exhibits a preference for the equine family and more rarely the bovine, while actinomycosis prefers cattle, and only exceptionally affects the horse.

20. Assuming that the fungus may exist in nature on plants, or in the air or water, I have not yet been able to learn of any cases of human infection, either through the digestive or respiratory passages, or, as in the case of the madura disease, through wounds in the foot or hand.

DESCRIPTION OF PLATES.

PLATE XIX.

Fig. 1. A piece cut from the lip of an affected horse, showing several diseased foci. (Somewhat reduced.)

2. From the same lip, but shows a larger infected area.

3. An isolated nodule showing the characteristic roughened, coral-like appearance of the mass. Enlarged about 10 diameters. The small figure to the left represents the natural size.

PLATE XX.

Fig. 1. A section of the skin showing the infiltration of the wandering cells from the inflammatory center into the dermal tissue.
(*a*) Wandering cells laden with spore-like bodies in the epidermis.
(*b*) Sebaceous gland.
(*c*) Hair sectioned obliquely.
(*d*) Hair follicle.
(*e*) Sebaceous gland in section.
(*f*) Connective tissue, infiltrated with wandering cells.
(*g*) Nodule.
Biondi-Ehrlich stain. No. 4 ocular (Zeiss) and 1-inch objective. Camera lucida.

2. Vacuolated wandering cells. Some showing one and others two nuclei. Biondi-Ehrlich stain. No. 4 ocular, 2 mm. objective (Zeiss). Camera lucida.

3. A field from the circumnodular tissue.
(*a*) Plasma cells.
(*b*) Vacuolated wandering cells.
(*c*) Vacuolated connective tissue cells: Toluidin blue stain. No. 4 ocular, 2 mm. objective. Camera lucida.

4. Spore (?) laden leucocytes, mononuclear and polynuclear forms, lying along fibers of elastic tissue.

PLATE XXI.

Fig. 1. Section through a nodule showing its nearly complete separation from the adjacent tissue. Within the nodule there are appearances of elastic fibers which have become somewhat calcified, presumably by the action of the fungus. No. 4 ocular, a^3 objective. Camera lucida.

2. Cross-section of a blood vessel showing the effects of inflammation and some of the vacuolated wandering cells inside. Biondi-Ehrlich stain. No. 4 ocular, 2 mm. objective. Camera lucida.

Fig. 1.

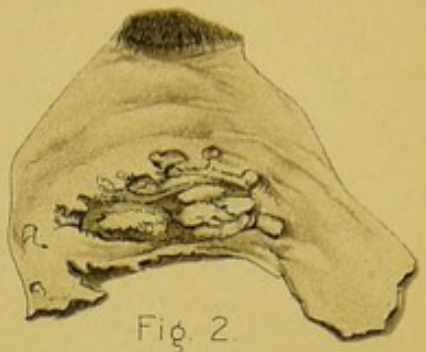


Fig. 2.

Fig. 3.

Haines, del.

LIP AND INFLAMMATORY GROWTH.

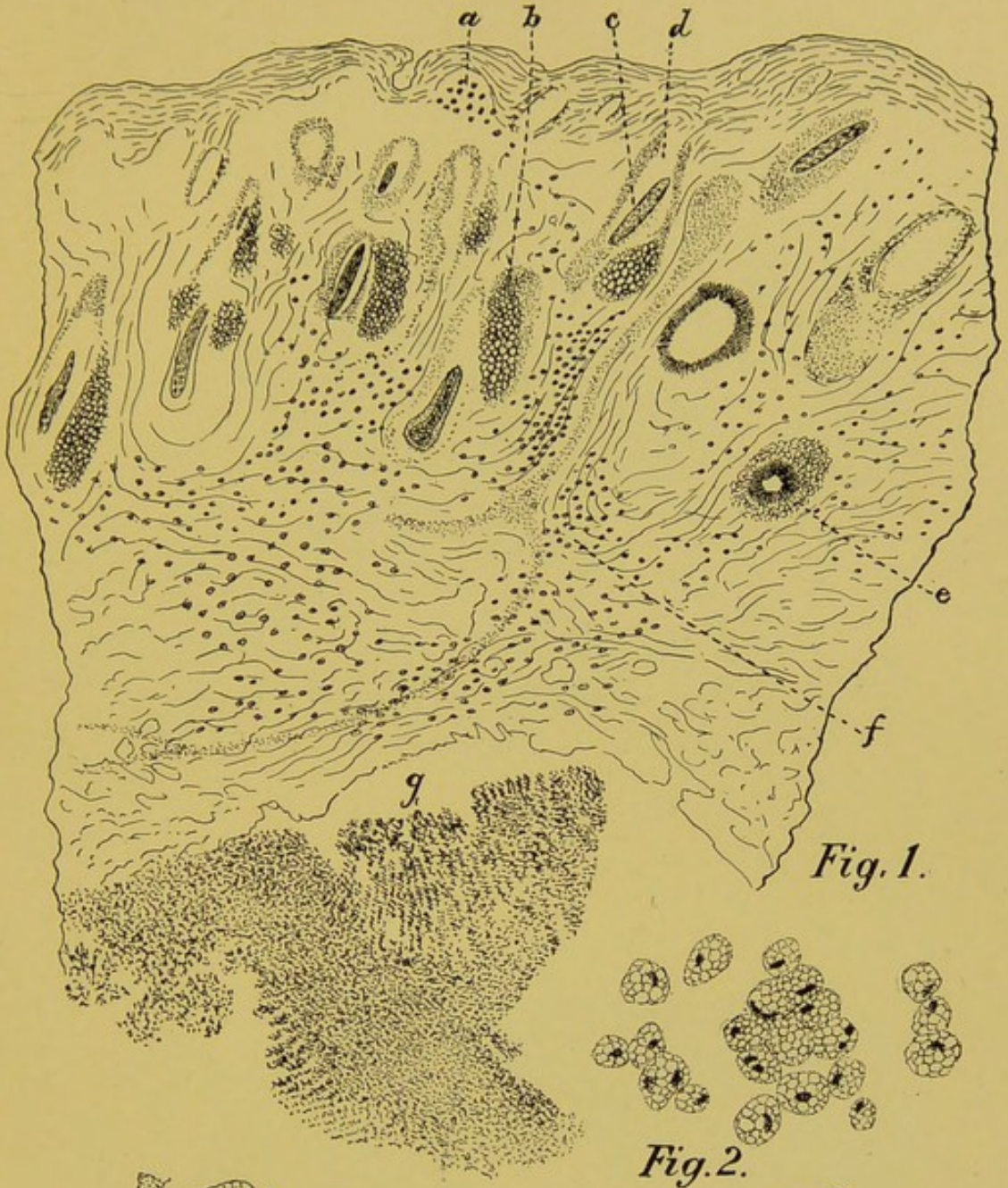


Fig. 1.

Fig. 2.

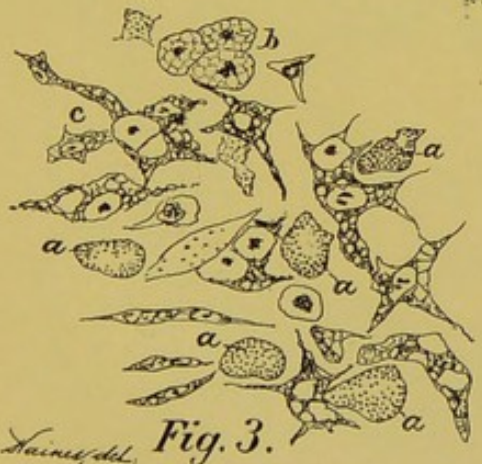
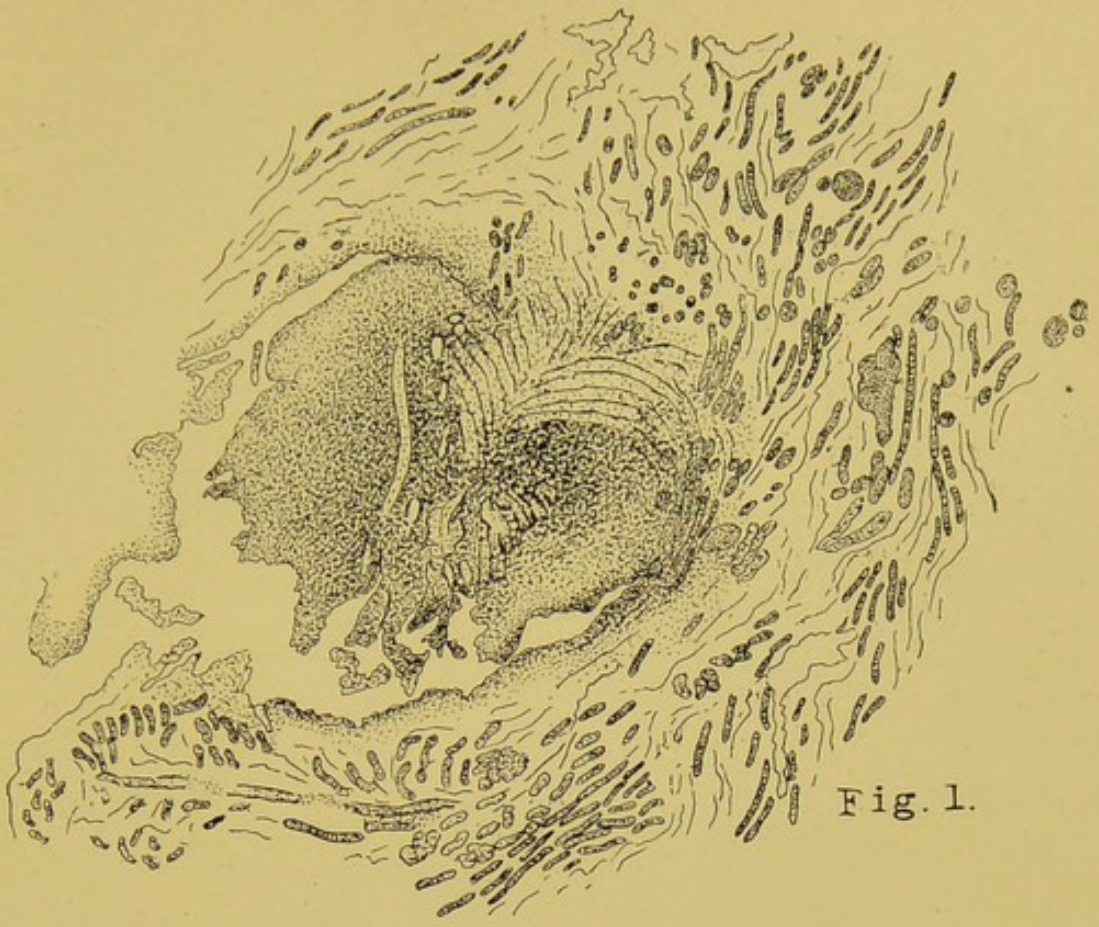


Fig. 3.

Fig. 4.

SKIN AND WANDERING CELLS.



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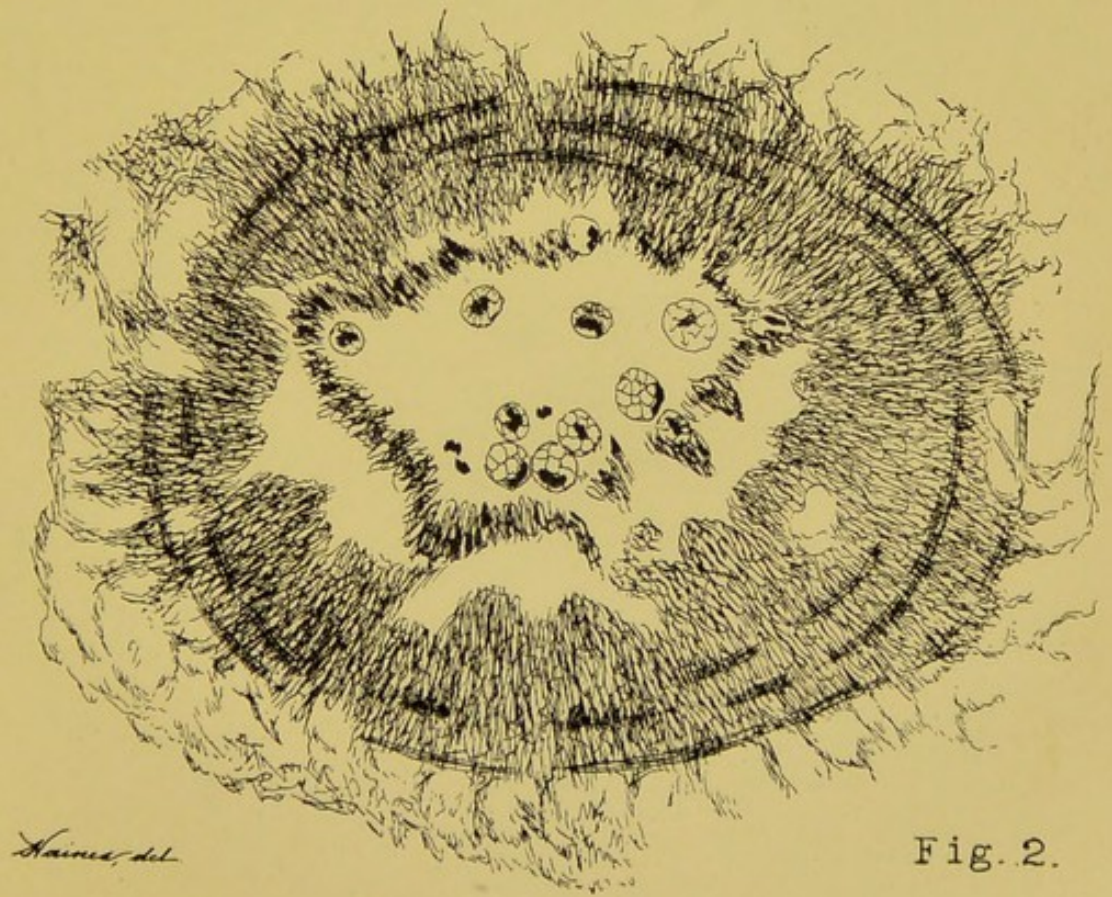
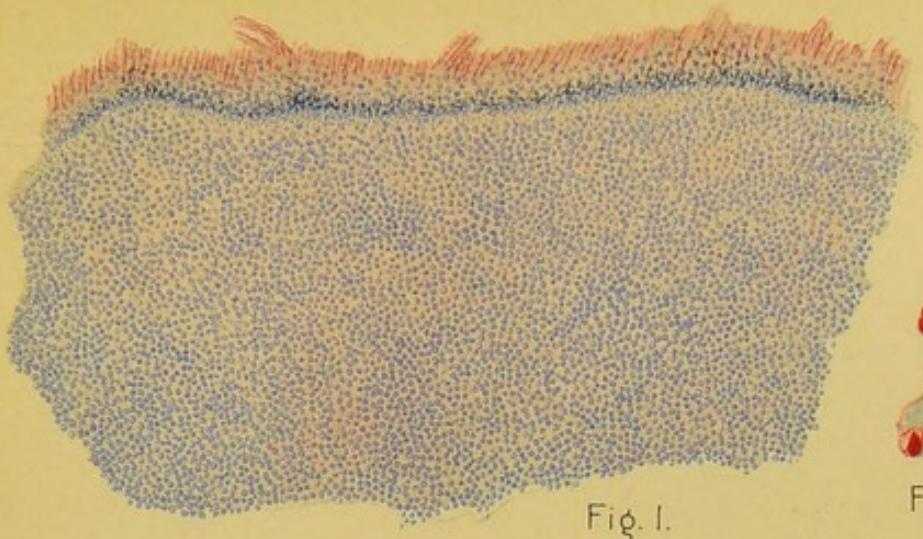


Fig. 2.

NODULE AND BLOOD VESSEL.



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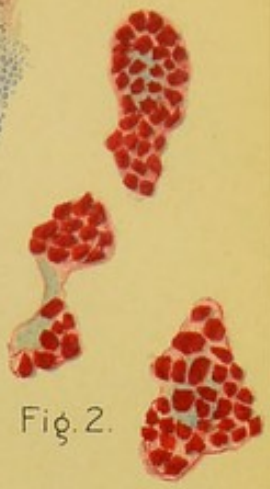

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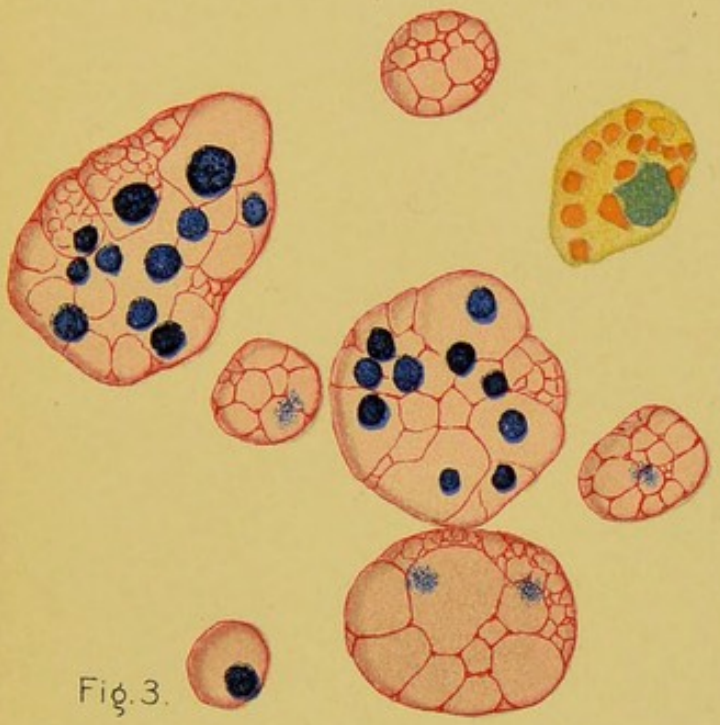

Fig. 3.

Fig. 4.

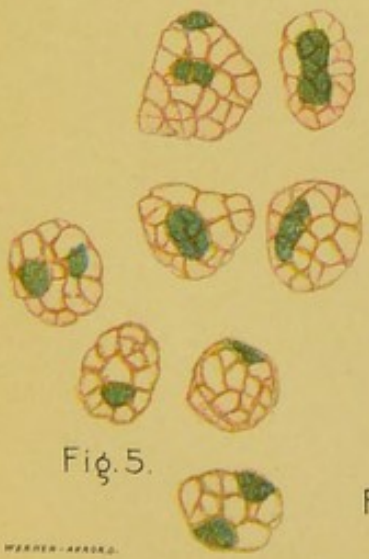

Fig. 5.

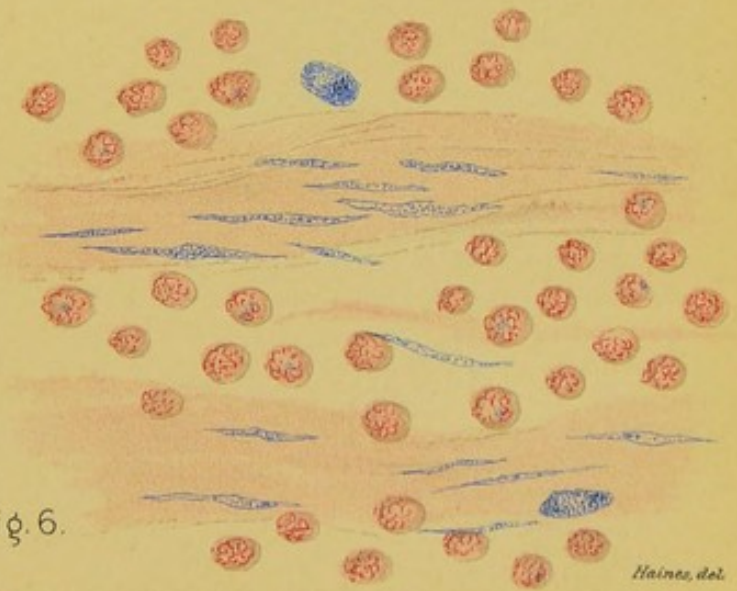

Fig. 6.

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NODULE AND WANDERING CELLS.

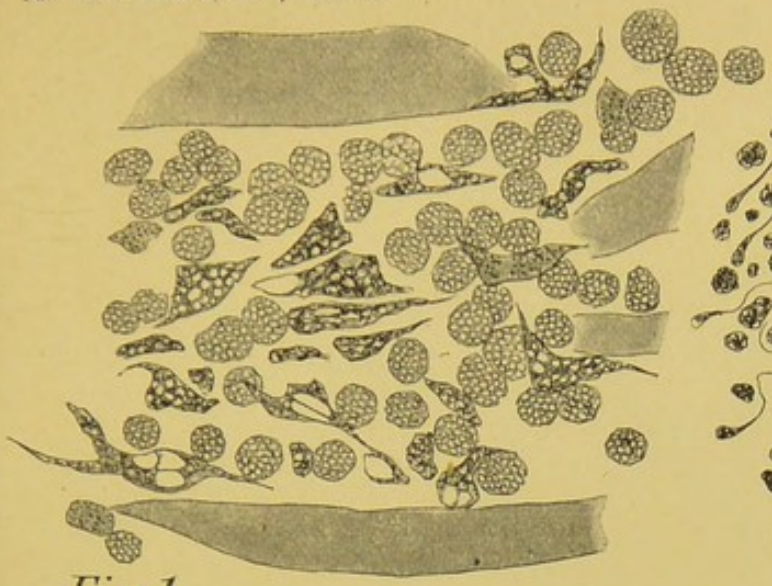

Fig. 1.

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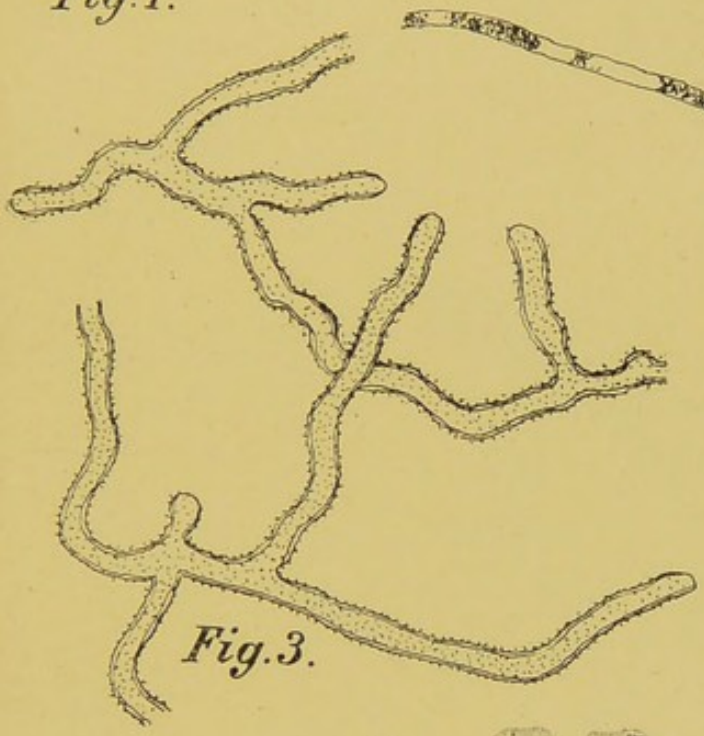

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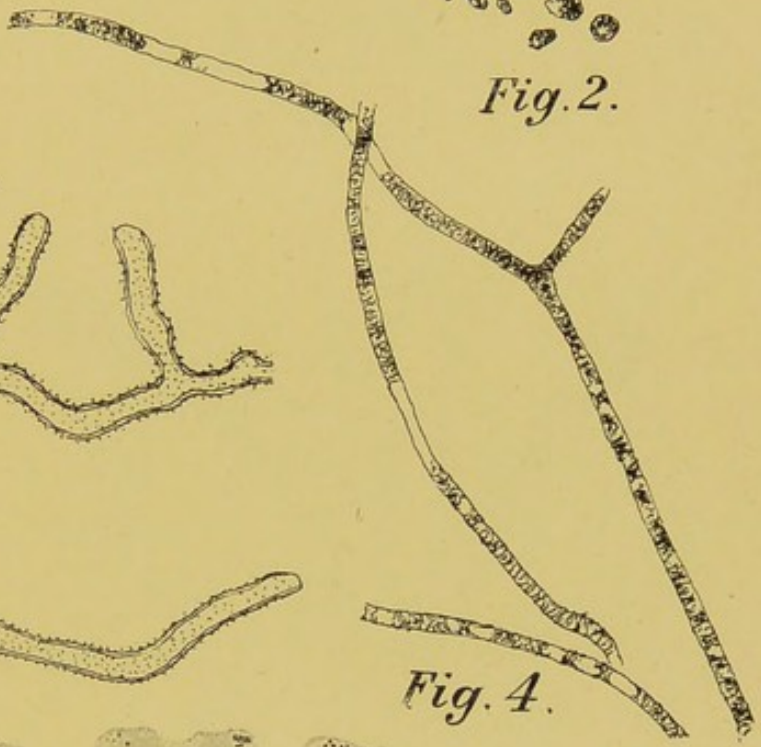

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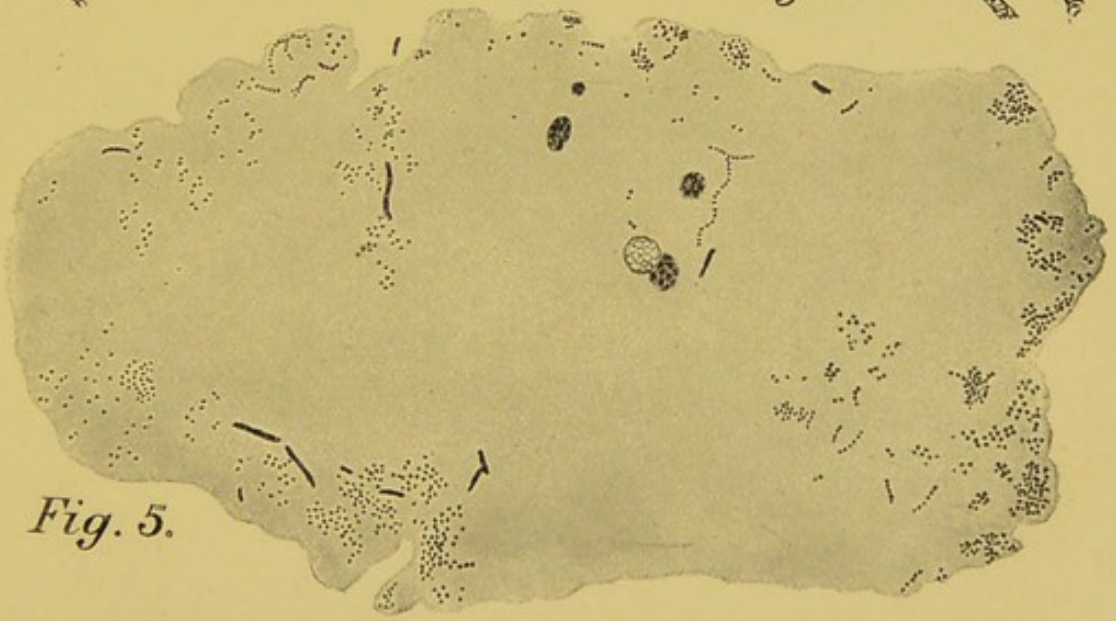

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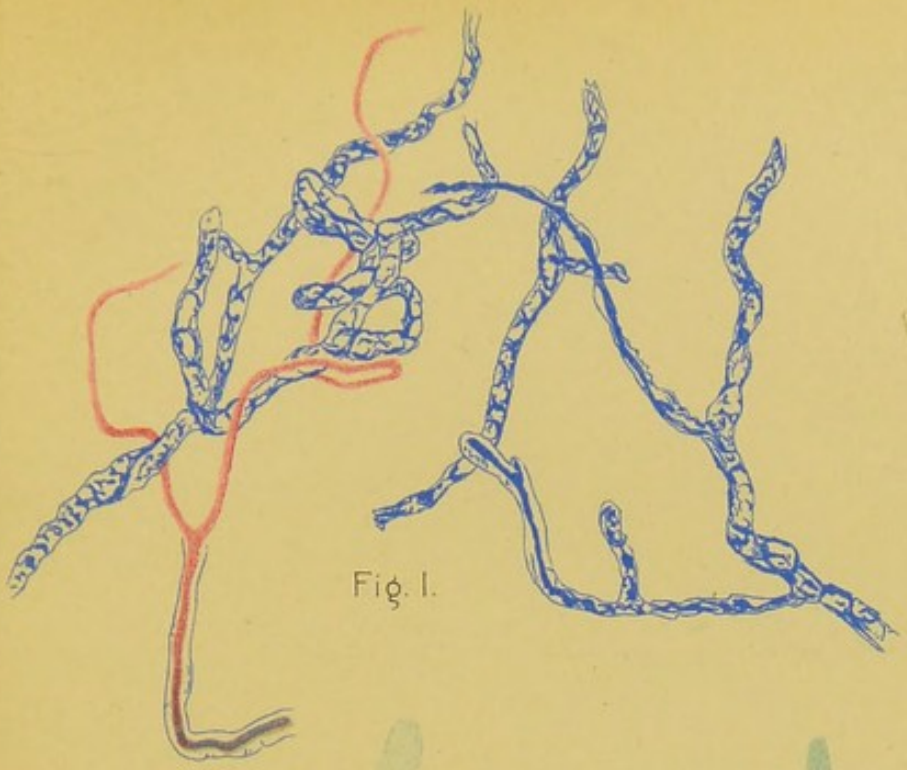

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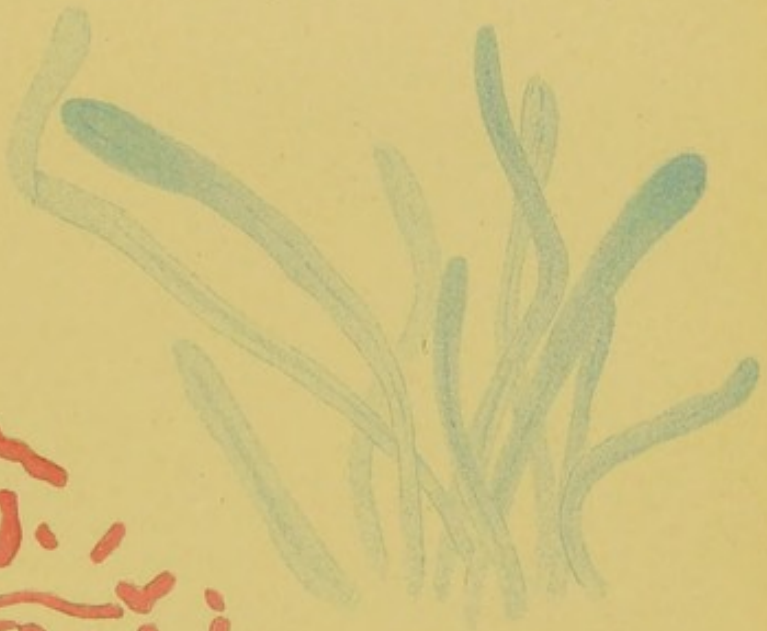

Fig. 3.

Fig. 4.

Fig. 5.

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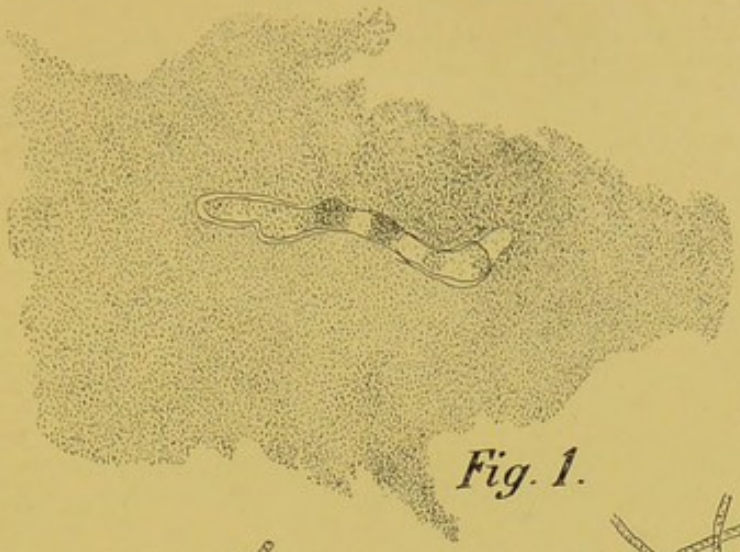
FUNGUS.



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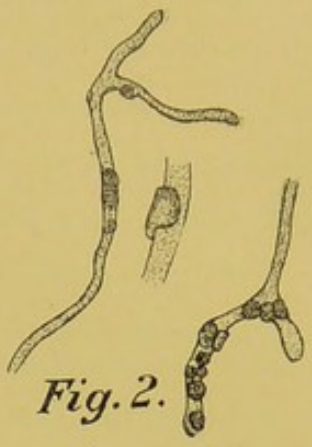

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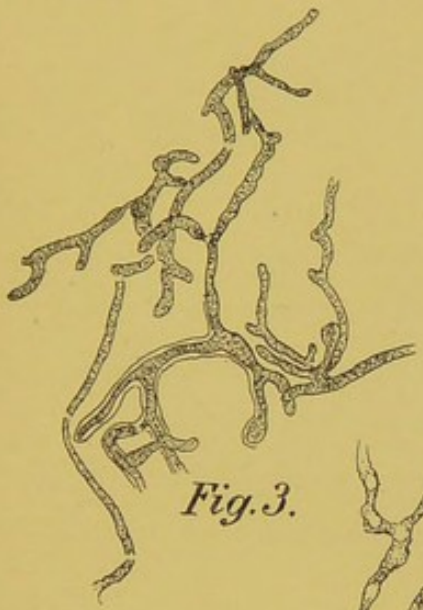

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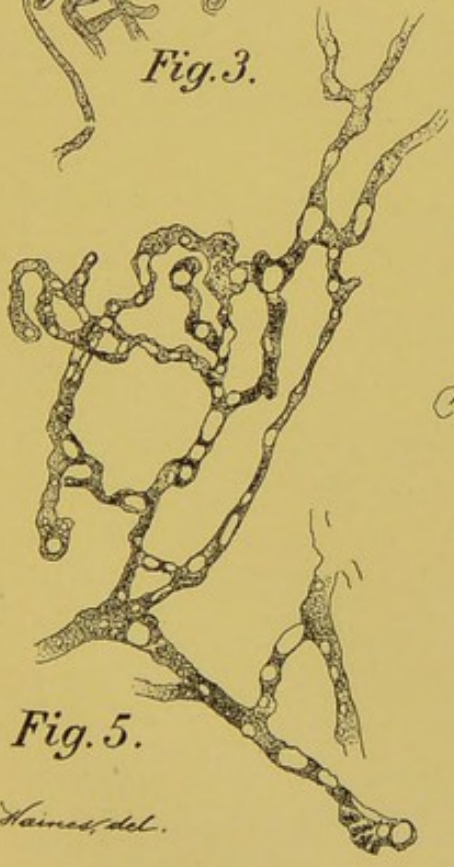

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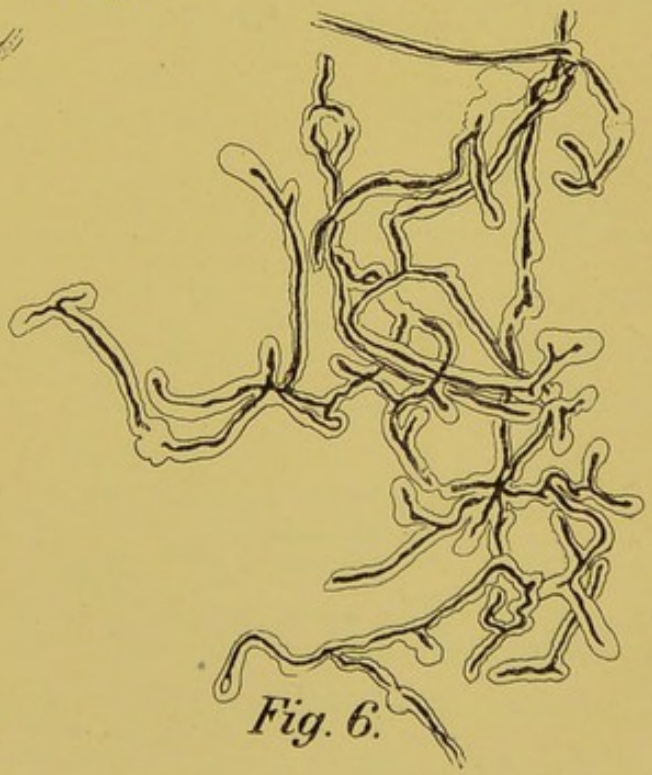

Fig. 6.

PLATE XXII.

Fig. 1. A section through a nodule showing its dense texture, with a portion of its peripheral zone made up of hyphæ with leucocytes interspersed. Methylen blue and eosin stain. No. 4 ocular, 8 mm. objective. Camera lucida.

2. Spore (?) laden leucocytes. Methylen blue and eosin stain. No. 8 ocular, 2 mm. objective. Camera lucida.

3. Giant (?) cells with nuclei deeply stained. Near them are some vacuolated cells with nuclei absent or faintly visible. One mononuclear leucocyte is shown. No. 8 ocular, 2 mm. objective. Camera lucida. Methylen blue and eosin stain.

4. Same as fig. 2, but stained with the Biondi-Ehrlich mixture. No. 8 ocular, 2 mm. objective. Camera lucida.

5. Vacuolated wandering cells, showing the mononuclear, polynuclear, and transition forms. Biondi-Ehrlich stain. No. 8 ocular, 2 mm. objective. Camera lucida.

6. Stroma of connective tissue with infiltrated cells. Methylen blue and eosin stain. No. 4 ocular, 2 mm. objective. Camera lucida.

PLATE XXIII.

Fig. 1. Fibers of elastic tissue with intervening connective tissue and wandering cells, showing the vacuolated condition in the latter and the absence of any distinct nuclei. Methylen blue and eosin. No. 4 ocular, 2 mm. objective. Camera lucida.

2. Portions of a nodule showing disorganized leucocytes, nuclei, and club-shaped fungal elements; their filaments could not be traced to their central connection. Hematoxylin preparation. No. 4 ocular, 2 mm. objective. Camera lucida.

3. Mycelium from a teased preparation. It shows a delicate transparent sheath and roughened surface. Stained with Biondi-Ehrlich mixture. Sketched.

4. Mycelium from a hardened and sectioned preparation. No sheath visible and contents of filament irregular. No. 4 ocular, 2 mm. objective. Methylen blue and eosin stain.

5. Section at the surface of the skin bordering the sore representing the bacilli, staphylococci, and streptococci found. There is also shown one vacuolated and three spore (?) laden leucocytes. Gram-eosin preparation. No 4 ocular, 2 mm. objective. Camera lucida.

PLATE XXIV.

Fig. 1. Involution forms of fungus. Gram-eosin preparation. No. 4 ocular, 2 mm. objective. Camera lucida.

2. Free ends of the fungus; one of them shows with some distinctness an appearance of a septum, another very vaguely represents a spore-like body at the end of the filament. No. 6 ocular, 2 mm. objective. Camera lucida. Hematoxylin and eosin preparation.

3. Free club-like ends (hyphæ) of the fungus. Toluidin blue preparation. No. 4 ocular, 2 mm. objective. Camera lucida.

4. Young forms of the fungus. Hematoxylin and eosin stain. No. 6 ocular, 2 mm. objective. Camera lucida.

5. Free ends of fungus. No. 6 ocular, 2 mm. objective. Hematoxylin and eosin stain.

PLATE XXV.

Fig. 1. Showing a young fungus embedded in the tissue. From the lip. No. 4 ocular, 2 mm. objective. Gram's method. Camera lucida.

2. Showing scale-like bodies embracing the filaments. No. 4 ocular, 2 mm. objective. Hematoxylin preparation.

3. Showing young branches of the fungus, and in one place the transparent sheath. No. 4 ocular, 2 mm. objective. Camera lucida. Hematoxylin preparation.

4. Mycelium with scale-like bodies lying among the filaments. Hematoxylin. No. 4 ocular, 2 mm. objective. Camera lucida.

5. Vacuolated filaments. Hematoxylin. No. 4 ocular, 2 mm. objective. Camera lucida.

6. Branches showing a well-developed transparent sheath. In places the axis of the branch is disconnected and occasionally there is a faint sign of a septum. Hematoxylin. No. 4 ocular, 2 mm. objective. Camera lucida.

PRELIMINARY INVESTIGATIONS CONCERNING THE NUMBER AND NATURE OF BACTERIA IN FRESHLY DRAWN MILK.

By VERANUS A. MOORE, B. S., M. D.,
Chief of Division of Animal Pathology.

It is well known that milk, under ordinary conditions, contains many bacteria. The large number is attributable to the multiplication of the bacteria which gain access to the milk either from the udder and the milk ducts themselves or from external contaminations. Among the latter there is a great variety of fecal and purely saprophytic organisms, some of which are capable of producing fermentations, and occasionally the specific organisms of certain diseases peculiar to mankind which find their way into the milk through contaminated water used in washing the milk utensils or otherwise. Among the former are the specific bacteria of certain diseases of cattle common to the human species, such as tuberculosis and anthrax, which are transmitted from the cow to her milk. There are, furthermore, bacteria which get into the milk ducts and multiply there after each milking, and which necessarily contaminate milk subsequently drawn. It is the extent of this milk-duct infection and the nature of the bacteria involved which especially need further investigation. The danger from the pathogenic organisms attending the indiscriminate use of milk has long since been established, and both the medical profession and the laity are in possession of methods whereby these organisms can be destroyed and the danger, for the greater part, be eliminated.

It is supposed by certain milk bacteriologists that, disregarding the age, the number of bacteria present depends largely upon the amount of dirt and filth which finds its way into the milk after it is drawn from the udder. Passing over the details concerning the well-known channels through which external contamination takes place, we come to the still more interesting fact, namely, that it has been found impossible in the majority of cases to obtain milk free from bacteria when it is drawn directly into sterile flasks and under rigid aseptic precautions. The nature of these bacteria, which constitute the normal bacterial flora of milk, and their effect upon the milk itself and indirectly upon the health of the consumer, have received comparatively little attention. The examinations to determine the number of bacteria in milk as it is delivered by the milkman is legion, but the inquiries into the number and nature of the organisms which gain access to the milk through their localization and multiplication in the milk ducts are exceedingly few. In the milk ducts of the teats are found peculiar conditions respecting temperature and oxygen, which undoubtedly favor the localization of certain species of micro-organisms. These multiply in the ducts, from which they are removed at the subsequent milking. These organisms must necessarily contaminate the milk irrespective of external conditions, although their number and variety

of species will unquestionably depend largely upon the conditions under which the animals are kept.

Among the earlier carefully conducted experiments to determine the extent of the milk-duct infection are those of Leopold Schultz[1] in 1892. He examined the milk bacteriologically at the first of the milking, in the middle of the milking, and at its close. The results of the different tests were quite uniform. The first milk usually contained many bacteria, 97,200 per cubic centimeter being found in one instance in the fore milk. The number at the middle of the milking was much less, the average being 9,000 per cubic centimeter. The cultures from the milk at the close of the milking remained clear in some cases, in others from 500 to 600 bacteria per cubic centimeter were found.

Gernhardt[2] made a series of similar examinations, but with quite different results. He found that the number of bacteria in the milk was at first moderately high, often reaching 600,000 per cubic centimeter. This number rapidly increased, so that the examination made at the middle of the milking showed as high as 7,000,000 bacteria per cubic centimeter, but toward the close the number diminished, so that frequently they were absent from the milk taken at the finish. His suggestions concerning the results are, that the bacteria grew up through the milk ducts of the teats into the udder itself, where they multiply rapidly in the acini of the mammary gland, and that the masses or colonies of bacteria thus produced are not readily removed. Gernhardt found many irregularities in his figures, which he explained on the supposition that in the cases where the number of bacteria were excessively large entire masses or colonies of the organisms were washed into the milk.

Russell states in his Dairy Bacteriology, 1894, that he has been unable to find the last milk perfectly free from bacteria.

At the meeting of the Association of American Physicians in Washington, 1894, Dr. T. M. Rotch presented a paper giving the results of the experiments made by Dr. Austin Peters near Boston, Mass., to determine the number of bacteria in freshly drawn milk. In these experiments every possible precaution was taken to avoid contamination from without. The fore milk and the milk near the close were taken, and also the milk drawn through a sterile cannula passed into the milk duct nearly if not quite through the teat. Four cows were thus examined with the following results:

No. I represents the milk in the first half of milking and drawn by hand directly into sterile bottles.

No. II represents milk drawn through sterile cannula into bottles.

Nos. III and IV represent milk drawn by hand after more than half the udder had been emptied.

The columns marked A, B, C, D, represent the milk from the four cows, the figures showing the number of colonies of bacteria in the plates made from each specimen of milk.

	A.	B.	C.	D.
I	141	167	19 1	53 2
II		6		
III			1	2
IV				

[1] Archiv. f. Hygiene, B. S., XIV (1892). [2] Inaug. Diss. Jurjew, 1893.

Rotch concludes from these examinations that the bacteria found in the cow's milk do not necessarily come from some part of the milk tract between the udder and the end of the teat.

He states further that these experiments provide us with a means of procuring a milk practically sterile without being sterilized. The few colonies obtained in the plates made from the milk in the last half of the milking are considered as possible contaminations between the "cow and the plates." He does not consider the character of the bacteria found in the fore milk, except to state that they were micrococci and small bacilli.

The important conclusion reached by Dr. Rotch that sterilized milk can be obtained by taking the liquid from the last half of the milking is not fully confirmed by the results of the writers previously mentioned, although it is evident that frequently such results would be possible. The results of these examinations, which have led to controverted rather than confirmatory conclusions and have been conducted with animals kept under quite different environments, have the disadvantage of being few in number. Russell has pointed out the interesting fact that the number and kind of bacteria differ according to the treatment the animals have received. He also states that when a reasonable degree of cleanliness is maintained those species of bacteria which predominate in numbers in any sample of milk are derived from the fore milk, i. e., they were present in the milk ducts.

At different times during the past two years I have had opportunity of making bacteriological examinations under favorable conditions of the freshly drawn milk of a few cows. Owing to the infrequency of slaughtering milch cows, I have been unable to make such an examination of the larger milk ducts and the acini of the udders themselves, and consequently facts relative to the assumption of Gernhardt concerning the presence of bacteria in the depths of the healthy glandular tissue have not been obtained. The circumstantial evidence, however, does not support such a theory.

BACTERIOLOGICAL EXAMINATIONS OF FRESHLY DRAWN MILK.

In collecting milk for these examinations the teats and the udders were washed with a solution of 1 to 1,000 corrosive sublimate. The hands and arms were likewise disinfected, and care was exercised to prevent the stirring up of dust. The milk was drawn directly into sterilized bottles. As yet a series of examinations from the milk drawn at different periods of the milking have not been made, as these examinations were made to collect facts bearing directly upon the determination of the number, nature, and constancy of the bacteria in the fore milk and the frequency with which sterilized milk could be obtained, if the milk during the last part of the milking was drawn directly into properly prepared flasks. In collecting the milk, from 5 to 10 c. c. of the fore milk was taken in separate bottles from each quarter of the udder and about 50 c. c. near the close of the milking except in the first five cases. The milk was taken directly to the laboratory and agar plates (in a few instances gelatin plates also) were made with definite quantities (from 0.1 to 1 c. c.) of the milk. The results of the cultivations are summarized in the following tables.

The number of bacteria per cubic centimeter in freshly drawn milk taken under aseptic precautions, as determined by the number of colonies on agar plates.

Cow number.	Colonies in the first milk.				Colonies in the last milk.			
	Quarter of the udder.							
	1	2	3	4	1	2	3	4
1	0	6	6,625	1,750	.....	.....	.....	.....
2	7	Fungi.	0	Fungi.	.....	.....	.....	.....
3	180	3,840	1,200	18	.....	.....	.....	.....
4	5	1,200	(*a*)	5	.....	.....	.....	.....
5	0	3,840	6	12	.....	.....	.....	.....
6	Very many.	Very many.	0	600	1,800	2,400	0	0
7	Very many.	7,200	8,400	940	546	0	504	0
8	455	0	390	0	2	0	0	0
9	4	8	0	8	0	4	0	0
9*b*	230	5	120	5	0	0	0	0

a Surface of plates grown over.

Gelatin plates.

Cow number.	Colonies in first milk.				Colonies in last milk.			
	Quarter of the udder.							
	1	2	3	4	1	2	3	4
1	Fungi.	Fungi.	1	690	.....	.....	.....	.....
2	280	0	0	0	.....	.....	.....	.....
3	0	0	0	0	.....	.....	.....	.....
9	8	4	0	8	20	0	0	44
9*b*	20	0	0	5	120	0	0	15

Cow No. 9*b* is the same animal as No. 9, the second examination being made about three weeks after the first.

It is a noticeable fact that in every case bacteria were found in the last milk taken from one or more quarters of the udder. While it is possible, as presumed by Dr. Rotch, that they are the result of contamination between the "cow and the plates," these results, as well as those heretofore reported, show an almost constant appearance of bacteria, often in small numbers to be sure, in the milk taken during the last part of the milking. Basing an opinion on the data in hand, it appears that the last milk can not be considered, generally speaking, free from micro-organisms, although it undoubtedly exists in such a condition in many instances. In nearly all the cases the bacteria in the last milk are so few that it is highly probable that they would not produce any appreciable effect upon the normal condition of the fluid if it is cooled rapidly, immediately after milking, to a temperature below that necessary for their multiplication. It is well known that numerous saprophytic bacteria, such as many of the water forms, will not multiply at the temperature of the animal body, and certain highly parasitic organisms like the tubercle bacillus fail to develop when placed in a temperature much below that of the living blood. Thus certain bacteria which gain access to and multiply in the milk ducts are unable to do so when placed in a low temperature where many purely saprophytic organisms thrive luxuriantly.

The properties of the bacteria found in the fore milk are of more importance than their numbers. It has been stated that the species of bacteria which predominate in milk when taken under ordinary conditions are those contained in the fore milk. Russell states that the organisms found in the teat are those that produce lactic acid.

Bolley and Hall, of the North Dakota Experiment Station, made a qualitative test of the fore milk of 10 healthy cows. They report 16 species, 12 of which produced an acid reaction in milk, 3 produced an alkaline reaction, and 1 species produced no appreciable change. They state that gas-engendering organisms were not found.

A thorough differential study of the bacteria which I have found in freshly drawn milk has not been completed. In all, 20 apparently different species were isolated. Of these, 3 were streptococci, 4 bacilli, and 13 micrococci. Among the bacilli *Bacillus cloacæ* occurred in one specimen. It is possible, as it occurred but once, that it was an accidental contamination from external sources. This was the only gas producer in the entire series. Nine of the apparent species were aerobic, indicating their presence near the end of the teat or milk duct, and the others were faculative anaerobic and could have come from much higher up in the teat.

With one exception the organisms isolated fermented lactose in both bouillon to which chemically pure lactose had been added, and in milk itself, giving a decidedly acid reaction. A chemical analysis has not been made to determine the nature of the acids produced, but it is presumed that lactic acid predominated. Six of the twenty species produced a firm coagulation of the milk within twenty hours. The others precipitated or coagulated the casein in from four to ten days. A few of the milk cultures emitted a very strong "sour-milk" odor, while the odor of the others was not perceptible or of a pungent or disagreeable nature. Eleven of these organisms liquefied gelatin. In some instances liquefaction occurred rapidly, in others more slowly, several days being required to complete the process.

The pathogenesis of these organisms was tested by the subcutaneous inoculation of 0.5 cc. of a fresh bouillon culture into guinea pigs, but invariably the animals remained well. In fact, a perceptible local lesion was not produced in any case.

Although there was a marked morphological difference in certain of the organisms found in these examinations, there was a striking resemblance between them in their behavior when subjected to certain physiological tests. The fact that several of them were very active in their power to ferment sugars offers a valuable suggestion concerning the time for sterilizing milk. As the number of bacteria in freshly drawn milk indicate that the multiplication of the fore-milk bacteria is progressing rapidly at the time of milking, there is every reason to suppose that this process continues until the milk is cooled to a low temperature, and if it is not brought to a point below that possible for their multiplication they continue to multiply, and consequent important chemical changes are produced in the milk. In view of these facts it seems very important that milk should be pasteurized, if its normal composition is to be retained, as soon as possible after being drawn. Milk that has stood under ordinary conditions for from four to twelve hours before its bacterial flora is destroyed must necessarily have undergone more or less deleterious changes, the extent of these alterations depending upon the temperature at which it is kept and the number and nature of the bacteria in the fore milk. It is well known that all milk pasteurized after it is delivered by the milk vender does not agree with all children. It is furthermore asserted that the milk from certain animals, and, in fact, entire dairies, is objectionable on account of its rapidly becoming acid. Undoubtedly the food of the cattle has much to do with the

quality of the milk, but the changes referable to the fermentation of the milk sugar are unquestionably dependent upon the action of certain of the fore-milk bacteria.

Although a considerable number of apparent species were found in the plate cultures, it should be stated that the majority of them occurred but once or twice, and then only in small numbers. All of the rapidly acid-producing organisms and those which coagulated the casein with the sour-milk odor were micrococci, and these were the forms most frequently encountered. They were destroyed at a low temperature, 50° to 56° C., when exposed for a period of twenty minutes. Spores were not found in any of the bacilli. Until an opportunity is afforded for a bacteriological study of the ducts and the acini of the mammary glands themselves, many important questions concerning the natural bacterial flora of milk must remain unanswered. These preliminary observations, considered in conjunction with the results of other investigations, lead to the following conclusions:

1. Freshly drawn fore milk contains a variable number of bacteria, varying in number from a few individuals to many thousand per cubic centimeter. These are distributed among several species. The last milk drawn at a regular milking contains, as compared with the fore milk, very few micro-organisms. It is the exception, however, to find a sample of milk that is free from micro-organisms unless it is taken during the latter part of the milking process from a single quarter of the udder.

2. The bacteria which become localized in the milk ducts, and which are necessarily carried into the milk are for the greater part rapidly acid-producing organisms, i. e., they ferment milk sugar forming acids. They do not produce gas. *Bacillus coli communis* and other gas-producing bacteria sometimes found in market milk are presumably the result of external contamination.

3. The fact that sugar-fermenting bacteria are ordinarily present in freshly drawn milk renders it necessary, if its normal composition is to be retained, that it should be pasteurized as soon as possible after it is drawn. This is necessary, regardless of the cleanliness of the stable, milking utensils, and surroundings generally. Much of the reported intestinal trouble arising from feeding infants ordinarily pasteurized milk is undoubtedly due to the presence of acids produced before the pasteurization by sugar-fermenting bacteria derived from the milk ducts.

A REPORT ON RABIES IN WASHINGTON, D. C.

By VERANUS A. MOORE, B. S., M. D., and PIERRE A. FISH, D. Sc., D. V. S.

The presence of rabies in the city of Washington was recognized in the spring of 1893. Special attention was drawn to this subject at that time on account of an investigation of an outbreak of rabies in cattle,[1] in which it was found desirable to make a few comparative inoculations with the virus of rabid dogs. This desire was made known to Dr. F. L. Kilborne, then director of the experiment station of this Bureau, and he requested several veterinarians in the city of Washington to bring all dogs suspected of rabies to the experiment station. As a result a considerable number of supposed rabid dogs were brought to the station or shot by Dr. Kilborne himself from March 24 to December 12, 1893. From the entire number eleven were demonstrated to be rabid.[2] In December, 1892, a negro died of rabies in Georgetown, and in the following January a horse was killed for this disease in Washington.[3]

As the investigations requiring virus from rabid dogs were closed in the summer of 1893, no further effort was made to procure cases, and very few were voluntarily reported. In the fall of 1895, however, a fatal case in a lady was reported by Dr. Behrend at the Medical Society of the District of Columbia. This case again attracted general attention to the disease, and arrangements were made by the Health Officer of the District of Columbia with the Chief of the Bureau of Animal Industry whereby all dogs or other animals suspected of having rabies were to be sent to this laboratory for the purpose of making a positive diagnosis. From September 27, 1895, to August 1, 1896, a period of three days more than ten months, 15 suspected dogs and 2 foxes were received. Unfortunately 3 of the dogs did not arrive until from one to two days after they had either died or been killed, and were consequently decomposed to such an extent that rabbit inoculations were precluded. Rabbits inoculated with the brains of two dogs which were thought to be sufficiently fresh to warrant the operation died of septicæmia within twenty-four hours. Thus in 5 of the dogs a diagnosis was rendered impossible. Of the remaining 10 dogs 8, or 80 per cent, and the 2 foxes, were demonstrated by the inoculation test to have had rabies.

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[1] Moore: A Disease in Cattle not Distinguishable from Rabies, Bulletin No. 10, B. A. I., Department of Agriculture, 1896, p. 71.

[2] As Dr. Kilborne made the majority of the original inoculations, we do not know the exact number from which negative results were obtained, but from the notes left at the laboratory it is evident that there were several.

[3] The diagnosis in these cases was made from rabbit inoculations in this laboratory.

The diagnosis of rabies in the street dog is an exceedingly difficult, if not impossible, task without the aid of the inoculation test. The variety of symptoms manifested by dogs affected with this disease and the occasional temporary existence of peculiar actions in healthy animals, especially in the summer season, formerly led to more or less controversy concerning the specific nature of this disease. However, when the large number of experiments made by Pasteur, and repeated by others proceeding after the same methods, were reported the distinctive character of the disease was generally admitted. These experiments showed that if rabbits were inoculated with a small quantity of a suspension of the brain or spinal cord of a rabid dog they would die in a certain length of time with definite and characteristic paralytic symptoms, while rabbits similarly inoculated with a suspension of the brain of healthy animals would remain well. Although the specific agent has not been isolated or studied in pure culture, the repeated inoculation experiments have demonstrated its specific nature and shown that rabies is transmissible from animal to animal by means of the bite of the affected individual or by direct inoculation. It has also been found that the saliva of the dog is virulent for a few days before the animal shows visible symptoms of the disease. While the accuracy of the diagnosis of rabies made from the apparent symptoms may frequently be questioned, when the diagnosis is based on the results obtained from animal inoculation it is as positive and reliable as any fact in biological or medical science.

In view of the fact that a few physicians have put themselves on record as disbelievers in the specific nature of rabies, and various publications have echoed their utterances with an air of triumph, it is important to again call attention to the methods for diagnosing this disease. The characteristic feature in the argument of those who wish to hold up rabies as a delusion is that "they have never seen a case," or if they have made inoculations they have ignored the length of time necessary for the development of the symptoms, and based their results and condemnatory epithets concerning the method upon the actions of the animals immediately after the inoculation. This is strikingly illustrated in the experiments made by Spitzka,[1] who attempted to reproduce symptoms of rabies by introducing nonrabic substances, and who recorded the unusual actions of the inoculated dogs during the first few days, but failed to give the final results. In these cases partial paralysis was noted within the first six days, which would indicate cerebral injury or pressure due to the effects of an unsuccessful operation or of the irritant or septic substances introduced. As these experiments were apparently made to discountenance the positive results by the inoculation test, it is only necessary to call attention to the all-important fact that in Liautard's case quoted by Spitzka the symptoms of rabies in the inoculated dog began on the sixteenth day and led to a fatal termination on the nineteenth day, which is in accordance with the results obtained by Pasteur and others, while Spitzka merely recorded the symptoms for the few days immediately following the inoculation. There is no evidence more conclusive than rabbit inoculations, made with aseptic precautions, from the brain or spinal cord of a rabid dog and followed by the different stages of paralysis which have developed after the proper period of incubation. Until after making these test inoculations no one is

[1] The Journal of Comparative Medicine and Surgery, Vol. VII (1886), p. 244.

justified in asserting with sweeping denials that the method is false and that the disease is a myth.

As rabies is not always readily detected, it is not strange that many physicians have never seen, or at least have never recognized, a case. Fleming,[1] who at one time considered this disease of little importance, discovered upon investigation that it was of much greater significance than he had formerly supposed, finding, as he did in 1866, 66 fatal cases in men in England alone. It is true with rabies, as it is with other contagious diseases, that all of the individuals inoculated do not become affected. It is also true that many dogs are supposed to be mad when they are not, and, conversely, many dogs, in which the disease is never suspected, die of rabies.[2] It is not rational, therefore, that one's belief in the disease should rest on either of the extremes. The facts are clear, and, when considered in the light of modern methods, are easily explained. An inherent dread of rabies exists in the minds of a few people and perhaps it may have led, in rare instances, to hysteria, and finally to fatal results. Fear of other diseases has led in some cases to such unfortunate results, and it may be freely admitted that the same may occur from fright concerning this probably the most dreaded of all maladies. It would be absurd, however, to conclude that because some persons have died from fear of rabies, the disease which they feared does not exist. The statement, so frequently made, that the imagination is the predisposing cause of the disease in man is not applicable in the case of animals where this attribute as an etiological factor may be safely eliminated. The fact can not be denied that since Aristotle described rabies there have been recorded in human history innumerable deaths, preceded by certain uniform and definite symptoms following the bite of rabid dogs and other animals. Furthermore, many more animals have succumbed from the same cause and they also have manifested uniform symptoms within well-defined limits. The examination of the cases which have come to this laboratory for diagnosis during the past year and which were pronounced rabies by different people in the city of Washington affords an excellent illustration of the existing facts concerning false and true rabies.

METHOD OF RABBIT INOCULATION FOR THE DIAGNOSIS OF RABIES.

The method of diagnosing rabies which we have followed, and which the experience of pathologists has shown to be the best, is the subdural inoculation of rabbits with a suspension of the brain or spinal cord of the suspected animal. The subdural inoculation with the brain tissue of rabid animals was first demonstrated by Pasteur to be more reliable and more rapid in its results than the subcutaneous injections. The procedure is simple. The brain of the suspected animal is removed with aseptic precautions as soon as possible after death. A small piece of the brain or spinal cord is placed in a sterile mortar and thoroughly ground with a few cubic centimeters of sterilized water or bouillon. This forms the suspension to be injected.

[1] Rabies and Hydrophobia. London, 1872.

[2] This condition is well illustrated in a case which occurred in Washington in the spring of 1893. A lady whose pet dog appeared to her to be suffering from indigestion, wrapped it in a blanket and carried it in her arms to a veterinarian. The dog died of rabies on the following day, and rabbit inoculations showed it to have died of a very virulent form of the disease.

The hands of the operator and all instruments are carefully disinfected. The rabbit is etherized, the hair clipped from the head between the eyes and ears, and the skin thoroughly washed and disinfected. A longitudinal incision is then made, the skin and subcutaneous tissue held back by means of a speculum, a crucial incision is made in the periosteum on one side of the median line to avoid hemorrhage from the longitudinal sinus, and the four parts of the periosteum reflected or pushed back. By the aid of a trephine a small button of bone is easily removed leaving the dura mater exposed. With a hypodermic syringe a drop or more of the rabid brain suspension is injected beneath the dura, the periosteum is replaced, the skin carefully sutured and disinfected, and the rabbit returned to its cage. As soon as the influence of the anæsthetic[1] has passed off the rabbit shows no appearance of discomfort. If the operation is performed in the forenoon the animal partakes of its evening meal with the usual relish. The inoculation wound heals rapidly and the rabbit exhibits every appearance of being in perfect health until the beginning of the specific symptoms, which occur ordinarily in from fifteen to thirty days after the inoculation. Occasionally the symptoms appear earlier than fifteen days, and in some cases the rabbits are not attacked for from one to three months.

Symptoms.—The symptoms following our inoculations have been quite uniform, the only pronounced difference being in the length of time the rabbits lived after the initial manifestation of the disease. The fact should be clearly stated that rabbits do not ordinarily become furious. In some instances they are somewhat nervous for a day or two preceding the paralysis. There appears to be marked hyperæsthesia. Usually the first indication of the disease is a partial paralysis of one or both hind limbs. This gradually advances until the rabbits are completely prostrated, the only evidence of life being a slight respiratory movement. The head occupies different positions. In some it is drawn backward as in tetanus; in others it is drawn down with the nose near the fore legs; and in still others it is extended as if the animal were sleeping. The period of this complete paralysis varies from a few hours to a few days, but ordinarily it has not exceeded twenty-four hours. Although these animals were unable to move voluntarily there was a reflex action of the limbs until a very short time before death.

During the period of incubation the temperature of the rabbits was normal. As the time approached for the first symptoms to appear there was, in the animals tested, an elevation of temperature of from 1 to 2 degrees, which continued for a variable length of time, but rarely longer than two days. This was followed by a gradual or usually a more rapid drop to the subnormal, which continued to the end. Dr. J. J. Kinyoun, of the Marine-Hospital Service, tells us that in his experience the sudden rise and fall of temperature in rabbits suffering from rabies have been constant.

The differential diagnosis in experimental animals is not difficult. Rabbits inoculated with several varieties of pathogenic bacteria frequently exhibit symptoms of paralysis for a brief period preceding death. In cases of injury to the brain or spinal cord there may also be

[1] Ether should be used in preference to chloroform for rabbits, as the latter frequently causes death, while the former can be administered with comparative safety.

paralysis, which in the absence of the history of the case might be taken for that of rabies. In these cases, however, the symptoms appear very soon after inoculation. This is especially true when the paralysis is due to mechanical injury of the brain or to irritating or septic substances, and in the case of the pathogenic bacteria if paralysis occurs at all it is almost invariably preceded by marked disability. This method of diagnosing rabies requires that the inoculated animals remain apparently well for a considerable length of time after the subdural inoculation and before the paralytic symptoms appear.

The lesions found on the post-mortem examination are also of much assistance in making a diagnosis. If the animal died from septicæmia or brain injury there will be lesions almost invariably recognizable in the brain or viscera. In the case of septicæmia a bacteriological examination will reveal the presence of micro-organisms. If the death was caused by rabies the inoculation wound in the head should be healed perfectly, there should be no abscess, and the meninges should be free from exudates and the brain itself should appear perfectly normal, except that in rare cases there may be a slight injection of the blood vessels. The viscera are ordinarily normal in appearance, with possibly the exception of the liver, which we have frequently found to be deeply reddened, and the gastric mucosa, which not infrequently shows dark patches, indications of disintegrated hemorrhagic areas. A bacteriological examination fails to reveal the presence of micro-organisms in either the tissues or blood. Another important point which we have noticed is the intense rigor mortis following death from rabies. Dr. Kinyoun says that this was a constant feature of this disease in all of the produced cases which have come under his observation.

A DETAILED ACCOUNT OF THE CASES RECEIVED.

The dogs suspected of having rabies and which were brought to this laboratory for the purpose of diagnosis were in every instance dead when received. Some of them had died; others had been killed. The diagnosis of rabies had been made by the owners of the affected animals or by the policeman who officiated as executioner. A few of the animals had been examined by veterinarians, who pronounced them rabid. In four cases the animals were much decomposed when received. In a few instances the dogs were sent to the experiment station of the Bureau, where Dr. Schroeder removed the brains and immediately sent them to this laboratory. Short delays in the inoculation of rabbits were occasionally necessary, but in these the suspected brains were kept on ice until used. Before injections were made, however, the substance was warmed to near the body temperature.

The history of the dogs is in nearly every instance very meager, the only point of interest obtained being the belief on the part of one or more individuals that the dog was mad.

Two rabid foxes were received from the Zoological Park. These are interesting. The source of infection was not determined in the first one; in the second fox the disease was produced by the bite of the first. Rabbits were not inoculated from the brain of the second fox, as the history and symptoms were sufficiently conclusive to indicate the nature of the disease.

The results of the inoculations are given in the following table:

Dogs and foxes received for diagnosis with data concerning the rabbit inoculations.

No.	Breed.	Date of death.	When received.	Inoculations.		
				Rabbit No.—	Date.	Result.
			1895.		1895.	
1	Mastiff	Sept. 26, 1895 (skull crushed).	Sept. 27	28	Sept. 27	No. 28 died of paralytic rabies Oct. 14, 1895.
				30	....do....	No. 30 died of paralytic rabies Oct. 16, 1895.
2	Irish setter	Nov. 22, 1895 (died).	Nov. 22	58	Nov. 23	No. 58 died of paralytic rabies Dec. 11, 1895.
				59	....do....	No. 59 paralyzed Dec. 11; chloroformed; would have died in a few hours.
				a 85	....do....	Guinea pig died of paralytic rabies Dec. 13.
3	Large mastiff	Nov. 30, 1895 (shot in head).	Nov. 30	62	Nov. 30	Negative.
				63	....do....	Do.
4	Irish setter	Dec. 13, 1895 (shot in head).	Dec. 13	12	Dec. 13	Died of paralytic rabies Dec. 25.
5	Eskimo	Dec. 14, 1895 (died).	Dec. 14	22	Dec. 14	No. 22 died of paralytic rabies Feb. 12, 1896.
				32	....do....	No. 32 died of paralytic rabies Feb. 11, 1896.
6	Collie	Dec. 19, 1895 (shot in head).	Dec. 19	4	Dec. 19	No. 4 died of septicæmia May 18, 1896. *b*
				5	....do....	No. 5 died of septicæmia Dec. 24, 1895.
			1896.		1896.	
7	Mastiff	Feb. 7, 1896 (shot).	Feb. 8	77	Feb. 8	No. 77 negative (alive).
				78	....do....	No. 78 died of rabies Mar. 1, 1896.
8	Brown setter	Apr. 8, 1896 (died).	Apr. 9	90	Apr. 10	No. 90 died of paralytic rabies May 13, 1896.
				91	....do....	No. 91 died of paralytic rabies May 17, 1896.
9	.....	Apr. 23, 1896 (died).	Apr. 24	.....	.....	Brain decomposed. No inoculation.
10	.....	May 8, 1896 (died).	May 9	108	May 9	No. 108 died of septicæmia May 10, 1896.
				109	....do....	No. 109 died of septicæmia May 10, 1896.
11	.....	May 21, 1896 (shot).	June 1	.....	.....	Brain decomposed. No inoculation.
12	Fox terrier	June 1, 1896 (shot).	June 2	.....	.....	Do.
13	Collie	June 1, 1896 (died).	June 18	106	June 18	No. 106 alive Aug. 31.
				127	....do....	No. 127 died of paralytic rabies July 14, 1896.
14	Small white poodle.	July 19, 1896 (shot in head).	July 20	118	July 20	Rabbit alive Sept. 10, 1896.
15	Small black terrier.	July 31, 1896 (skull crushed).	July 31	151	July 31	No. 151 died of paralytic rabies Aug. 29, 1896.
				152	....do....	No. 152 died of paralytic rabies Aug. 29, 1896.
			1895.		1895.	
1	Red fox	Nov. 10, 1895 (died).	Nov. 10	51	Nov. 11	No. 51 died of paralytic rabies Dec. 3, 1895.
				c 52	....do....	No. 52 died of paralytic rabies Jan. 18, 1896.
d 2	.....do.....	Nov. 21, 1895.....	Nov. 21			

a Guinea pig.
b This rabbit was badly bitten on its back by another rabbit put in same cage about May 1. It is highly probable that this was the source of infection.
c Inoculated subcutaneously with part of parotid gland.
d Rabbits were not inoculated, as this fox was bitten by No. 1.

The facts brought out in the above table are exceedingly interesting. Of the 15 dogs received 3 were so badly decomposed that rabbits were not inoculated, and those inoculated from 2 others died of septicæmia on account of the bad condition of the brain. A diagnosis was not made in five cases. Of the remaining 10, in which a diagnosis was

made, 8 (or 80 per cent) unquestionably had rabies. The details concerning the fatal cases are more fully brought out in the following table:

Details concerning the disease in rabbits caused by the inoculation with the brain from rabid dogs (street rabies).

Virus from dog—	No. of rabbit inoculated.	Date of rabbit inoculation.	Date of first symptoms.	Date of death.	Period of incubation.	Duration of symptoms (paralysis).	Time from inoculation to death.
		1895.	1895.		*Days.*		*Days.*
No. 1	28	Sept. 27	Oct. 14	Oct. 15	17	About 30 hours	18
	30	do	Oct. 15	Oct. 16	18	do	19
No. 2	58	Nov. 23	Dec. 11	Dec. 11	18	Less than 24 hours	18
	59	do	do	do	18	(*a*)	19
No. 4	12	Dec. 13	Dec. 24	Dec. 25	11	About 30 hours	12
			1896.				
No. 5	22	Dec. 14	Feb. 11	Feb. 12	58	do	59
	23	do	Feb. 9	Feb. 11	56	About 48 hours	58
		1896.					
No. 7	77	Feb. 8					
	78	do	Feb. 29	Mar. 1	21	About 36 hours	22
No. 8	90	Apr. 10	May 13	May 13	33	About 12 hours	33
	91	do	May 16	May 17	36	About 36 hours	37
No. 12	106	June 18					
	127	do	July 14	July 14	26	Found dead	26
No. 15	151	July 31	Aug. 29	Aug. 29	29	About 12 hours	30
	152	do	Aug. 27	Aug. 29	27	About 60 hours	30

a Rabbit chloroformed after becoming completely paralyzed.

Rabbits Nos. 77 and 106, inoculated with the brains from dogs Nos. 7 and 12, remained well. The two other rabbits inoculated from these dogs died of paralytic rabies. An explanation for this may be found in one of two possibilities, namely, these rabbits may not have been susceptible to the disease, or the small quantity of the virus injected beneath the dura may have been washed out through the puncture made by the needle, as there was some hemorrhage, and the disinfection of the wound would, presumably, have destroyed the virus if it had escaped from beneath the meninges. The fact that one of the rabbits inoculated from each of these dogs (Nos. 7 and 12) died of paralytic rabies is sufficient to demonstrate the presence of rabies in these animals. A brief history of the dogs examined, as given to us by the health officer, is appended.

Dog No. 1.—This is the most important case in the series, as the owner was bitten by the dog and died of rabies. The animal was somewhat ferocious, and had been kept tied in a barn for some time prior to this incident. On the morning of September 26 its owner, Mrs. S., went into the barn, and as she passed by the dog he jumped upon her, biting her on the right hand, left forearm, and upper lip. The dog was not known to have been bitten by any other dog. It was killed immediately and sent later in the day to the experiment station of this Bureau, where Dr. Schroeder removed the brain and sent it to the laboratory.

From Dr. Behrend's report[1] of the case the following is taken:

Mrs. S., age 56, a widow, in good health, was bitten on the right hand, left forearm, and upper lip by her dog on September 26, 1895. She consulted me eight hours after she had been bitten. The wounds were cauterized and dressed and nothing more was heard from the case until October 11, when she summoned me to the house. She was walking about her room in a very nervous and excited condition. She complained of not being able to sleep and in having trouble in swallowing.

[1] Journal of Practical Medicine, VI (1896), p. 224.

She said she had felt bad for two or three days and had not taken food for twenty-four hours. The pulse was full, the temperature 98.8° F. Twenty grains of chloral were ordered with whisky and an egg every three hours. Inquiries were then made concerning the inoculated rabbits, but finding them still well the very grave question arose, Is it rabies or hysteria? October 12 the patient appeared to be about the same. She had taken the medicine, but reported trouble in swallowing water. She was told that the rabbits were well, and that she could not possibly have rabies, but was suffering from a bilious and nervous attack. Calomel and bromide of potassium were prescribed. To the family I expressed my doubts of the case, for I must state that this was the first case of hydrophobia I ever saw, and, furthermore, I never saw such peculiar symptoms in hysteria. October 13, on entering the house about 10 a. m., the woman ran with an excited, maniacal cry toward me, exclaiming with clenched fists: "You must give me relief from this thirst; I am burning up." She complained also of a peculiar oppression and ejected a viscid saliva with great effort. She was induced to lie down, and a half grain of morphine was administered hypodermically. Consultation was requested and Dr. Lovejoy met me at 1 p. m.

The patient was then a little more tranquil, temperature 100.4° F. She gave very coherent answers to all of Dr. Lovejoy's questions. In his presence she gulped down a wineglassful of water at one swallow, and with a spasmodic action threw the glass from her. She was very excitable. Morphine and chloral were administered. At 5 p. m. she was sitting up in bed, very restless, pulse 105°, skin cold and clammy, no cyanosis. At 10 p. m., in an effort to vomit, she fell back and expired.

Dr. Lamb made a post-mortem examination. He removed a small piece of the medulla, with which Dr. Reed, of the Army Medical Museum, inoculated three rabbits. These died of rabies on the seventeenth and eighteenth days after inoculation.[1]

Dog No. 2.—This dog was sent to the health office November 22, 1895, by Dr. Cecil French, who gave the following history of the case in a letter addressed to the health officer. The letter, which was referred to us, is in part as follows:

The Irish setter I am sending by the dog wagon was brought to my infirmary last night almost dead. The owner, Mr. McL., of the Government Printing Office, who resides at Langdon, stated that it had eaten foreign substances, had manifested a paralyzed jaw with salivation, and had absented itself from home a day or two ago. It was unable to prehend any nourishment. The temperature last night was subnormal. Death occurred in the night. There are no other reports as to its actions. * * *

Dog No. 3.—This dog was received from the health officer without a statement concerning its history other than that it was thought to be mad and was shot. The rabbits inoculated with its brain remained well.

Dog No. 4.—This dog belonged to Mrs. S., who resides on Sixteenth street. It had been sick for three days. It bit a horse and dog and was shot by Dr. Kimpton, who believed it to be mad.

Post-mortem: Mucosa of the larynx of a pinkish color. The lungs were slightly œdematous; considerable serum escaped upon section. The liver was abnormally reddened. Spleen normal. Kidneys hyperæmic. Stomach contracted, contents semifluid, and the mucous membranes were pale. As the dog was shot in the head, little could be determined concerning the existence of brain lesions. The spinal cord appeared to be normal. But one rabbit was inoculated from the brain of this dog. It died in twelve days of paralytic rabies.

Dog No. 5.—A large Eskimo dog. No history was obtained. The post-mortem examination showed the brain to be apparently normal in appearance. Mouth, larynx, and pharynx normal. The lungs were

[1] The rabbits inoculated by us from the brain of the dog died eighteen and nineteen days, respectively, after their inoculation.

considerably pigmented. Liver dark and sprinkled with small grayish areas. Stomach contracted; contents small in quantity and largely liquid.

The rabbits inoculated with the brain from this dog lived an unusually long time, but eventually died of paralytic rabies.

Dog No. 6.—This dog was sent to us without its history. The post-mortem showed its organs to be normal in appearance. The brain was badly injured from the bullet. The rabbits inoculated with the brain substance died of septicæmia.

Dog No. 7.—A large mastiff killed at the pound February 7. The head only was received. February 8 two rabbits were inoculated. One of these (No. 78) died of rabies on the twenty-second day, but the other (No. 77) remained well.

Dog No. 8.—The appended letter addressed to the Chief of this Bureau by the health officer of the District contains all the information we have concerning this case:

> Replying to yours of the 11th instant, requesting all obtainable facts relative to the supposed rabid dog, the brain of which was sent to the experiment station on the 9th instant, I have the honor to state that this dog was said by the owner to be, so far as was noticeable, in perfect health up to the morning on which she died, the 9th instant. On that date, when taking the dog out for a walk, as was his custom, he noticed that she ran around and barked at horses and people, something which he had never known her to do before. She suddenly fell, and when he reached her she was rigid and frothing at the mouth. As he attempted to put his hand on her she bit him, drawing the blood, and died in a few moments afterwards. The dog was a brown setter and had given birth to pups about two months before.

The brain of the dog was received during the afternoon of April 9 and rabbits were inoculated with it the following morning. They died of paralytic rabies in thirty-three and thirty-seven days, respectively.

Dog No. 9.—This dog was sent to the experiment station April 23. It had been dead about twenty-four hours and decomposition was so far advanced that rabbit inoculation could not be made. The post-mortem examination was made by Dr. Schroeder. The mouth was full of earth and coal ashes. Mucous membrane of the trachea deeply reddened. Abdominal organs much decomposed.

Dog No. 10.—This dog was reported to Dr. Schroeder as acting queerly. It was biting boards and boxes, chewing earth, and trying to bite the children of the household. If another dog appeared it seemed to be excited. The owner was told to keep it confined in such a manner that it could not bite anyone. The dog died during the night and was brought to the experiment station at 6 p. m. on the following day. The brain was removed and brought to the laboratory. On the day following two rabbits were inoculated, but subsequently died of septicæmia caused by a streptococcus infection.

Dog No. 11.—This dog was received from the health office in a badly decomposed condition. It had been shot by a policeman for rabies, but otherwise no history was obtained.

Dog No. 12.—The brain of this dog was sent to the experiment station, from which we received it. It had become so much changed from decomposition that rabbits were not inoculated.

Dog No. 13.—This dog was sent to us by Dr. French, who had carefully examined it before death. The post-mortem showed the mucous membranes of the mouth and pharynx to be deeply reddened. Otherwise organs appeared to be normal. Two rabbits were inoculated with the brain. One of them died suddenly twenty-four days afterwards; the other remained well.

Dog No. 14.—This dog was thought by the owner to be mad, as it snapped at passing objects and was consequently shot. The organs appeared to be normal and the rabbits inoculated with the brain remained well.

Dog No. 15.—The history of this dog is contained in the appended letter addressed to the Chief of the Bureau by the health officer of the District:

I have to state that the supposed rabid dog (black terrier), delivered on the 31st ultimo by the poundmaster, was discovered by Officer Upperman running on R street, between First and North Capitol NW., frothing at the mouth and snapping at passers-by. The officer says its appearance and actions were those of a rabid dog, and he accordingly shot it. Ownership not known.

The two rabbits inoculated died of rabies on the thirtieth day.

It is important to note that in the case of dog No. 1 the human subject developed the disease before it appeared in the inoculated rabbits. The short period of incubation is explained in part at least by the position of the wounds, as it has long been known that where they are on the head the disease develops much more rapidly. As shown in this case, and it is probably the rule, that when the bites are on the head it is not safe for the patient to await the results of the diagnosis by rabbit inoculations before availing themselves of the Pasteur treatment. When wounds inflicted by the supposed rabid dog are on the lower extremities and usually when on the hands the positive diagnosis can ordinarily be made by Pasteur's method in time for the subsequent treatment if the dog proves to be rabid. The death rate is so high (about 80 per cent) in subjects bitten in the face or about the neck or head that it is not safe to postpone the treatment. Dr. J. J. Kinyoun tells us of a case in a boy who, in 1892, resided near Falls Church, Va. The boy was badly bitten about the head, and through Dr. Kinyoun's efforts he was sent to New York for the Pasteur treatment. The dog was not killed, but it died with paralytic rabies the day after biting the boy. He inoculated two rabbits with the brain and on the day the boy received the last treatment the rabbits died of paralytic rabies. The boy is still alive and well. The fatalities of rabies in those bitten by mad dogs and who take the Pasteur treatment is, according to late reports, less than 1 per cent.

During the month of August, 1896, two dogs supposed to be rabid were received from the health officer. One of these had died, according to Dr. French, of paralytic rabies, and the other was a suspicious case which had been killed. Two rabbits were inoculated from each. Those inoculated with the brain of one of these died of rabies on the fifteenth and eighteenth days, respectively. Those inoculated from the other dog are well at the time of this writing (September 8).

PATHOLOGY OF RABIES.

The investigations which we have made into the pathology of rabies have been for the greater part restricted to the microscopical study of the brain tissue. The dogs received were frequently in a condition unsuitable for making observations, and in other cases the work under way at the time of receipt necessitated the omission of a careful post-mortem examination. The rabbits, however, were carefully examined. In these a hyperæmic condition of the liver was the most conspicuous lesion. The intestines were sometimes sprinkled with punctiform hemorrhages. The brains were normal in appearance. The examination of the sections of the brains of a few of the dogs and several of the

rabbits failed to reveal the existence of any foci of cell infiltration indicative of bacterial or protozoan irritation. The changes which appeared have been restricted to certain apparent alterations in the structure of the nerve cells. The interpretation of the changes is exceedingly difficult, being complicated as it is with possible errors in technique and, as yet, undetermined influences of the reagents used upon the nerve texture. The intensity of the virus is not constant even when of a sufficient degree of virulence to cause the death of the victim, and, as often occurs in the case of dogs, the animal is killed perhaps before the extent of the structural changes has been attained. Such a condition as this is likely to exert more or less influence upon the distinctness of any lesion that may exist. Post-mortem changes should be scrupulously guarded against, since molecular alterations of the cell have been described by some writers as existing in rabid material, and some confusion may result from the similarity of the effects due to the two different causes.

Previous experience in the treatment of nervous tissue for sectioning has caused us to prefer the collodion to the paraffin method, believing that with such delicate structures less change from the normal may be effected. The tissues after being embedded in collodion were clarified in the castor-thyme oil mixture,[1] cut and fastened to the slide, and stained in hematoxylin, fuchsin, methylen blue and eosin, toluidin blue, and neutral red.

Weigert's method for the medullated nerve fibers and the silver-bichromate method for the cell processes were also employed. The fresh tissue was also teased apart, stained, and examined under the microscope.

The material was obtained in as fresh a state as possible. In one control experiment the brain was removed and immersed in the fixing fluid within half an hour after death, and while the brain of this one was being removed a healthy rabbit of the same size and weight was being killed, and within half an hour again the control brain of rabbit No. 2 was in the same fixing fluid as that of No. 1. Three kinds of fluids were used—corrosive sublimate, 10 per cent formalin, and 10 per cent formalin in 95 per cent alcohol. The brains were divided and similar portions of each put into the different reagents, the aim being to carry the diseased and healthy tissues along under exactly the same conditions during the hardening and embedding processes, even to cutting the two specimens with the same stroke of the knife, allowing exactly the same duration of time for the action of the staining, dehydrating, and clearing media, and finally mounting the sections upon the same slide. The diseased rabbit from which this brain was obtained did not develop paralytic symptoms until a month after its inoculation.

The desirability of an immediate and positive diagnosis of rabies in a suspected animal is unquestioned, and if this can be determined by an histological examination of the nervous system the amount of time saved in the beginning of the treatment of the party bitten would, in many cases, be of inestimable value. The inoculation test of lower animals, which is our most reliable method at present, is not always rapid enough in its action, as cases have been reported where the patient developed the symptoms as soon as the rabbits or earlier.

[1] P. A. Fish, "A new clearer for collodionized objects," Proceedings Amer. Micr. Soc., XV, pp. 1–4, 1893.

Notwithstanding the fact that certain investigators have found histological lesions, which to their minds have been pathognomonic of the disease, the close simulation of nervous lesions due to other diseases, and the possibility of greater or less post-mortem changes will foster an element of doubt in the minds of the majority of working histologists. This doubt instead of diminishing shows a tendency to grow when a review is taken of the conflicting results and opinions held by those who have already investigated this field. It also appears that some portions of the nervous system may exhibit lesions of a pronounced character, other portions very slightly, and still others none at all, thus presenting additional difficulties.

Some difference of opinion seems to exist among observers as to the importance of these lesions. Certain of them believe that no evidence has been produced in the way of tissue change that may not be duplicated by some other complication, while others believe that changes may be found which are peculiar and absolutely characteristic of rabies.

One of the most common lesions observed is of an inflammatory character, the congested blood vessels frequently showing diapedesis, and, according to some, a perivascular exudation of a granular or hyaline substance. Hyperæmia and lymph-stasis, although of not so much significance when taken by themselves, have been taken into consideration along with other changes. The blood vessels quite as much or even more than the nerve structures have been noted as the locus of some of the most marked changes, among which are the proliferation of the epithelial cells and of the connective tissue elements of the outer coat, with the infiltration by lymphoid cells. Such lesions may be nodular primarily, but later become diffuse. The inflammatory processes may progress to such an extent as to obliterate certain vessels.

Pathological miliary centers have been noted not only in the axial portions of the nervous system, but in the gray matter as well. These centers being formed by lymph cells which accumulate notably around the blood vessels (perivascular), and the nerve cells (pericellular) as well, the lesions, when present, are observed most frequently in the motor centers of the oblongata and spinal cord.

The following results observed by Babes,[1] and first published in 1887, are here given in full:

1. In animals dead from street rabies there are found usually a hyperæmia and an acute generalized œdema of the cerebral meninges, acute hemorrhages localized around certain vessels, as well as inflammatory lesions. On microscopic examination we find an increase of the plasma cells, augmentation of the reticular substance, fibrinous in character, between the several layers of the meninges.

2. The epithelium of the cerebro-spinal central canal has proliferated. In the gray matter which surrounds the canal, and especially in that of the floor, hemorrhages, sometimes symmetrical, are often found. Microscopically, we often find an obliteration or thrombosis of a vessel by a reticulated, hyaline, pigmented material, or by leucocytes or hyaline globules, and sometimes a hyaline degeneration or even inflammation of the vascular tunics. The extravasated blood also contains much of the hyaline material. The hemorrhages are often limited by the lymphatic sheath of the vessels. At the same time the epithelium of the ventricles and central canal may be partially lost. This last is occasionally filled with blood or plugs, either granular or hyaline in character.

3. With the naked eye small centers of degeneration may sometimes be noted in the gray matter, but often they may be sought for in vain.

4. The most constant lesions are microscopic in character; they are found more

[1] Sur Certains Caractères des Lésions Histologiques de la Rage, Ann. de l'Institut Pasteur, VI, 209–223.

especially in the gray matter surrounding the cerebro-spinal canal, and in the motor centers of the medulla and spinal cord. These lesions consist at first in hyperæmia and accumulations of embryonic cells around the small vessels, perithelial or migratory in origin, often showing indirect division; finally there are also found lesions of nerve cells.

5. The lesion of the nervous elements of the parts indicated is quite characteristic; it consists in signs of proliferation, namely, in the presence of several small cells in place of one large one, or in a uniform degeneration, and often in the appearance of vacuoles with a reduction in size or disappearance of the nucleus, or, again, its chromatic network disappears. These cells frequently contain pigment. Round uninuclear, more rarely multinuclear, elements of a lymphatic origin often invade the protoplasm even of the cell, and fill out the dilated pericellular lymphatic spaces by a multiplication of small nuclei.

6. The lesion of medullary substance is less pronounced, it consists chiefly in an œdema of the medullary sheath of the nerve fibers.

7. In certain plasma cells, in the interior of and around vessels sometimes in leucocytes, in lymphatic spaces, in the altered parts of certain nerve cells, and in the dilated sheath of nerve fibers may be seen round or amœboid granules about 1μ in diameter, pigmented or stainable by aniline dyes, and which in part seem to posses the power of movement.

Babes in a more recent work has noted, besides the lesions above mentioned, that the alteration of the nerve cell is usually accompanied by a modification of their protoplasmic network and concludes that "Whilst admitting that the lesions of rabies are not absolutely characteristic, and that it may be that in a case of diffuse, very acute myelitis similar lesions may be found, it is necessary all the same to state, that neither in writing nor in my personal experience have I ever met with a similar case, so that at present we may consider the lesion of rabies as characteristic. In other infectious diseases there have also been found histological lesions characteristic as a whole, although composed of elements not absolutely specific."

Golgi[1] draws attention to the following morbid changes in rabies:

(1) Changes in the structure of the nucleus, all the various phases of karyokinesis may be simulated, yet no true nuclear division may take place. (2) Changes in the body of the cells, such as vacuole formation, bladder-like transformation of the cells. Changes may also be recognized by methods directed to the study of the outer form of the cell. Here varicose appearances of the cell processes may be seen. Granular fatty changes may also be present. An important change lies in the displacement of the nucleus. The periphery of the cell becomes homogeneous. Granular fatty changes are also seen in the neuroglia cells. (3) Changes in the intervertebral ganglia. The author would look upon these anatomico-pathological changes found by him as characteristic, while here not only the sum total of the changes, but also their order of occurrence and mutual interdependence are taken into consideration.

The morbid process is a parenchymatous encephalo-myelitis, of which the exact exciting cause is as yet unknown. The changes are thus grouped: (1) Appearance of nuclear chromatin, peculiar cell division (neuroglia cells and vascular endothelium), nuclear movements also in nerve cells, diffuse vascular distension, and leucocyte infiltration, revealing a condition of irritation; (2) swelling, vacuolation, changes of form, granular appearance of nerve cells, and neuroglia; and (3) more advanced changes in the nerve elements. The changes in the first group may be seen as early as five days after inoculation.

In a more recent article by Germano and Capobianco[2] attention is called to the fact that the destruction of some of the nerve cells in rabid animals is not accepted by everybody, but that in their researches they have been able to confirm the statements made by Golgi, who is among the most recent to investigate this matter, that instances of the complete disappearance of nerve cells have been observed, while other cells showing fatty degeneration and partial destruction of their entirety

[1] Berl. klin. Wochenschrift, April, 1894.
[2] Ann. de l'Institut Pasteur, IX, 625-635, No. 8, August, 1895.

represent intermediate stages between the normal cell and its total disappearance. The alteration of the nucleus may precede or follow that of the cell body.

The nerve fibers, either in the white or gray matter, undergo a certain amount of change. In a longitudinal section of the myel, especially through the ventro-lateral columns, there were noticed marked changes in the axis cylinders. In some cases they appeared uniformly swollen their whole length, while in others there were varicose enlargements. In the swollen portions there were frequently observed small vacuoles which interrupted the continuity of the axis cylinder.

A certain amount of importance is attached to the neuroglia in acute inflammation of the central nervous system. The stimulus, which tends toward the destruction of the nerve fibers and cells, favors the production of neuroglia, and perhaps contributes, by the pressure resulting from its increased growth, to the destruction of the nerve elements.

In the present investigation the central nervous systems of rabbits were mostly used because they could be obtained in a more perfectly fresh or living condition, although a few favorable specimens of dogs' brains were also utilized. Portions of the cerebrum (hippocampal region), cerebellum, oblongata, and cervical myel were examined after the tissue had been prepared by the methods already mentioned.

Of the specimens examined, those of the dogs showed perhaps the more pronounced lesions, the less marked changes in the case of the rabbits possibly being due to a slight weakening of the virus.

The well-known fact that the virus grows stronger during its passage through a great many rabbits until it reaches what is known as the "fixed" virus, would lead us to expect that from the first to the last rabbit the virus as it increases in intensity would concomitantly intensify the lesions.

In general, the pathological conditions incident to the congestion of the blood vessels and some of the other alterations dependent upon inflammation, although to a lesser extent, were noted as described by previous investigators. Of the portions of the brain examined, that of the cerebellum showed less departure from the normal structure than any of the other parts. The motor cells of the oblongata and the myel in most instances showed a marked vacuolation of the cell body, as did also those of the different layers of the cerebral cortex. This vacuolated degeneration often appeared to have usurped the whole cytoplasm and in some places to have dissolved the continuity of the cell boundary, giving it a very irregular and distorted outline. Cells of this character and those showing a lesser degree of vacuolation were invariably chromophobic, taking the stain but slightly.

The Golgi method, which brought out with great distinctness the cell processes for their whole extent, showed numerous varicosities occurring on either the dendrites or neurites. These were particularly noticeable on certain of the cells (sensory cells?) (fig. 3, Pl. XXVIII) from the dorsal horn of the myel and appeared more pronounced than certain similar varicosities which have been observed on the processes of apparently normal cells.

The neuroglia cells seemed to be involved. Their processes in places have become very considerably swollen, and the swollen areas as well as that of the cell body also show the extensively vacuolated condition. The medullated nerve fibers of the central nervous system also showed, by the Weigert method, occasional varicosities along their course, but the fact that similar swellings are met with in nervous tissue not supposed to be rabid would tend to weaken this characteristic

as a pathognomonic sign. In some of the teased preparations somewhat more pronounced swellings were noticed, which might prove of more value for diagnostic purposes.

The conditions noted seem to indicate that the true nervous lesions were, in a measure at least, dependent upon circulatory changes, and that such changes causing an interference with the proper nutrition of the parts effected the degeneration of the nerve elements, while, on the other hand, there may be some ground for the belief that the degeneration of the nerve cells due to some irritant is primary and the vascular changes are secondary.

At this time the result of our investigations will not permit us to say that we have found certain peculiar and constant changes in the histology of the rabid nervous system which are absolutely characteristic of that disease and none other; but we do believe that a histological examination is useful in a negative way, since the absence of the various lesions already enumerated would strongly indicate the absence of the disease.

ETIOLOGY.

Although rabies has become recognized as a specific disease, in which the virus selects the nervous system as its most favorable location, its specific agent or organism has not been demonstrated, although several investigators have described minute granules in the brain substance which at first were thought to be the etiological factor, and still others have found various forms of bacteria which were believed to have a causal relation to this disease.

In the fall of 1892 one of us made a very large number of cultures on alkaline agar, gelatin, and in bouillon from the brains of cattle which died of rabies, and in the spring of 1893 they were repeated in rabbits with the brains of produced cases of canine rabies. The results of these bacteriological investigations were negative, and the microscopical examination of the brain tissue, both in a fresh condition and in sections stained for bacteria, failed to reveal the presence of any bacterial or protozoan bodies. It was thought, however, that perhaps the failure to obtain results in these investigations was due to the fact that the media used were not suited to the requirements of this disease, and other media and methods of cultivation were planned, but as yet we have tested but one of these, namely, the use of media in which normal brain substance was used in place of meat or beef extract in preparing the bouillon, agar, and gelatin. The results were negative.

Two very interesting papers[1] on the etiology of rabies have just appeared, in which each author has found a different kind of organism as the supposed cause and has reproduced by inoculation a disease which, in the mind of each of the investigators, was thought to be rabies. Bruschettini considers the specific organism to be a bacillus which he has isolated and with which the disease has been produced experimentally by him. Memmo has found a fungus (Blastomycetes) to be the etiological factor. He has located these organisms in the brain, cerebro-spinal fluid, aqueous humor, stroma of the parotid glands and the saliva. He finds the growth more favorable on acid media and claims to have reproduced the disease from subcutaneous intra-abdominal and subdural inoculations into guinea pigs, rabbits, and dogs.

[1] Centralblatt f. Bakteriol. u. Parasitenk. u. Infekt. Erste Abtheilung, XX, 209-213, 214-217.

DESCRIPTION OF PLATES.

PLATE XXVI.

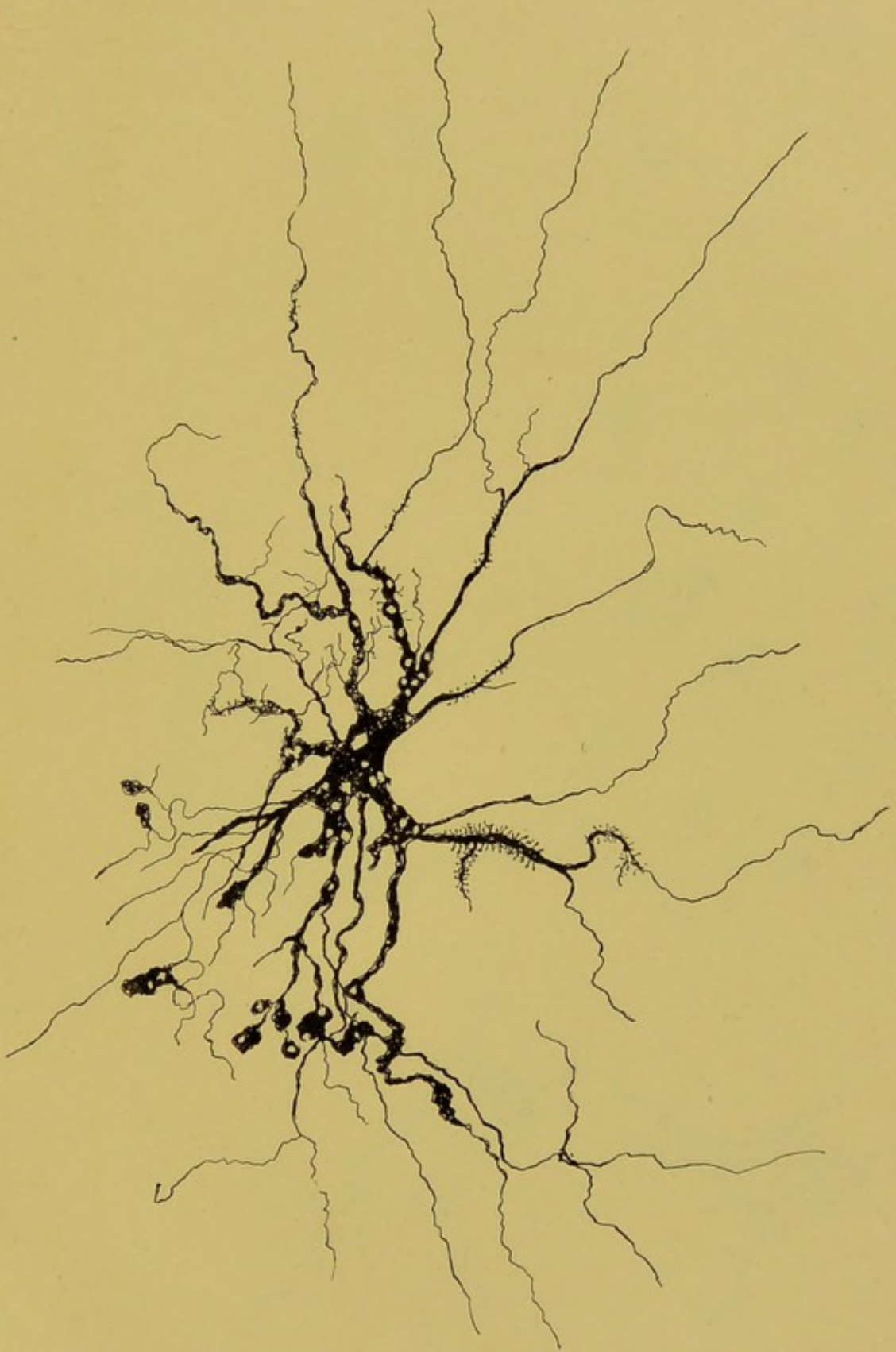
Neuroglia cell from the cervical region of the myel of rabbit No. 30. Numerous vacuoles are shown in the cell body and in some of the irregularly swollen processes. Drawn with a camera lucida. Zeiss. No. 4 compensating ocular and 2 mm. apochromatic objective. (Golgi method.)

PLATE XXVII.

Nerve cells *a a a* (figs. 1, 2, and 4) from the cortex of the hippocampal region. Brain of rabbit No. 2. Showing the presence of vacuoles and the gradual disintegration of the cell body resulting from their increase. *b b* (figs. 3 and 4), motor cells from the oblongata of rabbit No. 2. The cell processes are distorted. One of the lighter cells shows the presence of vacuoles *v* (fig. 5). Drawn with the camera lucida. Zeiss. No. 4 compensating ocular and 2 mm. apochromatic objective.

PLATE XXVIII.

Figs. 1, 2, and 3 are cells from the dorsal horn of the myel of rabbit No. 28. The cell processes show numerous small enlargements. Golgi method. Camera lucida. Zeiss. No. 4 compensating ocular and 2 mm. apochromatic objective.



Neuroglia Cell.

Fig. 1.

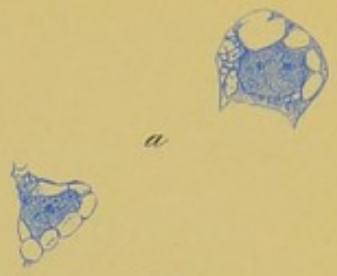


Fig. 2.

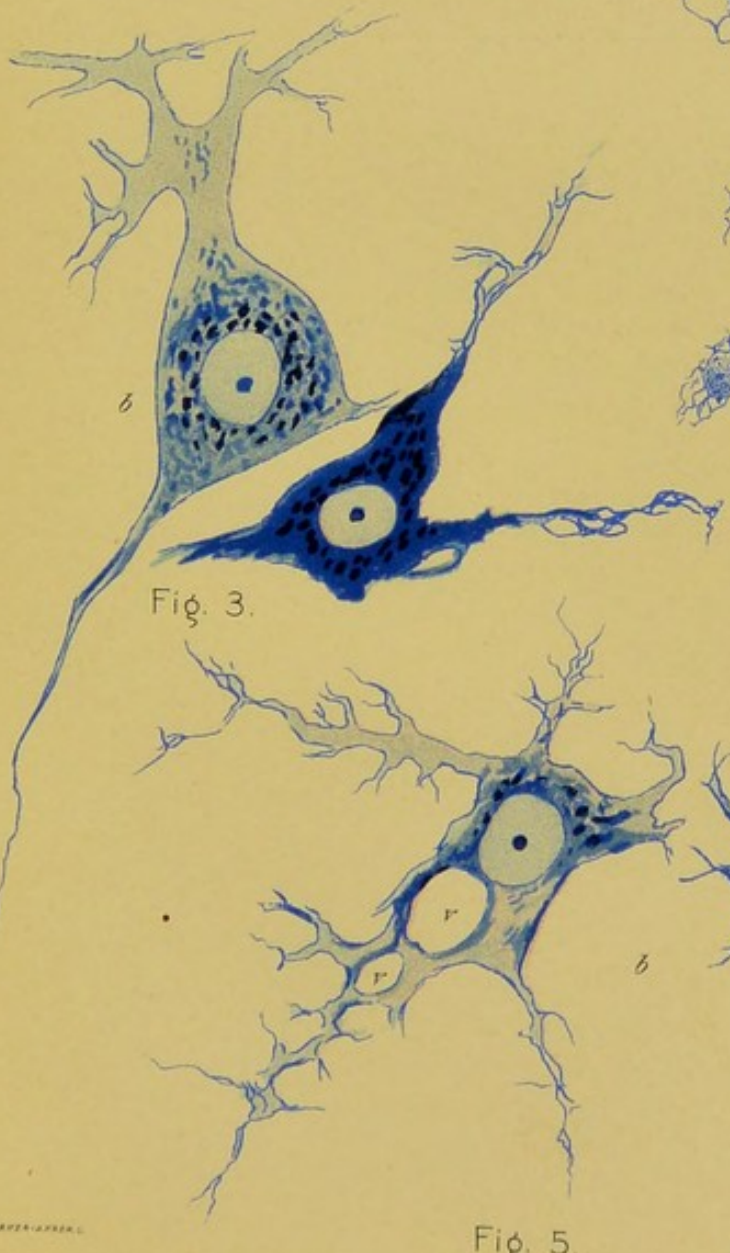


Fig. 3.

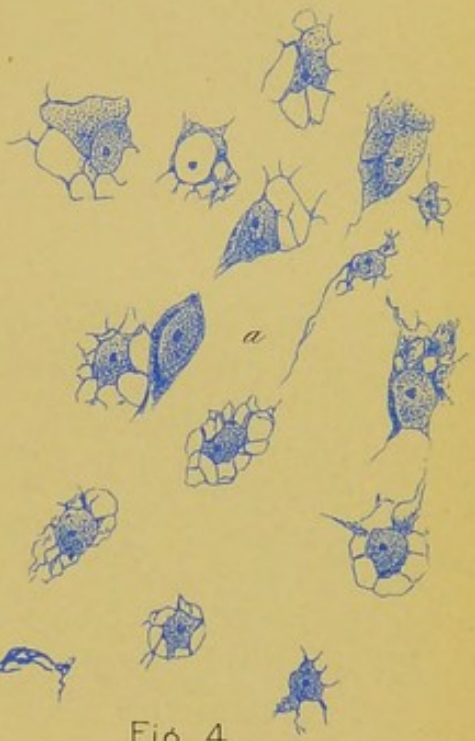


Fig. 4.

Fig. 5.

Haines, del.

NERVE CELLS FROM BRAIN AND MYEL.

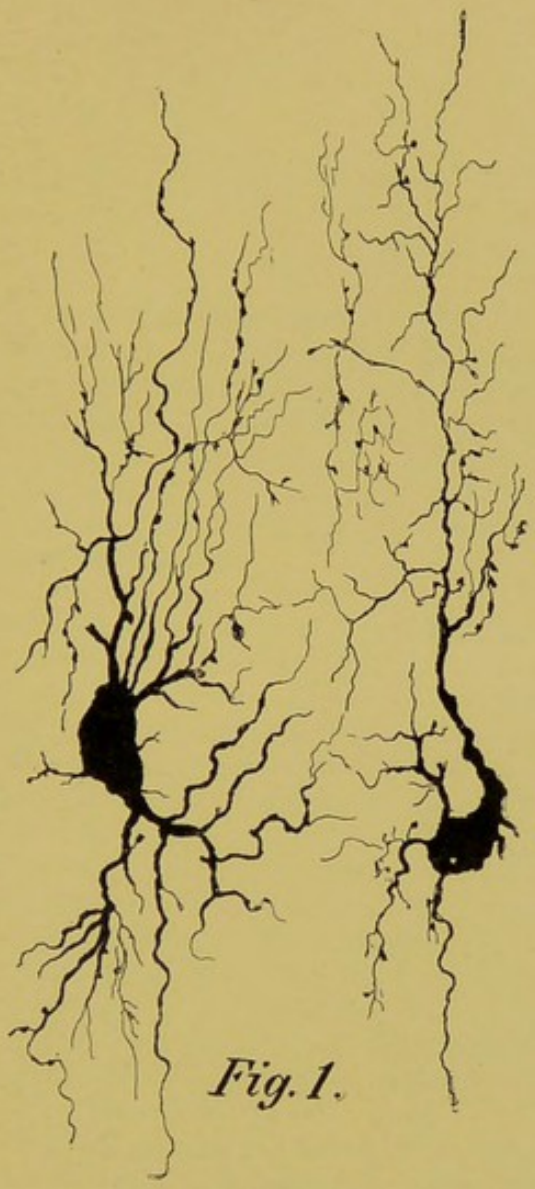


Fig. 1.

Fig. 2.

Fig. 3.

NERVE CELLS FROM MYEL.

THE DETECTION OF TUBERCULOSIS IN CATTLE.

By Cooper Curtice, D. V. S., M. D.

The tuberculin test for tuberculosis has proven itself of such a benefit to stock owners and is so easy of application when its details are understood that a detailed presentation of the subject seems advisable. With this information each stock owner may either test his own cattle or employ a veterinarian, as most men favorably situated will do, and in the latter case be in a position to know whether approved methods are pursued and correct deductions drawn from the results obtained.

Such is the scarcity of competent, reliable veterinarians in many sections of this country that it is impossible for stock owners to employ one. Often the testing of but a few cows is desired, and the expense of employing one from a distance is so great as to prove prohibitive.

Stock owners will when they are favorably situated find it to their interest to employ specialists, owing to the necessities of trade and the prevention of possible legal controversies. Competent veterinarians are disinterested experts upon whose certificates the parties in trade may rely and their evidence will have greater weight with the court. It is almost needless to add that strangers will accept from each other only certificates from honest, capable, qualified men, whose characters are in a measure guaranteed either by the community in which they reside or by the State or Federal Government.

This description is especially written for those owners who wish either from choice or compulsory circumstances to test their own cattle.

An examination of a herd of cows for tuberculosis embraces—

(1) Ascertaining the history and present physical condition of the individuals composing it, especially their temperatures or body heat;

(2) The injection of a specific agent called tuberculin beneath the skin;

(3) The determination of the variations of temperature during a limited period after injection; and

(4) Deductions from these variations, determining the presence or absence of tuberculosis.

DESCRIPTION OF INSTRUMENTS AND CHEMICALS, WITH METHODS OF RECORDING RESULTS.

Thermometers.—The temperature of the animals to be tested should be taken with an ordinary 4-inch physician's clinical self-registering thermometer (Pl. XXIX, fig. 2). This should have a large, smooth bulb, a wide column of mercury, or a magnifying lens front, and a broad, flat, plainly graduated stem. Sharp-pointed bulbs catch in the mucous membrane of the rectum when inserted. Indistinct markings and a narrow column of mercury are difficult to see in dark stables or by lantern light. There is a so-called veterinarian's thermometer,

which is larger than the physician's, but the size is frequently a disadvantage. The especially curved thermometers are more expensive and offer no special advantages.

Inspectors who are constantly using thermometers should secure from a half dozen to a dozen to save cost in purchase and to prevent a sudden and annoying termination of the test through often unavoidable breakage of the thermometer. When but 10 or 12 cattle are to be tested, one or two thermometers are sufficient, but when 30 or 40 are to be tested, the simultaneous use of three or four will save considerable time.[1]

A self-registering thermometer is one in which a constriction of the caliber of the tube at the neck prevents the return of the mercury into the bulb when the glass cools, unless it is intentionally shaken back. Although the return of the mercury index in the stem is easily effected, a description of the method will doubtless prove an aid to many readers:

Seize the stem of the thermometer between the thumb and forefingers with its length at right angles to the forearm. Hold the hand on its back and quickly turn it downward, holding the forearm stiff, ending with a snap and a slight throw of the hand. The mercury disappears from the stem by the influence of the centrifugal motion and the sudden arrest of the movement.

In a second method the stem is grasped with the bulb downward like a pen. A swift downward throw of the forearm with the wrist immovable and sudden stop returns the mercury. The stop should be sharp and snappy. It may be aided by striking the hand upon the opposite fist or knee.

Syringe.—The hypodermic syringe (Pl. XXIX, fig. 1) suitable for this use is like the ordinary physician's hypodermic, but larger and stronger, and adapted and graduated for the more severe use intended; indeed, the physician's aspirator makes an admirable though rather large substitute.

A good syringe should have the graduation plainly marked either on the glass cylinder or on the piston rod, and preferably in the metric system. The English system is about the only one in use in this country and must be used until instrument makers place upon the market syringes graduated in the Continental or European system. The capacity of the syringe should be about 5 c. c., or 2 drams, if intended for two or three doses, or about 3 c. c., or 1 dram, if intended for one dose.

One style of syringe, costing about $3, has a jam nut running on a graduated piston rod, which may be used to divide the fluid taken into the syringe into the appropriate doses by setting and resetting the nut. This device saves recharging. Some prefer, however, to charge with but one dose at a time. The cylinder should have either straight arms at the top or large thumb rings as in the figure.

The packings of a syringe are usually leather. It is said that asbestus is now being used. Should this material prove an efficient packing it will be of great value, as sterilizing liquids which soon destroy the elasticity of leather would have no effect on it. The packings should always work snugly and smoothly, and washers should be frequently replaced. All should be kept well soaked with carbolized oil.

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[1] Thermometers cost from $9 per dozen upward. A good serviceable thermometer which is certified to be accurate within the limits specified can be bought for about $1. They may be purchased with other glassware at any city drug store.

The needle should be about 2 inches in length, strong, and made of steel; iron will bend and its soft point dulls too quickly. The caliber should be sufficiently large to permit the easy flow of a glycerin preparation of tuberculin in cold weather. Extra points should be provided with each syringe, as they dull easily. The points should be used only when sharp, otherwise great resistance by the cattle may be expected.

To fully charge the syringe some of the fluid is drawn in, the syringe inverted, the piston still further withdrawn to clear the needle, and the piston then pushed in to expel the air until the fluid appears at the end of the needle. The remainder of the dose may then be drawn into the syringe without air.

Tuberculin.—The material with which cattle are injected in the test for tuberculosis is called Tuberculinum Kochii. It is made by different firms and placed upon the market under their names, and each brand seems to vary somewhat in appearance and effects, but not so materially as to seriously change the results. One great difference is in the character of diluent used. When it is put up in a dose ready for use the original material is diluted with glycerin or carbolized water. When preliminary preparation is required 0.5 per cent carbolized sterilized water should be added to it, usually in the proportion of 1 part tuberculin, 9 parts 0.5 per cent carbolized solution. The amount of necessary dilution is one of the great factors in making a difference in price.

The tuberculin manufactured by this Bureau is diluted with glycerin solution so that 2 c. c. may be given for each dose. This tuberculin is used in State work by boards of health and cattle commissioners, but is not distributed to individual veterinarians or cattle owners and can not be bought on the market.

Since the public will appreciate the necessity for purity and will desire a reliable, guaranteed article, it would seem politic for the different experimental stations to undertake the manufacture of tuberculin and distribute it to farmers at a slight advance upon the cost price; or, if they so decide, distribute it free of cost when the inspection is to be done under the supervision of an approved inspector.

There are at present but few firms dispensing tuberculin in this country whose advertisements are available: Victor Koechl & Co., 79 Murray street, New York, agents for Libbertz's T. Kochii and Lucius & Bruning's tuberculin; Pasteur Anthrax Vaccine Company (Limited), No. 56 Fifth avenue, Chicago, agents for tuberculin from Pasteur laboratory, Paris; New York Biological and Vaccinal Institute in Pasteur Institute Building, No. 1 West Ninety-seventh street, New York; and the Oakland Chemical Company, No. 465 West Broadway, New York, who probably manufacture their own material.

The price of tuberculin per dose varies greatly. Owing in part to the tariff and in part to the percentage of profit to dealers, the European brand costs more here than there. The Libbertz variety costs here $4 for each 5 c. c., or 20 cents for each 0.25 c. c. dose. However, Kral I, Kleiner Ring 11, Prague, Austria, advertised (1895) 10 c. c. of tuberculin at 10 shillings, which is about $2.50. The German price shows the marked cheapness at which tuberculin can there be made and sold.

When the dose is put up for immediate use there is probably small chance for reduction in the prices. There is an advantage to be gained in buying the undiluted tuberculin. In practice it has been found that two drops, or 0.12 c. c. doses, of Libbertz tuberculin diluted

in the one-half of 1 per cent carbolized solution to a 10 per cent solution gives good results on first injections. Some inspectors claim good results from even smaller quantities, but the complete efficiency of the latter has not been demonstrated, and it seems best to adopt diminished doses very cautiously, thus preventing a decrease in the reliability of the diagnosis.

In this connection too much stress can not be laid upon the unknown strength of various manufactures of tuberculin, for their use alone will enable a comparison to be made with known standards. Since the formulæ for making and the assumed means of testing the strength are well known to makers, it is not likely that various brands materially differ.

The undiluted material is, therefore, much the cheaper. Work is also much more satisfactory when the character and quantity of the diluent, as well as the amount of the standard article, are known.

In using the Libbertz material as a standard, the writer does so with no disparagement of other brands. It is the oldest on the market and has been most used by himself and others, thus offering the only available unit of comparison.

Bottles, graduates, and pipettes.—Where only a few cattle are to be tested no extra bottles are required. The syringe may be used to measure the liquids, the tuberculin be dropped from the stopper, and teacups be used for containers.

When an inspector uses an outfit daily a wide-mouthed, glass-capped (Pl. XXIX, fig. 7) or stoppered 2-ounce bottle is a great convenience. In the absence of this a wide-mouthed bottle with cotton-batting plugs or cork stoppers will suffice.

An ordinary medicine dropper (Pl. XXIX, fig. 6) may be used for counting drops. The greatest care should be taken in keeping the dropper sterile, otherwise each time it is put in the main bottle of tuberculin it may carry contaminating germs which will destroy the usefulness of the tuberculin. With sufficient expertness in dropping medicine from a bottle such should be done, as there is then very little probability of contaminating its contents.

A metric fluid measure or graduate (Pl. XXIX, fig. 5) is necessary in daily practice. One containing 10 c. c. will answer for all ordinary purposes. As these are not always accessible the common 2-ounce druggist's graduates, subdivided into one-half drams, will answer. Volumes indicated in the metric system can be converted into the English system by recollecting that there are about 32 c. c. in 1 ounce. Little else is needed save tin dishes or porcelain bowls for the carbolic solution, and perhaps bottles for holding the carbolic disinfectants prepared for immediate use.

Lanterns.—Some part of the work, especially in the short winter days, must be done before daylight or after dark. Often stables are dark. While lanterns are to be found at every farmhouse, it is well to assure plenty of light for seeing the obscure thread of mercury in the thermometers. Professional inspectors should provide at least one suitable lantern. The variety with the reflector and dashboard attachment, serviceable for hanging on boards, etc., is best. Great care should be taken in holding these around the cows. When injecting it is best to have the lantern separated from the cow under treatment by at least the cow next her.

Marking implements.—Farmers are so familiar with their cattle that for them no method of external marking is necessary. When

strangers inspect herds they must either study the cattle so that they may be remembered or attach some distinguishing tag. The best device seems to be either the flat metal band in the ear or a metal tag so attached by a ring to the upper border of the ear that it hangs in the shell with the lettering in full view. This is necessary because of the importance of permanently fixing the individual records to the proper cows and thus preventing serious mistakes. The hog-ringers and round rings found on the market are convenient affairs with which to attach the tag.

Disinfection.—The materials and apparatus necessary for disinfection in this operation are very simple. The necessary materials are heat, water, pure carbolic acid, absolute alcohol, and possibly corrosive sublimate. The water and heat are at every farmhouse, carbolic acid and corrosive sublimate can be bought at any drug store. The vessels used should be carefully cleaned and then disinfected by boiling in water five or ten minutes.

The syringe may be disinfected by drawing into it at top and bottom absolute alcohol or a 5 per cent solution of carbolic acid, after first rinsing with water and putting it into some of the disinfecting fluid and leaving it about an hour. It should then be rinsed with boiled water and is ready for use. If previously sterilized, simple rinsing of the syringe in boiled water before using is sufficient. The needles should be emptied of water and dried, and the fine wire that accompanies each syringe be inserted in each needle before putting away.

When disinfection is practiced during injection, an assistant should carry a small bottle of the 5 per cent carbolic solution in which to wash the point after each insertion.

To make 5 per cent carbolic acid solution use 1 c. c. of acid crystals to 19 c. c. of boiling water or 1 dram of acid to 2½ ounces of boiling water. Hot water dissolves the acid more quickly than cold.

When necessary to dilute tuberculin use a one-half of 1 per cent carbolic solution in boiled water. This may be made by taking 1 part of the newly prepared 5 per cent solution and adding 4 parts of boiled water, then adding an equal volume of the latter, or it will suffice to put 10 drops of acid to each 2 ounces of boiled water. Unused tuberculin, or that which is suspected of being contaminated, may be sterilized by inserting its container and stopper in warm water, and permitting the water to come to a boiling temperature for five minutes. The bottle should then be stoppered and sealed with paraffin or wax. Avoid suddenly plunging the container into hot water.

For disinfecting the skin at the point of injection either use absolute alcohol or 1 part corrosive sublimate to 1,000 parts of water. The latter should be handled in glass bottles or earthenware dishes. Either have the desired quantity of corrosive sublimate weighed out in convenient packages by the druggist or buy the prepared sublimate tablets. One of the tablets is usually used to a pint of water. It is always best to wash the hands in this solution before injecting. Dissolve the tablets or powders in hot water, and remember that they are poisonous and should not be left around within reach of children.

If care is taken in disinfection or sterilization of the syringe, needle, and fluid for injection, little or no trouble may be expected from an omission to disinfect the skin at the point of puncture. Many operators, however, advise such disinfection, and when employed the hairs should be closely clipped over a space of about 4 square inches. This spot should be thoroughly wet with the disinfectant a half hour before

and also immediately preceding the injection. The hands should also be carefully disinfected each time after touching other than sterilized objects.

Treatment of cattle.—Handle the cattle quietly and allow them to cool off if driven far on a hot day, or when suffering from the noonday heat. Before beginning the preliminary temperature test the cattle should be put in their accustomed places in the stables, and if afterwards released must be returned to the same place. This will avoid confusion in recording the notes of tests.

Feeding.—Feeding may occasion a slight rise in body heat, but this is so slight that the cattle may be fed as usual.

Watering.—Watering may produce a decrease in body heat of from 1° to 3° F. No large volumes of cold water should be permitted from the sixth hour after the injection until the inspection is completed. When the cattle have been accustomed to water standing constantly before them, it should not be withheld. When they are watered before taking the preliminary temperature, it must be expected that they may be found from 1° to 3° F. below normal, and a subsequent rise of this amount after the injection within normal limits can not be regarded as being caused by the tuberculin.

Pasture.—In summer time, when the cattle are accustomed to go to pasture after milking, there seems to be no good reason why they can not be released from the time of injection until about a half hour before the time selected for taking the first temperature, say about eight hours after injection.

Milking.—The milking should proceed as usual. In healthy herds the quantity will be but slightly diminished, or only so much as may be accounted for by the irritation and the loss of the succulent pasturage and the accustomed water. Some owners remedy this by cutting green fodder, such as corn, rye, or clover, and by watering occasionally with lukewarm water. When fevers are excited in tuberculous cattle, the loss in milk production is considerable.

Stables.—In fly time it will be advisable to darken the stables on account of the incessant tail switching. The stable should always be well ventilated, and in summer time be as cool as possible. The manure should be removed before each taking of temperature.

Provision for recording tests.—A careful and systematic record should be made and preserved in a notebook of ample size, and this record should include the number or name, history, age, breed, weight, condition, times of testing, and temperature of each cow, as well as the amount of tuberculin injected.

Time of testing.—Any portion of the year is suitable, excepting the extremely hot weather, for making the test. The nighttime is theoretically the best time of the day for observing the temperature of the animal, because it is then most constant, but daylight is practically indispensable for most of the test.

Many inspectors advise that the temperature should be taken each hour or two throughout the day of injection. This method is very satisfactory, but it unnecessarily confines and worries the cattle. One careful temperature record at 4 p. m. and another just before injection seems to serve every practical purpose.

The time of the injection, preferably in the evening, is at the will of the operator and depends largely upon the character of the tuberculin used. Seven or 8 p. m. is the best for that manufactured by this Bureau; 10 p. m. seems a good time to use the Libbertz variety of Koch's tuberculin when the dose is 0.25 cc. Temperature taking after

injection may begin at 6 or 7 o'clock of the following morning, or in eleven and eight hours, respectively, after the use of each variety. The temperature should be taken every two hours, or, better, hourly, until twenty to twenty-four hours have elapsed from the time of injection.

All cattle except those manifestly too ill should be tested. Even cattle in the last stages of pregnancy do not seem to react from the injection unless they are tuberculous. When the preliminary temperature is above 104°, the animal may be temporarily passed, or, if it is suspected that the high temperature is due to a tubercular condition, it may also be injected.

Order of procedure.—It will be found convenient and conducive to success to observe the following order in making tuberculin tests:

1. Confine and mark the cattle.
2. Take the preliminary temperature each two hours on the day of injection.[1]
3. Inject the tuberculin.
4. Take temperatures from 6 a. m. to 6 p. m. of the following day, or until there is no rise of temperature and the normal level has been resumed.

Method of testing.—The temperature of cattle, as well as all other of the lower animals, is taken within the rectum. A few minutes before beginning all the cattle should be made to stand so that they will have time to unload the rectum before the thermometer is inserted. Often the animal appears about to strain, and time may be gained by raising the tail and irritating the mucous membrane with the thermometer. This stimulates peristalsis and hastens the motion. Before inserting the thermometer make sure that the mercury stands below 97° F. Always look at it just before inserting. By following a fixed rule the work becomes more automatic and greater accuracy is obtained. Success depends in large part upon the mechanical proficiency attained.

With the left hand firmly but gently grasp the cow's tail about 9 inches from its root; lift it slowly just high enough to permit access to the anus. Move quietly and with deliberation, as much depends on quiet treatment.

The bulb and lower part of the thermometer may be moistened with saliva, lard, glycerin, oil, or vaseline to prevent its sticking to the mucous membrane. When inserting the thermometer, avoid catching it in a fold of mucous membrane by changing its direction either to one side or upward or downward, as the occasion requires. Push it into the rectum until about an inch projects; this will permit easy withdrawal and yet escape the swinging motion of the cow's tail when the latter is released. Should the thermometer slip inside, recover it by inserting either the first two fingers or the entire hand. Often slowly raising the tail and waiting until the sphincter muscles relax brings the glass into sight.

The thermometer should remain in position three minutes, although two minutes often suffice for full registration if the anus is well closed. When three or more thermometers are used, insert the second and third in the respective cows before returning to the first; then read, record, and insert in rotation. The thermometers and hands should be cleansed after each withdrawal.

In taking temperatures some cattle may kick, jump from side to side, buck, or draw in their tails. They are usually more manageable in stanchions and when tolerably close together, and when they have

[1] The experienced inspector may confine himself to an afternoon and evening preliminary temperature.

been accustomed to gentle treatment. The operator should stand directly back of the cow, lean slightly forward, and at a sufficient distance to escape harm from kicking, but not far enough away to work at arm's length. Kicking cows may be tied, but this is usually unnecessary. The assistant should be one of the milkers. His duty with the more restless cows should be to stand at the right of the cow, placing the left hand on the right hip, and with the right smooth the udder and strip the teat as if milking while the operator inserts the thermometer. This act usually quiets the cow. Occasionally one must be summarily dealt with each time approached. The assistant should hold her head firmly, by inserting his thumb and fingers in the nostrils.

To prevent breakage of thermometers when testing calves greater care must be exercised than with cows. Usually they should be tested singly, holding the calf's tail close upon the thermometer after it is in position.

Injecting.—When the syringe is filled, the proper dose set, and the last bubble of air expelled, the operator should take his place by the right shoulder of the cow, or on the side opposite to that in which he intends to inject. Reaching over the cow with the left hand, he must pinch the skin firmly at the chosen point with the left thumb and forefinger. With the syringe resting in his right palm, the needle between the thumb and forefinger, he pierces the skin with a quick thrust, retaining hold of the folded skin with the left hand. The piston is taken in the right hand and the contents of the syringe slowly introduced.

To avoid mishap, it is better to slightly lean against the cow and rest the elbows upon her body. If she is confined in a stanchion, no assistance will be necessary, but if tied it is often necessary for an assistant to hold her by the nose. Few cattle resist when sharp-pointed needles are used. Many operators prefer to stand on the side upon which they inject, following the same course as when standing upon the opposite side of the animal. They run the risk of being hit by the head or kicked, and of having the cow draw away, all of which is avoided by taking a position on the side opposite to that in which the injection is made.

There seems to be little difference what place is chosen for injection. Operators choose the point over the shoulder blade where the skin is thick, or in the neck before the shoulder, or behind the shoulder, over the heart, or in the flank, and have equally good results. The writer prefers the location over the shoulder blade, as it is a very safe place, there is little subcutaneous fat, and the pressure exerted by the tense skin seems to facilitate absorption of the injected substance. Bulls rarely kick, but when any trouble is to be expected from getting too near their heads a flank should be selected for the injection.

Interpretation of results.—The detection of tuberculosis in cattle by tuberculin depends upon the characteristic elevation of temperature produced in a tuberculous cow within a limited period after injection. In determining that an abnormal rise in temperature has taken place due allowance must be made for variations within the normal range of temperature and for those produced by causes other than the injection of tuberculin. The elevation produced by the latter usually ranges from below 102.5° F. to over 104° F., beginning from eight to twelve hours after injection and lasting from ten to twenty hours or more. The elevation is so decided that there is little difficulty in knowing that an unusual reaction has been produced.

The method of making charts of temperatures shown in Pl. XXX is very satisfactory and greatly assists in deciding which animals are tuberculous. The degrees are represented as spaced off on the vertical lines. The time of taking the temperature is represented by setting these vertical lines at equal distances apart. The lines are numbered at the top to show the time since injection, and at the bottom in Roman characters to show the time of day. In making charts points are selected on the vertical lines which correspond to the temperature at the time of injection, and connecting lines are drawn. These lines make what is called "the temperature curve," and this curve is most pronounced when the times of observation are closest together. Two tables are shown, so that the degrees between 100° and 107° F. are repeated twice. In the upper table is shown the temperature of five cows and in the lower part that of ten cows (two entire herds).

The great difference in height in the upper plate between the line numbered 77 and those in the group at the bottom is apparent. No. 77 was slaughtered and was tuberculous. The lower plate shows that the temperature of four (Nos. 96, 87, 91, and 68) rose above 104° F. They were killed and found tuberculous. The temperature of the remainder ran closely together, and the cows were probably all healthy. They were not killed.

Not all herds give so well-defined reaction as these, because in many herds some minor troubles may exist which will cause an elevation of temperature a little above the normal, and because the normal range is wider where there are more cattle. Cows may in the last stages of pregnancy reach 104° F. Some healthy cows may reach as high as 103.3° F. After feeding they may range as high as 102.5° F. If when the temperatures are platted they show a decided elevation for some hours above 104° F. and the curve resembles others which have risen, they may be safely regarded as tuberculous. If the curve departs in a marked degree from the general shape of other curves, it may be regarded with suspicion.

There are occasionally cows suffering from tuberculosis which give but a low curve of reaction, or but a slight continued elevation. Physical diagnosis will detect some of these, especially if tuberculosis is present in other cattle of the same herd.

Pl. XXXI, Plat IV shows the temperature of a few cattle which seem to have had a rise of temperature, but which were not found to be tuberculous when slaughtered. The curves delineated depart in a marked degree from those of Plats I and II.[1]

In Pl. XXXI, Plat III are shown temperatures of tuberculous cows which departed from the usual rule.[2]

The following tables are taken from actual dairy inspection work, and are those which are delineated in Plats I and II, Pl. XXIX.

[1] Curve 23 was from a cow whose temperature on the preceding day was 104° F. at 4 and 10 p. m., evidently due to inflammation of the udder. Curves 340 and 321 were from cows heavy with calf, and which were included in the Soldiers' Home herd (Bulletin No. 7, Bovine Tuberculosis). Curves 32 and 108 were from cows that were not slaughtered. Curve 175 is suspicious and was from a herd in which 13 out of 16 head proved tuberculous. Curve 23, recorded by Dr. Page, was killed on suspicion, but was declared free from tubercle. These curves are the majority of such exceptions found in over 200 cases, and differ widely from the typical curves.

[2] Curves 17 and 238 were from bad cases; 145, 187, and 155 from mild cases; 157 from a medium case. These all differ from the normal curves of their respective herds, and all occurred in tuberculous herds. Nos. 187 and 145 developed the elevation of temperature later than others.

These dairies contained no extremely bad cases, as proved by post-mortem examinations. One, however, contained cows which disseminated the germs from their lungs and were a standing menace to others. They were of the character which every farmer should shun, either for purchase or for retention in his herd.

Herd No. 5, injected with 2 c. c. Bureau tuberculin at 10 p. m. October 22.

No of cow.	Age (years).	Breed.	Time in herd.	Pregnant.	Temperatures taken October 22 and 23, 1894.							
					Time before.		Hours after injection.					
					4 p. m.	9 p. m.	8	10	12	14	16	18
74	10	Native.	1 month...	.....	100.3	100	100.7	101	100.8	101.2	101	101.8
75	11	....do...	5 months..	.....	101	100.8	100.8	101	100.8	101.4	101.2	101.7
76	13	....do...	1 month...	.....	101	100.2	100	100.6	100.6	101	101.2	101.8
77	8	....do...	1 year.....	.....	101.7	101.2	101	**102.4**	**105.9**	**106.9**	**106**	**105**
78	10	....do...	1 month...	Calf by side.	101.2	100.8	101	101.1	101.2	101.6	101.4	101.6

Herd No. 6, injected with 2 c. c. Bureau tuberculin at 10 p. m. October 23.

No. of cow.	Age (years).	Breed.	Time in herd.	Pregnant.	Temperatures taken October 23 and 24, 1894.							
					Time before.		Hours after injection.					
					4 p. m.	10 p. m.	8	10	12	14	16	18
a 87	9	Native.	6 months.	.....	101.8	101.4	101.6	**102.9**	**103.3**	**105.6**	**106.6**	**105.3**
88	9	....do...	5 years...	.....	101.4	99.6	**102.4**	**104.6**	**105.6**	**106.8**	**106**	**107**
89	10	....do...	2 years...	.....	101.4	101.4	101	101	100.6	101.5	101.5	101.2
b 90	10	....do...	6 years...	9 months.	102.8	105	102.2	101.9	101	101.7	101.8	101.6
91	9	....do...	9 years...	.....	101	101.4	102.2	**104.5**	**105.5**	**106.6**	**106.6**	**106.5**
92	10	....do...	10 years..	.....	101.4	101.2	102.2	**102.8**	**103.4**	**104.4**	**104.5**	**104.5**
93	9	....do...	3 months.	.....	101.6	101.4	101.6	101.5	101.3	101.2	101.6	101.2
94	8	....do...	2 years...	.....	102.3	101.8	101.8	101.4	101.5	101.7	102	101.6
95	6	....do...	10 months	.....	101.6	101	101.5	101.2	101.4	101.2	101.4	101.6
96	7	....do...	7 years...	.....	101.4	101.2	101.6	101.8	101.6	101.6	101.9	101.2

a Left hind quarter of udder small and hard, with but little milk.
b This cow was due to calve; no milk.

The following post-mortem notes taken by Dr. Theobald Smith are inserted to show the character of lesion accompanying the observed elevations of temperature:

No. 77 of herd 5. Weight, 850 pounds. Age, 8 years. Killed October 29, 1894. Animal fat.

Left bronchial gland about twice normal size, and contained a softened, caseous focus 1 by three-fourths inch in diameter. Adjoining this a one-half inch focus not yet softened. In the periphery about 12¼-inch, necrotic, partly softened foci. Remaining gland tissue not hyperplastic.

Several mesenteric glands contained each a worm tubercle consisting of a mortar-like, brittle, rather dry mass embedded in the gland tissue.

No. 87 of herd 6. Weight, 800 pounds. Age, 9 years. Killed November 2, 1894.

Left half of udder firmer and smaller than right. On section no milk flows; tissue denser. From small and large ducts masses of a grayish pasty consistency were forced by slight pressure.

The first dorsal mediastinal gland 8 by 3 by 1½ inches was irregular in size. On section there was uniform pinkish gray tuberculous infiltration containing minute necrotic points and lines forming an incomplete network. The smaller glands of the chain each contained a small focus. In gland at level of bifurcation one minute recent focus. In gland at level right supernumerary bronchus a one-fourth inch slightly necrosed focus.

In about the center of caudal lobe of right lung there was a focus 2 by 3 inches,

consisting of a central cavity and a dense infiltrated zone. The cavity was 1 to 1½ inches in diameter and contained yellowish flakes suspended in some fluid. The surrounding recent infiltration was from one-half to 1 inch thick. No discharge into bronchi evident.

No. 88 of herd 6. Weight, 750 pounds. Age, 9 years. Killed October 29, 1894.

The first dorsal mediastinal gland contained a focus 1 by 1 by 3 inches, consisting of crowded one-eighth to one-fourth inch soft, cheesy foci. The next gland, about 1 inch in diameter, was entirely permeated by such foci. One of the cephalic glands of the series contained a one-half inch focus with recent necrosis. Left bronchial gland about normal in size, contained a one-half inch necrosed but firm focus.

Adhesion of ventral lobe of liver to diaphragm. In the caudal margin of this lobe there was an abscess about three-fourths inch in diameter with thick-walled capsule containing pus. A fatty area on border adjoining this abscess. Near the margin of this lobe a depressed cicatricial line. In the main lobe of liver near caudal margin, 1 inch from gall bladder, a second abscess projecting above surface. Pus offensive in odor. A third abscess about three-fourths inch in diameter was near middle of liver.

In six mesenteric glands there were worm tubercles, consisting of mortar-like masses one-fourth to three-eighths inch diameter.

Uterus nearly normal in size. Horns contain a small quantity of blood-stained fluid. Neck sprinkled with minute ecchymoses.

No. 91 of herd 6. Weight, 700 pounds. Age, 9 years. Killed October 30, 1894.

In first dorsal mediastinal gland, slightly enlarged, there were a considerable number of yellow foci up to three-sixteenths inch in diameter projecting above cut surface; calcareous particles centrally. In a second small gland one such focus and in one of the upper series about six 1-inch foci.

Lungs: Right ventral and azygos lobe firmly adherent to diaphragm and to pericardium. In left caudal lobe a tuberculous focus of irregular outline, probably 2 inches across, consisting of a completely caseous focus and a hepatized region containing dilated air tubes choked with yellowish mucopus. Whether the softened focus communicated with bronchus was not determined, though this is probably the case. The mouths of the bronchi of this lobe contained only foam.

Embedded in azygos lobe was a mass of cartilaginous firmness and covered with connective tissue where it was adherent to diaphragm. It contained one one-half inch cavity filled with an earthy-looking, bulby, offensive fluid mass.

Pericardium firmly adherent to left ventricle of heart.

Ventral lobe of liver adherent to diaphragm. Considerable inflammatory adhesion of second stomach to diaphragm. Foreign body not traced. Some wires removed from stomach. Mesenteric glands generally slightly larger than normal. In three of them were a few one-eighth inch yellow tuberculous foci like those in mediastinal gland.

No. 92 of herd 6. Weight, 700 pounds. Age, 10 years. Killed November 1, 1894.

In the first dorsal mediastinal gland, about twice the normal size, the central half was occupied by a focus 2 by one-half by one-half inch made of multiple, small, yellow foci intermingled with a few larger somewhat softened foci. The tip of the gland was occupied by a small recent focus.

In right caudal lobe on the lateral margin, about 7 inches from caudal tip, was a triangular focus (about 1 inch to a side) with the pleura thickened and covered with connective tissue shreds. On section the focus was found to be composed of a uniformly tuberculous mass in which necrosis was general. A second small focus was contiguous to it. No discharge into a bronchus.

Extensive adhesion of convex surface of liver to diaphragm.

THE DISPOSAL OF TUBERCULOUS CATTLE.

The disposition to be made of tuberculous cattle is an important question, whether considered from the standpoint of the Federal and State government or of the cattle owner, and should be thoroughly planned by all concerned before proceeding to extremes in any direction.

There are three classes of dairies: First, city milk dairies—those in which the cattle are constantly changing; second, those which are essentially breeding dairies, but make milk and butter; and, third, the ordinary general-purpose dairy.

The breeding dairy is the one upon which most work must be expended, the one on which the greatest loss may fall, and the one most quickly recuperating losses.

In many of our large herds it is very desirable to keep the particular strain of blood embodied in the various well-known features of form, color, beef, milk, and butter fat production before entirely exterminating the tuberculous cattle.

In our large city dairies the presence of one or more tuberculous cows is far too common, but these are soon killed and are replaced by healthy stock. Over these periodic watch must be kept.

It is found on post-mortem examination that a few of the reacting cattle are in bad beef condition. Many may give off germs through the trachea and drooling saliva. By milk experiments it is proven that a few give off tubercle bacilli in their milk. While most of the cattle may therefore be regarded as of no positive immediate damage to the milk supply, but prospectively so, a few are absolutely dangerous. Some may in addition afford positive menace to the health of the other unaffected cattle and to the health of human beings through the germs contained in the floating dust. Unfortunately, there is no means of separating the dangerous from the nondangerous tuberculous cattle, and all must be considered dangerous.

In view of the desire felt by farmers to cleanse their herds with the least loss, and of the obligations to the public both in the exchange of animals and the sale of products, and also of the desirability of preserving certain strains of well-known and productive blood, the following course is proposed:

Carefully test the herd with tuberculin. If it contains a single tuberculous animal—

1. Separate the tuberculous from the sound animals. Isolate them in different pastures and stables.

2. If there is some doubt as to the reaction or the amount of tuberculosis is questionable, either save for a retest or fatten and have the cattle slaughtered for beef, with the understanding that if the internal organs are tuberculous the meat is to be condemned. Though the layman may find nothing, the disease is present in more than 95 per cent of reacting animals, and would be found by an expert. According to good authority, however, much of this meat is not to be condemned.[1]

3. Disinfect the stable with a 1:1,000 solution of corrosive sublimate after thoroughly cleansing the mangers, stanchions, ceilings, and floors, and then whitewash it throughout.

4. Have the reacting cattle thoroughly examined physically and kill those not passing physical tests unless retained for breeders. If very valuable cattle are pregnant at the time of testing and it is desirable to raise the calf, the cows may be used as breeders, especially if the calves are themselves tested after birth.

5. Continue to breed from the valuable cows, and as soon as a calf is dropped remove it to uninfected quarters, and either put it to another cow or put it on the sterilized milk from its tuberculous dam. The risk of leaving the suckling with the dam for the first day or two may well be taken, provided her udder seems to be sound.

6. Sterilize all the milk from reacting cows by heating it to 160° F. for ten minutes for all uses.

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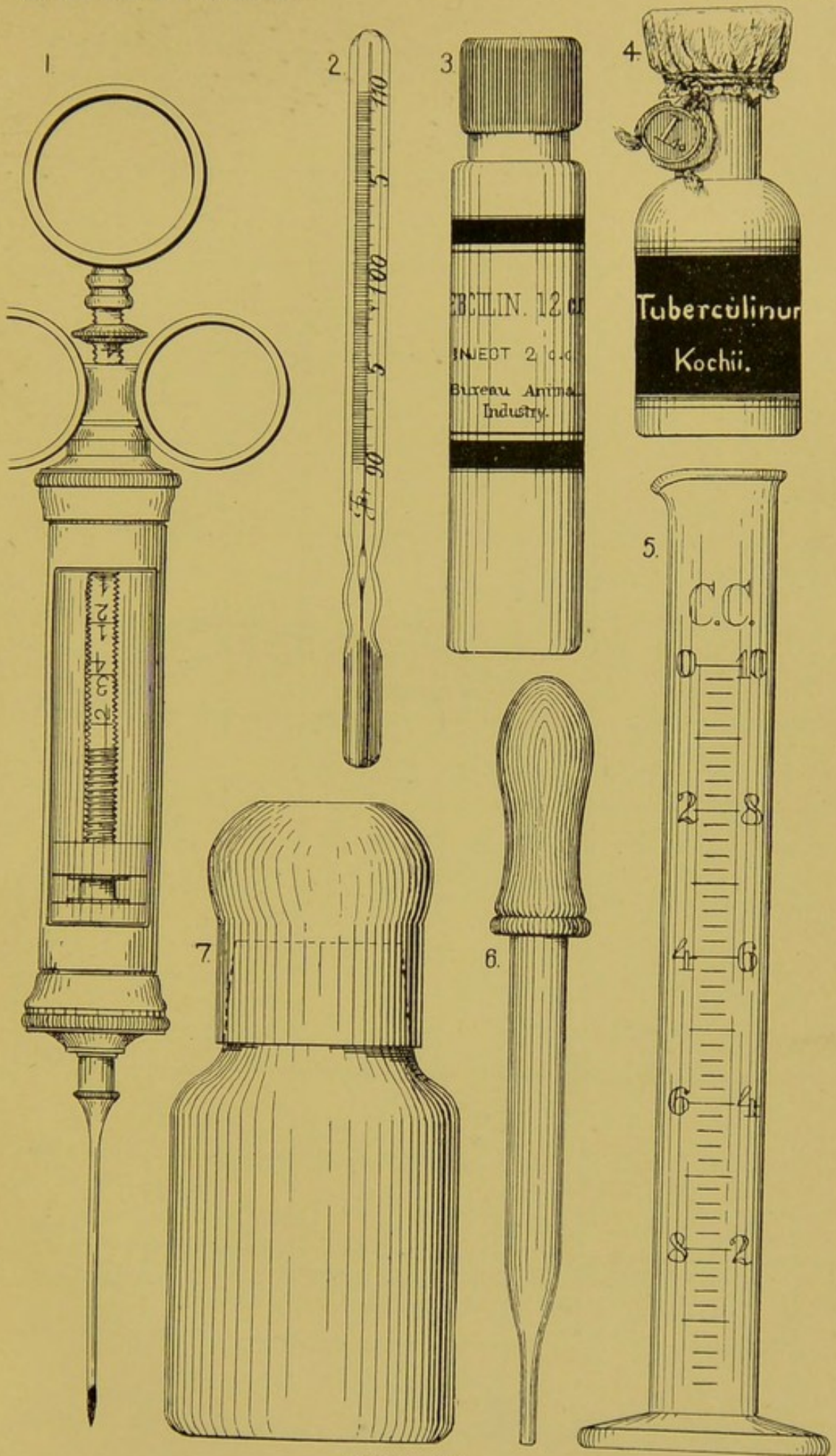
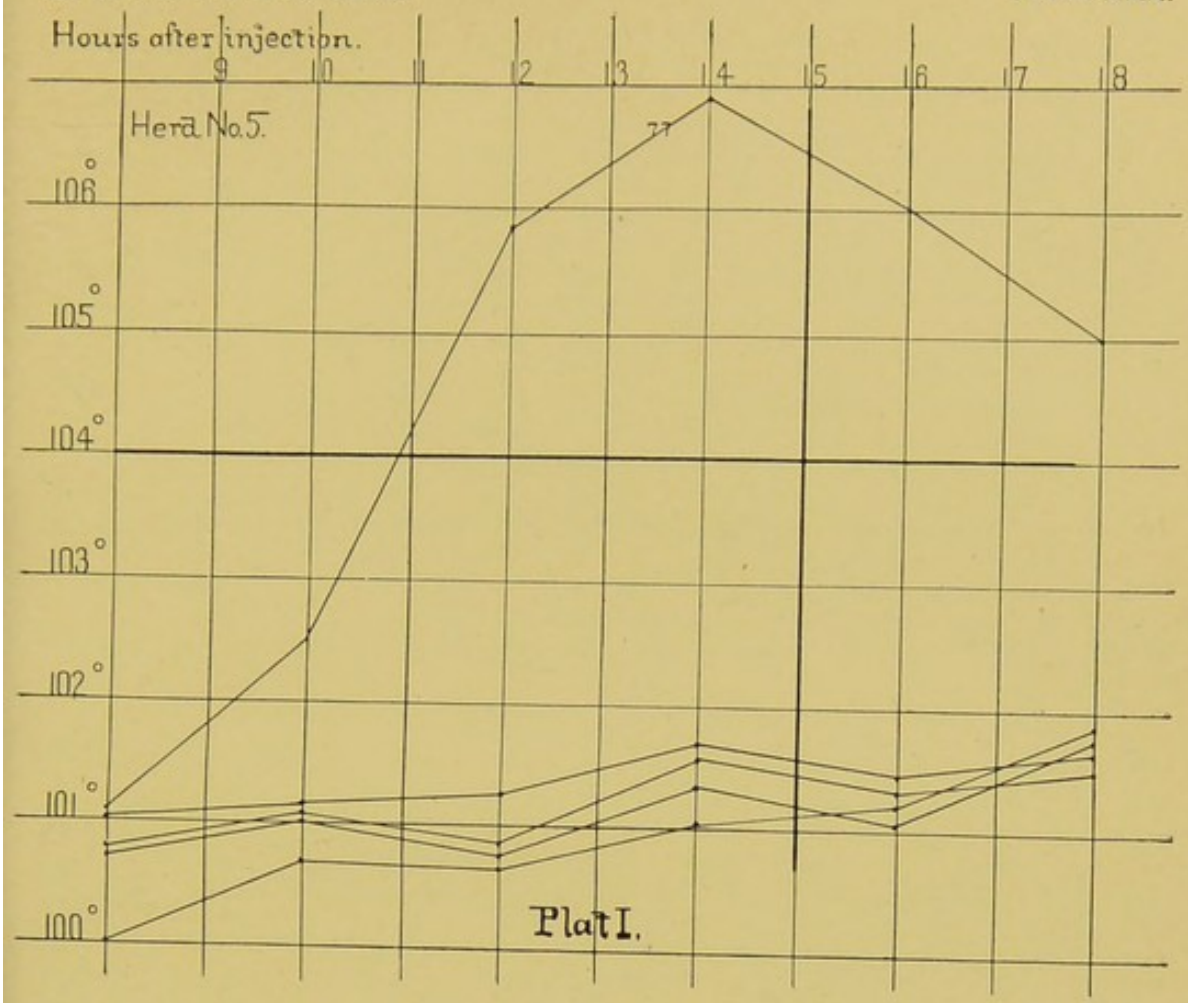
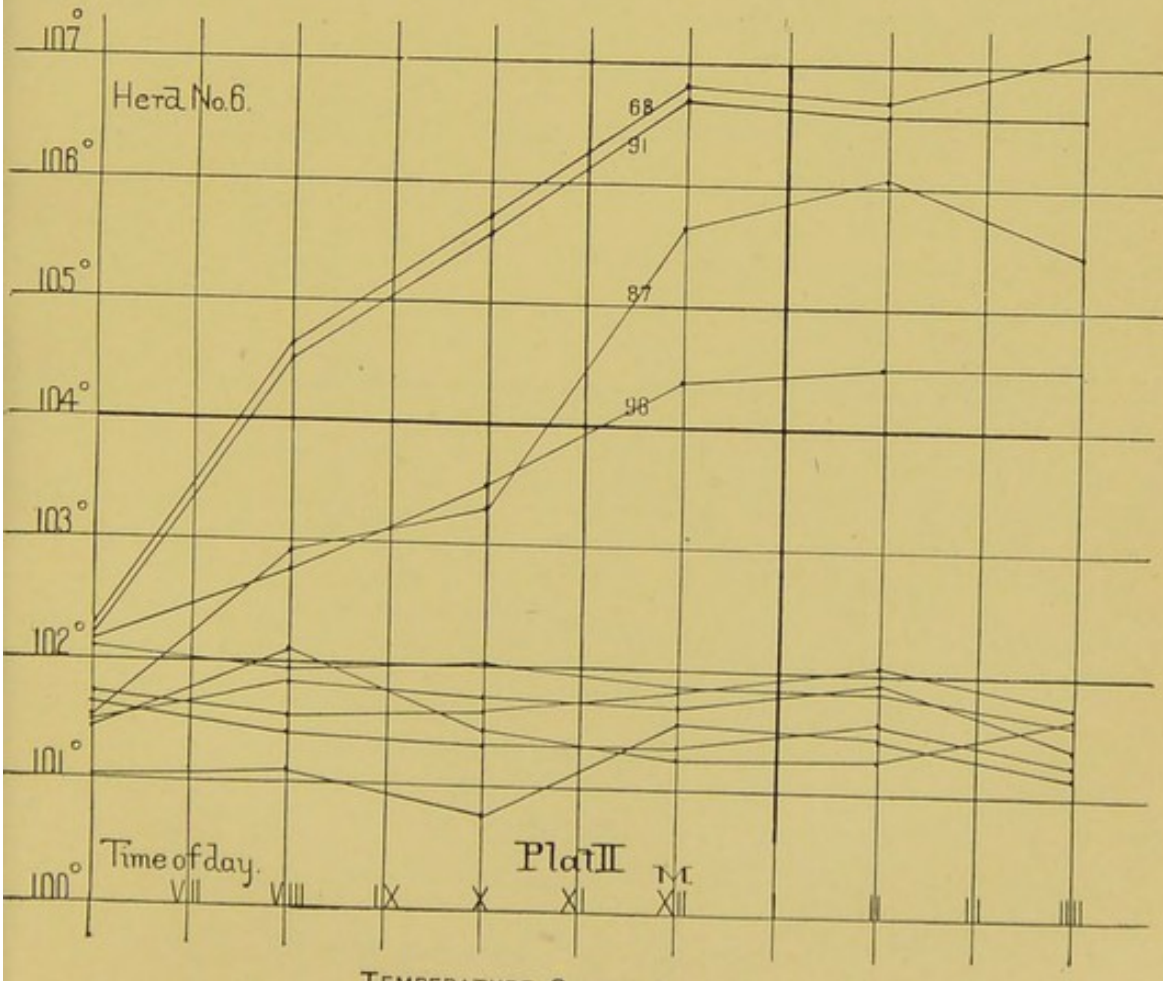
[1] The regulations of the Department of Agriculture, issued June 14, 1895, direct the condemnation of carcasses affected with "extensive or generalized tuberculosis."

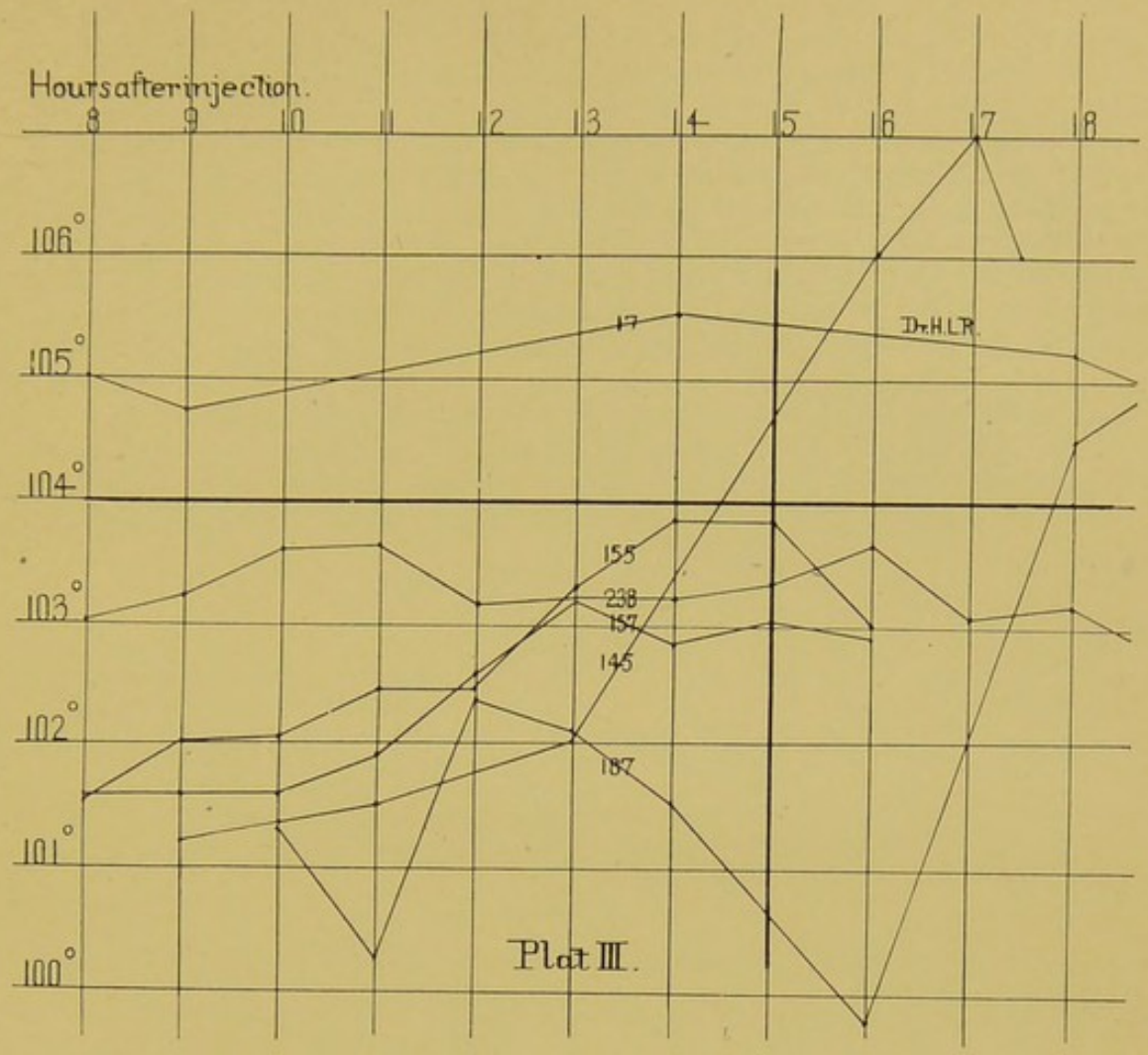


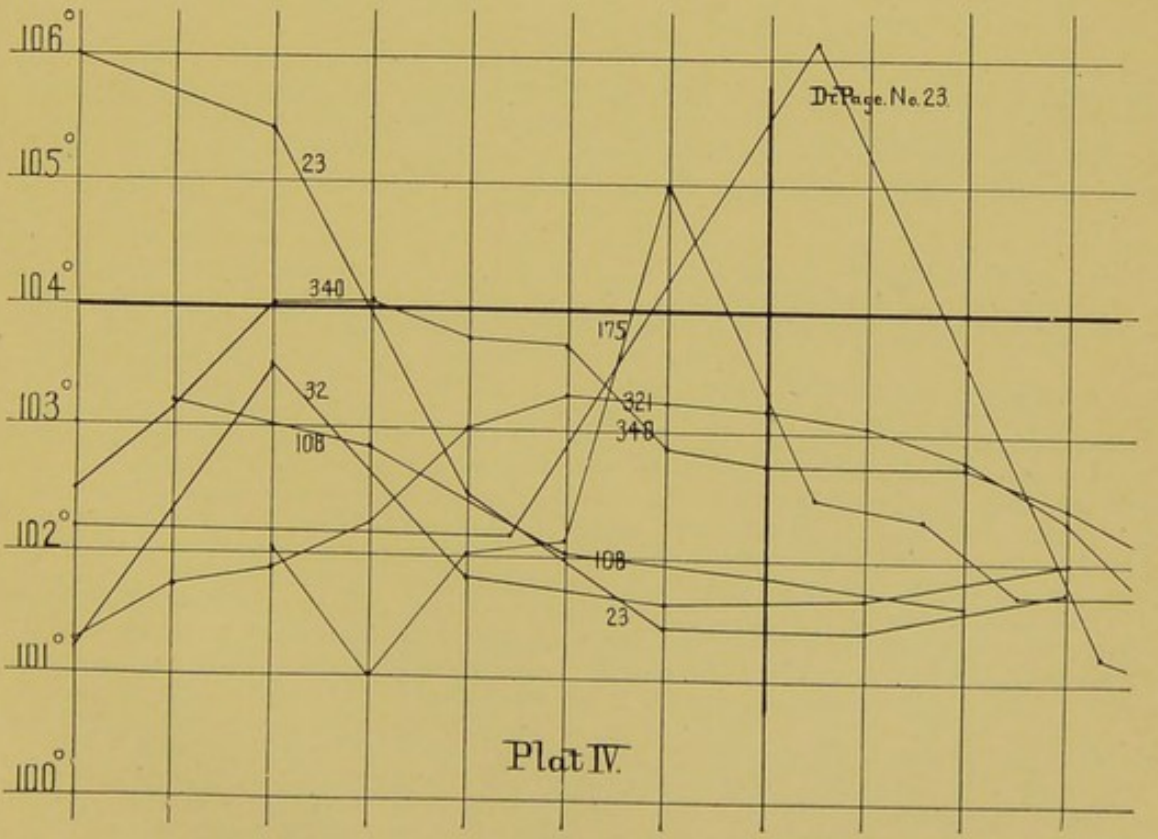
INSTRUMENTS.





TEMPERATURE CHARTS I AND II.





TEMPERATURE CHARTS III AND IV.

7. Calculate closely the difference in the breeding, the probable meat, and the milk values[1] of the cattle, and dispose of them either by slaughter before or after fattening or by retention as breeders, or as producers of milk to be sold only when sterilized, and as such.

8. After six months, retest the herd and again separate as before. Each time the herd is tested fewer cattle will react, until within two or three years a new uninfected herd will have grown from the old one.

9. Particular care should be taken with the infected stable in at least weekly cleansing and disinfecting all its parts. Sprinkling the floor with water before sweeping is advisable, and will prevent the raising of dust.

In following such a course as the one outlined the farmer will incur a minimum of expense. If, as is the better way, the public steps in to share the loss and eradicate the disease, it is possible that an entirely different mode of procedure will be found better.

DESCRIPTION OF PLATES.

PLATE XXIX.

Fig. 1. Hypodermic syringe.
2. Clinical thermometer.
3. Sample bottle of Bureau tuberculin.
4. Sample bottle of Libbertz's tuberculin.
5. Metric graduate, holding 10 c. c.
6. Pipette with rubber cap, used for dropper.
7. Glass-capped bottle, used for container.

PLATE XXX.

Plat I. Test temperature curves of 5 cows, all those of herd No. 5. No. 77 was tuberculous.

II. Test temperature curves of 10 cows, all of herd No. 6. Nos. 87, 88, 91, and 96 were tuberculous.

PLATE XXXI.

Plat III. Six temperature curves of tuberculous cattle which depart from the usual form. These are the exceptions.

IV. Seven temperature curves of nontuberculous cattle. Nos. 32 and 108 were not slaughtered. These are the majority of such cases in over 200 tests.

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[1] In nearly all States there is a law against the sale of diseased cattle and of their milk. In most States tuberculosis is designated by law as a contagious or infectious disease. Cattle affected by tuberculosis in the least degree are diseased and milk coming from them is from diseased cattle.

Tuberculous cattle must not be sold. Since pasteurization or sterilization of milk from tuberculous cows prevents spread of disease, it would seem that such pasteurized milk, being no longer harmful, might be sold by common consent, when the seller makes known its nature. Since authorities declare that meat from cattle with but few tubercles in their viscera is harmless, it would seem that such meat can not be considered as diseased. The sale of condemned animals to the butcher should not be completed until their exact condition as regards tubercle is learned after slaughter. The slaughter should take place in the presence of health authorities, their representatives, or of other competent disinterested witnesses, thus shielding the owner and the butcher from imputations of selling diseased meat from condemned cattle. While the driving of tuberculous cattle over the highways causes practically little danger to others, such cattle when driven should be kept entirely separate.

Federal regulations issued by the Secretary of Agriculture December 13, 1895, require that animals affected with tuberculosis shall not enter into interstate trade, into stockyards, or other places where animals are handled for interstate trade. Both the Federal regulations and the State regulations should be carefully observed in all instances.

CREAMERIES OR BUTTER FACTORIES; ADVANTAGES, LOCATION, ORGANIZATION, AND EQUIPMENT.

By Henry E. Alvord, C. E.,
Chief of Dairy Division.

The system of making butter in creameries or factories is in many respects a vast improvement over the ordinary farm-dairy practice. Where the conditions are favorable, a well-managed creamery is a source of profit to the farmers of the neighborhood and a real blessing to their families. But favorable conditions do not always prevail by any means, and many communities have suffered serious loss as the result of not making a thorough study of the subject before embarking in the creamery enterprise. The question of starting a creamery has two sides to it, and both should be fairly considered. Although this matter has been frequently and exhaustively discussed, there are parts of the country in which the subject of starting creameries is now being first agitated, and it may be well to again present the same, and especially those points which should receive attention by the farmers in any locality where such a project is under consideration.

The establishment of the creamery as a central place and improved plan for making butter for a number of farms, is as natural a development and as distinctly a forward movement as the substitution of the gristmill and the woolen factory for the old methods of grinding, spinning and weaving upon the farm. Regarding milk or cream as raw farm products, like grain and wool, the same arguments apply in favor of manipulating or manufacturing them in one place instead of in several or many different places. Whether the change is made by reason of cooperative effort among the producers or as a business enterprise introduced by one or more persons, the probabilities are that the result will be economy in manufacture and improvement in the average quality of the product. There may be poor gristmills, or too many of them, and unsuccessful factories, but these are not sufficient arguments to cause a return to farm grinding or the home loom. The operation of the creamery, like the mill, is manufacturing and not farming. All such establishments must be judiciously located and properly managed on business principles. They are subject, like all business ventures, to ups and downs, periods of prosperity and of depression, and those who supply the raw materials will find the prices received to vary accordingly. But spinning wheels do not reappear with decline in the price of wool, and the same is true of churns in the well-developed creamery districts. There are cases in which farming is conducted on so large a scale, or where power is available or

cheaply supplied, or the uses are such to which the grist is applied, that the farm mill is desirable and economical. There are likewise cases, and a good many of them, where the conditions for butter-making on the farm are so satisfactory, the product so good, and the opportunities for delivery and sale so exceptional, that the creamery offers few advantages; in these instances a change of system is not to be advised. The creamery plan or factory method is a positive advance and a permanent one, in butter making. It will gradually supersede farm dairying in a great majority of cases in most of the States. And yet, in every locality and in the case of every farmer, the conditions and circumstances should be studied, the advantages and disadvantages reviewed, and the question decided upon its merits.

The introduction of the creamery system in any community is of the greatest benefit to those who make butter which is poor, common-place, or not above the average farm-dairy product in quality, and such makers are generally in the majority. It is of less advantage to those who make a superior article of butter and have a fancy market for it. And yet the latter are pretty certain to become, in the course of time, patrons of any successful creamery in their vicinity and among its stanchest supporters.

In nearly all manufactures the cost of production per unit decreases with increase in the quantity produced. And as system in the work proceeds and the methods and appliances improve, the quality of the product becomes better and more uniform. These business principles apply forcibly to the creamery. It is reasonable to believe, and has been abundantly proved, that if the milk or cream produced on a hundred farms is taken to one place and made into butter, the work can be done at a less cost per pound than if made on the hundred farms. And if the factory is well equipped and well conducted, with an expert butter maker, the butter produced is certain to be of higher quality than the average of the butter from the hundred farms, with their many different makers; and of course the product is more uniform. In every dairy there is always more or less waste and loss in handling the materials, no matter how well managed, and unless churning is done daily there is much cream churned when too old or not old enough. At the creamery the proportion of waste and loss is much less and all cream can be churned at exactly the right time. The result is that the creamery can make more pounds of butter from a quantity of milk or cream than can be done if the same material is worked up on many different farms. The cost per pound of sending to market and selling a thousand pounds of butter in one lot is less than if handled in a hundred different lots, and if the one large lot is of uniform quality, while the smaller lots differ greatly in appearance and grade, the average selling price will be decidedly higher for the former. It is not business-like or according to common sense to have going on at a number of different places, some of them unsuitable and inconvenient, the work of churning and slopping and cleaning, with printing or packing and selling, by different persons of whom a fair share are certain to be indifferent makers, when the whole can be done much better at one place especially prepared and fitted for the work and in charge of an experienced and successful butter maker. These are old and time-worn arguments in support of the factory system of making butter, but they are well sustained by experience.

The particulars might be given of hundreds of cases in which a creamery has been substituted for farm butter making in a stated

community or neighborhood with these definite and satisfactory results:

1. The quality of the whole factory product equal to the best of the single dairies of the same community.
2. A greater quantity of butter produced from the same cows.
3. The average selling price and the net return per pound of butter considerably increased.
4. The gains sufficient to cover the whole cost of running the creamery, thus causing—
5. The cash income to the farmers, from a given number of cows, as much as by the old system and often more, while at the same time
6. All the labor, trouble, and expense of making and marketing the butter are removed from the farms and the households.
7. Relief from the labor of caring for milk and cream usually results in adding to the number of cows, and the effect of the friendly rivalry between the patrons of the creamery, with the frequent money measure of the butter capacity of the cows, tends to a constant improvement in the quality of the latter and in the consequent profit.

A single example is as good as many. In the year 1884 a creamery was located in a certain town not especially noted for good butter. It began with 17 patrons owning 100 cows, and the daily product was 79 pounds. In 1893 it had 116 patrons holding 1,000 cows, and its annual product per cow was 289 pounds of butter. The product has sold at the highest market price and the cow owners have received a greater net income per cow than ever experienced before. Nothing would induce these patrons to return to farm dairying. They received from the factory during nine years $458,000 in monthly cash installments. The market for their butter is at their doors and they can pay cash for all their supplies. Farms and farm buildings have been improved. Dairy stocks have been doubled and trebled. The community has taken on a new inspiration. A large grange has been formed and has become a power for good. It is alive to the most progressive measures for intellectual and moral culture, and through it many business matters have been most economically transacted upon the cooperative plan.

The creamery[1] or butter factory is an institution of American origin. The butter factory followed the cheese factory and was a natural development. Cheese factories having been in operation in the State of New York and elsewhere for several years, the first creameries were established as follows: In New York, 1864; in Illinois, 1867; in Iowa, 1871; and in Massachusetts, 1880. During the last fifteen years the growth of this form of dairy industry has been very rapid, as shown by the number of factories reported by several States in the years named, as follows:

Iowa—1874, 45; 1880, 244; 1888, 468; 1895, 855.
Illinois—1874, 45; 1880, 285; 1890, 396; 1894, 530.
New York—1865, 484; 1874, 1,018; 1880, 1,652; 1894, 1,624.
Vermont—1874, 32; 1880, 85; 1890, 124; 1895, 250.

Cheese factories are included in these returns, but they are not numerous in any of the States named excepting New York. In Iowa,

[1] It must be understood that the word "creamery" is properly applied only to an industrial establishment at which the milk or cream is received from a number of producing farms and disposed of (ordinarily) by making into butter. It is a frequent error to designate as a "creamery" a milk setting or creaming apparatus for use in a private dairy. Such ought to be called a "creamer," but the butter from it is not entitled to be called "creamery butter."

for example, of the 855 establishments last reporting, 64 were cheese factories, and in 17 both butter and cheese were made. Iowa is now the leading State of the Union in number of creameries, in the quantity of creamery butter annually made, and also in total butter produced. Yet, notwithstanding the large number of creameries in operation, the proportion of all the butter produced which is made in factories is still comparatively small. Iowa makes a little more than one-tenth of all the yearly butter product of the country, and yet less than half of this comes from her creameries. In Illinois, which produces 7 per cent of the butter crop of the United States, about one-third is factory made. New York produces over 9 per cent of the butter of the country, but of this only one-eighth is from creameries.

Taking the country as a whole, no butter factories and no factory butter were reported by the census of 1870. In 1880 the creamery butter reported was only 3½ per cent of the total, but in 1890 the proportion of butter made in creameries had increased to over 15 per cent. It is estimated that the butter produced in the United States during the year 1896 will amount to 1,400,000,000 pounds, of which something more than one-fifth, or about 300,000,000 pounds, will be made in the factories or creameries, of which there are now nearly 10,000 in operation.

Creameries differ in their form of organization and system of management, and also in the way in which their operations are conducted. In the first respect, they may be divided into three classes, (1) purely cooperative, (2) joint stock companies, and (3) proprietary concerns. In the first form the owners of the cows which contribute the raw material also own the factory. These "patrons" share in the expense of starting the concern, according to the number of cows they respectively keep, or upon other agreed basis, and the management is vested in a committee or board chosen from their own number. The cost of running the factory and the proceeds of sales are divided pro rata according to the milk, cream, or butter fat contributed by the patrons. In many respects this is the most desirable plan of creamery organization. The success of the enterprise depends upon the farmers themselves; the profits all go to the cow owners, and if it fails they have none but themselves to blame. But such cooperation can not be successful unless it is thoroughly harmonious; the patrons must find good men among their own number to manage the business, or else employ a capable manager, and must then unitedly sustain the management and strictly conform to all needed regulations.

A joint-stock company may be formed to establish, build, equip, and carry on a creamery the same as any other business; the stockholders may be residents of the neighborhood or entire strangers, and may or may not include the cow owners who are to be the patrons. The raw material is bought of the producers under some form of monthly or yearly contract and the profits or losses apportioned among the stockholders. Or the factory may make butter and dispose of it for its patrons for a fixed price per pound. Such a concern is likely to be most prosperous if the stock is mainly held by farmers who are also patrons, and if some of the experienced and enterprising business men of the community also have an interest.

The proprietary or "independent" creamery resembles other industrial enterprises. An individual or a firm furnishes the capital, conducts the business and takes all risks, and the relations of the proprietors and the farmers or patrons are simply those of buyer and seller. Those producing milk or cream dispose of it to the factory or

not at pleasure and under such terms as may be mutually agreed upon. In some instances the factory does the work at so much a pound. In communities where the farmers are not disposed to pull together very well (and such are to be found occasionally), or where the right sort of managers are lacking, this proprietary form of creamery organization is found the most desirable.

A plan which has proved successful in many places, unites features of the joint-stock and cooperative systems. The creamery being established, owned, and started by a joint-stock company which includes some of the principal patrons, it is then managed upon the cooperative plan, a reasonable dividend being paid on the capital stock as a part of the current expenses, or the factory receives a stipulated price per pound for making and selling the butter, which is deducted from the gross receipts, and this covers all operating expenses and interest on the plant. In such cases provision should be made for a slowly accumulating contingent fund to maintain the plant and meet unexpected needs or losses.

The three classes into which creameries may be divided according to methods of operation are, (1) separator creameries, (2) gathered-cream factories, and (3) whole-milk creameries.

In most of the active creamery districts the separator factory is now the favorite system. It is based upon the use of the dairy centrifuge or mechanical separator for extracting cream from the milk. The whole milk is brought to the factory or separator station while it is sweet, and immediately passed through the centrifugal separator. The skim milk may or may not be returned to the producing farms. The milk brought to the creamery may be paid for at a uniform rate per hundredweight, or upon the basis of the fat which it contains (that is, its butter value), determined by one of the several methods in use. This form of creamery possesses the advantage of requiring storage for cream only, and also of taking the milk from the farms while fresh, thus relieving the producers from the necessity of having a supply of ice or cold water to preserve the milk while creaming. It is therefore especially adapted to warm climates. But the labor of hauling the milk daily from farm to factory and the skim milk back again, as should always be done, is very objectionable. In almost all cases the hauling devolves upon the milk producer. Another advantage of this system is that one concern may establish any number of "separating stations" located conveniently for the patrons and to which they haul their milk. These places may be small and inexpensive, the power, the separator, and temporary storage for cream being about all that is needed. One factory is located for a group of stations within reach, and the cream from the latter hauled to the factory by its own team or teams and the butter making done at this central plant.

The cream-gathering plan originated in Wisconsin, was first widely adopted in Iowa, and was the basis upon which creameries were established in New England, where it continues to be the favorite operating system. For some years this plan required all the farms contributing to any one factory to have uniform creaming appliances and treat their milk exactly alike. The milk was set on the farms where produced and the cream daily measured, removed (skimmed), and taken to the factory by its agents, known as cream gatherers. The cream was paid for equally, according to measure, volume, or "space," it being assumed that where the milk was all treated alike equal quantities of cream from different farms and herds had practically the same

butter value. The "space" was an inch or other unit of depth in cans of uniform size. An improvement on this plan is to test every lot of cream gathered or taken to the factory and pay for it according to its ascertained percentage of butter or butter fat. This has led to allowing every patron to handle his milk to suit himself, and to do his own skimming, if desired. One farmer may now use deep open setting and another some patent creamer, while a third has a small farm separator. The cream may still be called for and hauled to the factory by the agent or gatherer, or may be sent by cooperation among neighbors, or it may be shipped by rail to a creamery at a considerable distance; this is frequently done. The cream may go to the factory daily, or every other day, or even longer intervals at some seasons. These varied possibilities make this the most independent and elastic of the systems in vogue. Cream becomes the raw material offered by its producer to any factory within reach and transported as may be most convenient. The producer manages his milk and cream to suit himself and every lot of the latter is accepted at the creamery after careful inspection and test and paid for at its actual butter value at the time and place of delivery. In its original form this cream-gathering plan necessitated an abundant and reliable supply of water and ice on the part of every patron. Under the modifications mentioned this is no longer essential, and with the centrifugal separator the farmer can prepare his cream for the factory as well in Mississippi as in Maine or Minnesota. The gathered-cream system has the very great advantage of never moving the milk from the farm where produced; the factory has no bother with milk; all the heavy hauling, usually both ways, is saved, and the skim milk remains where it can be used in its best condition and to the best advantage. As an offset to the hauling saved, the farm must care for the milk-setting apparatus or for operating the separator.

The old style of whole-milk creamery has practically disappeared. Taking pattern from the cheese factory, the first creameries established received the milk fresh from the farms, daily or twice a day, paid for all alike by the hundred pounds, set the milk in large shallow pans or in deep cans immersed in a pool of cold water—depended upon gravity for separation of the cream—and skimmed by hand. The skim milk was a waste product, and often hard to dispose of to advantage. Of course the storage of large quantities of milk, with the water and ice incidentally required, necessitated a larger and more expensive factory than essential to either of the other forms of creamery already described.

Farmers contemplating the establishment of a creamery in their locality will do well to carefully consider the cooperative system and the cream-gathering plan modified as described, with payments upon the fat test of cream received.

STARTING A CREAMERY.

The best place for the successful introduction of the creamery or cooperative system of butter making is a farming district where dairying has been long in practice, with butter as a leading product and yet not of the highest reputation as to quality. Such conditions insure the cows needed, and those of the right kind, with proper care of the animals and knowledge of the details involved.

There are many creamery buildings standing useless in different parts of the country, representing unproductive investments by neighboring farmers, which aggregate some millions of dollars, because the

conditions necessary to success were not recognized in advance. These creameries have been built in communities having no knowledge whatever of dairying, the farmers unfamiliar with cows and their care and, if accustomed to cattle at all, having those of a kind unsuited to milk production.

If the question of starting a creamery becomes one of local interest anywhere, the best course to pursue is as follows: Let an informal meeting be held of those interested in the subject. Endeavor to bring to this meeting the owners of the largest herds of cows or other cattle in the vicinity, whether they are favorable to the project or not. The experience of such men, on various points involved, will be of value. Also invite to the meeting a few of the local business men, whose interests are closely connected with those of the farmers, and whose business training and advice will be useful. If possible, secure the presence at this meeting of some successful creamery manager or a farmer who is a creamery patron and perhaps on the board of management. Even if such a person has to be sent for some distance and at considerable expense, it will be a wise and economical preliminary step to take; he will be able to give the experience of those who are engaged in a like enterprise elsewhere and to answer many practical questions in connection with the matter. The man who is not wanted at this meeting is the one whose business it is to "get up creameries," build them, and equip them and get the farmers' money. The influence of the professional "creamery promoter" is to be avoided at all stages of the movement.

After a general conference and interchange of opinions, with such reliable information on the subject as can be obtained in the way suggested, if it appears that the project is so favorably received as to warrant further action, let the meeting appoint a representative committee of three or five men to make a thorough investigation of the subject as a business proposition. This committee should include a representation of the business men of the community, and a subscription should be made to pay the expenses. This provisional committee should be authorized to call a formal meeting for organization, if such action becomes expedient.

The committee should carefully consider the conditions essential to success in starting and conducting a creamery, and whether these conditions exist or can be supplied in the locality concerned. It will be well for the committee, or some members of it, to visit a creamery in active and successful operation and confer with its superintendent or board of managers. Farmers who are patrons of this creamery should also be visited, to get their opinion of its operations and results, its advantages and disadvantages to the cow owners.

The following are among the conditions which are essential to a new creamery or which will largely contribute to its success:

The first requirement is a sufficient number of cows, owned within reach of the proposed location of the creamery, and whose milk is absolutely guaranteed for six months or more, to start the concern. It is folly to establish the creamery first and expect the cows to be forthcoming later. If well conducted, the creamery will cause the number of cows within reach of it to increase, beyond a doubt, but there must be enough of them and their milk must be available the day the factory starts, and be secure until it is fairly established, to allow the work to be conducted on a paying basis from the outset.

Large creameries are usually more profitable than small ones, because they can be more economically conducted. Upon the cost per pound

of making and marketing the butter depends the return which can be made to the farmer for his milk or cream. A thousand-cow creamery can usually do better on this account than a smaller one. The total cost of collecting the milk or cream, running the creamery, and marketing the product, including interest on the plant or investment and provision for a moderate sinking or contingent fund, should never exceed 4 cents for every pound of butter made. This can be and should be reduced to 3 cents under favorable conditions, and it is claimed that creamery administration can be so perfected as to reduce the cost to less than 2 cents. This is practicable where patrons deliver their own milk or cream at the factory. As a small creamery can not be operated, especially when all is new, at less than $4 or $5 per day, it becomes evident that the daily product should be over 100 pounds of butter. A safe minimum is 150 pounds for every working day; 200 pounds is better. Consequently no creamery should start its operations unless having control of the milk or cream from 250 cows at the very least; 300 cows is a much safer minimum. The greater the number of contributing cows, up to 1,000 or 1,200, the greater the likelihood of a successful enterprise; and the better the quality of the cows as butter producers, the more certain the success of the creamery. Every factory must have a reliable and sufficient supply of raw material to manufacture, and the higher its quality the more satisfactory the results.

It is necessary to have the farms of the cow owners who patronize the creamery accessible to the latter. No exact maximum distance can be named, because much depends upon the condition of the roads and the system of operating the factory. The closer or more compact the location of creamery and contributing farms, the better for the business. The cream-gathering plan is the best suited to long hauls between farm and factory and to a sparsely settled district. In some cases factories send gatherers to farms 12 or 15 miles distant for cream, making gathering routes nearly 30 miles long. If the farmers have to haul their milk to the factory daily and wait while it is separated, the patrons should all be located within 4 or 5 miles, measured by the roads necessarily used. As the creamery ought to be operated throughout the year, the condition of the roads at all seasons must be considered.

If the farmers who expect to become patrons are not cow owners already, it must be ascertained that they have suitable provisions for keeping cows, as to stables, pastures, water supply, and facilities for producing an abundance of good forage.

A good location for the creamery itself is of the utmost importance. It must have a reliable supply of good water and ample provision for good drainage. The water must be pure and guarded against future contamination. If ice can be abundantly and cheaply supplied, the temperature of the water is immaterial; otherwise the water should be available in the working room at a temperature not exceeding 60° F. at all seasons and the nearer to 50°, the better. The wastes of a creamery are large in quantity and very offensive and troublesome if not disposed of quickly and thoroughly. The drains must not only carry the waste to a safe distance from the creamery, but so dispose of it as to avoid complaint. Creameries have been closed as nuisances by legal process because drainage found lodgment at a distance of half a mile or more. Too much attention can not be given to this feature of the location. The surroundings should provide pure air and freedom from dust; exposure to direct sunlight should be suitably modified. Location with reference to the producing farms, the point of shipment

for products, and the markets to which the products go, deserve attention, although comparatively unimportant.

The cost of real estate and the question of renting, rather than buying or building, demand consideration. In numerous cases a new enterprise of this sort, especially if cooperative, will find it expedient to lease some building which can be fitted to its purposes without too much expense, and defer building until the business is well established and its needs demonstrated. Many a creamery enterprise inaugurated under otherwise promising circumstances has been handicapped by starting with too heavy a load of real estate, in kind and cost.

A creamery having the capacity to handle the milk or cream of 300 to 500 cows can be built for from $800 to $1,500, and the equipment will cost about as much more. It is, therefore, possible to start a 250 or 300 cow creamery with an investment of $1,500 to $2,000, which may be increased with some advantage to $2,500 or even $3,000. Of course much depends upon the local cost of materials and building expenses in general.

If the provisional committee decides, after a thorough investigation of the general subject, that the local conditions are favorable to establishing a creamery and making a success of it, two papers should be circulated for signature. One should pledge the milk from a stated number of cows, to be contributed to the factory for at least six months, and preferably for a year, upon terms to be fixed and equitable for all patrons. The other should be subscriptions to the capital stock necessary to start the creamery; whether the latter shall be confined to cow owners and prospective patrons, or whether others in the neighborhood who incline to help the enterprise shall be permitted to subscribe, is a matter for local decision.

A plan which has been found satisfactory in some places is to have but one subscription paper, or "organization agreement," substantially of the form given in Appendix A. This provides for borrowing the necessary capital upon the joint security of the patrons. This debt is then gradually paid off by setting apart a certain percentage of the gross receipts from sales until the company owns its factory. A model form for full articles of agreement and by-laws, according to this plan, is given in Appendix B.

Cows and capital being secured, a meeting for organization should be called. Property rights and business questions being involved, it is usual to permit only those who have subscribed to the capital to take part in the organization. In nearly all cases it is advisable for the enterprise to be incorporated, and as State laws differ much as to corporations it is impossible to prescribe an exact line of procedure to be followed. Legal advice should be obtained in order that all action may be regular and future trouble avoided. By way of example, a plan of organization is appended which is very simple and has been found sufficient and satisfactory after use for years by a factory run on the cream-gathering plan. This may serve as a general guide, but will doubtless have to be modified to meet legal requirements and accord with local conditions. (See Appendix C.)

The administration of the business will be simplified, economized, and rendered most effective if a single executive board is created to be responsible for directing all affairs of the company. This board should not be too large, seven at most, and five are enough. In selecting these directors or members of the board of management, all personal ambitions and jealousies must be laid aside and the very best

men chosen for the service to be performed. Success upon the cooperative plan is impossible unless absolute harmony prevails and the board has good business capacity.

Whatever the form of organization, it is usually found to be expedient and true economy, provided the business is large enough to justify the expense, to employ a thoroughly competent manager to look after all business affairs. The manager should serve under the supervision of the directors or governing board, but be given full discretion in all matters of detail. He should deal with the patrons, control the plant, employ and direct the butter maker and all help, purchase supplies, make sales and shipments, keep the accounts, and attend to all collections and payments. In short, he should be the actual and active head of the enterprise, and as such be cordially supported by directors and patrons.

It is sometimes possible with small creameries to secure a butter maker who is competent to act as business agent also, and it is well, in all cases, for the butter maker to know about the markets for his product, the selling prices, tastes of consumers, and the minor points which affect sales.

CONSTRUCTION AND EQUIPMENT.

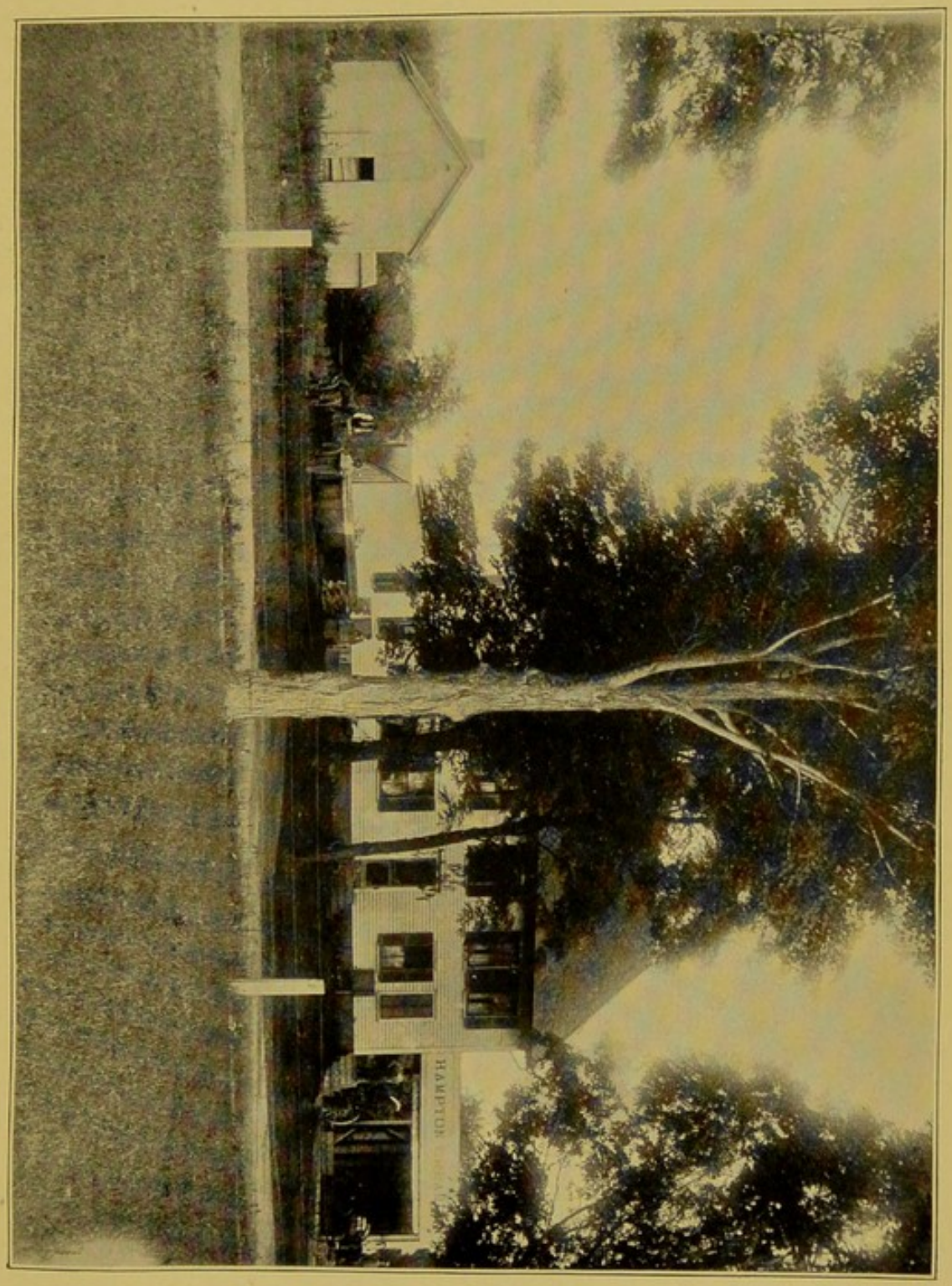
Organization being accomplished, one of the first duties of the directors will be to provide a suitable building for the work and equip it with the necessary machinery and appliances. In doing this the experience of successful creameries should be sought and they should be such as have been in operation long enough to determine the suitability of their own accommodations, and wherein changes, if any, are desirable. Several of the large and reputable creamery supply houses are prepared to furnish a variety of plans and specifications for creamery buildings, with approximate cost, from which selection can be made.

In some localities it will be found expedient to have the main working and storage rooms on the same level, and in others a two-story or hillside pattern will be more suitable. There is great convenience in having easy access to one room above the floor used for most of the work. The milk or cream can thus be received in the upper room, being lifted only when taken from the wagon, and then can be run by spouts and conductors to vats, separator, and churn below. This can be accomplished with a ground floor and basement for working room, or, by a high foundation, receiving room on main floor and a semi-basement for work. Lifting milk and cream is very heavy labor and should be avoided; so, also, should pumps and elevators for milk or cream.

If the factory is intended for making both butter and cheese, plans must be modified accordingly. In this case a good deal more room is needed for storing and handling milk in quantity, and a cheese-curing room must be added and constructed with care, to secure control of temperature and ventilation.

Plans having been agreed upon, experience proves that better results are usually obtained by having the building done by local workmen rather than by contract with professional creamery builders from a distance. The same general course should be followed in the selection and purchase of equipment. Determine by consulting with experienced and disinterested persons what is necessary or desirable,

DWELLING HOUSE IN WHICH THE CELLAR AND FIRST FLOOR HAVE BEEN REFITTED AS A CREAMERY.



Front and End of House in Plate XXXII, showing Position of Engine and Boiler Room.

and then submit a schedule of articles wanted to a number of supply houses for competitive bids.

There is no economy in starting operations in an inconvenient or crowded condition or with poor tools. But there should be no unnecessary investment in real estate or appliances at the start. Let the plans adopted provide in advance for suitable addition to the accommodations when required by the expansion of the business. Implements can be improved or added to as becomes necessary. A frequent error, which should be guarded against, is starting with an engine and boiler too small in size. This is an expensive part of the outfit, and changes cost too much. It is true economy to anticipate the time when more work will be done, and secure an engine of ample power. The boiler should be three or four sizes larger than the engine requires, as extra steam in considerable quantity is constantly wanted for cleaning, heating water and vats, running a tester, pumping, and like purposes.

If the local directors will give sufficient time and attention to the matters of building and equipment, obtaining advice from the right kind of people, the total investment can be held closely to the necessities of the case, and a high quality can be secured in the property acquired. In most cases where professional "promoters" go into a community, "get up a creamery," build and equip it, complete, they get from the farmers two or three times as much as the property is worth. And then, after the factory has started and the local managers begin to gain experience and learn their actual wants, they find the equipment deficient in parts, poor in quality, and inadequate for the work in hand.

Lists are appended of the articles of equipment which may be recommended for a creamery handling the milk or cream of 300 to 500 cows, and operated either as a separator factory or upon the gathered-cream plan. The prices attached are only approximate. Discounts from those named can usually be obtained for cash payments. The additional cost of transportation and of having the machinery set in place and put in operation by experienced workmen, must be considered; this varies so much with circumstances as to make estimates impracticable. (See Appendix D.)

By way of example, illustrations follow of two factories started in rather different ways, with brief descriptions of the circumstances in each case.

The Hampton Cooperative Creamery Association was one of the very first organized in New England, in the year 1881. This was on a purely cooperative basis. The cows were pledged, the articles of agreement adopted (as per Appendix C), and stock subscribed to the amount of $2,000. This was divided into 100 shares of $20 each and distributed as widely as possible among the cow owners who agreed to become patrons. Half of the capital was invested in a lot of 2 acres at the extreme end of the village, upon which was an old but substantial dwelling house of two full stories, large storage attic, and an excellent cellar, high and dry. In the cellar was a well of uncommonly good water. The two engravings from photographs (Pls. XXXII and XXXIII) show this building, which has been changed very little in general appearance during the past fifteen years. It was believed that this property could be fitted cheaply to answer the early needs of the work, and that the alterations, fittings, and equipment would not cost over $1,000. This was actually exceeded by $400, so that the

enterprise started with a debt of that amount. The sum of $12.50 was then set aside monthly as a fund for betterment of the property, payment of interest, and reduction of debt. This has enabled substantial

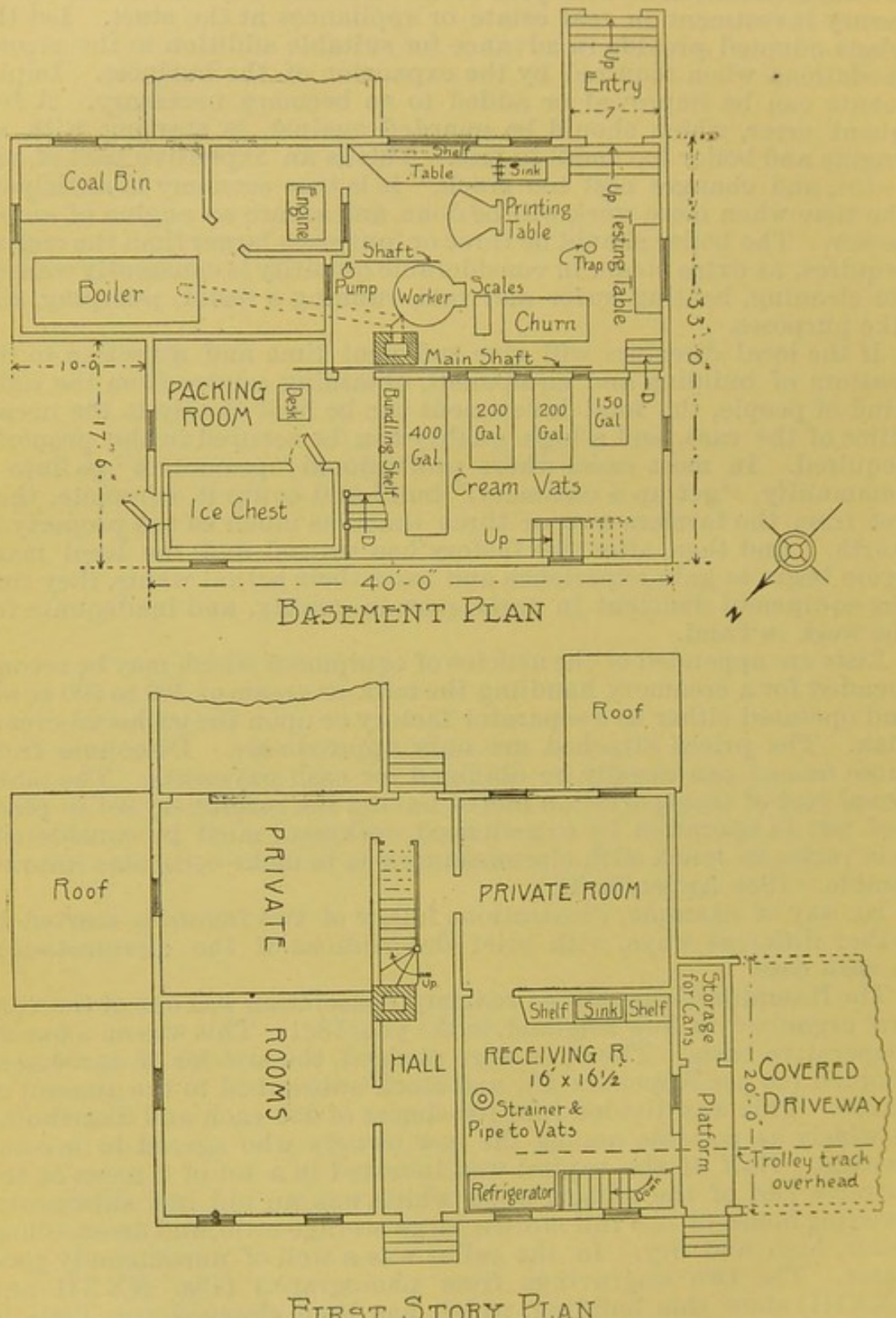
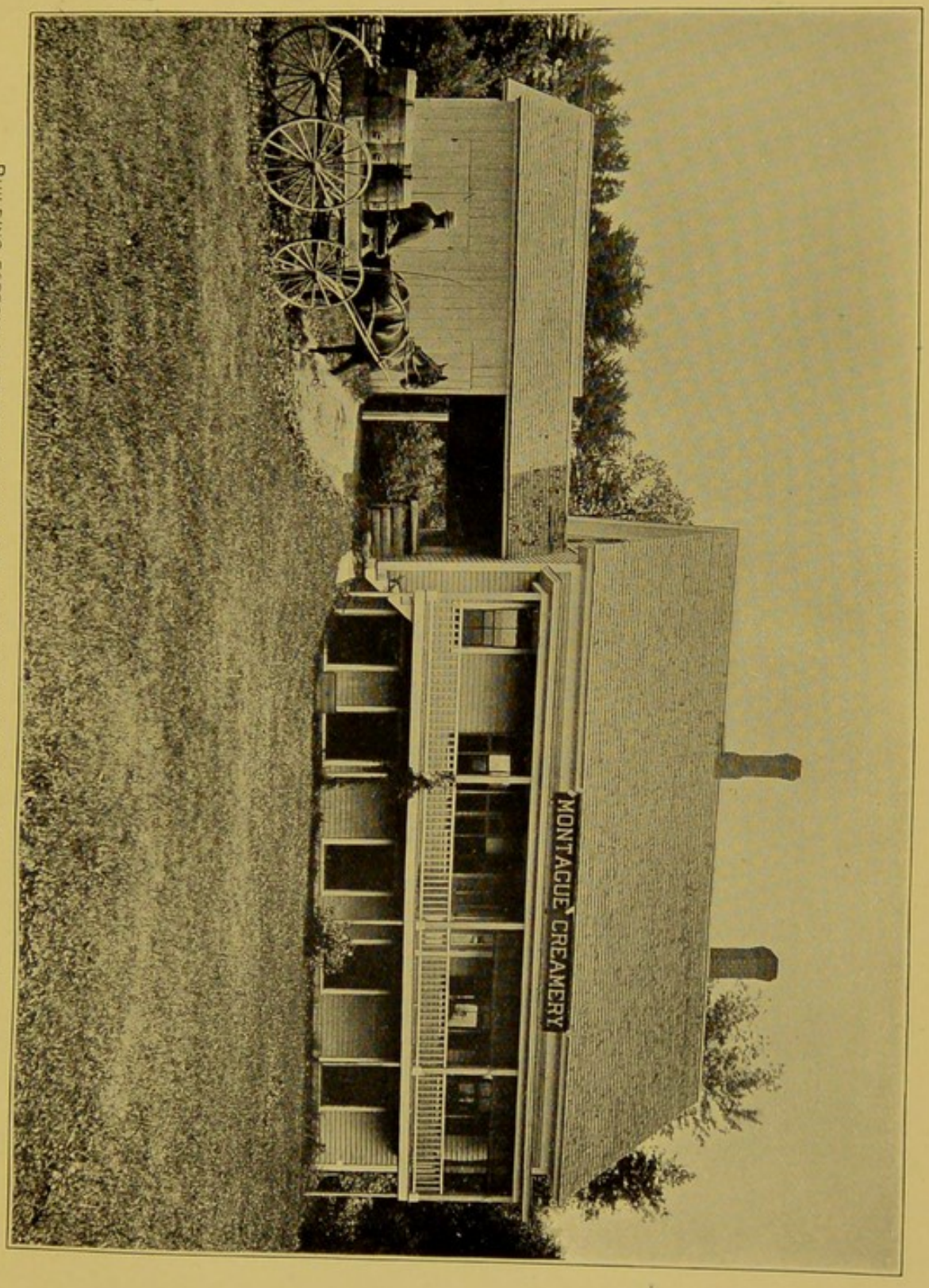


FIG. 2.—First story and basement of the building shown in Plates XXXII and XXXIII.

improvements to be made; an ice house was built and cold storage arranged in it, as well as a cold room in the house basement; this



Building especially designed for a Creamery, with Dwelling Rooms above; Front View.

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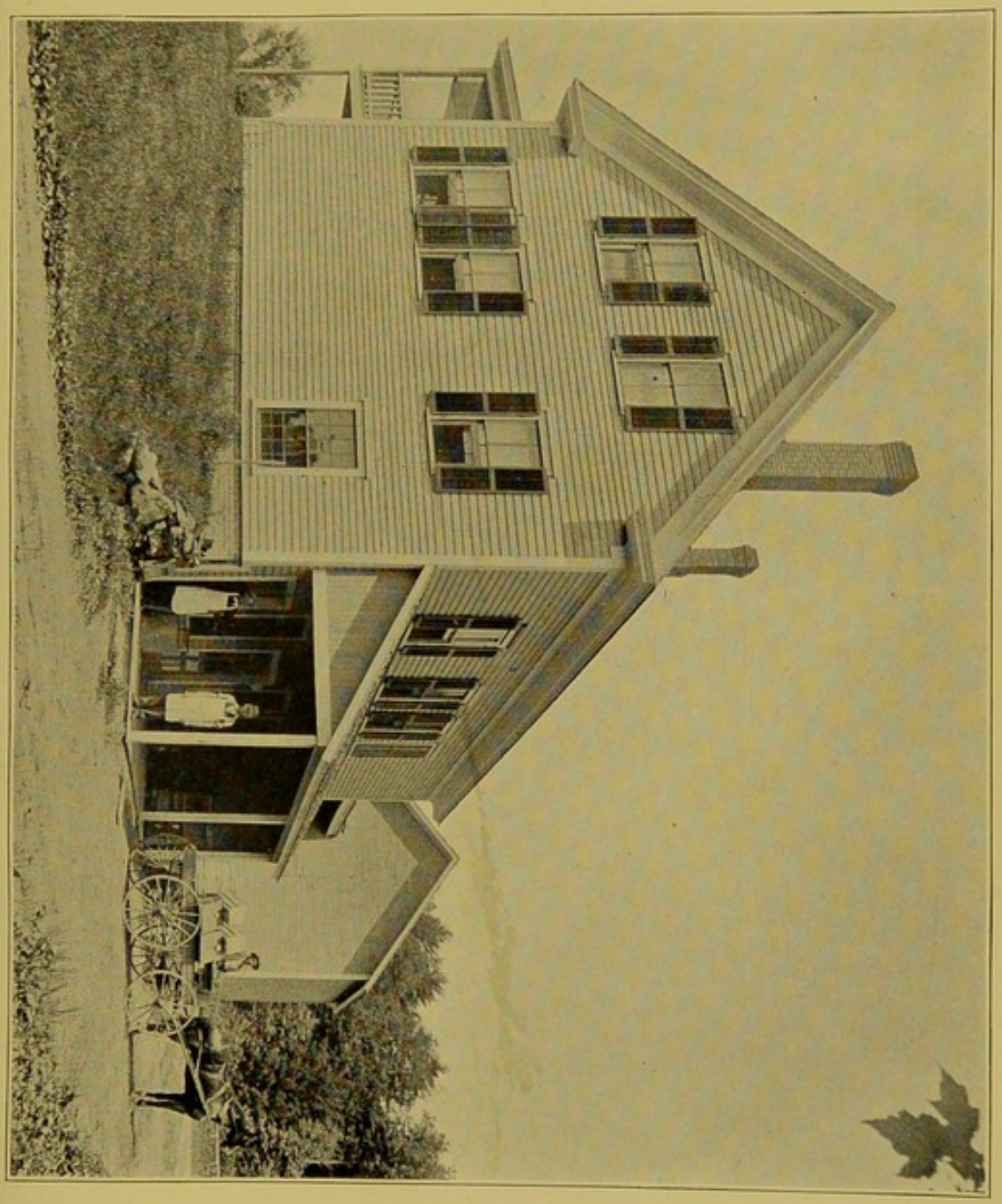
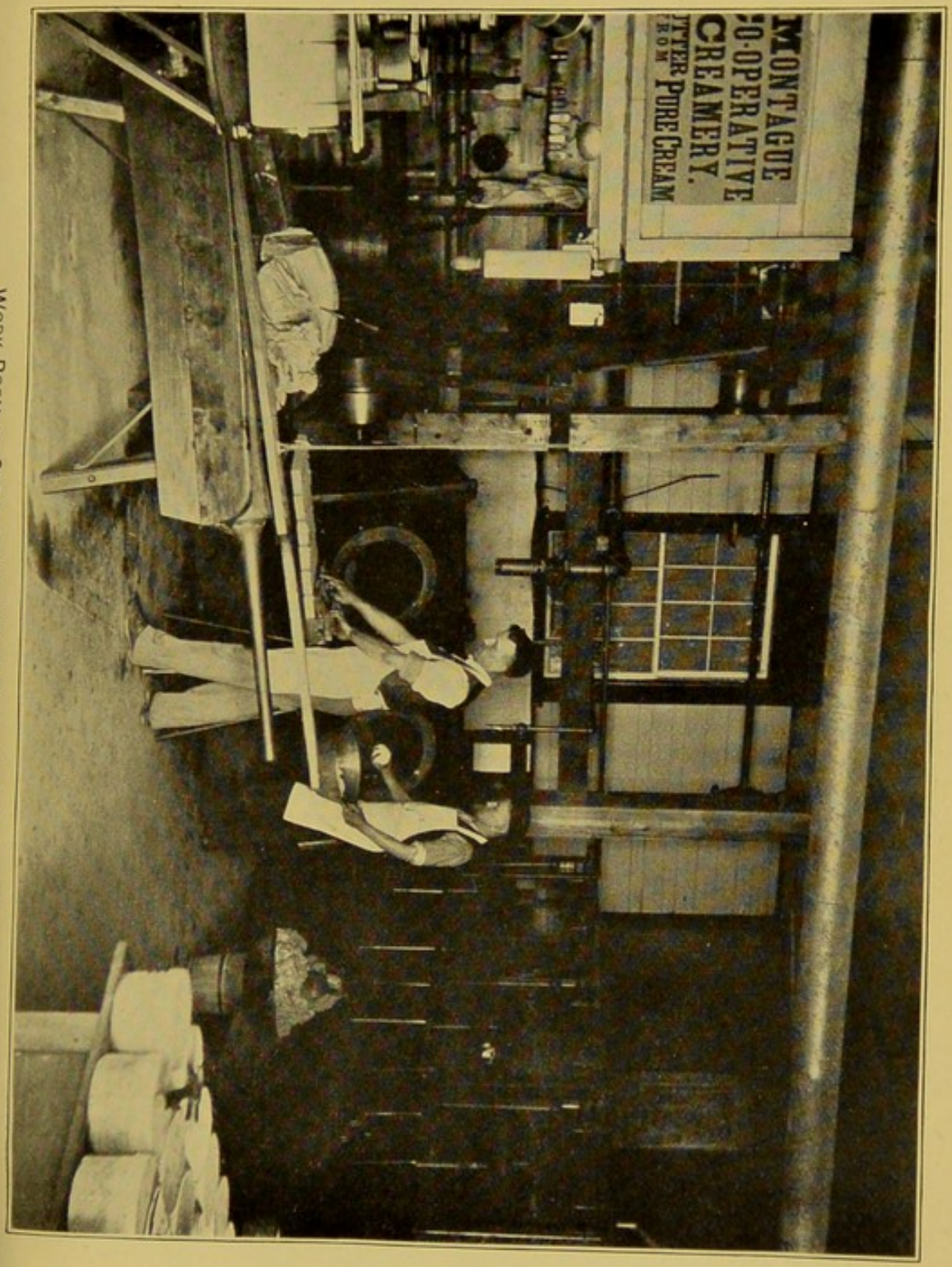
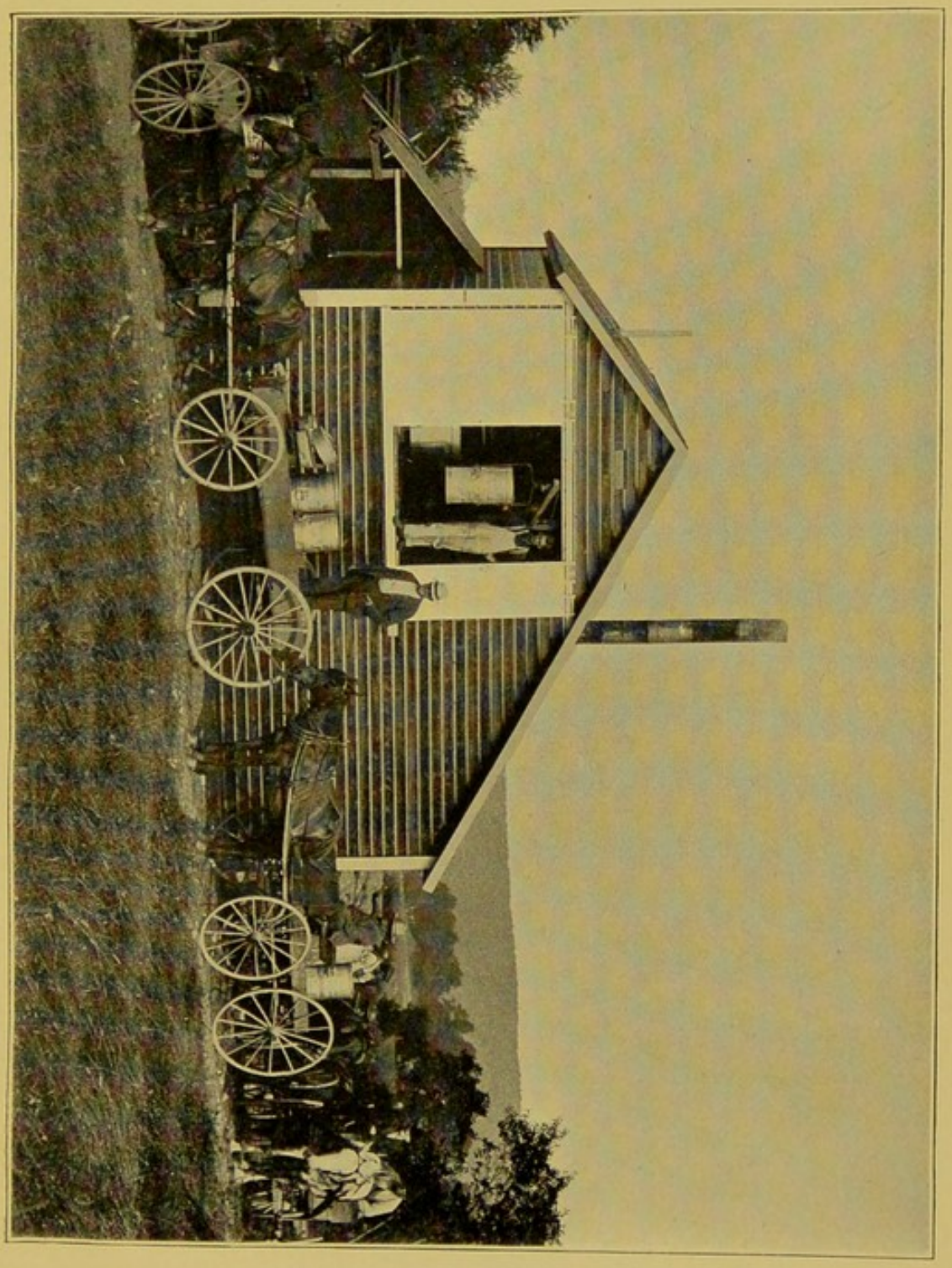


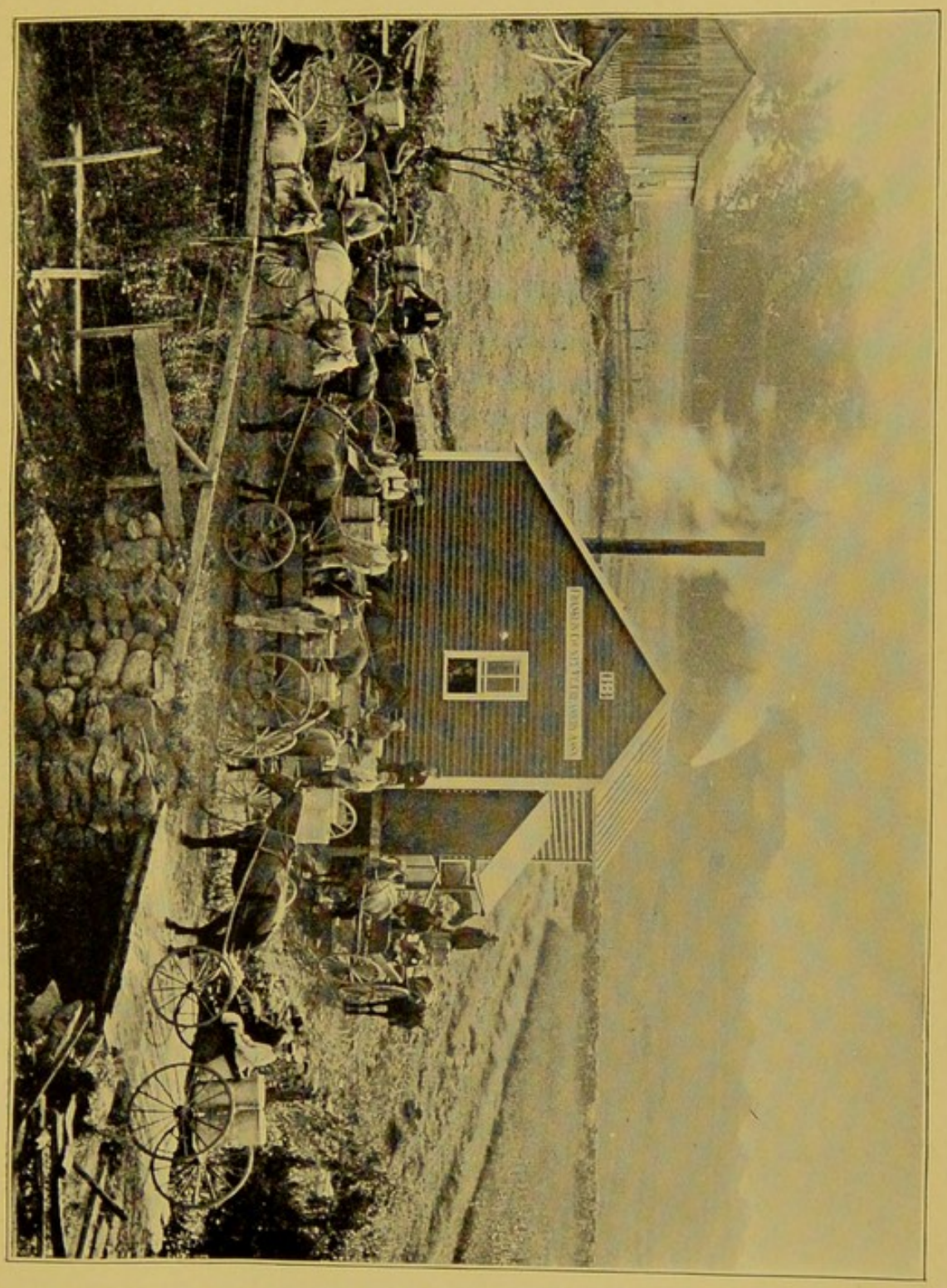
Rear and End View of Building in Plate XXXIV.



Work Room of Creamery shown in Plates XXXIV and XXXV.



Separating Station, Receiving Milk.



Separating Station and Patrons

cost $500. In 1893 the boiler room was enlarged and a new boiler put in, at an expense of $600. A year later, a covered driveway was built at the door of the receiving room and a track, hoist, and carrier arranged for unloading cream from gatherers' wagons; cost, $200. These features are all plainly shown in the illustrations. In 1895, a new engine was bought and the work generally refitted and rearranged, the whole costing about $700. New vats, churn, etc., have been bought as needed. In all, about $2,100 has been thus expended during fifteen years. Finally, in order to get entirely out of debt, new stock has been issued to the amount of $500.

This creamery fronts northwest. The two views and plans of ground floor and basement (Fig. 2, p. 308) show the general arrangement. The cream, upon arrival, is unloaded under the covered shed at west corner of the building, carried into receiving room at that corner of main floor, and is thence conducted to the vats directly below. These are upon a raised platform, so when the cream is ready it can be run directly to the churn. The extension of the cellar made for the boiler room and coal room is seen at northeast end of the building. At this end, also, ice is put in for the cooling room, and the shipping cases of butter are passed out of a window, to be carried away. The ice house is in the yard a short distance east from main building. Good drainage was early secured to the rear or eastward. The arrangements, as a whole, are not quite as complete and convenient as if twice as large a capital had been raised and a building specially erected, but the business has been pretty well provided for and the necessary changes and improvements have been easily accomplished as needed. Whereas this factory made at the start less than 200 pounds of butter a day, its operations have grown so that 700 to 800 pounds are now turned out daily. Three teams gather the cream, each making a daily trip of 18 to 27 miles in length. The building, besides accommodating the business (although the basement is now rather crowded), affords ample dwelling rooms for the family of the butter maker. The capital stock is now $2,500, upon which a 6 per cent annual dividend is paid, and the property is valued at $4,000.

The Montague Cooperative Creamery Association was organized in 1890, with a capital of $2,500, divided into shares of $10 each, placed mainly among the prospective patrons. Land was purchased, plans carefully prepared, and the creamery erected under contract with a competent local architect and builder, as shown by Pls. XXXIV-XXXVI. When completed and equipped and operations began in October, 1891, the entire plant had cost $3,750. Of the debt $1,000 is carried for the present as a mortgage on the property, and the rest has been paid from current receipts. The illustrations give a good idea of the exterior and interior arrangements of the creamery and outbuildings. The accommodations for receiving and storing the cream, making and removing the butter, are ample and convenient. Ice can be easily moved from ice house to cooling room. There is a good tenement for the butter maker, and the front balcony shades the most exposed side of the work room. An abundance of soft water is brought from a fine spring but a short distance in rear of the creamery and owned by the company. All waste is drained to a quick-running stream near by, and the buttermilk tank is located at a sufficient distance, near the stream, and kept in good order.

This creamery has storage capacity for 1,000 gallons of cream and makes about 2,500 pounds of butter a week, working only six days.

All the patrons have creamers and the factory was operated on the "space" system for four years. Then, by vote of the stockholders and patrons, a change was made to payment upon the basis of butter fat, determined by the Babcock test. Much satisfaction and improvement followed this change. Two teams gather the cream. One has a route fully 30 miles in length, but makes the trip only four times a week. It has been found that the cost of bringing the cream from the producing farms to the factory is from 1¼ to 1½ cents for every pound of butter made. The cream of every patron is sampled daily and composite samples made, these being tested two or three times a month. This creamery is located in a region from which milk is shipped to the Boston market, but the quality of butter produced is so good and its sales are so well managed, that the patrons receive as much for the cream of their milk as their milk-shipping neighbors do for their whole milk.

Two other examples, not illustrated, may be briefly mentioned. One creamery was started on a capital of $1,500 in a rented building similar to that purchased by the Hampton Association (above), and cheaply fitted up for the purpose. This made 200 pounds of butter a day, growing to 400, and got along very well for some years, when the capital stock was increased and a good creamery was built. The other raised $5,000 at the beginning, bought an exceptionally good location, and erected a substantial two-story brick building with large hillside half basement and an ice house and cold-storage room attached. Later a house was built for the butter maker, costing $1,800, and other improvements made, the capital stock being increased to $7,000; upon this 6 per cent interest is paid, and charged, as usual, to the general running expenses.

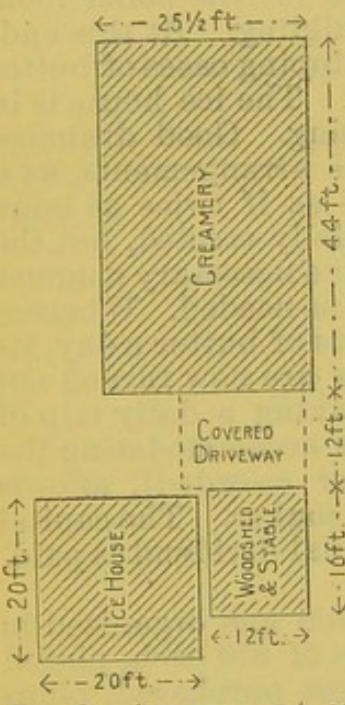
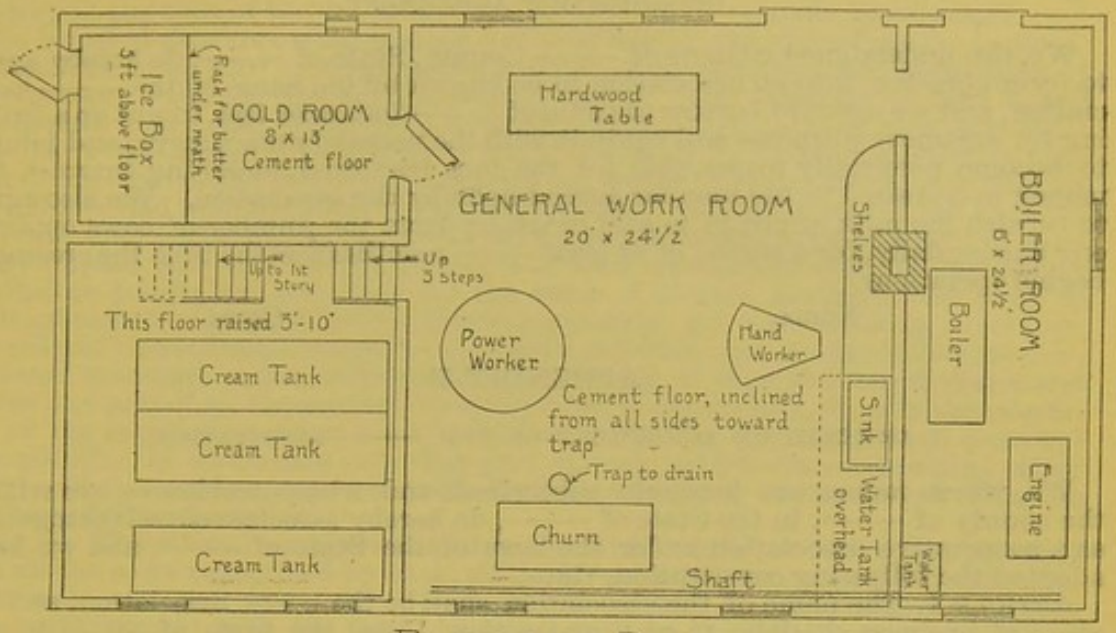


FIG. 3.—Arrangement of creamery and outbuildings shown in plates XXXIV and XXXV.

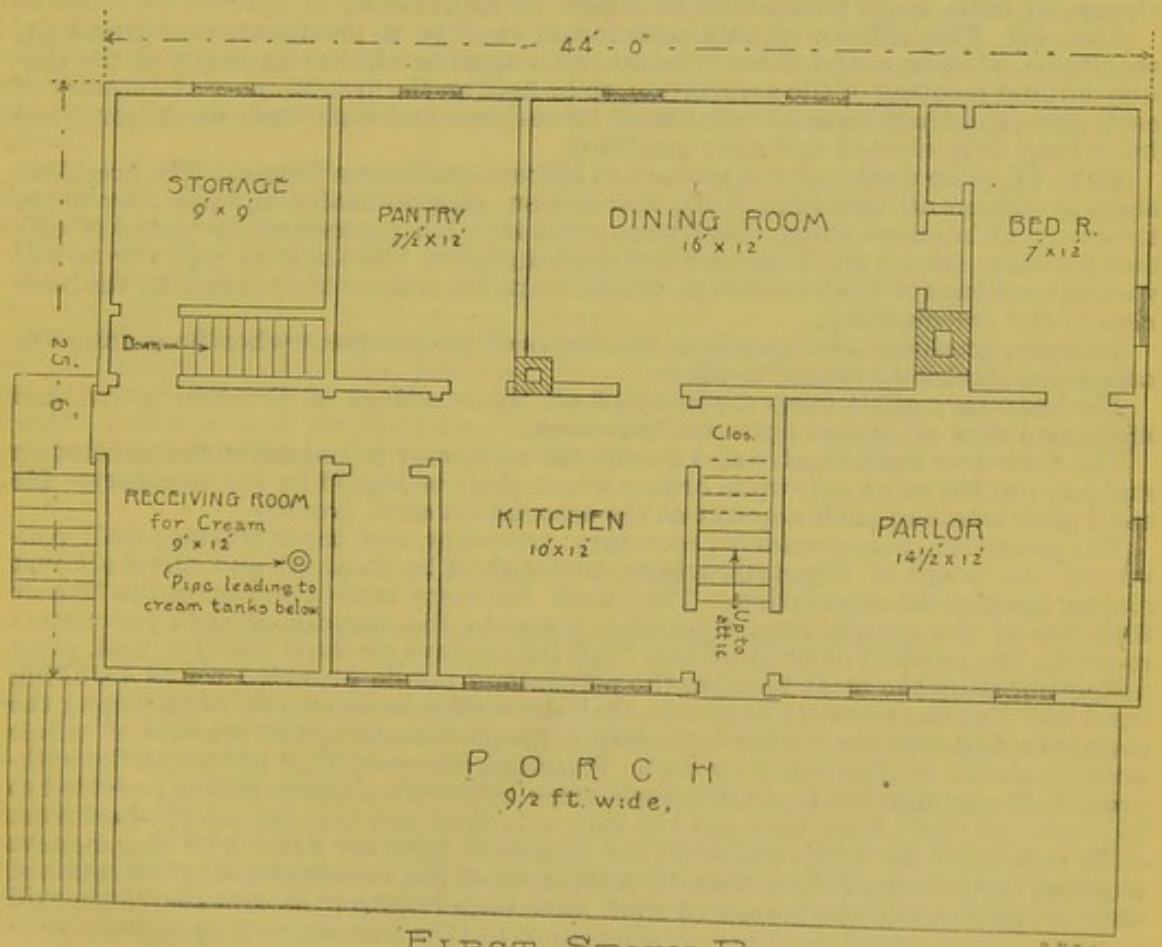
The foregoing examples are all creameries operated upon the cream-gathering plan. For the separator system the buildings and equipment must be substantially the same. A little additional room would be wanted for the centrifugal machine or cream separator, with its attachments, and this would be an additional expense. As a partial offset, the carrying cans and outfits for gatherers would not be required. The differences between such a factory and those already described are not sufficient to call for further plans and illustrations.

A separating station is a comparatively simple establishment. To such the milk is hauled by the farmers producing it, who usually wait until their contributions are run through the separator, and then haul home their shares of the skim milk. The building requires weigh cans, scales, receiving vat, tempering vat, separator, cream vat or cans, and provisions for the necessary power to operate the separator. The cream is usually sent from such a station, as soon as possible, by the most convenient conveyance, to the central factory or creamery, where the butter is made, and to which the station is tributary. A station of this kind is shown by Pls. XXXVII and XXXVIII. These views well illustrate the chief objection to this system, the crowding of

patrons about the station or factory and the almost daily loss of time by men and teams while awaiting their turn to deliver milk, get it



BASEMENT PLAN



FIRST STORY PLAN

FIG. 4.—First story and basement of the creamery shown in plates XXXIV and XXXV.

separated, and secure their share of skim milk to take home. This is a very serious matter.

APPENDIX A.

ORGANIZATION AGREEMENT.

We, the undersigned citizens of ——— County, State of ———, do hereby agree to form ourselves into an association to be known by the name of the ——— association, and we agree to borrow the sum of ——— dollars or less, to put up a building for creamery purposes and equip it with the necessary machinery, and jointly to become personally responsible for the sum borrowed, including interest, the money to be raised in the manner agreed upon by the association. We also agree to furnish the milk or cream to said creamery from the number of cows opposite our names, daily, for a period of at least ——— months from the time the creamery begins operations.

Name.	Cows.

APPENDIX B.

ARTICLES OF AGREEMENT OF THE ——— ASSOCIATION.

We, whose names are hereunto subscribed, and whose residences are within the county of ———, in the State of ———, do hereby associate ourselves together as a cooperative association under the laws of the State of ———, and we have adopted the following constitution, viz:

ARTICLE I. The name of the association shall be the ——— association, and its place of business shall be at or near (section —, in) the town of ——— in said ——— county.

ART. II. The object of this association shall be the manufacture of butter or cheese, or both, from whole milk or cream, at actual cost.

ART. III. The officers of this association shall be a president, vice-president, secretary, treasurer, and three trustees, who shall be elected annually at the regular annual meeting of the association, to be held on the first Monday of January of each year, and their term of office shall be one year and until their successors shall have been duly elected and have qualified.

ART. IV. The duties of the respective officers shall be as follows: The president shall preside at all meetings of the association, sign all drafts, and pay over to the treasurer all moneys which shall have come into his possession by virtue of official position, taking the treasurer's receipt therefor. He shall have power to call special meetings of the association whenever in his judgment required by the business of the association.

The vice-president shall perform the duties of the president when he is absent or otherwise unable to attend to them.

The secretary shall keep a record of all the meetings of the association, and make and sign all orders upon the treasurer.

The treasurer shall receive and receipt for all money belonging to the association and pay out the same only upon orders which shall be signed by the secretary. He shall give bonds in such amount as the association shall provide.

The president, vice-president, secretary, treasurer, and three trustees shall constitute the board of directors, whose duties shall be to audit and allow all just claims against the association. They shall for every month and upon the 19th or 20th day of the month next following compute the amount of milk (or cream) receipts, the amount of product sold, and the moneys received therefor, and, after deducting from the total receipts the percentage herein provided for as a sinking fund and also the running expenses, shall divide the remaining receipts among the members and patrons of the association proportionally to the amount of whole milk or butter fat furnished by each: *Provided, however*, That in case of the withdrawal of any member from this association before the moneys herein provided to be borrowed shall have been paid in full, principal and interest, all product from milk furnished by such withdrawing members then on hand, and any moneys received from such product then in possession of the association shall be retained until all said moneys so borrowed shall have been fully repaid, and thereafter said moneys or any remainder thereof, after applying the just share of such withdrawing members therefrom to the repayment of any balance of such indebtedness not paid from the sinking fund, shall be paid over to him or his assigns.

The board of directors shall cause the secretary to make a report in writing to the annual meeting of the association, setting forth in detail the gross amount of milk (or cream) receipts, the net amount of receipts from product sold, and all other receipts, the amount paid out for running expenses, the sums, if any, paid

out for milk (or cream), and all other matters pertaining to the business of the association. A like statement containing the gross amount of milk (or cream) receipts, the net receipts from product sold, and all running expenses of the creamery, shall be made each month and posted conspicuously in the creamery building at the time of the division of the prior month's receipts, as aforesaid.

The board of directors shall borrow a sum of money not exceeding ——— thousand dollars, to be used by them in the erection and completion and furnishing of the creamery building, and for no other purpose. The members of said board may borrow said money on their individual responsibility, and in case they shall do so, then the sinking fund herein provided for shall by them be applied in payment of such borrowed moneys as the same fall due in the same manner as though said moneys had been borrowed by the association. Said members of the board in such case shall be held to be the creditors of the association to the amount of such money unpaid, and the several members of said association shall be personally responsible, jointly and severally, for the same: *Provided, however*, That prior to any legal assertion of such individual responsibility, the entire sinking fund then accrued and on hand shall be applied upon such indebtedness: *And provided further*, That said members so borrowing said moneys may, if they so elect, demand and receive any part of all the moneys received from products sold, then in the possession of the association, upon such indebtedness before enforcing such personal responsibility, in which case only that part of such indebtedness remaining, after applying thereon all sums so received, shall be recovered or demanded from the members of the association.

ART. V. The several members shall furnish all the milk (or the cream thereof) from all the cows subscribed by each, all milk (or cream) to be sound, fresh, unadulterated, pure, and unskimmed, and patrons of the association not members may, by agreement with the board of trustees, furnish such amounts of milk or cream as may be so agreed upon. The association shall receive all such milk and cream so furnished, manufacture the same into butter, cheese, or both, and sell and receive all money from the product; and from the moneys so received deduct such a percentage thereof, or such a number of cents per one hundred pounds of milk, or cents per pound of butter sold, as shall have been agreed upon by the association in the by-laws or otherwise, and also deduct the running expenses of the creamery, the remainder thereof to be distributed as provided in Article IV hereof.

ART. VI. Each member shall be entitled to one vote only at any meeting of the association. New members may be admitted as provided in the by-laws. Members shall be permitted to withdraw only as provided in the by-laws.

ART. VII. The first officers and board of trustees shall be as follows: ——— ———, president; ——— ———, vice-president; ——— ———, secretary; ——— ———, treasurer; ——— ———, ——— ———, ——— ———, trustees.

ART. VIII. The constitution may be amended at any annual meeting, or at any special meeting called for that purpose: *Provided*, That two-thirds of all members present vote in favor of such change: *And provided further*, That at least one month's notice of such proposed amendment shall have been given in such manner as may be provided in the by-laws, or otherwise by the association.

Signatures to this constitution:

* * * * * * *

BY-LAWS OF THE ——— ASSOCIATION.

(Applicable to a separator creamery not operated on Sunday.)

ARTICLE I. The treasurer shall give bonds in the sum of ——— dollars, the bond to be approved by the board of directors.

ART. II. Five cents on each one hundred pounds of milk received at the creamery shall be reserved to form a sinking fund.

ART. III. No milk shall be received or business of any kind transacted at the creamery on Sundays.

ART. IV. During the interval between the twentieth day of May and the twentieth day of September of each season all milk shall be delivered at the creamery as early at least as nine o'clock a. m.; during the remaining portion of the season as early as ten o'clock a. m.

ART. V. All milk delivered shall be sweet and in good condition; if any be found otherwise, the operator may condemn and refuse to receive the same, and in such case he shall as soon as possible notify the president. The operator shall preserve samples of every delivery of each patron's milk, testing the same at proper intervals on the composite sample plan for tests for butter fat in milk.

ART. VI. Any member or patron of the association found skimming, watering, or in any manner adulterating his milk offered at the creamery shall forfeit to the association as follows: For the first offense, ten dollars; for the second offense, twenty-five dollars; for the third offense, he or she shall forfeit all interest in the association and also all claims for milk theretofore delivered to the association. But no such forfeiture shall be adjudged without first offering to the member or patron charged with so having skimmed, watered, or adulterated his milk full opportunity to defend himself from such charge. Any member sending to the creamery any bloody or unhealthy milk, or any milk from any cow within four days after calving, shall, if convicted of having so done knowingly, forfeit as described above in this section. The directors shall act as a board of judges in such cases.

ART. VII. Members and patrons furnishing whole milk may take from the separator or the tank at the creamery four-fifths of the quantity of milk (in pounds or quantity) delivered at the creamery on that day. Any member taking therefrom more than such amount shall forfeit to the association the sum of five dollars for each such taking.

ART. VIII. Withdrawals from the association shall be allowed only as follows: The member desiring to withdraw shall give at least one month's notice of his application therefor. Such application shall only be allowed on a vote of two-thirds of all members present and voting at any meeting for hearing at which such application shall have been noticed: *Provided, however*, That any member living more than three miles by the nearest road from the creamery building may make application to the board of directors, who in their discretion may grant permission to such member to withdraw from the association.

ART. IX. Any member refusing to deliver at the creamery the milk (or cream) agreed to be there delivered shall, without reason satisfactory therefor to the association, forfeit all interest in the product on hand.

ART. X. Notice of any proposed amendment to the constitution or by-laws shall be in writing or printing and shall be kept posted prominently in the creamery building and also on the walls of the delivery department for the reception of milk.

APPENDIX C.

BY-LAWS OF THE ——— COOPERATIVE CREAMERY ASSOCIATION.

(Originally framed and adopted in April, 1881.)

(Suited to a purely cooperative factory on the cream-gathering plan.)

ARTICLE I. This corporation shall be called the ——— Cooperative Creamery Association and shall be established in the town of ———, the location to be fixed by its directors: *Provided*, That the original location be approved by a stockholders' meeting. The object of the association shall be to receive milk or cream from its members and other patrons, or to purchase milk and its products, and to manufacture and sell the products of milk, purchasing, leasing, holding, and using real and personal estate necessary for the proper transaction of the business of the corporation.

ART. II. The stock capital of the corporation shall be two thousand dollars ($2,000), divided into eighty shares of twenty-five dollars each. The members of the association shall be owners of its capital stock. Each stockholder shall be entitled to one vote in the meetings of the association, irrespective of the number of shares owned, and may vote in person or by proxy, subject to the provisions of the law. A majority of the stockholders of record present at any meeting of which regular notice has been given shall constitute a quorum for the transaction of business.

ART. III. The annual meeting of the association shall be held on the last Wednesday in January, in each year, at one o'clock in the afternoon, at such place as the directors may appoint, after at least seven days' notice by advertisement or by mail to each stockholder. Special meetings shall be ordered by the president, and shall be held after notice as provided for the annual meeting, whenever voted by a majority of the directors, or in writing requested by one-third of the stockholders.

ART. IV. The members present at the annual meeting shall choose by ballot a clerk, a treasurer, and not less than three directors, and the persons so chosen shall hold office till the next annual meeting or till others are chosen and qualified in their places. The clerk and treasurer may be chosen directors. A majority of the directors in office shall constitute a quorum of the board. Any vacancies in these positions may be filled by the board of directors, such appointment to hold

until the next meeting of the corporation, when the stockholders shall fill the said vacancy.

ART. V. The directors shall choose one of their number as president, and the president shall be the presiding officer of the board of directors and at meetings of the stockholders. The directors shall appoint all other officers, agents, and employees of the association needed in its business, and shall fix the compensation for all service performed for the association. The directors shall procure and adopt a corporate seal and shall determine the form of certificate and transfer of stock.

ART. VI. Certificates of stock, duly numbered and signed by the president and treasurer, shall be issued to stockholders, and upon transfer and surrender of any former certificates new ones shall be issued therefor, if desired, and all transfers and issues shall be duly recorded in the proper books of the corporation.

ART. VII. The treasurer shall be the transfer clerk for stock and, subject to the control of the directors, the custodian of the property of the association, and its financial agent and accountant. He shall give bonds, approved by the directors, in a sum of not less than two thousand five hundred dollars ($2,500) for the faithful performance of the duties of his office.

ART. VIII. The clerk, who shall be duly sworn, shall keep a true record of the proceedings in all stockholders' meetings and of the stated meetings of the directors, and shall give all notices. In the absence of the clerk, a clerk pro tempore may be chosen and qualified by the directors.

ART. IX. This corporation shall be cooperative. Milk or cream may be accepted or purchased from any member and also from any producer not a stockholder upon the same terms and regulations as may be prescribed for stockholders, and after deducting all actual expenses of the business from the gross receipts, the remaining income, profits, and earnings shall be divided among the members and other contributing patrons, pro rata, according to the quantity of milk or cream furnished by each, or according to the quantity of actual butter fat furnished, as determined by proper test, under regulations therefor made by the directors; and such division of profits shall be made and paid monthly or bimonthly, as the directors may decide. The actual expenses shall include the disbursements properly made in prosecuting the business, such sums as may be legally required for a contingent fund, and an amount equal to six per centum per annum upon the par value of the capital stock; the last-mentioned interest to be paid annually to the stockholders of record at noon of the day next before each date fixed for such payment: *Provided*, That the directors may deduct the sum necessary for this interest, at their discretion, from the gross receipts of any portion of the business year.

ART. X. The directors shall cause to be kept a correct account of all purchases, all expenses, and all receipts from sales or otherwise, also of all milk or cream furnished by stockholders and other patrons. The board shall prescribe the rules and regulations governing the collection and delivery of milk and cream, may cause the quality of such to be tested as often as may be expedient, may in person or by authorized agent inspect the premises and methods of any person from whom milk or cream is received, and may reject or refuse to collect or receive any milk or cream that is unsatisfactory or not furnished according to the prescribed regulations. The directors shall establish prices and shall have full power over the business of the association, and shall make a full statement of the business and of the doings of the board at each annual meeting, or whenever called for by vote of the stockholders. Meetings of the board of directors, of which records shall be kept, shall be held whenever called by the president, or requested in writing by any two directors.

ART. XI. These by-laws may be altered or amended by a two-thirds vote of the members present at any annual meeting, or at any special meeting of stockholders, provided the fact of the proposed change be stated in the notice.

APPENDIX D.

CREAMERY EQUIPMENT.

The following is a list of the principal apparatus needed to equip a butter factory for 500 cows or less, with the usual prices at place of shipment, subject to discount for cash payment:

1.	One 15-horsepower boiler, with fixtures	$200.00
2.	One 10-horsepower engine, with fixtures	175.00
3.	Shafting, pulleys, hangers, belting, etc., about	100.00
4.	One set of hoisting-crane fixtures and track	20.00
5.	Twenty carrying cans, 20 gallons each	120.00
6.	One 600-pound platform scale, compound beam	25.00
7.	One 70-gallon weighing can	10.00
8.	One 500-gallon milk-receiving vat	35.00
9.	One milk-tempering vat, copper	15.00
10.	One cream separator, 2,600 to 3,000 pounds per hour capacity	475.00
11.	One milk or cream conductor head and pipe	3.00
12.	One 300-gallon twin cream vat, steam and water and ice boxes	70.00
13.	One 400-gallon factory churn, center belt	45.00
14.	Cream and buttermilk strainers	4.00
15.	Four tubs for granular butter, 125 pounds each	8.00
16.	One power butter worker	50.00
17.	One printing table and two butter printers	20.00
18.	Butter ladles, large and small, packers, and butter bowls	3.00
19.	One counter scale (to weigh half ounce)	12.00
20.	Glassware, floating thermometers, graduates, etc	3.00
21.	One 30-bottle Babcock tester, and fittings complete	25.00
22.	One plunger pump for water	20.00
23.	One water tank, 600 gallons	25.00
24.	One hot-water tank, 300 gallons	20.00
25.	One milk and buttermilk pump	15.00
26.	One skim milk or buttermilk tank, 300 gallons	17.00
27.	Two wash-sinks and connections	13.00
28.	Water pails, dippers, and other tinware	5.00
29.	Hose, mops, aprons, brooms, brushes, stencils, etc	17.00
30.	Fifty print-butter carriers, 24 to 84 pounds each	150.00
	Total	1,700.00

NOTE.—For a cream-gathering factory, omit from this list Nos. 8, 9, and 10, amounting to $525, leaving cost of outfit less than $1,200. For a separator creamery, omit No. 5; amount, $120. Nonreturnable packages are so largely used that the last item ($150) may generally be omitted. For cheese-making apparatus an additional outlay of about $250 would be necessary.

MISCELLANEOUS REPORTS OF INSPECTORS AND CORRESPONDENTS.

ERGOTISM IN KANSAS.

In April, 1895, a committee of cattle feeders requested Mr. Albert Dean, live stock agent in charge at Kansas City, Mo., to institute an investigation into the nature of a disease then existing among the cattle of Thomas Steele, at Selma, Kans. Stock Examiner W. N. D. Bird, to whom was assigned the task, reports as follows:

I proceeded at once to the farm of Mr. Steele, which is a mile and a half northeast of Selma, Anderson County, Kans. After a thorough investigation of the cattle and their feed I found the disease to be ergotism, caused by the cattle eating wild rye on which was ergot in considerable quantities. Hay containing this wild rye had been fed to the cattle prior to the outbreak of the disease.

History of cattle and disease.—Mr. Steele purchased about the middle of January last in the Kansas City (Mo.) stock yards 100 head of 3-year-old native dehorned steers and shipped them to his farm. He could not give me their former history. After their arrival on the farm he added one more steer of same age, raised in the neighborhood. Mr. Steele placed these 101 steers in his feed lots, consisting of one sheltered lot of about an acre, fenced with a high stone wall on the east, north, and west, and board and wire on the south; a ravine ran diagonally through the lot. Adjoining this lot on the east was a pasture of 35 acres of which the cattle had the run. In the eastern part of this pasture was a creek of water from which the cattle obtained their drink. In very cold weather the ice had to be cut to enable the cattle to drink. These two lots were more or less stony and rough. Mr. Steele commenced by feeding the steers shock corn in the morning. The corn on this was fairly good and probably would yield 20 to 25 bushels to the acre. The fodder was quite bright and the corn free from any smut. In the afternoon he fed all hay, consisting of timothy and clover, with some redtop and some prairie slough hay containing a great proportion of wild rye. This way of feeding continued until his shock corn was exhausted. He then kept the racks full of hay and fed some ear corn. Mr. Steele says he only had about an acre of this slough hay, amounting to about 3 or 4 tons, and had fed out the greater part of it previous to the outbreak of the disease. About the last of January it became very cold, the thermometer dropping to 12° or 15° below zero.

After this storm Mr. Steele noticed 3 steers so lame they would not go to water without driving. He turned them out on his meadow and they fed at the hay stacks of timothy and clover.

He thought at first that the steers had frozen their feet, and he continued in that opinion until a number more became lame and were turned out of the lots. Up to April 1 Mr. Steele had turned out 25 head, and 1 steer had lost both feet above the os coronæ and was killed. Several others had lost one foot at the joint between the os pedis and os coronæ and several had lost one toe.

Upon arrival at Mr. Steele's farm I noticed 24 head of steers feeding at the hay stacks or grazing on the meadow. On moving them I noticed all were more or less lame, in the various stages of the disease. One steer had lost all of one foot below the os coronæ, 2 or 3 had lost one toe, and would probably lose the other; 8 or 10 more will probably lose one toe. I pulled the toe of one steer off, which was hanging only by a little skin. All the steers were affected only in one foot; the other feet were apparently well. The lame steers with only two exceptions were all doing well and appeared to be thriving. I put one steer in the branding chute, and made a thorough examination of the mucous membrane of the mouth, nose, and rectum, and found them in perfectly healthy condition.

All the steers had a good appetite during the progress of the disease, their bowels were perfectly healthy, and in none of them were any indications of fever present. I found also that some of the sick steers and a few of those in the main lot, at least 10 or 12, had lost the bushes of their tails.

On investigation I learned that the slough hay which was fed before the outbreak of the disease and contained a large proportion of wild rye had all been fed out except one stack, which consisted of the rakings of timothy, clover, and prairie hay. In this I found considerable wild rye, and on examination of this wild rye I found quite a proportion of it contained well-developed ergot. I found no ergot in the timothy, clover, or redtop. After showing Mr. Steele the heads of wild rye with the ergot on them he said the hay which he had fed the cattle he remembered to have contained many heads of rye black with ergot. After making this thorough examination of the hay I believed that there was sufficient ergot on the wild rye, with the climatic conditions, to cause the disease.

Mr. Steele said the first indication of the cattle losing their toes had been a slight crack in the horn of the heel; this would gradually lengthen out, showing little or no pus, until it gradually would fall off.

The foot of the steer from which I pulled the toe showed no considerable inflamed condition, with a very little pus.

Mr. Steele said that in no stage of the disease were there any indications of soreness of the mouth or derangement of the bowels.

Mr. Steele did nothing in a medicinal way to cure them. He did not change the feed or their locality until the hay with wild rye and corn fodder had been fed out.

On an adjoining farm to Mr. Steele's lived Mr. Alfred Pillsbury, who had one small steer become lame, but he changed the feed, and had no new cases.

TEXAS FEVER IN KANSAS.

In 1894 an outbreak of Texas fever occurred among a few herds of cattle in Kansas. A careful study of the environments of the affected animals at once gave rise to a suspicion that the source of the disease might be traced to southern California through a few shipments of cattle that had been made to Kansas from that section. To verify this suspicion became a subject of exceptional interest from the fact that the cattle tick, the direct medium of the contagion, had not heretofore been officially recognized as existing in California; in other words, the Texas fever line had not been extended so as to include any part of that State.

An investigation resulted in the discovery of abundant cattle ticks in the lower altitudes of the southern California coast, and traced to this section, beyond reasonable doubt, the origin of the outbreak of Texas fever in Kansas. The recommendation to extend the Texas fever line so as to include all of that portion of California south of an irregular line extending from Lake Tahoe to San Francisco has since been carried into effect.

The following report of Live Stock Agent W. E. Hill gives a detailed history of the investigations in California:

Hon. J. Sterling Morton,
Washington, D. C.

Sir: In compliance with your instructions, dated November 17, 1894, to visit such parts of southern California as might be necessary to investigate the prevalence of Texas fever among cattle, I beg leave to report that on arriving at Los Angeles, Cal., I made the acquaintance of Mr. B. Weller, live stock agent of the Santa Fe Railroad and a live stock commission merchant; Mr. Simon Maier and Mr. Lewis Sentous, butchers of Central and Orleans markets; W. H. Newhalls, and Messrs. Vail & Gates, cattlemen, and many other gentlemen interested in the cattle business of southern California.

They informed me that, so far as they were advised, there had been no disease among the cattle of that section during the season of 1894, but that in previous years, at various times and in separate localities, sometimes in one place and then

in another, large numbers of cattle had been taken sick and the loss by death had generally been heavy. They also informed me that there was plenty of ticks on the cattle during the warm weather of summer and fall, and that I could obtain all I desired by visiting the various cattle ranches along the coast. Mr. Weller, who at one time was in the cattle business in Texas, said he believed them to be the same kind of ticks he had seen in former years on cattle in Texas.

After gaining what information I could in Los Angeles, I started down the coast for the purpose of visiting some of the ranches and obtaining ticks from the cattle. The first ranch visited was the San Joaquin, below Santa Anna, managed by Mr. George Irvine. On account of a severe rainstorm that prevailed at the time and the cattle being from 5 to 10 miles off in the Coast Range hills, I was unable to obtain ticks at this ranch. Mr. Irvine said if I had been present about November 1, when they removed the ticks from the cattle with a currycomb and saturated their bodies with crude petroleum, I could have obtained a bushel of ticks.

Mr. Irvine also informed me that of late years they had brought no cattle (except bulls) from other ranches; that in the spring of 1893 he brought from Puente ranch, elevation 1,200 to 1,500 feet, 19 bulls, all of which died during the following summer. On a later occasion they brought to their ranch a number of yearling bulls, about one-half of which died. It is said this ranch is free from ticks.

I went next to the following ranches where I obtained ticks: From the cattle of Marcus Foster, at Capastrano, and from the cattle of Mr. O'Neal, at Las Flores, on the Santa Margarita ranch, also near Lakeside, in El Cajon valley, 22 miles inland, and at Tia Juana, where I found an abundance of ticks on both sides of the boundary line between the Republic of Mexico and the United States.

From all these places I sent ticks to Kansas City, Mo., and one case of ticks from Las Flores, on the Santa Margarita ranch, to Washington, D. C. I met here Mr. L. J. Ware, formerly of Massachusetts; he said that, to his personal knowledge, ticks had been in the country since 1869, but not so plentiful as now, when cattle have become more numerous.

Mr. Manuel Lucero (a Mexican) said that he at one time lived near the Santa Cruz Mountain, south of San Francisco, and that he had seen plenty of the same kind of ticks in that section of the country. Another gentleman (he did not give his name) told me he had cattle on Mexican territory, 60 miles south, near the coast, and that there was plenty of ticks in that section and that his cattle were infested. Mr. Gates, of the firm of Vail & Gates, also informed me that eight or nine years ago he was engaged in the cattle business in the Salinas Valley, between Soledad and Monterey, Cal., and at that time there was plenty of ticks in the valley and that his cattle were infested.

Having completed my investigations at San Diego and its vicinity, and having obtained all the ticks desired, I returned to Los Angeles. After obtaining all the information possible from persons at Los Angeles concerning shipments of cattle made by L. J. Rose, C. R. Willoughby, and H. C. Hooker to Kansas, during the summer of 1894, I left that city for a trip up the coast for the purpose of visiting these gentlemen and obtaining from them the full particulars.

I first called upon Mr. Rose at his home near New Jerusalem, 17 miles southeast from Ventura, Cal. Mr. Rose informed me of the losses which he and his partner, Mr. Hobson, had sustained during the summer of 1893, they losing over 900 head of cattle from some disease to them unknown. He said that the losses occurred among cattle recently brought from high elevations to ranches much lower and near the coast, which had been infested with cattle ticks for years; that from the survivors he shipped about May 20, 1894, 340 head to the Barse Commission Company of Kansas City. These, he had been informed, had been sold to Mr. Jerome Pitney. The symptoms of the cattle when sick, as he explained them, were those usually present in Texas fever.

This is the shipment of cattle that arrived at Hymer Station, Kans., about May 27, 1894, and was put into one of the Berry pastures where they remained ten or twelve days and infected it with Texas fever; they were then sold to Jerome Pitney and removed to his pasture near Belvue, Kans., which they also infected. A loss of more than 50 head of cattle from Texas fever occurred in and near the two pastures.

Mr. Rose also informed me that 154 head from the original infected herd of cattle were sold to C. R. Willoughby, of Ventura, Cal., and with other cattle were shipped to Kansas City, Mo., the last of May, 1894. This is the shipment that arrived at the Kansas City stock yards June 5, 1894, 31 head of which were sold to Mr. Blackwell and taken to his pasture near Lees Summit, Mo., where Texas fever soon developed and resulted in the loss of 10 or 12 head of native cattle.

One hundred and forty head from the same shipment were bought on the same date (June 5, 1894) by O'Donald & Son and taken to their pasture near Gridley,

Kans., where 3 died. The remaining 137 were returned to the Kansas City stock yards one month later, when they were bought by Mr. Gillespie and taken to Paradise, Russell County, Kans., where Texas fever developed and resulted in the loss of several head; one or two of them, it is said, were of the California shipment.

I met Mr. Willoughby at Ventura, Cal.; he seemed reticent about his cattle shipments to Kansas, excused himself as having an important engagement, and promised that he would call the next day, but failed to do so.

I did not see Mr. H. C. Hooker; he having disposed of all his cattle, had returned to his ranch in Arizona. At Ventura and Santa Barbara I obtained what information was possible from other persons concerning his cattle shipments from California to Kansas during the summer of 1894, to wit: He sent three trains; two of them were from the Foxen or Bell ranch, near Los Olivos, Cal.; the third, or last train, was from the Santa Anita or Holister ranch, near the Santa Ynez Mountain, bordering on the coast. These shipments were made the last of May, 1894. So far as known, no harm resulted from the first two shipments, although Mr. Bell informed me that his ranch near Los Olivos was infested with some kind of ticks that got on cattle, horses, deer, and rabbits. It is supposed that from the other shipment (Santa Anita ranch) Texas fever developed near Bazar, Kans., which resulted in considerable loss. A portion of the cattle in this shipment came from the Foxen or Bell to the Santa Anita or Holister ranch about February 1, 1894, the remainder came from Tucson, Ariz., to the Santa Anita ranch in March, 1894.

Up to the time of the shipment of this train load to Kansas about 100 head of the cattle thus introduced had died, but none of the original herd on this ranch had been sick. From the symptoms of those that were sick and post-mortem examinations of those that died, as explained by Mr. Holister, no doubt remains in my mind that the disease was Texas fever, and that those that were shipped to Kansas infected the country in and around Bazar.

The ticks sent you inclosed in a redwood box came from the Santa Anita or Holister ranch, where the cattle in the last Hooker shipment came from. I regret exceedingly that, owing to the lateness of the season and the cold, rainy weather, I was unable, after leaving Santa Barbara on my trip up the coast to Niles, then across to Stockton and up the San Joaquin Valley to Bakersfield and Los Angeles, Cal., to obtain or see any cattle ticks. Had I been two months earlier I could have known to a certainty if cattle ticks existed in the several localities visited, and would not have been dependent upon the testimony of others. Seeing is believing and far more convincing than being otherwise informed; but I was assured by many persons (whom I have no reason to doubt) at the different places which I visited, and by parties I met on the train, that the whole coast as far north as San Francisco was infested with ticks during the warm weather of summer. Two of these persons told me that the coast far above San Francisco was infested with ticks. One of them, J. H. Flickengar, of San Jose, Cal., declared that to his personal knowledge they could be found at least as far north as the northern limit of California, and likewise along the foothills of the mountains on both sides of the San Joaquin Valley and in many of its alfalfa pastures.

At Hanford, Cal., Dr. William Carmichale, V. S., informed me that in the summer of 1894 a herd of cattle was brought from Cholame, on the west side of San Rafael Mountains, near the head of the Salinas Valley, to the Barber pasture near Manford; here they remained two or three weeks, after which they were slaughtered. Within three or four weeks thereafter a dairy herd of 30 cows was put in the same pasture and 17 of them died in about three weeks. Dr. Carmichale was called to see these sick cattle. The symptoms of the disease, as he described them, were glassy, staring eyes; drooping head, with ears lopped; the urine a dark, bloody color. Two of the sick animals were slaughtered. A post-mortem of these, and also of those that died from sickness, showed enlarged spleen, liver somewhat enlarged and of a mahogany color, gall bladder full of dark bile, and the contents of the third stomach, or manifolds, impact and very dry.

Dr. Carmichale, having recently emigrated from Canada and having had but little experience with diseases among cattle in this country, was unable to designate the disease. He did not recollect having seen any ticks on their bodies, and, never having heard of the cattle tick, he did not make any examination in search of them. Here in the Barber pasture and among the Hooker cattle on the Santa Anita or Holister ranch, occurred the only outbreaks of disease of any note during the summer of 1894, so far as I was able to learn.

There is no doubt in my mind that the Barber pasture was infected, and that the loss of the 17 cows of the dairy herd was caused by the introduction of ticks by the herd brought from near Cholame.

It is a conceded fact in that section that the Salinas Valley has been infested with cattle ticks for many years. I learned while at Hanford and other towns

near by that at different times and places in 1893, and previously, there was quite a number of similar attacks of sickness among the cattle of the valley. Bakersfield is the headquarters for the Kern County Cattle Company and the Miller & Lux Cattle Company; both firms have large cattle interests in the San Joaquin Valley. Mr. W. F. Wible, manager of the latter company, was away and I did not see him.

The Kern County Cattle Company claims to have about 75,000 acres of alfalfa pastures and grainfields, all or nearly all under irrigation. Owing to the rain and impassable roads, I was unable to visit the several cattle pastures. I was informed that there had been no unusual losses among cattle in that section for about two years, but that for several years previous to that time a disease unknown to the people, which had baffled the skill of all, had appeared among cattle in separate places and at various times during warm weather, and that hundreds had died, while cattle in other pastures in near localities would seem perfectly healthy.

I exhibited some ticks I had in a box and asked persons if the cattle in their pastures were infested with ticks like those. The reply was that ticks were on the cattle in some of the pastures and in others not. Mr. C. L. Conner, superintendent of the Greenfield cattle ranch, said that in his vicinity during the summer there was plenty of such ticks on the cattle.

The people of California have never suspected that the tick in any way was the cause of disease, or even an injury to their cattle, except by the loss of blood it caused, thus producing a weakness of the system. To remedy this evil, many of them with a currycomb cleaned the ticks from their cattle once or twice during the year. I was told at Bakersfield and many other places that the losses generally occurred among cattle brought during the latter part of winter, spring, and summer from Arizona, New Mexico, and other high elevations to the low elevations along the coast and to the San Joaquin Valley; but that if they were brought in during the fall and early winter the mortality was far less during the following summer.

Some thought the disease was murrain, others that it might be Texas fever; some attributed it to climatic conditions and change of feed and water; others seemed to think the cattle must have been diseased when they were brought to their pastures.

I could not hear of any disease among the cattle that had been taken from the coast and placed on the alfalfa pastures in the San Joaquin Valley. The reason is easily understood when we know that all the coast cattle have ticks, and have therefore acquired immunity against Texas fever.

The fact, as they say, that some of the alfalfa pastures in the San Joaquin Valley are not infected or unhealthy, as they term it, may be attributed to the fact that no infectious (ticky) cattle have been placed on such pastures since they were seeded down to alfalfa. Mr. Lewis Sentous, of Orleans market, Los Angeles, told me that he had two ranches, one on the coast near Santa Monica, elevation from 300 to 500 feet, covered with small brush, and during the summer his cattle there were covered with ticks. His other ranch was at Puente, 20 miles east, at an elevation from 1,200 to 1,500 feet, and on this there were no ticks.

Mr. Sentous said about one-half of the cattle he had slaughtered came from Arizona, and in the summer, if this class of cattle were placed on the Puente ranch, he lost none by sickness; but if put on the coast ranch, many of them would die if they remained there three or four weeks. These facts, he said, he had learned from experience.

I visited many other places and obtained much information, from parties of whom I have made no mention, which is only confirmatory of the facts already stated.

I think sufficient evidence has been furnished to establish the fact that the Southern cattle tick (*Boophilus bovis*) does exist in southern California, and that Texas fever is prevalent whenever the proper conditions are present for the transmission to susceptible animals (cattle from high elevations) of the infectious agent (micro-parasite), which exists in the red blood corpuscles of the cattle along the coast of southern California. In justice to other States and Territories, I would recommend the following quarantine line:

Commencing at the intersection of the southern boundary line of California with the Colorado River, thence in a northerly direction following the eastern boundary line of California to Lake Tahoe, thence in a westerly direction along the northern line of Eldorado and Sacramento counties to the Sacramento River, thence down said river to the Bay of San Francisco.

Yours, respectfully,

W. E. HILL,
Live-Stock Agent.

KANSAS CITY, MO., *February 9, 1895.*

AN OUTBREAK OF CATTLE DISEASE IN KANSAS.

An unfortunate and, as after events proved, a groundless alarm over a seeming outbreak of cattle disease startled the stockmen and farmers of eastern central Kansas early in March, 1895. During the preceding summer an unusually severe drought had prevailed over this entire section, and in consequence much of the fodder had been harvested before maturity. In handling, the blades not only broke up badly into chaff and dust, but as a food it was almost devoid of nutrition. In the month of February following occurred the worst dust and sand storm that has been known in that section for years. The dust and sand were beaten into the shocks of corn, and the fodder, already in poor condition, was rendered almost unfit for use. Owing, however, to the failure of the corn crop to mature, this damaged, innutritious fodder was the average farmer's and stockman's main resource for wintering his stock.

Naturally the general use of this quality of food was productive of bad results in all cattle whose subsistence depended upon it; they fell off in flesh; some, on account of the dust and sand, became affected with a cough, and an occasional animal in a herd here and there died. From this condition of the stock a report, the responsibility for which seems not to be definitely fixed, obtained wide circulation that pleuro-pneumonia existed among the cattle herds of Morris and Geary counties, Kans. As is usual with reports of misfortunes, this one lost nothing from reiteration, and a feeling of uneasiness, if not alarm, over such an impending calamity pervaded all persons interested. Immediately all precautions were taken to prevent the spread of disease pending investigation. The State live-stock sanitary commission quarantined the suspected herds, and the governor of the State asked the Secretary of Agriculture for investigation by an expert who had experience in diseases of this character. A thorough investigation revealed the baselessness of all fears, and in less than a fortnight from its establishment the quarantine was raised. The following letter of United States Inspector W. S. Devoe gives a full history of the investigation and the conclusions deduced:

Hon. J. STERLING MORTON,
Secretary of Agriculture.

SIR: In compliance with your instructions I beg leave to submit the following report in regard to an investigation of the alleged outbreak of contagious pleuro-pneumonia in the counties of Geary and Morris, State of Kansas.

On March 20, 1895, Prof. N. S. Mayo made a post-mortem examination on a steer owned by J. J. Furney, Beman, Morris County, Kans. He removed a portion of what seemed to be one of the ventral lobes of the lung and took it to the laboratory of the Kansas State Agricultural College.

On March 26, 1895, this part was shown to me, and the diseased portion, a small part of the inferior border, was examined by Professor Mayo and myself. There was no pleurisy. A slight thickening of interstitial connective-tissue bands was apparent and there was a slight hepatization. The tissue of affected lobules was spongy, but there was no characteristic lesion of contagious pleuro-pneumonia. From the appearance of this portion of the lung and from the coughing of the animals and the false reports of sickness in the herds, made by E. N. Turner, ex-chairman of Kansas State live-stock sanitary commission, originated the rumor of contagious pleuro-pneumonia in the herds of Geary and Morris counties, Kans.

In the afternoon of March 26 I visited the farm of John J. Furney, Beman, Morris County, Kans., in company with Prof. N. S. Mayo, J. W. Johnson, J. W. Moore, and J. I. Brown, members of Kansas live-stock sanitary board. Dr. Daniel Le May, veterinary surgeon Seventh United States Cavalry, stationed at Fort Riley, Kans., accompanied me. The latter was formerly employed by the State of Maryland in

investigating outbreaks of contagious pleuro-pneumonia and has good knowledge in regard to the disease. Mr. Furney had on hand 58 steers, 3 cows, and 4 calves. Of this number, 20 steers were purchased of John Hooper, Alta Vista, Kans., about the month of September, 1894; 7 steers were purchased of J. Bolten, Council Grove, Kans., September, 1894, and 7 steers were purchased of Chris. Longwaith, Alta Vista, September, 1894. The remainder of the steers has been on the farm for some time. The cows and calves were raised by him. He first noticed the animals coughing about December, 1894. They lost flesh and did not thrive. The coughing of the animals was more noticeable in the mornings after feeding. On March 1, 1895, 1 died suddenly, and on March 21 Professor Mayo killed 1 and made a post-mortem examination. On March 26, 1 steer was killed for a like purpose. On arriving at the farm on this date and making an ante-mortem examination of the cattle in pasture no signs of sickness were visible. The animals were in poor condition, but on chasing and driving and compelling them to run not much coughing was heard.

Professor Mayo was requested to pick out what he considered the animal most affected, and he ordered a 2-year-old steer to be killed. In endeavoring to catch the animal it was necessary for a man on horseback to ride the animal down, as, with the assistance of half a dozen men, it was found impossible to catch it. The steer broke out of the field and ran a mile down the road and back before it was caught and thrown. With all this driving hardly a cough came from the animal. On post-mortem there was no sign of pleurisy, no adhesions, and only a small portion of the right cephalic lobe of the lung showed pneumonic spots. This part was sent to Washington. This steer, the owner says, was taken sick and refused to eat about December, 1894. Its food had been only corn fodder. As soon as the owner changed its food to millet it began to recover and was improving up to the date of killing. E. N. Turner, ex-chairman of board, reported to Governor E. N. Morrill, of Kansas, that 50 per cent of the animals were sick and that there was 1 dead. None has died on the place except the one which died suddenly March 1 and the 2 which were killed for post-mortem examination. The owner reports the condition of the feed given to the animals to have been bad and dusty.

On the same day we visited the farm of John Bond, Beman, Morris County, Kans. We made a post-mortem examination on a steer which had been reported affected. It was a 2-year-old black steer afflicted with what appeared to be a hock injury, and we found, on questioning the owner, that he, on noticing a small swelling some time ago on the inside and outside of the hock joint, had punctured it with a jackknife, and the result was an opening of the bursæ and synovitis followed. It was the nigh hind leg affected, and the animal laid upon its right side continually. On the post-mortem we found a portion of the right caudal lobe of the lung discolored with hemorrhagic areas which were thought due to the imperfect bleeding and to the animal lying on that side. A small portion of the right ventral lobe showed some hepatization, but the lobules were softened. This part was sent to Washington. Mr. Bond had on hand 40 steers, 5 cows, and 1 bull, all of which have been on his place since October, 1894. He has purchased none and has noticed no sickness in his herd, with the exception of the injured steer killed for post-mortem. Mr. Bond states that the feed has been very bad this winter.

On March 28 I visited the farm of J. E. Carnahan, Alta Vista, Kans., in company with J. W. Johnson, chairman of the Kansas live-stock sanitary commission. We made a post-mortem examination on a 2-year-old red steer raised on that place, and found the mediastinal glands affected with tuberculosis. This animal was raised on the place and had been losing flesh and not thriving since October, 1894. The owner had noticed it coughing about that time, and it appeared ill during the winter. A red cow was seen on the place with glands of submaxillary space enlarged. This animal was purchased at a public sale at John Woodward's farm, 3 miles southeast of Beman, last summer. It could not be caught for the purpose of a close examination.

Mr. Carnahan has 10 steers, 6 cows, and 2 calves, all raised by him, with the exception of the cow purchased from John Woodward.

Another red cow was found coughing and emaciated, showing external appearance of tuberculosis.

E. N. Turner, ex-chairman, reports in regard to the J. E. Carnahan farm that he had 13 head, 3 of which are sick and 4 dead. There was none dead on the place and Mr. Carnahan had lost none. The animals were in a poor condition. The owner states that the feed this winter was exceedingly dirty and of poor quality.

The next place visited was the J. J. Furney farm. Here were 13 head mixed cattle. Mr. Turner reported that this man had 4 dead and 3 sick cattle. There was none dead on the place and no evidence of sickness in the herd. The animals

were in poor condition. Mr. Furney informed me that he attempted to tell Mr. Turner that 2 calves that were born during the night were found dead in the morning by the side of their mothers, having probably died from the exposure of the severe weather. Mr. Turner reports these as 2 of the 4 dead. The owner lost 2 cows in the early part of March, 1895. The animals refused to eat and died suddenly. The owner says the cause is bad feed and that he has heard no animal cough since he had changed from fodder to millet.

I next visited W. A. McCoy, Beman, Morris County, Kans. Mr. Turner reported that Mr. McCoy had 2 dead. Mr. McCoy has 1 cow and 1 heifer but they have never been sick. No animals have died on his place.

The next place visited was the corral of William and Henry Furney. I found 39 head of steers and yearlings, but on examination failed to find evidence of sickness. The owner lost a 2-year-old steer about January 5, 1895. Mr. Turner reports a number sick.

On March 29, 1895, in company with J. W. Johnson and J. W. Moore, members of the Kansas live-stock sanitary commission, I visited the farm of H. C. Church, Weston, Geary County, Kans. He had on hand March 29, 1895, 40 head mixed cattle. Mr. Turner reports that he found "5 dead and 1 sick with a bad cough." Mr. Church says that Mr. Turner simply stood on a rail fence and counted his cattle.

Professor Mayo had previously visited the farm and advised Mr. Turner to quarantine them. Mr. Church says that Professor Mayo was never within 1,000 feet of his cattle, and made no examination whatever. Mr. Church had one animal down, which had been injured, so he states, in jumping a ditch to get to water and had been trampled on. This cow showed paralysis of hind extremities and was unable to rise. No animal has died from sickness on his place, but the owner killed one in the early part of last January because she would not eat and was in poor condition. He has heard no animals cough except at feeding times, and he attributed it then to the dust in corn fodder. This man claims to be a veterinary surgeon and practices in his neighborhood. He is also the township assessor and knows all the owners and the number of cattle on their farms. He states there is no sickness to speak of among cattle in his vicinity, as his position as township assessor would enable him to know of it.

I next visited the farm of J. S. Clark, Welcome, Geary County, Kans. He had on hand 4 steers, 4 cows, 2 2-year-olds, and 12 yearlings. Mr. Turner reported 4 sick on this place. There was none showing evidence of sickness. The owner lost one steer in February during the dust storm. He has lost none before that for two years. A red cow which had been reported affected with pleuro-pneumonia was examined, but no sounds of lung trouble could be heard on auscultation and percussion. This animal was examined previously by Professor Mayo, who had reported it to the owner as a suspected case. During her sickness, which the owner states lasted one month, the animal never ceased lactation and the milk was used by the owner in his family with the exception of two days.

On March 30, 1895, in company with Professor Mayo, I visited the farm of Chris. Zuhanly, Dwight, Morris County, Kans. Mr. Turner reported that there were 22 head on this place, 3 dead and 1 sick. There were only 8 head on the place and none sick. One cow was approaching parturition; she was uneasy, but there were no visible signs of sickness. There was none dead on the place. The owner killed a steer about December, 1894, which had broken its leg. Two died during the winter from impaction. The owner made a post-mortem examination and found the rumen and omasum thoroughly distended and packed with cornstalks and dry feed. The symptoms as described by him pointed to that trouble. The animals became crazy, ran at objects, and appeared dizzy. The lungs were perfectly clear and normal in appearance. Mr. Zuhanly has had experience in slaughtering animals.

I next visited the farm of Thomas Foster, Dwight, Morris County, Kans. Mr. Turner reports that on this farm he found two dead. Mr. Foster says that Mr. Turner never visited his farm. He has on hand 3 cows and 1 calf. He lost one cow about February 15, 1895. The animal appeared in good health one night, ate her feed and gave milk, and the next morning on being called to feed, dropped dead. Mr. Zuhanly made a post-mortem at the time of death, at the request of Mr. Foster, and reports the lungs clear and sound. Another cow had its leg broken about last October and was destroyed. He has raised two of his cows and purchased one of Mr. Brainard, Alta Vista, in June, 1894.

On March 31, 1895, I visited the farm of H. Vietse, Alta Vista, Geary County, Kans. He has 31 head of mixed cattle. He raised 29 of them and purchased 2 in March, 1894, of John Garry, Alta Vista, Kans. He has lost none in two years, but has one cow that coughs in the morning when first turned out and has done so for three months. I examined her, but could detect nothing abnormal in the lung

sounds. Mr. Furney had been feeding fodder to his cattle, which were not thriving and were in poor condition up to March 1. He then changed to millet, and the animals were now improving and doing well. On examination of the herd I could find no evidence of sickness. Mr. Turner never visited this farm, but reported a sick animal on the place.

I visited the farm of W. W. Parker, 7 miles northwest of Alta Vista, Geary County, Kans. The owner has on hand 4 cows and 3 heifers. He raised them all and has purchased none for over a year. A cow last September died suddenly, but she never stopped eating or lactation never ceased. I examined the herd and found no evidence of sickness. The owner reports a bad condition of the fodder and the animals losing flesh while feeding on it, but improvement was shown as soon as feed was changed to millet. Mr. Turner did not visit this farm, but reported sickness there in cattle.

I next visited the farm of R. B. Hampton, Alta Vista, Geary County, Kans. The owner has on hand 40 head mixed cattle; raised them all. He has purchased none in two years. Sickness was reported in the herd, but on examination I found no evidence of it. The owner reports the animals coughing while given dusty fodder, but that has ceased since changing to millet.

I next visited E. H. Pettijohn, Alta Vista, Geary County, Kans. The owner had on hand 10 yearlings and 5 cows. I could find no evidence of sickness. The owner lost one cow in December, 1894, which died suddenly. She began to fail when dry feed was given to her in October, and was milked up to the time of death. The owner has noticed no sickness in his herd except the coughing of animals when given the dusty fodder, and at no other time.

Respectfully submitted.

W. S. DEVOE, *Inspector.*

TOPEKA, KANS., *April 1, 1895.*

THE COMMUNICATION OF HOG CHOLERA BY CARRION CROWS.

Hon. J. STERLING MORTON,
Secretary of Agriculture.

SIR: Your pamphlet treating of hog cholera and swine plague has just fallen into my hands. I have read it with great interest, as its importance demands. I sincerely hope that it will be widely distributed in the South, where the ravages of this disease in the past have so discouraged the farmers that they for a long time felt that it was hopeless to undertake to raise their own meat. I am prompted to write you by the recollection of a very interesting statement made to me by a farmer in this county some time since, and it is submitted to you with the expectation that it may lead to some further suggestions in the future on your part along the line of preventing the spread of the disease.

Mr. E. J. Pool, a farmer who lives some 4 miles from this place, a few years ago had a large number of hogs in the fall and set about the task of hog killing, and, being short of help, the entrails of the slaughtered animals were thrown over the fence. Presently he observed that his hogs had come in from the woods and begun to eat the offal, and soon they were assisted by a large number of carrion crows and turkey buzzards. He observed the buzzards running about over and among the offal, trampling on it with their feet, and the hogs and vultures all eating together. Within a week a fearful epidemic of hog cholera broke out and swept away nearly his entire stock of hogs. At that time there was an epidemic of the disease raging in another part of the county across Pearl River, a distance of 8 or 10 miles. Mr. Poole is sure that the vultures brought the germs of hog cholera on their feet and legs from animals dead of the disease, and in that way communicated the contagion to the offal, and the hogs in eating it were infected. I fully concur with him in this opinion, and bring the incident to your attention in the hope that you will give it such publicity as will call the attention of swine raisers to this source of infection. In devouring carrion the birds take their stand on the carcass and chase each other over and about the remains, and if they happen to have any of the germs on their feet from a former repast, and there is any particle of carrion left, the germs would be sure to be left scattered on the ground, thence to be washed into ponds and streams from which the infection might pass into the bodies of other animals. Epidemics (or rather in this case, epizootics) of cholera frequently break out sporadically without any known channel of communication between the diseased and the healthy animals. May it not be true that the germs are conveyed in such instances as they were in Mr. Poole's case?

The indications are that all animals—cattle and horses as well as swine—dying of hog cholera or of any other disease should be promptly burned or buried, so that the carrion-eating birds and animals may not have these opportunities of spreading the contagion.

Yours, very truly, T. S. FORD.

COLUMBIA, MISS., *July 1, 1895.*

NOTES ON THE CATTLE SHOW OF THE SMITHFIELD CLUB.

At the ninety-eighth annual live-stock show of the Smithfield Club, held in Islington, England, on December 9 to 14, 1895, a new and instructive feature was introduced. Under the title "Special slaughter classes" four new classes of stock were instituted—two for cattle and two for sheep. The animals entered in these classes were exhibited during the first day of the show alive. They were then removed for slaughter, and on the morning of the third day the carcasses were returned—one side whole and one side quartered—and awards were made on the quality of meat, the proportion of lean to fat in the most valuable parts, the percentage of carcass to gross live weight, and other minor merits. Their value was then further measured indirectly by a sale to dealers at auction, but the carcasses remained on exhibition until the close of the show. In the cattle contest, which is of more especial interest in the United States, the details of the competition are given in the tabulated statement on the opposite page.

Details of carcass competition.

CLASS 102.—STEERS NOT EXCEEDING 3 YEARS OLD.

Name of exhibitor.	Live weight on arrival.	Carcass weight.	Suet, caul, and reed fat.	Fat, gut fat, and trimmings.	Tongue and tail.	Head and feet.	Heart, liver, and lights.	Tripe, feck, and reed.	Hide.	Blood.	Intestines.	Award.
	Cwt. qrs. lbs.	*Cwt. qrs. lbs.*	*Qrs. lbs.*	*Qrs. lbs.*	*Lbs.*	*Qrs. lbs.*	*Qrs. lbs.*	*Cwt. qrs. lbs.*	*Cwt. qrs. lbs.*	*Qrs. lbs.*	*Qrs. lbs.*	
Earl of Tankerville's crossbred Experiment.	16 1 19	11 0 4	0 27	2 3	14	2 7	1 3	1 0 4	1 0 17	1 13	1 0	
T. Biggar & Sons' Galloway	12 3 8	8 3 1	0 27	2 5	14	1 25	1 6	0 3 13	0 2 22	1 2	0 18	Reserved and highly commended.
R. D. Jameson's crossbred	15 3 24	10 3 10	1 2	1 25	13	2 8	1 13	1 0 19	0 3 25	1 7	0 27	
Lord Hasting's Red Poll	12 2 2	8 2 0	0 20	1 6	10	1 25	1 0	0 3 8	0 3 3	1 8	0 19	Commended.
Earl of Rosebery's crossbred	16 2 12	11 0 8	1 4	3 5	13	2 7	1 3	1 0 12	0 3 8	1 13	1 0	
H. H. Murr[illegible] Stewart's Galloway	15 0 9	8 1 3	0 25	2 19	12	2 5	1 3	1 0 6	0 3 13	1 12	1 2	Second prize.
George Bruce's Aberdeen-Angus	14 2 20	9 2 1	1 3	2 14	13	2 0	1 3	1 0 20	0 2 18	1 7	0 25	Commended.
Earl of Cawdor's Highlander	12 1 24	9 2 16	0 20	1 13	11	1 26	0 25	0 3 14	1 0 14	1 1	0 21	First prize.
J. Douglas Fletcher's Aberdeen-Angus.	15 3 0	10 1 26	0 26	2 24	14	2 5	1 5	1 0 1	0 3 8	1 19	1 0	
W. Parkin Moore's Galloway	14 3 6	9 2 1	1 0	2 6	13	2 5	1 1	1 0 21	0 3 21	1 21	0 27	Third prize.

CLASS 103.—STEERS NOT EXCEEDING 2 YEARS OLD.

H. R. H. Prince of Wales's Shorthorn.	12 1 26	8 0 25	0 15	1 22	10	1 23	1 3	0 2 25	0 3 0	1 2	0 20	Third prize.
W. H. Cooke's Hereford	12 2 27	8 0 24	0 19	1 13	13	1 24	0 26	0 3 16	0 3 18	1 3	0 19	
Earl of Rosebery's Aberdeen-Angus	11 0 14	7 0 2	0 17	1 13	11	1 23	0 26	0 3 13	0 2 20	1 4	0 18	Reserved and highly commended.
John Ross's crossbred	11 3 4	7 3 2	0 27	2 1	12	1 21	0 26	0 2 26	0 2 22	1 4	0 20	First prize.
Lieut. Col. M. H. Lambert's Aberdeen-Angus.	12 0 4	7 3 18	0 14	1 5	10	1 27	0 25	0 3 12	0 2 24	1 10	0 19	
Joseph Godman's Sussex	13 3 16	9 0 16	0 21	2 6	12	2 3	1 3	0 3 5	0 3 20	1 7	0 27	Second prize.
Baron F. J. de Rothschild's Shorthorn.	1. 1 5	7 2 0	0 19	1 13	11	1 21	1 3	0 3 11	0 2 16	1 0	0 22	Commended.
W. Wood, jr.'s Sussex	12 0 9	8 0 0	1 2	1 19	11	1 22	0 27	0 2 14	0 3 10	1 7	0 20	

The deductions that have been made in England from the results of this competition have led to a great amount of discussion on the subject of the profitable fattening of stock. A knowledge of the opinions expressed may be made a source of profit to all who cater to the English trade.

To appreciate the discussion fully it must first be understood that the typical beef, according to the English taste, consists of a great depth of well-marbled lean flesh. With this fact in mind one can readily see that an inevitable conclusion from the tabulated results of the competition was that the superabundance of fat, which makes the living animal so attractive to the eye of the breeder, is a certain source of waste and loss to the butcher who buys the animal for slaughter and the profits of English trade.

The trend of English opinion on this phase of the discussion is given by a writer in the Live Stock Journal (London) of December 20, 1895. It may prevent confusion to note that he speaks of a different class than that referred to in the above tabulation. He writes:

With your permission I should like to call the attention of your readers to the way in which the prizes are awarded at the Smithfield and other shows of a like character, for, unless some better method be pursued, I fear we can no longer pride ourselves, as we like to do, on being a practical people. What is now being done is a distinct discouragement to profitable production, and is detrimental alike to the interests of the breeder, the feeder, and the consumer. It is actually putting a premium on folly.

The primary object of the Smithfield Club, as set forth on page 4 of the catalogue, is "to encourage the selection and breeding of the best and most useful animals for the production of meat * * * and to supply the * * * markets * * * with the cheapest and best meat."

The following case will show how our cattle judges and those who instruct them proceed to attain this primary object.

In class 2, Devon steers under 3 years old, 15 animals were shown; a very strong class, and, according to a reliable authority, about the best ever exhibited at Smithfield, and presumably a fourth prize was given because of the general excellence of the class.

The prizes were given as follows:

	Age.				Weight.			Gain per day.
	Years.	*Months.*	*Weeks.*	*Days.*	*Cwt.*	*Qrs.*	*Lbs.*	*Lbs.*
First, to No. 8	2	7	0	0	11	2	12	1.38
Second, to No. 21	2	5	1	1	12	1	22	1.58
Third, to No. 14	2	10	1	2	14	0	7	1.51
Fourth, to No. 7	2	9	1	0	11	3	15	1.31

With the exception of No. 14, which secured the third prize, all these animals were, for all practical purposes, much overfed, and when slaughtered their carcasses would be found to contain so large a proportion of fat as to be unsalable, without the aid of a prize, except at a low price, and if shown in the carcass class they would have been condemned.

Now, in the same class were other animals which showed a far more rapid growth and higher gain per day, as will be seen below, and which if placed in the market under equal conditions would fetch a much higher price per pound. For instance:

	Age.				Weight.			Gain per day.
	Years.	*Months.*	*Weeks.*	*Days.*	*Cwt.*	*Qrs.*	*Lbs.*	*Lbs.*
No. 16	2	9	2	0	15	1	19	1.70
No. 18	2	5	2	5	13	2	22	1.70

These steers were equally as well bred as the prize winners, and quite as handsome; in fact, more so; for who likes to see an animal so fattened as to be hardly

able to walk? If these two had been overfed like the prize winners they would have presented the same rounded and level appearance, apparently so dear to judges generally, but so little valued by the butcher and consumer.

I have no reason to think the judging of this class of Devons has been conducted on different lines from those generally followed.

Now, who gains by this system, or want of system? Certainly not the consumer, who abhors this fat meat; nor the feeder, if the gain per day is any indication of the cost of production and if the low price be taken into account he must be a heavy loser. Well, then, I suppose it must be the breeder. But what breeder? Not the hard-working, practical farmer who has to get his living thereby, and who, in my opinion, is the man to be encouraged, but a select few who own what may be termed fashionable herds. These few, under the present wasteful system, win prizes in the showyard, it is true, but the large majority of their animals would, if placed for sale in any ordinary auction or market, be looked upon as unprofitable for any other purpose.

I fear I have trespassed too long on your space, but unless something be done, and that soon, instead of being regarded as the producers of the very best meat, and doing this at the lowest possible cost, we shall in this, as in some other things, have to play second fiddle to the foreigner. I venture to suggest that the councils of our societies and clubs should seriously consider this question, and see that such rules and regulations are adopted and carried out as will secure the object for which the societies exist.

A like opinion was expressed by Mr. T. T. Cridlan, of the Butchers Trade Society, in a speech at the last annual meeting of the Smithfield Club. Referring to the recent establishment of the "special slaughter classes," he said:

We also thank the council for the modification of the old classes of cattle, and hope soon to hear of their complete abolition, as they have been a grave cause of complaint and serious loss to the butcher; they are not wanted for the consuming public for whom they cater, and as it is apparent that breeders can produce cattle of 18 hundredweight and even 19 hundredweight at under three years, another year's feeding entails not only a heavy loss on the feeder, but the additional fat is superfluous and not required by butcher or consumer.

Such expressions of opinion as the above have been centered solely upon the object lesson taught by the carcass competition of the Smithfield Club show. They are, however, capable of a much broader range of application, and may be pertinently directed to the entire meat and stock market of England. In a letter recently received by this Department from Mr. George Rogers, the London correspondent of a leading Australian journal, this broader view of the subject furnishes the theme for a discussion upon the relative merits of the imported and native beef found in the London markets. It is a source of gratification to note that American beef ranks first in all desirable points among the imported meats offered in the English market. Mr. Rogers writes:

Hon. J. Sterling Morton,
Secretary of Agriculture, Washington, D. C.:

At the close of the Smithfield Club live stock exhibit, which was opened at Islington on the 9th instant, 21 steers were killed for block-test purposes, and some report upon the results may be interesting.

Heretofore the aim of breeders has been to win prizes on judges' and not on butchers' points. But the club this year decided that the value of the animals for trade should be ascertained.

This block test has opened breeders' eyes, and indicates that American meat exporters understand English requirements better than do meat raisers here.

Without going into detail, it may be said that all the beef carcasses were far too fat for domestic use, and at any other time than Christmas would have shown a heavy loss to sellers; a recognition of this fact will serve as a good object lesson for the future.

Twenty-one sheep also were slaughtered in the block-test competition; these turned out more unsatisfactory than did the steers, and were more fit for rendering than for consumption. In spite of the prices received for the carcasses—if the

prizes which most of the sheep had taken are excepted—it is doubtful whether owners saw their money back. Such stock is quite unsuitable for other purposes than illustrating "how not to do well" and advertising.

It occurred to me to go through our markets and note the display and character of imported meats and make a comparison between them and the English product, and I venture to present my brief report.

The quantity of meat which went through our central market last week exceeded 10,000 tons, some 830 tons more than were received in the corresponding week of 1894. What quantity of this block meat was American I have not ascertained, but from observation I am satisfied that it was considerable. Deptford sent plenty of fresh beef. There were also liberal shipments from Liverpool, 500 carcasses coming thence in one consignment. The selection of the fresh beef was such that every expert had to admit it to be all that could be desired. It suited the London trade demand exactly, and had buyers not known it was American none could have recognized it as other than prime home-grown beef.

I do not, however, mean to hint that consumers ever can detect any difference between the American and the English product; on the contrary, I assert that not even experts could have found a difference between the two after an examination of the English meat offered at Smithfield last week.

Beef of finer quality, better fed for the table, and more attractive in appearance than the imported above referred to Londoners do not need. There was that proportion of fat and lean which is appreciated here, and, to use a phrase quite understandable, joints were cut in "come-again" sorts. The color of the meat certified to the excellent health of the steer, and it was impossible after scrutiny not to be sure that the age of the animal was right. A finer exhibit of fresh beef has not been seen in the London market.

I might repeat pretty nearly all I have said above regarding the American chilled beef which has been offered and sold for fair prices here during the past ten days. It was as a rule excellent, and most prejudiced indeed must be the judge who could differ from the opinion I express. Possibly some could insist that the Scotch sides were superior to it, and national predilections would excuse them. There were some excellent Scotch meats, and they sold for high prices; the quality was exceptionally good, but the American chilled meat was also choice and superior, and there was none who did not say so. Of course, the latter sold for less in the markets, but such meat has revealed to buyers generally that imported beef is not what many suppose. True, it is cheaper than home produced, but it is quite as good, and from the butcher's point of view it is better; the English demand for such beef can be relied upon as permanent. America has not only found out what quality of meat suits the English taste, but she can also furnish the quantity required. And on the other hand, England, through the demand of both dealer and consumer, is determined to have the American product, and have it, too, in larger quantities. It is the cheapest and best beef found on sale, and competes with home grown solely on the basis of merit.

American beef, compared with what I saw and know to be regularly coming from Australia, is vastly better. The hard freezing of the colonial beef handicaps it; that must be said. But even when it is thawed out and the two are placed side by side a half-blind man can instantly see the inferiority of the Australian. Color, texture, character, value for use and consequently for sale, one has, the other has not. The American beats the colonial on all points, and both butcher and consumer lay stress upon that fact every time.

Mr. Nelson, of the Colonial Consignment and Distributing Company, has invented a process for thawing meat, and I carefully inspected sides treated by him. If I could I would influence that ingenious inventor to drop his experiments and chill, as your exporters do. I am not surprised that the American agents of your great producers smile at his process and hope that he will persevere. As a humorous agent recently remarked, "It will take Mr. Nelson more than a year, and what it costs then for thawing each side is a profit to every American packer on all pieces he sells." Still, were the Australians to adopt the refrigerating system practiced by the big American houses they could not put on this market such fine-grade beef as your packers do. They are not half so alive to the advantage of a marketable appearance for their cows as are Americans. Your beef looks bright, clean, and externally faultless; theirs, the exact opposite. In a word, there is evidence in your product of gumption, much intelligent handling, very careful selection, and of an idea that what is meant for sale shall be worth buying and worth buying often. As the tradesmen put it, "If you try what I recommend you will not go over the street, but always come to me, sir." And one feels like taking this advice when standing at the butcher's store who has American beef, if a few minutes previously one has passed another shop where Australian beef is ticketed up 4

cents a pound less. Cheap and of poor quality is the verdict, and that is mine after a trip of investigation. As the evidence happens to be just to the point, supports my argument, and is obtained from a quarter where the man swears by Australian beef, I give it because its value will be unquestioned.

I may therefore sum up this report by saying: However choice English home-grown meats were—beef I especially allude to—American, as far as I have been able to discover in my very careful examination, has given great satisfaction. "English all over" may be grand from the showman's standpoint, and breeders can console themselves as they gaze upon silver cups, gold medals, and such rewards, but when we get down to the real prize winners—that is, the men who prefer to win the daily orders for the nation's food—your stockraisers are supplying beef which takes the solid gold coin. And while your people are improving their stock and discovering how cleverly they can please our buyers and how easily they can meet any demand made, we over here do not just know where we stand. One thing is certain, your best beef is good enough for the best of us, and our best is good enough to take medals, but not to make money when sent to the butcher shop. So we shall have to climb down as fast as we can; and while the English, who are slow and heavy, are feeling their way to the ground, Americans, as usual, are under the trees and gathering the ripe fruit.

The conclusion of the whole matter is, Great Britain is a market where American beef is believed to be fit to make tiptop Englishmen, and they don't want a better article.

I am, dear sir, yours, obediently,

GEORGE ROGERS.

EATON VILLAS, LAUGHTON, ESSEX, ENGLAND, *December 27, 1895.*

LAWS OF STATES AND TERRITORIES FOR THE CONTROL OF CONTAGIOUS ANIMAL DISEASES.

Laws of the States and Territories for the control of contagious and communicable diseases of domestic animals not heretofore published in the annual reports of this Bureau will be found below.

ALABAMA.

AN ACT to prevent the running at large in the State of Alabama of rabid dogs, or dogs bitten, or supposed to have been, by a rabid dog.

Be it enacted by the general assembly of Alabama, That from and after the passage of this act it shall be unlawful for any person to allow a dog or dogs which belong to him, or over which he has control, which becomes rabid, or a dog which he has information of or good reason to believe has been bitten by a rabid dog, to run at large in the State for a period of six months thereafter.

SEC. 2. That any person who shall knowingly and wilfully violate the provisions of this act shall be guilty of a misdemeanor, and upon conviction thereof shall be fined not less than fifty dollars nor more than five hundred dollars.

Approved, February 16, 1895.

ARIZONA.

AN ACT to amend Chapter Sixth, Title LIX, of the Revised Statutes of Arizona, entitled "Sheep quarantine."

Be it enacted by the legislative assembly of the Territory of Arizona, That within thirty days after the passage of this act the governor of this Territory shall appoint an inspector of sheep in and for each of the counties of this Territory. Such inspector of sheep shall hold such office until the first day of January, 1897, and until his successor is duly appointed and qualified.

Such inspector of sheep shall be a resident of the county for which he is appointed, and shall be a practical sheepman. Before entering upon the discharge of his duties he shall execute a bond payable to the Territory of Arizona, in the sum of one thousand ($1,000) dollars, with at least two good and sufficient sureties, to be approved by the board of supervisors, conditioned for the faithful discharge of the duties of his office. Such inspector may appoint one or more deputies, within his county, for whose official acts he and his official bond shall be responsible. It shall be the duty of such inspectors at any time, upon the affidavit of any citizen of his county, having in charge or owning sheep, that sheep in such county owned by or in charge of any other person, giving location thereof, are infected with scab, scabbies, or other contagious or infectious disease, or upon personal knowledge of such facts, to examine such sheep without delay, and if he shall find such sheep to be infected with any such diseases he shall notify the owner or party in charge thereof in writing, to doctor, dip, or cure them forthwith by the use of some standard curative medicine. If such owner or party in charge fail or refuse to so doctor, dip, or cure said sheep for ten days after notice, said inspector shall take said sheep into his possession at once and cure them or cause them to be cured. He may call to his assistance such aid as may be necessary for that purpose, and the owner or owners of such diseased sheep shall be liable to said inspector for all necessary expense, costs, and charges incurred in curing such sheep, including a compensation of five ($5) dollars per day to such inspector for every day or part of a day in

which he shall be necessarily employed, and fifteen (15 cts.) cents per mile for each and every mile traveled to and from such place: *Provided*, That if such complaint shall be false the party complaining shall be liable to said inspector for such mileage and per diem, such sum to be recovered in any court of competent jurisdiction: *Provided further*, That any party wilfully and maliciously making false affidavits under this act shall be guilty of perjury and punished accordingly.

SEC. 2. Any person, company, or corporation bringing or causing to be brought to any county of this Territory any sheep or band of sheep must first procure from some inspector appointed under this act a certificate that such sheep or band of sheep are sound and free from scab, scabbies, and all other contagious and infectious diseases, before entering said county.

SEC. 3. Whenever on examination of any herd or band of sheep within any county of this Territory said inspector shall find such sheep or any part of them infected by any of said diseases he shall forthwith take all and every necessary measure and precaution to prevent such disease from spreading, and the owner or owners of such diseased sheep shall immediately proceed to treat such sheep for the cure of such disease, and any person who shall refuse or neglect to immediately observe the directions of such inspector, as provided in this section or either of the two preceding sections of this act, shall be guilty of a misdemeanor, punished on conviction by a fine of not less than one hundred ($100) dollars and not more two hundred and fifty ($250) dollars.

SEC. 4. It shall be the duty of every owner of sheep or other party in this Territory wishing to change or alter the mark or to re-mark any sheep to give notice to the said inspector in writing, giving the approximate number of sheep to be so marked, their present marks and the mark or marks to which they are to be changed and the point at which such sheep are located, and the inspector shall on receipt of such notice agree with such owner or party so notifying him or in charge of such sheep upon a date and place at which said marking shall be done, and shall on the day so fixed be present at the place named, in person or in deputy, and shall inspect every sheep so re-marked, and shall keep a complete record of the mark changed and the number re-marked and the marks into which they are changed, and shall file each month with the county recorder a statement showing these facts and the name of the owner of the sheep and the parties doing the marking, and shall deliver a certificate of such facts to the owners or party in charge of such sheep, and no person shall change or re-mark a sheep without the presence of an inspector or deputy after written notice as above required. Every person so re-marking or under whose directions or at whose instance such marking shall be done without the notice above provided for and the presence of the inspector or deputy as above provided, shall be guilty of a misdemeanor and subject to a fine of not less than twenty-five (25 cts.) cents nor more than two ($2) dollars for each sheep so re-marked, and such facts when known shall be prima facie evidence of fraud in any civil action wherein the identity of the sheep or original mark shall be the question at issue, and possession of sheep so re-marked without the required certificate of inspection shall be prima facie proof of such marking by the party in charge thereof.

SEC. 5. All legal fees, charges, and expenses of such inspector under this act shall be a first lien upon any such diseased or infected sheep, in whosesoever possession they may be found, for sixty days after treatment or inspection, as herein provided, and in case the owner or owners or party in charge representing such owner or owners, in their absence, shall fail or refuse to pay any legal charges, fees, mileage, or expenses, upon the completion of such inspection or treatment, as provided in this act, such inspector may recover such fees, charges, and expenses from the owner or owners of such sheep by an action in any court of competent jurisdiction, or he may seize and hold such sheep, or any part thereof, for such payment, and if such fees, charges, or expenses are not paid within ten days after such inspection or treatment was completed, then such inspector may sell at public or private sale sufficient of such sheep to pay all legal fees, charges, and expenses, including expense of such seizure and holding, and five ($5) dollars per day for his time during such seizure and holding: *Provided*, That no person, company or corporation shall be required to treat or dip any band of ewes, or any part of them in which there are ewes with lamb, at any time from the first day of March to the first day of June in any year.

SEC. 6. The fees of inspector of sheep shall be as follows: For inspecting and granting certificate of inspection five ($5) dollars each, and fifteen (15 cts.) cents per mile for every mile necessarily traveled in making such inspection and enforcing the requirements of this act, or any duty or requirement thereunder, and five ($5) dollars per day for each day or part of a day more than one which he shall be so engaged in the inspection and handling or treatment of such sheep, including

the time he shall hold such sheep after treatment before payment of his legal fees, expenses, and charges.

SEC. 7. Any person, company, or corporation violating any of the provisions of this act shall be liable in a civil action for all damages sustained by any person, company, or corporation in consequence of such violation.

SEC. 8. It shall be the duty of such inspector and his deputies to institute prosecution for all violation of this act, but nothing herein contained shall prevent other persons from so doing.

SEC. 9. Upon the arrival of any band or herd of sheep in this Territory the owner or person in charge shall immediately report such sheep to the inspector in the county where such sheep may be for inspection. Upon failure to do so such owner or person in charge of such sheep shall upon conviction be punished by a fine of not less than one hundred ($100) dollars and not more than two hundred and fifty ($250) dollars.

SEC. 10. All acts and parts of acts in conflict with this act are hereby repealed.

SEC. 11. This act shall take effect and be in force from and after its passage.

Approved, March 21, 1895.

KENTUCKY.

[Chapter 5, Article II, Kentucky Statutes.]

SECTION 48. *Contagious diseases—State board of health to suppress.*—Whenever any contagious or infectious disease affecting cattle shall exist in this State, it shall be the duty of the State board of health to take measures to promptly suppress and effectively prevent the same from spreading.

SEC. 49. *Board of health—Powers and duties in suppressing contagious diseases.*—For such purposes the State board of health shall have power to issue their proclamation, stating that infectious or contagious disease exists in any county or counties of the State, and warning all persons to seclude all animals in their possession that are affected with such disease, or have been exposed thereto, and ordering all persons to take such precautions against the spreading of such disease as the nature thereof may, in their judgment, render necessary or expedient; to order that any premises, farm or farms where such disease exists, or has existed, be put in quarantine at the owner's expense, so that no cattle be removed from or brought to the premises so quarantined, and to prescribe such regulations concerning the mode of quarantine as they may judge necessary or expedient to prevent infection or contagion being communicated in any way from the places so quarantined; to call on the sheriffs and deputy sheriffs to assist in enforcing and carrying out the provisions of said proclamations and orders, whose duty it shall be to observe and obey all of said orders and proclamations; to employ a veterinary surgeon and practitioner, and such other persons as may be necessary, from time to time, in performing their duties under this article, and to prescribe regulations for the destruction of such animals so affected, and for the disposition of their hides and carcasses, and all objects which might convey infection or contagion, at the owner's expense. But no animal shall be destroyed by said board unless first examined by a veterinary practitioner acting in the employ or under the direction of said board, or unless the owner thereof knows that such cattle are so affected. Said board shall also have power to prescribe regulations for the disinfection of all premises, buildings, and railway cars, and all objects by or from which infection may take place or be conveyed; to alter, modify, cancel, or withdraw any of said proclamations, orders, or regulations whenever they may deem it proper so to do.

SEC. 50. *Duty of owner of diseased cattle—Penalty—Duty of board.*—Any person knowingly transgressing or failing to comply with the terms of any proclamation, order, or regulation issued or prescribed by the board shall be guilty of a misdemeanor and on indictment be fined in a sum not less than two hundred nor more than one thousand dollars; and the owner of any cattle affected with the contagious disease known as pleuro-pneumonia, and knowing the same to be so affected, who fails to kill and bury or burn them, or fails to report the same at once to the State board of health, shall be fined for each offense in a sum not less than two hundred nor more than one thousand dollars; and the said board, upon the failure of any such person to immediately kill said diseased cattle when ordered by the board so to do, shall have the right to kill the same at the owner's expense.

SEC. 51. *Pleuro-pneumonia—Penalty for importing cattle affected with.*—Any person bringing cattle into this Commonwealth having the disease pleuro-pneumonia, and knowing or having reasonable grounds to believe the same to be so

affected, shall be deemed guilty of a felony, and on indictment and conviction be confined in the penitentiary not less than one nor more than four years.

SEC. 52. *Penalty for selling or driving such cattle.*—Any owner of cattle affected with pleuro-pneumonia, and knowing the same to be so affected, or knowing the same to have been exposed thereto, who drives or causes the same to be driven upon the public highway, or sells or transfers the same, shall on indictment and conviction be fined in a sum not less than two hundred nor more than one thousand dollars.

SEC. 53. *Veterinarian may be employed—Compensation—Penalty.*—The State board of health shall have the power to employ a veterinarian, who shall be a regular practitioner and graduate of some college of veterinary surgery and practice, and who shall be known as the State veterinarian, whose duty it shall be to render such service under this law as the board may direct. Said veterinarian shall be entitled for the services rendered to a sum not exceeding five dollars per day and traveling expenses for the time he is actually engaged at work for said board, to be paid by the county court of the county in which the disease is prevailing. The veterinarian shall receive nothing from any other person for examinations and work done at the instance of said board, and in the event he does he shall upon indictment and conviction be fined in a sum not less than two hundred nor more than one thousand dollars.

SEC. 54. *State board may cooperate with Federal Government.*—In order to effectually carry out the provisions and intentions of this law, the board may engage and obtain the services of any veterinarian in the employ of the United States, and otherwise cooperate with the proper department of the Federal Government in the suppression of said disease in this Commonwealth.

SEC. 55. *Governor to appoint three persons to act with State board.*—The governor shall, in addition to the number of persons now on said State board of health, appoint three discreet and intelligent housekeepers engaged in the cattle business, whose duty it shall be to serve on said board without any charge for services, but said three persons shall only have the right and power to act thereon with reference to matters embraced in this article.

SEC. 56. *Power of jury and court in trial of cases.*—If any person shall be convicted of a violation of fiftieth, fifty-first, or fifty-second sections of this article, the jury shall have the power to determine by their verdict whether or not such person is at the time of his trial and conviction the owner of any cattle which have been exposed to said disease by having been within the same inclosure with a diseased animal within four months next before trial, and if they find he has such cattle they will find the number, sex, and distinguishing marks, and it shall be the duty of the court to order the destruction of said cattle by the sheriff at the owner's expense, and the expense thereof shall be taxed as costs in the case.

SEC. 57. *Appropriation to aid in suppressing pleuro-pneumonia.*—In order to more effectually stamp out said disease in this Commonwealth, and to encourage those persons whose cattle may be affected therewith or exposed thereto to make the same known, the sum of three thousand dollars is appropriated out of any money that may be in the treasury not otherwise appropriated, for which sum the auditor shall, at the request of the State board of health, draw his warrant in their favor upon the treasurer; said board may use said sum, or any part of it in their discretion, for the purchase and destruction of any cattle which may have been exposed to said disease, but which do not actually have the same, and if all of said sum is not so used they shall return the remainder to the treasury.

SEC. 58. *Compensation for cattle killed—When to be paid.*—In purchasing said cattle said board shall in no case pay more than thirty dollars for any one animal, nor shall they pay for any cattle whose owner has concealed the existence of said disease among his cattle or on his premises, or has in any way, by intentional act or by willful neglect, contributed to the spread of the disease.

SEC. 59. *Appropriation to suppress disease by governor—Limit of.*—Should said disease make its appearance hereafter in any portion of the State where it has not heretofore existed, the governor, if the general assembly is not in session, shall have the power to direct the application by said board of a sum not exceeding three thousand dollars for the purpose of paying for such cattle as may be exposed to but not actually affected by said disease, upon such conditions and limitations as he may prescribe; said sum, upon the written order of the governor, shall be paid to said board out of the treasury upon the warrant of the auditor. But the whole appropriation made shall not exceed six thousand dollars.

SEC. 60. *Board to keep account of expenses, and report.*—The board of health shall keep and report to the general assembly an itemized account of their expenditures under this article.

SEC. 61. *Quarantine may be established.*—Whenever the State board of health shall have information of the existence of pleuro-pneumonia in any other State or

county they shall have power to quarantine against the cattle of such State or locality to prevent the importation of said disease into this Commonwealth, upon such terms and conditions as the board may prescribe.

SEC. 62. *Diseased cattle to be confined—Duty of owner—Penalty.*—If the owner of any distempered cattle, or hogs diseased with hog cholera, shall permit them to run at large outside of his inclosure, or shall drive the same into or through any part of this Commonwealth, unless it be from one portion of his own inclosure to another, he shall forfeit and pay the sum of ten dollars for each head, and be liable by civil action for any damage that may occur by the spreading of the disease; and when any such cattle or hogs shall die, the owner thereof shall cause them to be burned or buried, and if he fail he shall be fined five dollars for each offense.

SEC. 63. *Diseased cattle—Duty of justice and constable concerning.*—If a justice of the peace be informed by affidavit that the owner of such cattle or hogs as are described in the preceding section has violated its provisions, it shall be his duty to issue his order in the name of the Commonwealth to such owner, commanding him to impound them; and if he fail or refuse to do so, or permit them to escape, he shall have power to order the cattle or hogs to be killed and burned or buried; and the constable or other officer who is directed to execute the order shall be paid by the owner two dollars for killing and burning or burying each head, and if he fail or refuse to execute the order he shall be fined two dollars in each case.

MASSACHUSETTS.

[Chapter 496, acts of the year 1895.]

AN ACT relative to inspection of domestic animals.

Be it enacted, etc., That section four of chapter four hundred and ninety-one of the acts of the year eighteen hundred and ninety-four is hereby amended by inserting in the second line, after the word "cattle," the words "sheep and swine," and by inserting in the twelfth line, after the word "animals," the words "and any barn, stable, or premises where any such animals are kept;" also by adding at the end of said section the words: "*Provided*, That nothing in this act shall apply to the inspection of sheep or swine slaughtered in wholesale slaughtering establishments, or to the obtaining of a license for the slaughtering of such sheep or swine," so as to read as follows:

"SEC. 1. Said inspectors shall make regular and thorough inspections of all neat cattle, sheep, and swine found within the limits of their several cities and towns. Such inspections shall be made at such times and in such manner as the board of cattle commissioners shall from time to time determine and direct. They shall also make from time to time inspections of all other domestic animals within the limits of their several cities and towns, whenever they have knowledge or reason to suspect that such animals are affected with or have been exposed to any contagious disease, and they shall immediately inspect any and all domestic animals and any barn, stable, or premises where any such animals are kept, whenever directed so to do by the board of cattle commissioners or any of its members: *Provided*, That nothing in this act shall apply to the inspection of sheep or swine slaughtered in wholesale slaughtering establishments, or to the obtaining of a license for the slaughtering of such sheep or swine."

SEC. 2. Section six of said chapter is hereby amended by inserting in the second line, after the word "cattle," the words "sheep or swine;" by striking out in said second line, after the word "such," the word "cattle," and inserting in place thereof the word "animals," so as to read as follows:

"SEC. 6. Whenever an inspector is satisfied, upon an examination of any neat cattle, sheep, or swine, that such animals are free from contagious disease, he shall deliver to the owner or to the person in charge thereof a written certificate of their wholesome condition, signed by him, which certificate shall be in such form as the board of cattle commissioners shall prescribe, and shall cause a copy of said certificate to be entered upon his records."

SEC. 3. Section seventeen of said chapter is hereby amended by striking out in the fifth line the word "cattle," and inserting in place thereof the words "neat cattle, sheep, or swine;" by striking out in the twentieth line the word "cattle," and inserting in place thereof the words "neat cattle, sheep, and swine," so as to read as follows:

"SEC. 17. The proprietor or proprietors of every slaughterhouse, canning, salting, smoking, or rendering establishment, and of every establishment used for the

manufacture of sausages or chopped meat of any kind, engaged in the slaughter of neat cattle, sheep, or swine, the carcass or any of the meat or product of which is to be sold or used for food, shall within thirty days after the passage of this act, and thereafter annually in the month of April, make application to the mayor and aldermen of the city or the selectmen of the town where such slaughterhouse or establishment is located for a license to carry on such business. Such application shall be in writing, signed by one or more of the owners thereof, or by one or more of the persons carrying on such business; if such owner or the person carrying on such business be a corporation, then by some officer thereof thereto duly authorized. Such application shall give the name in full and address of all the owners or persons carrying on said business, the location of the slaughterhouse or establishment where said business is to be conducted, shall state the estimated number of neat cattle, sheep, and swine to be slaughtered per week, the day or days of the week upon which it is intended to slaughter the same, and the nature of the product or products thereof to be sold or used for food. Every such application shall be sworn to before a justice of the peace."

SEC. 4. Section eighteen of said chapter is hereby amended by striking out in the fifth line the word "cattle," and inserting in place thereof the words "neat cattle, sheep, or swine;" and by adding at the end thereof the words "The board or officer of every city or town authorized to issue said licenses shall, on or before the first day of June in each year, send to the board of cattle commissioners a copy of every application made to them under section seventeen of this act, and shall state the doings of said board or officer upon said application; and shall futher send to the board of cattle commissioners the names and addresses of all persons required to make application under section seventeen, who were engaged in such business on the last day of the previous April, and who have failed to make application as provided in said section," so as to read as follows:

"SEC. 18. The mayor and aldermen of cities and the selectmen of towns, or such other board of officers as they shall designate, may annually issue to persons applying therefor licenses to carry on the business of slaughtering neat cattle, sheep, or swine, and there shall be paid to such city or town for every such license, by the person or persons obtaining the same, a fee of one dollar. Every such license shall name the person or persons licensed to conduct such business, the building or establishment where the same is to be carried on, and such license shall continue until the first day of May of the year next ensuing, or until sooner forfeited or rendered void. The board or officer of every city or town authorized to issue licenses hereby shall keep a record of all applications for licenses under section seventeen, and shall record therein every license issued by him or them, and such records shall be competent evidence in any court of the issue of any such license. The board or officer of every city or town authorized to issue said licenses shall, on or before the first day of June in each year, send to the board of cattle commissioners a copy of every application made to them under section seventeen of this act, and shall state the doings of said board or officer upon said application; and shall further send to the board of cattle commissioners the names and addresses of all persons required to make application under section seventeen, who were engaged in such business on the last day of the previous April, and who have failed to make application as provided in said section."

SEC. 5. Section nineteen of said chapter is hereby amended by striking out, in the fourth line, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, or swine," and by striking out, in the seventh line, the word "cattle" and inserting in place thereof the word "animals" so as to read as follows:

"SEC. 19. No person or persons licensed under the preceding section shall slaughter or cause or authorize to be slaughtered at such slaughter house or establishment, except in the presence of an inspector, any neat cattle, sheep, or swine on any day or days other than those specified in the application for such license, except that such licensee may at any time change the day or days for slaughtering such animals by giving at least seven days' written notice of such change to the board or person authorized to issue licenses under the provisions of section eighteen; and such board or person shall immediately give written notice of such change to all inspectors appointed by such city or town."

SEC. 6. Section twenty of said chapter is hereby amended by striking out, in the twelfth line, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, and swine;" by striking out, in the fourteenth line, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, and swine," so as to read as follows:

"SEC. 20. It shall be the duty of the inspectors of animals and provisions of the several cities and towns, or when there is more than one inspector appointed for any such city or town and any one of them has been duly selected and ordered so

to do by the board of health of such city or town, then of such inspector so selected and ordered to be present at all licensed slaughter houses or establishments upon the day or days designated for slaughter in the application for such license, or at such other day or days as may be designated under the provisions of section nineteen, and there carefully examine at the time of slaughter the carcasses of all neat cattle, sheep, and swine slaughtered thereat. And it shall be the duty of such inspectors also to examine at the time of slaughter any and all neat cattle, sheep, and swine slaughtered as provided in section twenty-one of this act, whenever notified so to do by the person slaughtering the same. Such inspection shall be made in such manner and under such rules and regulations as the board of cattle commissioners may, from time to time, determine and direct. Whenever, in the opinion of any inspector, any of said carcasses are diseased or any meat or product thereof is diseased, corrupted, unwholesome, or unfit for food, he shall seize the same and cause it to be destroyed as provided in section ten of this act, subject, however, to the provisions therein contained concerning appeal and the disposal of money."

SEC. 7. Section twenty-one of said chapter is hereby amended by striking out, in the fifth line thereof, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, or swine;" by inserting, in the eleventh line, after the word "animal," the words "is less than six months old or" so as to read as follows:

"SEC. 21. None of the provisions of sections seventeen, eighteen, nineteen, and twenty of this act shall apply to the slaughter from time to time by any person not engaged in such business, as provided in section seventeen, of one or more of his own neat cattle, sheep, or swine, when the same are slaughtered upon his own premises other than a slaughter house or establishment mentioned in section seventeen, but such persons shall cause such carcass to be inspected at the time of slaughter by an inspector of animals and provisions appointed under the provisions of this act, unless said animal is less than six months old or has been duly inspected under the provisions of this act within six months prior to such slaughter and a certificate of health has been delivered to the owner or person in charge thereof, as provided in section six."

SEC. 8. Section twenty-two of said chapter is hereby amended by striking out, in the fourth line, the word "cattle" where it occurs therein, and inserting in place thereof the words "neat cattle, sheep, or swine;" by striking out, in the tenth line, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, or swine;" by striking out, in the thirteenth line, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, or swine;" by striking out, in the eighteenth line, the word "cattle" and inserting in place thereof the words "neat cattle, sheep, or swine" so as to read as follows:

"SEC. 22. Any person violating any of the provisions of sections eighteen, nineteen, twenty, and twenty-one, or who, being engaged in the business of slaughtering neat cattle, sheep, or swine, shall slaughter any neat cattle, sheep, or swine, or shall knowingly authorize or cause the same to be slaughtered, with the intent of selling the carcass or any of the meat or product thereof for food without first having applied for and obtained a license as provided in sections seventeen and eighteen, or who, having obtained such license, slaughters or knowingly authorizes or causes to be slaughtered any neat cattle, sheep, or swine without causing the carcass thereof to be inspected as provided in section twenty, or who sells or authorizes or causes to be sold any carcass, meat, or product of any neat cattle, sheep, or swine, knowing that such carcass or the carcass from which such meat or product was obtained had not been inspected according to the provisions of sections twenty or twenty-one of this act, or who slaughters or knowingly authorizes or causes to be slaughtered any neat cattle, sheep, or swine upon his own premises other than a slaughter house or establishment mentioned in section seventeen without causing the carcass of such animal to be inspected, except as provided in section twenty-one, or who sells or authorizes or causes to be sold the carcass or any meat or product thereof of any such animal slaughtered upon his own premises, knowing that the same has not been inspected as provided in section twenty-one, shall be punished by a fine not exceeding five hundred dollars or by imprisonment in jail for not exceeding sixty days, or by both such fine and imprisonment."

SEC. 9. Section twenty-seven of said chapter is hereby amended by striking out all of said section after the word "possession," in the sixth line thereof, and inserting in place thereof the following: "thereof; but whenever specific animals are quarantined or isolated under the provisions of sections seven, twenty-six, and forty-five of this act more than ten days upon such premises, as suspected of being afflicted with a contagious disease, and the owner is forbidden to sell any of

the product thereof for food, or whenever any animals are quarantined, collected, or isolated on any premises other than those of such owner or person in possession thereof, the expense of such quarantine shall be paid by the Commonwealth" so as to read as follows:

"SEC. 27. When any animals are quarantined, collected, or isolated under the provisions of this act upon the premises of the owner or of the person in possession thereof at the time such quarantine is imposed, the expense thereof shall be paid by such owner or person in possession thereof; but whenever specific animals are quarantined or isolated under the provisions of sections seven, twenty-six, and forty-five of this act more than ten days upon such premises, as suspected of being afflicted with a contagious disease, and the owner is forbidden to sell any of the product thereof for food, or whenever any animals are quarantined, collected, or isolated on any premises other than those of such owner or person in possession thereof, the expense of such quarantine shall be paid by the Commonwealth."

SEC. 10. Section forty-five of said chapter is hereby amended by striking out, in the sixth and seventh lines, the words "at the expense of the owner;" by striking out, in the twenty-second line, the words "one-half of" and inserting in place thereof the word "full;" by striking out, in the twenty-third and twenty-fifth lines, the words "slaughter for food or milk purposes, and without taking into consideration the existence of such disease," and inserting in place thereof the words "condemnation not exceeding the sum of sixty dollars for any one animal;" by inserting in the twenty-seventh line, after the word "been," the word "owned;" and by striking out all after the word "thereto," in the twenty-ninth line, and inserting in place thereof the words "in the judgment of the cattle commissioners, by willful act or neglect, contributed to the spread of tuberculosis; but such decision on the part of the commissioners shall not deprive the owner of the right of arbitration as hereinafter provided" so as to read as follows:

"SEC. 45. When the board of cattle commissioners, or any of its members, by an examination of a case of contagious disease among domestic animals becomes satisfied that the public good requires it, such board or commissioner shall cause such animal or animals affected therewith to be securely isolated, or shall cause it or them to be killed without appraisal or payment. Such order of killing shall be in writing, and may be directed to the board of health, inspector, or other person, and shall contain such direction as to the examination and disposal of the carcass and the cleansing and disinfecting of the premises where such animal was condemned as such board or commissioner shall deem expedient. A reasonable sum may be paid out of the treasury of the Commonwealth for the expense of such killing and burial. If it shall subsequently appear, upon post-mortem examination or otherwise, that such animal was free from the disease for which it was condemned, a reasonable sum therefor shall be paid to the owner thereof by the Commonwealth: *Provided, however*, That whenever any cattle condemned as afflicted with the disease of tuberculosis are killed under the provisions of this section the full value thereof at the time of condemnation, not exceeding the sum of sixty dollars for any one animal, shall be paid to the owner thereof out of the treasury of the Commonwealth if such animal has been owned within the State six months continuously prior to its being killed, provided such person shall not have prior thereto, in the judgment of the cattle commissioners, by wilful act or neglect, contributed to the spread of tuberculosis; but such decision on the part of the commissioners shall not deprive the owner of the right of arbitration, as hereinbefore provided."

SEC. 11. If the owner who is entitled to compensation for an animal destroyed as affected with tuberculosis under section forty-five of said chapter four hundred and ninety-one and the commissioner condemning the same can not agree as to the value of the animal so condemned, the question of such value shall be determined by arbitrators, one to be selected by the commissioner, one to be selected by the owner; or if the owner neglects or refuses for twenty-four hours to select an arbitrator, the one already selected shall select a second, and if these two can not agree, a third to be selected by the two arbitrators first selected. Such arbitrators shall be sworn faithfully to discharge the duties of their office, and shall determine the value of such animal according to the provisions of said section forty-five, and the full value so determined shall be paid to the owner as provided in said section. Either party aggrieved by the doings of the cattle commissioners, or any of its members, under the provisions of said section forty-five, or by the award of such arbitrators, may petition the superior court for the county where such animal was killed, or for the county of Suffolk, to have the damages assessed; such petition shall be by or against the board of cattle commissioners, and a copy thereof shall be served upon the defendant, or if the petition is against said board

of cattle commissioners, upon one of the commissioners, in the same manner as is provided for the service of other civil process. Such petition shall be filed in the clerk's office of the superior court for said county within thirty days after the killing of such animal or animals. Such petition shall be subject to the provisions of section sixty-nine of chapter one hundred and sixty-seven of the public statutes, and a trial may be had thereon at the bar of the court in the same manner as other civil cases are tried. If upon such trial it shall be determined that such animal was not affected with the disease for which it was condemned, reasonable compensation may be recovered therefor, and if the owner recovers damages in excess of the amount previously awarded him by the arbitrators, or allowed him by the commissioners, he shall recover his costs; otherwise he shall pay costs. The damages, costs, and expenses incurred by the commissioners in prosecuting or defending any such action shall be paid by the Commonwealth.

SEC. 12. The commissioners may examine under oath all persons believed to possess knowledge of material facts concerning the existence or dissemination or danger of dissemination of contagious diseases among domestic animals, or concerning any other matter within the provisions of this act and said chapter four hundred and ninety-one, and each of said commissioners shall have all the powers vested in justices of the peace to take depositions, to compel witnesses to attend and testify before said commission, and to administer oaths for any of the purposes of this act by chapters one hundred and fifty-five and one hundred and sixty-nine of the public statutes. The fees for such witnesses for attendance and travel shall be the same as for witnesses before the superior court. All costs and expenses incurred in procuring the attendance of such witnesses shall be allowed and paid by the Commonwealth. Copies of the records of the board of cattle commissioners or of any regulation or order issued by said board or any of its members under the provisions of this act, when duly certified by the secretary of said board, and any certificate by said secretary of the issuing, recording, delivering, or publishing of any such orders or regulations under the provisions of section forty, shall be competent evidence of such fact in any tribunal.

SEC. 13. The amount to be expended under this act shall not exceed one hundred thousand dollars for the current year.

SEC. 14. Until June first, eighteen hundred and ninety-six, the use of tuberculin as a diagnostic agent for the detection of the disease known as tuberculosis in domestic animals shall be restricted to cattle brought into the Commonwealth from any point without its limits and to all cattle held in quarantine at Brighton, Watertown, and Somerville: *Provided, however*, That tuberculin may be used as such diagnostic agent on any animal or animals in any other portion of the State upon the consent in writing of the owner or person in possession thereof, and upon any animals condemned as tuberculous upon physical examination by a competent veterinarian.

SEC. 15. Sections forty-six and forty-nine of chapter four hundred and ninety-one of the acts of the year eighteen hundred and ninety-four are hereby repealed.

SEC. 16. This act shall take effect upon its passage.

Approved, June 5, 1895.

MONTANA.

[Laws relating to the control of contagious and infectious diseases of domesticated animals passed by the fourth legislative assembly of the State of Montana, and in full force and effect from and after the 1st day of July, A. D. 1895.]

POLITICAL CODE.—ARTICLE V.

SECTION 1986. The governor is authorized to nominate, and, by and with the advice and consent of the senate, appoint a competent veterinary surgeon, who is known as the "State veterinary surgeon," who holds his office for two years, and must execute a bond in the sum of five thousand dollars, and who, before entering on his duties, must take and subscribe the oath of office prescribed by the constitution.

SEC. 1987. The duties of the State veterinary surgeon are: (1) To investigate all cases of contagious and infectious diseases among cattle, horses, mules, and asses in this State of which he may have a knowledge, or which may be brought to his notice by any resident in the locality where such disease exists; and in the absence of specific information to make visits of inspection to any locality where he may have reason to suspect that there is any contagious or infectious

disease. (2) To inspect, under the regulations of this article, all such animals which may be brought into this State in any manner whatever from or through such State, Territory, or foreign country as the governor may declare by proclamation, upon the recommendation of the board of stock commissioners, or otherwise, must be held in quarantine for the purpose of inspection for contagious or infectious diseases.

SEC. 1988. After the making of such proclamation the owner or person in charge of any such animals, arriving in this State from or through any State, Territory, or foreign country, against which quarantine has been declared, must notify the State veterinary surgeon without delay, and must not allow such animals to leave the place of arrival until they have been examined by the veterinary surgeon, and his certificate obtained that all such animals are free from disease; and no animal pronounced unsound from disease by the veterinary surgeon must be turned loose or allowed to run at large, or removed or permitted to escape, but must be held subject to the order of the veterinary surgeon. Any person failing to comply with the provisions of this section is punishable as provided in section 730 of the penal code, and is liable for any damage and loss that may be sustained by any person by reason of the failure of such owner to comply with the provisions of this section.

SEC. 1989. The owner of such animals, ridden under the saddle or driven in harness into this State, or under yoke, and any person coming into this State with his own team or teams, is not required to notify the veterinary surgeon or await the inspection of the animals, but he is liable for all loss or damage to any person by reason of any contagious or infectious disease brought into the State by his animals, and no such animals must be held in quarantine for a longer period than ninety days, unless contagious or infectious disease is found to exist among them.

SEC. 1990. In all cases of contagious or infectious disease among domestic animals or Texas cattle in this State, the veterinary surgeon has authority to order the quarantine of the infected premises, and in case such disease becomes epidemic in any locality in this State, the veterinary surgeon must immediately notify the governor, who must thereupon issue his proclamation forbidding any animal of the kind among which said epidemic exists to be transferred from said locality without a certificate from the veterinary surgeon showing such animal to be healthy. The expenses of holding, feeding, and taking care of all animals quarantined under the provisions of this article must be paid by the owner, agent, or person in charge of such animals.

SEC. 1991. In case of any epidemic disease where premises have been previously quarantined by the veterinary surgeon, as before provided, he is further authorized and empowered, when in his judgment necessary, to order the slaughter of any and all such diseased animals upon said premises, and all such animals as have been exposed to contagion or infection, under the following restrictions: The order must be a written one, and must be made in duplicate, and there must be a separate order and duplicate for each owner of the animals condemned, the original of each order to be filed by the veterinary surgeon with the secretary of state, and the duplicate given to the owner. Before slaughtering any animal that has been exposed only and does not show disease, the veterinary surgeon must call in consultation with him two practicing veterinary surgeons or physicians, residents of the State, or if this is impossible, then two stock owners, residents of the State, and he must have written endorsements upon his order of at least one of the consulting persons, stating that such action is necessary, before the animal is slaughtered.

SEC. 1992. Whenever, as in this article provided, the veterinary surgeon orders the slaughter of one or more animals, he must at the time of making such order notify in writing the nearest available justice of the peace, who must thereupon summon three disinterested citizens, who are stock owners in the neighborhood, to act as appraisers of the value of the animal. The appraisers before entering upon the discharge of their duties must be sworn to make a true and faithful appraisement without prejudice or favor. They must, after making their appraisement, return certified copies of their valuation, a separate one being made for each owner, together with an accurate description of each animal slaughtered (giving all brands, earmarks, wattles, age, sex, and class, as to whether American, half-breed or Texan), to the justice of the peace by whom they were summoned, who must, after entering the same upon his record and making an indorsement upon each showing it to have been properly recorded, return it, together with a duplicate order of the veterinary surgeon, to the person owning the animal slaughtered; and it is the duty of the veterinary surgeon to superintend the slaughter of such animals as may be condemned and also the destruction of the carcass, which latter must be by burning to ashes or burying in the earth to the depth of not less than

six feet, and which must include every part of the animal and hide and also excrement as far as possible. If the owner of any animal found diseased by the veterinary surgeon is killed, or consents to its being killed by the veterinary surgeon without appraisement, then the veterinary surgeon must burn or bury it as herein provided.

SEC. 1993. The veterinary surgeon must make an annual report on or before the first day of October to the State board of stock commissioners of all matters connected with his work, and the board must make the same a part of their annual report to the governor, and they must also transmit to the several boards of county commissioners such parts of the report as they consider necessary and of general interest to the breeders of live stock. The board must also give information in writing, as soon as it is obtained, to the governor and to the various boards of county commissioners, of each case or supposed case of disease in each locality, the cause, if known, and the measures adopted to check it.

SEC. 1994. Whenever the governor has good reason to believe that any disease mentioned in this article has become epidemic in certain localities in another State or Territory, or that conditions exist that render domestic animals and Texas cattle likely to convey disease, he must, by proclamation, designate such localities and prohibit the importation therefrom of any live stock of the kind diseased into this State, except under such restrictions as he, after consultation with the veterinary surgeon, may deem proper. Any person who, after the publication of such proclamation, knowingly receives in charge any animal from any of the prohibited districts and transports or conveys the same within the limits of this State is punishable as provided in section 739 of the Penal Code, and is further liable for any and all damages and loss that may be sustained by any person by reason of the importation or transportation of such prohibited animals.

SEC. 1995. It is the duty of any person who has upon his premises, or upon the public domain, any case of contagious or infectious disease among such animals, to immediately report the same to the veterinary surgeon, and a failure so to do, or any attempt to conceal the existence of such disease, or to willfully or maliciously obstruct or resist the veterinary surgeon in the discharge of his duty, is punishable as prescribed in section 740 of the Penal Code, and forfeits all claims to indemnity for loss from the State.

SEC. 1996. The following regulations must be observed in all cases of disease mentioned in this article:

1. It is unlawful to sell, give away, or in any manner part with any animal affected with or suspected of being affected with contagious or infectious disease, and in case of an animal that may be known to have been affected with or exposed to any such disease within one year prior to such disposal, due notice of the fact must be given in writing to the party receiving the animal.

2. It is unlawful to kill, for the purpose of selling the meat, any such animal, or to sell, give away, or use any part of it, or its milk, or to remove any part of the skin. A failure to observe these provisions is punishable as provided in section 740 of the Penal Code. It is the duty of the owner or the person having in charge any such animal affected with or suspected of being affected with any contagious or infectious disease to immediately confine the same in a safe place, isolated from other animals and with all necessary restrictions to prevent dissemination of the disease, until the arrival of the veterinary surgeon. These regulations apply as well to animals in transit through the State as those resident therein, and the veterinary surgeon or his duly authorized agent has authority to examine in car, yard, pasture, or stables, or upon the public domain, all such animals, and on detection of disease to take possession of and treat and dispose of the animals in the same manner as provided by this article.

SEC. 1997. All claims arising from the slaughter of animals under the provisions of this article, together with the order of the veterinary surgeon and the valuation of the appraisers in each case, must be submitted to the State auditor, and for each claim that he finds to be equitable and entitled to indemnity under this article must issue to the person entitled thereto his warrant on the stock indemnity fund in the State treasury for the sum named in the appraisers' report. In auditing any claim under this article the auditor must satisfy himself that it does not come under any class for which indemnity is prohibited by this article, and he must require the affidavit of the claimant to this fact, or if the claimant be not cognizant thereof, then of some reputable person who is cognizant thereof; and also the certificate of the veterinary surgeon, whose duty it is to inform himself fully of the fact that in his opinion the claim is legal and just, and the auditor may, in his discretion, require further proof.

SEC. 1998. The indemnity granted is the value of the animal as determined by

the appraisers with reference to its diminished value because of being diseased or having been exposed to disease. The indemnity must be paid to the owner upon his application and the presentation of the proofs prescribed herein, and such application must be made within six months after the slaughter of the animal or the claim is barred. The right to indemnity under this article is limited to animals destroyed by reason of the existence of some epizootic disease generally fatal and incurable, such as rinderpest, hoof-and-mouth disease, pleuro-pneumonia, anthrax, or Texas fever among bovines, and glanders among horses, mules, and asses. For the ordinary contagious diseases not in their nature fatal, such as epizootic and influenza in horses, no indemnity must be paid. The right to indemnity does not exist and the payment of such must not be made in the following cases:

1. For animals belonging to the United States.
2. For animals that are brought into the State contrary to the provisions of this article.
3. For animals that are found to be diseased, or that are destroyed because they have been exposed to disease before or at the time of their arrival in the State.
4. When an animal was previously affected by any other disease which from its nature and development was incurable and necessarily fatal.
5. When an owner or person in charge has knowingly or negligently omitted to comply with the provisions of sections 1995 and 1996 of this article.
6. When an owner or claimant at the time of coming into possession of the animal knew it to be diseased or received the notice specified in the first clause of section 1896 of this article.
7. When the animal has been brought into the State within ninety days immediately preceding the outbreak of disease on account of which said animal was killed.

SEC. 1999. The veterinary surgeon receives for his services an annual salary of three thousand dollars. No person must receive the appointment of State veterinary surgeon who is not a graduate in good standing of a recognized college of veterinary surgeons, either in the United States, Canada, or Europe.

SEC. 2000. The appraisers mentioned in this article receive three dollars for each day or part of a day they are actually employed, which must be paid from the State treasury out of the stock-indemnity fund in this article provided upon vouchers which bear the certificate of the justice who summoned them. The justice receives his ordinary fee for issuing a summons, to be paid out of the stock-indemnity fund. The persons called in consultation by the veterinary surgeon each receive three dollars for each day or part of a day they are actually employed and ten cents per mile for distances actually traveled, which sums must be paid from the State treasury out of the stock-indemnity fund upon vouchers certified by the veterinary surgeon. The incidental expenses in causing animals to be slaughtered and their carcasses to be burned and disinfecting infected premises must be paid from the State treasury out of the stock-indemnity fund upon vouchers.

SEC. 2001. The liability for indemnity for animals destroyed and for fees, costs, and expenses incurred under the provisions of this article in any year is limited by, and must in no case exceed, the amount especially designated for the purpose and for that period, by the terms of this article; nor must the veterinary surgeon or anyone else incur any liability under the provisions of this article in excess of the surplus in the stock-indemnity fund hereinafter provided, nor must any act be performed or property taken under the provisions of this article become a charge against the State.

SEC. 2002. The board of county commissioners of each county must at the time of making the annual assessment levy a special tax, not exceeding one-half of one mill on the dollar, upon the assessed value of all cattle, horses, mules, and asses in the county, to be known as the "stock-indemnity fund;" said tax must be collected and paid to the State treasurer in the manner provided by law for the levying, collection, and payment of other State taxes, which fund constitutes the indemnity fund specified by this article to be used in paying for animals destroyed and for fees, costs, and expenses provided under the provisions therefor. It must be used exclusively for that purpose, and must be paid out by the State treasurer as provided in this article.

SEC. 2003. The veterinary surgeon must select the place where stock must be quarantined.

SEC. 2004. The veterinary surgeon has power to appoint from time to time deputies, not exceeding four in number, at any time he can not personally attend to all the duties required by his office, at a salary not to exceed five dollars per day for each day actually employed, to be paid out of the stock-indemnity fund, and must designate the county for which each deputy is to act.

POLITICAL CODE—ARTICLE VI.

SEC. 2022. The State veterinary surgeon, upon the request of the president or secretary of any organized woolgrowers' association in any county in the State, or of any three sheep owners in any county, must appoint a capable person as deputy inspector in such county, who holds his office during the pleasure of the veterinary surgeon and must perform the duties hereinafter prescribed.

SEC. 2023. The deputy inspector must be a resident of the county for which he is appointed. He must before entering upon the duties of his office take the constitutional oath of office.

SEC. 2024. The deputy inspector must inspect all sheep within his county of which he may receive notice, as provided in the next section, and in case he finds the same are not diseased he must make and issue a certificate stating such fact. But if the sheep are diseased, or have been herded upon the range or in corrals which have within the past ninety days previous thereto been used or occupied by any diseased or infected sheep, the regulations for their quarantine, holding, and keeping must at once be made by such deputy. Each deputy inspector so appointed must personally supervise the dipping of every band of scabby sheep within his county and appoint the date for each and every dipping. He has the right to determine and superintend the proportion and mixture of materials and must cause all sheep quarantined to be distinctly marked.

SEC. 2025. Upon receipt of information in writing of any of the facts mentioned in the preceding section, the deputy inspector must immediately cause the diseased sheep, and all sheep running in the same flock with them, to be examined, and if found so diseased to be quarantined and held within a certain limit or place to be defined by him, and such sheep must be held in quarantine until the owner or person in charge has eradicated such scab or infectious disease. The expense of feeding, holding, dipping, marking, and taking care of all sheep quarantined under the provisions of this article must be paid by the owner, agent, or person in charge of such sheep.

SEC. 2026. Whenever the governor by proclamation quarantines sheep for inspection, as provided in the next section, any sheep brought into Montana, the deputy inspector of the county in which such sheep may come must immediately inspect the same, and if he finds that they are infected with scab or any other infectious disease, he must cause the same to be held within a certain limit or place in his said county, to be defined by him, until such disease has been eradicated, as provided in the next preceding section.

SEC. 2027. Whenever the governor has reason to believe that any disease mentioned by this article has become epidemic in certain localities in any other State or Territory, or that conditions exist that render sheep likely to convey disease, he must thereupon by proclamation designate such localities and prohibit the importation from them of any sheep into this State, except under such restrictions as he, after consultation with the veterinary surgeon, may deem proper. Any person who, after publication of such proclamation, knowingly receives in charge any such sheep from any of the prohibited districts and transports or conveys the same to and within the limits of any of the counties of this State, is punishable as provided in Chapter II, Title XV, Part I, of the penal code, and is liable for all damages that may be sustained by any person by reason of the importation or transportation of such prohibited sheep.

SEC. 2028. Upon issuing such proclamation the owners or persons in charge of any sheep being shipped into Montana against which quarantine has been declared must forthwith notify the deputy inspector of the county into which such sheep first came of such arrival, and such owner or person in charge must not allow any sheep so quarantined to pass over or upon any public highway, or upon the ranges occupied by other sheep, or within five miles of any corral in which sheep are usually corralled, until such sheep have first been inspected, and any person failing to comply with the provisions of this section is punishable as provided in Chapter II, Title XV, Part I, of the penal code, and is liable for all damages sustained by any person by reason of the failure to comply with the provisions of this section.

SEC. 2029. In no case must any scabby sheep be allowed to be removed from one point to another within any county, or from one county to another, or any sheep that have within one year been scabby, without a written certificate from the deputy inspector. Such sheep may be transferred and removed with the written consent of all the sheep owners or managers along the route and in the vicinity of the proposed location, except those mentioned in the preceding section. Any person violating the provisions of this section is punishable as prescribed in Chapter II, Title XV, Part I, of the penal code.

SEC. 2030. Upon the arrival of any sheep into this State from any other country, State or Territory, the owner or agent in charge must immediately report to the deputy inspector of the county in which such sheep first came for inspection, and such deputy must immediately inspect the same. If the owner or agent fails to report for inspection, the person so offending is punishable as provided in Chapter II, Title XV, Part I, of the penal code. The expense of such inspection must be borne (?) by the owner, and is a lien upon the sheep, which may be sold to satisfy the lien as provided by law.

SEC. 2031. The deputy inspector in each county receives for his services while necessarily employed in inspection not exceeding $8 per day, which includes all traveling expenses of whatever kind and nature, incurred in going to and from the places where such inspection is had. When a deputy veterinary surgeon as provided in section 2004 of this code is permanently located in a county it is his duty to perform the duties imposed in this article upon deputy inspectors and for his services receives the same compensation as the deputy inspector, which must be paid in the same manner.

SEC. 2032. Whenever any deputy inspector files in the office of the State auditor proper vouchers duly approved by the veterinary surgeon setting forth—

1. The name in full of such deputy inspector.
2. The kind and nature of the services rendered.
3. The particular locality where the work was done.
4. The time when and the length of time employed.
5. The number of sheep inspected and the name of the owner or person in charge.
6. The disease or diseases treated, and the number treated for each disease, and the length of time of such treatment and the result.
7. The amount claimed and the value of such services.

The State auditor must audit the same, and if found correct draw a warrant in favor of such deputy inspector, payable out of any moneys in the "sheep inspector and indemnity fund."

SEC. 2033. Every deputy appointed under the provisions of this article must keep a book to be known as the Inspection Record, in which he must enter and record all his official acts and proceedings. Such record must particularly show the name of the owner of every flock of sheep inspected, when the same was inspected, and the number in each flock, the result of such inspection, the names of the persons to whom certificates have been granted, and when, and all orders and directions made in relation to any matters herein designated.

SEC. 2034. Any person who fails to comply with or disregards any order or direction made by any deputy inspector under the provisions of this article is punishable as provided in Chapter II, Title XV, Part I, of the penal code.

SEC. 2035. It is unlawful for any person to bring into the State any sheep infected with the scab or any other contagious disease. Every person so offending is punishable as provided in Chapter II, Title XV, Part I, of the penal code.

SEC. 2036. Every deputy inspector must on or before the first Monday of August each year report to the State veterinary surgeon in writing, showing from his inspection record particularly the matters therein contained since his last report, and the veterinary surgeon must embody the information thus given in his report to the governor.

SEC. 2037. The veterinary surgeon, if necessary, may appoint more than one deputy inspector in a county, and may define the particular part of the county in which a deputy is to perform his duties, and any deputy appointed under the provisions of this article who, under and by virtue of the powers conferred upon him by reason of such appointment, oppresses, wrongs, or injures any person, is punishable as provided in section 187 of the penal code.

SEC. 2038. The board of county commissioners at the time of the annual levy of taxes must levy a special tax not exceeding one-half of one mill on a dollar, or so much thereof as is necessary, on the assessed value of all sheep in the county, and the money collected from such tax constitutes the "sheep inspector and indemnity fund." Such tax must be collected in the same manner as other taxes and paid into the State treasury as other State taxes are. The money in the "sheep inspector and indemnity fund" must be used in the payment of the salaries and expenses of the deputy sheep inspector as provided in this article and all other expenses arising thereunder except the salary of the State veterinary surgeon. All other salaries and expenses must not be a charge against the State.

SEC. 2047. It is unlawful for the owner or for any person having in charge any horse, mule, ass, sheep or cattle affected with any contagious disease to allow such diseased animal to run on any range, or within any enclosure where such animals

may come in contact with any other animal not so diseased. All animals so affected with contagious disease must be at once removed by the owner thereof, or the person in charge of the same, to some secure inside enclosure, where contact with other animals by reaching over or through the fence of said enclosure will be impossible, or must be strictly herded six miles away from any farm or from any other stock running at large or being herded. Every person who knowingly neglects or refuses to remove or so enclose or herd away from farms or other stock such diseased animals affected with contagious disease, after having received notice of their diseased condition, is punishable as provided in section 435 of the penal code, and is liable for damages to the party injured.

SEC. 733. Any person who removes from one point to another in any of the counties of this State, or from one county to another, any scabby sheep, or any sheep that have been scabby within one year, without the written certificate of the sheep inspector, or the written consent of all the sheep owners or managers along the route and in the vicinity of the proposed location, is punishable by a fine not exceeding one thousand dollars. This section does not apply to scabby sheep imported into the State and against which quarantine has been declared.

SEC. 734. Every person who brings into this State sheep infected with scab or other infectious disease, or any horses, mules, asses, or cattle infected with any contagious disease, is punishable by a fine not exceeding five hundred dollars.

SEC. 735. Every person who fails to comply with or disregards any lawful order or direction made by the State veterinary surgeon, or deputy, or deputy sheep inspector, under the provisions of the political code concerning scab and other contagious diseases among sheep, or to prevent the spread of disease among cattle, is punishable by a fine not exceeding five hundred dollars.

SEC. 736. Every person who, after the publication of the proclamation of the governor of this State prohibiting the importation of diseased sheep into this State, knowingly receives any such sheep from any of the prohibited districts, or transports the same within the limits of the State, is punishable by a fine not exceeding five hundred dollars.

SEC. 737. Every person in charge of sheep being shipped into this State against which quarantine has been declared, as specified in the last preceding section, and fails to notify the deputy inspector of the county in which such sheep are brought, or allows any such sheep to pass over or upon any public highway, or upon the ranges occupied by other sheep, or within five miles of any corral in which sheep are regularly corralled, before such sheep are inspected as provided by law, is punishable by a fine not exceeding five hundred dollars.

SEC. 738. Every person who imports into this State any cattle, horses, mules, or asses after the governor has made proclamation holding in quarantine for the purpose of inspection for contagious or infectious diseases such animals, and allows the same or any of them to leave the place of their first arrival in this State until they have been examined by the State veterinary surgeon and a certificate has been obtained therefrom that such animals are free from disease, or permits any of such animals to run at large or to be removed or to escape before such certificate has been received, is punishable by fine not exceeding five hundred dollars. This section does not apply to any animals driven in harness or under yoke or ridden by their owners into this State.

SEC. 739. Every person who, after the publication of such proclamation, knowingly receives or transports within the limits of this State any animal mentioned in the preceding section before the certificate mentioned therein has been given, is punishable by a fine not exceeding ten thousand dollars.

SEC. 740. Every person who owns or has the custody of any cattle, horses, mules, or asses infected with a contagious disease and fails to immediately report the same to the State veterinary surgeon, or conceals the existence of such disease or attempts so to do, or willfully obstructs or resists said veterinary surgeon in the discharge of his duty as provided by law, or sells, gives away, or uses the meat or milk or removes the skin or any part of such animal, is punishable by a fine not exceeding five hundred dollars.

SEC. 750. It is unlawful for any person having in charge any horse, mule, ass, sheep, hog, or cattle affected with a contagious disease to allow such animal to run on any range or to be within any inclosure where they may come in contact with any other animal not so diseased.

All animals so affected must be immediately removed to an inside inclosure secure from other animals or must be herded six miles away from any farm or ranch or from any other stock running at large or being herded. Every person who neglects or refuses to remove or inclose or herd as aforesaid such diseased animals is guilty of a misdemeanor and liable in damages to the party injured.

NEW JERSEY.

A further supplement to an act entitled "An act concerning contagious and infectious diseases among animals, and to repeal certain acts relating thereto," approved May fourth, one thousand eight hundred and eighty-six.

Be it enacted by the senate and general assembly of the State of New Jersey, That the second section of the act entitled "A supplement to an act entitled 'An act concerning contagious and infectious diseases among animals, and to repeal certain acts relating thereto,'" approved May fourth, one tnousand eight hundred and eighty-six, which supplement was approved May twenty-second, one thousand eight hundred and ninety-four, be, and the same is hereby, amended to read as follows:

SEC. 1. That it shall be lawful for the State tuberculosis commission to employ one of their number as secretary of the commission, and to fix by resolution such compensation for his services as they, in their judgment, may deem reasonable, which compensation shall be paid in monthly installments, out of the appropriation to said commission, by the State treasurer upon the warrant of the State comptroller.

SEC. 2. That when any animal or animals shall be slaughtered by direction of said commission the value of the same shall be ascertained and appraised by three disinterested freeholders, resident in this State, who shall make and sign certificates thereof in the presence of a witness, who shall attest the same; such appraisement shall be made on the basis of the market value of the animal or animals slaughtered, and shall be limited to the sum of one hundred dollars for registered animals and to forty dollars for all others; three-fourths of the valuation so ascertained shall be paid by the State on the presentation of such certificate, with the approval of the said commission indorsed thereon, to the owner or owners: *Provided*, No compensation shall be made for animals considered by the commission to be of no value.

SEC. 3. That whenever the State tuberculosis commission shall have made, or caused to be made, any examination of any animal or herd of animals within this State, and shall have ascertained such animal or herd of animals to be sound and in good health, they shall, upon request from the owner thereof, give to him a certificate in writing, signed by the president and secretary of said commission, certifying to the fact of such examination and of the good health and condition of such animal or herd of animals.

SEC. 4. That the said State tuberculosis commission shall have the power to cooperate with the Bureau of Animal Industry of the United States in any general national system which may be adopted by such Bureau for the prevention of the spread of bovine tuberculosis and its eradication in the United States and its Territories.

SEC. 5. That there shall be appropriated to the said State tuberculosis commission the sum of five thousand dollars for defraying its expenses and for payment of the proportion of the appraised value of slaughtered animals required to be paid out of the treasury of this State, all which payments and expenses shall be made by the treasurer of this State, upon the warrants of the State comptroller; that in cases of emergency the said commission may, with the consent of the governor, comptroller, and treasurer, in addition to the sum of money hereby appropriated, expend such further sums of money for the purposes of this act, not to exceed in the whole the sum of five thousand dollars in any one year.

SEC. 6. That all acts and parts of acts inconsistent with this act be, and the same are hereby, repealed, and that this act shall take effect immediately.

Approved, March 28, 1895.

NEW MEXICO.

AN ACT to amend an act entitled "An act to prevent the introduction of diseased cattle into New Mexico."

Be it enacted by the legislative assembly of the Territory of New Mexico, That section 2 of an act entitled "An act to prevent the introduction of diseased cattle into New Mexico," which became a law on the twenty-eighth day of February, A. D. 1889, be, and the same is hereby, amended so as to hereafter read as follows:

"SEC. 2. A sanitary board consisting of five persons, each of whom shall be a practical raiser and owner of neat cattle in this Territory, one to be appointed in and for each of the following districts: District No. 1, to be formed by the counties of Santa Fe, San Miguel, and Taos; district No. 2, of the counties of Bernalillo, Valencia, Rio Arriba, and San Juan; district No. 3, of the counties of Mora,

Colfax, and Union; district No. 4, of the counties of Dona Ana, Grant, Socorro, and Sierra; district No. 5, of the counties of Lincoln, Chavez, Eddy, and Guadalupe (the limits of each as now constituted) hereby is created, to be known as the 'cattle sanitary board of New Mexico.' The term of office of each member of said board shall be two years from and after his appointment and until his successor shall have been appointed and qualified. Each of the members of said board shall be nominated by the governor of the Territory and appointed by and with the consent of the legislative council. In case of any vacancy in the membership of said board from death, resignation, or otherwise, the governor shall fill such vacancy by appointment, and the appointee shall hold such office only during the unexpired term of the office so becoming vacant."

SEC. 3. That section 4 of said act is hereby amended so as to hereafter read as follows:

"SEC. 4. Said sanitary board is hereby authorized, empowered, and required to prevent the introduction into this Territory or the spreading therein of Texas or splenic fever, contagious pleuro-pneumonia, tuberculosis, or any other contagious or infectious diseases affecting cattle, and to investigate and stamp out any such diseases among cattle wherever the same may be found to exist in this Territory, and to adopt, publish, and enforce such quarantine rules and regulations as may be necessary to carry into effect the provisions of this act and not inconsistent therewith, and to make and enforce such rules and regulations as may be necessary to provide for the inspection of cattle for sale and slaughter."

SEC. 5. That this act shall take effect and be in force from and after its passage.

Approved, February 8, 1895.

NORTH DAKOTA.

AN ACT with reference to driving stock into or through the State.

Be it enacted by the legislative assembly of the State of North Dakota, That all drovers of horses, mules, cattle, or sheep which may hereafter be driven from any other State or Territory of the United States or any foreign country into or through any county or counties of this State shall be plainly branded or marked with one uniform brand or mark.

SEC. 2. All such horses, mules, and cattle shall be so branded with one distinct ranch or road brand of the owner or owners so as to show distinctly, in such place or places as the owner may adopt.

SEC. 3. All such sheep shall be marked distinctly with such mark or device as may be sufficient to distinguish the same readily should they become intermixed or mingled with other flocks of sheep in this State.

SEC. 4. Any such owner or owners, person or persons in charge of such drove of stock which may be driven into or through this State who shall fail to comply with the provisions of this act shall be fined in a sum not less than fifty nor more than three hundred dollars, together with costs of suit.

SEC. 5. It shall be the special duty of the county auditor, sheriff, and any constable of each and every county of this State to enforce the provisions of this act.

SEC. 6. All fines collected under the provisions of this act shall be paid into the general school fund of the county in which judgment thereof is recovered.

SEC. 7. All acts and parts of acts in conflict with this act are hereby repealed.

Approved March 14, 1895.

AN ACT to prevent the spread of contagious, infectious, and epidemic diseases among domestic animals, creating the office of chief State veterinarian, prescribing the duties thereof, and appropriating money for the necessary expenses thereof.

Be it enacted by the legislative assembly of the State of North Dakota, That the professor of veterinary science of the State agricultural college is hereby made chief State veterinarian, who shall serve as such without salary, and who shall, upon entering upon his duties, take an oath to well and truly perform all the duties required of him by law, which said oath shall be taken before any judge of a district court or notary public within the State, and shall be filed with the secretary of state.

SEC. 2. The State shall be divided into seven "veterinarian districts," in each of which there shall be appointed by the governor, by and with the consent of the

senate, one competent veterinarian, who shall be known as the "district veterinarian," who shall hold their office for a term of two years from the date of their appointments, respectively, unless sooner removed for cause, and who, upon entering upon their duties, shall each take an oath to well and truly perform their duties as provided by law, which said oath shall be taken before any judge of the district court or notary public within the district of the State for which they may be appointed, and shall be filed with the secretary of state.

SEC. 3. District No. 1 shall consist of the first judicial district.
District No. 2 shall consist of the second judicial district.
District No. 3 shall consist of the third judicial district.
District No. 4 shall consist of the fourth judicial district.
District No. 5 shall consist of the fifth judicial district.
District No. 6 shall consist of the sixth judicial district.
District No. 7 shall consist of the seventh judicial district.

SEC. 4. The duties of said chief State veterinarian shall be to ascertain by personal examination, or through report from the district veterinarian, in such manner as he shall prescribe, all information that he can obtain regarding the existence of any or all contagious, infectious, and epidemic diseases in the State. He shall also make a complete and permanent record of all reports of the district veterinarians; shall make an examination of all diseased animals or portions of any such that may be forwarded to him by the district veterinarians, and, upon completion of such examination, shall instruct the district veterinarians in such way as he may deem proper in regard to the treatment of similar cases. It shall also be his duty to furnish material, as far as lies in his power, for the diagnosis of contagious diseases and instruction as to its uses. In case that remedies are discovered for the prevention or cure of contagious diseases, such as glanders, tuberculosis, anthrax, hog cholera, foot and mouth disease, and foot rot, it shall be his duty to furnish the district veterinarians or any person or persons he may see fit to appoint the remedies so discovered, with full directions for application. He shall also be empowered to make quarantine regulations and enforce the same after approval and authority by the governor. He shall further prescribe, with the consent of the governor, the rules and regulations necessary to carry out the purposes of this act.

SEC. 5. The duties of said "district veterinarians" shall be as follows:

First. To investigate in person any and all cases of contagious, infectious, and epidemic diseases among cattle, horses, mules, sheep, asses, and other domestic animals within his district of which he may have knowledge, and which may be brought to his notice by any resident, or any other person, in any locality within his said district where such disease may exist, and it shall also be his duty in the absence of specific information to make visits of inspection to any locality within his district where he may have reason to believe that there are contagious or infectious diseases existing among such domestic animals.

Second. To seize and inspect in person at the State line bordering on his district any horses, mules, cattle, asses, sheep, or other domestic animals which may be unloaded temporarily or consigned to any point within his district of the State when the owner, agent, or person in charge thereof shall not upon demand produce certificates of health of such animals satisfactory to him from a duly authorized State or district veterinarian or examiner of the State from which said animals have been shipped.

Third. To examine in person, so often as he may deem reasonable, all pens, enclosures, and cars within the district within which domestic animals may be confined or transported, and to require the owner, agent, or person in charge of all such pens, enclosures, and cars to keep the same in proper sanitary condition.

Fourth. To require in person the owner, agent, or person in charge of all pens, enclosures, or cars, within which domestic animals may be confined or transported, to cleanse, fumigate, and disinfect all pens, enclosures, or cars within which such domestic animals may be confined or transported, within two days after written notice, when, in his opinion, such cleansing, fumigating, and disinfection shall be necessary for the prevention of the spread or outbreak of any contagious or infectious disease among such animals.

Fifth. It shall also be the duty of the district veterinarian in person to seize and inspect all domestic animals coming into and to remain within his district of the State without a certificate of the health of such animals from a duly authorized State or district veterinarian or examiner from the State from which said animals have been shipped and before such animals shall be allowed by the district veterinarian to be transported into and to remain within the State. In addition to such inspection, he shall, in person, require from the owner, agent, or person in charge of such animals an affidavit to the effect that such animals have not

been exposed to any infectious or contagious disease for a period of at least ninety days prior to the making of such affidavit, and in case that the district veterinarian shall have reason to believe that any domestic animals have been exposed to or have contracted any contagious or infectious disease it shall be his duty to seize and inspect such animals, notwithstanding any certificate of their health by any veterinarian or examiner of any other State, and report the same to the chief State veterinarian.

SEC. 6. Whenever any domestic animals are seized and inspected under the provisions of this act by the district veterinarian, while such animals are being transported in cars, on shipboard, or brought into the State in any other manner, the district veterinarian making such seizure and inspections shall require the owner, agent, or person in charge of such animals to pay one-half cent each for the inspection of sheep and twenty-five cents each for all other animals named therein. All money so collected shall be immediately transmitted to the chief State veterinarian, together with a detailed report of the seizure and inspection, and it shall be the duty of the chief State veterinarian to transmit monthly all money collected as inspection fees under the provisions of this act to the State treasurer, who shall receipt to the chief State veterinarian. All such fees shall be paid by the State treasurer into the State treasury general fund: *Provided*, That no inspection shall be made by any district veterinarian of any domestic animals in transit through the State without special instructions from the chief State veterinarian, where the owner, agent, or person in charge thereof shall produce certificates of the health of such animals from a duly authorized veterinarian or examiner from the State from which said animals have been shipped.

SEC. 7. In all cases of contagious or infectious diseases among domestic animals in this State the district veterinarian shall have authority to order the quarantine of the infected premises and animals within his district, and upon such order to immediately report the same to the chief State veterinarian, and in case such disease shall become epidemic in any locality within the State it shall be the duty of the district veterinarian of the district where such epidemic may exist or become known to immediately notify the chief State veterinarian, who shall thereupon have authority to enforce a permanent quarantine and prevent the removal therefrom of any animals of the kind among which said epidemic exists until the district veterinarian of such district locality shall report such animals to be in healthy condition, and upon such report a certificate shall be issued by the chief State veterinarian permitting the removal of the animals that are reported to be healthy. The expense of holding and taking care of all animals quarantined under the provisions of this act shall be paid by the owner, agent, or person in charge of the same.

SEC. 8. In case of any epidemic diseases where premises and animals have been previously quarantined by order of the chief State veterinarian or by the district veterinarian as hereinbefore provided, the district veterinarian is further authorized and empowered, when in his judgment it is necessary, to order that any and all diseased animals shall be quarantined at such places and in such manner as he may direct, and shall be held in such quarantine until released by certificate of the chief State veterinarian, as provided in section 7 of this act, and in case the district veterinarian shall find that any one or more of the animals so quarantined and so diseased that it becomes necessary to destroy the same to prevent the spread of such disease to other animals, he shall at once serve, in person, a written notice of his intention to destroy upon the owner, agent, or person in charge of the animals so quarantined and condemned, and if such owner, agent, or person in charge of such animals feels aggrieved by the decision of the district veterinarian, and shall desire a consultation of veterinarians, notice in writing to that effect must within twenty-four hours thereafter be served upon the district veterinarian issuing the notice, and it is hereby made the duty of the resident district veterinarian to summon two district veterinarians from adjoining districts to appear and assist in diagnosing and pronouncing upon the character of the disease with which said animal or animals are supposed to be infected, and in case all three district veterinarians, or any two of them, declare said disease to be contagious or epidemic in its character, and that such animal or animals should be destroyed to prevent the spread of such disease to other animals, the district veterinarian of the district wherein the animal or animals are located shall immediately slaughter such animal or animals and not otherwise, and shall then make in duplicate a written statement, setting forth distinctly the nature of the disease for which such animals were condemned and destroyed, to be served on each owner thereof, the original of each order to be filed by the district veterinarian with the chief State veterinarian and the duplicate thereof given to the said owner, agent, or person in charge of said condemned animals. It shall be the duty of the owner, agent, or

person in charge of any and all animals slaughtered under the provisions of this act to immediately bury the carcasses of such slaughtered animals in a trench at least six feet in depth and at least four feet beneath the surface of the ground, or burn and consume such carcasses under the direction of the district veterinarian; and it is hereby made the duty of the district veterinarian, in person, to require the owner, agent, or person in charge of such slaughtered animals within his district to immediately bury or burn under his personal supervision the carcasses of such slaughtered animals as herein provided, except in all cases where the cause of death is due to anthrax, when they shall immediately be burned.

SEC. 9. Each district veterinarian shall make a report at the end of every three months, and at such other times as may be required, to the chief State veterinarian of all matters connected with his work, the forms of such reports to be furnished by the chief State veterinarian, and the chief State veterinarian shall transmit to the several boards of county commissioners, as often as he deems necessary, such parts of said reports as may be of general interest to the breeders of live stock, and he shall also give information in writing, as soon as he obtains it, to the various boards of county commissioners of each case of suspicion or fresh outbreak of disease in any locality, its causes, and the measures adopted to check it.

SEC. 10. It shall be the duty of any owner, agent, or person in charge of any cattle, horses, mules, asses, sheep, or other domestic animals, where such owner, agent, or person in charge thereof intends to bring any such animals into this State for distribution, sale, transportation, or permanent location therein, without a certificate of their health from a duly authorized veterinarian or examiner of the State from which such animals are shipped, to give notice in writing to the district veterinarian of the district of the State bordering on the State line from which said animals [are] brought at least three days before such animals are brought into this State beyond the quarantine station at the State line of such district, and it shall be the duty of any person or persons who shall have knowledge or suspect that there is upon his or their premises or upon the public domain any case of contagious, infectious, and epidemic disease among domestic animals to immediately report the same to the district veterinarian of the district wherein such animals or cattle may be; and a failure so to do, or any attempt to conceal the existence of such diseases, or a failure to give notice before passing the quarantine station at the State line of said district as in this section required, or to willfully or maliciously obstruct or resist or disobey any order issued by the chief State veterinarian or the district veterinarian, or in any way interfere in the discharge of their duties as set forth in this act, shall be deemed a misdemeanor, and any person or persons who shall be convicted of any one of the above acts or omissions shall be fined not less than fifty dollars nor more than two thousand dollars for each and every such offense; and upon conviction of such offense a second time shall, in addition to the above-named fine, be imprisoned in the county jail of the county wherein convicted, or as otherwise provided by law, for a term of not less than ninety days nor more than one year.

SEC. 11. The following resolutions shall be observed in all cases of disease covered by this act:

First. It shall be unlawful to sell, give away, or in any manner part with any animal affected with or suspected of being affected with any contagious or infectious disease, and in case of any animal that may be known to have been affected with or exposed to any such disease within one year prior to such disposal, due notice of the fact shall be given in writing to the party receiving the animal.

Second. It shall be unlawful to kill for butcher purposes any such animal, to sell, give, or use any part of it, or its milk, or to remove any part of the skin. A failure to observe these provisions shall be deemed a misdemeanor, and on conviction shall be punished by a fine not less than one hundred dollars nor exceeding two thousand dollars; in addition to the above-named fine, be imprisoned in the county jail for a term of not less than ninety days nor more than one year. It shall be the duty of the owner, agent, or person having in charge any animal infected with or suspected with being infected with any contagious or infectious disease to immediately confine the same in a safe place, isolated from all other animals, and with all necessary restrictions to prevent the dissemination of the disease until the arrival of the district veterinarian within and for the district wherein the same may be at the time. The above regulations shall apply as well to animals in transit through the State as to those resident therein, and the district veterinarian shall have full authority within his district to examine, whether in yard, pasture, or stables, or upon the public domain, all animals passing through the State, within his district, or any part of it, and on detection or suspicion of disease take possession of and treat and dispose of such animals in the same manner as is prescribed for animals resident within this State.

SEC. 12. Each of said district veterinarians shall receive for their services the sum of six hundred dollars per annum. The payment of such salary shall be made from any funds in the State treasury not otherwise appropriated monthly, upon itemized vouchers signed and sworn to by each for his separate district and submitted to the State auditor, who shall draw warrants upon the State treasurer for the amount thereof, if found correct, separately. No person shall be competent under this act to receive the appointment of district veterinarian who is not, at the date of his appointment, a graduate in good standing of a recognized college of veterinary surgeons, or who has not practiced veterinary surgery within this State for at least five years. Before entering upon the discharge of his duties he shall give a bond to the State of North Dakota, with good and sufficient surety, in the sum of two thousand dollars, conditioned for the proper discharge of the same. No constructive mileage shall be paid under this act nor shall the district veterinarian receive any mileage except when called in cases of consultation as hereinbefore provided, when he shall receive actual expenses paid by him.

SEC. 13. The district veterinarians shall select the place or places within their respective districts at which all animals referred to herein shall be quarantined.

SEC. 14. All fines collected under the provisions of this act shall be paid into the general funds of the State.

SEC. 15. It is hereby made the duty of the attorney-general or State's attorney of the respective counties of the veterinary district to prosecute any case complained of by the district veterinarian of such district for prosecution in any justice or district court within the jurisdiction of which any violation of this act may have been had, and on conviction of violation of any of the provisions of this act the court, in addition to the penalties prescribed by law, shall add thereto reasonable attorney's fee as it may be determined just in the premises.

SEC. 16. It shall, in addition to their duties already defined by law, be the duty of all sheep inspectors, and the district veterinarians, who are hereby authorized to appoint such inspectors, shall require all sheep inspectors within their respective districts to report to them in writing, at the end of each calendar month, any knowledge or information such sheep inspectors may possess relative to any diseased sheep which may be within his own or adjacent counties within the veterinarian district wherein said county or adjacent counties may form a part, and the district veterinarian shall report to the chief State veterinarian all the information that he obtains from the reports received from the sheep inspectors; and whenever in the opinion of the district veterinarian any sheep inspector within his district is incompetent to, neglects, or refuses to attend in a proper manner to his duties, the district veterinarian of such district shall take charge of any diseased sheep in such county and dip and treat them in the manner provided for in the law relating to sheep inspectors (chapter 135, general laws of 1885), and when such action shall become necessary he shall report the same to the chief State veterinarian, who shall give such assistance as is in his power; and in addition thereto the district veterinarian shall, when, by reason of incompetency or neglect to perform his duties as such sheep inspector, the district veterinarian is hereby required to remove said inspector and to appoint some competent person in his place. The owner, agent, or person in charge of such sheep shall be required by the district veterinarian, upon his performance of duty as set forth in this section, to pay a fee of five dollars per day, together with the necessary expenses, and said fees shall be a lien upon the sheep inspected, subject to foreclosure the same as chattel mortgages. All fees or moneys collected by the district veterinarian under the provisions of this act shall be remitted, turned over, and receipted for the same as other funds that may pass through their hands as prescribed by section 6 of this act.

SEC. 17. In all the counties of this State where a sheep inspector has been or may be appointed, as provided for by law, the resident sheep therein shall be under the supervision and inspection of such sheep inspector: *Provided, however*, That upon a written application, signed by not less than three sheep owners, the district veterinarian shall visit such county and take such authority or give such directions as in his judgment is necessary.

SEC. 18. The inspector shall receive for his services five dollars per day while necessariiy employed in inspecting, which shall be paid out of the county general fund in the same manner and form as claims against the county are paid: *Provided, however*, That the board of county commissioners shall require such sheep inspector to present an itemized statement of the number of sheep inspected and the number of days actually employed in the performance of his official duties, such statement to be approved by the district veterinarian of the district in which such inspector is engaged.

SEC. 19. In addition to the duties of the chief State veterinarian hereinbefore described, he shall make an annual report to the governor on or before the first

day of December of all matters connected with his work, and in addition thereto may from time to time, as in his judgment seems best, publish bulletins for general distribution, giving information as to the existence of animal diseases in the State, and such suggestions thereto as to care and treatment as he thinks proper.

SEC. 20. For the purpose of carrying out the provisions of this act as herein set forth there shall be appropriated out of any money in the State treasury not otherwise appropriated, an annual sum of thirty-six hundred dollars with which to pay the salaries of the district veterinarians, and the further annual amount of five hundred dollars for stationery, clerk hire, and all traveling and other necessary expenses of the chief State veterinarian.

SEC. 21. In case of any serious outbreak of any contagious, infectious, or epidemic diseases among domestic animals which can not be supervised by the district veterinarian the chief State veterinarian shall at once notify the governor, who shall thereupon appoint a sufficient number of deputies to perform the required duties, at such compensation as he may deem proper, not to exceed five dollars per day for the actual time employed, the same to be paid out of the general fund of the State upon vouchers duly approved by the governor and the chief State veterinarian.

SEC. 22. All acts and parts of acts in conflict with the provisions of this act are hereby repealed.

SEC. 23. An emergency exists in that the existing law is inadequate to prevent the spread of contagious and infectious diseases among domestic animals within the State, and this act should take effect prior to July first, one thousand eight hundred and ninety-five; therefore this act shall take effect and be in force from and after its passage and approval.

Approved March 23, 1895.

PENNSYLVANIA.

AN ACT to establish the State live stock sanitary board of Pennsylvania, and to provide for the control and suppression of dangerous, contagious, and infectious diseases of domestic animals.

Be it enacted, etc., That a board is hereby established, to be known as "the State live stock sanitary board." This board shall consist of the governor of the Commonwealth, the secretary of agriculture, the State dairy and food commissioner, and the State veterinarian, who shall be a competent and qualified person, as provided in the act entitled "An act to create a department of agriculture and define its duties."

SEC. 2. That it shall be the duty of the State live stock sanitary board to protect the health of the domestic animals of the State, to determine and employ the most efficient and practical means for the prevention, suppression, control, or eradication of dangerous, contagious, or infectious diseases among domestic animals, and for this purpose it is hereby authorized and empowered to establish, maintain, enforce, and regulate such quarantine and other measures relating to the movements and care of animals and their products, the disinfection of suspected localities and articles, and the destruction of animals as it may deem necessary, and to adopt from time to time all such regulations as may be necessary and proper for carrying out the purposes of this act: *Provided, however*, That in case of any slowly contagious diseases only suspected or diseased animals shall be quarantined.

SEC. 3. That when it shall be deemed necessary to condemn and kill any animal or animals to prevent the further spread of the disease, and an agreement can not be made with the owners for the value thereof, three appraisers shall be appointed, one by the owner, one by the commission or its authorized agent, and the third by the two so appointed, who shall, under oath or affirmation, appraise the animal or animals, taking into consideration their actual value and condition at the time of appraisement, and such appraised price shall be paid in the same manner as other expenses under this act are provided for: *Provided*, That under such appraisement not more than twenty-five dollars shall be paid for any infected animal of grade or common stock, and not more than fifty dollars for any infected animal of registered stock, nor more than forty dollars for any horse or mule of common or grade stock, and not to exceed fifty per cent of the appraised value of any standard bred, registered, or imported horses.

SEC. 4. That the board or any member thereof, or any of their duly authorized agents, shall at all times have the right to enter any premises, farms, fields, pens, abattoirs, slaughterhouses, buildings, cars, or vessels where any domestic animal is at the time quartered, or wherever the carcass of any may be, for the purpose

of examining it in any way that may be deemed necessary to determine whether they are or were the subjects of any contagious or infectious diseases.

SEC. 5. That any person or persons wilfully violating any of the provisions of this act or any regulation of the State live stock sanitary board, or wilfully interfering with officers appointed under this act, shall be deemed guilty of misdemeanor, and shall upon conviction be punished by a fine not exceeding one hundred dollars or by imprisonment not exceeding one month, or both, at the discretion of the court.

SEC. 6. That the State live stock sanitary board is hereby empowered to appoint and employ such assistants and agents and to purchase such supplies and materials as may be necessary in carrying out the provisions of this act, and the board and the members thereof are hereby empowered to administer oaths or affirmations to the appraisers appointed under this act; and they may order and conduct such examinations into the condition of the live stock of the State in relation to contagious diseases, including the milk supplies of cities, boroughs, and villages, as may seem necessary, and to take proper measures to protect such milk supplies from contamination.

SEC. 7. That all necessary expenses under the provisions of this act shall, after approval in writing by the governor and secretary of agriculture, be paid by the State treasurer upon the warrant of the auditor-general in the manner now provided by law.

SEC. 8. That this act shall take effect June first, one thousand eight hundred and ninety-five, and all acts or parts of acts inconsistent herewith are hereby repealed.

Approved May 21, 1895.

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