THE PATHOLOGY

AND

DIFFERENTIAL DIAGNOSIS

OF

INFECTIOUS DISEASES OF ANIMALS

Prepared for Students and Practitioners of
Veterinary Medicine

By VERANUS ALVA MOORE, B.S., M.D., V.M.D.

Professor of Comparative Pathology, Bacteriology and Meat Inspection, New York State
Veterinary College at Cornell University, and Dean of the College

FOURTH EDITION REVISED AND ENLARGED

WITH 120 ILLUSTRATIONS

NEW YORK
THE MACMILLAN COMPANY
1916

TO DANIEL ELMER SALMON

LARGELY THROUGH WHOSE LABORS THERE WAS
ESTABLISHED IN THE UNITED STATES DEPARTMENT
OF AGRICULTURE THE BUREAU OF ANIMAL INDUSTRY
WHICH HAS MADE POSSIBLE EXTENSIVE INVESTIGA-
TIONS INTO THE NATURE OF EPIZOÖTIC DISEASES IN
AMERICA AND WHO FOR TWENTY-ONE YEARS DIRECTED
THESE INVESTIGATIONS

THIS VOLUME IS DEDICATED.

PREFACE

The purpose of the Fourth Edition of this book is to provide students and practitioners with a text that will give them information on the etiology and morbid anatomy of the specific infectious diseases of animals and the methods available for their diagnosis. It is possible, within the limits of a workable text book, to include but a small part of the available knowledge on each of these diseases. To supplement the necessarily brief account, a list of the more important publications is appended to the description of each. It is believed that these references will give the key to the literature thereby making it possible for the student to familiarize himself with the present knowledge of the subject.

The sanitary significance and the economic importance of the infectious diseases of animals are calling for a better understanding of their nature and more efficient methods for their control. These will be attained only through a more definite and specific knowledge of the etiology and morbid anatomy of each of these maladies.

This edition has been carefully revised, much of it rewritten and numerous additions made. It has been kept, however, within the limits of a text book. Two appendices have been added, one on the requirements for interstate shipment of live stock and the other on the Federal regulations for the veterinary inspection of meat. These may be of much assistance to veterinarians. The diseases not indigenous to, or imported into, this country have been accorded much less space than those existing here. The desire is to emphasize the nature of the diseases our veterinarians are liable to encounter and, at the same time, give the characteristics of the others. The same plan of presenting the subject and of grouping the diseases according to their etiology that was followed in the previous editions has been retained in this.

I desire to express my appreciation of the kind reception accorded the third edition, and for the helpful suggestions received from its readers. It is hoped that this edition will be still more useful to the student and practitioner. My thanks are especially due to Dr. C. P. Fitch for making several of the photographs particularly those of glanders, reproduced in the the text.

V. A. M.

PREFACE TO THE FIRST EDITION

The literature on infection and the etiology and morbid anatomy of infectious diseases of animals is exceedingly rich in the results of new discoveries and important investigations. However, students just beginning this study and following a prescribed curriculum have not the time nor are they prepared to read with profit the detailed records of original research. Such publications seem to be better adapted for those doing advanced or graduate work. Furthermore, many of these publications are out of print and are only available for consultation. For these reasons it is believed that a volume containing the rudiments of the subject will be of use to the student and an aid to the teacher. It is also believed that such a work will be of assistance to practitioners.

In preparing this volume the aim has been to bring together in a concise form the fundamental facts in the pathology of the more common infectious diseases of animals, especially those existing in the United States, with which sanitarians and the practitioners of comparative medicine must contend. To this end the current literature, the reports of the investigations made at various institutions and experiment stations, as well as the standard works on comparative pathology have been freely drawn upon, to all of which full acknowledgment is hereby made.

In order to bring into consideration the clinical value of a knowledge of morbid anatomy a few of the symptoms or antemortem manifestations have been included. It is hoped that this correlation of symptoms and lesions will stimulate a deeper interest in the study of comparative pathology and thus render it of more permanent and practical value for those entering into the practice of veterinary medicine.

In selecting the subject matter care has been taken to avoid, as far as possible, the introduction of results concerning which there are controversies. It has seemed best to deal with those facts about which at the present time there is little or no doubt. After the discussion of each disease a few references to the literature are appended. These are intended simply to bring the attention of the student to a few publications respecting the cause and morbid anatomy, considered in the light of modern etiology, of the disease in question and to a few articles containing the results of original research.

In order not to complicate or unnecessarily expand this text, a knowledge of general pathology and the principles of bacteriology has been taken for granted.

The difficulties involved in the preparation of such a text are both numerous and obvious. The indication of errors or omissions with any other criticisms that would tend to better the volume and increase its efficiency for the student will be thankfully received.

V. A. M.

TABLE OF CONTENTS

CHAPTER I

ETIOLOGY, INFECTION AND SPECIFIC INFECTIOUS DISEASES

CHAPTER II

DISEASES CAUSED BY BACTERIA—GENUS STREPTOCOCCUS

CHAPTER III

DISEASES CAUSED BY BACTERIA—GENUS MICROCOCCUS

CHAPTER IV

DISEASES CAUSED BY BACTERIA—GENUS BACTERIUM

CHAPTER V

DISEASES CAUSED BY BACTERIA—GENUS BACILLUS

CHAPTER VI

DISEASES CAUSED BY HIGHER BACTERIA—GENUS ACTINOMYCES

CHAPTER VII

DISEASES CAUSED BY FUNGI

CHAPTER VIII

DISEASES CAUSED BY PROTOZOA—GENUS SPIROCHAETA

CHAPTER IX
DISEASES CAUSED BY PROTOZOA—GENUS AMEBA

CHAPTER X
DISEASES CAUSED BY PROTOZOA—GENUS PIROPLASMA

CHAPTER XI
DISEASES CAUSED BY PROTOZOA—GENUS TRYPANOSOMA

CHAPTER XII
DISEASES CAUSED BY PROTOZOA—SUB-ORDER MICROSPORIDIA

CHAPTER XIII
INFECTIOUS DISEASES FOR WHICH THE SPECIFIC CAUSE IS NOT DETERMINED

CHAPTER XIV

IMMUNITY AND PROTECTIVE INOCULATION

CHAPTER XV

DISINFECTION

APPENDICES.

ILLUSTRATIONS

FIGURES IN TEXT

A LIST OF REFERENCE BOOKS

BOULEY ET REYNAL—Nouveau Dictionaire pratique de Médecine de Chirurgie et d'Hygiène Vétérinaires.

CADÉAC—Encyclopédie Vétérinaire.

DIECKERHOFF—Lehrbuch der speciellen Pathologie und Therapie für Thierärzte.

ELLENBERGER, SCHÜTZ UND BAUM—Jahresbericht über die Leistungen auf dem Gebiete der Veterinär-Medicin.

FLEMING—A manual of veterinary sanitary science and police.

FLEMING,—Animal plagues; Their history. nature and prevention.

FRIEDBERGER UND FRÖHNER—Lehrbuch der speziellen Pathologie u. Therapie der Haustiere.

GALTIER—Traité des maladies contagieuses et de la police sanitaire des animaux domestiques.

HUTYRA UND MAREK—Spezielle Pathologie und Therapie der Haustiere.

KITT—Lehrbuch der pathologischen Anatomie der Hausthiere.

KOLLE UND WASSERMANN—Handbuch der pathogenen Mikroorganismen.

KOLMER—Infection, immunity and specific therapy.

KRAUS AND LEVADITI—Handbuch der Technik und Methodik der Immunitätsforschung.

LAW—Veterinary Medicine. (Especially Vol. IV.)

LUBARSCH UND OSTERTAG—Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie des Menschen und der Tiere.

NOCARD ET LECLAINCHE—Les maladies microbiennes des animaux.

OSTERTAG—Handbuch der Fleischbeschau.

REYNAL—Traité de la Police Sanitaire des Animaux Domestiques.

SCHNEIDEMÜHL—Lehrbuch der vergleichenden Pathologie und Therapie des Menschen und der Hausthiere.

WALLEY—The four bovine scourges.

For bibliography on all medical subjects, see Index-catalogue of the Library of the Surgeon-General's office.

Annual reports, Special Reports and Bulletins on Animal Diseases issued by the Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Proceedings of the American Veterinary Medical Association.

The Bulletins on Animal Diseases issued by the various State Agricultural Experiment Stations.

CHAPTER I

ETIOLOGY, INFECTION AND SPECIFIC INFECTIOUS DISEASES

Etiology. The development of the sciences of bacteriology and of protozoölogy has shown that a large number of the infectious diseases are the direct result of the invasion of the animal body by certain species of microörganisms. A specific etiology which teaches that for each of the various epizoötics we have a single, definite cause is recognized and accepted by all pathologists. Although there are a number of distinct diseases for which such a specific agent has not been found, the evidence in the very nature of the maladies is conclusive that for each of them such an etiological factor exists.

In studying the pathology of infectious diseases the idea of a definite and adequate cause should be kept in mind. For many years the etiology of these maladies was thought to be unfavorable environment, poor hygiene or insanitary surroundings. While these conditions may favor the spread of disease producing organisms, they cannot cause the malady. The etiology of each of the specific infectious diseases of animals is a definite virus which alone can produce the trouble. For many of the infections and epizoötics the properties of the specific organism are well understood. For certain of the others, such as rinderpest and Foot-and-Mouth Disease, the exact nature of the virus is not understood but its location in the body of the infected animal is known. With the morbid tissues the disease can be produced in susceptible animals and without this definite infection, no matter what the surrounding conditions are, the disease cannot be made to develop. These facts argue against extraneous conditions as exciting causes.

The mystery which formerly surrounded the origin, the course and the disappearance of epizoötics has in a large degree been cleared away; and in its place we are confronted with the problems involved in the life history and the possiblities of invading microörganisms. In fact, during recent years the biological sciences have been brought into actual use by the pathologists. Etiology has become permanently linked to microbiology so that in seeking for the specific

1

cause of an infectious disease we look for some species of micro-
organism which may belong either to the animal or to the vegetable
kingdom. The fact that certain microscopic animals and plants
have become, if they were not in the beginning, parasitic on larger
and higher forms of life has long been recognized; but the idea
came later, that the various infections giving rise to a wide series
of phenomena, known as symptoms and morbid anatomy, were
the direct results of the invasion of the individual with living micro-
scopic plants (bacteria) or animals (protozoa). It is likewise true
that for many general disorders the cause may be found in the condi-
tions of life under which the individual has been forced to exist.
Etiology, therefore, in a broad sense, includes both the infecting
microörganisms that cause the specific infectious diseases and poor
hygiene, insanitary conditions and physical forces that may produce
non-specific morbid changes often sufficient to cause death.

Infection. The term infection is generally understood to mean
the entrance into the animal body, from without, of living micro-
örganisms capable of multiplying within the living tissues and of
producing in consequence of this multiplication a local or a general
diseased condition and perhaps the death of the individual. The
invading microörganisms may belong to any one of three groups of
microscopic life, namely, bacteria, higher fungi, and protozoa.
It is customary and convenient, if not altogether logical, to limit
the term microörganisms to these forms, excluding altogether the
entozoa and other animal parasites, most of which are not micro-
scopic in size.

Intoxication. A diseased condition produced by substances not
capable of reproducing themselves, as, for example, organic or inor-
ganic chemical compounds, is an intoxicative process. In an infec-
tion, the immediate cause of the symptoms and morbid changes
in the tissues affected may be an intoxication due to the action of
the metabolic products (toxins) of the invading microörganisms.
The theories of the mechanical interference of the invading organ-
isms with the normal functions of the body or that they absorb
the nutriment, thus depriving the tissues of necessary food, wait for
demonstration. The results of infection vary in their manifestations.

Wound Infection. If the invading organisms remain at the point
of entrance and produce local tissue changes, the condition is spoken
of as a wound infection.

Bacteriemia. If the invading bacteria multiply in the blood and become widely distributed in the circulation and tissues, the condition is known as septicemia or bacteriemia.

Toxemia. If the infecting bacteria remain at the point of entrance and multiply there, elaborating a toxin which is absorbed and which causes the symptoms and possibly death, the condition is a toxemia.

Sapremia. If there is a febrile condition, resulting from the absorption of the products or ptomains produced by putrefactive bacteria, the condition is called sapremia or septic intoxication.

A Specific, Infectious Disease. If the invading organism is one possessed of definite pathogenic properties, such as the bacterium of anthrax, giving rise to a definite series of symptoms and lesions, the affection is designated a specific, infectious disease.

Through the agency of metastasis, invading microörganisms may be carried from the point of introduction to other parts of the body, where they may become localized, multiply, and give rise to any one of many forms of lesions. It may happen that the point of entrance is so obscure that the resulting morbid changes are not easily traced to an external infection. There are many illustrations of this in comparative pathology, such for example as suppurative cellulitis. For convenience in discussion, infections may be divided into two clinical groups, namely: wound infections and specific infectious diseases, although in certain instances they cannot be entirely separated.

In arriving at a clear understanding of the nature of infections, it is well not to be too closely circumscribed by classifications. It is better to look upon infections as a series of processes going on in the animal world due to the activities of infecting or parasitic microörganisms. In other words, the lesions following an infection are the result of a microbian parasitism.

In the study of the various forms of infection in the lower animals, lesions have been found to contain, apparently as their causative factors, bacteria which suggest at least that certain of the supposed saprophytic organisms may, under certain conditions, become parasitic and cause more or less local or general tissue disturbance. Many lesions seem to be produced by bacteria which are harbored normally upon the skin. When these organisms are introduced by accident into the living tissues they multiply and acquire, if they did not already possess it, the power to produce tissue changes.

We cannot, therefore, dismiss the subject of infection without a
passing consideration of the possible etiological significance, under
certain conditions, of many species of bacteria ordinarily not clas-
sified among the pathogenic forms but which more or less constantly
surround the body.   In the search for the cause of many lesions
supposed from their nature to be infectious, or in applying methods
for their prevention, it is well to take into consideration microörgan-
isms which might possibly be the causative factors and not limit
the search to the already recognized pathogenic species.   Recent
investigations point to the conclusion that domesticated animals
quite frequently suffer as the result of the invasion of bacteria at
present not listed among the pathogenic microörganisms.

Wound Infection. Wound infections are the direct results of
the entrance of certain microörganisms into traumatisms and opera-
tive incisions.   They fall very naturally into two classes:

Infections producing local, acute or more chronic inflammatory
processes usually leading to suppuration and finally healing by
granulation.   This is the form most frequently encountered clini-
cally.   The lesions are those described under acute or chronic inflam-
mation.

Infections which may in the beginning appear like the first or
which may cause so little disturbance as to be unnoticed at the time,
but later result in a local or remotely situated lesion or lesions.
Frequently these are recognized as distinct diseases although in
some cases, such as scirrhous cord, the origin is easily traced to an
operation where infection was possible.

In wound infection, the invading organism is not always of the
same species.   It is because wound infection lesions anatomically
of a similar character may be caused by a number of different bacteria
that they are not classed among the specific infectious diseases.
It has been observed further, that in many lesions two or more
species of bacteria have been associated and probably share the
responsibility for the results.   There is no symptom, or manifesta-
tion of tissue changes, by which one can determine positively the
exciting cause, without a bacteriological examination.

Another class of diseases that are sometimes called wound infec-
tions should be mentioned, namely, those specific diseases, such as
tetanus, where the virus is introduced through a wound either in the
skin or a mucous membrane.

Bacteria causing wound infection. A number of species of bacteria and a few fungi are included among the organisms that are known to produce wound infections in animals. Usually, however, the forms encountered are *Micrococcus pyogenes, Streptococcus pyogenes, Bacillus pyogenes,* * *Bacillus pyogenes Suis* (Grips), *Bacillus pyogenes bovis* (Künnemann), a few other bacilli, more especially those belonging to the colon group, and a few species of the genus pseudomonas. Fungi, especially the actinomyces, are rarely found in acute wound infections. Protozoa are very rare excepting in specific diseases which they cause and where their entrance to the body is by means of the bites of insects. It is true in the domesticated animals as in man, that the pyogenic bacteria are the most common cause of wound infection. In open wounds they are often associated with a number of ordinary saprophytic bacteria. Frequently in closed lesions nonpathogenic organisms are present in
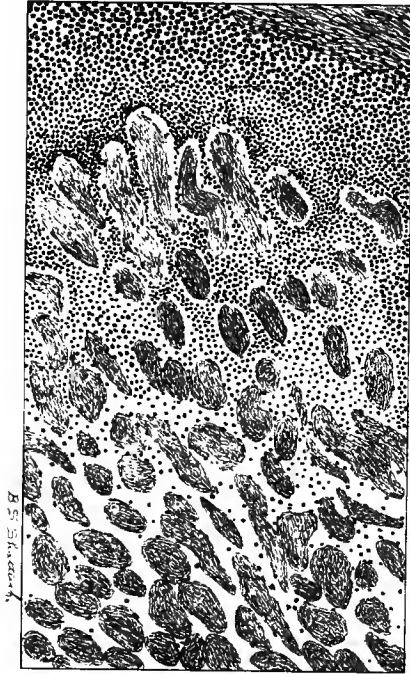
FIG. 1. WOUND INFECTION. A SECTION FROM THE WALL OF AN ABSCESS IN A HORSE SHOWING THE INFILTRATION OF THE INTERMUSCULAR TISSUE WITH PUS CORPUSCLES. DRAWING MADE WITH 1 INCH OCULAR AND 2-3 INCH OBJECTIVE.

---

*Holt considers *B. pyogenes* to be identical with the bacillus of polyarthritis described by von Pods in 1897. He has found it present in a number of inflammations in animals which deviated somewhat from those caused by other pyogenic bacteria. These inflammations are characterized by their chronic and slow course and they are rarely followed by pyemia. The lesions differ from other abscess forming inflammations in that before the necrosis of the tissue occurs, a distinct proliferation of the same takes place which leads to the formation of a tumorlike swelling. According to Grips, "The development of the abscess begins with the formation of small, solid tubercles of a spherical or linseed shape. These tubercles consist of a yellowish, hard tissue and show on section at the beginning, a symmetrical structure. In somewhat larger tubercles one can recognize two zones, a yellow center and a gray-white periphery. At the beginning of the purulent dissolution which sets in at the central part one sees small drops of pus come out under pressure. The abscess consists of a strong indurated capsule and thick viscid pus of greenish or yellowish green color." This organism has been found in a variety of pathological conditions in swine and cattle. In swine it seems to be intimately associated with arthritis. It is suggested by this work that *B. pyogenes* may be an important factor in many morbid conditions that thus far have not been ascribed to any specific organism.

addition to those causing the trouble. It should be noted that in wound infections it is impossible to predict from the general character of the lesions the species of bacteria which are producing them although certain bacteria cause tissue changes that seem to be somewhat peculiar to their species. The infecting organisms can usually be found microscopically without trouble in properly stained cover-glass preparations made from the lesions and most of them can be readily cultivated on ordinary media. *B. pyogenes*, however, requires special media for its development and most characteristic growth.

Morbid anatomy of wound infection. The tissue changes resulting from an infection of the body with various microörganisms belong with those considered in general pathology under the headings of inflammation, degeneration, other retrograde disturbances and regenerative processes. The pathology of ordinary wound infections consists of an acute inflammation usually leading to suppuration, necrosis or gangrene; sometimes the lesions are chronic in nature resulting in a productive inflammation, and again they exhibit the various forms of exudates. In other instances infection results in hemorrhages of varying degrees. Occasionally the lesions may become localized, as in pneumonia, lymphangitis, intestinal ulcers, nephritis, hepatitis and the like. The morbid anatomy of wound infection includes the variety of changes found in different forms of inflammation. Because of this, the emphasis of the pathology of infection rests on the *etiology* and the distribution within the body of the morbid changes. It has been shown that certain infections are slow in bringing about tissue changes and consequently many lesions resulting from wound infection may, when they appear, be considered as distinct maladies.

Fig. 2. MICROCOCCUS PYOGENES. DRAWING FROM A COVER-GLASS PREPARATION OF A BOUILLON CULTURE. HIGHLY MAGNIFIED.

Prevention of infection. In surgical operations, wound infection may be prevented by properly disinfecting the field of operation,

instruments, dressings and the hands of the operator. This is a much more difficult task than it appears. The habit of bacteria of growing down into the hair follicles and sweat glands and beneath the dead epithelial cells on the surface of the skin, renders it necessary to use a disinfectant of much penetrating power in order to disinfect the skin. In case of traumatic infection the wound itself must be disinfected.

A specific infectious disease. A specific infectious disease is the result of the multiplication within the animal*body of a single species of microörganism. The lesion may be local or general, but the cause producing them is always the same. Thus, *Bacterium anthracis* will produce a disease which is called anthrax, no other agent can produce it and no matter how much the lesions may vary in different individuals if they are produced by this species of bacteria the disease is anthrax. It is clear, therefore, that there is no hard and fast line between a simple wound infection and a specific infectious disease, except in the nature of the invading organism. The course of the disease may vary in different individuals and usually it does, especially in different species of animals. If a man becomes accidentally infected by a cut from a knife with which he is making a

FIG. 3. STREPTOCOCCUS PYOGENES. DRAWING MADE FROM A COVER-GLASS PREPARATION FROM A BOUIL-LON CULTURE. HIGHLY MAGNIFIED.

post-mortem on an animal dead from anthrax, the lesion is liable to be restricted to the point of inoculation, and while it is anthrax (malignant pustule) it might be considered as a wound infection. If an accidental inoculation should occur in a guinea pig, the disease would not be recognized as a local lesion, but the animal would develop a general bacteriemia.

As a class, the specific diseases are differentiated from the lesions known clinically as wound infections in a number of ways. The bacteria of the epizoötic diseases do not ordinarily produce wound

*Plants suffer from specific infectious diseases caused by bacteria and fungi, quite as much as animals.

infections following accidental injuries or surgical operations, although there are exceptions. Again, there is usually a difference in the mode of infection. The virus of an epizoötic disease is ordinarily introduced through the digestive or respiratory tract or by means of insects, while in wound infection the virus is introduced, as the term implies, through the injured integument or mucosa.

The differential characters of a specific infectious or epizoötic disease. It is very important not to mistake for an infectious disease some form of body disturbance due to another cause. Animals often suffer from improper food and the conditions of life under which they are compelled to live. It frequently happens that when all of the animals in a given herd are subjected to like conditions of life, a number of them, perhaps all, will manifest simultaneously very similar symptoms and more or less of them die. Such an occurrence often gives rise to the supposition that the cause of death is some specific infection. Deaths from such causes or under such conditions should be carefully distinguished from an epizoötic. In differentiating a non-infectious disorder from a specific disease, it is important, and usually sufficient, to take into account the appended characteristics of an infectious disease.

Cause. An infectious disease is produced by a specific virus. This necessitates as the first requisite an exposure to and an infection with the specific organism. Ordinarily but a few animals in a herd are infected simultaneously.

Period of incubation. The infection must be followed by a certain period of incubation before the development of symptoms. This is the time necessary for the invading microörganisms to become established in the body and to bring about the first symptoms of the disease. According to Vaughn it is the time required for the infecting organism to sensitize the tissues. The incubation period varies in different diseases, and, to a certain degree, in the same disease, according to the mode of infection and the resistance of the individual. Usually, however, the incubation period of a given disease is practically the same for all individuals of the same species when subjected to the same mode of infection. Exceptions may occur.

Lesions. The morbid anatomy of an infectious disease is usually nearly the same in animals suffering in the same outbreak. Each pathogenic organism brings about tissue changes more or less peculiar

to itself. They may vary within rather definite limits. They may also be acute or chronic in nature. They are due to the action upon the tissues of exogenous and endogenous toxins, bacterial proteins, or mechanical blocking of vessels. In many epizoötics, the disease appears in an acute form in the first animals attacked while those attacked later in the course of the outbreak suffer from a chronic or modified form of the affection. In other outbreaks, the first cases are chronic in nature and the later ones acute. It is important to distinguish between the lesions due to the virus and secondary tissue changes that may take place.

Duration. In animals, as in man, most of the infectious diseases are self limiting, but, as a rule, the percentage of fatal cases is larger in epizoötics than in epidemics. It frequently happens that the course of the disease is so changed that an acute case which appears to recover, or at least to pass into the stage of convalescence, becomes chronic or subchronic in nature and eventually terminates in death. The lateness in the development of the modified lesions often causes the nature of the terminal disease to go unrecognized.

Transmission by inoculation. Finally, it is necessary in making a positive diagnosis to find the specific organism, or to prove the transmissibility of the malady from the sick or dead animals to healthy ones. The extent of the spread of the virus through the available channels for its dissemination will also aid in determining the infectious or noninfectious nature of the malady in an outbreak among animals.

In diagnosing an epizoötic disease, investigations have shown that too much reliance can not be placed on the period of incubation, or the morbid anatomy. There are many possibilities, therefore, that an erroneous diagnosis may be made when the clinical and post-mortem evidences of the disease are alone considered. It has also been determined that certain non-infectious disorders often assimilate, in their more general manifestations, the character of infectious maladies. This necessitates care in the differentiation of outbreaks of animal diseases.

The dietary and other non-infectious disorders do not exhibit definite, uniform differential characters excepting perhaps in cases of those caused by a few mineral poisons or by eating certain plants. As examples of these, lead poisoning and the Pictou or Winton disease of horses and cattle caused by eating a ragwort (*Senecio*

jacoboea) may be mentioned. The non-infectious disorders are differentiated from the infectious ones largely by eliminating the characters of the latter and finding, if possible, the causative agent.

The necessity for an early and positive diagnosis in all outbreaks of epizoötic disease, is to assure the enforcement of all possible measures to prevent its further spread. The essential problem for the practitioner in the presence of these diseases, is to restrict the number of cases to the individuals already infected. In order to do this, it is of much importance that modified or chronic cases should not escape detection as there is danger of their spreading the virus to susceptible animals.

Dissemination of infectious diseases. Although the discussion of the means by which each of the various diseases are disseminated will be found under the descriptions of the individual affections, it is important to consider the general ways and means by which these different vital, causative factors are spread from an infected individual to a non-infected one in the same herd and also from one herd to another. Each virus is dependent for its perpetuation upon its escape from its host (sick or dead) to a susceptible one. As these organisms are without power of their own for such migration, they are dependent upon other forces and carriers to take them. In explaining their spread, we must know how they escape from the infected individual, how they are carried from one individual to another and how they gain entrance to the bodies of healthy individuals.

Escape of virus from infected animals. The infected organisms escape from the living body of the infected animal either with (a) the excreta, (b) the external discharge of ulcers or abscesses or both, and (c) the blood drawn by sucking insects. After the death of the host they can escape only after the disintegration of the dead body or by its being consumed as food by other animals or birds. The bacteria of several diseases may pass through the digestive tract of such animals uninjured.

Dissemination of infecting organisms after they leave the body. Pathogenic bacteria are spread, after they escape from the body, in a number of ways. The following are the more common:

They are carried on the hands, shoes or clothing of attendants, and on farm implements, such as shovels and hoes, in crates and cars that have contained the infected animals.

They are carried in streams receiving the excreta or disintegrating dead bodies of the infected animals.

They are scattered with the excreta of birds that feed upon dead carcasses. Other animals, such as dogs and foxes, are also charged with the scattering of the virus by the same method.

The virus may be carried from one herd or flock to another (a) by animals suffering with chronic forms of the disease; (b) by those already infected but in which the symptoms have not yet appeared; and (c) by the "carriers" of the virus, after recovery has taken place.

The pathogenic protozoa are transferred from infected to non-infected individuals by means of insects. They are carried from one locality to another in infected animals or intermediate hosts.

Channels of infection. All infecting agents (bacteria, fungi and protozoa) must gain entrance to the tissues of the healthy animal before they can produce their respective disease. It is possible for a healthy animal to come in close contact with a sick one and yet not become actually infected. Such an exposure does not necessarily mean that infection has occurred. Infecting agents gain acecss to the living tissues by being brought by some agency into the digestive, respiratory or generative tracts or by being introduced directly into the tissues. Most viruses are brought into the body with the food, infected dust-laden air, by copulation or by inoculations. The common channels of infection, therefore, are:

The digestive tract. Bacteria brought into the intestinal tract with food or water may pass through the mucosa of the intestine thereby gaining entrance into the walls of the intestine, or through the lymphatics or blood-vessels obtain passage to other organs or tissues.

Respiratory tract. Infected, dust-laden air may be taken into the lungs where the infecting organism may either multiply or be taken by means of metastasis to other parts of the body.

Generative tract. The viruses of certain diseases may be transmitted from one individual to another by copulation.

By inoculation. Inoculation often takes place through abrasions or wounds produced accidentally on the skin or mucous membrane. In these cases the infecting organisms may be already lodged on the skin or membrane at the point of injury or they may be brought on the object causing the wound.

Insects. Certain insects are carriers of pathogenic bacteria. The most important are, perhaps, the mosquito that carries the plasmodium of malaria and which is an intermediate host for the parasite, and the cattle tick that becomes infected with the piroplasma of Texas fever. In some stage, the tick transmits the parasite to its offspring which in turn infect susceptible cattle upon which they crawl. Many diseases, especially those caused by protozoa, are transmitted through the agencies of insects. It is also true that certain pathogenic bacteria are carried by flies and other insects.

Transmission of the virus from the parent to the fetus. Occasionally the young of diseased parents are born infected with the disease with which one or both of its parents were suffering. In these cases the specific organisms were transmitted either from the sire at the time of coition, or later to the fetus in the uterus from the dam. It is important not to confuse these rare cases with those in which the offspring are born uninfected but subsequently contract the disease. Many of the so-called hereditary diseases are the result of post-natal infection.

The growing knowledge of infectious diseases is tending to the conclusion that the virus of each of them has, through the assistance of natural agencies, some one or at most very few, common, so-called natural means of gaining entrance to the tissues of a new host. While there may be extraneous ways by which infection takes place they are, in most instances, relatively unimportant. It is essential, therefore, that the more definite manner of dissemination of each of the specific diseases of animals be thoroughly understood. This involves knowledge of when in the course of the disease the virus escapes from the infected animal, what becomes of it after it leaves its host and how it gains entrance into the uninfected.

Diagnosis of a specific infectious disease. The accurate diagnosis of a specific infectious disease is made by taking into account one or more of the following:

The symptoms.

The lesions or morbid anatomy.

The specific cause.

Specific reactions.

Symptoms. The value of symptoms in making a positive diagnosis varies with the disease and often with the individual case. While each disease exhibits a somewhat constant chain of symptoms it

is also true that there is a striking similarity between the symptoms exhibited in many diseases. The principle was laid down by French* "that a particular symptom attracts special notice in a given case, and that the diagnosis has to be established by differentiating between the various diseases to which this symptom may be due." As a rule it is difficult to make a positive diagnosis of a specific, infectious disease from the symptoms alone.

Tissue changes. · The changes produced in the tissues by a specific microörganism are usually constant in their nature. They represent the result of the reaction of the body tissues to the particular invading virus. In the invasion of the animal body certain viruses seem to select some definite tissue or organs in which to locate. On the other hand, the location of the lesions due to other organisms is largely determined by the channel through which they gained entrance and the place where they first became lodged. The diagnostic value of the specific lesions, or other tissue changes, therefore, may be relied upon in a large number of cases with greater certainty than the symptoms. In some diseases, like rabies, the characteristic tissue changes are microscopic and cannot be determined from the gross examination of the organs. It is very important in making a diagnosis from the lesions, that the specific tissue changes be present and that they be carefully differentiated from secondary ones that often occur in such diseases.

In making a diagnosis from the morbid tissues it is necessary that sufficient time should have elapsed for the changes to have taken place. If the microörganism should be very virulent, death may take place before the usual changes have occurred. Likewise, if an animal is killed in the earlier stages of the disease, the characteristic lesions are very liable not to be found because they have not as yet had time to develop.

The specific cause. The positive diagnosis is made when the microörganism that caused the disease is demonstrated in the morbid tissues. In some diseases this can be done by very simple bacteriological methods. With other affections it requires very careful and special methods to isolate, cultivate or even to demonstrate the cause. There are several epizoötic diseases for which a specific organism has not as yet been found. In order to use the cause, for diagnostic

*Index of Differential Diagnosis of Main Symptoms by various writers edited by Herbert French, M.A., M.D., Wm. Wood & Co., New York, 1913.

purposes, it is necessary to know how it can be detected and the best method for its cultivation or demonstration.

Specific reactions. In the study of immunity it has been found that animals suffering from certain infectious diseases will react to the injection into the body of the specific protein of the organism with which they are infected. Thus, if mallein is injected into a horse suffering from glanders a definite reaction will occur which can be considered specific for glanders. There are also certain substances given off by the tissues into its blood that have a specific affinity for the antigen or species of organisms that cause the infection. There are a number of these so-called specific reactions employed in diagnosis. They are reactions to mallein, tuberculin and other like substances, the agglutination, the precipitation, complement fixation and others. These are all quite largely employed in diagnosis. When a reaction takes place with any of these tests it is generally considered positive evidence of infection.

The limitations of the specific tests are not determined, but experience has shown that with certain of them, especially tuberculin, a failure to react is not conclusive evidence of the absence of infection while a reaction is a very definite indication that the animal is tuberculous. The sera tests seem to be more accurate in negative cases. The methods employed in making a diagnosis will be described in connection with the diseases to which they apply.

Control. The knowledge necessary for the control of specific infectious diseases is one of the most important acquisitions of the veterinarian. It is obtained by a careful study of the etiology and morbid anatomy of these affections. The control is accomplished by following out definite procedures along one or both of the following lines, namely: (*1*) by preventing the spread of the virus or (*2*) by immunizing susceptible animals against it.

By checking the spread of the virus. This involves an intimate knowledge of the life cycle of the virus for each of the diseases. It requires that one should know, (*1*) when in the course of the disease the virus is eliminated and the channels through which it escapes. (*2*) What happens to the virus after it leaves the body and how long it will resist the action of external influences such as drying and sunlight. (*3*) Through what channels it gains entrance to the body of the uninfected animal. When the facts regarding these points are ascertained it is not difficult to determine what

should be done to prevent the virus from spreading from the animals in one herd to those in another, or from one individual to another in the same herd. The extermination of the virus, or its restriction to known places, is dependent upon a knowledge of the ability of the specific microörganism to resist destructive influences or to be carried from one place to another. It also involves a knowledge of the tissue changes and the protective forces of the individuals invaded. These determine under what conditions the virus can escape and when infected individuals are immediately dangerous. The specific morbid anatomy of the infectious diseases is essential for an understanding of this phase of the subject.

Immunization. Immunization of animals against either sporadic or epizoötic infectious diseases has been found to be practicable with certain diseases such as "black leg," hog cholera and a few others. This method is valuable when it is possible to establish an artificial immunity. The method of vaccination, however, cannot be employed in the control of infectious diseases except with those affections where efficient immunizing methods are known. The danger of the method is, that where it is employed precautions against infection are usually minimized and consequently if the protection is not complete it gives a false security. To successfully employ immunizing processes for the control of infectious diseases it is essential to have a thorough knowledge of the principles of immunity, its varieties and methods for its production.

Cause for variations in the course of an infectious disease. It is recognized that there is much variation in the course of infectious diseases in different epizoötics and often marked individual variations occur in the same outbreak. In explaining this interesting phenomenon, it is important to take into account the question of individual resistance or immunity,—partial or more complete. It was found in case of certain diseases that when an individual was partially immunized and then infected the lesions were very much modified. The teachings of a specific etiology point to this phenomenon as a result of certain biological or vital differences existing either in the invading organism, or in the host, or possibly in both. The results of the investigations already made along this line suggest as a probable explanation, that the course of the disease varies on the one side with the immunity possessed by or resistance of the host and on the

other with the degree of virulence of the infecting microörganism. This has been expressed in the formula

$$D = \frac{V}{R}$$

in which D = the disease, V = the virulence of the infecting organism and R = the resistance of the host or the individual attacked. As V or R change the disease is modified. For example, rabbits that are partially immunized against swine plague bacteria, when inoculated with a virulent culture of that organism, will live for several days and perhaps weeks, and then die of peritonitis, pleuritis or extensive pus formations, instead of perishing within twenty-four hours with bacteriemia as they would if they had not been partially protected against this organism. In chronic cases of swine plague, as found in certain outbreaks, the bacteria are often attenuated so that when inoculated into susceptible rabbits the result is the same as when the rabbits protected by partial immunization were inoculated with virulent cultures. The above simple formula which was worked out and demonstrated for certain swine diseases seems to apply to infectious diseases generally.

Classification or grouping of the infectious diseases. It will be found in the study of the morbid anatòmy of the various specific maladies that the lesions in a given disease vary in different species and to a marked degree in individuals of the same species. This fact precludes the possibility of classifying or arranging them after their morbid anatomy. If the infectious diseases are to be considered as parasitisms, as they appear to be, the logical method of classifying them for purposes of study would seem to be the one suggested by their etiology, namely, that they shall be placed in groups corresponding to the classification of their etiological factors. Thus a single lesion found in the glands of the head, in the lungs, in the liver, in the mesenteric glands, in the skin, joints, generative organs or elsewhere in the body would be called tuberculous if the bacterium of tuberculosis could be demonstrated to be its cause. The same conclusion would be maintained regardless of the character of the lesion, whether it consisted of a clump of cpitheliod cells, purulent, caseous or calcareous tissue. These facts are enough to indicate that the most direct method of arranging these diseases for purposes of study is in groups composed of like generic etiological factors.

MIGULA'S CLASSIFICATION

LOWER BACTERIA

COCCACEÆ

Streptococcus.......   . .   . .

Micrococcus .. ..

Sarcina ....

Planococcus ....

Planosarcina ....…......

BACTERIACEÆ

Bacterium.......................................….....

Bacillus...

Pseudomonas....................….........…..... ... .

SPIRILLACEÆ

Spirosoma ....…................... ....

Microspira... ... .

Spirillum ....

Spirochaeta ... .
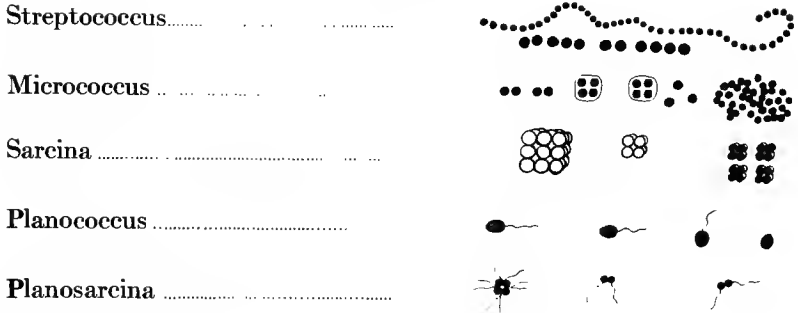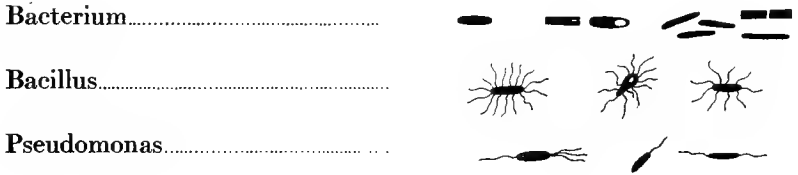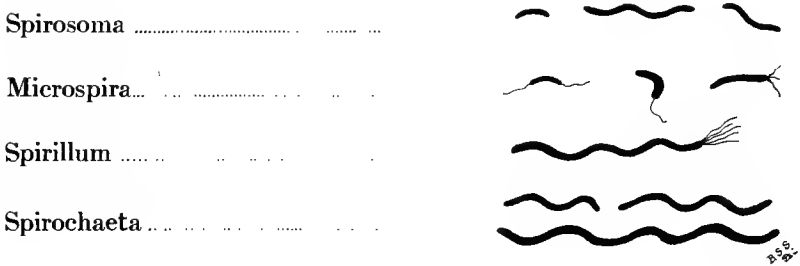
Most of the known specific causes of the infectious diseases of animals are bacteria. It is necessary, therefore, in carrying out the above plan to choose from among the numerous classifications of bacteria one to be followed in grouping the diseases according to the genera of bacteria producing them. Of these the one by Migula seems to be the simplest and best adapted for this purpose and consequently it is selected. The only radical difference between this classification and the older ones, so far as pathogenic forms are concerned, rests in the fact that the old genus *Bacterium* is revived, but with a new meaning. All rod-shaped, non-motile bacteria are placed in this genus. This causes a change of the generic name from *Bacillus* to *Bacterium* of a number of pathogenic bacteria, such as those of tuberculosis, anthrax, swine plague and a few others.

The same system should be followed with the diseases caused by fungi and protozoa. There are as yet so few species of fungi known to produce diseases in animals that their classification is not essential in discussing the few morbid conditions which they cause. The classification of protozoa by Calkins as found in his excellent work, "The Protozoa," is observed for the grouping of the protozoan diseases.

Migula's classification of bacteria. The genera of the five families are included.

<div align="center">FAMILIES</div>

I. Cells globose in a free state, not elongating in any direction before division into 1, 2, or 3 planes . . . . 1. COCCACEÆ.
II. Cells cylindrical, longer or shorter, and dividing in only 1 plane, and elongating to twice the normal length before the division.
(1) Cells straight, rod-shaped, without sheath, non-motile, or motile by means of flagella . . . . 2. BACTERIACEÆ.
(2) Cells crooked without sheath . . . . 3. SPIRILLACEÆ.
(3) Cells enclosed in a sheath . . . . . . 4. CHLAMYDOBACTERIACEÆ.
(4) Cells destitute of a sheath, united into threads, motile by means of an undulating membrane . . . . 5. BEGGIATOACEÆ.
    COCCACEÆ, cells without organs of motion.
        a. Division in 1 plane . . . . *Streptococcus.*
        b. Division in 2 planes . . . . *Micrococcus.*
        c. Division in 3 planes . . . . *Sarcina.*
    Cells with organs of motion.
        a. Division in 2 planes . . . . *Planococcus.*
        b. Division in 3 planes . . . . *Planosarcina.*
    BACTERIACEÆ, cells without organs of motion . . . . . . *Bacterium.*

Cells with organs of motion (flagella).
    a.   Flagella distributed over the whole body......*Bacillus.*
    b.   Flagella polar...........................*Pseudomonas.*
3.   SPIRILLACEÆ.   Cells rigid, not snakelike or flexuous.
    a.   Cells without organs of motion............*Spirosoma.*
    b.   Cells with organs of motion (flagella).
          1.   Cells with one, very rarely 2–3 polar flagella
                                      Microspira.
          2.   Cells of polar flagella, in tufts of from 5–20
                                    Spirillum.
Cells flexuous............ ....*Spirochæta.*
4.   CHLAMYDOBACTERIACEÆ.   Cell contents without granules of sulphur.
    A.   Cell threads unbranched.
          1.   Cell division always only in 1 plane....*Streptothrix.*
          2.   Cell division in 3 planes previous to the formation of conidia.
                a.   Cells surrounded by a very delicate, scarcely visible sheath (marine)................. *Phragmidiothrix.*
                b.   Sheath clearly visible (in fresh water)...... *Crenothrix.*
    B.   Cell threads branched (pseudo-branches)... ... *Cladothrix.*
Cell contents containing sulphur granules .... *Thiothrix.*
5.   BEGGIATOACEÆ.   Only one genus known (*Beggiatoa* Trev.), which is scarcely separable from *Oscillaria.* Character as given under the family.

Lesions due to infections that have been attributed to one or more species of microörganisms. It has already been stated that the lesions following wound infections may be brought about by a variety of bacteria and also that certain of these disease processes are sometimes recognized as distinct maladies. There are a number of affections which belong to this class. From some of these, such as botryomycosis, a supposedly specific organism has been isolated and described. A number of workers, however, have found that other bacteria may produce apparently the same morbid changes. It seems wise to consider briefly the pathology of the more important of these infections. It should be stated, however, that the amount of work that has been done on these subjects is not sufficient to preclude the possibility of a specific etiological factor for each. The association with the lesions of certain species of bacteria suggests that possibly they are the result of a number of organisms acting either singly or together as mixed infections.

Botryomycosis. This name was given to a variety of lesions found more commonly in the horse, but occurring also in cattle,

swine and other animals.   The thickened spermatic cord (scirrhous
cord) which sometimes follows castration is the most common form.
Practitioners often designate as botryomycosis certain closed abces-
ses occurring in the subcutaneous or intermuscular tissue.   Abscesses
and nodules found in the internal organs have been included under
this caption.   The recent literature contains numerous references
to lesions belonging to this group of morbid changes.   Several
investigators have isolated from the lesions a microörganism which
appeared to stand in a causative relation to them.   It was first
described in 1870 as *Zoögloea pulmonis equi* by Bollinger who found
it in the nodules in the lungs of a horse.   Later he renamed it *Botryoco-
ccus ascoformans*.   Rivolta designated it *Discomyces equi*.   Rabe
proposed the name *Micrococcus botryogenes* and Johne has called it
M. ascoformans.   The results of other investigations throw some
doubt upon the specific nature of these lesions.   Kitt, Hell, deJong,
Gay and others have found in the lesions micrococci which do not
differ from *M. pyogenes*.   The writer has failed to find *M. ascoformans*
but has isolated in its stead pyogenic micrococci and streptococci.
It is possible, however, that *Botryococcus ascoformans* was present.
In one very interesting case of thickened cord, he found masses of
a fungus resembling somewhat that of actinomyces within the pockets
of spongy tissue sprinkled throughout the thickened fibrous cord.
Bacteria were not found in this case.

   In the closed abcesses in the connective tissue pyogenic bacteria
have been found excepting in certain cases of long standing where
the cultures gave negative results.   Investigations of the bacterial
flora of the skin of the horse show that pyogenic bacteria are fre-
quently present in the deeper layers of the epidermis.   With the
possibility of infection from the integument plus all the other chances
of having pyogenic and other bacteria introduced into the wound
there are abundant opportunities for infection with a variety of
species.   The evidence at hand points to the conclusion that botry-
omycosis especially the form known as scirrhous cord is the result
of an infection with one or more species of microörganisms.   It is
very desirable that further investigations be made on this subject.

   Omphalo-phlebitis or navel-ill.   This affection consists of suppura-
tive lesions in young animals caused by pyogenic bacteria.   In the
colt the lesions are most often localized in the joints of the limbs.
In other animals they are quite as likely to be located elsewhere in

the body. In some cases the morbid changes consist of subcutaneous and intermuscular suppurative cellulitis.

The infection takes place through the umbilicus. As the cord is broken in the field or stable many species of bacteria may gain access to the exposed and freshly severed surface. In colts a streptococcus seems to be the most common species of bacteria capable of invading the body and producing the joint abscesses. In the lamb, a variety of the colon bacillus has been associated, apparently as the etiological factor, with the subcutaneous cellulitis. The lesions resulting from navel infection illustrate in a most excellent manner the extent to which certain pyogenic bacteria, gaining access to the body, may extend by metastasis to places remote from their entrance and produce diseased foci. In some cases the lesions are said to be of a different nature. There is swelling and necrosis of the navel and surrounding tissues caused by *B. necrophorus*. This organism has also been found in the joint lesions by Mettam.

In the case of navel-ill, the umbilical vein usually contains a number of bacteria. Moore found that in colts very few if any of the streptococci producing the joint lesions could be found in the parenchymatous organs. Occasionally one or two of many tubes of media inoculated from the liver would develop cultures of the infecting organism. In this disease, where many species of bacteria come into competition, one is impressed with the fact that seemingly very few of the extraneous bacteria are capable of gaining entrance into the general circulation or, at least, are possessed of vital powers sufficient to resist the destructive forces of the living animal body.

The lesions consist of some form of inflammation. In the joints it is usually the suppurative type (pyemicarthritis). In the intermuscular tissue it may be suppurative or of a serous or fibrinous nature. Bacteriemia may occur.

White scours or diarrhea in calves. This is a disease affecting calves from a few hours to as many days old, with a mortality ranging from 50 to 90 per cent. The investigations which have been made in this country have suggested that it is often due to umbilical infection with certain forms of the colon bacillus. Other species such as *B. aerogenes*, *Ps. pyocyaneus*, *B. proteus*, varieties of the colon group and *Bact. bovisepticum* have been described as etiological factors in this disease. It has been suggested, and Williams has some data to confirm it, that the Bang abortion organism

is the cause of a specific white scours and pneumonia of calves. It is also very likely that infection occurs through ingestion. In these investigations characteristic lesions were not found.

Nocard reported the results of his investigation of apparently a similar disease of calves in Ireland. He found that they usually died during the first week. In the more chronic cases, lung lesions were found. His inquiries tend to show that this is primarily due to a wound infection. He states in his report concerning the nature of this disease that it usually lasts from 3 to 6 days and is characterized by an intense intestinal discharge. The calves lose flesh rapidly, their flanks are hollow, abdomen retracted, back arched, eyes sunken, and hair dull; they make violent expulsive efforts, the nose is hot and dry with slight discharge of mucus, and the temperature is elevated.

In other cases, but not so commonly, the symptoms are less severe and recovery seems to take place; but most of the calves die several weeks later with pulmonary lesions.

Nocard stated that it is not rare to see, in these cases, the discharges mixed with blood in various quantities. In more chronic forms one may observe acute, multiple and very painful arthritis.

The lesions found by Nocard at the autopsy varied according to the course of the disease. Usually the umbilicus was large and the umbilical blood-vessels had indurated walls, and contained blood clots which were soft and purulent. Bloody extravasations were observed, sometimes very extensive, along the umbilical vessels and the urachus, extending sometimes to the posterior third of the bladder.

In rapidly progressing cases, lesions of true hemorrhagic septicemia were found. All the organs were congested; their surfaces were covered with petechiae, ecchymoses or sub-serous blood infiltrations; the capillary network of the peritoneum, the omentum, the pleura and the pericardium were very much injected.

Nocard described the lungs and articular lesions as follows:

"The lungs are rarely entirely healthy; most often they present here and there little diffuse centers of catarrhal pneumonia, nodular broncho-pneumonia or only atelectasis. The lesions are much more constant, extensive and dense when the animals have resisted longer; they represent then the transition between the simple atelectasis of the beginning and the suppurative lesion of lung disease.

"Articular lesions, when they exist, are very interesting. The periarticular tissues are infiltrated with yellowish and somewhat gelatinous liquid; the synovial serous membrane is covered with a rich vascular injection, which seems to extend to the borders of the cartilages of the articular surfaces; the culs-de-sac of the synovials are distended by a great quantity of thick and limpid synovia, strongly yellow or red-

dish in color, and in which more or less dense and abundant clots of fibrinous exudate are floating. When the lesion is older, instead of synovia, there are thick, dense, and firm fibrinous exudates, which fill the culs-de-sac of the serous membrane and are infiltrated between the articular surfaces. In these cases the lesion resembles exactly those of peripneumonic arthritis of sucking calves."

He found a microörganism (Pasteurella) in the organs and blood of the calves, with which he was able to produce the disease. After convincing himself that he had found the cause, he sought for the source of infection, which he found in the *umbilicus*. He advanced three suppositions concerning the time and mode of entrance: 1. intra-uterine, 2. vaginal, 3. after delivery when the calf drops on the ground or floor and when the ruptured cord comes in contact with the fecal matter or dust of the stable. The latter he believes to be the actual method.

He states that "white scours is ordinarily the result of *umbilical infection* which takes place at the time of birth, through the wound made by the rupture of the cord."

The disease described by Nocard does not seem to differ in many respects from the diarrhea in young calves in this country. Lesage and Delmar have described it in France. Ward and Fisher tested Nocard's method of treatment by properly disinfecting the ruptured cord, with quite satisfactory results. A number of experiences in the thorough washing of the cows and disinfecting the teats before parturition have been successful in checking this trouble. The lung complications do not always occur.

The bacteriological examination of young calves that died of this trouble showed that their blood and organs were teeming with a variety of *B. coli*. We have not found *Bact. bovisepticum* (Pasteurella) in any of our cases. This suggests the possibility of umbilical infection with members of other groups of bacteria. The important findings of Nocard should stimulate further investigations into this important disease. The remedy which he recommends, and which has given good results, is simply one of prevention. To what extent infection takes place through the digestive tract has not been determined but it is believed to be considerable. It is not impossible that intra-uterine infection from the dam sometimes occurs. Williams has found the abortion bacillus in the pneumonic lungs of the new-born calf.

Titze and Weichel suggest the possibility that the cause of white scours is an ultra-visible virus. In their study of this disease they found in the tissues *B. coli commune, Pseudocoli bacilli, Paracoli*

bacilli, Bacillus enteritidis, Bacillus paratyphosus A and B. and
Bacillus typhi.

Infectious suppurative cellulitis of the limbs. Cattle and sheep
occasionally suffer from an inflammatory condition of the sub-
cutaneous tissues, of the lower extremities. Frequently the morbid
process extends beneath the hoof, causing it to slough or to undergo
resulting disintegration. When this condition exists, the affection
is frequently called "foot rot." If the inflammatory process attacks
the skin also, the condition is designated erysipelas. If it becomes
circumscribed, resulting in a local suppuration, an abscess or an ulcer
may be the result. The investigations which have been made
concerning the cause of these lesions point to an infection, probably
through some slight abrasion of the skin near the hoof. Thus far,
the results show streptococci* to be the etiological factors in the
majority of these cases. It frequently happens that a number of
animals subjected to the same conditions are attacked at the same
time, giving rise to a condition resembling an epizoötic. In cattle
the lesions that have been described were, within certain limits,
uniform in all of the affected animals. Usually but one foot or leg
was attacked, although there were numerous exceptions. The first
symptom noticed was a swelling, which usually appeared in the lower
part of the leg, most often in the pastern. In some animals the
swelling was restricted to a small area, but often it extended up
the leg to and even above the knee or hock joint. There was evi-
dence of pain. As the inflammatory process continued the sub-
cutaneous tissue became indurated, the skin thick and dry, and later
it would crack, usually but not always, below the dew claws, and a
thick creamy pus would be discharged. After discharging, the swell-
ing subsided and the normal condition was rapidly restored. The
time necessary for the suppurative process and recovery to take
place varied in different animals, but as a rule from ten to fifteen days
were required. The exceptions were largely in those cases where
the inflammatory process extended down to the coronary cushion.
In these there was more or less sloughing of the hoof. These cases
were the most serious.

---

*Lucet has reported the results of bacteriological examinations of fifty-two abscesses
in cattle. From nine of these steptococci were obtained in pure culture, and in ten
cases they were associated with other bacteria.—*Annales de l' Institut Pasteur. Vol.
VII (1893), p. 324 .

In the so-called foot rot of sheep, we have, in the cases which have come to our notice, conditions similar to those found in the cattle that were suffering from suppurative cellulitis. These cases are to be differentiated from necrosis of the skin above the coronary band due to *Bacillus necrophorus*. (See "Lip and leg" ulceration).

Fistulous withers and poll-evil. Recent investigations indicate that these very common and troublesome local diseases are either directly or indirectly the result of bacterial invasion. This conclusion is drawn from the fact that the bacteriological examinations made from the pus and from recent lesions in these affections invariably reveal the presence of streptococci or micrococci, or both. Gay found a streptococcus in each of seven cases of common fistulous withers and in two cases of poll-evil. It was invariably associated with a micrococcus. He found in five cases of deep seated shoulder abscesses *M. pyogenes* only. It is instructive to note, that bacteria closely resembling this organism have frequently been found in the deeper layers of the skin of the horse. The mechanical injuries commonly attributed as the primary cause consist usually of little more than skin irritation from ill fitting harnesses, saddles or from blows. While these are mechanically not extensive, they are sufficient to liberate into the juices of the subjacent tissues the bacteria deeply seated in the integument. It is not unlikely that in some cases the lesions are due to infection, through metastasis, from bacteria that gain entrance through the walls of the digestive tract. The inflammatory process leading to suppuration, the formation of fistulæ, the new formation of fibrous tissue in the affected parts, and even the bone necrosis occasionally seen are all possible results of the activities of the pyogenic bacteria found in the lesions. There is nothing in their character to suggest causative agencies other than microörganisms. The tissue changes involved in the deposition of fibrous tissue and the abscess formation are known as the results of infection as well as the inflammatory processes following them. These affections are mentioned in this connection simply because the accumulating evidence tends to strengthen the working hypothesis that they are the result of bacterial invasion.

The morbid changes in the tissues are those of acute or more chronic inflammation.

Infectious mastitis. Cattle suffer frequently from an acute inflammation of the udder as the result apparently of an invasion by

a number of bacteria. The results of the investigations of this affection thus far reported suggest that the form which is transmitted from animal to animal is caused by a streptococcus. It is, however, impossible without a bacteriological examination to distinguish between this affection and those caused by other bacteria. It seems likely that many cases are primarily brought about by mechanical injuries which render possible the entrance into the fresh tissues of the bacteria of the skin or of the milk ducts. Other cases may be due to infection through the teat of bacteria capable of producing, by means of their metabolic products, the inflammatory condition without a distinct injury to the mucous membrane. The former view that there 'was a sphincter muscle near the base of the teat which closed the duct sufficiently to prevent the entrance of bacteria to the secreting portions of the gland was not founded on anatomical facts.

The inflammatory affections of the udder fall very naturally into two general groups, namely: (1) those in which the parenchyma is most affected and (2) those in which the stroma or fibrous tissue is involved. In many chronic inflammations they are both included. The form of mastitis more frequently encountered as an infectious (transmissible) disease is characterized by very marked changes in the milk, accompanied by the usual symptoms of parenchymatous inflammation of the gland itself. The discharge from the affected part of the udder usually contains flaky masses held in suspension in the clear or perhaps cloudy serum. The color varies, and occasionally the fluid is blood-stained. The microscopic examination shows the presence of agglutinated fat globules, pus cells and often red blood corpuscles.

A number of bacteria* considered of more or less etiological value have been associated with lesions of doubtful origin. The results of Kitt, Nocard, Mollereau, Guillebeau, Zschokke, Bang and still others, in which a *Bacterium*, a *Bacillus*, a *Micrococcus*, a *Staphylococcus*, and a *Streptococcus* have been found and reported as standing in a causal relation to the trouble, indicate that a variety of microörganisms are active in producing those affections which are frequently grouped without distinction as infectious mastitis. The

---

*Among the bacteria which have been found in udder trouble and described as a possible or perhaps the more probable cause the following species may be mentioned: *Bacterium phlegmasiæ uberis*, *Streptococcus agalactisæ contagiosæ*, *Staphylococcus mastitidis*, *Galactococcus versicolor*, *G. fulvus*, *G. albus*.

review of much of the literature on this subject shows that a number of cases reported as infectious were isolated or sporadic ones, that is, they were in dairies where the disease did not spread to other animals. While these may be truly infectious in their nature they should be differentiated from the rapidly spreading phlegmons which are easily recognized as infectious (contagious) mastitis.

If we take into account the variety of anatomical changes which have been described in the various udder affections, we can reasonably admit that different agencies may have been instrumental in their production. The various species of bacteria which have been isolated from the udder lesions may very likely have been of etiological importance in their respective cases. Bang, Hess, Kitt, Lucet and Nocard have pointed out that infectious mastitis is caused by bacteria which gain access to the udder through (1) the milk duct, (2) the blood circulation and (3) the lymphatic system.

Already the facts have been pointed out, that the udder is normally more or less extensively invaded with bacteria and that certain species seem to persist in the milk ducts of the glands when once they become localized there. If these results apply to cows generally as rigidly as they did to those examined, an explanation for the presence of a variety of bacteria in the affected udders is not difficult to find. Whether these particular organisms, under certain conditions, would become primarily responsible for udder disease is not known. The evidence suggests that a number of bacteria, heretofore described as the cause of mammitis, were in the affected glands by virtue of their presence in the normal udder. Concerning these points the results of additional investigations are much needed.*

*The writer has examined the milk secretions from the affected cows in two quite serious outbreaks of mastitis. In the first, the milk was drawn in sterile bottles after the udders and the hands of the milker had been thoroughly washed in a 1 to 1000 solution of corrosive sublimate. In all, there were eight samples of milk taken from as many different cows. In six of the eight specimens streptococci appeared in pure culture. In the other two cases micrococci were associated with the streptococcus. In the second outbreak, the milk from four diseased udders was drawn with aseptic precautions directly into tubes containing slant agar and promptly sent to the laboratory, where it was carefully examined. From two cases pure cultures of streptococci were obtained, while those from the others were impure. The streptococci obtained from the twelve cases appeared to be identical and the clinical aspect of the disease in the different animals was the same.

In a dairy that was under close observation by Ward, one cow was found to be troubled in one quarter of the udder with an inflammatory process which produced thickened masses in the blood-stained milk. From this milk a streptococcus was isolated in pure culture. It could not be differentiated from the one isolated from the cows in the outbreaks mentioned. Another cow in this herd was found to have her udder permanently infected with a streptococcus. Another animal in the same dairy suffered repeatedly from acute streptococcus mastitis.

There are a large number of morbid conditions more or less frequently encountered in domesticated animals, which seem to be due to infection of some kind but which are not demonstrated to be of such an origin. These will continue to be attributed by some to infection and by others to various general causes until the truth concerning their etiology is revealed.

Contagious agalactia. This is a disease reported to be peculiar to the goat and ewe, complicated with local manifestations in the eye, udder, and articulations. It has been classed as epizoötic or rheumatoid arthritis. It is observed in the mountainous regions, especially the Alps. It appears most often in the spring. It seems to be communicable. Oreste and Marcone have described four organisms associated with it, two of which are micrococci.

Miscellaneous infections. Attention should be called to the many morbid conditions, resulting from infection, that are encountered in different species of animals and are liable to be attributed to other agencies. Usually such lesions are referred to general pathological conditions, but a more careful inquiry will reveal the presence of infection. Among these, may be mentioned pericarditis in cattle, so frequently associated with punctures by foreign bodies. The extensive exudative inflammations in these cases are frequently associated with micrococci. The same has been true of certain cases of localized endocarditis resulting in the formation of fungoid, purulent, or necrotic masses about the valves of the heart. When one considers the possibilities of infection from accidental causes, in the intestinal mucosa as well as from the skin, together with the agency of metastasis, it is not difficult to understand how such a variety of morbid conditions can come about. Infection, therefore, forms an important part of pathology, outside of those specific organisms that cause epizoötics of greater or less severity.

REFERENCES

1. BOLLINGER. Mycosis der Lunge beim Pferd. *Archiv für pathol. Anat.*, Bd. XLIX (1870), S. 583.

2. BERGER. Vergleichende Untersuchungen über den Bacillus pyogenes bovis und den Bacillus pyogenes suis. *Zeitsch f. Infektionskrankh. parasitare Krankh. und Hygiene der Haustiere*, Bd. III (1907), S. 101.

3. GRIPS. Ueber eine mit multipler Abszessbildung verlaufende Pleuritis und Peritonitis der Schweine und deren Erreger. *Zeitschr. f. Fleisch-u Milchhygiene, 1898.*

4. GRIPS. Ueber einen pyogenen Mikroorganismus des Schweines. *Inaugural-Dissertation.* Giessen, 1902.

5. KUNNEMANN. Ein Beitrag zur Kenntniss der Eitererreger des Rindes. *Arch. f. wiss. u. prakt. Tierheilk., 1903.* Bd. 29, 30., S. 128.

6. MIGULA. *System der Bacterien.* 1897.

7. SMITH AND MOORE. On the variability of the infectious diseases as illustrated by hog cholera and swine plague. *Bulletin No. 6. U. S. Bureau of Animal Industry,* 1894. p. 81.

8. WELCH. General bacteriology of surgical infections. *Dennis' System of Surgery.* Vol. 1, p. 249.

9. DEJONG. Untersuchungen über Botryomyces. *Thèse de Giessen,* 1899.

10. DUBOIS. An enzooty of acute streptococcic mammitis. *Jour. of Compr. Pathology and Therap.,* Vol. 17 (1904), p. 159.

11. FROHNER. Ein Fall von generalisirter Botryomykose beim Pferd mit Metastasen in der Lunge. *Monatshefte für Thierheilk.,* Bd. VIII (1897), S. 171.

12. GAY. A bacteriological study of fistulous withers, botryomycosis and infected wounds in the horse. *Amer. Vet. Review,* Vol. XXIV (1901), p. 877.

13. JENSEN. Die vom nekrosebacillus (bacillus necroseos) hervorgerufenen krankheiten, In *Kolle and Wassermann's Handbuch der pathogenen mikroorganismen.* Vol. 2 (1903), p. 693–706.

14. JOHNE. Zur Aktinomykose des Samenstranges. *Deutsche Zeitschr. für Thierm.,* Bd. XII (1885), S. 73.

15. LUCET. Recherches bacteriologique sus la Suppuration. *Ann. de l' Institut Pasteur.* Vol. VI (1893) p. 324.

16. McFADYEAN. Disseminated necrosis of the liver of the ox and sheep. *Journ. Comp. Path. and Therap.* Vol. 4 (1891), p. 46–53.

17. M'FADYEAN. Metastatic lesions in Discomycosis. *The Jour. Compr. Path. and Therap.,* Vol. XIII (1900), p. 337.

18. METTAM. On certain septicaemias and some other infections of young animals. *Vet. Rec.* Vol. 16 (1903), p. 293–296.

19. MEYER. Untersuchungen über die multiple Nekrose der Leber des Rindes. Inaug. diss. Giessen (1903).

20. MIGULA. *System der Bacterien.* 1897.

21. MOORE. Suppurative cellulitis in the limbs of cattle due to streptococcus infection. *Amer. Vet. Review,* June, 1898. p. 169, Vol. 22.

22. NOCARD. A New Pasteurellose: White scours and lung disease of calves in Ireland. *Amer. Vet. Review.,* Vol. XXV (1901), p. 326.

23. SMITH AND DAWSON. Injuries to cattle from swallowing pointed objects. *Ann. Rept. U. S. Bureau of Animal Industry,* 1893–4, p. 78.

24. WARD. The invasion of the udder by bacteria. *Bulletin No. 178, Cornell Univ. Agric. Exp. Station,* 1900.

25. WILLIAMS. Reports N. Y. State Veterinary College. 1913–14 and 1914–15.

26. LEBLANC. The diseases of the Mammary Gland of the Domesticated Animals, 1904.

CHAPTER II

DISEASES CAUSED BY BACTERIA
GENUS STREPTOCOCCUS

General discussion of streptococci. The genus *Streptococcus* is based, according to Migula, on its method of reproduction or division. Streptococci are spherical bacteria that divide in one plane. The segments do not separate but are held together in short or longer chains, although the divisions seem to be complete. Just how the segments are held together is not fully determined. According to older and more commonly encountered classifications, a streptococcus is simply a number of micrococci (spherical bacteria) united in the form of a chain. In some of the supposedly different species the segments are oblong and vary in size. Frequently, however, the segments vary in size and form in the same chain.

The more usually observed cultural characters and biochemic properties of pathogenic streptococci are quite similar although it is difficult to obtain two cultures that will exactly agree in all of their manifestations when grown on a large number of media. Their disease-producing powers, however, vary within wide limits. While variations in the physiological properties and pathogenesis are true for different cultures (species?), it has been found that there is a possibility of much variation in the subcultures of the same species. As with certain other bacteria, their virulence is the first to suffer change. In differentiating species, therefore, the fact must not be overlooked, that the existing characters and properties possessed by the streptococcus in hand may have been more or less influenced by its conditions of life. When, for example, two streptococci appear to be identical under the majority of tests, a slight deviation in a single property cannot be considered of great differential value especially if this particular manifestation is among those most subject to change. A fundamental difficulty in differentiating species among streptococci seems to be a lack of information concerning the possible variations brought about by different environments. The further difficulty of identifying any of the very large number of forms which have been assigned specific names is due to the brevity of their

30

STREPTOCOCCI 31

description and the failure of the authors to mention any character or property, or combination of the same, which would distinguish them from each other.

Classification of streptococci. A few investigators have tried to eliminate the confusion concerning species by classifying streptococci according to distinct morphologic characters and pathogenic properties. More recently several investigators have attempted to classify them according to their cultural properties, especially their action upon the sugars. The studies of Kurth, Pasquale, von Lingelsheim, Andrews and Horder and Winslow, Palmer and Broadhurst are worthy of careful consideration in this connection.

Distribution of streptococci in nature. The fact has been pointed out in many publications that streptococci are quite widely distributed in nature. The results of the bacteriologic examinations of normal mucous membranes show that they are frequently included in the bacterial flora of the mouth, throat, nares, intestines, vagina, and in a few cases they have been found in the bronchioles of the horse and rabbit. They are also present in greater or less numbers on the skin, especially in the deeper layers, presumably in the ducts of the sweat and sebaceous glands and along the hair shafts and follicles. This explains their frequent appearance in certain wound infections. They have been found in soil and in water, and occasionally these forms are quite as delicate in their morphology and equally as sensitive to the influence of environment as those isolated from diseased animal tissues. In view of their wide distribution, the presence of streptococci in morbid tissues cannot be considered necessarily as specific infections. In many diseases, such as diphtheria and tuberculosis, streptococci frequently appear in the lesions. In these cases, they are accidental or secondary invaders, although in some of these maladies, such as tuberculosis, they are believed to be of more or less importance. When, however, the specific cause of the disease is not positively known, and streptococci which possess certain pathogenic powers for experimental animals are constantly present, the pathologist is confronted with a puzzling problem in trying to determine their etiological importance. In streptococcic infection leading at once to bacteriemia, peritonitis or suppuration, the explanation is more simple than in the epizoötic diseases, such as *Brustseuche*, where the presence of streptococci in the lesions can be quite as easily explained on the ground of their invasion of the parts

affected from the normal habitat in the upper air passages as on the hypothesis of a specific infection. It is in these instances that we are seeking for the crucial test for specific streptococcic etiology.

In cases of infection resulting in bacteriemia, or in those where the disease is more localized, as in strangles or mastitis, and possibly in others where the affection spreads more or less rapidly as in erysipelas, we cannot well escape from the feeling that the streptococci, present in such large numbers, must either stand in a causal relation to the disease or be accounted for by their rapid proliferation in the tissues in association with the true etiological factor. Their natural distribution is so wide and their virulence so capricious that a secondary invasion, which seems always to be possible, renders the fixing of etiological responsibility upon a streptococcus isolated from any diseased tissue a somewhat difficult task. The problems in this connection which concern us most and which need more extended investigation pertain (1) to the determination of the pathogenic possibilities of streptococci existing in their natural habitat and (2) to the distinction, if it exists, between streptococci that are able to produce local inflammatory processes leading to suppuration and those which produce specific diseases, such as erysipelas and strangles.

Pathogenesis. The pyogenic streptococci (*Strep. pyogenes* and its varieties) are the most important pathogenic species for animals in this genus. It has been found to be impossible to differentiate this species from several streptococci that have been considered the cause of certain diseases such as strangles in horses and erysipelas in cattle. In the absence of verified results to prove the non-specific relation of these streptococci to the diseases which have with reasonable certainty been attributed to this genus of bacteria, they are included among the specific streptococci maladies. It is very important, especially when the use of antistreptococcic serums are in question, to take into account the apparently large number of forms, or species, commonly included in the general statement of a streptococcus infection. In 1897, Van de Velde, in a very exhaustive series of experiments, showed that an antitoxin produced from one streptococcus will not immunize against another, save to a very slight degree. Better results are reported by the use of polyvalent serums.

There are a number of acute disorders, such as vaginitis in cows, that have been attributed to this genus.

REFERENCES

1. ANDREWS AND HORDER. A study of the streptococci pathogenic for man. *Lancet*, Vol. CLXXI (1906), p. 708.

2. BROADHURST. Environmental studies of streptococci. *Jour. Infect. Diseases*, Vol. XVII (1915), p. 277.

3. KLEIN. *Seventeenth Annual Report of the Local Government Board. Supplement containing report of Medical Officer.* London. 1887.

4. KURTH. *Arbeiten a. d. Kaiserlichen Gesundheitsamte*, Bd. VII (1891), S. 389.

5. MOORE. *Bulletin No. 3. U. S. Bureau of Animal Industry*, 1893, p. 9.

6. PASQUALE. *Beiträge zur path. Anat. u. zur allgemeinen Pathologie*, Bd. XII (1893), S. 433.

7. PETRUSCHKY. Untersuchungen uber Infection mit Pyogenen Kokken. *Zeitschrift f. Hygiene*, Bd. XVII, S. 59.

8. VON LINGELSHEIM. *Zeitschrift f. Hygiene*, Bd. X (1891), S. 331.

9. WELCH. *The Amer. Jour. of Med. Sciences*, Vol. CII (1891), p. 439.

10. WINSLOW AND PALMER. A comparative study of intestinal streptcoccoi from the horse, the cow and man. *Jour. of Infect. Diseases*, Vol. VII (1910), p. 1.

11. WINSLOW AND WINSLOW. The Systematic Relationships of the Coccaceæ. 1908.

STRANGLES

Synonyms. Adenitis equorum; Coryza contagiosa equorum; Distemper; *Gourme; Druse der Pferde.*

Characterization. Strangles is an acute infectious disease of horses, asses and their hybrids occurring sporadically and in epizoötics. It is characterized principally by a fever, followed by an acute catarrh of the mucosa of the upper air passages, especially of the nares, and a suppurative inflammation of the lymph glands of the submaxillary and pharyngeal regions. *Streptococcus equi* is present in the pus from the inflamed glands. It is a disease of young animals. Horses over 5 years of age are rarely attacked. It usually occurs between the ages of six months and two years.

Nagg has observed a disease in cattle in which the symptoms and lesions were like those of strangles. Starcovici observed a condition in young swine that was characterized by suppurating lumph glands about the head. Fröhner refers to a rare affection of dogs which he designated "dog strangles." In these cases the results of a careful bacteriological examination was not recorded and the diagnosis was based on the general symptoms and lesions.

History. Strangles was among the first equine diseases to be recognized. In 1664, Solleysel states that it had been known for a long time. Its infectious (contagious) nature was determined experimentally in 1790 by Lafosse and since that time his findings have been confirmed by other investigators. In 1873, Rivolta found in the pus of the abscesses a micrococcus which appeared in chains of from three to five segments. The specific cause, *Streptococcus equi*,

was described first by Schütz and later in the same year (1888) by
Sand and Jensen. This discovery has been confirmed by Poels,
Lupka and others. Lignières has discovered a "coccobacillus"
which he believed to be the primary cause. His conclusions have not
been confirmed.

Geographical distribution. Strangles is a wide spread disease
among young horses. It exists in all countries where horses are raised.
It is more prevalent in breeding districts than elsewhere. It is
reported, however, that Argentina and Ireland are free.

Etiology. Strangles is caused by *Streptococcus equi.* With pure
cultures of this organism Schütz was able to produce the disease in
healthy horses. This streptococcus is fatal to mice, a maximum
virulent virus destroying life in three days. Rabbits and guinea pigs
are less susceptible. Injected into horses subcutaneously a suppu-
rating inflammation follows. In the writer's experience streptococci
are the only bacteria that have been found in the abscesses.

The period of incubation varies. The usual time is from four to
eight days.

Symptoms. The first indication of this disease is a rise of tempera-
ture. There is loss of appetite, depression, and often great weakness.
The general symptoms may continue for a few days before the locali-
zation of the lesions is apparent. The first local manifestation con-
sists usually in a catarrh of the nasal mucosa or swelling of the sub-
maxillary and pharyngeal lymphatic glands. The nasal discharge is
at first serous and somewhat viscid, but in from 3 to 5 days it becomes
purulent and of a yellowish green color. The catarrhal condition
may exist in one or both nostrils. It may extend into the pharynx,
larynx, trachea and even to the bronchi. In most cases, swelling
of the sub-maxillary glands appears concurrently with the purulent
nasal catarrh. The spreading of the inflammation to the connective
tissue which surrounds the glands, and the stasis of the lymph in the
efferent lymph vessels, often cause the development, from the sub-
maxillary lymph glands, of extensive swellings that may occupy the
entire inter-maxillary space, and may spread even to the outer side
of the maxilla. Abscesses form in most cases.

In exceptional cases, strangles may present catarrhal symptoms
without suppuration of the lymph glands. Jensen states that it
may first assume the form of pharyngitis, purulent pneumonia, and
pleuritis without any well marked morbid affection of the lymph

glands. The urine generally remains alkaline; it frequently contains a considerable quantity of albumen.

At times, strangles is accompanied by a cutaneous exanthema which takes the form of an eruption of wheals, nodules, vesicles and even pustules; these may appear, chiefly on the sides of the neck, shoulders and sides of the chest. These exanthemata are characterized by their sudden appearance, and often by their equally rapid disappearance. An eruption of vesicles may break out on the nasal mucous membrane. · The contents of the vesicles is at first limpid, but later it becomes purulent. Rabe states that the streptococcus of strangles can produce ulcers on the nasal mucous membrane.

Morbid anatomy. The lesions in strangles are interesting from the fact that in the beginning the disease is general but later in its course it presents a series, exceedingly variable in different individuals, of localized tissue changes. The lymphatic glands are most often affected, although any organ may be involved. As indicated by the symptoms, the lesions in most cases are characterized by an acute inflammatory process followed by suppuration.

The glandular swellings about the head usually terminate in suppuration, the pus discharging either externally or into the oral cavity. In other cases the pus undergoes caseation. Frequently the inflamed glands become confluent, resulting in a single large abscess. Small abscesses may occur under the pharyngeal mucosa.

The inflammation may extend to the superficial lymph vessels of the skin, especially of the head, resulting in the formation of a large number of small abscesses. This may be followed by a diffuse phlegmonous swelling of the parts. Metastatic abscesses are liable to occur in a great variety of organs. The metastasis seems to take place through both lymph and blood vessels although the lymphatic glands are most often affected. Suppurating foci have been described in nearly every lymphatic gland in the body. The discharge of pus from the bronchial, mesenteric or other glands, within or adjacent to the pleura or peritoneal cavities, may give rise to a fatal pleuritis or peritonitis. There is no organ of the body free from possible suppurative lesions as a result of metastasis (bastard strangles).

Brudeaud and Demé have described a case of strangles which began as polysynovitis. Bouet reports that encephalitis is not exceptionally rare.

Strangles may become chronic, especially when the nasal catarrh extends into the sinuses of the head, the guttural pouches, or

pharyngeal cavity. In these cases the animal becomes emaciated. The lesions in these cases resemble somewhat those of chronic glanders. Many complications are liable to arise. Mixed infections and secondary lesions often occur. The prognosis, however, is favorable.

Death from strangles is caused usually by either septicemia, pyemia, pleuritis, peritonitis or suppurating (metastatic) pneumonia.

The duration of the disease varies according to its severity and the localization of the lesions. In mild cases convalescence begins in a few days, but in other cases restoration may require weeks and even months.

The mortality, according to available statistics, does not exceed four per cent. In certain epizoötics it may be higher. Often it is below two per cent.

Diagnosis. Strangles is to be diagnosed by the symptoms, lesions and a bacteriological examination. There are no specific tests that can be used for its detection. Strangles is to be differentiated from the following affections, namely:

Purulent nasal catarrh. In this affection, there are rarely suppurating sub-maxillary glands, although occasionally these glands may be swollen.

Glanders. In glanders, the tissue changes are more persistent and the skin lesions, if they exist, do not heal as rapidly as in strangles. In chronic cases, the diagnosis is quite difficult. Here animal inoculation must be resorted to. Mice inoculated subcutaneously with the nasal discharge succumb to the streptococcus of strangles but they are resistant to the bacterium of glanders. Guinea pigs inoculated in a like manner will, in case of glanders, develop that disease from the lesions of which pure cultures of *Bacterium mallei* may be obtained. The specific tests for glanders may be applied.

Parotiditis. In this affection the swelling is localized in the parotid gland and suppuration does not often occur.

Abscesses due to pyogenic bacteria. The cases are rare where there would be any question as to diagnosis. The bacteriological examination including the inoculation of animals would give positive aid unless the pyogenic organism happened to be a virulent streptococcus in which case a differentiation might be difficult. In these cases the nasal mucosa is not likely to be involved.

Prevention. This is best obtained by isolating the infected horses and removing the well ones from the infected premises. Colts are

reported to suffer more severely from strangles than adult horses. It is recommended that young horses should be carefully protected against infection, notwithstanding the immunity that seems to follow recovery. All infected stables should be thoroughly and repeatedly disinfected.

A number of attempts have been made to produce immunity against strangles but thus far they do not seem to be successful. Viborg and Toggia, a century ago, inoculated horses by rubbing in the nostrils discharges from cases of mild strangles for the purpose of producing a light attack of the disease followed by a resistance to natural infection. Jess and Piorkowski tried to produce immunity with a "shake-extract of cultures of strangles streptococci." Otto reports satisfactory results with its use in 694 horses. The use of killed cultures of streptococci, and mixtures of concentrated cultures and pleural exudates (aggressins) have been tried without success.

Specific biologic treatment. Anti-strangles serums have been carefully tested by Dassonville and Visgocqi, Jess and Piorkowski and others with not entirely satisfactory results. *Gurmin*, or Ruffel's strangles serum, has been reported to be of considerable curative value. Cederberg found the use of serum of value in treating strangles in the Danish army horses. Vicchi and Gatti have made a serum that is used in Italy for treating strangles. There does not seem to be an efficient serum for the treatment of this disease.

REFERENCES

1. *Boudeaud et Demé.* Un curieux cas de gourme. *Rev. Gén. de Méd.* Vét. Vol. XVI, (1910), p 508.

2. LIGNIERES. The etiology of equine influenza or infectious pneumonia, *Jour. Compr. Path. and Therap.*, Vol. XI (1898), p. 312. Translated from Recueil de Méd. Vét., Vol. IV (1897).

3. POELS. Die Mikrokokken der Druse der Pferde. *Fortschr. der Med.*, Bd. Vol. (1888), S. 4.

4. REEKS. Intra-cranial strangles abscess in a mare. *Jour. Compr. Path. and Therap.*, Vol. XII (1899), p. 178.

5. SAND AND JENSEN. Die Aetiologie der Druse. *Deutsche Zeit. für Thiermed.*, Bd. XIII (1888), S. 437.

6. SCHUTZ. Der Streptococcus der Druse des Pferdes. *Arch. für Thierkeilkunde* Bd. XIV (1888), S. 172.

APOPLECTIFORM SEPTICEMIA IN CHICKENS

Characterization. A rapidly fatal septicemia in chickens caused by a streptococcus.

History. This disease was recently discovered and described by Nörgaard and Mohler. Although the symptoms and lesions given correspond somewhat closely to those mentioned by Mazza and Rabieux, there is a marked difference in the etiological factor. It is caused by a streptococcus which is readily obtained from the blood or organs.

Geographical distribution. This disease has been described from Virginia, New York, Hawaiian Islands, and several places in Europe.

Etiology. This disease is due to a streptococcus which grows in short or longer chains with segments varying from 0.6 to 0.8μ in diameter. In some cases elongated forms are observed. It is an ærobe, and a facultative anærobe. When cultivated on artificial media it does not liquefy gelatin, it does not change the appearance of milk, but causes slight acidity and thickening of the lower stratum without coagulation of the casein. The reaction of alkaline bouillon is changed to an acid one. It does not give a visible growth on potato. It stains by Gram's and Gram-Weigert's methods. In bouillon it grows in somewhat flaky masses while the medium remains clear. It was fatal to fowls, mice, rabbits and swine; guinea pigs, dogs and sheep were not destroyed by inoculation. This streptococcus has not been specifically named.

The period of incubation is very short.

Symptoms. It is not at all uncommon to find the fowls dead and lying under the roosts. Occasionally capons were observed to be sick for from 12 to 24 hours prior to death. In these cases the feathers became ruffled and the fowl showed evidence of extreme depression. The onset of the disease is very sudden and its course a very rapid one, usually terminating in death.

Morbid anatomy. Nörgaard and Mohler described the morbid anatomy as follows: "The spleen is more or less enlarged, showing hyperplasia of the Malpighian corpuscles. The pulp contains numerous areas of extravasated blood. When a stained section is examined by means of a hand lens a number of circular semi-transparent foci, the size of a pin hole, may be noted. These are found on microscopic examination to be centers of necrobiosis, consisting of

parenchyma which has undergone coagulation necrosis, and surrounded by a more or less well defined capsule of embryonic and further developed connective tissue cells and filaments.

"On microscopic examination, the kidneys show slightly swollen epithelial cells of a beginning parenchymatous degeneration to well pronounced disintegration of the renal epithelium of acute nephritis. The degree of degeneration depends, as a rule, upon the course of the disease. If a bird succumbs suddenly or in the course of a few hours the morbid changes are either not apparent at all or but slightly pronounced, while, on the other hand, the duration of three or four days to a week results in an acute exudative nephritis. The swollen or degenerate epithelium of the tubules surrounds irregular masses of coagulated exudate and white blood corpuscles, among which are numerous short chains of streptococci. In very acute cases with sudden death the liver shows extreme hyperemia. The cells have a slightly granular appearance in addition to the fatty infiltration usually seen in the liver of well kept fowls. When death does not occur until after twenty-four hours the liver cells also show parenchymatous or fatty degeneration; their outlines become indistinct, the body very granular, and the nucleus takes the stain but faintly. Interlobular and intralobular collections of round cells and leucocytes appear, and in more chronic cases centers of coagulation necrosis may be seen. The lungs become hepatized. The walls of the bronchioles are thickened and the streptococci may be seen in the minute capillaries. The air cells are filled with plasma, red blood corpuscles and epithelium, among which the microörganism is easily detected." Magnusson reports the same lesions.

Diagnosis. This affection must be diagnosed by the bacteriological examination of the blood and tissues. It is to be differentiated from fowl typhoid and chicken cholera.

Prevention. The separation of the well fowls from the diseased ones and placing them in uninfected houses or yards is of the first importance. Nörgaard and Mohler found that immunity may be produced by the filtrate or sterilized bouillon cultures and the serum of immunized animals.

REFERENCES

1. MAGNUSSON. Apoplektiforme Septikæmia bei Hühnern. *Sevensk veterinär Tidskrift.* Bd. XV, S. 60. Ref. Ellenbergers Jahesbericht, 1910.

2. NÖRGAARD AND MOHLER. Apoplectiform septicemia in chickens. *Bulletin No. U. S. Bureau of Animal Industry,* 1902.

STREPTOCOCCIC MASTITIS

Characterization. The term "streptococcic mastitis" has been given to an infectious disease of the udder of cows caused by a streptococcus. It is characterized by hard foci in the gland.

History. As early as 1848, Brennwold observed in Switzerland an enzoötic mastitis that was difficult to cure. The affection was called *"gelber Galt."* Since that time this affection has been found in nearly, if not, all countries. Among the more recent writers on this subject may be mentioned Hess and Borgeaud in Switzerland, Nocard and Mollereau in France, and Zschokke in Vienna. In America it does not appear to have been studied independently of the infectious form of mastitis described on page 25. The epizoötic mastitis occasionally reported in this country may be identical with this supposed distinctively specific disease. Zschokke found the streptococcus in 297 of the 444 cases of altered milk examined.

Geographical distribution. This affection has been reported from nearly every country where cows are kept.

Etiology. The organism that causes this disease was described by Kitt as *Streptococcus agalactiæ*, and by Guillebeau as *Streptococcus contagiosæ*. It enters the udder through the ducts of the teats.

The writer has been unable to differentiate this streptococcus from the one he has found in cases of mastitis, and also in the milk of cows with healthy udders. Ward and Reed produced mastitis in a healthy udder with the streptococcus that they had isolated from a normal udder.

The period of incubation is very short, one to three days in the produced cases.

Symptoms. The first symptom is the diminution in the quantity of milk, usually in but one-quarter of the udder. This is quickly followed by indurated foci in the affected glands. The part becomes inflamed. The discharge or secretion is thin, more or less colored, and contains pus cells and clumps of streptococci. The lesions develop slowly, and one quarter after another of the udder becomes involved. Later the milk secretion is liable to stop entirely.

Morbid anatomy. The tissue changes are described as those of catarrhal inflammation of a mucous surface, followed by the development of new formed tissue and atrophy of the parenchymatous tissue.

The gland is hard and in time becomes enlarged, due to the new formed tissue. The microscopic examination shows a thickened intertubular tissue, and the epithelial cells more or less disintegrated and sloughed from the tubular walls. The lymphatic glands and other organs of the body are not involved. The lesions are localized in the udder.

The period of duration is variable, but always long.

The prognosis is grave for the gland itself. It is rarely fatal to the animal.

Diagnosis. The diagnosis is made by finding the streptococcus in the secretions. It is to be differentiated from cases of mastitis caused primarily by some injury, and the infectious mastitis caused by other bacteria. This can readily be done from a bacteriological examination of the udder secretions.

Prevention. This disease is spread from the infected to the non-infected largely by the hands of the milkers. Recognizing this fact, the spread can be stopped by disinfecting the hands (washing in a disinfectant) of the milker after each animal. The diseased animals should be isolated from the others. It is not a difficult infection to control.

REFERENCES

1. BRENNWALD. Chronische Euterentzündung, *Archiv. f. Thierheilk.* Bd. X. (1848), S. 40.

2. DUBOIS. An enzoöty of acute streptococcic mammitis. *Jour. Comp. Path. and Therap.*, Vol. XVII (1894), p. 159.

3. NOCARD ET MOLLEREAU. Sur une mammite contagieuse des vaches laitières. *Bulletin de la Sociètè centr. de Méd. vét.*, 1884, p. 188. *Ibid. Ann. de l'Institut Pasteur*, Vol. I (1887), p. 109.

4. REED AND WARD. The significance of the presence of streptococci in market milk. *American Medicine*, Vol. VII (1903), p. 256.

5. ZSCHOKKE. Weitere Untersuchungen uber den gelben Galt. *Schweizer-Archiv für Thierheilk*, Bd. XXXIX (1897), S. 145.

CHAPTER III

DISEASES CAUSED BY BACTERIA
GENUS MICROCOCCUS

General discussion of the genus micrococcus. The *genus Micrococcus* includes the spherical bacteria that divide in two planes. The micrococci, therefore, may exist as single spherical organisms or they may be united in pairs (diplococcus), in fours (tetracoccus), or in small clumps or masses (staphylococcus). This genus contains many important species but for lower animals they are largely restricted to those producing wound infections, such as *Micrococcus pyogenes* and its varieties. These infections are not characteristic in their manifestations and consequently the disturbances they produce are not classed among the specific infectious diseases. At present, we seem to have recognized but one specific malady of animals attributed to them. In man a number of diseases are caused by micrococci.

TAKOSIS

Characterization. Takosis, meaning "to waste," is a destructive, infectious disease of angora goats. It is characterized by great emaciation and weakness, with symptoms of diarrhea and pneumonia.

History. The name Takosis was given to this disease of goats by Mohler and Washburn who described it in 1903.

In 1875, a disease was reported among angora goats in Virginia which may have been this infection. Pegler describes it somewhat fully in his work entitled "The book of the goat" as "a disease peculiar to goats." The identity of this disease and the various affections of goats, especially pneumonia, described by Duquesnoy, Hutchens, Pusch, Steele and others is not established.* Mohler and Washburn seem to be the only investigators in this country who have carefully studied this affection.

---

*In 1913, we studied a disease of goats that resembled in its symptoms Takosis. It was tentatively diagnosed as such. The post mortem examination revealed large numbers of the larval form presumably of *Dictyocaulus filaria*, in the lungs. From some cultures we obtained a micrococcus but it was not uniformly present. It was not *M. caprinus*. There seems to be a little doubt as to the identity of the diseases of goats studied by different authors and it is possible that some of them may have been parasitic in nature.

Geographical distribution. Takosis is reported from a number of localities in this country, more especially in the Northern States.

Etiology. According to Mohler and Washburn this disease is caused by *M. caprinus*. It is pathogenic for goats, chickens, rabbits, guinea pigs and white mice, but not for sheep, dogs or rats. It usually appears in pairs. It has been isolated in pure culture from the heart's blood, spleen, kidneys and pericardial fluid. It was not obtained in cultures from the spinal cord.

Symptoms. The first observable symptom is a listless and languid appearance of the animal. The affected goats lag behind the flock. Frequently there is drooping of the ears and a drowsy appearance of the eyes. There is slight elevation of the temperature in the beginning, but later in the course of the disease it becomes sub- normal. As the disease advances the goats move about in a desultory manner, the back arched, neck drawn down toward the sternum, and the gait staggering. Rumination is seldom impaired. The appetite is usually good but capricious. The exposed mucous membranes are pale. The respirations are accelerated and labored. The affected animals soon become so weak that they can stand with difficulty, and often they are knocked down and trampled by their companions. They shrink often to nearly half their normal weight. There is usually a fluid discharge from the bowels of a very offensive odor during the last few days. The goat groans occasionally and the head is usually bent around to one side. Death follows in from eight days to ten weeks. Recoveries have not been observed. The young are reported to be more susceptible to the disease than the older animals.

Morbid anatomy. According to Mohler and Washburn emacia- tion and anemia are the most striking lesions. The lungs usually contain areas of pneumonia. Their surface is mottled by areas of congestion and iron gray patches. On section these areas show a frothy mucus in the bronchioles. The heart muscle is pale, dull, soft and flabby. Inflamed hemorrhagic areas may appear on the epi- cardium. Sometimes they are present in the endocardium especially that lining the ventricles. The pericardium is slightly thickened and usually contains a small quantity of blood-stained fluid. The gall bladder is frequently distended with a pale-yellow watery bile. The liver appears to be unaffected. The kidneys are anemic and softened. The cortex is pale and contrasts strongly with the dark pyramids.

The capsule is easily removed. The spleen appears to be atrophied and indurated, the fibrous portions exceeding the spleen pulp. The spleen may be attached to the diaphragm or neighboring organs by adhesions. The mucosa of the intestines gives the appearance of a chronic catarrh associated with necrosis of the mucosa.

The microscopic study shows the terminal bronchioles and alveolar passages to have swollen walls and to contain various amounts of mucus and desquamated cells as a result of the catarrhal inflammation. The blood vessels in the interalveolar tissue are distended and surrounded by migrated leucocytes. The kidneys show a catarrhal or parenchymatous nephritis with the most pronounced changes occurring in the cortex. The intercapsular space is dilated and contains an albuminous exudate. The convoluted tubules show the epithelium to be swollen and granular and occasionally desquamated. The tubules may contain an albuminous deposit. As the specific micrococcus has not been found in the kidneys, Mohler and Washburn considered the lesions in this organ to be of toxic origin.

The heart shows parenchymatous degeneration of isolated fibers or groups of fibers. The spleen shows an increase in fibrous tissue. Sections of the intestines, especially of the duodenum, show a productive inflammation with exudation. There is often desquamation of the mucosa.

The blood count in experimental cases shows an increase in the number of red corpuscles. Mohler and Washburn report the examination of the blood in two such cases and one of natural infection as follows:

No. 1. Red corpuscles 11,190,000, white corpuscles 20,560 per cubic millimeter.

No. 2. Red corpuscles 12,160,000, white corpuscles 20,000 per cubic millimeter.

No. 3. (Natural infection) red corpuscles 10,208,000, white corpuscles 14, 860 per cubic millimeter.

They give the normal red corpuscles as 9,976,000, white corpuscles 9,200 per cubic millimeter.

The increase in the white cells they state is due to an increased number of polymorphonuclear leucocytes and eosinophiles. They found the specific gravity of the blood to be 1.031 and hemoglobin 56.

Diagnosis. Takosis is to be diagnosed by its infectious nature and by finding the specific organism, *M. caprinus*, in the tissues. It is

to be differentiated from the morbid condition resulting from various animal parasites, anemia caused by some previously existing disease such as chronic pneumonia, or poor nutrition.

The symptoms caused by parasites frequently resemble quite closely those of takosis. In takosis symptoms of pneumonia will frequently be noted, especially the labored breathing or rapid respiration. The luster of the fleece is less affected in takosis, while diarrhea is more frequently noted. Continuous coughing and snuffling, while diagnostic of the presence of lung worms, are not characteristic of takosis.

Hutcheon writes concerning the contagious pneumonia of goats as follows:

"It was a specific infectious form of pleuro-pneumonia, affecting goats only. Cattle and sheep remaining free from infection although constantly exposed to it. The disease was introduced into Cape Colony by a shipload of angora goats from Asia Minor, where the disease is represented as being indigenous."

Prevention. The study of this disease by Mohler and Washburn brought out very clearly certain preventive measures that should be carefully noted.

The most destructive outbreaks have occurred among goats that had recently been shipped from a Southern locality to a Northern latitude. Sudden climatic changes should be avoided. Hobson states that the natives of Asia Minor assert that the goat cannot be transported from one village to another of higher altitude without suffering some deterioration.

Angora goats should be provided with stables that are perfectly dry. These should be accessible to them at all times, as rains are very injurious to them. So averse are they to wetting that they will seldom be caught out in a shower if shelter is within reach.

Careful feeding.

When the disease appears, remove all well animals from the sick ones.

Immunity seems to have been established by the injection subcutaneously of sterilized cultures of the specific organism. The method, however, is still in the experimental stage.

REFERENCES

1. Hobson. Angora goat farming. *Agricultural Journal, Cape Colony,* Vol. VIII (1894), p. 81.

2. Holzendorff. Lungen-Brustfellentzündung bei Ziegen. *Archiv für Thierheilk.* Bd. XXII (1896), p. 345.

3. HUTCHEON.   Contagious pleuro-pneumonia in Angora goats.   *The Veterinary Journal*, Vol. XIII (1881), p. 171.

4. HUTCHEON.   Contagious pleuro-pneumonia in goats at Cape Colony, South Africa.   *Ibid.* Vol. XXIX (1889), p. 399.

5. MOHLER AND WASHBURN.   Takosis, a contagious disease of goats.   *Bulletin No. 45, U. S. Bureau of Animal Industry, Washington, D. C.*, 1903.

6. NICOL ET REFIK-BEY.   La pneumonie des chèvres d' Anatolie.   *Ann. de l'Inst. Pasteur*, Vol. X (1896), p. 321.

7. PEGLER.   The book of the goat.   1885.

CHAPTER IV

DISEASES CAUSED BY BACTERIA
GENUS BACTERIUM

General discussion of the genus bacterium. The genus *Bacterium* includes all rod-shaped, non-motile bacteria. The absence of motility appears to be a logical, natural and sufficient reason to place these organisms in a genus by themselves. The only objection, that can reasonably be urged against it, from the pathologist' spoint of view, is the changing of the generic name of a number of important pathogenic bacteria, such as those of anthrax, glanders, tuberculosis and others, from *Bacillus* to *Bacterium*.* This, however, is not serious but should be gladly welcomed if it enables us to bring into groups for study diseases that are etiologically more closely related. It is for that reason that this classification is adopted. The further subdivision of the non-motile, rod-shaped bacteria into several genera, as found in more recent classifications, suggests the desirability of a more restricted grouping of diseases for study and comparison than the genera of their etiological organisms provide. In order to group the diseases due to closely related bacteria, Lignières introduced the term Pasteurelloses to include the diseases in different species of animals caused by bacteria represented by the bacterium of fowl cholera. Trevisan gave the generic name *Pasteurella* to this group of organisms. Hueppe designated them under the heading of *Bacillus septicemiae hemorrhagicæ*. He seems to have taken for his type the bacterium of *Schweineseuche*.

Lignières† grouped the diseases caused by the *Pasteurella* Trev. according to the animals affected. Thus, fowl cholera, rabbit septicæmia and swine plague are designated as the Pasteurelloses of birds, Pasteurelloses of rabbits, and Pasteurelloses of swine. He carries this classification to include all the diseases in all species of

---

*It is important not to confuse the genus bacterium as revived by Migula with the same genus of early writers who characterized it as composed of non-spore bearing, rod-shaped organisms.

†LIGNIÈRES. Contribution à l' étude et à la classification des septicemies hemorrhagiques les "Pasteurelloses." *Ann. de l' Instit. Pasteur*, Vol. XV (1901), p. 734.

animals caused by members of the genus Pasteurella.   Nocard and
Leclainche accepted this grouping.   Another illustration of grouping
diseases due to closely related bacteria is found in tuberculosis.   With
a better knowledge of the specific organisms, the infectious diseases
will undoubtedly be more generally studied in groups according to
their etiology.

REFERENCES

CHAMBERLAND ET JOUAN.   Les Pasteurella.   *Annales de L' institut Pasteur*, Vol.
XX (1906), p. 81.

LIGNIÈRES.   *Contribution à l' étude et à la classification des septicémies hémorrhagiques,*
Buenos-Ayres, 1900, p. 8.

NOCARD.   Les Pasteurelloses.   Leçon faite à l'Institut Pasteur.   *Revue générale*
Médecine vétérinaire, T. II, (1903,) p. 188.

SWINE PLAGUE*

Synonyms.   Infectious pneumo-enteritis; Pasteurellosis suis; *Pas-*
teurellose du porc; Septicemie du porc; Pneumonie contagieuse du porc;
Schweineseuche.

Characterization.   Swine plague is an infectious disease of swine
occurring sporadically and in enzoötics.   It appears usually as a
septicemia, or a pneumonia in which there is marked consolidation of
the ventral and cephalic lobes and the cephalic portion of the principal
lobe of one or both lungs.   There may or may not be pleuritis.
There may be marked changes in the intestine, consisting of super-
ficial necrosis of the mucosa especially the ileum and cecum.   On
this account it has been considered an infectious pneumo-enteritis.
Petechial hemorrhages in the kidney and on the heart have been
observed.

History.   In 1886, Smith found in a pig in the state of Illinois a
disease which differed from hog cholera, and from the lesions he
isolated a bacterium identical with that of *Schweineseuche*.   Later
other cases of this disease were found not only in the state of Illinois
but in various places in the eastern part of the United States.   In
1885, Lœffler had described the cause of an infectious pneumonia in
swine (*Schweineseuche*) and had differentiated it from swine erysipe-
las.   The first publication on this disease in the United States is in the
Annual Report of the Bureau of Animal Industry for 1886.

---

*For an explanation of the confusion existing concerning the nomenclature of
swine plague and hog cholera see hog cholera.

Smith described swine plague as an independent disease, although it was often associated with hog cholera in the same animal. In Europe, Schütz, Jensen, Bang, and Preisz established its independent nature. On account of its frequent association with hog cholera, it was thought by some to be a secondary infection only. Several outbreaks were studied which indicated that it was an epizoötic disease. It was differentiated from cholera by the lesions and bacteriological examination only. The discovery of the filterable virus of hog cholera has thrown doubt upon the conclusions of the earlier study of epizoötic swine plague. The consensus of opinion seems to be that swine plague is a sporadic disease which rarely if ever occurs in epizoötic form. Certain lesions, such as petechial hemorrhages in the kidney, found in pigs dead from the septicemic form of swine plague are now known to be quite characteristic of hog cholera. For that reason, lesions that were formerly supposed to be due to *Bact. suisepticum* may have been caused by the filterable virus of hog cholera. The lesions resulting from, or peculiar to, *Bact. suisepticum* infection must be more definitely determined from future investigations.

Geographical distribution. Swine plague occurs more or less frequently in every state in the Union. It is quite widely distributed in Europe. It does not appear to be restricted to any country.
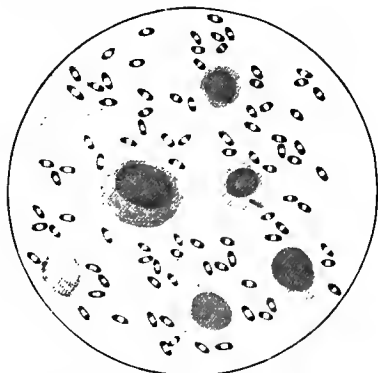
Fig. 4. BACTERIUM SUISEPTICUM FROM A COVER-GLASS PREPARATION OF A RABBIT'S LIVER.

Etiology. Swine plague is caused by a non-motile, elongated, oval bacterium first described by Lœffler in 1885. Hueppe proposed the name *Bacterium septicemiae hemorrhagicae* for this organism. Migula called it *Bacterium suisepticum*.

Hutyra has found a filterable virus in the blood and other parts of pigs suffering with *Schweineseuche*. The possibility of a mixed infection in this case is not excluded. Other European observers have reported similar findings.

Bacterium suisepticum and its closely related varieties attacking other animals have not been systematically studied and classified.

It has already been noted that the bacteria of rabbit septicemia, fowl cholera, *Wildseuche* and septicemia hemorrhagica of cattle are closely related to it.   In human pathology, there is a striking resemblance between *M. lanceolatus* and *Bact. suisepticum*, especially in its varied pathogenic possibilities in rabbits and their frequent presence in normal saliva.

It should be recognized that experimentally the different varieties or forms of this group (*Bacterium septicemiae hemorragicae* Hueppe, *Pasteurella* Trev.) are not interchangeable in their pathogenesis except for the rabbit.   Thus an epizoötic form of fowl cholera has not been produced with the bacterium of swine plague or of rabbit septicemia.   Further, it has been shown that in the upper air passages of healthy swine, cattle, horses, cats and dogs* there are bacteria not distinguishable by the usual methods in their cultural characters and in their effect upon rabbits from the swine plague bacterium.   The presence of this organism in the trachea of healthy pigs explains the frequent association of this bacterium with hog cholera and other maladies.   The conditions necessary for this organism to produce disease in its host have not been explained.

If the rabbit is taken as the animal on which to test the pathogenesis of the bacteria belonging to the swine plague group, we find that those from different sources are very similar.   In nature, the bacteria of swine plague, rabbit septicemia, fowl cholera, and those located in the normal upper air passages of the various species of animals mentioned exist, possessed of marked variation in virulence, that is, there are those that will kill a rabbit in from 16 to 24 hours when inoculated subcutaneously with a pure culture and those that require from 3 to 10 days, or even weeks, to destroy life.   With the variations in the time period, we have corresponding differences in les'ons.   The virulent forms produce bacteriemia while the attenuated varieties excite a severe purulent infiltration about the place of inoculation and exudates on one or more of the serous membranes. Conversely, it has been shown that rabbits possessed of a certain amount of natural or artificially produced resistance will, when inoculated with a virulent culture, die after the same period of time and with lesions similar to those produced by the attenuated virus in the susceptible rabbit.

---

*The investigations thus far made show these bacteria to be present in 48 per cent. of healthy swine, 80 per cent. of cattle, 50 per cent. of sheep, 16 per cent. of horses, 90 per cent. of cats, and 30 per cent. of dogs.

The effect of swine-plague bacteria on rabbits. In 1894, Smith and Moore described the effect of swine-plague bacteria on rabbits and also the effect of resistance on the part of the rabbit on the form of the resulting lesions.

Among the forms of disease observed after the subcutaneous inoculation of rabbits with swine-plague bacteria from different sources (epizoötics) were bacteriemia, peritonitis, pleuritis (usually with pericarditis), pleuritis (usually with pericarditis and peritonitis) and local lesion only.

In bacteriemia, death ensues within eighteen or twenty-four hours. The local lesion produced at the seat of inoculation is slight. Bacteria are abundant in the parenchyma (blood vessels) of the various organs. In the form characterized by peritonitis death occurs in three to seven days. The local lesion, which in all these forms of diseases increases in extent with the prolongation of the life of the animal, is here characterized by more or less suppurative infiltration of the skin and the subcutis. The peritonitis in its earlier stages is characterized by punctiform hemorrhages on the cecum and a fibrinous or cellular exudate. It always contains immense numbers of bacteria. When pleuritis is also present the exudate usually involves the pericardium as well. It varies in amount according to the duration of the disease and is essentially the same as the peritoneal exudate.

The form characterized by pleuritis and pericarditis without peritonitis is interesting in so far as the seat of inoculation does not explain the localization, for, in every case, the inoculation was made in the region of the abdomen. The lungs may become hepatized secondarily through invasion from the pleura if the animal lives long enough.

Lastly, the form of disease in which the only localization is a very extensive suppurative infiltration associated with hemorrhage and edema of the subcutaneous tissue is not common.

Period of Incubation. In artifically produced cases it is very short, the symptoms appearing in from 1 to 2 days. In natural infections it is also believed to be short.

Symptoms. The peculiarities of swine render it exceedingly difficult to obtain evidence on physical examination of lung disease or general infections distinctive of swine plague. Sometimes this affection runs a very rapid course, the animal dying of bacteriemia. Usually it is more protracted, lasting from a few days to one or two weeks. There is a rise of temperature. As it advances the pigs become weak. In its very acute septicæmic form the temperature is high and the duration of the disease is very short. Animals affected with the more chronic form eat very little or refuse food altogether. They cough considerably, especially when forced to run. The back is usually arched and the groins sunken. The whites of the eyes are

reddened.   The skin over the ventral surface of the body, nose and
ears is frequently flushed.   The cough, however, is the most reliable
indication we have of swine plague; but in some cases of hog cholera
the coexistence of broncho-pneumonia also causes the animal to
cough when forced to move rapidly.

Morbid anatomy.   There are many known variations in the
appearance of the internal organs of hogs which have died of swine
plague.   The most characteristic lesions are to be found in the lungs.
Frequently the abdominal viscera appear to be normal, although a
careful examination will usually reveal slight changes.   In the lungs,
however, the disease is ordinarily obvious.

The variety of lesions produced by the inoculation of swine plague
bacteria is not so great as that observed in the naturally contracted
disease.   While there are outbreaks in which considerable uniformity
is observed, there are others in which each animal is a surprise to the
pathologist.   In general it may be stated that the lungs and the diges-
tive tract are the chief seats of the disease, though other organs,
notably the lymphatic glands, are secondarily involved.   The lesions
are localized in the lungs and in the digestive tract probably because
the bacteria gain entrance through the respiratory and digestive
passages.

The lungs have been found diseased in nearly every outbreak
which has been investigated.   In some outbreaks the lung lesions
predominated and pneumonia was the direct cause of death.   In
individual cases, pneumonia is absent but pleuritis and interlobular
edema are generally present.   In a few instances interlobular emphy-
sema of the lungs has been observed.   With pneumonia the seat of
the lesion varies: usually the ventral lobes are first attacked, then
the cephalic and azygos, and lastly the principal lobes.   This move-
ment of the disease seems to depend on gravity, inasmuch as the dis-
eased parts are marked off from the healthy portion by a nearly
horizontal line.   In other words, the most dependent portions of the
lungs are the ones affected first, and as the disease progresses upwards
only a small port'on of the principal lobe directly under the back of
the animal remains pervious, provided the life of the animal is main-
tained up to this point.   In cases where disease is caused by lung
worms or by embolism, the pneumonia involves portions of the
principal lobes not contiguous to the ventral lobes.

Two kinds of pneumonia are encountered, namely, lobar and catarrhal or broncho-pneumonia. In the former the vesicular portion of the lung substance is chiefly affected; in the latter the smaller bronchioles are primarily attacked and the alveoli secondarily. In croupous-pneumonia, there is, following the stage of congestion, an emigration of red blood corpuscles, some leucocytes, and an exudate of fibrin into the air spaces. These elements are firmly matted together by the coagulating fibrin, making the diseased lung firm to
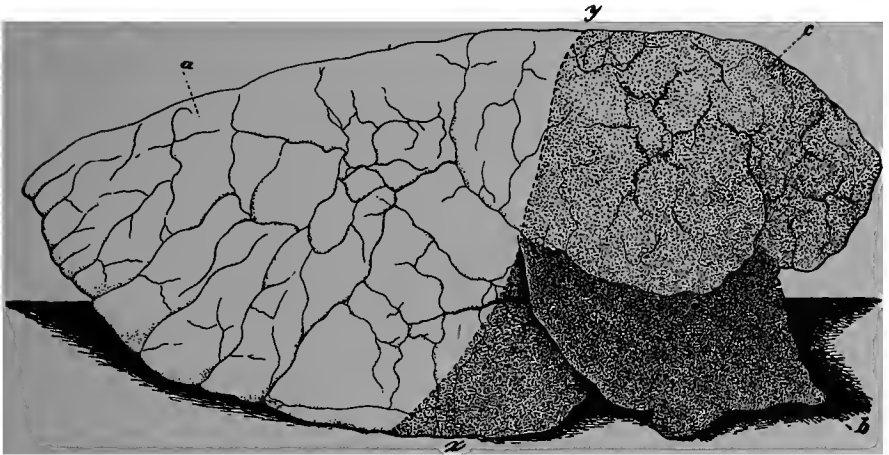
FIG. 5. RIGHT LUNG OF PIG. THE STIPPLED PORTION IS USUALLY INVOLVED IN CASES OF INFECTIOUS PNEUMONIA OR SWINE PLAGUE. (b) VENTRAL LOBE, (c) CEPHALIC LOBE, (a) PRINCIPAL LOBE. THE VENTRAL LOBE IS USUALLY THE SEAT OF THE MORE ADVANCED DISEASE AND CONSEQUENTLY THE FIRST TO BECOME HEPATIZED. THE CEPHALIC PORTION OF THE PRINCIPAL LOBE (x) IS USUALLY HEPATIZED AND THE REMAINING PORTION DEEPLY REDDENED.

the touch. In broncho-pneumonia the catarrhal condition of the smaller air tubes makes them impervious to air. The lung tissue which they supply is gradually emptied of air and assumes the appearance of red flesh, owing to the collapse of the walls of the alveoli and the distended condition of the capillary network. Subsequently the inflammation extends into the alveoli, which then become distended with cellular masses.

The nature of the lung disease will depend more or less upon the mode of entrance of the virus. If it enters only by way of the air tubes it will appear perhaps as a broncho-pneumonia. If it enters the lung tissue through the circulation we may have more or less

scattered centers of hepatization (embolic pneumonia). If it enters by way of the pleura, the virus will creep along the interlobular and peribronchial tissue before it invades the parenchyma proper.

In natural infection of swine plague, bacteria seem to enter the lung tissue chiefly by way of the air passages. At the same time it is not improbable that occasionally they may enter the serous cavities first, i. e., invade the pleural cavities and thence the lungs. This probability is shown by inoculation in which intravenous injections produced exudative pleuritis and pneumonia of the most dependent portions of the lungs covered by the pleural exudate. It is not improbable that even in the natural disease the bacteria which have gained access to a portion of the lung tissue by way of the air tubes reach the pleura covering this portion, and may then by this route invade other portions of the lungs. It may be that in this way a pneumonia originally single may become double. It has been observed that the first pneumonic infiltration of the principal lobe was at the point of contact with the diseased ventral lobe, and that the resting of a lobe against an inflamed serous surface, such as the peri-cardium, caused a pneumonic infiltration at the point of contact.

The character and seat of the lung lesions are somewhat variable. It is difficult to find two lungs exactly alike so far as gross appearances go. This to be sure may be due largely to the fact that animals die in different stages of the disease. Yet there are differences evidently not dependent on this fact, which must be left for special pathological investigation.

In general the cephalic (anterior) half of a swine-plague lung is hepatized, of a dark-red or grayish-red color and firm to the touch. The pleura is more or less thickened and opaque, and possibly covered with easily removable, friable, false membranes. In the more recently affected regions a faint but quite regular, delicate mottling with yellow is observed to shine through the pleura when not thick-ened. These minute hazy, yellowish dots usually occur in groups of four. Occasionally whitish or yellowish patches varying much in size are seen, perhaps more frequently in the ventral lobes. These correspond to homogeneous dead masses of lung tissue.

When such lungs are cut open, the section presents much the same appearance, both as regards color and mottling, as when viewed from the surface, excepting that the details are less distinct. In some cases, in the most recently invaded areas in the principal lobe and nearer the dorsum in the other lobes, the dark or grayish-red cut

surface shows grayish lines usually arranged in curves and circles. These, so far as determined, represent the cut outlines of the inter-lobular and peribronchial tissue infiltrated with cells. It has already been stated that these lines may represent the paths along which the swine-plague bacteria invade the lungs from the pleural surface.

The cut ends of the bronchi of the ventral lobes are frequently occluded with thick, whitish pus; in the other lobes a reddish froth is usually present. Rarely they also contain thick glairy mucus in which particles of dry pus and lung worms are imbedded. The con-tents of the air tubes in the ventral lobes may have been derived from the overdistended alveoli, or else a broncho-pneumonia may have preceded the swine-plague pneumonia.

In microscopic sections of diseased lung tissue the alveoli and smallest air tubes are found distended with cell masses consisting chiefly of leucocytes. Usually there is very little fibrin and very few red corpuscles in the alveoli, even in cases in which the disease was quite recent. It may be that the stage represented in ordinary croupous-pneumonia by the presence of fibrin in connection with the cellular elements is very brief, and that it is speedily replaced by large numbers of leucocytes. The large predominance of these elements in some portions of the lungs, as well as beginning fatty degeneration, is probably the cause of the regular mottling of the lungs, as seen from the surface. The little yellowish hazy dots represent alveoli surrounded by the hyperemic walls.

The necrotic and caseous changes so frequent in swine plague are most interesting. The latter are usually quite small and disseminated in large numbers over the diseased lobes. The former represent larger masses from a marble to a horsechestnut in size. They repre-sent tissue which has been destroyed by the rapid multiplication of swine-plague bacteria in particular localities. Hence they are found in all stages of the pneumonia. The large caseous masses may be considered as the result of a slow death of larger areas of lung tissue, due primarily to the gradual overdistention of the tissue by leuco-cytes, and hence the gradual cutting off of the blood supply. One is a rapid death due directly to highly virulent bacteria, the other a slow death, or a kind of dry suppuration in the later stages of the pneu-monia, characteristic of the pig, and due indirectly to the irritation of perhaps more attenuated races of bacteria. In some cases there are extensive hemorrhages in the interlobular connective tissue.

The inflammation of the pleura frequently extends to the peri-
cardium. This membrane is opaque, thickened and its vessels dis-
tended. It may be glued to the contiguous lobes of the lungs and
covered by a false membrane, smooth or roughened, which extends
upon the large vessels emerging at its base.

Disease of the digestive tract in a considerable proportion of
animals inoculated with swine-plague cultures consisted in a severe
catarrhal inflammation of the lining membrane of the stomach.
The hyperemia was very intense, bordering on hemorrhage. Occa-
sionally the extension of the peritonitis, produced by intra-abdominal
inoculation along the mesentery, causes a severe inflammation, with
exudation on the mucosa of the small intestine. A case is reported
where all the Peyer's patches of the small intestine were in a hypere-
mic and partly hemorrhagic condition.

In the naturally contracted disease extensive hyperemia of the
mucosa of the large intestine, bordering on a hemorrhagic condition,
has been observed. In other cases a peculiar croupous exudation
appeared, which seemingly resulted from the effect of swine-plague
bacteria in the large intestine.

The production of intestinal lesions by *Bact. suisepticum* may be
supposed to go on as follows: The bacteria first attack the lung
tissues and there produce more or less hepatization. The blood
through the lungs finds its path partly obstructed. This reacts on
the blood in the right side of the heart and the venous blood entering
it. Hence there may be more or less stasis of blood in the portal
circulation which in turn impairs the digestive functions of the
stomach. The swine-plague bacteria in the lungs in the later stages
of the pneumonia may be coughed up in the contents of the bronchial
tubes, swallowed and passed through the impaired stomach unharmed
into the intestines. The stagnation of the feces in the large intestine
furnishes the bacteria an opportunity to cause inflammation with
exudation on the mucous membrane. The tendency of swine-plague
bacteria to cause fibrinous inflammatory deposits on serous mem-
branes may serve to explain such action on mucous membranes.

There is general congestion with resulting degeneration of the
parenchyma of the spleen, kidneys and liver in the acute septicemic
forms of the disease. In these cases the specific bacterium is easily
obtained from the abdominal organs. In brief, the lesions of swine
plague as they appear in various outbreaks may be summarized in
four classes, namely:

The acute bacteriemia form in which the lesions are characterized by a general hyperemic condition of the serous membranes and parenchymatous organs. Not infrequently hemorrhages, especially the punctiform variety, occur. No localized lesions.

Cases of pneumonia with or without pleuritis. The other organs remain normal in appearance.

Cases where either in addition to, or possibly in the absence of, the lung lesions there are marked anatomical changes in the mucosa of the digestive tract and possibly in the lymphatic glands.

Cases of mixed infection, especially with hog cholera, where in addition to the swine-plague lesions which may be more or less modified, there are those, especially of the digestive tract, characteristic of the accompanying disease.

The course of the disease varies in acute cases from one to three weeks. In chronic or complicated cases it is indefinite.

The prognosis in swine plague is very unfavorable. Most of the affected animals die, and those that recover are usually not thrifty.

Diagnosis. Swine plague is diagnosed from the symptoms, lesions and the bacteriological examination. There are no specific reactions that have been found to be satisfactory in making a diagnosis. Swine plague is to be differentiated from broncho-pneumonia due to other causes than the swine-plague bacterium. Pneumonia of a non-specific nature is often associated with deaths due to dietary or other causes. Pneumonia frequently produces death in chronic cases of other diseases (terminal pneumonia).

In enzoötics or outbreaks, it is to be distinguished from hog cholera accompanied with catarrhal pneumonia.

Pneumonia resulting from lung worms (*Strongylus paradoxus*) is to be distinguished by a careful examination of the contents of the bronchioles.

In case of coexistence of hog cholera and swine plague a bacteriological examination and also the inoculation of pigs with the filtered blood serums, are necessary to determine the presence of the two diseases, owing to the possibility of an accompanying or terminal pneumonia with hog cholera.

The question has arisen as to whether the presence of *Bact. suisepticum* in the hepatized lung constitutes a diagnosis of swine plague. As understood at the present time it would seem that the presence of this species of bacteria would indicate the nature of the disease. It

must be remembered, however, that bacteria not readily distinguishable from the swine-plague organism exist in the normal upper air passages, from whence they could be brought into the lung and in such a case it might appear as a secondary invader only, or, it might have been primarily the cause of the lesions. It is not improbable that the disease may start from these sporadic cases, although conclusive proof of this is still wanting. It seems, however, that the presence of this organism in the lung tissue of a sporadic case should be considered in the light of the distribution of these organisms and not necessarily as the beginning of an enzoötic.

Prevention. The well animals should be promptly separated from the sick and placed in suitable pens or yards, protected against subsequent infection, and given wholesome food and water.* It is well to remove the sick animals to other pens. The infected pens should be thoroughly disinfected before they are again occupied.

Specific biologic treatment. Although several attempts have been made to produce a specific biological remedy for swine plague, they have not been successful.

REFERENCES

1. DE SCHWEINITZ. Serum therapy. *Proceedings Society for the Promotion of Agricultural Science*, 1896, p. 47.

2. DE SCHWEINITZ. The serum treatment of swine plague and hog cholera. *Bulletin No. 23, U. S. Bureau of Animal Industry*, 1899.

3. EVANS. Hämorrhagische Septikämie des Elephanten. *The Jour. of Tropical Vet. Science*, Vol. 1, p. 283.

4. JOEST. *Schweineseuche und Schweinepest.* Jena, 1906.

5. LOEFFLER. *Arbeiten a. d. Kaiserlichen Gesundheitsamte*, Bd. I (1885), S. 51.

6. MOORE. Pathogenic and toxicogenic bacteria in the upper air passages of domesticated animals. *Bulletin No. 3. U. S. Bureau of Animal Industry*, 1893.

7. MOORE. Concerning the nature of infectious swine diseases in the State of New York with practical suggestions for their prevention and treatment. *Report of the New York State Commissioner of Agriculture*, 1897.

8. SMITH. Preliminary investigations concerning infectious pneumonia in swine (Swine plague). *Ann. Rpt. Bureau of Animal Industry, U. S. Dept, of Agriculture*, 1886, p. 76.

9. SMITH. Special report on swine plague. *Bureau of Animal Industry, U. S, Dept. of Agriculture*, 1891, p. 47.

10. SMITH AND MOORE. Experiments on the production of immunity in rabbits and guinea pigs with reference to hog-cholera and swine-plague bacteria. *Bulletin No. 6, Bureau of Animal Industry, U. S. Dept. of Agriculture*, 1894, p. 65.

11. WELCH AND CLEMENTS. Remarks on hog cholera and swine plague. *First International Veterinary Congress of America.* Chicago, 1893.

*As swine plague is frequently associated with hog cholera. it is recommended by many that the well pigs should be immunized against that disease.

HEMORRHAGIC SEPTICEMIA IN CATTLE

Synonyms. Wild und Rinderseuche: Pasteurellosis bovum; *Sépti-cémie hémorrhagique du boeuf; Septicaemia pluriformis.*

Characterization. Hemorrhagic septicemia in cattle is determined by an acute attack usually running a rapid course and terminating fatally. The lesions consist largely of hemorrhagic areas more or less widely distributed throughout the body and due to the presence of *Bacterium bovisepticum*. It occurs more frequently in cattle. It has been described, however, in other ruminants, horses and mules. Men, dogs and fowls are reported by Gaiger to be immune.

History. In 1878, Bollinger described under the name of *Wild-und Rinderseuche* an epizoötic disease which killed a large number of wild boars and deer in the Royal game preserves of Munich. After the disease in these animals had died out, the domestic cattle in the neighborhood began to die of the same or a very similar affection. He reports it to be sudden in its onset and rapidly fatal in its course, with a mortality of 90 per cent. Death occurred in from 12 hours to a few days after the appearance of symptoms.*

In 1885, Kitt studied an epizoötic disease in cattle and swine in Sincbach. From this disease he isolated a short, polar staining, non-motile, rod-shaped organism, fatal to cattle, horses, pigs, sheep, goats, dogs and rabbits. In the following year Oreste and Armanni reported a destructive disease of young buffaloes in Italy with symptoms and lesions similar to those reported by Bollinger and Kitt. This disease had been known in Italy for a century or more, where in certain districts it is reported to have recurred with great regularity, destroying both old and young animals. In the same year (1885) Poels described a septic pleuro-pneumonia in calves which prevailed in the vicinity of Rotterdam. It was of a septicemic nature. From the organs he

---

*An exanthematous and a pectoral form are described. In the exanthematous form there are large and small hemorrhages disseminated throughout the muscles and viscera. The intestines exhibit large numbers of ecchymotic areas, while the submucous tissue is infiltrated with a serous exudate. Large hemorrhagic tumors infiltrated with serum are abundant in the subcutaneous tissue, often extending into the muscles. The mucous membranes of the tongue, larynx and pharynx, and the lymphatic glands of these regions, are swollen and infiltrated with more or less bloody serum. In the pectoral form, there is a hemorrhagic lobular pneumonia, with considerable infiltration into the interlobular tissue of a serofibrinous exudate. The pleura is infiltrated and inflamed and covered with a fibrinous exudate. The pleural cavities contain from two to twenty-five litres of liquid. At the same time there may exist a hemorrhagic enteritis and the widely disseminated hemorrhagic lesions common to the preceding form.

obtained an organism belonging to the *Bacillus septicemiae hemor-rhagicae* group of bacteria. In 1889, Jensen described a similar disease affecting calves in Jutland. In the same year Piot reported the presence of "barbone" in the buffaloes and domestic cattle in Egypt. In some districts 40 per cent. of the horned cattle are said to have died in a single year. It is reported as being more prevalent in the wet season.

In 1890, Van Ecke described a hemorrhagic septicemia in cattle in Dutch India, particularly in Java, the lesions of which were similar to those first described by Bollinger. The specific organism was virulent for rabbits, mice, turtle doves, calves, horses and swine; sheep and asses were almost immune. In the following year Galtier described the same disease imported from Algiers to Lyons. A number of other investigators have studied and reported cases of this disease. In 1890, Nocard isolated from cases of broncho-pneumonia in American cattle landed at La Villette, France, an organism similar to that described as the cause of septicemia hemorrhagica. In 1896, Smith called attention to a similar organism which he found in cases of sporadic pneumonia in cattle. These cases have been referred to as the pectoral form of the disease. As early as 1891, Smith published the discovery of the presence of an organism, morphologically and in its cultural characters identical with that of swine plague, in the upper air passages of healthy swine. The same year Moore reported the presence of a like organism in the upper air passages of cattle, horses, sheep, dogs and cats. The following year, Fiocca described a pathogenic bacterium resembling that of rabbit septicemia in the saliva of healthy cats and dogs.

Hueppe proposed the name *Bacillus septicemiae hemorrhagicae* for this group of organisms and septicemia hemorrhagica for the disease they produce. Lignières has designated the diseases caused by this group as *Pasteurelloses*. While there may be objections to this unifying name, there seems to be no serious reason for not accepting it as a working hypothesis. In 1898, Fennimore described under the name of "Wild and Cattle Disease" a malady occurring in Eastern Tennessee. Its serious nature caused an investigation to be made by the Tennessee Agricultural Experiment Station. Nörgaard, who assisted in this investigation, recognized it as the same disease as that described by Bollinger in 1878. Fennimore states that it has occurred to a considerable extent in his practice. In 1901, it was carefully studied by Wilson and Brimhall for the Minnesota State

Board of Health. They report 64 cases of this affection which they have examined in cattle in the state of Minnesota. In 1903, Reynolds described an investigation into several outbreaks of this disease in the same state.

Geographical distribution. It will be seen from the history that this disease is a wide spread malady occurring in nearly every country. It appears to be more or less prevalent in the temperate and tropical countries. It has been reported from many places in the United States.

Etiology. Septicemia hemorrhagica in cattle is caused by an organism belonging to the group of bacteria designated by Hueppe as the hemorrhagic septicemia group and specifically named *Bacillus bovisepticus* by Kruse. This, according to Migula's classification, should be *Bacterium bovisepticum*. A brief description of the organism as given by Wilson and Brimhall is appended.

"The organism has a strong tendency to show polar staining in tissues and to form chains of much shortened individuals in liquid media, which causes it to be mistaken in examinations of a single specimen for a diplococcus or a streptococcus. Sometimes in cover-glass preparations from solid organs and very frequently in those from body fluids and liquid cultures, the bacteria were found in chains of three to twelve individuals. In cover-glass preparations the bacteria are from 0.6μ to 0.8μ broad and from 1.0 to 1.5μ in length. In tissues which have been fixed in 96 per cent. alcohol, they are somewhat smaller. In cultures, especially in fluid media, they are apt to be much smaller and approach diplococci in appearance. The ends are rounded. In stained preparations directly from the tissues most of the bacteria have the ends intensely stained and the central portion but faintly so. In some chains in rapidly growing broth cultures this is not the case, many of the individual bacteria being evenly stained throughout and somewhat pointed at the ends. They do not retain the stain by Gram's method. The organism is non-motile. It is aerobic, but prefers the depths rather than the surfaces of the media. It grows best at the body temperature and more slowly at room temperature. In plain and dextrose broth a growth appears in 24 hours. In Dunham's solution a small amount of indol is formed in 48 hours. No coagulation of milk. On Löffler's blood serum, direct from the diseased tissues, it failed to grow well. On potato no appreciable growth has been obtained. In gelatin plates small, granular, white to slightly yellowish colonies appear after 48 hours. In gelatin stab cultures a light growth occurs on the surface, while along the needle tract numerous colonies like those in the deep portions of the plate cultures develop. The bacteria are destroyed in fluids at $58°$ C. in 7 or 8 minutes, by 1 to 5,000 mercuric chloride in one minute, and by a solution of lime water as weak as 0.04 per cent. almost immediately."

The period of incubation is supposed to be very short. The method of infection is not known.

Symptoms. The animals observed at the onset of the disease by Wilson and Brimhall appeared to be dumpish and out of sorts. There is sudden stopping of the milk secretion in milch cows. As a rule the affected animals refuse food. Of the few that make an attempt to eat, those with affected throats are unable to swallow except with much difficulty. These cases also breathe very heavily. The animals show marked disinclination to move and when incited to do so, exhibit stiffness, and in some instances actual lameness. Animals have been observed to drop to the ground and die in a short time, apparently without pain. Other animals live for several hours in great pain as indicated by groans and spasms of the muscles. The paroxysms of pain are apparently intermittent. There is extremely rapid loss of flesh in the animals that are sick for any length of time.

Reynolds observed the symptoms in a few cases. He describes three stages. First (24–36 hours), general dullness and checking of milk secretion. Second, diarrheal discharge dark in color, and of disagreeable odor. The breath was noticed to be offensive. In some cases nervous symptoms developed. The temperature remained about normal during this period. Third, in this stage the eyes are wild, there are grinding of the jaws and convulsions of the face and neck muscles followed by a period of intense restlessness and activity.

He also records the observation that the cases that appeared to be the worst in the beginning lived longer than those that appeared to be mild.

A temperature of 104° to 106° F. may be followed by a rapid decline. There are accelerated pulse, dullness and rough coat. Painful edematous swellings about the legs, shoulders and under the throat are noted as early symptoms. The intestinal discharges are often streaked with blood. In other cases the feces are black, tarry or of a bloody, serous nature. Bloody urine and a bloody serous discharge from the nose have been reported. The vaginal and rectal mucous membranes are intensely congested.

The marked swelling of the face, stomatitis, glossitis, and convulsive movements of the jaws in the pneumonic form of the disease, described by European writers, more especially Bollinger, were not observed by Wilson and Brimhall or by Reynolds. The edematous form is stated by Hutyra to be more rapidly fatal.

The pectoral form exhibits symptoms of an acute pleuro-pneumonia. There is a dry and rather painful cough. This form has been mistaken for contagious pleuro-pneumonia.

The duration of the disease is short. Often the animals are found dead.

The prognosis is unfavorable. The mortality is placed at from 80 to 90 per cent. of the animals affected.

Morbid anatomy. The characteristic lesions of the disease are widely distributed areas of hemorrhage, varying in size from a pin point to several centimeters in diameter. They vary in color from light red to almost black. They are frequently accompanied with a sero-fibrinous exudate, usually yellow, but occasionally dark red in color. The hemorrhagic areas in the animals just dead are not so dark as those in animals that have been dead for some hours. The large areas, some centimeters in diameter, are apparently due, in some instances at least, to single hemorrhages, infiltrating an extensive mass of tissue, and in others to a number of minute hemorrhages closely placed and partially coalescing. Gas is not present in the subcutaneous connective tissue except in cases where extensive postmortem changes have occurred.

There is extensive fullness of the vessels of the subcutaneous connective tissue in the acute cases, especially in those animals which are not killed by bleeding. In animals which live until emaciation is marked, there is no engorgement of the vessels.

Reynolds reports one outbreak in which meningitis involving the spinal cord, brain or both of these organs was invariably present.

All cases show some hemorrhagic areas in the subcutaneous tissue, though the number and size of these vary greatly in different individuals. Some animals exhibit very few, while others, on removing the skin, present hemorrhagic areas or petechiae in large numbers and so extensive that a large fraction, possibly one-eighth, of the body surface appears to be involved. The large hemorrhages in the subcutaneous connective tissue appear to be of the composite type noted above.

The location of the superficial lesions varies in different animals. In most cases the parts about the shoulder are most affected. A few animals show marked lesions in the gluteal and inguinal regions.

At first sight the muscle tissue in some cases seems to be much involved. A closer examination, however, usually shows that while some of the minute hemorrhages are in the muscle proper, the larger ones are in the intermuscular connective tissue. They are usually accompanied by a considerable quantity of yellowish or blood-stained

serous exudate.   The intermuscular connective tissue appears to be
quite as much involved as the subcutaneous connective tissues.

The lymphatic g'ands are frequently though not uniformly en-
larged.   Those that are enlarged are edematous or hemorrhagic.
The cervical and prescapular glands are most seriously affected.
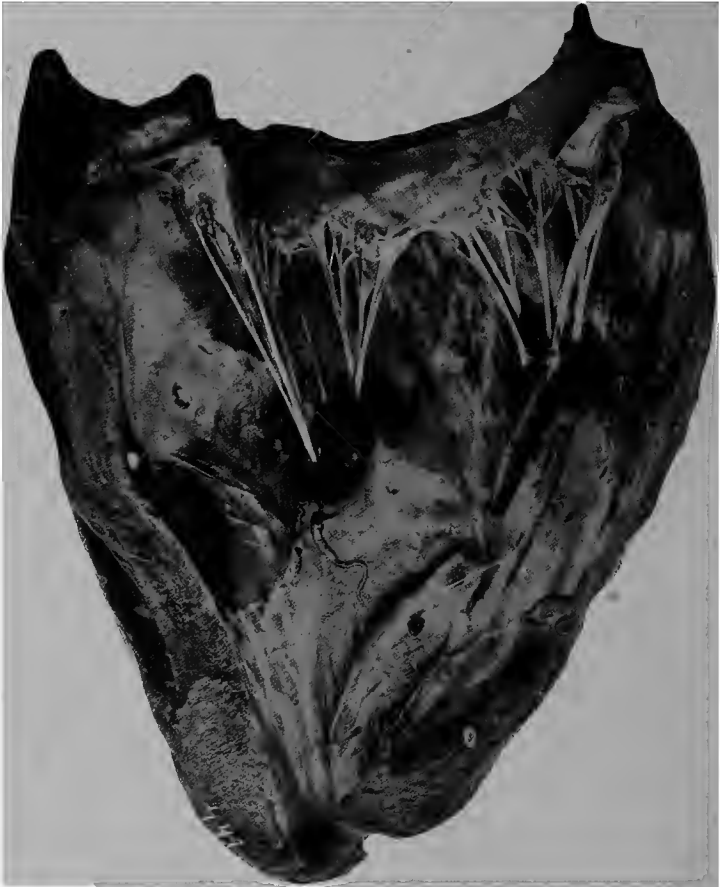
Fɪɢ. 6.   Pʜᴏᴛᴏɢʀᴀᴘʜ sʜᴏᴡɪɴɢ ʜᴇᴍᴏʀʀʜᴀɢᴇs ʙᴇɴᴇᴀᴛʜ ᴛʜᴇ ᴇɴᴅᴏᴄᴀʀᴅɪᴜᴍ ᴏғ ᴛʜᴇ
RIGHT VENTRICLE (REYNOLDS).

The nasal mucous membrane in some cases is congested, and a
bloody serous discharge from the nostrils is present.   The tissues
around the larynx are hemorrhagic and infiltrated with blood-stained
serum.   The mucous membranes of the larynx and trachea are more

or less congested and covered with a frothy mucus, sometimes streaked with blood. In some instances no lesions are observable in these organs. The lungs, except in the pectoral form, are in general almost free from lesions. Occasionally there are a small number of hemorrhagic areas, pyramidal in shape with their bases on the pleura. In most cattle the parietal pleura is studded with small hemorrhages. The diaphragm sometimes contains very large hemorrhagic areas. In the pectoral form of the disease there is a broncho-pneumonia together with interlobular edema. The edematous fluid is usually clear but rich in the specific organisms.

The pericardial sac usually shows small, sometimes very numerous, hemorrhages in the walls, and in many instances contains a blood-stained serum.

The heart walls, with but few exceptions, contain ecchymoses and petechiae. These sometimes extend deep into the muscle. Similar areas of hemorrhage are also visible in the endocardium. The heart usually contains post-mortem blood clots.

The blood, in animals just dead, is said to be somewhat lighter than normal in color. When post-mortem changes set in, the blood is darker in color, but reddens on exposure to the air.

The spleen shows on its surface a few small hemorrhagic areas. It is usually normal in size, color and consistency.

Stomatitis and glossitis are rarely observed. The pharynx is usually congested.

The stomach walls contain a few or many hemorrhagic areas. These are sometimes extremely large, especially on the third stomach. As a rule the larger hemorrhages penetrate the entire thickness of the walls. The smaller ones are confined to the subserous or mucous coats. The stomach contents are apparently normal.

The intestinal walls are constantly affected. Hemorrhagic areas involving all the coats are frequently present. Smaller ones, visible only from the inner or outer surface, are always present. General enteritis and peritonitis are constantly observed. Localized enteritis is frequent.

The bowel contents are in some cases dark and tarry; in others the feces are apparently normal in color and consistency but streaked with bloody mucus.

The kidneys are usually but slightly affected. When lesions are present they consist of pin point hemorrhages and mostly confined to

the cortical substance, though a few are found in the walls of the pelvis and ureters. The urine is bloody in some instances.

The vaginal mucous membrane is congested in many cases. Wilson and Brimhall saw one animal that was four months pregnant which exhibited small hemorrhagic areas in the placental membranes.

The udder is congested in some cases. It may be hemorrhagic. Occasionally there are hemorrhages in the dura. A few cases are recorded of hemorrhages on the joint surfaces. Brimhall reported an outbreak where the autopsies showed very few hemorrhagic areas in the subcutis and internal organs but there were lesions in the spinal cord. The spleens were enlarged. In nine outbreaks, *Bact. borisepticum* was present.

Wilson and Brimhall fixed portions of the subcutaneous tissue, skeletal muscles, lymphatic glands, lung, heart wall, stomach wall, and spleen in 95 per cent. alcohol and in 4 per cent. formaldehyde solution and stained by various methods. In general the lesions found were enormous extravasations of blood, some recent and some showing coagulation of fibrin. In the areas of less recent hemorrhage, the surrounding tissues showed varying degrees of ordinary coagulation necrosis. This was particularly marked in the affected muscles, lymph glands and portions of the lungs. In the borders of such necrosed areas leucocytic infiltration was not infrequent. In the spleen, in which the hemorrhagic areas were neither numerous nor large, there was in some instances an apparent destruction or shrinkage of the parenchyma.

A very important feature in this disease has been brought out, namely, that it is necessary, in order to obtain cultures of the bacterium producing it, that the media should be inoculated very soon after death. With this precaution cultures should be obtained. It is reported that the lymphatic glands are the most reliable organs from which to make cultures.

Diagnosis. Septicemia hemorrhagica in cattle is diagnosed by the symptoms, the lesions and the bacteriological examination. There are no specific tests that can be applied with satisfactory results. It is to be differentiated from anthrax, symptomatic anthrax, death due to accidental causes, poisoning, or the effect of over eating of grain or green fodder (hoven). Death from any of these causes may be very sudden. It is necessary, therefore, that in all cases, especially with the first animals to die, careful postmortem and bacteriological examinations should be made.

In case of septicemia hemorrhagica, the cultures will usually reveal the presence of *Bact. bovisepticum*. The lesions will be hemorrhagic in nature.

With anthrax and symptomatic anthrax, the specific bacteria will be found and with anthrax the Ascoli test may be tried as well as the M'Fadyean stain.

Prevention. When this disease occurs, it is important to remove the unaffected animals to other fields or enclosures. It is well to divide them into small groups if possible. The carcasses of animals that die should be burned or buried deeply with a good covering of a disinfectant, such, for example, as quicklime. Should death occur in a stable, all contaminated litter should be burned or thoroughly disinfected as well as the floors, mangers and walls.

Mohler has reported success in immunizing buffalo against this disease by means of a bacterin prepared from a culture of the organism. Bliss and Carrougeau also recommend immunization.

Control. Septicemia hemorrhagica is considered by Hutyra and others as a disease of the soil. This renders unnecessary extensive measures restricting cattle traffic. In outbreaks the well animals should be separated from the infected. The specific organism dies soon after drying. Hides from animals that have died become innocuous as soon as they become dry. Because of the rapid death of the organism and the probable soil origin of the germ it is not necessary to impose rigid traffic restrictions.

Septicemia hemorrhagica in other species. There is considerable literature on the presence of septicemia hemorrhagica in sheep and more rarely in goats, horses and swine. In most cases the lesions seem to take on the form usually found in cattle but modifications are numerous.

Miessner and Schern give the following in reference to this disease in sheep: "The acute stage is for the most part observed in lambs. The animals die within 24 hours without having shown any striking symptoms of the disease. The post mortem examination shows a hemorrhagic infiltration of the subcutis, stasis in the mucous membranes of the head, hemorrhagic lymphadenitis in the region of the head, sometimes hemorrhagic tracheitis.

"The subacute stage is characterized by a discharge from the eyes and nose and dyspnœa. It terminates fatally as a rule within 14–21 days. The most important post mortem changes consist in a pneumonia and pleuritis.

"The chronic stage occurs in young and older animals, and is characterized by emaciation and respiratory disturbances. On post mortem examination one finds a watery condition of the muscles, fluid in the serous cavities, and sometimes residues of a pneumonia and pleuritis.

"Sometimes one observes complications such as ulcers of the gums, falling out of the teeth, proliferations and ulcerous changes on the lips and the udder.

"The work shows further, that the septicemia pluriformis ovium observed in Germany, resembles very much the disease occurring in France and Argentina under the name of Pasteurella or Lombritz. The authors consider these diseases as identical."

REFERENCES

1. BOLLINGER. Ueber eine neue Wild und Rinderseuche. München. 1878.

2. BRIMHALL. Haemorrhagic septicemia in cattle. *Amer. Vet. Rev.*, Vol. XXVII (1903–4), p. 103.

3. FENNIMORE. Wild and cattle diseases. *Jour. of Comp. Med. and Vet. Archiv.*, Vol. XIX (1898), p. 625.

4. GAIGER. Beitrag zum Studium der Hæmorrhagischen Septikämie. *Jour. of Tropical Vet. Sci.*, Vol. IV.

5. GALTIER. Nouveaux faits tendant à établir que la pneumo-enterite infectieuse existe sur les grands et les petits ruminants en algerie. *Recueil de Méd. Vét.*, 7 serie, Vol. VIII (1891), p. 97.

6. HUEPPE. Ueber die Wildseuche. *Berlin klinische Wochenschrift*, 1886, S. 753.

7. KITT. Ueber eine Experimentelle, der Rinderseuche (Bollinger) ähnliche Infectionskrankheit. *Sitzungsberichte der Gesellschaft für Morphologie und Physiologie in München*, Bd. I (1885), S. 240.

8. MIESSNER AND SCHERN. Septicemia pluriformis ovium. *Archiv. f. wissen. u prak. Tierheilk.* Bd. XXXVI, S. 44 u. 208.

9. POELS. Septische Pleuro-Pneumonie der Kälber. *Fortschr. d. Med.*, 1886, S. 388.

10. REYNOLDS. Hæmorrhagic Septicemia. *Am. Vet. Review.* Vol. XXVI (1902), p. 819.

11. REYNOLDS. Hæmorrhagic Septicemia. *Bulletin No. 82, Minn. Agric. Experiment Station*, 1903.

12. WILSON AND BRIMHALL. Sixty-four cases of hemorrhagic septicemia in cattle due to bacillus hovisepticus. *Report State Board of Health of Minnesota*, 1901. (Very full bibliography.)

13. WOOLLEY AND JOBLING. A report on hemorrhagic septicemia in animals in the Philippine Islands. 1903. *No. 9. Bureau of Government Laboratories, Manila*, P. I.

FOWL CHOLERA

Synonyms. Chicken cholera; cholera gallinarum; *Hühner cholera; Pasteurellosis avium; choléra des poules.*

Characterization. This is an infectious disease of fowls caused by bacteria, and transmissable by cohabitation and inoculation. It is determined by a high fever, great weakness and prostration, and usually terminates in the death of the affected bird. It is reported that it attacks all varieties of domesticated poultry (chickens, ducks, geese, pigeons, turkeys), and caged birds such as parrots and canaries. It also attacks some species of wild birds. It is communicable by inoculation to rabbits and mice. Guinea pigs are less susceptible.

History. This disease is mentioned in some of the oldest works treating of the diseases of animals. Fowl cholera was studied by Chabert in 1782, who regarded it as a form of anthrax. Since 1825, it has frequently been observed in France where it caused enormous losses in 1830, in 1850 and in 1860. About 1830, it became known in Russia, Bohemia and Austria. In 1851, Benjamin considered it to be a contagious disease but remarked that people and dogs might consume with impunity the meat of affected fowls. Delafond observed that it might be transmitted to birds and rabbits by using blood, secretions, and portions of the flesh. It was also recognized that the excrement plays an important part in the dissemination of the virus.

During recent years it has been observed in nearly all of the countries of Europe as well as in the United States. It has been reported from many places in the United States, but its presence seems to have been determined by scientific investigation in but a very few of these. Salmon investigated it in South Carolina in 1879–80, and Higgins in 1898 reported it from Canada. Salmon gave special attention to vaccination and the effect of disinfectants in destroying the virus. In 1904, Ward reported an outbreak in California.

Perroncito was among the first to describe the specific cause of the disease. This was followed by the contributions of Pasteur, who, in 1880, cultivated the bacterium in chicken broth and showed that its virulence might be reduced to such an extent that it could safely be used for vaccination. This was the first time that a virulent organism was successfully modified in a laboratory and used as a vaccine. It was the forerunner of the preparation of vaccines for a number of diseases, more particularly for anthrax, black quarter, and rabies.

Geographical distribution. Fowl cholera seems to be widely distributed in Europe and it has been found in many places in the United States and in Canada. In Germany it is the cause of heavy losses among poultry. In 1903 it is reported to have killed over 48,000 fowls and 23,000 geese, besides other poultry.

Etiology. Fowl cholera is caused by a specific bacterium, *Bact. cholerae-gallinarum* (*Bacillus* (*bipolaris*) *avisepticus, Pasteurella avium*), which is not distinguishable morphologically or in its cultural manifestations from the other members of the Pasteurella. According to Gærtner, it will remain alive in manure for at least three months. It is reported to live in putrefactive carcasses and in garden soil for an equal length of time. Kitt states they resist freezing for at least 14 days. Natural infection usually takes place by ingestion.

The period of incubation is placed by European writers at from 18 to 48 hours. In the case of 40 fowls inoculated by Salmon, it varied from 4 to 20 days, the average period being 8 days. Ward fed viscera of dead fowls to 10 healthy ones. They died in from 24 hours to 6 days.

Symptoms. The symptoms described for this disease in Europe differ somewhat from those reported by Salmon. The appetite is often affected and occasionally the fowls continue to eat almost to the time of death. The earliest indication of the disease is a yellow coloration of the urates. In health, these are a pure white though they are frequently tinted with yellow as the result of disorders other than cholera.

Occasionally the first symptom is a diarrhea in which the excrement is passed in large quantities and consists almost entirely of white urates mixed with colorless mucus.

Very soon after the first symptoms appear the bird separates itself from the flock, it no longer stands erect, the feathers are roughened, the wings droop, the head is drawn down towards the body and the general outline of the bird becomes spherical or ball-shaped. At this period there is great weakness, the affected bird becomes drowsy and may sink into a sleep which lasts during the last day or two of its life, and from which it is almost impossible to arouse it.

The crop is nearly always distended with food and apparently paralyzed. There is in most cases intense thirst. If the birds are aroused and caused to walk, there is at first an abundant discharge of excrement followed at short intervals by scanty evacuation.

With the beginning of diarrhea the body temperature has been found to rise to 109 to 110° F. Ward states that in advanced stages it ranges from 109 to 112° F. The comb loses its bright hue and becomes pale and bloodless. European writers describe the comb as dark blue, purple, or black, and some writers in the United States have referred to it in the same terms. Salmon reports that he never observed it.

Diseased birds rapidly lose in weight. They become so weak that they walk with great difficulty, a slight touch causes them to fall over. The fowls become very much emaciated. Death may occur without a struggle or there may be convulsive movements and cries.

This disease may run rapidly through a flock destroying the greater part of the birds in a week, or it may assume a more chronic form, spreading slowly, and remain upon the premises for several weeks or months.

The duration of the disease varies from a few hours to several days.

The prognosis is unfavorable. The mortality is very high, often 100 per cent.

Morbid anatomy. The comb is pale and bloodless. The superficial vessels usually contain but little blood. In cases of acute cholera there is a tendency to hemorrhagic inflammation of the intestines.

The liver is usually enlarged, softened, and the blood vessels are engorged. The gall bladder is distended with thick, dark bile.

The crop is usually distended with food. The stomach often presents externally a number of circular discolorations, about three millimeters in diameter, which on section are found to be extravasated blood. The small intestines are congested.

The rectum and cloaca usually present deep, red lines upon their mucous membrane, evidently the first stage of inflammation, which results, in chronic cases, in thickening of the walls, especially of the rectum, the desquamation of the mucous membrane and the formation of large ulcerated surfaces.

The mesentery is generally congested, often greatly thickened and reddened, and rendered opaque by inflammation. The ureters are distended with yellow urates; the kidneys seem engorged, and on section accumulations of the tenacious, yellow urates are frequently

seen. The spleen is generally normal in size and appearance, though frequently enlarged and softened.

The pericardium is sometimes distended with effusions, in which case there is noticeable hyperemia of the surface of the heart. The lungs are often, though not generally, engorged with dark blood; they are seldom, if ever, hepatized.

The blood vessels are sometimes filled with a firm clot, and contain but little liquid; at other times the blood does not coagulate at all. It seems to be those cases in which the duration of the disease is the longest, that the blood loses its power to coagulate.

Ward has summarized the morbid anatomy in the fowls examined by him as follows:

"At death, or some hours previous, the comb frequently takes on a dark purple color, but this does not always occur. Very often the comb is pale and bloodless. The skin of the breast and abdomen is frequently reddened.

"In post-mortem examinations a congestion of the blood-vessels of the liver, kidney, mesentery, or intestines is noticeable to some degree in all cases. Punctiform hemorrhages are found upon the heart with almost absolute uniformity. The liver is very frequently marked with punctiform whitish areas of necrosis. Stained sections show these necrotic foci throughout the substance of the liver, and besides reveal a congestion of the blood-vessels of that organ. The next most striking lesions occur in the first and second duodenal flexures. The mucosa is deeply reddened and studded with hemorrhages varying in size, but seldom exceeding one millimeter in diameter. These involve the intestinal coats to an extent that makes them distinctly visible on the peritoneal surface. The contents of the duodenum consist of a pasty mass, more or less thickly intermingled with blood clots. The intestinal contents sometimes consist of a cream-colored pasty mass, or may be brownish red or even green in color. Lesions are very rarely observed in other portions of the intestines. The ureters are noticeable in practically all cases by reason of the yellow-colored urates that they contain. The nasal cavity, pharynx and oral cavity frequently contain a viscous mucous fluid, probably regurgitated from the crop.

"The field notes on twenty-one post-mortem examinations refer to hemorrhages in the heart in twenty-one cases; punctiform necroses in the liver, fifteen cases; hemorrhages in duodenum, seven cases; the discoloration of the skin in six cases. The presence of a gelatinous exudate within the pericardium was noted twice. A fibrinous exudate in the pericardium occurred the same number of times. Hemorrhages in the peritoneum other than those visible through the mucosa of the duodenum occurred but twice. In one case hemorrhages were abundantly scattered throughout the muscles of the trunk and legs.

"Fowls inoculated subcutaneously with cultures exhibit on post-mortem examination the punctiform hemorrhages on the heart and the hemorrhages in the mucosa of the duodenum exactly as in cases infected naturally."

BLOOD COUNTS OF FOWLS INFECTED BY INGESTION AND INFECTED NATURALLY

Fowl.	White Corpuscles.	Red Corpuscles.	Remarks.	Temperature.
No. 3	23,000	2,290,000 per cmm.	3 days after exposure to infection	...44.8° C.
No. 3	20,000	2,800,000 " "	4 " " " " "	...43.7° C.
No. 6	37,000	3,930,000 " "	3 " " " " "	...43.3° C.
No. 8	87,000	4,490,000 " "	3 " " " " "	...42.8° C.
No. 8	101,000	2,960,000 " "	4 " " " " "	...42.2° C.
A 58,000		1,710,000 " ··	Naturally infected	...........42.8° C.
B 45,000		1,925,000 " "	" "	....

BLOOD COUNTS OF APPARENTLY HEALTHY FOWLS

Fowl.	White Corpuscles.	Red Corpuscles.
No. 11	24,000	2,980,000 per cmm.
No. 12	26,300	2,987,000 " "
No. 14	36,000	3,115,000 " "
No. 15	52,000	3,980,000 " "
No. 16	61,000	3,920,000 " "
No. 17	30,000	2,380,000 " "
No. 18	24,000	2,620,000 " "

Diagnosis. Fowl cholera is diagnosed by the symptoms, lesions and bacteriological examination. The specific cause is not difficult to isolate. Fowl cholera is to be differentiated from:

A number of dietary disorders which cause the death of a large number of fowls. Such cases are often thought to be chicken cholera and so reported by the owners. A diagnosis is to be made from the bacteriological findings.

It is to be differentiated from fowl typhoid. There are a number of resemblances in the clinical history of the two diseases. There are, however, marked differences in both the morbid anatomy and etiology For a comparison see fowl typhoid.

Prevention. Pasteur introduced a preventive inoculation or vaccine for this disease. Kitt found that the eggs of fowls unknown to this disease possessed a substance somewhat similar to antitoxin. He immunized fowls by injecting them simultaneously with from four to eight cubic centimeters of the whites of such eggs. More recently he has obtained a horse serum that promises to be of immunizing value. Jensen obtained good results in immunizing geese against this disease by the use of serums. There does not appear, however, to be a reliable immunizing agent. Hadley has used subcutaneous injections of diluted carbolic acid.

Good sanitary conditions, isolation of the well from the sick fowls and thorough disinfection seem to be the most satisfactory procedure. It is important not to introduce the disease with newly purchased fowls, or to expose healthy ones to the disease either at or in transportation to various poultry exhibits.

Control. Fowl cholera is a reportable disease in Germany, Austria and Hungary. In those countries the infected premises are quarantined against traffic in fowls. The infected places should be thoroughly disinfected. Care should be taken not to introduce infected fowls into healthy flocks. The fowls that appear to be sound but which are in either the period of incubation or have recovered from the disease are very liable to spread the infection.

REFERENCES

1. HADLEY. Fowl cholera and methods of combating it. *Bulletin 144, R. I. Agr. Exp. Station*, 1910.

2. HIGGINS. Notes upon an epidemic of fowl cholera and upon the comparative production of acid by allied bacteria. *Jour. of Experimental Medicine*, Vol. III (1898), p. 651.

3. KITT. Die Serumimpfung gegen Geflügelcholera. *Monatshefte für praktische Tierheilk.*, Vol. XVI (1904), S. 1.

4. PERRONCITO. Ueber das epizootische Typhoid der Hühner. *Arch. für wiss. u. prackt. Thierheilk.*, 1879.

5. PASTEUR. De l'attenuation du virus du cholera des poules. *Comptes rendus des Seances de l' Academie des Sciences*, Vol. XCI (1880), p. 673.

6. PASTEUR. Sur les maladies virulentes, et en particulier sur la maladie appelée vulgairement choléra des poules. *Ibid.* Vol. XC (1880), p. 239.

7. SALMON. *Annual Reports of the U. S. Commissioner of Agriculture*, 1880–82.

8. SALMON. The diseases of poultry. Washington, D. C. 1889, p. 232.

9. WARD. Fowl cholera. *Bulletin No. 156. College of Agric., Calif. Agric. Exp. Station*, 1904.

GOOSE SEPTICEMIA

Characterization. The disease is an acute septicemia causing the death of the infected goose in a few hours after there are evidences of sickness.

History. In 1902, Curtice described this disease as causing considerable loss in Rhode Island. The following note by T. Smith, dated October 31, 1900, quoted by Curtice, is significant in explaining the condition under which the disease appeared.

"Geese born in April and May and collected during the summer and fall for fattening, kept in open yards, crowded together but able to move about; about 500 in a pen. Fed on a mixture of corn meal and meat and beef scraps. Epidemic began in midsummer. Deaths up to twenty a day (one workman says sixty one day); about 3,000 lost to date."

Etiology. The cause of this disease is a bacterium belonging to the septicemia hemorrhagica group. It is stated to have "the characters of the fowl cholera type." It killed rabbits when they were inoculated with 0.2 cc. of a bouillon culture.

Symptoms. The symptoms are indefinite. In the outbreak described the geese were often found dead. The description of the disease by Curtice is appended.

"Few symptoms of disease were seen, those noted pertaining mainly to the death struggles. Very few that died were noticed to be sick more than an hour or two before death, and, as the experimental investigation demonstrated, the disease could not have lasted, in the majority of the geese, more than thirty-six hours. An uncertain gait, a burrowing of the head in the dirt, twisting it around, or actions indicating spasms of the throat, were the earliest symptoms. Some geese were observed to die within five minutes or after the first seizure." There are few chronic cases and recoveries are not recorded. Some show no other symptom than being slower in action, and separating themselves somewhat from the flock. However, this sign is quite important when the wild nature and gregarious habit of the goose are taken into account.

Morbid anatomy. The tissue changes, as given by Curtice, are as follows:

"There was considerable mucus in the throat and mouth, and a very tenacious mucus in the nose. The veins of the head were usually congested, as though the animal had died of asphyxia. This, together with spasm of the throat, indicates a spasmodic closure of the glottis. The digestive tract was found to be full of food in nearly all stages of digestion. In some cases the catarrhal products of the intestines contained petechiæ. Sometimes these points were collected in more or less extensive patches. Perhaps more than half of the livers showed yellow spots of from a pin point to a pin head in size. These discolorations were found on section to extend into the substance of the liver, and were evidently dead tissue, or necroses. In one example the heart disclosed severe inflammation, both epicarditis and pericarditis being present. In one case the lungs were affected. In all, fifteen cases were examined, and from these this composite description of the post mortem appearances is drawn."

Hemorrhages on the serous membranes and punctate necroses in the liver seem to be quite characteristic lesions.

Post mortem notes.—These are a few taken from Curtice's publication:

"Goose No. 1. Died last night; quite fat. Right lung, ventral portion quite firm, whitish. Some flocculi of exudate in peritoneal cavity. Liver shows numerous point-like necrotic foci. Blood thick, blackish and tarry. Mucus glassy on dusky mucosæ of nose and throat.

"Goose No. 2. Died last night. Somewhat thinner than No. 1. Ecchymoses on fat in abdomen and gizzard and on heart muscle; necrosis in liver. Blood thick, tarry. Mucus in nasal passages.

"Gander No. 7. Died last night; now cold. No well marked hemorrhagic lesion in pleuroperitoneal cavity. Whitish points in liver. Hemorrhagic or extremely hyperemic condition of duodenum. Jejunum, or second coil of intestine, filled with a glairy mucous fluid in which are suspended shreds and patches of food (?). Few if any necroses in liver."

Diagnosis. Goose septicemia is to be diagnosed from the bacteriological examination. It is caused by a *pasteurella* which resembles that of fowl cholera. A diagnosis, therefore, is made positive by finding this organism in the tissue of the sick and dead geese. It is to be differentiated from other forms of infection from which geese may suffer.

M'Fadyean has described a disease under this title causing the death of many geese in which he found the blood swarming with bacteria suggesting *Bact. septicemiae hemorrhagicae* but morphologically different. He could not induce it to grow on any of several media in cultures under both aërobic and anaërobic conditions. It appears that this is a different disease from that described by Curtice.

Prevention. The procedure that can be suggested at present is isolation of the well from the sick, repeating the separations as often as new cases appear. The infected pens should be thoroughly disinfected before being reoccupied.

REFERENCES

1. CURTICE. Goose septicemia. *Bulletin No. 86, R. I. Agr. Exp. Station,* 1902.
2. M'FADYEAN. A remarkable outbreak of goose septicemia. *Jour. Compar. Path. and Therap.,* Vol. XV (1902), p. 162.

Fowls and poultry of all kinds are subject to infections that are interesting in their nature and often fatal in their results. There is a large literature on the subject. The general statement may be made that all poultry seem to be susceptible to the *Pasteurella.*

FOWL TYPHOID

Characterization. A specific disease of fowls caused by *Bacterium sanguinarium*. It is not known whether or not other species of domesticated birds are susceptible.

History. This disease was briefly described by Moore in 1895. At that time it had been studied in but a few fowls and these the last to die in their respective flocks. In the following year other fowls were examined very carefully from two outbreaks of the disease and it is upon the data obtained in this investigation together with those procured from many produced cases that the description of the disease is based. It was described as an infectious leukemia. Further investigation, however, has shown that the excess of white corpuscles was due to a leucocytosis brought about by the infecting organism and that the disease is not a leukemia.

It was found by Smith in 1894, on Block Island, R. I. In 1898, Dawson found it to be the cause of very serious losses among poultry near Baltimore, Md. In all of the outbreaks studied, the owners of the fowls first reported the disease as chicken cholera. In 1902, Curtice investigated an outbreak in Rhode Island.

Geographical distribution. It was first studied in fowls taken from an outbreak in Virginia. Since then, it has been identified in Maryland, the District of Columbia, and the State of Rhode Island. There is good evidence in the numerous reports of destructive fowl diseases to believe that it is quite widespread in the United States.

Etiology. Moore isolated and described a pathogenic bacterium which he designated *Bacterium sanguinarium*. With this organism the disease has been produced in healthy fowls both by feeding cultures and by intravenous injections. Its etiological relation to the disease is, therefore, quite clearly established. It is possible that certain accompanying conditions may be necessary in conjunction with the organism to cause the disease to spread rapidly in a flock. Experimentally it did not spread from diseased (inoculated or fed) to healthy fowls when kept in the same yard.

Symptoms. From the statement of the owners of the diseased fowls in the different outbreaks and from the appearance of those in which the disease was artificially produced, little can be positively stated concerning the early symptoms. There is a pronounced anemic condition of the mucosa of the head. An examination of the

blood shows a marked diminution in the number of red corpuscles
and an increase in the number of white ones.   In the disease pro-
duced artificially by feeding cultures of the specific organism there
is, in most cases, a marked drowsiness and general debility mani-
fested from one to four days before death occurs.   The period during
which the prostration continues varies from a few hours to two days.
The mucous membranes and skin about the head become pale.   There
is an elevation of from 1 to 4 degrees in temperature.   The fever is of
a continuous type, as shown in the appended temperature chart of
two fowls in which the disease was produced artificially.

Although the course of the disease in different fowls is usually
constant, there are many variations.   The time required for fatal
results is from three to fifteen days, but ordinarily death occurs in
about eight days after feeding the cultures.   The rise in temperature
can be 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 follow-
ing the intravenous injection of the virus were, as would be expected,
considerably modified from those fowls which contracted the disease
by the ingestion of cultures of the specific bacterium.
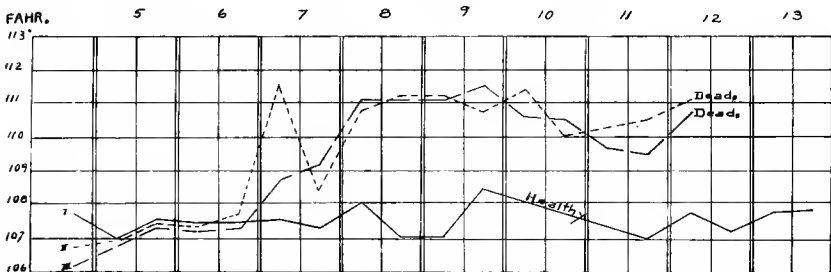
FIG. 7.   TEMPERATURE CHART OF TWO FATAL CASES ARTIFICIALLY PRODUCED IN FOWLS.

Morbid anatomy.   The only constant lesions found in the fowls
which contract the disease naturally, as well as in those fed upon the
virus, are in the liver and blood.   The liver is somewhat enlarged
and dark colored.   A close inspection shows the surface to be sprinkled
with minute grayish areas.   The microscopic examination shows the
blood spaces to be distended.   The hepatic cells often stain **very**

feebly. Not infrequently the cells are isolated and their outlines indistinct. Occasionally foci are observed in which the liver cells appear to be dead and the intervening spaces infiltrated with round cells. The changes in the hepatic tissues are presumably secondary to the engorgement of the organ with blood.

The rareness with which the intestinal tract is affected in both the natural and artificially produced cases is exceedingly interesting and important for the differential diagnosis. There is in most cases a hyperemia of the mucous membrane of the colon, but this condition is not uncommon in the healthy individual. The kidneys are generally but not uniformly pale. They are streaked with reddish lines, due to the injection of blood vessels. In section the tubular epithelium appears to be normal. The kidneys seem to be, from the number of bacteria in the cover-glass preparations, especially favorable for the localization of the specific organism. The spleen is rarely discolored or engorged with blood. The lymphatic glands were not appreciably enlarged in any individual examined. The lungs except in chronic cases are normal. The brain and spinal cord are unaffected.

The heart muscle is usually pale and sprinkled with grayish points due to cell infiltration and necrosis. These lesions are so common that it seems safe to consider them characteristic manifestations. Death usually occurs in systole, the auricles containing very thin, unclotted blood.

The most important alterations are found in the blood. These consist, in the progress of the disease, of the gradual disappearance of the red corpuscles and increase in the number of white ones, as determined by blood counts made daily or every other day, from the time of inoculation, or of feeding the virus, until the day of death.

The diminution in the number of red corpuscles and the increase in the number of white ones are illustrated in the blood count of two cases of artificially produced disease.

In carefully heated cover-glass preparations of healthy fowl's blood stained with methylene-blue and eosin, the nuclei are colored a deep blue, and the cellular protoplasm surrounding the nucleus is stained by the eosin. In similar preparations made from the blood of the affected fowls there are a greater or less number of cells which do not take the eosin stain. These were called spindle cells by Van Recklinghausen, blood plates by Bizzozero, and hematoblasts by Hayem. More recently Dekhuyzen has called them thrombocytes.

In these the portion of the cell body surrounding the nucleus remains unstained or becomes slightly tinted with blue. Occasionally they contain one or more vacuoles, and the margin is frequently broken. The apparent dissolving away of the red corpuscles has been frequently observed and corpuscles showing the intermediate stages are readily detected in carefully prepared specimens. These must be differentiated from the blood plates.

FIG. 8. BLOOD FROM A WELL ADVANCED CASE OF FOWL TYPHOID SHOWING RED CORPUSCLES, BLOOD PLATES AND INCREASE IN THE NUMBER OF LEUCOCYTES.

The cause of the destruction of the red corpuscles is not satisfactorily explained. In his report on fowl cholera, Salmon illustrates leucocytes surrounding the red corpuscles, but the marked diminution of the red cells was not determined. He speaks, however, of the pale color of the blood. In fresh preparations of the blood, portions of red cells may be seen within the leucocytes, those containing spindle-shaped granules. The determination of the extent of this mode of destruction of the red corpuscles necessitates further investigation.

TABLE SHOWING CHANGES IN THE NUMBER OF CORPUSCLES

Fowl inoculated in the wing vein February 6

Date	Temperature (F°.)	Number of red corpuscles per c. mm.	Number of white corpuscles per c. mm.	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 fed culture March 26				
Date	Temperature (F°)	Number of red corpuscles per c. mm.	Number of white corpuscles per c. mm.	Remarks
Mar. 26	106.2	3,535,000	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	245,000	Very weak; many red corpuscles attacked by leucocytes.
4	....	....	....	Found dead.

In fresh preparations of the blood of affected fowls examined in Toisson's fluid, red corpuscles which take the violet stain more or less intensely throughout are frequently observed.

In the blood of poultry two distinct classes of white corpuscles are conspicuous. The first, which predominates in numbers, contains nuclei with from one to four lobes, and the cytoplasm is sprinkled with a variable number of round, elongated, or spider-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 takes the blue stain feebly. Usually it is difficult to detect the nucleus, although it is occasionally distinct. Between these two types there are many varieties. The leucocytes containing the spindle-shaped bodies appear to be the phagocytes, as they were the only ones which were observed to engulf 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 comparatively large part in their destruction. Another hypothesis is also suggested, namely, that a toxin produced during the multiplication of the specific organism has this effect on the red corpuscles. In the fresh preparations we can observe the phagocytes attacking the red cells. 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 an open question.

In the blood from healthy fowls it is comparatively rare to see one of the white corpuscles engulfing 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 number of the leucocytes. It is an interesting and as yet unexplained fact that the increase in the corpuscles is apparently restricted to those containing the spindle-shaped bodies.

Diagnosis. Fowl typhoid is diagnosed by the symptoms, lesions and the finding of *Bact. sanguinarium* in the organs. It is to be differentiated from intestinal disturbances, especially diarrhea and fowl cholera.

A comparison of the important changes in the morbid anatomy in fowl cholera, as described by European writers, and in the disease under consideration, can be made from the appended columns, in which their more characteristic lesions are contrasted:

LESIONS IN FOWL CHOLERA	LESIONS IN FOWL TYPHOID
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 not common.
4. Intestines deeply reddened.	4. Intestines pale.
5. Intestinal contents liquid, muco-purulent, 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, hyperemic 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.

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. Salmon observed many changes which may have been similar to or identical with those herein recorded. Ward found an increase in the number of white corpuscles and in some cases a decrease in the number of red ones in cases of fowl cholera.

The difference between the specific organisms of these two diseases can readily be appreciated by a comparison of the more diagnostic properties of each; they are 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 microörganisms. These facts render positive differentiation dependent upon a careful bacteriological and pathological examination. In fowl cholera the course of the disease is more rapid than in fowl typhoid.

Prevention. Prompt isolation of the well from the sick fowls and thorough disinfection of the houses and yards.

In reference to preventing its introduction, Curtice makes the following observation:

"Inasmuch as one possible method of introducing the disease is through purchases, it will always be necessary for purchasers to inquire into the history of the flock from which additions are to be made, and especially to examine into the condition of the fowls. It is better in any case to keep new purchases by themselves for some weeks or until it is apparent that they are healthy."

REFERENCES

1. CURTICE. Fowl typhoid. *Bulletin 87. Agr. Exp. Station of the R. I. College of Agric. and Mech. Arts*, 1902.

2. MOORE. A study of a bacillus obtained from three outbreaks of fowl cholera. *Bulletin No. 8, U. S. Bureau of Animal Industry*, 1895.

3. MOORE. Infectious leukemia in fowls—A bacterial disease frequently mistaken for fowl cholera. *Annual Report of the Bureau of Animal Industry*, 1895–96.

SWINE ERYSIPELAS

Synonyms. Red fever of swine; *rouget du porc*; *Rotlauf.*

Characterization. This disease, peculiar to swine, is determined by a rise of temperature, cerebral disturbances and pronounced reddening of areas of the skin. It is a disease of adult life. It is stated that pigs are rarely attacked under three months or over three years of age. Lydtin and Schottelius found some differences in the degree of susceptibility of certain breeds of swine. The common country pig was least susceptible.

History. This disease has been known in Europe for many years. It was not distinguished from other infections until studied by Pasteur and Thuillier. Smith found a bacterium in rabbits inoculated with the organs of pigs that had died of an undetermined disease in Minnesota, which was either the bacterium of swine erysipelas or of mouse septicemia. The latter organism had been recorded on two previous occasions from pigs in this country.

Geographical distribution. Swine erysipelas occurs enzoötically and in epizoötics in most of the countries of Europe. It was formerly restricted in Bavaria to the districts along the Danube, and was entirely unknown in southern Bavaria (Kitt). It is stated that the disease tends to become enzoötic chiefly in valleys and low-lying plains which have slow-flowing streams and heavy, damp, clay soil; and that sandy and granite soils are comparatively free from it. It occurs chiefly during the months of July, August and September, although it appears sporadically during the winter months. It has not been described from the United States.

Etiology. Loeffler and Schütz pointed out in 1885 that swine erysipelas was caused by a very slender bacterium (*Bact. rhusiopathiae*) 1 to 2μ long and 0.3 to 0.4μ broad, straight or slightly curved, ends not rounded and in cultures often appearing in filaments. It is

very closely related to the bacterium of mouse septicemia described by Koch in 1878. There is much uncertainty concerning the relationship of the bacterium of mouse septicemia to that of this disease. Smith has suggested that possibly the bacterium which has been found in this country may gain virulence sufficient to produce epizoötics, if such is not already the case. It is exceedingly important that careful search be made for this organism in the outbreaks among swine where the nature of the disease is not clearly determined. House mice and pigeons are susceptible to the bacterium of swine erysipelas; guinea pigs and fowls are immune. The bacterium of swine erysipelas is to be differentiated from that of mouse septicemia.

The period of incubation is stated to be at least three days. It is apparently longer than that in many cases.

Symptoms. The disease usually begins suddenly and violently. The animal refuses food, makes efforts to vomit, has a rise of temperature, manifests severe nervous disturbance, is very weak, torpid and indifferent to its surroundings. When approached it tries to hide itself under its bedding. The hind quarters become weak and paralyzed. Muscular spasms and grinding of the teeth are sometimes observed. At first there is constipation, the conjunctiva is of a dark red or brownish-red color, and the eyelids are sometimes swollen. Usually a day or two after the first symptoms develop, or, perhaps, from the first, reddish spots appear on the thin parts of the skin, such as the region of the navel, lower surface of the chest, perineum, inner surface of the thighs, ears and throat. These spots, which at first are bright red and about the size of a man's hand, become, later on, dark red or purple, and soon unite into large, irregularly-shaped patches. As a rule, they are neither painful to the touch nor prominent, but sometimes they show a slight inflammatory swelling. The skin of the red spots, especially of the ears, may suffer from an eruption of vesicles and may even slough. Gangrene of the skin sometimes occurs. The reddening of the skin may be very slight in severe cases, or may appear only immediately before, or even after death. Death takes place usually on the third or fourth day. In the very severe form, the animal may die in twenty-four hours, otherwise the disease requires a week or longer to run its course.

Jensen considers that this disease, instead of being uniform in its clinical aspects, manifests itself in the following forms, which differ from each other by well-marked peculiarities. The forms recognized

as varieties of this disease but more generally considered as distinct maladies and known by different names are as follows:

True erysipelas.
Swine urticaria.
Erysipelas without redness of the skin.
Diffuse necrotic erysipelas of the skin.
Endocarditis of erysipelas.

He also maintains that there may sometimes be transitional forms between the respective varieties which he enumerates. Different forms of epizoötic erysipelas have also been described by Cornevin, Hess and others.

The duration of the disease varies from 1 to 10 days. In types of moderate severity it runs from 3 to 4 weeks.

The prognosis is unfavorable. There is from 20 to 80 per cent. mortality.

Morbid anatomy. In the ordinary form of epizoötic erysipelas there is a septicemic condition without any well marked morbid changes of separate organs. In less acute cases the septicemia may give way to hemorrhagic and diphtheritic gastro-enteritis, considerable swelling of the lymphatic system, hemorrhagic or parenchymatous nephritis, and hepatitis, acute swelling of the spleen and myositis. The hemorrhagic gastro-enteritis consists at first of excessive inflammation of the mucous membrane of the stomach in the region of the fundus. The mucosa shows a dark-red discoloration which is partly diffuse and partly in spots. The cells suffer from cloudy swelling and the mucous membrane is covered with a viscid layer of mucus. The intestinal mucous membrane is swollen, especially on the top of the folds and in the nieghborhood of Peyer's patches. It is infiltrated with blood and sometimes shows superficial scabs. Less frequently, circumscribed parts of the mucosa of the cecum and the anterior parts of the colon suffer from a diphtheritic affection.

The solitary follicles and Peyer's patches appear as prominently raised patches. Sometimes they are infiltrated with blood and surrounded by a reddish band. There is ulceration and cicatrization of the solitary and agminated follicles. The mesenteric glands become more swollen than the other glands of the body, of a dark red color, and show softening. The surface of fresh sections is dun-colored with interspersed dark-red areas. The paraglandular tissue is hyperemic and infiltrated with blood.

The kidneys are enlarged, the cortex of a grayish-red and the medullary portion of a very dark-red color. Frequently catarrhal nephritis occurs as a complication.

The acute swelling of the spleen arises in consequence of an acute hyperemia, with an increase of the cellular constituents of the pulp, in which case the organ is enlarged, but not softened as in anthrax. The pulp is of a purple color, moderately soft and free from hemorrhages.

There is cloudy swelling and enlargement of the liver. The surface of sections has a grayish-brown color, and the acini are widened. The muscles are gray in color, soft, flaccid, watery, glistening and sometimes they are sprinkled with hemorrhages. They give the general appearance of boiled flesh. The myocardium shows similar spotted changes, and punctiform hemorrhages beneath the endocardium. In the abdominal and thoracic cavities and pericardium, there may be found small quantities of an orange-colored, clear fluid, which may be mixed with a flaky coagulum.

Many English veterinarians regard the occurrence of more or less luxuriant vegetations on the valves of the heart to be so common that it is to be considered almost diagnostic. It would appear from the literature that this endocarditis is not nearly so common in continental Europe. The lungs remain unchanged, or at most exhibit a post-mortem edema. By microscopic examination, the specific bacteria are found everywhere in the body, especially in the spleen and kidneys, and to a less extent in the blood.

Diagnosis. Swine erysipelas is diagnosed from the symptoms, lesions and the isolation of its specific cause. The Ascoli thermo-precipitation test may be used. It is stated by Gsawizky to be strongly specific and Seibold reports favorably on its use. It is the only specific test for indicating this infection. Swine erysipelas is to be differentiated from:

Hog cholera and swine plague. The frequent reddening of the skin in these diseases together with the modified lesions so frequently observed may cause confusion. The bacteriological examination will enable the positive diagnosis to be made.

Anthrax, which is very rare in swine. Here, in addition to the bacteriological examination, specific tests for anthrax can be employed (see anthrax).

Erythemata due to various dietary causes. The significance of a deep reddening of the skin about the head, abdomen and thighs of

pigs is not fully determined. It is clear, however, that such a condition often occurs in the absence, so far as present knowledge goes, of a specific infection. It is frequently found in pigs suffering from digestive troubles, or poisoning from eating decomposed offal.

Prevention. Swine infected with this disease readily transmit it to others. The organism is reported to remain for a considerable time in the pharynx and nostrils and also to have been found in healthy swine. It is stated also to exist in the soil in a saprophytic form. These facts render prevention difficult. The general precaution of removing the sick from the well and not placing healthy swine in infected yards for some months after the recovery or removal of sick ones should be observed.

Pasteur's preventive inoculation was until recently the chief prophylactic means employed against epizoötic erysipelas. Metchnikoff found that the blood of immunized rabbits was antitoxic, and Lorenz maintains that the serum of swine that have recovered from swine erysipelas is also antitoxic, and will produce immunity in other animals. The treatment introduced by Lorenz is to inject the immunizing serum in the proportion of 1 cc. to every 10 kilograms of the body weight of the animal. Two days afterward 0.5 to 1.0 cc. of virulent culture is injected, and after twelve days the dose is doubled. The use of the immunizing serum is reported to be very successful.

Specific biologic treatment. The serum prepared by Lorenz is reported to give excellent results in acute cases.

REFERENCES

1. BANG. Ueber Rotlauf-Endocarditis bei Schweinen. *Deutsche Zeitschr. f. Thiermed.*, Bd. XVIII (1891), S. 27.

2. JENSEN. Die Aetiologie des Nesselfiebers und der diffusen Hautnekrose des Schweines. *Deutsche Zeitschr. f. Thiermed.*, 1892, S. 278.

3. LOEFFLER. Experimentelle Untersuchungen über Schweine-Rotlauf. *Arbeiten aus d. Kaiserlichen Gesundheitsamte*, Bd. 1 (1885), S. 46.

4. LORENZ. Die Schutzimpfung gegen Schweinerotlauf mit Anwendung eines aus Blutserum immunisirter Thiere hergestellten Impfstoffes. *Deutsche Zeitschr. f. Thiermed.*, Bd. XX (1894), S. 1.

5. LORENZ. Die Veterinärpolizeiliche Behandlung des Schweinerothlaufes und die Schutzimpfung. *Berliner thierarz. Wochen.*, 1897, S. 574.

6. LORENZ. Schutzimpfungen gegen den Rotlauf der Schweine. *Ibid.*, 1897, S. 109.

7. MOORE. Mouse septicemia bacilli in a pig's spleen with some observations on their pathogenic properties. *Jour. of Comp. Med. and Vet. Archives*, Vol. XIII (1892), p. 333.

8. PASTEUR ET THUILLIER. La vaccination du rouget des porcs á Paide du virus mortel attenué de cette maladie. *Comp. rendus Acod. des Sciences*, Vol. XCVII (1883), p. 1163.

9. SALMON. An Examination of Pasteur's Vaccine for Rouget. *Annual Report U. S. Bureau of Animal Industry*, 1885, p. 187.

10. SCHUTZ. Ueber den Rotlauf der Schweine und die Impfung mit demselben. *Arbeit a. d. Kaiserlichen Gesundheistamte*, Bd. I (1885), S. 56.

11. SEIBOLD. Beitrag zur Feststellung des Rotlaufs der Schweine mit Hilfe der Thermo-präcipitinreaktions nach Ascoli. *Zeit. f. infek. d. Haust.*, Bd. XIII, S. 91.

12. SMITH. Swine erysipelas or mouse septicemia bacilli from an outbreak of swine disease. *Annual Rept. U. S. Bureau of Animal Industry*, 1895–96, p, 166.

ANTHRAX

Synonyms. Splenic fever; splenic apoplexy; wool sorters' disease; malignant pustule; anthracemia; mycosis intestinalis; *charbon; Milzbrand.*

Characterization. Anthrax is an infectious disease occurring sporadically and in epizoötics in herbivora and omnivora and communicable to nearly all warm-blooded animals, and to man. It is characterized by a high temperature, the presence in the diseased tissues or liquids of *Bacterium anthracis*, by an enlarged spleen, blood extravasations and by local gangrene. It usually occurs in the acute form.

Nearly all species of animals suffer from anthrax. The herbivora and rodents are most susceptible. Horses and mules often suffer from it. M'Fadyean has reported outbreaks aggregating 54 cases, of which 49 were cattle, 4 horses and 1 pig. He states also that for a period of 5 years there had been reported 192 cases in horses and 3,390 in cattle. It is interesting to note that the Algerian race of sheep are immune. A satisfactory explanation for this striking exception has not been recorded. It has been stated that a single bacterium introduced into the subcutaneous connective tissue of a guinea pig or mouse is sufficient to kill it. Cats, tame and wild rabbits and hares are the next most susceptible species. It is stated that dogs, pigs and foxes are very slightly susceptible. Rats, fowls and pigeons are reported to be immune. Fish and amphibia are rarely attacked.

History. Anthrax is among the oldest of the known infectious diseases of animals. Descriptions of epidemics and epizoötics of this disease are given by Homer, Plutarch, Livy and other writers before the Christian Era. The Arab physicians designated it as "Persian Fire." Extensive outbreaks are mentioned in the literature of the fifteenth, sixteenth, seventeenth, eighteenth and nineteenth centuries. Chabert pointed out in 1780 that the various kinds or forms of the

disease, which had previously been described as independent affections, were all one disease. As late as 1805, Kausch gave a good description of anthrax but denied its contagiousness. Delafond and Gerlach thoroughly investigated ovine anthrax in 1854 and its contagiousness was experimentally shown by Gerlach. In 1850, Heusinger published a very comprehensive treatise on anthrax which deals at length with its history and geographical distribution.

Much new information concerning the nature of anthrax was acquired during the fifth decade of the last century. In 1855, Pollander announced the discovery, which he first made in 1849, of minute unbranched rod-shaped bodies in the blood of cattle dead of anthrax. Davaine observed similar bodies in 1850. Then followed a long series of observations and somewhat controversial discussions on the bacterial origin of the disease, culminating by Robert Koch's careful description of the morphology of its specific organism including the spore formation in 1876 (1877 Pasteur). Cohn, however, seems to have been the first to have called the organism a *Bacillus* and to have suspected the existence of spores. Toussaint, in 1880, and Pasteur in 1881, published results of investigations directed toward protective inoculation. Since that time, the literature on the cause, morbid anatomy and prevention of anthrax has become very extensive.

Geographical distribution. Anthrax is a widely disseminated disease. The continent of Europe has perhaps suffered most from its ravages. It occurs, also, in Northern, Eastern and Central Africa, where in recent years it has become a great plague. In Siberia, it has caused fearful destruction, and in that country it is still known as the "Siberian Plague." It has frequently appeared in England. Russia, India and Australia are also infected. South America is also reported to suffer much from its ravages. It has occurred in the United States in many localities. There are very few, if any, countries where this disease has not been found. A knowledge of its specific cause, with the methods of properly disposing of dead animals, isolation and disinfection, as well as the preventive inoculations now in vogue, have made it possible to prevent wide-spread epizoötics. In America it is looked upon as a comparatively rare disease, excepting in certain very restricted infected districts.

Etiology. Anthrax is caused by a microörganism, *Bacterium anthracis*. This organism can usually be found in the diseased organs

of affected animals. Its spores are very resistant to the normal destructive agencies in nature. Consequently when anthrax is once introduced into a locality it tends to remain there for many years, possibly causing from time to time a few cases or more serious epizoötics, or epidemics. The spores are frequently carried in the wool, hair, hides, hoofs and horns taken from animals sick or dead of anthrax. Through these agencies anthrax has been introduced into distant localities.

Bacterium anthracis is a rod-shaped organism varying in length from 1 to 4 μ, but having a quite uniform breadth of about one micron. In a suitable medium it grows out in long flexible filaments which combine to form thread-like bundles. When examined, the ends of the rod seem to be square cut. In preparations from animal tissues there appear sometimes to be slight concavities in the ends of the segments when two of them are united. In the preparations, capsules are easily recognized. It is believed by Kodama and others that the capsule is a protection to the bacterium against phagocytes but that it does not protect against the bacteriological action of the blood serum. In cultures spores are formed. These are oval, highly refractive bodies held within the cellular envelopes of the filaments, but later they are set free by the dissolution of this membrane. They stain readily with the aniline dyes and also by Gram's method.

The bacterium of anthrax itself is not an especially hardy organism. On the contrary it is easily destroyed by weak disinfectants and it has a low thermal death point. Its spores, however, are among the most hardy of bacterial life to resist chemical and thermal agents. They resist drying for months or years and often boiling for a half-hour or longer does not destroy them. On that account it is very difficult to eliminate the virus from infected pasture lands, especially if they are wet or marshy. Hutyra and Marek state that the spores may pass through the digestive tract without germinating.

As the spores may remain on the soil in a dormant condition for many years, it sometimes happens that the disease does not appear until long after the introduction of the virus. Anthrax has been known to break out among cattle grazing on a field in which the carcasses or hides from affected animals were buried many years before. Through some means the spores seem to be able to get to the surface and contaminate the grass. The virus may be introduced with blood or bone fertilizers, hides, hair or wool from infected countries. When the extent of this traffic is realized, it is easy to under-

stand how anthrax has been brought to this country and why it occasionally appears here and there over a large part of the continent. Many outbreaks, as well as isolated cases, illustrating this common method of dissemination are on record.

The period of incubation is very short. In inoculated animals it ranges from 1 to 5 days.

Channels of infection. Three common modes of infection are recognized for anthrax, namely: through the digestive tract, by the skin and by the lungs. In cattle the infection seems to be largely through the alimentary canal; in horses and sheep by the skin or digestive tract; in men through wounds of the skin and the respiratory tract. Although these are the usual methods there are many exceptions with each species.

Infection through the alimentary canal. This is the more common mode of infection in cattle. The resulting disease has been designated by various names, among which are "intestinal anthrax," "fodder anthrax," "spontaneous anthrax," "internal anthrax," "anthrax fever," and anthrax without external manifestations. In these cases the infecting organisms, either the spores or the vegetating bacteria themselves, are taken into the body with food or drinking water. M'Fadyean has shown that infected food-stuffs are often responsible for the infection. It is stated that the infection takes place in most cases in the small intestine, the mucosa of which, it is stated, need not necessarily be injured. It is highly probable that the gastric juice destroys most of the bacteria while the free spores are not injuriously affected by it. In the infected districts, the spores exist at or upon the surface of the soil and possibly on the blades of grass, from which they are easily taken up by grazing animals. In lands thus infected, the specific organism has been introduced at some previous time either by the burying of anthrax animals in these fields, by the use of infected tannery or slaughter house refuse as fertilizers, by flooding from infected streams, or by the bringing of the organism in the droppings of birds or other small animals which have fed upon anthrax carcasses. It is reported that the spores will find their way to the surface even when the dead animals have been buried at a considerable depth. There has been some controversy in the writings of Pasteur, Koch and Bollinger concerning the method by which the spores reach the surface. Pasteur supposed that they were brought by earth worms from the buried carcasses. Koch believed this impossible because of the

low temperature of the ground at the depth at which the animals are buried. Bollinger has shown experimentally the possibility of Pasteur's views. Karliniski and others have found that the spores of anthrax may be disseminated by slugs, insects and larvæ which are found on untanned infected skins.

Infection through the skin. In animals, this mode of infection occurs less frequently than in man. Anthrax produced in this way is usually characterized by local manifestations known as "carbuncle disease," or "malignant pustule." In this mode of infection the bacteria penetrate through wounds in the skin and exposed mucous membranes into the living tissues by means of infected utensils, the use of infected instruments, and insects, especially the house fly (*Musca domestica*). Dalrymple has called attention to the spread of this disease among animals in the lower Mississippi Valley by means of the horse fly (*Tabanidae*). In man many cases of the disease occur from injuries or cuts made at the post-mortem of anthrax animals or by the infection of skin wounds while handling infected hides or wool. Maligant pustule is reported to be quite common among the employes of certain tanneries and upholstering establishments where hides and hair imported from infected districts or countries are used.

Infection through the respiratory tract. Faser, Buchner, Lemke, and other writers have shown experimentally that the disease can be produced by the inhalation of spores. In man this form of infection is quite common among the wool sorters. In Great Britain, where much foreign wool is handled, it has been reported as causing as many as 500 deaths annually. It is known as "wool-sorters' disease."

Symptoms. In anthrax, the symptoms vary not only in different species of animals but also in different individuals according to the location of the disease. Again there is often considerable variation when the lesions are apparently the same. The most characteristic features of the disease are the suddenness of the attack, the grave general disturbances, high elevation of temperature, a tendency to ecchymoses of the mucous membranes and local manifestations, such as carbuncles and edema of the skin, digestive disturbances, brain complications and difficult respiration.

Anthrax has been classified according to its course as peracute, acute and subacute. It has also been divided according to the site of its manifestations as anthrax with visible localization and anthrax without visible localization.

Anthrax without visible localization. This form is generally due to ordinary infection presumably by spores. It includes the peracute, acute, and subacute.

The peracute or apoplectic anthrax gives rise to symptoms of cerebral apoplexy. The animal becomes suddenly ill, staggers about for a brief period and falls. There is often a bloody discharge from the mouth, nostrils and anus. Death usually ensues in from a few minutes to an hour. Usually there are convulsions. Sheep and cattle suffer most frequently with this form. They are often found dead. This is especially true in the beginning of an epizoötic.

In the acute form, the disease runs a somewhat slower course, lasting usually not to exceed twenty-four hours. The temperature rises rapidly to from 105 to 108° F., dropping suddenly just before death. With this there are signs of congestion either of the brain or of the lungs. If the brain is affected the animal becomes restless, excited, stamps the ground, rears in the air, bellows, runs to and fro and finally goes into convulsions followed by stupor and death. If the lungs are congested there is difficulty in breathing, more or less wheezing, panting, groaning, palpitation of the heart, small and frequent pulse, cyanosis of the mucosa of the head, bloody discharges, hematuria, staggering and finally convulsions and death from suffocation. Occasionally there is a partial remission of the symptoms, followed by relapse. It has been observed that occasionally there are premonitory symptoms preceding the acute attack consisting of slight digestive disturbances and diminished vivacity. Burnett found the anthrax bacteria in large numbers in the blood during this stage. He likewise found them to be present in the blood of the more chronic cases during the febrile period.

The subacute form is known as anthrax fever or intermittent anthrax. The symptoms are the same as in the other forms, except that they are more sharply defined and the course is longer. The disease lasts from one to seven or eight days. The high temperature, the congestion of the lungs or brain complicated with intestinal disturbances, especially colic, are usually well marked. In epizoötics where the peracute or acute form ushers in the disease, the later cases usually are of the subacute variety.

Anthrax with visible localization. These forms usually result from infection of the skin and mucous membranes. This form is common in horses and sometimes it occurs in cattle. It is reported to occur in other species. The lesions are circumscribed, cutaneous swellings

which are at first hard, hot and painful. Later they become cold and painless, with a tendency to become gangrenous. The edematous tissue becomes doughy, cold to the touch and painless. Frequently fluctuating swellings of the skin occur. The duration of this form of the disease varies from four to fifteen days. Ordinarily it is not so fatal as internal anthrax.

When the infection is on the mucous membrane of the mouth or pharynx the animal suffers from fever, dyspnea, difficulty in swallowing and cyanosis, together with the immediate local effects. Death occurs much sooner than when the disease is located in the skin. It is stated that dogs and swine suffer from this form more than from the acute types.

In horses, anthrax usually runs an acute or subacute course. The first symptom is rise of temperature with a rapid, feeble pulse. There may be chills and muscular spasms. The mucosa of the head becomes cyanotic and lacrymation is often present. The animal has a dull, stupid look, appears to be stunned and walks with a staggering gait. In some cases there are symptoms of cerebral congestion, such as restlessness or convulsions. Colic is a very characteristic symptom in the horse, otherwise the symptoms are the same as in cattle. Infection of the skin usually occurs on the hypogastrium, lower part of the breast, inner surface of the fore and hind quarters. Swelling of the hind quarters often causes lameness.

In sheep and goats the disease is usually of the acute or apoplectic form. The animals appear as if suddenly stricken with apoplexy. If death does not occur within a very short time, symptoms already described for this form of the disease may be recognized. Subacute anthrax is said to be very rare in sheep.

In swine, anthrax is ordinarily characterized by local lesions on the mucous membrane of the larynx and pharynx. The animals have a rise of temperature and the intermaxillary space is generally swollen. The swelling may spread along the trachea, giving rise to difficulty in swallowing, hoarseness, cyanosis of the mucosa of the mouth, dyspnea and rapid breathing. The animal shows signs of paralysis. Death occurs from suffocation. Frequently the tongue becomes the seat of the disease.

In dogs and cats, the disease usually runs a very rapid course. The fact that they are usually infected by eating the meat of animals dead of anthrax causes them to suffer largely from the intestinal form. It

has been stated that probably much of the so-called anthrax in dogs may be ptomaine poisoning.

It is reported that in birds anthrax usually runs a very rapid and usually fatal course.   Toward the end they stagger, tremble, or go into convulsions and die with bloody discharges from the mouth, nostrils and anus.   From the first the birds are depressed, weak, and their feathers ruffled.   There is evidence of dyspnea.   Carbuncles are said to appear on the comb, wattles, conjunctiva, tongue and extremities.

It has been stated that the milk from cows suffering with anthrax contains *Bact. anthracis*.   Moore found in the examinations made in one epizoötic that they were present in considerable numbers in the milk just before or immediately after death, but they were not found in the milk of animals in the earlier stages of the disease.

The duration varies from a few hours to a week or even longer.

The prognosis is unfavorable.   In some herds the mortality is 100 per cent. while in others a number of animals may recover.*   The average mortality is placed at about 70 per cent. in animals.   In the human species many persons recover from its local form (malignant pustule).

Morbid anatomy.   The nature and extent of the tissue changes depend upon the course of the disease.   When experimentally produced it is ordinarily a septicemia.   This form often occurs in domesticated animals when they contract the disease naturally.   The more common anatomical changes, except in the most acute cases and in the strictly localized lesions or carbuncles, are: ·

Hemorrhages varying in amount from petechiæ to blood extravasations, with more or less serous, gelatinous and hemorrhagic infiltration of the submucous, subserous and subcutaneous tissue.

The capillaries are distended and frequently there are hemorrhages due to changes in the walls of the capillaries.   The anthrax bacteria are often present in large numbers in the smaller blood-vessels.   It is believed by many that the capsules absorb much of the body liquid,

---

*M'Fadyean has reported this disease in 39 consecutive outbreaks in which a total of 54 animals died.   In New York the disease existed in 1904 in 15 herds in one locality. There were more than 30 deaths.   In one herd of 21 animals, 20 had the disease, 16 died and 4 recovered.   In another dairy 4 out of 7 died, but in the others one or two animals in each were affected.   In 1903 anthrax occurred on 84 different farms in the same county.   There were 170 fatal cases of which 33 were in horses, 123 in cattle, 11 in sheep, and 3 in hogs (Burnett).   These facts are interesting in showing that the disease does not always cause heavy losses in the infected herds.

causing them to swell and preventing the fluid from reentering the circulation. This gives rise to the edematous swellings. The subcutis may be sprinkled with ecchymoses. Frequently there are gelatinous effusions of a rather firm consistency and of varying size. The color also differs, ranging between a deep yellow and a yellowish brown. Often these edematous areas are sprinkled with hemorrhagic foci. A simple serous edema may occur.

The lymphatic glands may be hemorrhagic, edematous or both. An edema of the connective tissues of the neck or about the trachea is often very marked.

The muscles vary in color but usually they are darker than normal, and, like the skin, they often become sprinkled with ecchymoses. The heart muscle suffers from parenchymatous changes.

In the larger cavities of the body, a sanguinolent fluid is found in moderate quantities. Blood extravasations of different sizes are seen under the serous membranes, particularly on the mesentery and mediastinum. The subserous connective tissue, especially on the mesentery, anterior mediastinum and in the neighborhood of the kidneys, is often infiltrated with a gelatinous substance. On this account the neighboring lymph glands are considerably swollen, filled with serum and sprinkled with hemorrhages. The internal organs contain a large quantity of blood. All the larger veins and the heart are filled, while the surrounding tissues show sanious imbibition.

The spleen is usually considerably enlarged (two to five times its normal size), either uniformly or by prominent tumor-like protuberances. The pulp is soft, more or less fluid, and stained a dark-red color. The capsule is always very tense. It is frequently sprinkled with ecchymoses. Occasionally this organ is slightly affected.

The liver and kidneys are highly congested and somewhat enlarged. The parenchyma contains areas of blood infiltration and the cells themselves manifest various kinds of degeneration. The portal lymph glands often appear to be enlarged, and the retroperitoneal tissue may be infiltrated with a serous, gelatinous fluid. The subperitoneal tissue of the intestines and of the abdominal walls may be similarly affected.

The nature of the lesions of the intestinal canal varies according as the disease is intestinal anthrax, or anthrax caused by inoculation. In case of inoculation anthrax, the intestine is frequently normal. In other cases there may be submucous and subserous hemorrhages, or swelling of the mesentric glands. The principal changes in intestinal

anthrax are always found in the small intestine, chiefly in the duodenum. In the milder cases of intestinal anthrax the mucous membrane is affected by circumscribed or diffuse swellings. The bacteria are often found in very large numbers on the surface of the mucous membrane. Necroses and ulcers appear in those parts where the bacteria are most thickly congregated. In very severe cases, the abomasum or the true stomach may be affected with a gelatinous and sanious infiltration of the mucous membrane. The mucosa of the abomasum, and especially of the duodenum, is, in consequence of excessive hyperemia, dark-red or almost black, and is covered with erosions and ulcers or necroses, which may extend down to the submucosa. The contents of the intestine are bloody, and the submucosa is infiltrated with a serous, gelatinous, or hemorrhagic exudate, so that the mucous membrane often projects in the form of large tumors into the lumen of the intestine. On the site of Peyer's patches and the solitary follicles we may find flat or prominent nodules, the surface of which are covered with diphtheritic crusts.

The lungs are greatly congested, edematous and show areas of ecchymoses. The entire respiratory mucous membrane is considerably reddened and ecchymotic. The mucous membrane of the pharynx and opening of the larynx is often so edematous that stenosis of the larynx takes place. The contents of the trachea and the bronchi consist mostly of bloody froth or mucus.

The brain is often studded with ecchymoses. The surface of its membranes often exhibits hemorrhages with an accumulation of sanious serum in the ventricles. Extravasations of blood sometimes occur in the anterior chamber of the eye and under the retina. All the other organs may contain hemorrhages, and the urine frequently contains blood.

The blood is usually dark. It has a tarry or varnish-like lustre, and shows little tendency to coagulate. It does not assume its normal red color when exposed to the air. Burnett studied the blood of a few cases of anthrax in 1904. The appended tables are taken from the results of his examinations.

RESULT OF THE EXAMINATION OF THE BLOOD OF FIVE CASES OF ANTHRAX IN CATTLE

Cow	First symptom observed	Date of examination	Temperature	Hemoglobin per cent.	Red Corpuscles per c. mm.	Leucocytes per c. mm.	Remarks
No. 8	July 8		107.5°				Anth. bact. in blood
" 8		July 9	106.6	60	4,072,000	20,000	Died July 9
" 4*	July 10		106.2				
" 4		July 11		56	5,471,000	4,814	
" 4		" 13	103.0	38	3,400,000	3,444	
" 4		" 19		56	2,086,000	9,876	Recovered
" 6†	July 7						Anth. bact. in blood
" 6		July 9	104.0	50	3,876,600	8,222	
" 6		" 11		60	3,954,000	5,210	
" 6		" 13	101.8				
" 6		" 14	101.2	47	3,484,000	5,666	Apparently recovered.
" 6		" 19		54	1,980,000	8,777	Died of anthrax Nov. 4
" 6		" 24		63	3,132,000	11,888	
" 3	June 29						
" 3		July 14	101.2	57	4,168,000	5,222	Recovered
" 1	July 15						
" 1		July 16	103.8	53	2,324,000	8,111	
" 1		" 17	101.0	58	2,632,000	5,333	
" 1		" 18	102.0			8,163	
" 1		" 19			5,940,000	11,000	
" 1		" 24		61		10,767	Recovered

*Temperature July 8, 102.1°   Temperature July 10, 100.0°
†Temperature July 8, 107.4°   Temperature July 9, 103.0°

THE DIFFERENTIAL COUNT OF THE LEUCOCYTES IN FIVE CASES OF ANTHRAX IN CATTLE

Cow	Date	Leucocytes per c. mm.	Lymphocytes		Large Mono nuclear		Polynuclear		Eosinophiles		Mast Cells	
			No.	%	No.	%	No.	%	No.	%	No.	%
Soper No. 8	July 9	20000	7080	35.4	2200	11.0	7120	35.6	3520	17.6	80	0.4
" 4	" 10			27.4		5.2		64.8		2.8		0.2
" 4	" 11	4814	1670	34.7	341	7.1	2200	45.7	548	11.4	48	1.0
" 4	" 13	3444	1432	41.6	261	7.6	1667	48.4	75	2.2	7	0.2
" 4	" 19	9876	4257	43.1	273	2.7	4696	47.5	636	6.4	11	0.2
" 6	" 9	8222	3930	47.8	296	3.6	3436	41.8	518	6.3	41	0.5
" 6	" 11	5210	2287	43.9	338	6.5	2115	40.6	432	8.3	36	0.7
" 6	" 13			40.1		4.0		47.2		7.8		0.7
" 6	" 14	5666	2833	50.0	119	2.1	2221	39.2	430	7.6	62	1.1
" 6	" 19	8777	3747	42.7	263	3.0	2800	31.9	1930	22.0	35	0.4
" 6	" 24	11888	6033	50.7	698	5.8	3120	26.2	1946	16.3	89	0.7
" 3	" 14	5222	3352	64.2	261	5.0	1451	27.8	146	2.8	10	0.2
Bradish No. 1	" 16	8111	3528	43.5	154	1.9	3033	37.4	1330	16.4	65	0.8
" 1	" 17	5333	2832	53.1	256	4.8	1568	29.4	640	12.0	36	0.7
" 1	" 18	8163	6375	78.1	138	1.7	914	11.2	734	9.0		
" 1	" 19	11000	6611	60.1	253	2.3	2585	23.5	1496	13.6	55	0.5
" 1	" 24	10767	5911	54.9	484	4.5	2186	20.3	2099	19.5	86	0.8

Burnett found that the number of red corpuscles and the percentage of hemoglobin are reduced. In the chronic cases they tend to return to the normal condition. There was an increase in the number of lymphocytes and a decrease in the number of polynuclear leucocytes. In some cases there was a marked increase in the number of eosinophiles. No change from the normal was noted in the large mononuclear leucocytes or in the mast cells.

The bodies of animals which have died from anthrax are often well nourished. Rigor mortis is absent and they decompose quickly. Very frequently blood flows from the natural openings of the body, and the rectum is sometimes prolapsed.

All the foregoing lesions may be absent in very acute apoplectic cases. The specific organism is, however, always present in the cadaver. It is important to note that occasionally the usual changes indicated by the symptoms and the duration of the disease are not found on post-mortem examination. In one epizoötic, the writer saw an animal dead from subacute anthrax in which the blood and tissues were teeming with anthrax bacteria, yet the organs examined microscopically appeared to be normal. Other animals in the same outbreak exhibited the more usual anatomical changes.

Diagnosis.* There are a number of methods for diagnosing anthrax. The symptoms and lesions are of value but often they are not sufficiently characteristic to enable one to make a positive determination. This must rest on the bacteriological examination and the specific reactions.

Bacteriological. It was believed for many years that the bacteriological examination for the diagnosis of anthrax was very simple and sure. That opinion is entertained by many veterinarians still. The facts are that many cases can be readily identified by this method if the tissues are fresh, but, on the other hand, there are those where it is impossible. The difficulties lie in several directions. The most important is the rapidity with which anthrax bacteria die in tissues where sporulation does not occur. For this reason it is not always possible to find the organisms in specimens sent to a laboratory. The

---

*Veterinarians should recognize that anthrax is often very difficult to diagnose in the laboratory. The lesions are frequently localized and in such cases the specific organism is not always present in the general circulation or in other organs. In such cases the man who makes the post mortem examination must locate the lesions and select parts of the affected tissues for bacteriological examination. While the typical cases are readily detected, there are those where the limitations of the laboratory methods and the neglect of the clinician in making the post mortem allow the disease to go undetected.

careful work of Fischœder has clearly pointed out the difficulties in this direction. Secondly, errors may occur because of the presence of what are called pseudo-anthrax bacteria from which a differentiation is not always easy. Fitch pointed out that these organisms could not be differentiated from *Bact. anthracis* from agar or gelatin cultures. Pokschischewsky has studied their biology and shown their very close resemblance, in certain particulars, to *Bact. anthracis*. He divides these organisms according to their growth on agar, gelatin and potato, into two types, namely, pseudo-anthrax and anthraxoid. Microscopically the presence of an organism resembling that of anthrax, often found in tissues some hours old, may be mistaken for that of anthrax. The diagnosis bacteriologically requires the isolation of the specific bacterium and its identification by cultural methods or animal inoculation.

Differential stain. M'Fadyean has described a peculiar staining reaction, first pointed out by Heins, which he considers of value for the microscopic diagnosis of this disease. The reaction is in evidence when films of blood, exudates, or tissue juice containing the bacteria are stained with a simple aqueous solution of methylene blue. The method as applied to blood is as follows:

Place a drop of the blood on a clean slide. The size of the drop should be about 2 mm. in diameter. It is spread quickly with a platinum needle until it covers an area about 12 mm. in diameter. Protect from dust and allow the slide to remain until all evidence of moisture has disappeared. When dry, heat the preparation by lowering it film upwards into the flame of a Bunsen burner or an alcohol lamp for a second. Repeat this three times or until the glass is too hot to be borne by the skin in the palm of the hand. Allow the slide to cool and then cover the film with 1 per cent. aqueous solution of methylene blue. After a few seconds pour off the free stain and wash the slide thoroughly in tap water. Dry the slide by pressing it gently between two layers of bibulous paper, and then more thoroughly by holding it in the current of hot air above the Bunsen flame. Finally, mount in Canada balsam.

The microscopic examinations (x 800 to 1000) will show an occasional leucocyte and the anthrax bacteria. There will appear no other visible formed elements. The nuclei of the corpuscles generally exhibit a greenish-blue tint, the anthrax rods are stained blue. The intensity of the stain depends upon the length of time after death before the films were made. Usually the segment character of all but the shortest rods will be apparent. If they are deeply stained this is not very distinct. *The peculiarity in the reaction lies in the color of the amorphous material which is present between and around the bacteria.* This material presents itself under the form of coarse or fine granules of a violet or reddish-purple color, which is in sharp contrast to the tint of the bacteria or cell nuclei, especially with brilliant lamp or gas light. These violet granules differ a good deal in form and size; sometimes they are very minute, and at others coarsely granular. When the bacteria are arranged in clumps the violet material is often in

greatest amount about them. Free-lying anthrax rods will be surrounded by a thick envelope of the same substance. M'Fadyean states that he has never found this reaction in animals dead from other diseases. The peculiar coloring, he states, in some cases may be observed without the aid of the microscope. Our experience with it has not been so satisfactory.

Thermoprecipitation. This method was formulated for practical work by Schutz and Pfeiler. It was especially advocated by Ascoli for the diagnosis of anthrax. It has been tested by a number of laboratory workers and as a rule highly recommended. The method is based on the fact that anthrax bacteria, or their decomposition products, present in the bodies of animals dead of anthrax, contain a precipitinogen which, when brought into contact with anthrax immune serum, produces a precipitate at the point of contact. Pickens describes the method in detail and concludes that it is a reliable means for the diagnosis of anthrax. It was found, however, by Pokschischewsky that this reaction took place in cases of infection with certain of the pseudo-anthrax organisms.

Method.—The principle of this test is based on the fact pointed out by Kraus in 1897, that an immune serum when brought into contact with its corresponding antigen will produce a precipitation at the point of union of the two fluids. The first fluid, the immune serum, is difficult to produce. The technic was first worked out by Ascoli.

The selection of the animal for the production of this serum is an important problem to decide. According to Ascoli, Schutz and Pfeiler and others, the ass is the most desirable animal. However, good sera have been produced from the horse, mule, cow and rabbit. Varying degrees of success have been obtained with the dog, sheep, goat and guinea pig.

The selection of the animal is important as only certain individuals produce a proper serum. It is stated by Schutz and Pfeiler that out of one lot of thirty animals only three were found that produced a satisfactory serum. According to Ascoli and others, the serum of certain normal animals, especially the horse, is apt to produce a precipitation when brought into contact with salt solution, carbol salt solution, distilled water or bouillon. The serum of the animal to be immunized should be tested with reference to this quality.

The test requires an immune serum and the antigen. The animals are immunized by repeated injections of attenuated, slightly virulent and virulent cultures of *Bact. anthracis* beginning with the attenuated culture. Several injections are necessary. They may be made subcutaneously, into the abdominal cavity or intravenously. The injections are made about a week apart. It requires several weeks to obtain a satisfactory serum. The blood should not be drawn from the immunized animal for at least ten days after the last injection.

To obtain the serum the blood is drawn in the usual way and allowed to clot and the clear serum drawn off. The serum must be perfectly clear and not colored with hemoglobin.

There are three different methods by which an antigen may be prepared from the suspected material, namely: The boiled extract, the shake extract and the slow process. He also describes a method for the preparation of an antigen made from a culture of the anthrax organism. This antigen should be used as a control upon the material to be tested by this reaction.

The Boiled Extract. This extract consists in taking a small piece of the material to be examined and placing it in a test tube, and adding to it four or five volumes of normal salt solution. The test tube is then placed in boiling water from five to fifteen minutes. This yields a cloudy brownish fluid which may be cleared by filtering through filter paper. Two or more filtrations may be necessary.

According to Schutz and Pfeiler, this antigen may be improved by the use of carbol salt solution instead of normal salt solution. They state that the carbolic acid will also preserve the extract so that it may be kept for later use.

The Shake Extract. This extract consists in placing a piece of tissue about the size of a hazelnut in a test tube to which is added about 10 c.c. of normal salt solution. It is then thoroughly shaken. This yields a dirty, reddish fluid, which is centrifuged for clearing purposes. It is then filtered through filter paper or asbestos. This process may have to be repeated several times to obtain a clear antigen.

The Slow Process. This consists in taking a piece of suspected material about the size of a hazelnut and triturating in a mortar with about ten grams of dry, white sand. To this is added enough chloroform to completely cover it. The chloroform is allowed to remain for several hours, after which it is poured off. The residue should then be thoroughly stirred with a glass rod and enough normal salt solution added to cover it. This should be stirred again and the fluid filtered into a test-tube through ordinary filter paper. The filtrate should be colorless or slightly yellow. If it is not clear, it should be refiltered until it is.

The chloroform does not have any effect upon the mixture except to precipitate the hemoglobin. The precipitating substance is not soluble in chloroform, and hence the final extract is not weakened by this process. In cases where the organs contain only a few bacteria, a longer extraction by the normal salt solution may be necessary.

The Culture Extract. For this extract, a 24-hour agar culture of the anthrax organism is used. Five to 10 c.c. of normal salt solution is poured over the surface of the culture and allowed to act for two hours at room temperature. At the expiration of that time, the fluid is removed and filtered through ordinary filter paper. The filtrate must be clear, or slightly yellowish in color.

Schutz and Pfeiler again prefer the carbol salt solution to the normal salt, as they think it produces a stronger antigen.

Of the three different methods for the preparation of the antigen, Ascoli favors the boiled extract. On the other hand Schutz, Pfeiler and Pickens believe the slow process to be the best. They contend that an antigen prepared by this method contains more precipitinogen than by either of the other processes.

For the preparation of the antigen, any of the tissues of the body may be used. The spleen, blood, serous or hemorrhagic exudates are preferable and in the order named. Of the remaining organs, the liver, lungs, kidney, muscle and skin should be mentioned. In case the skin is used, plenty of subcutaneous tissue should be included with it. According to Negroni, the presence of anthrax may be detected in imported skins by this method.

The quality of the immune serum is roughly estimated as follows: The serum that will produce a reaction immediately when brought into contact with an anthrax antigen is considered univalent. It has been learned that good sera will show the presence of an anthrax antigen in a normal unit and, also, in fractions of the same. A serum that reacts to an antigen diluted 100 times is a hundred fold serum. This is called a standard serum.

In testing the material for the presence of anthrax by the Ascoli reaction, it is not known whether the precipitinogen is present or not, hence its titration is impossible. But in case of the titration of the immune serum where the antigen is prepared from known anthrax material, some kind of a standard is necessary. For this standard, the culture extract is used.

TECHNIC OF THE TEST

According to Ascoli, the serum should first be placed in a test tube after which the extract, the lighter of the two fluids, should be placed beneath it by means of a capillary pipette. According to Schutz and Pfeiler, just as good results are obtained by placing the heavier fluid, the immune serum, in the tube first and then placing the antigen on top of it. They use the same technic used by Pfeiler in his diagnosis of glanders by the precipitation reaction. With an ordinary pipette, they place the serum drop by drop on the edge of a small test tube, about 6 millimeters in diameter and 12 centimeters long. The serum is allowed to run slowly down the inside of the tube to the bottom. Enough serum should be added to bring the top of the fluid up about one-half a centimeter in height. After the serum has reached the bottom and remains stationary, the antigen should be placed on the edge of the tube in the same manner as the serum, and allowed to run slowly down the tube until it reaches the serum. It is advantageous to have the antigen take the same course down the tube as the serum. When the extract reaches the serum it should form a sharply defined layer above it. If the extract mixes with the serum, at the point of contact, the test will be ruined. However, if the extract does not mix with the serum, and the two fluids form, at the point of contact, a sharply defined division line, the technic has been properly executed. In case the antigen was prepared from anthrax material, a cloudy, grayish white ring will form immediately at the point of contact of the two fluids. This ring gradually increases in density for some time. It may be seen best in an oblique light. The test tubes used in this reaction do not necessarily have to be sterile, but it is imperative that they should be scrupulously clean, for if they are not clear the ring may not be seen.

In case the antigen is prepared from material taken from an animal that did not have anthrax, this ring will not occur, or if it does occur will not take place under fifteen minutes. In case the reaction is positive, the ring remains visible for two hours, and longer, after which it disappears entirely. A test, however, may be read on the succeeding day by the presence of sediment in the bottom of the tubes containing the positive reaction. The tubes containing negative reactions will show no precipitation.

Control. Schutz and Pfeiler state that with reliable sera the control tests may seem superfluous, but still they do not wish to discard them. They make control tests only with suspicious material, and not with material known to be negative or positive. They make the statement that the extract, in every case, should be tested with normal serum from the same species of animal from which the antigen originates. They also use a positive and negative control for their antigen in routine work. In all, this makes three controls which are, the normal serum, the anthrax extract and the normal organ extract.

Anthrax is to be differentiated from certain specific diseases such as *symptomatic anthrax (black leg)*, malignant edema, and septicemia hemorrhagica. Rabies is not infrequently mistaken for anthrax. These diseases can as a rule be readily diagnosed by the methods applicable to each. In addition to the specific infections, anthrax has been confused with certain dietary troubles and poisoning.

Protective inoculation. Toussaint was the first to make use of protective inoculations in anthrax. He heated defibrinated blood to a temperature of 55° C. for 10 minutes. Better results were obtained by heating the blood to 60° C. for 3 or 4 times before using it. Pasteur, however, was the first to prove that immunity could be obtained by the use of cultures of attenuated bacteria. Several methods of attenuating the specific organisms were proposed by Pasteur, Toussaint, Chaveau, Chamberland, Arloing and others.

Pasteur's method consists in inoculating the animal with a small quantity of culture which has been grown at a high temperature— 42 to 43° C.—for several days. This deprives the bacteria of their virulence. To strengthen the resistance, the animals are again inoculated 12 days later with a stronger virus.* After the two inoculations, they are said to be protected against the most virulent anthrax; but the immunity is of short duration. Chamberland reported in 1894 that a total of 1,988,677 animals were treated by this method in France, and that the loss from anthrax had diminished from 10 per cent. in sheep and 5 per cent. in cattle to less than 1 per cent. Cope, in his report to the English Board of Agriculture, regards the conclusions of Chamberland as somewhat fallacious, because in order to prove that the animals inoculated received immunity, it should be shown that they were subsequently exposed to the risks of natural infection. The excellent work which has been done by Neal and Chester, at the Delaware College Experiment Station, has shown the possible efficiency of this method. Of the 331 cows which they vaccinated against anthrax, two died of the disease, giving a death rate of less than 1 per cent. and this in a territory so saturated with the virus that it was practically impossible to keep cattle at all before its

---

*The first vaccine is a culture of anthrax bacteria that has been cultivated so that it will kill mice but has no ill effect on rabbits and sheep. The second vaccine, given 12 days later, is more virulent. It will kill mice and guinea-pigs and occasionally rabbits. Immunity is established 12 days after the second inoculation.

The French recommend the following plan of injection. The first vaccine is injected into the internal surface of the right thigh and the second into the internal surface of the left thigh. The vaccine should be used as soon as it is procured. A contaminated vaccine should not be injected.

use. The objection to this method is, that it requires the use of the living bacteria, which later may become virulent and consequently cause a subsequent outbreak. The scattering of pathogenic organisms, even in an attenuated condition, should be avoided if possible. It must be admitted, however, that Pasteur's method has done much good and helped to rob anthrax of much of its former terror, especially for the farmers of Europe. In America the spread of anthrax has been checked in many districts by its use. Dalrymple has pointed out its success in the lower Mississippi Valley. Chester and Neal used it successfully in Delaware. They pointed out that a vaccine which succeeded at one time proved fatal at a subsequent time. Notwithstanding, it is highly probable that the spreading of a knowledge of the specific cause of this disease with instructions for the proper disposition of dead animals has also exerted much influence for good in checking its ravages.

In Germany and England the stamping-out system is considered superior to vaccination. According to Crookshank, in England it is regarded as the only reliable means of suppressing the disease. To this end rigid laws have been enacted. In this country as rigid measures as possible for its eradication should accompany the use of methods for establishing a tolerance for its existence.

The simultaneous method. This method consists in the injection of anthrax serum* together with a small quantity of virulent anthrax bacteria. It has proven to be very satisfactory. It has the advantage of being administered at one time. This method of protection against anthrax seems to have been first proposed by Sobernheim in 1899. He reports excellent results from its use in immunizing cattle and sheep against anthrax in South America. Sclavo has produced serums which seem to have a therapeutic as well as prophylactic value. It has been used in treating human anthrax since 1897.

Prevention. In all cases the well animals should be removed from the barns or yards containing the sick ones and from pasture lands on which the sick became infected. The temperature of the apparently healthy animals should be taken morning and evening from one to two weeks after they are removed and all of those showing an elevation of temperature should be isolated. *By careful isolation and safe*

---

*Horses are immunized in from 10 to 12 days. They are injected with 5 cc. of the serum and are from 0.25 to 0.30 cc. of a culture of anthrax bacteria in different parts of the body. According to Sobernheim the successful use of the serum depends upon using the virus in combinations with it.

disposition of the dead animals the spread of the disease can be checked.
Animals do not, as a rule, spread the virus when the first symptom
(rise of temperature) can be detected. All infected stables and yards
should be thoroughly disinfected.

The disposition of dead animals in an outbreak of anthrax is a matter
of much importance. In all cases they should be burned if possible,
if not, they should be deeply buried and covered with quick lime
before the dirt is replaced. The ground over the place where they are
buried should be fenced in to prevent other animals from grazing over
it, and the surface should be burned annually for some years to
destroy spores should they be brought to the surface.

Control. Owing to the long resistance of the anthrax spores, it is
impossible to render an infected field safe for pasture land. After a
number of years such fields may become harmless. The safe disposal
of all carcasses by burying deep or cremation; the thorough disinfec-
tion of all stables and paddocks; and the burning of all litter that
might have become infected are precautions that should be taken.

In localities where there are frequent outbreaks of the disease
regular vaccination of all cattle, horses, sheep and swine is advocated.
It is all that can be done beyond preventing exposure to known in-
fected localities. Anthrax should be reported but a quarantine
against healthy animals does not seem to be necessary as the disease
is not ordinarily communicated directly from one animal to another.
There is a possibility of removing animals while in the period of
incubation, thereby infecting other places. Little is known about
the "carriers" in this disease.

REFERENCES

1. Ascoli. Die Prazipitindiagnose bei Milzbrand. *Centralbl, f. Bakt. u. Parasitenk.,*
Bd. LVIII (1811), S. 63.

2. BURNETT. The control of an outbreak of anthrax. *Am. Vet. Review,* Vol.
XXXIII (1908), p. 136.

3. CHESTER. Anthrax, bacteriological work. *Report Del. Agr. Expt. Station,*
1895, p. 64.

4. CHESTER. Protective inoculation against anthrax. *Proceedings of the Society
for the Promotion of Agricultural Science,* 1896, p. 52.

5. DALRYMPLE. Anthrax and protective inoculation in Louisiana. *Proceedings
of the Am. Vet. Med. Assn.,* 1901, p. 147.

6. DAVAINE. Recherches sur les infusoires du sang dans la maladies connue sous
le nom de sang de rate. *Compt. Rend. de l'Acad. des Sc.,* 1863, 1864, 1865.

7. EICHHORN. Experiments in vaccination against anthrax. *Bulletin No. 340,*
U. S. Dept. of Agric., 1915.

8. FISCHOEDER. *Berliner Tierärztliche Wochenschrift,* Bd. XXIX (1913), Nos. 36,
37, 38.

9. Fitch. Organisms morphologically resembling anthrax bacteria. *Report N. Y. State Vet. College at Cornell University*, 1909–10, p. 200.

10. Koch. Die Aetiologie der Milzbrand-Krankheit begründet auf die Entwicke-lungsgeschichte des Bacillus Anthracis. *Cohn's Beitr. zur Biol. der Pflanzen*, Bd. II (1876), S. 277.

11. Kodama. Ursache der natürlichen Immunität gegen Milzbrandbacillen. *Centralbl. f. Bakt. u. Parasitenk.*, Bd. LXVIII (1913), S. 373.

12. M'Fadyean. Anthrax. *Jour. Compar. Path. and Therap.*, Vol. XI (1898), p. 51.

13. M'Fadyean. A peculiar staining reaction of the blood of animals dead of anthrax. *Jour. of Compor. Path. and Therap.*, Vol. XVI (1903), p. 35.

14. M'Fadyean. Extraneous sources of infection in outbreaks of anthrax. *Jour. Compar. Path. and Therap.*, Vol. XVI, p. 346.

15. Moore. Report of an outbreak of anthrax. *Annual Report, Commissioner of Agriculture of the State of New York*. 1897, p. 550.

16. Pasteur, Chamberland et Roux. De l'attenuation des virus et de leur retour à la virulence. *Comp. Rend. d. Acad. des Sc.*, Vol. XCII (1881), p. 427.

17. Pasteur. La vaccin du charbon. *Ibid.* p. 666.

18. Pickens. The determination of anthrax by means of the thermo-precipita-tion reaction. *Report New York State Veterinary College at Cornell University*, 1914, p. 220.

19. Pokschischewsky. Uber die Biologie der Pseudomilzbrandbacillen Beiträge zur Differentialdiagnose der Milzbrand und Pseudomilzbrand bazillen. *Arbeiten a. d. Kaiserlichen Gesundheitsamte*, Bd. XLVII, S 541.

20. Schutz and Pfeiler. Der Nachweis des Milzbrandes mittels der Präzipi-tationsmethode. *Archiv. f. wissen. u. prakt. Tierheilkunde*, Bd. XXXVIII (1912).

21. Russell. Outbreak of anthrax fever traceable to tannery refuse. *The 17th Annual report of the Wis. Agric. Exp. Station*, 1889.

22. Sobernheim. Ueber das Milzbrandserum und seine praktische Anwendung. *Deut. med. Wochenschr.*, 1904. No. 26 u. 27. (First publication. *Zeit. für Hygiene*, 1899, Bd. XXXI).

GLANDERS

Synonyms. Malleus; farcy; *morve: Rotzkrankheit.*

Characterization. Glanders is one of the most important diseases of horses, asses and mules. It is communicable to man. It runs an acute or chronic course, attacking the lymphatic system more espec-ially in the upper air passages, lungs or skin. The disease is charac-terized by a strong tendency to the formation of small neoplasms or nodules which are likely to degenerate into ulcers from which exudes a peculiar sticky discharge. In the very acute cases a considerable rise of temperature and general debility may accompany the forma-tion of the lesions. Glanders of the skin is known as farcy.

By direct inoculation several species of animals may be infected. Thus the disease has been reported in goats, rabbits, sheep, guinea pigs, field mice, and several of the wild animals, especially those of the cat tribe. Swine and pigeons are very slightly susceptible. Cattle, white mice, rats and domestic fowls seem to be immune.

History.   Glanders is reported to have been known long before the Christian Era. The name malleus was given to it by Aristotle. The theory of the contagiousness of glanders was much doubted at the beginning of the last century.   The view taken by the veterinarians at the Alfort Veterinary College was that glanders might arise spontaneously from an attack of strangles.   This view was far more widely accepted than the theory of its contagiousness, which was stoutly supported by the authorities at the Veterinary College of Lyons.   It was not until Rayer (1837) had demonstrated the transmissibility of glanders to man, and Chauveau (1868) had shown that the virus was contained chiefly in the firm component parts of the infective material, that the fact of the infectious nature of the disease was accepted.

The theory of the spontaneous origin of glanders was widely accepted in Germany. It was believed that glanders could be produced by the injection of pus, and that strangles could develop into glanders.   Glanders was looked upon as a tubercular disease, scrofula, pyemia, diphtheritis, general dyscrasia and cachexia respectively. Virchow was the first to declare that the nodules of glanders were independent, anatomical formations, which he placed under the heading of granulation tumors.   Gerlach was the strong advocate for the exclusively infectious origin of the disease.   Leisering appears to have been the first to give an accurate description of the lesions.

The first biological researches into its nature were made in 1868 by Zurn and Hallier, who found a fungus which they believed to be its cause.   In 1882, Lœffler and Schütz succeeded in finding the bacterium of glanders, in cultivating it, and in transmitting the disease to other animals by inoculating them with pure cultures of the organism.   Their researches furnished the positive proof that glanders is a specific, infectious disease, produced exclusively by *Bacterium mallei.*

Geographical distribution.   Glanders exists in the greater part of the civilized world.   It is more common in the temperate zones, where traffic in horses is active.   In the United States it was largely confined to the Northern States before 1861, but it spread over the South in connection with the civil war.   It is said to have entered Mexico with the American cavalry in 1847.   Similarly, Portugal is said to have been exempt until the invasion by Napoleon in 1797.   Central Hindoostan was said to be free from it until the war with Afghanistan in 1878.   In all these cases, the movements of cavalry, artillery and of commissary trains were responsible for the introduction of the

disease into new territory. In our own case the sale of horses and mules at the close of the civil war produced a very general diffusion of this disease, from which the country is still suffering.

Insular places, especially if far from the main land and free from importation of horses, usually escape. Thus glanders is very rare in Iceland and in the Faroe islands. In Australia, Tasmania and New Zealand it is reported to be unknown.

Etiology. *Bacterium mallei*, the specific cause of glanders, was discovered and isolated in pure culture almost at the same time (1882) by Lœffler, Schütz, Isræl, Bouchard, Charrin, Weichselbaum, Kauzfeld and Kitt. It is found in the recent nodules, in the discharge from the nostrils, pus from the specific ulcers, and occasionally in the blood of animals affected with acute glanders.

Morphologically it is a small organism with rounded or pointed ends. It varies in breadth from 0.25μ to 0.4μ and from 1.5μ to 3μ in length. It is usually single but pairs and long filaments, especially on potato cultures, are not rare. It frequently breaks into short, almost coccus-like elements. Galli-Valerio found great variations in its morphology when grown under certain different conditions. Branching forms were numerous.

It stains with some difficulty. Of the aniline dyes the best results are obtained with the aqueous solutions, when they are made feebly alkaline. It is decolorized by Gram's method.

It grows well, but slowly, at the body temperature on acid-glycerin agar, in acid-glycerin bouillon, on blood-serum and on potato.

Of the test animals guinea pigs and field mice are the most susceptible. In guinea pigs, subcutaneous injections are followed in four or five days by swelling at the point of inoculation and sloughing of the skin, which are followed by the formation of a chronic, purulent ulcer. The lymphatic glands become inflamed and symptoms of general infection develop in from two to four weeks; the glands suppurate and in males the testicles are involved. A purulent inflammation of the joints may occur. The formation of the specific ulcers upon the nasal mucous membrane, which forms one of the characteristics of the disease in the horse, rarely occurs in the guinea pig as a result of inoculation. The disease is often prolonged for several weeks or months. Guinea pigs succumb usually in from eight to ten days when injected into the peritoneal cavity with a virulent culture. In males, the testicles are invariably affected. The inoculation of male guinea pigs and diagnosis of glanders by the orchitis that follows is

known as the Strauss method. Hallopian and Bureau observed an orchitis following the inoculation of pus from a case of human mycosis into the peritoneal cavity of a guinea pig. Nocard recorded nineteen cases of a slightly contagious, farcy-like lymphangitis in horses due to a bacterium which produced an orchitis when inoculated into guinea pigs but which was different from *Bact. mallei* both in its cultural characteristics and its reaction to the Gram stain (Benson). The inoculation of the guinea pig in making a diagnosis can be consid-

FIG. 9. THE SO-CALLED "GLANDERS EXPRESSION."

ered as only one factor. The bacterium must be obtained from the lesions and identified. Infection takes place through the digestive tract. Experimentally it has been induced through the respiratory organs.

The period of incubation is not definitely known. It varies from a few to many days, depending upon the method of infection and the virulence of the organisms, as well as the resistance of the animal.

Symptoms. Two forms of glanders have been recognized, namely, acute and chronic.

Acute glanders. Acute glanders is common in the ass and mule, but less frequent in the horse. After a short period of incubation the animal has a chill, elevation of temperature, a profuse muco-purulent,

sticky discharge, sometimes mixed with blood, from the nose. Particles of food arrested in the pharynx occasionally appear in the nasal discharge. If unilateral the margin of the nostril swells, the mucosa is dark red, infiltrated, marked with pea-like, yellowish elevations with red areolæ, which in a few days become eroded, thus forming spreading ulcers. The submaxillary lymphatic glands on the affected side become enlarged. There may, however, be a uniform swelling of the intermaxillary space. The course is rapid and death may occur in from the sixth to the fifteenth day. The acute form rarely if ever becomes chronic.

Chronic glanders. In the horse, this form of the disease may begin with a chill but usually the onset is very insidious. There may be a muco-purulent, sticky discharge, sometimes streaked with blood, from one or both nostrils. There may be intermittent or continued lameness, arthritis, edema of a limb, swelling of a testicle, cough, or epistaxis. There is usually a nodular but comparatively painless swelling of the submaxillary lymph gland on the affected side. On palpation the swelling imparts a sensation suggestive of a number of peas. They are adherent to the adjacent structures. The nasal mucosa is congested, of a dark reddish color and sprinkled with superficial or deep ulcers either clean or covered with crusts.

Rarely the submaxillary glands only are apparently diseased. In other cases, there is only a cough, the lesions being confined to the lungs. Occasionally, the lesions are restricted to one or both testicles, the spleen, or other internal organ. Objective symptoms may or may not be present. Chronic glanders may terminate in the acute form.

In chronic, cutaneous glanders, with or without edema of the limbs, there may be one or many nodules on the fetlock, or elsewhere on the line of the lymphatic vessels, with induration of the lymphatics extending from it. The nodules may be suppurating and discharging, or they may be closed. The period of incubation and duration found after a lecture on morbid anatomy.

Morbid anatomy. In chronic glanders the most frequent locations of the lesions are on the respiratory mucous membrane, in the lungs, lymph glands and skin. M'Fadyean states that he has never seen a case of glanders in which the lungs were not affected if any lesions were found. Other organs are more rarely invaded. The mucous membrane of the upper respiratory passages is the usual seat of the lesions. Glanders occurs in two forms, (1) as circumscribed nodules with the formation of ulcers and cicatrices; and (2) as diffuse or infiltrated lesions.

In nodular glanders, which is the common form, the lesions are most frequently situated on the upper portion of the nasal septum and in the cavities of the turbinated bones. The affection begins with the appearance of nodules varying in size from a grain of sand to a millet seed. They are more or less translucent, of a roundish or oval shape, and of a dirty gray or grayish-red color. The nodules, which may attain to the maximum size of a pea, project somewhat above the surface of the mucous membrane. They are surrounded by a reddish ring. Some of them are isolated and others are arranged in groups. Microscopically they consist of a large number of lymphoid cells, which disintegrate in the centre of the nodule. In consequence of the central fatty and purulent degeneration, the nodules become yellowish in color, discharge and form ulcers. These ulcers are sometimes superficial, sometimes deep, lenticular or crateriform, surrounded by a hard, indurated edge, and frequently becoming confluent, with irregularly serrated and eroded edges. They are sometimes covered with a brownish crust. The ulcers may increase in area or in depth and may even involve the underlying cartilage or bone, causing perforation of the septum nasi, and distensions of the maxillary or exostoses on the turbinated bones. The shallow lenticular ulcers may heal without leaving any visible

Fig. 10.   Nasal septum showing ulcers.

changes; but the deeper ones, after granulating, leave a radiating, star-shaped cicatrix which is either smooth or horny, and which, according to the shape of the ulcer, may be of an irregular or oblong
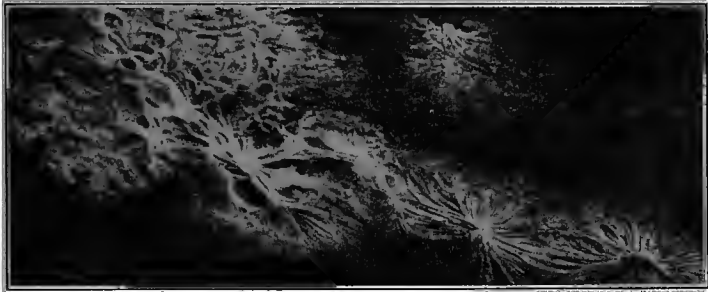
FIG. 11.   SCAR TISSUE FOLLOWING GLANDERS ULCERS, NASAL SEPTUM HORSE. (AFTER JOEST.)
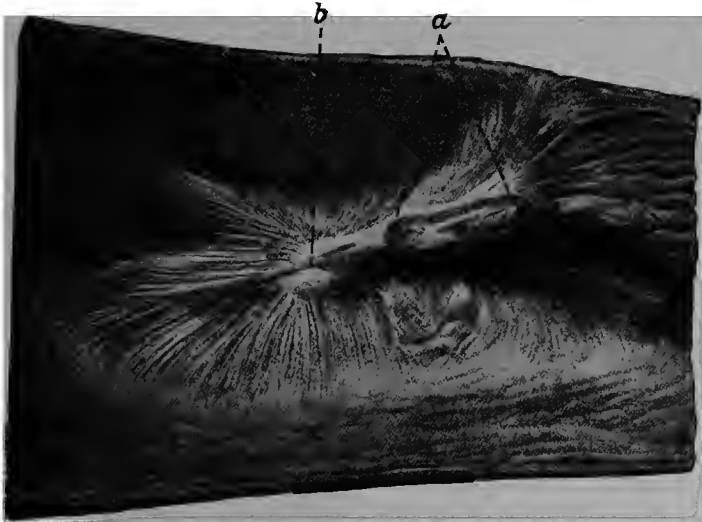
FIG. 12.   SCARS FROM NON-GLANDERED LESIONS ON NASAL SEPTUM HORSE. (AFTER JOEST.)

form. The nasal septum is frequently covered with these scars. The ulcers and cicatrices are sometimes found in the maxillary and frontal sinuses, in the guttural pouches and in the eustachian tubes. They may also occur in the larynx, especially in the region of the vocal chords. In the trachea and even in the bronchi, particularly on the anterior surface, numerous long, oval ulcers or long, pointed,

serrated scars are occasionally found. In addition to the ulcers,
a catarrhal inflammation of the mucous membrane is very apt to
be present.

Diffuse glanders manifests itself as a diffuse catarrh of the
mucous membrane of the nasal and neighboring cavities, with
superficial ulceration, thrombosis of the veins, inflammatory in-
filtration of the submucosa, considerable thickening of the mu-
cous membrane and the formation of a peculiar, radiating cicatrix.

Both the nodular and infiltrated forms are found in the lungs.

In the nodular form, the lungs contain nodules* varying in size
from a millet seed to a pea. They are gray by transmitted light,
glassy and pearl gray by reflected light, and are surrounded by a con-
gested or a hemorrhagic ring.   The center of the nodule shows a pale
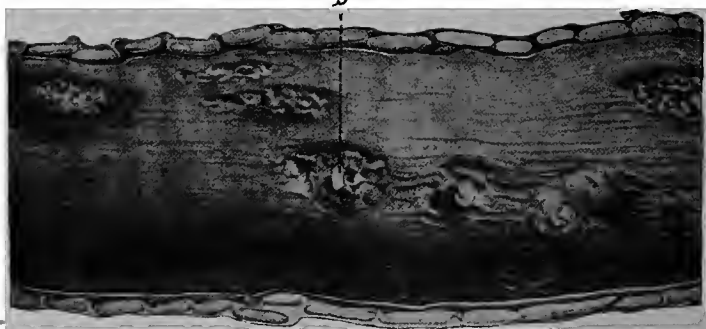
FIG. 13.   GLANDERS ULCERS IN THE TRACHEA (A) PERFORATION.
(AFTER JOEST.)

yellow point in consequence of caseation and disintegration of the
innermost cells.   These nodules are of different sizes, of varying
numbers, and of different ages.   The formation of a capsule by a con-
nective tissue membrane is induced by a reactive inflammation in the
tissue surrounding the nodule.   The nodules may be of an embolic
origin, situated principally in the periphery of the lung, their structure
being the same as that of the nodules in the nasal mucosa.   Some-
times the lung nodules represent lobular pneumonic foci, in which the
alveoli are filled with red and white blood corpuscles and with des-

*Nocard showed that when glandered horses are treated with mallein, a certain pro-
portion of them recover, in which case nodules that are present in the lungs cease to
contain living bacteria, a fact he has fully proved by inoculation.   On postmortem
examination the nodules may be readily felt by passing the hand with firm pressure
over the surface of the lung, which, when badly diseased, will feel like a bag full of shot
or peas.

quamated epithelium of the lungs. Central disintegration occurs very early. These areas are surrounded by a membrane resulting from a reactive inflammation which manifests itself and out of which a connective tissue capsule develops later on. There are two theories

FIG. 14. LUNG OF HORSE SHOWING SMALL AND LARGER GLANDERS NODULES.

concerning the structure of the early nodules. One is, that the first cells are epithelial in nature, thus closely resembling a tubercle. The other is that the first stage of the nodules consists of air cells filled with leucocytes.

M'Fadyean has called attention to the structure of the lung nodules, in which he finds a central part composed of leucocytes that have filled the air spaces, the walls of which have disappeared as if by liquefaction. This is surrounded by a zone of epithelioid cells. A third zone surrounds this, in which the walls of the air vesicles are

FIG. 15. (a) MASS OF FIBROUS TISSUE SURROUNDING QUITE LARGE BRONCHI IN GLANDERED LUNG; (b) GLANDERS NODULE.

recognizable. The walls are thickened. The fourth zone is composed of air vesicles filled with a fibrinous exudate, which entangles a few leucocytes. Frequently the exudate is free from red blood corpuscles, but at times it contains much blood. In older nodules the third and outermost zone is composed of cirrhotic lung tissue, in which can be distinguished the remains of the air cells. This zone passes gradually into the normal tissue. In the last stage the central
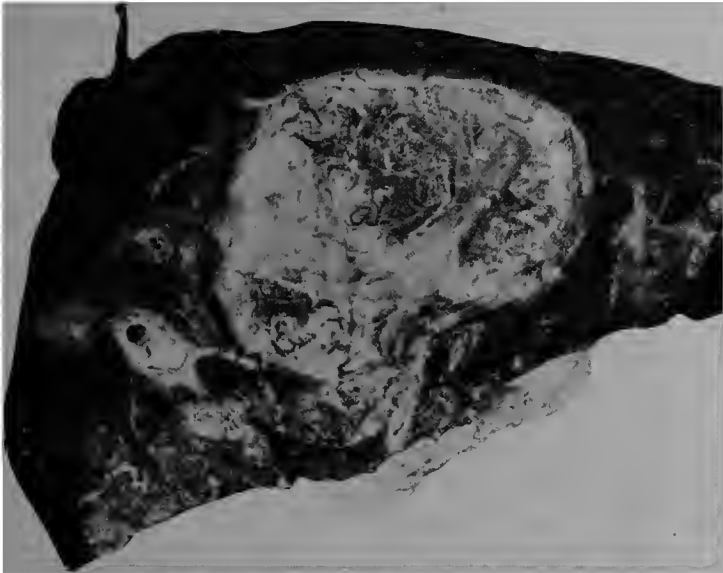
Fig. 16. Large glanders nodule undergoing organization (its development of fibrous tissue).
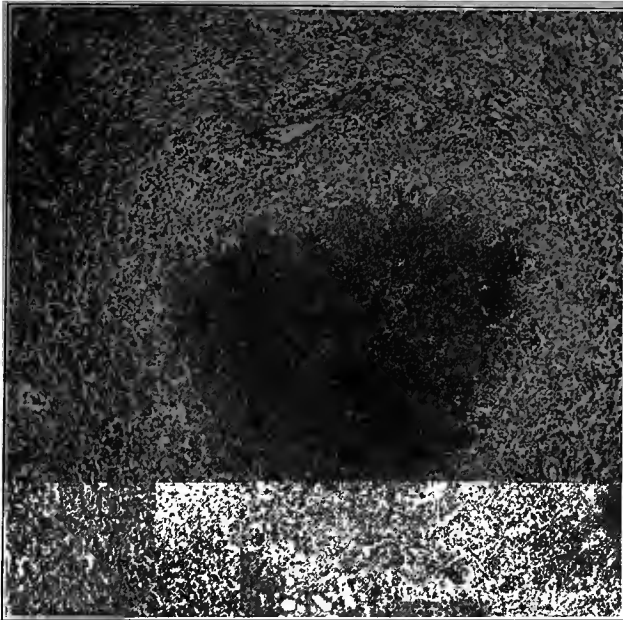
Fig. 17. Glanders nodule. Low magnification

area shrinks and becomes calcified, while the other zones become con-verted into a distinct fibrous capsule. Other observers have not reported the calcification. It has not occurred in the writer's observation. The cell necrosis in glanders has been designated by
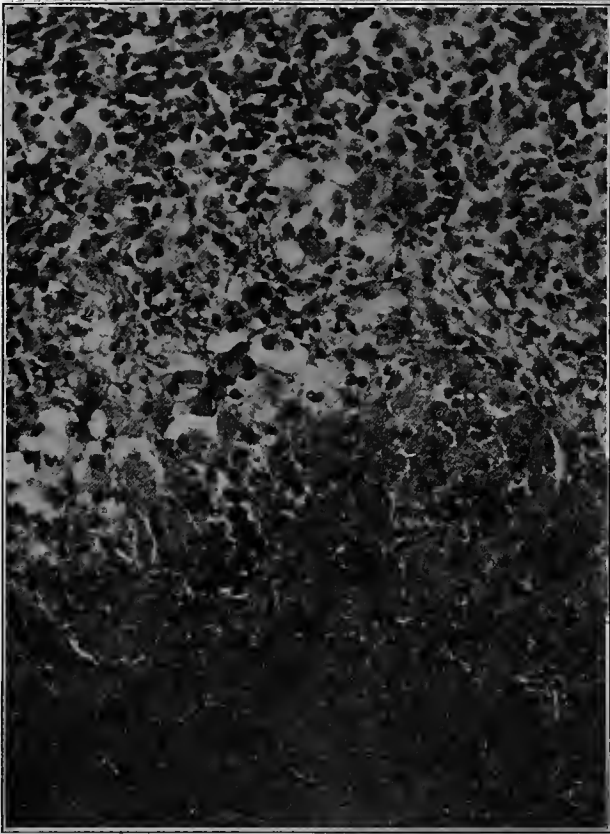
FIG. 18. A PORTION OF A GLANDERS NODULE (FIG. 17) EXTENDING FROM THE NECROTIC CENTER TO THE NORMAL TISSUE. X 380.

Unna as chromatolasis which consists in the disintegration of the nucleus before the destruction of the cell body and the retention of the staining property of the broken, nuclear chromatin. This gives the dark color in the central part of a stained nodule.

Besides these nodules, there are often chronic bronchitis, peribron-chitis, parabronchitis, atelectasis, inflammation of the tissue of the lung and less frequently circumscribed or exudative pleuritis.

Infiltrated glanders of the lungs forms tumors from the size of a walnut to that of a child's head, consisting of a diffuse glanderous infiltration of the alveoli and of the interstitial connective tissue. Frequently on section the infiltrated parts of the lungs resemble very closely a soft sarcoma. They are of a dirty white color, of a gelatinous, juicy consistency and irregular in shape. They may either become indurated so as to form hard, connective tissue-like new growths (fibroma-like tumors of glanders, according to Gerlach), or they may become gangrenous. At times there appear masses of connective tissue of varying size at the borders of which glanders bacteria are found. In nodular and in infiltrated glanders of the lungs, the bronchial glands and frequently the mediastinal glands become enlarged, indurated and studded with small foci of cell infiltration.

In glanders of the skin (farcy) the nodules are found in the papillary layer, in the cutis and in the subcutaneous and superficial intermuscular tissue. The cutaneous nodules vary in size from a hemp seed up to a pea. They suppurate rapidly and form small ulcers. The nodules in the subcutis are inflammatory (metastatic) tumors from the size of a pea to that of a hen's egg. They change into large abscesses and discharge externally. In the region of the nodules the lymphatic vessels are inflamed, swollen, and frequently resemble a rosary or knotted cord. Ulcers often develop from these secondary nodes. The neighboring lymph glands are at first swollen and soft, but later they become indurated by the growth of connective tissue and studded with dirty white nodules about as large as a pin head, or with yellow foci of

FIG. 19. SECTION OF A GLANDERS NODULE IN THE LUNG OF A HORSE: (a) NECROTIC CENTER, (c) ZONE OF GIANT CELLS, (b) CAPSULE SURROUNDING THE NODULE (Schutz).

caseation. The capsule around the

lymph glands becomes infiltrated with small cells and subsequently thickened. In rare cases secondary chronic farcy occurs. It is marked by a large, diffuse new growth of connective tissue with nodular thickening of the skin. This condition is termed glanderous elephantiasis or pachyderma. It chiefly affects the limbs and head.

Of the abdominal organs, the spleen is most frequently attacked. It then contains embolic nodules, which vary in size and either suppurate or become calcareous. Similar nodules occur, though not so often, in the liver, kidneys, testicles, brain, muscles, heart and bones. In the bones, the lesions consist of a cellular infiltration of the medulla and purulent breaking down of the osseous tissue. Ulcers are
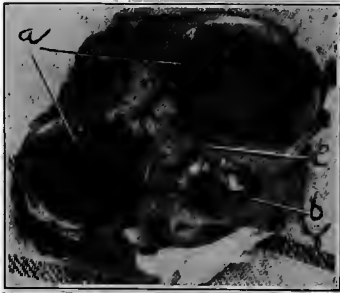
FIG. 20.   GLANDERS NODULES IN LYMPH GLAND DISCHARGING INTO BRONCHUS.   (a) BRONCHI, (b) LYMPH GLAND, (c) OPENING INTO BRONCHUS.
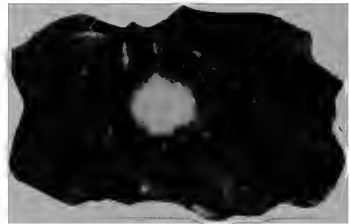
FIG. 21. GLANDERS NODULE IN SPLEEN OF HORSE.   NATURAL SIZE.

very rare on the mucous membranes of the eyes, stomach and vagina. The blood shows signs of slight leucocytosis. The specific bacteria are found in the blood only in cases of acute general infection.

The anatomical changes in acute glanders consist chiefly in a disintegration of the respiratory mucous membrane, in a serous infiltration of the submucosa, subcutis, and intermuscular tissue, with inflammation and suppuration of the lymph vessels and glands. There are also metastatic formations in the skin and lungs. The nasal mucous membranes are covered with rapidly spreading ulcers with considerable infiltration into the submucosa. The mucous membrane of the larynx and pharynx may be swollen and covered with ulcers. The lungs are studded with purulent metastatic foci or fresh nodules. The skin is excessively swollen and covered with glanderous nodes. Sometimes diffuse gangrene of the skin occurs.

Glanders in man. The symptoms of glanders in man are of much importance to the veterinarian. Although the susceptibility to the disease is usually not very great, cases of human glanders unfortunately occur, especially among veterinary surgeons and those having the care of horses. Human glanders is reported to be quite common in Russia. Robins has reported 156 cases collected from the

FIG. 22.   SKIN GLANDERS (FARCY).

literature. The parts usually first affected are the hands, nasal mucous membrane, lips and conjunctiva. After a period of incubation of from three to five days the infected part becomes swollen and painful, with subsequent inflammation of the lymph vessels and swelling of the glands. Fever is often the first symptom, and it is nearly always followed by a nasal discharge, ulcers on the nasal mucous membrane, pustules and abscesses in the skin, ulcers in the oral cavity, larynx, and conjunctiva, articular swellings, and grave general disturbances. Sometimes there is intense gastro-intestinal trouble. Nodules occur in the lungs in some cases. As a rule, death

takes place in from two to four weeks. In other instances, the disease becomes chronic, lasting for months or years. *Bact. mallei* has been found in the blood in cases of acute glanders. The positive diagnosis depends on the possibility of infection having taken place, on inoculation in guinea pigs, the proof of the presence of *Bact. mallei* or positive results with sera tests. Treatment is usually of no avail. The only hopeful cases are those that are purely local in their manifestation. A few of these are reported to have been cured by applying deep cauterization.

Diagnosis. Glanders is to be diagnosed by the symptoms, lesions, cause and specific reactions of which there are several. In somewhat "typical" and advanced cases the diagnosis may be very accurately made from the manifestations. The positive diagnosis, however, must be made from the identification of *Bact. mallei,* or from one or more of the specific tests. In the dead animal the histology of the lesions may reveal the nature of the disease.

Lesions. The lesions exhibited in a living animal are not sufficiently characteristic to positively identify the disease in very many, if in any, cases. The histological structure of the nodules may, however, enable one to do so.

Bacteriological examinations. The examination of the lesions for *Bact. mallei* can be made by cultures or guinea pig inoculation. In case of nodules in internal organs cultures on acid-glycerine agar or bouillon or on potato are quite satisfactory.

Animal inoculation. Male guinea pigs should be used. The material for inoculation usually consists of the nasal discharge from the suspected glandered horse, bits of scrapings from the ulcers, or pieces of other diseased tissue may be injected subcutaneously or into the abdominal cavity. The first symptom of glanders noticed is usually orchitis. The lymphatic glands in the groin are also enlarged. After the orchitis becomes well advanced, the guinea pig may be chloroformed and examined. Pure cultures of the specific organism can be obtained in most cases from the suppurating foci in the testicle. The spleen is usually enlarged and sprinkled with grayish nodules. Other organs may be involved. The diagnosis by the inoculation of a male guinea pig is known as the Strauss method. It is important to note that the orchitis alone is not sufficient to make a positive diagnosis but the specific organism must be found and identified.

Mallein. Mallein is prepared in the same way as tuberculin. It was used by Kaling and Helman independently as a diagnostic agent.

It consists of the glycerinated bouillon in which the glanders bacteria have grown and in which are the products resulting from their multiplication. It has a somewhat fetid odor. Mallein is applied in two ways, namely, subcutaneously and on the conjunctiva. The latter is called the ophthalmic method.

Subcutaneous method. In applying mallein the horse is injected usually in the neck with from 0.5 to 2 cc. of mallein, the quantity depending upon the degree of concentration. If a concentrated mallein is used it should be diluted with a 1 per cent. carbolic acid solution to at least 2 cc. The reaction is as follows. In a few hours there forms at the place of injection a hot, inflammatory swelling. It is very painful and in case of glanders quite large. From all sides of the swelling there may radiate wavy lines consisting of swollen lymphatics, hot and painful when touched, extending toward the adjoining glands. When the mallein injection is made aseptically, this swelling never suppurates, but it may increase in size during a period of from 24 to 36 hours and persists for several days, when it gradually diminishes and finally disappears at the end of eight or ten days. With the appearance of the local swelling the patient becomes dull and dejected, the eyes have an anxious expression, the coat is lusterless, the flanks contracted, the respiration hurried and the appetite is impaired. Frequent shudders are observed to pass through the muscles of the fore legs and sometimes the trunk is subject to violent convulsive movements. The most active and fractious horses become listless and indifferent to their surroundings. These general phenomena constitute what the French call the "organic reaction," but they are not always so clearly marked. Differences in their intensity are observed but they are never completely absent.

The temperature reaction seldom fails to show itself. In about eight hours after the injection the temperature of a glandered horse gradually rise 1.5°, 2° or 2.5° F., and even more above the normal. The rise in temperature usually attains its maximum between the tenth and twelfth hour, occasionally not till the fifteenth, and more rarely not until about the eighteenth hour. An important fact to note is that the reaction called forth in glandered horses by the injection of mallein persists for from 24 to 48 hours and in some cases the temperature remains above the normal for an even longer time. In practice it is advisable to take the temperature of the suspected animals two or three times before the injection of the mallein, and every two hours, beginning at the eighth and going to the twentieth

hour after the injection. It is often sufficient for diagnostic purposes to take the temperature but four times, viz., at 9, 12, 15, and 18 hours after the injection, but a longer observation would be more reliable.
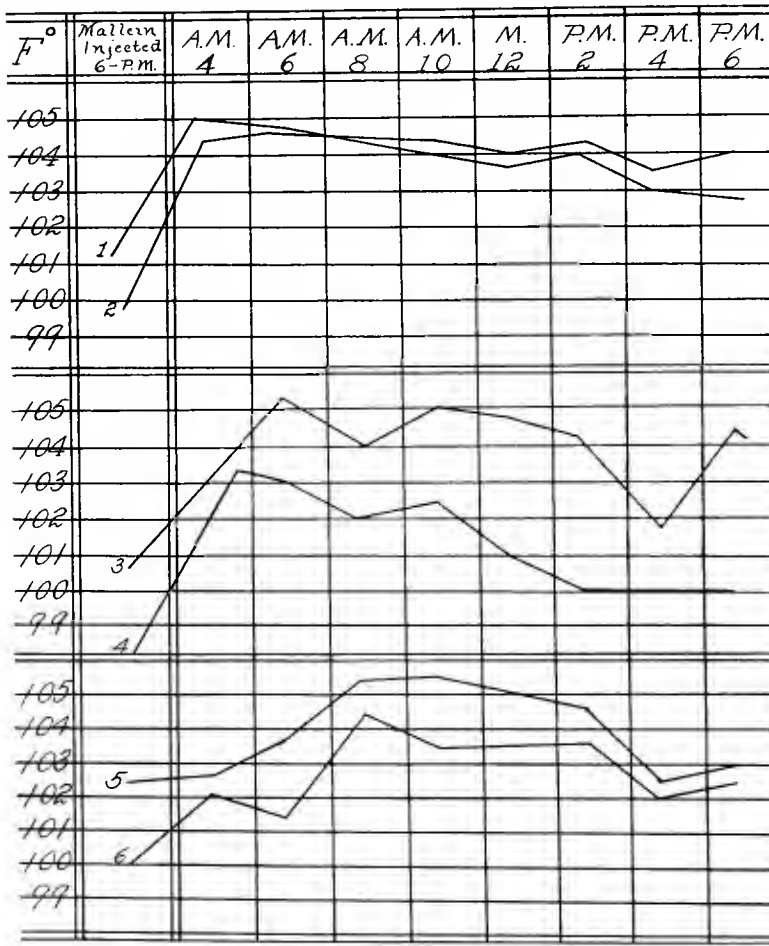

FIG. 23. MALLEIN REACTION. TEMPERATURE CURVES OF SIX HORSES FOR 24 HOURS AFTER INJECTING MALLEIN. THE HORSES WERE IN ONE STABLE FROM WHICH A WELL DEVELOPED CASE OF GLANDERS HAD BEEN REMOVED.

In healthy horses the injection of mallein, even in a much larger dose, produces no effect on the temperature or the general condition of the animal. There is produced, however, at the point of injection,

a small edematous swelling, somewhat hot and painful to the touch, but the edema instead of increasing, diminishes rapidly and disappears in less than 24 hours.

The reaction called forth by the injection of mallein in a glandered animal is quite specific. When it occurs one is enabled to state at once and with certainty that glanders exists, although the lesions may be quite minute or obscure. When the reaction does not take place it is generally considered that the animal tested is not glandered, although the physical examination may suggest it. Notwithstanding the specific action of mallein, its administration can give really useful indications "only when, and as far as, we can remove the causes of error that have been pointed out by experience." For example, it would be imprudent to use mallein in case of animals already suffering with an abnormally high or low temperature. The further precautions should be taken that the animals subjected to the test be removed as far as possible from atmospheric variations and the influence of strong sunlight, fog, rain and currents of air. If it be true that the majority of horses are not susceptible or slightly so, to these influences, there are still some that are affected by them. A sudden rise and fall of temperature due to other causes must be differentiated from a mallein reaction.

*Ophthalmic use of mallein.** Schnürer in Vienna and Fröhner in Berlin recommend this method of using mallein. The mallein is applied to the eye with a camel's hair brush in the following way: The eyelids are opened with the index finger and the thumb, as is customary when examining the conjunctiva of the eye. Then the camel's-hair brush, which has been submerged in the mallein, is drawn once forward and again backward over the eye. Only one eye is used, the other serving as a control. Immediately after the application of the mallein to the eye in most of the animals lacrimation, increased reddening, and twinkling of the eye appear; these primary reactions are not specific and disappear in the following few hours. The specific reaction commences as a rule 5 or 6 hours after the application of the test and lasts from 36 to 48 hours, occasionally even longer. It consists in a suppurative conjunctivitis, with reddening, swelling, and suppurative secretions. Of these signs only a sup-

---

*The ophthalmic use of mallein was first reported in this country by Dr. C. J. Marshall (Proceedings of the A. V. M. A. 1912). At the same time Moore and Fitch were using this method for the diagnosis of glanders in New York City (Report New York State Veterinary College, 1911–12).

purative secretion should be taken into consideration. The results
are interpreted as follows: (1) The reaction is positive if a suppura-
tive secretion is observed in varying quantities. If the secretion is
present in only a small quantity, it is principally visible at the inner
canthus of the eye. (2) The reaction is negative in the absence of any
secretion. (3) The reaction is doubtful when there is present a slimy
secretion or lacrimation after 24 hours.

The diagnosis should be made
not earlier than 12 hours and
not later than 24 hours after the
application of the test. The
examination should be made in
a good light. A positive result
indicates with certainty the
presence of glanders; negative
results, however, should not
eliminate the possibility of the
presence of the disease, and only
a repeated negative test after
three weeks excludes suspicion
of the disease.

Generally the positive oph-
thalmo reactions are not ac-
companied by fever or systemic
disturbances. Occasionally,
however, affected horses are
hypersensitive, so that often a
trace of mallein which enters
the circulation produces fever.
Accordingly, it is advisable to
accompany the ophthalmo reac-
tion with temperature measure-

FIG. 24.   EYE, FOLLOWING OPHTHALMIC USE
OF MALLEIN.

ments. For this purpose the temperature should be taken at least
twice, the first time when the mallein is applied and the second time
when it is judged. In a doubtful eye reaction where there is a rising
temperature of over 101.5° F., the test should be considered positive if
the animal had a normal temperature at the time the mallein was
applied.

The ophthalmic test is used officially in Austria. They employ the
Pasteur "Mallin brute," 0.75 cc. being used on 10 horses. In this

country both the concentrated mallein and a 5 to 7% solution of the precipitated mallein (Mallein Siccum Foth) are used. The ordinary mallein used for subcutaneous test cannot be relied upon.

This test should not be made in the presence of a conjunctivitis. It is also important, where there is a reaction, that the purulent discharge be not wiped out of the eye thereby leaving one in doubt. Like the subcutaneous method, there is no definite relation between the extent of the reaction and the amount of pathological changes that exist.

The cutaneous and intradermal application of mallein do not give uniformly satisfactory results.

The agglutination method or serum diagnosis. Rabieaux found that the difference which exists between the agglutinating power of a serum from a glandered and from a healthy horse may be used as the basis of a method for diagnosing glanders. He collected the serum as pure as possible, diluted it with sterile, distilled water to 1 in 10, or to 1 in 500. The diluted serum was then mixed in a small sterile tube with an equal volume of a 24 to 72 hour culture of *Bact. mallei* in peptonized bouillon (without glycerin). The mixture was placed in an incubator at a temperature of $35°$ to $37°$ C. and examined at variable times under the microscope. In dilutions of from 1 in 10 to 1 in 50 the agglutination occurred in 20 minutes to 3 hours. In serum of a non-glandered horse from 2 to 6 hours were required to produce the agglutination. In weaker dilutions the differences were more marked. The development of the method can be followed from the writings of M'Fadyean, Bourget and Méry, Arpàd, Fedorowsky, Reinecke, Bonome, Schütz and Miessner, Schnürer and Moore, Taylor and Giltner.

The method consists in the preparation of a test fluid from a suitable culture of *Bact. mallei* to which is added the diluted serum.

The "test-fluid" is prepared by washing the growth from a 72 hour acid-agar culture by the aid of a sterile wire loop into distilled water containing 0.85 per cent. sodium chloride and 0.5 per cent. carbolic acid crystals. This suspension is then placed in a thermostat at $60°$ C for two hours, which kills the bacteria. Three cubic centimeters of the "test-fluid" are placed in each of several small test-tubes. With a sterile pipette, the diluted serum is added to the tubes of test-fluid and thoroughly mixed. In making the different dilutions, the amount of diluted serum to be used is readily ascertained by the following table:

Dilution of Serum	Amount of Diluted Serum	Amount of Test Fluid	Dilution
1–40	1.2 cc.	3 cc.	1–100
1–40	0.6	3	1–200
1–40	0.405	3	1–300
1–40	0.3	3	1–400
1–40	0.24	3	1–500
1–40	0.195	3	1–600
1–40	0.15	3	1–800
1–40	0.12	3	1–1,000
1–40	0.105	3	1–1,200
1–40	0.09	3	1–1,500
1–40	0.06	3	1–2,000
1–40	0.03	3	1–4.000
1–40	0.015	3	1–8,000

Where dilutions greater than 1–1000 are made, a serum diluted 1–80 may be used to better advantage, unless the pipette employed is very finely graduated. In this case the amount of diluted serum for a certain dilution must be double that indicated in the table.

The mixture thus prepared is placed in an incubator at 37° C. for 24–30 hours. A temperature higher than 37° C. interferes with the agglutination.

The reaction consists of a layer of the agglutinated bacteria covering the entire convexity at the bottom of the tube. This film-like sediment may become so dense that it rolls in at the periphery. The supernatant fluid becomes clear in the lower dilutions, but in the higher ones the clarification may not be complete, showing that all the bacteria have not become agglutinated. This is further evidenced by the fact that the layer is less dense in the higher dilutions. The reaction may begin in six hours, but cannot be considered complete until 24 to 36 hours have elapsed. If no reaction appears in 24 hours it cannot be considered negative, as it may occur in from 30 to 40 hours after setting. Often, however, a reaction appears in less than 24 hours.

After the agglutination is completed, further standing produced no visible change in the test fluid.

A negative result shows a small round concentrated spot of sediment in the center of the convexity at the bottom of the tube, the test fluid remaining apparently unchanged even after several weeks. Animals whose blood serum agglutinates in dilutions of 1–500 are

suspicious and a reaction in dilutions of 1–800 or higher indicates an infection with *Bact. mallei.*

As pointed out by Bonome and confirmed by Taylor, there seems to be little or no change produced in the precipitating power of the serum of the blood taken before, during or after the mallein reaction but the agglutinating power as determined microscopically is very much increased during the mallein reaction.

This is shown by the appended table:

TABLE SHOWING BOTH MACROSCOPICALLY AND MICROSCOPICALLY THE AGGLUTINATION OF DEAD GLANDERS BACTERIA WITH BLOOD SERUM FROM HORSES TAKEN BEFORE, DURING AND AFTER THE MALLEIN REACTION.

No.	Blood taken the day previous to malleination Feb. 21		Blood taken during the reaction Feb. 22		Blood taken after the temperature had returned to normal Mar. 2	
	Macroscopic	Microscopic	Macroscopic	Microscopic	Macroscopic	Microscopic
1	1–800	1–1000	1–800	1–1800	1–800	1–1000
4	1–800	1–1200	1–1000	1–2000	1–1000	1–1500
5	1–1500	1–1600	1–1500	1–2500	1–1000	1–1200
6	1–1200	1–1500	1–1200	1–2000	1–1800	1–1800
7	1–1000	1–1200	1–1000	1–1800	1–1000	1–1400
8	1–500	1–500	1–800	1–1600	1–500	1–600
9	1–800	1–1000	1–800	1–1400	1–500	1–800
10	1–500	1–600	1–500	1–1000	1–500	1–800
12	1–200	1–300	1–500	1–800	1–500	1–750
13	1–1000	1–1200	1–500	1–1000	1–800	1–1000
14	1–500	1–600	1–500	1–800	1–500	1–800
15	1–1200	1–1400	1–1200	1–2500	1–1200	1–1800
16	1–1000	1–1200	1–1000	1–2400	1–1000	1–2200
17	1–500	1–800	1–800	1–1500	1–800	1–1400
18	1–1000	1–1400	1–1000	1–1800	1–800	1–1500
19	1–1000	1–1000	1–800	1–1200	1–1000	1–1200
20	1–500	1–600	1–500	1–800	1–500	1–800
21	1–500	1–750	1–500	1–1000	1–800	1–1200
22	1–1000	1–1400	1–1000	1–2500	1–800	1–2200
23	1–200	1–200	1–200	1–1000	1–800	1–1000
24	1–1000	1–1200	1–1000	1–2200	1–1200	1–2000
24	1–1000	1–1200	1–1000	1–2200	1–1200	1–2000
25	1–800	1–800	1–500	1–1200	1–800	1–1000
26	1–1200	1–1500	1–1000	1–2200	1–1200	1–2000
27	1–500	1–1000	1–1000	1–1800	1–800	1–2000
28	1–500	1–600	1–500	1–1200	1–1000	1–1500
29	1–800	1–1000	1–500	1–1200	1–1200	1–2000
30	1–1000	1–1200	1–800	1–1800	1–800	1–1600
31	1–500	1–600	1–800	1–1400	1–1000	1–1500

The agglutination in higher dilutions with the living organisms as determined microscopically was pointed out by Taylor. A comparison of the agglutination of the living and killed bacteria with the serum from glandered horses, as shown by the mallein reaction, is given in the appended table:

MACROSCOPIC AND MICROSCOPIC AGGLUTINATION OF BACTERIUM MALLEI WITH HORSE SERUM BY THE USE OF KILLED AND LIVING CULTURES.

Number of Horse	Macroscopic. Dead bacteria, 24 hours at 37° C.	Microscopic. Dead bacteria, 12 hours at 37° C.	Microscopic. Live bacteria, 12 hours at 37° C.
1	1–8000	1–12000	1–30000
2	1–2000	1–3000	1–12000
3	1–800	1–1000	1–10000
4	1–1600	1–1800	1–6000
5	1–500	1–600	1–5000
6	1–1000	1–1250	1–25000
7	1–800	1–1000	1–8000
8	1–800	1–1200	1–12000
9	1–1600	1–1800	1–24000
10	1–500	1–800	1–7500

The method as pointed out by Schütz and Miessner is a macroscopic one. It depends upon the precipitation of the agglutinated masses of bacteria. Normal horse's serum agglutinates glanders organisms in high dilutions as determined microscopically. This, however, does not appear to be of diagnostic value.

Complement fixation. This is strictly a laboratory method and cannot be applied in the field. It requires the collecting of the blood from the suspected animals and sending it to the laboratory as quickly as possible. The method is fully described by Mohler and Eichhorn in Bulletin 136 of the Bureau of Animal Industry. Schütz and Schubert, after applying this method and comparing it with others, recommend it as the most accurate means for diagnosing glanders. It is widely used in this country usually in conjunction with one or more of the other tests, especially the ophthalmic use of mallein.

The test requires five different substances: (1) washed red blood corpuscles of a sheep, (2) hemolytic amboceptor, (3) complement, (4) antigen (bacterial extract), (5) serum from the blood of the suspected animal to be tested.

Red blood cells. The washed red blood corpuscles of a sheep are obtained by bleeding a vigorous sheep from the jugular vein under antiseptic precautions. The blood is preferably collected in a sterile flask containing a few glass beads. The blood is shaken, defibrinated and filtered through sterile gauze into a glass tube. The tube is then filled

with physiological salt solution and the mixture centrifuged*. The supernatant fluid is poured off and the process repeated until *all* the serum is removed from the corpuscles. Enough salt solution is added to the sedimented corpuscles to make the volume of blood equal the original amount filtered into the tube.

Hemolytic amboceptor. The hemolytic amboceptor is obtained from the serum of a rabbit which has been immunized to the washed red blood corpuscles of a sheep. A strong vigorous rabbit is selected and injected with the washed red corpuscles. The injections are preferably made intraperitoneally and at intervals of 4–5 days. Two cc., 4 cc., 8 cc., and 12 cc. of the corpuscles are respectively used for each injection. In seven or eight days after the last injection a small amount of blood is taken from the rabbit (by bleeding from an ear vein) and the serum titrated to determine whether its hemolytic action is sufficient; that is, whether it will readily dissolve the hemoglobin from the corpuscles of a sheep. If it is found satisfactory the animal is bled from the carotid and the serum collected and stored in small bottles. It is preferable not to put more than 2 cc. in each bottle. The serum may be preserved by adding 0.5 per cent. of carbolic acid in a 5 per cent. dilution. If the carbolized serum is not used for three days after adding the carbolic acid it will not require inactivation (heating at 56° C. for ½ hour). Noguchi prefers adding 2 drops of chloroform to each 2 cc. of hemolytic serum and in this case in order to get the best results the serum should be inactivated before using. Serum preserved in this way can be kept in the ice-box for from three months to a year. It must, however, be titrated at intervals of a few weeks as the titre may change.

Titration of hemolytic rabbit serum. Dilutions of the hemolytic serum are made in eight test tubes, according to the following table. It is best to use pipettes to measure the required amounts.

TABLE No. I

DILUTIONS OF HEMOLYTIC AMBOCEPTOR (RABBIT SERUM).

Tube No.	NaCl Solution	Serum	Gives Dilution	Remarks
1	9 cc.	1 cc.	1–10	This is a 1–10 basic dilution.
2	9 cc.	1 cc. of 1–10 dilution	1–100	
3	8 cc.	2 cc. of 1-100 dil.	1–500	
4	9 cc.	1 cc. of 1–100 dil.	1–1000	
5	1 cc.	2 cc. of 1–1000 dil.	1- 1500	
6	1 cc.	1 cc. of 1–1000 dil.	1–2000	
7	3 cc.	1 cc. of 1–1000 dil.	1–4000	
8	8 cc.	2 cc. of 1–1000 dil.	1–5000	

*For hemolytic work 0.85 per cent. (Ehrlich) to 0.9 per cent. (Madsen) salt solution is universally employed. We prefer the latter concentration.

The titration proper is then made in the following manner: Eight additional test tubes are each filled with 2.5 cc. of salt solution to which is then added the hemolytic serum (amboceptor) in quantities of 1 cc. of the different dilutions (Table No. I) to each tube. Afterwards the complement of the guinea pig serum is added in quantities of 0.5 cc. of a 10% dilution to each tube, and finally 1 cc. of a five per cent. suspension of washed sheep corpuscles in salt solution is placed in each tube.

Besides these eight tubes there are also three control tubes, one to show that the complement alone will not produce hemolysis (without the amboceptor), the second that the amboceptor alone without the complement will not produce hemolysis, and the third that the salt solution alone will not produce hemolysis. Thus in the first control tube we add 3.5 cc. of salt solution, 0.5 cc. of complement, and 1 cc. suspension of sheep corpuscles. In the second control tube we add 3 cc. salt solution, 1 cc. of the 1–100 dilution of the amboceptor and 1 cc. suspension of sheep corpuscles. In the third control tube we add 4 cc. salt solution and 1 cc. sheep corpuscles. A test tube rack with two rows of holes is very convenient for holding the tubes for these tests.

TABLE No. II
TITRATION OF RABBIT SERUM (HEMOLYTIC AMBOCEPTOR).

Tube No.	(a) NaCl solution	Amboceptor	(b) Complement	(c) Blood corpuscles	Remarks
1	2.5 cc.	1 cc. of 1–10 dil.	0.5 cc.	1 cc.	
2	2.5 cc.	1 cc. of 1–100 dil.	0.5 cc.	1 cc.	
3	2.5 cc.	1 cc. of 1–500 dil.	0.5 cc.	1 cc.	
4	2.5 cc.	1 cc. of 1–1000 dil.	0.5 cc.	1 cc.	
5	2.5 cc.	1 cc. of 1–1500 dil.	0.5 cc.	1 cc.	
6	2.5 cc.	1 cc. of 1-2000 dil.	0.5 cc.	1 cc.	
7	2.5 cc.	1 cc. of 1–4000 dil.	0.5 cc.	1 cc.	
8	2.5 cc.	1 cc. of 1-5000 dil.	0.5 cc.	1 cc.	
9	3.5 cc.	controls	0.5 cc.	1 cc.	Complement control (no hemolysis should occur)
10	3.0 cc.	1 cc. of 1–100 dil.		1 cc.	Amboceptor control (no hemolysis should occur)
11	4.0 cc.			1 cc.	Salt solution control (no hemolysis should occur)

a. 0.9% NaCl solution.
b. 0.5 cc. of a 10% solution of the complement.
c. 5% suspension of washed sheep corpuscles in salt solution.

As can be seen from Table II the final volume in each tube is always uniformly 5 cc. Thus the different amounts of the blood derivatives used are always made up to 5 cc. by adding salt solution.

After adding the substances to the test tubes in the order given, the tubes are well shaken, placed in the test tube rack, and put in an incubator at 37° C. for two hours. Then the tubes are removed from the incubator and the results read.*

The highest dilution in which complete hemolysis has taken place represents the titre of the hemolytic amboceptor. Thus if complete hemolysis has taken place up to and including the tube where the dilution of the rabbit serum was 1–2000 (Tube No. 6, Table II), the hemolytic titre of this serum is represented by 1–2000. This dilution, however, is not used in the glanders test; but rather *its double strength*, which would be 1–1000. The titre of the hemolytic amboceptor for use in the diagnosis of glanders should not be less than 1–1000 and therefore if the rabbit serum should prove to be of a lower titre it should be discarded. It often happens that some rabbits are found to be unsuited for the production of amboceptor and the titre of their serum can not be raised high enough even after repeated inoculations with washed sheep corpuscles.

It is advisable to preserve the hemolytic amboceptor in small vials containing 1 to 2 cc. of the rabbit serum, and seal the corks with paraffin or sealing wax.

Complement. The complement which is contained in the blood serum of a normal guinea pig is employed. The complement should be titrated to determine its efficiency to act with the hemolytic amboceptor.

Titration of complement. By titration of the complement there is aimed to be established a complement unit which is the smallest quantity of complement necessary

TABLE No. III

TITRATION OF COMPLEMENT.

Tube No.	NaCl solution (a)	Complement from 1–10 dilution (b)	Ambocep-tor (c)	Blood corpuscles (d)
1	2.5 cc.	0.5 cc.	1 cc.	1 cc.
2	2.6 cc.	0.4 cc.	1 cc.	1 cc.
3	2.7 cc.	0.3 cc.	1 cc.	1 cc.
4	2.8 cc.	0.2 cc.	1 cc.	1 cc.
5	2.9 cc.	0.1 cc.	1 cc.	1 cc.
6	3.5 cc.	controls 0.5 cc.		1 cc. complement control (no hemolysis should occur)
7	3.0 cc.		1 cc.	1 cc. amboceptor control (no hemolysis should occur)
8	4.0 cc.			1 cc. salt solution control (no hemolysis should occur)

a. 0.9% NaCl solution.
b. Guinea pig serum in diminishing quantities.
c. Of previously titrated hemolytic serum, double dissolving quantity.
d. 5% suspension of washed sheep blood corpuscles in salt solution.

*If the tubes are placed in a water bath kept at 37° C. the time here may be shortened to one-half hour.

to produce complete hemolysis in the presence of one amboceptor unit and a suspension of sheep corpuscles.* The serum from each guinea pig should be titrated in the following manner before it is used. A basic dilution of 1–10 is made of the complement by adding 0.3 cc. of the complement to 2.7 cc. of salt solution in a test tube.

From this dilution certain definite amounts are added to each of five other tubes according to Table III. To the complement thus distributed to each tube, is added 1 cc. of hemolytic amboceptor of which the titre has already been determined† and likewise 1 cc. of a 5% solution of sheep blood corpuscles. The amount of fluid in each tube is made up to 5 cc. by the addition of salt solution. Three controls are used. The first control tube contains 3.5 cc. salt solution, 0.5 cc. of the 10% basic dilution of complement and 1 cc. of a suspension of sheep corpuscles. In the second control 3 cc. salt solution, 1 cc. of the amboceptor dilution and 1 cc. of the suspension of corpuscles; in the third control 4 cc. of salt solution and 1 cc. of the suspension of sheep's corpuscles are used. The order of adding each product and the amount of each are given in Table III.

After shaking each tube, they are placed in a rack and then in an incubator at 37° C. for two hours.‡ After the expiration of this time they are removed and the results noted. The highest dilution of complement in the tube in which the hemolysis is complete indicates the titre of the complement. For example, if hemolysis is complete in the tube where 0.3 cc. of the 10% basic dilution of the complement was used (Tube 3, Table III) and the hemolysis is incomplete in the tube in which 0.2 cc. of the same dilution was employed (Tube 4, Table III) then the titre of the complement is 0.3 cc., inasmuch as a 10% basic dilution of the complement was employed. Thus, in the tests using this complement it would be necessary to employ a 3% complement dilution, that is, 3 cc. of the serum of the guinea pig in 97 cc. of salt solution.

The amboceptor used in this titration should be inactivated when fresh, or when preserved with chloroform. The carbolized amboceptor may be used without inactivation after it is 3 days old.

Antigen. In testing for glanders this consists of an extract of *Bact. mallei* prepared as follows. Cultures are made on acid glycerin agar and allowed to incubate for 48–72 hours. The growth is then washed off with salt solution. This salt solution suspension of the organisms is kept at 60° C. for four hours in order to kill the bacteria. The suspension is then placed in a shaking machine and shook at frequent intervals for four days. It is then centrifuged at high speed (3000–5000 revolutions per minute), the clear supernatant liquid drawn off and ten per cent. of a 5% solution of carbolic acid added. Before use the antigen must be titrated to obtain its anti-complementary action. It would be well also to determine its antigenic and hemolytic properties as well. However, this may be left until the test proper.

Titration of the extract or antigen. The titration of the extract is carried out in order to determine the quantity of the extract which no longer prevents hemolysis

---

*The serum is obtained by bleeding a strong vigorous guinea pig by cutting the throat. The pig is first anesthetized and the ventral portions of the neck shaved and disinfected. The blood is collected preferably in a Petri dish.

†That is, if the titre was found to be 1–3000 by the previous titration you would add 1 cc. of a 1–1500 dilution, as double the quantity of amboceptor is added which has been stated before.

‡Here again the time may be shortened to one-half hour by the use of a water bath at 37° C.

(the anti-complementary action). A 1-10 dilution of the extract is first made by adding 1 cc. of the extract to 9 cc. of salt solution. From this 1-10 dilution other dilutions are prepared as follows:

TABLE No. IV

DILUTIONS OF THE ANTIGEN.

Tube No.	NaCl solution	Amount of Antigen	Give a dilution	
1	2 cc.	1 cc. of 1-10 dil.	1-30	
2	4 cc.	1 cc. of -110 dil.	1-50	
3	5 cc.	1 cc. of 1-10 dil.	1-60	
4	7 cc.	1 cc. of 1-10 dil.	1-80	
5	9 cc.	1 cc. of 1-10 dil.	1-100	
6	1 cc.	1 cc. of 1-100 dil.	1-200	
7	1½ cc.	1 cc. of 1-100 dil.	1-250	
8	2 cc.	1 cc. of 1-100 dil.	1-300	
9	3 cc.	1 cc. of 1-100 dil.	1- 400	
10	4 cc.	1 cc. of 1-100 dil.	1-500	

The titration proper is carried out as follows. Eleven test tubes and three for controls are used. To each tube 1 cc. of salt solution is added, then 1 cc. of the complement of the previously determined smallest quantity established by the titration done before. To each tube add 1 cc. of the different dilutions (1-10, 1-30, 1-50, etc.) of the extract as obtained in Table IV. The tubes are shaken, placed in a rack and the rack placed in an incubator at 37° C. for one hour.* The tubes are then removed and 1 cc. of a double quantity of the previously titrated amboceptor is added to each tube, and finally 1 cc. of a 5% suspension of washed sheep blood corpuscles. Three control tubes are used. The first serves for a control to show that the complement alone (without the amboceptor) does not produce hemolysis, the second to show that the amboceptor alone does not produce hemolysis and the third that the salt alone does not produce hemolysis. In the first control tube 3 cc. of salt solution, 1 cc. of complement and 1 cc. of blood corpuscles are added. The second tube contains 3 cc. of salt solution, 1 cc. of amboceptor and 1 cc. of blood corpuscles, while the third contains 4 cc. of salt solution and 1 cc. of suspension of sheep blood corpuscles. The following table (Table V) gives a summary of the foregoing method.

Each tube is shaken and all the tubes placed in the incubator at 37° C. for two hours.† The results are then read. The tube in which hemolysis is no longer prevented represents the titre of the extract. In the glanders test, however, *one-half* that quantity is used. For example, if the results of the titration should be that the first tube

*May place in water bath at 37° C. for ½ hour.

†Or water bath at 37° C. for one hour.

which does not prevent hemolysis contains a dilution of 1–80 (tube 5, Table V), then a *dilution* of 1–160 of the extract is used for the glanders test. Antigen which possesses too much anti-complementary substance should not be used; that is, where hemolysis is prevented in as high dilutions as 1–300, 1–400, 1–500, etc.

Suspected serum. The suspected glandered horse is bled usually through the jugular and about 20 cc. of the blood is collected in a small bottle. The blood is then allowed to coagulate and the serum to collect. If the blood came from an animal affected with glanders, the serum contains the specific glanders bacteriolytic amboceptor or immune bodies. The amount of these immune bodies in the blood of glandered horses is not

TABLE No. V

TITRATION OF GLANDERS BACTERIA EXTRACT.

Tube No.	NaCl solution (a)	Complement (b)	Extract (c)	Amboceptor (d)	Blood corpuscles (e)	
1	1 cc.	1 cc.	1 cc. of 1–10	1 cc.	1 cc.	
2	1 cc.	1 cc.	1 cc. of 1–30	1 cc.	1 cc.	
3	1 cc.	1 cc.	1 cc. of 1–50	1 cc.	1 cc.	
4	1 cc.	1 cc.	1 cc. of 1–60	1 cc.	1 cc.	
5	1 cc.	1 cc.	1 cc. of 1–80	1 cc.	1 cc.	
6	1 cc.	1 cc.	1 cc. of 1–100	1 cc.	1 cc.	
7	1 cc.	1 cc.	1 cc. of 1–200	1 cc.	1 cc.	
8	1 cc.	1 cc.	1 cc. of 1–250	1 cc.	1 cc.	
9	1 cc.	1 cc.	1 cc. of 1–300	1 cc.	1 cc.	
10	1 cc.	1 cc.	1 cc. of 1–400	1 cc.	1 cc.	
11	1 cc.	1 cc.	1 cc. of 1–500	1 cc.	1 cc.	
12	3 cc.	1 cc.	controls		1 cc.	Comp. control (no hemolysis should occur)
13	3 cc.			1 cc.	1 cc.	Ambocep. control (no hemolysis should occur)
14	4 cc.				1 cc.	Salt solu. control (no hemolysis should occur)

a.   NaCl solution.
b.   The determined smallest quantity established according to the primary test.
c.   Dilutions made according to Table IV.
d.   Double the quantity previously determined by titration.
e.   5% suspension of washed sheep blood corpuscles.

uniform and probably depends to some extent on the degree of infection present, therefore it is advisable to use in the test such quantities of the serum as will prove sufficient for the reaction to take place.

TABLE No. VI

THE FINAL TEST FOR GLANDERS.

Tube No.	NaCl solution 1	Suspected horse serum 2	Glanders bacteria extract 3	Complement 4	Amboceptor 5	Blood corpuscles 6	Remarks
1	cc. 1	cc. 0.1	cc. 1	cc. 1*	cc. 1	cc. 1	Test tube for the dose 0.1 cc. of suspected serum
2	2	0.1	......	1	1	1	Serum control for the dose 0.1 cc. suspected serum
3	1	0.2	1	1	1	1	Test tube for the dose 0.2 cc. of of suspected serum
4	2	0.2	......	1	1	1	Serum control for the dose 0.2 cc. suspected serum
5	1	......	1	1	1	1	Control for the quantity of extract used (hemolysis)
6	......	......	2	1	1	1	Control for the double quantity of ext. for gr. accuracy (hemo.)
7	2	......	......	1	1	1	Control of the hemolytic system (hemolysis)
8	3	......	......	1	......	1	Control of the complement (no hemolysis)
9	3	......	......	......	1	1	Control of amboceptor (no hemolysis)
10	4	......	......	......	......	1	Control of salt solution (no hemolysis)

1. 0.9 per cent. NaCl solution.
2. Suspected horse serum to be inactivated for 30 minutes at 56°-57° C. in water bath, in order to destroy the complement which is present in the serum of every horse.
3. One-half of the quantity which does not prevent hemolysis and established by titration (See Table V).
4. The determined smallest quantity established according to the preliminary test (See Table III).
5. Double the quantity previously determined by titration (See Table II).
6. 5% suspension of washed sheep blood corpuscles.

The necessary quantity has been established by Schütz and Schubert as 0.2 and 0.1 cc. placed in two different tubes. In some instances the tube containing 0.2 cc. may show a fixation of the complement, while the tube containing 0.1 cc. of the same serum may show only a partial fixation or hemolysis. In the majority of cases, however, the fixation is usually manifested in both tubes.

Method of performing test. Four tubes are employed for testing the serum of each animal. Two tubes are used for the test proper and two for controls. The tubes are

*Place for 1 hour in incubator or one-half hour in water bath at 37° C.

numbered 1, 2, 3, and 4. One cc. of a physiological salt solution is added to tubes Nos. 1 and 3. Two cc. is placed in tubes 2 and 4. The serum of the suspected horse is then added. This serum has been previously rendered inactive, that is, the complement has been "inactivated" by heating for one-half hour at 56–57° C. One-tenth cc. of this inactivated serum is added to tubes 1 and 2. Two-tenths cc. is added to tubes 3 and 4. The antigen (glanders bacteria extract) is now added to tubes Nos. 1 and 3. One cc. of an established dilution is used. Tubes 2 and 4 are controls to see whether the suspected horse's serum will influence hemolysis.

To each tube is now added 1 cc. of a dilution of the complement (blood serum of normal guinea pigs). The proper dilution has been determined by titration as mentioned before.

A series of six controls should be made in connection with each series of tests carried out. One set of controls will do for a single day's testing as long as the same substances (antigen, complement, amboceptor) are used in each test. It is also well to set what are called "positive" and "negative" controls in connection with each series of tests made. That is, the blood from a known glandered animal and likewise the blood from a healthy animal should be tested in connection with the blood from the suspected animals.

The six controls mentioned above are made according to Table VI beginning with tube No. 5.

Each tube is shaken carefully and placed in an incubator for 1 hour (or water bath at 37° C. for ½ hour). This is done in order to allow the union or fixing of the complement which will become locked up with the antigen and the bacteriolytic amboceptor in case the suspected serum came from a glandered animal. If the bacteriolytic amboceptor is not present or the suspected serum was from a healthy animal the complement will not become locked up or fixed.

After the required incubation period the tubes are removed from the incubator and to each tube is added 1 cc. of the previously titrated rabbit serum (hemolytic amboceptor). This serum has previously been inactivated by heating to 56°–57° C. for one-half hour, provided it has not been carbolized and kept for three days. Finally 1 cc. of a 5% suspension of the washed red corpuscles of a sheep is added to each tube. The tubes are now replaced in the incubator and left for 10 hours, when the results may be read. If put in a water bath at 37° C. and left for 1 hour and then removed and kept at room temperature for from 3–5 hours the results may be read. Some workers prefer reading the results as soon as removed from the water bath. If the horse was affected with glanders, that is, the serum of the animal contained bacteriolytic amboceptor, no hemolysis will have taken place in tubes 1 and 3. The red corpuscles will have settled to the bottom and the upper liquid will be clear. The controls Nos. 2 and 4 should show complete hemolysis, that is, the fluid in the tubes should be uniformly red. If, however, the suspected serum came from a healthy horse and did not contain the bacteriolytic amboceptor, hemolysis should take place in tubes Nos. 1 and 2. Mohler gives the following advice in the interpretation of the results of the test:

"Horses in which the serum produces a complete fixation of the complement in the quantities of 0.1 cc. and 0.2 cc. should be considered as glandered.

"Horses in which the serum gives a complete fixation in the quantity of 0.2 cc. and an incomplete fixation in the quantity of 0.1 cc. should likewise be considered glandered.

"Horses in which the serum produces an incomplete fixation of the complement in the quantities of 0.1 cc. and 0.2 cc. should also be considered as glandered.

"Horses in which the serum shows no fixation of the complement in either tube should be considered free of glanders."

In order to reduce the possibility of error to a minimum the agglutination test may be applied to the latter cases, and if this shows a value of 1 to 1,000 or over, the animal should be considered as glandered. However, such cases are extremely rare.

Conglutination. Recently this test has been introduced by Pfeiler and Weber for the diagnosis of glanders. They claim that it has certain advantages over the complement fixation test especially in that mule serum does not have the same highly anti-complementary action as it does in the fixation of the complement. It is based on the phenomenon first described by Ehrlich and Sachs in 1902 that if one brings together the washed red corpuscles of a guinea pig, fresh horse serum, and inactive (heated to 56° C. for ½ hour) cow serum, hemolysis results. Bordet and Gay in 1906 showed that this action was also attended by a very vigorous agglutination of the red corpuscles and the substance in the cow serum which was responsible for the agglutination was named by Bordet and Strenge "Konagglutinin." It is purely a laboratory test and cannot be applied in the field.

Glanders is to be differentiated from a variety of nasal and lymphatic disorders more or less common in the horse kind. Before the discovery of the specific bacterium of glanders and the specific tests, it was necessary to determine as closely as possible the differential anatomical characters between glanders and those of other affections, such as chronic nasal catarrh, strangles, lymphangitis, follicular ulceration of the nasal mucosa, cancer, sarcoma, actinomycosis, and the like. With the modern methods of diagnosis it is not necessary to attempt the often impossible differentiation between glanders and these lesions from the morbid changes alone.

Strong has described a disease in the Philippine Islands, which first appears in nodules, that resembles glanders very closely. It is caused by a blastomyces. It occasionally attacks cattle as well as horses.

Epizoötic lymphangitis is the disease most liable to be mistaken for farcy or skin glanders. It is caused by a yeast-like fungus (*Saccharomyces farciminosus*). This disease was discovered by Pearson in the State of Pennsylvania.

Edwards has described a disease in mules resembling glanders. It is characterized by lymphangitis, laryngitis and extensive ulceration, gangrenous pneumonia but no formation of nodules. The mules

did not react to mallein. A bacillus resembling *Bact. mallei* was isolated.

Tuberculosis and other lesions of the nares. Jœst has called attention to the difficulty in differentiating glanders from tuberculous ulcers* on the nasal septum and tuberculous nodules in the lungs, cicatricial scars on the nasal septum due to injuries, local amyloid tumor formation and hemorrhagic nodule-like lesions on the nasal mucosa and pressure ulcers in the larynx.

Parasitic nodules. In post mortem examinations, nodules are often found in the lungs, and occasionally in other organs, that are parasitic in nature but which resemble very closely those of glanders. Pathologists have long recognized parasitic nodules and their positive chemotactic action toward eosinophiles. There is a large literature on the differentiation of parasitic nodules from those of specific diseases such as glanders. Angeloff found that the "gray transparent nodules" in the lungs of horses were of parasitic nature and that the larva of a nematode usually *Sclerostoma bidentatum* could be found in the center of the nodules. Histologically the parasitic nodules may be recognized by the eosinophilic leucocytes which surround them. The studies of Moore and Fitch led to the conclusion that macroscopically it is difficult and often impossible to differentiate between the nodules due to parasites and those caused by *Bact. mallei* but that microscopically the lesions due to parasites are characterized by a variable eosinophilic infiltration. The existence of eosinophilia in parasitic lesions has been pointed out by Howard, Jœst and others.†

Prevention. The physical cases of glanders are practically all spreaders and should be promptly destroyed and their stables thoroughly disinfected including harness and watering bucket. The exposed animals should be tested and those that respond should be destroyed. On this point, however, there is a difference of opinion. Some authorities have affirmed that if the reacting animals are

---

*In this country tuberculosis in horses is very rare but in Denmark it is quite common. We have had one case of tuberculosis in the lung of a horse sent to the laboratory for diagnosis. It was sent in as a suspicious case of glanders.

†A few observers have noticed eosinophilia in old nodules of supposed glanders origin. The reports, however, on this subject are not sufficiently conclusive to exclude parasites. It is not supposed that all parasites give rise to eosinophilia. Moore, Haring and Cady pointed out (Proceedings A. V. M. A., 1904) that the blood of horses infested with *Sclerostoma bidentatum* exhibited eosinophilia. Because of the seriousness of infestion with this parasite, they suggested the desirability of a blood examination as a procedure in *examiniug horses* for soundness.

segregated, worked together in pairs, or singly and watered from individual buckets only, it is safe to keep and use them until physical symptoms develop when they should be promptly destroyed. This opinion is entertained because many horses appear eventually to recover that give a reaction to a specific test.

Glanders has undoubtedly been spread by reacting animals that have been brought into a community where they developed symptoms, became spreaders and transmitted the virus to healthy horses. Among the causes for the spread of glanders are the common watering trough*, the interchange of feeding bags and the retention of open cases of the disease. Great care should be taken to protect healthy horses against each and every channel of infection.

Immunization. Marxer found that heat sterilized virus has never given any satisfactory immunizing results. Experiments with glanders bacteria treated with 80% glycerine or 10% urea, first used by Levy, Blumenthal and Marxer, have been more satisfactory. The organisms treated by these substances give a product called "Farase."

According to Marxer it is prepared by shaking glanders bacteria in a concentration of 0.1 gm. of the bacteria to 4 cc. of a 10% urea solution for seventeen hours at 37° C. This process kills the organisms. "Farase" has been used with considerable success in the experimental immunization of cats, guinea pigs and horses against glanders. He tried "Farase" on horses that were exposed to glanders under natural conditions with promising results. Dediulin has used it with success.

A large Russian breeding establishment where upwards of 3000 horses were constantly kept and where during the harvest season 10,000 peasant horses were hired and stabled was selected in which to try the immunization experiment. During the year 1909, 276 horses had died of glanders.

Six hundred horses were selected and injected with "Farase." As a result of this work the following facts were noted:

One year and four months after the immunization none of the treated horses developed the disease, while during this time 14 new animals not treated with "Farase" which were kept with these died of glanders. In the meantime the treated horses were tested with mallein without obtaining any reactions.

---

*Dr. Luckey reports great success in the control of glanders in Missouri by eliminating the open watering fountains and providing hydrants where drivers can draw water for their horses in individual buckets. He states, "It is impossible to control glanders among horses which are watered out of a common trough or basin." Monthly Bulletin, Sept., 1914. Missouri State Board of Agriculture.

Specific biological treatment. A number of serums and vaccines have been tried but as yet they are unsatisfactory. A few workers have advocated the repeated injection of mallein as a remedy. The immunity established by an attack of glanders seems to be very transitory.

Control. The laws of each state and country prescribe the course to be followed when glandered horses are encountered. It is a reportable disease and practitioners should conform to the law and regulations regarding it. With glanders, as with other infectious diseases, it is not possible to state what consideration shall be given the occult cases. The disease is governed by definite laws of nature and not until these are accurately interpreted can the best procedure be formulated for its control. The more general practice is to slaughter the reacting animals and thoroughly disinfect the premises. The consensus of opinion seems to be that it is not safe to keep occult cases.

REFERENCES

1. ANGELOFF. Die grauen durch scheinenden Knötchen in den Pferdelungen und ihre Beziehung zu der Rotzkrankheit. *Arch. f. wiss u. prak. Tierch.*, Bd. XXXIV (1908), S. 41.

2. BABES. Observations sur la morve. *Arch. de Méd. expér. et d' Anat. path.*, Vol. III (1891), p. 619.

3. BERNS AND WAY. Practical Application and Results of the Agglutination Method of Diagnosing Glanders in One Hundred and Fifty-two Cases. *Amer. Vet. Rev.*, Vol. XXX (1906), p. 822.

4. BONOME. Ueber die Schwankungen des Agglutinin und Präzipitingehaltes des Blutes während der Rotzinfektion, *Centralbl. f. Bakt.*, Bd. XXXVIII (1905), S. 601.

5. BORDET AND STRENG. Des phénomènes d'absorption et la conagglutinine sérum de bœuf. *Centralbl. f. Bakt.*, Bd. XLIX (1909), S. 260.

6. BOURGET ET MÉRY. Sur le séradiagnostique de la morve. *La Semaine Med.*, 1898, p. 61.

7. BUTLER. Glanders. *Bulletin No. 16. Miss. Agr. Exp. Station*, 1891.

8. CARY. Glanders. *Bulletin No. 35. Ala. Agr. Expt. Station of the Agricultural and Mechanical College*, 1892.

9. DAWSON. Equine glanders and its eradication. *Bulletin No. 77. Florida Agric. Exp. Station*, 1905.

10. DE SCHWEINITZ AND KILBORNE. The use of mallein for the diagnosis of glanders in horses and experiments with an albumose extracted from cultures of bacillus mallei. *Am. Vet. Review*, Vol. XVI (1892), p. 439.

11. EDWARDS. A disease of mules simulating glanders. *Veterinary Journal*, Vol. LXIX (1915), p. 70.

12. FITCH. Glanders in man. *Cornell Veterinarian*, Vol. IV (1914), p. 86.

13. FRANCIS. Glanders, tests with mallein. *Bulletin No. 30. Texas Agri. Exp. Station*, 1894.

14. FROHNER. Klinische Untersuchungen über den diagnostischen Wert der Ophthalmoreaktion beim Rotz. *Monats. f. prak. Tierheilk.*, Bd. XXIII (1912), S. 1.

15. FROTHINGHAM. The diagnosis of glanders by the Strauss method. *Jour. of Medical Research*, Vol. VI (1901), p. 331.

16. GALLI-VALERIO. Contribution à l' étude de la morphologie du Bacillus mallei. *Centralbl. für Bakt.*, Bd. XXVI (1899), S. 177.

17. HIGGINS. Glanders and mallein. *Proceedings Amer. Vet. Med. Asso.*, 1904, p. 135.

18. HUNTING. Glanders. A clinical treatise. London, 1908.

19. JOEST. Zur Frage der lokalen Eosinophilie bei zooparasitären Organerkrankungen. *Deut. Tier. Woch.*, Jr., 17 (1909), S. 346.

20. JOEST. Uber einige rotzähnliche Erkrankungen des Respirations wege des Pferdes. *Zeits. f. Infektionsk. parasit. Krankheiten u. Hygiene*, Bd. XVI (1915), p. 238.

21. LANGER. Untersuchung über die differential diagnostische Bedeutung der Rotzagglutination u. s. w. *Monatshefte für prak. Tierheilkunde*, Bd. XVI (1905), S. 241.

22. LOEFFLER AND SCHÜTZ. The bacillus of glanders. *Deutsche Med. Wochenschrift*, Dec., 1882. Translated, Bd. CXV (1886), New Sydenham Society.

23. LORENZ. Versuche über den diagnostischen Wert der Ophthalmoreaktion beim Rotz. *Berl. Tier. Woch. Jahrg.*, 29 (1913), S. 252.

24. M'FADYEAN. The pulmonary lesions of glanders. *Jour. of Compar. Path. and Therap.*, Vol. VIII (1895), p. 50.

25. M'FADYEAN. Glanders. *Jour. Compar. Path. and Therap.*, Vol. XVII (1904), p. 295.

26. MARXER. Die aktive Immunisierung gegen Malleus. *Archiv. f. wissensch. u. prak. Tierheilk.*, Bd. XLI (1914–15), S. 272.

27. MOHLER AND EICHBORN. The diagnosis of glanders by complement fixation. *B. A. I. Bulletin No. 136*, 1911.

28. MOORE AND FITCH. The differentiation between nodules due to glanders and those caused by parasites. *Report New York State Veterinary College at Cornell University*, 1912–13, p. 115.

29. MOORE, TAYLOR AND GILTNER. The Agglutination Method for the Diagnosis of Glanders. *Amer. Veter, Rev.*, Vol. XXX (1906), p. 803.

30. NOCARD. The value of mallein as a means of diagnosis in doubtful cases of · glanders. *Jour. Compar. Path. and Therap.*, Vol. VIII (1895), p 227.

31. PFEILER AND WEBER. Vergleichende Untersuchungen der Sera von 100 Pferden mittels der agglutinations—, Komplement-ablenkungs-und Konglutinationsmethode zur Erkennung der Rotzkrankheit. *Zeits. f. Infekkrankh. der Haust.*, Bd. XII (1912), S. 397.

32. RABIEAUX. Serum diagnosis of glanders. *Abstract Jour. Compar. Path and Therap.*, Vol. XVI (1903), p. 59. Orig. *Jour. de Méd. Vét.*, 1902.

33. REYNOLDS. State control of glanders in Minnesota. *Jour. of Compar. Med. and Vet. Archives*, Vol. XX (1899).

34. ROBINS. A study of chronic glanders in man with report of a case. *Studies from the Royal Victoria Hospital, Montreal*, Vol. 2, No. 1.

35. RUTHERFORD. Glanders. *Proceedings Am. Vet. Med. Asso.*, 1906, p. 215.

36. SCHNÜRER. Die Diagnose der ansteckenden Tierkrankheiten mittels der neueren Immunitätsreaktionen. *Ninth International Tierärzt-Kongress im Haal.*, 1908.

37. SCHÜTZ UND SCHUBERT. Die Ermittelung der Rotzkrankheit mit Hilfe der Komplementablenkungsmethode. *Archiv. f. Wissensch. u. Prak. Tierheilkunde.*, Bd. XXXV (1909), S. 44.

38. SCHÜTZ. A contribution to the subject of glanders. *Jour. of Compar. Path. and Therap.*, Vol. XI (1898), p. 1.

39. SCHÜTZ. Zur Lehre vom Rotze. *Archiv. für wiss. u. prakt. Thierheilkunde,* Bd. XXIV (1898), S. 1.

40. SCHUTZ UND MIESSNER. Zur Serodiagnose der Rotzkrankheit. *Archiv. für wiss u. prakt. Tierhielkunde*, Bd. XXXI (1905), S. 353.

41. SMITH. On the influence of slight modifications of culture media on the growth of bacteria as illustrated by the glanders bacillus. *Journal of Comparative Medicine*, Vol. XI (1890), p. 158.

42. STRONG. Preliminary report of the appearance in the Philippine Islands of a disease clinically resembling glanders. 1902. *No. 1, Bureau of Government Laboratories*, Manila.

43. STRAUSS. Sur un moyen diagnostique rapide de la morve. *Arch. de Méd. expér. èt d'Anat. path.*, Vol. III (1889), p. 460.

44. VAN ES. Glanders. *Bulletin No. 85, N. Dak. Agric. Exp. Station*. 1909.

45. WAY. The practical application of the agglutination method for the diagnosis of glanders. *Am. Vet. Review*, Vol. XXXI (1907), p. 709.

46. WHERRY. Glanders: Its diagnosis and Prevention. *Bulletin No. 24. Bureau of Government Laboratories*, Manila. 1904.

47. WILLIAMS. Glanders. *Bul. No. 4. Mont. Agr. Exp. Sta.*, 1894.

48. WRIGHT. The histological lesions of acute glanders in man and of experimental glanders in the guinea pig. *Jour. of Exp., Med.*, Vol. I (1896), p. 577.

TUBERCULOSIS

Synonyms. Consumption; pearl disease; grapes; phthisis; scrofula; tabes; "The great white plague."

Characterization. Tuberculosis is an infectious disease of man and domesticated animals. Cattle and swine suffer most, but, under favorable conditions, all species including fish and amphibians are attacked. It is a disease of slow development, involving either primarily, or in association with other organs, the lymphatic system. It is characterized in the beginning by the formation of small, non-vascular nodules, or tubercles which have a tendency to central degeneration. It destroys life by a chronic and long continued systemic poisoning and by the destruction of tissue in organs necessary to life. It is caused by *Bacterium tuberculosis.*

History. Tuberculosis is one of the oldest diseases affecting cattle of which there are identifying records. It seems to have been known to the Jewish people during their Egyptian captivity and the ecclesiastical laws for many centuries contained numerous enactments against the consumption of flesh from tuberculous animals. In 1370, it was forbidden in Munich to have on sale the flesh of animals affected with tuberculosis. A number of other cities passed similar ordinances. In 1702, Florinus described the disease and emphasized the then existing opinion that it was identical with syphilis. This led to the practice of destroying all tuberculous animals. In 1783, the Berlin Board of Health rejected the theory of the connection of tuberculosis

and syphilis and declared the flesh of affected animals to be fit for food. This led finally to the changing of all laws throughout Prussia against the use for food of flesh from animals affected with the disease. Tscheulin, in 1816, recognized in reference to the infection of meat three degrees of bovine tuberculosis, viz.: (1), in which the tubercles were to be removed; (2), in which the diseased parts were to be destroyed and the meat sold at a low price; and (3), those cases in which the lesions were so extensive that the whole carcass must be rejected.

The study of the lesions themselves gave rise to a number of beliefs concerning their nature. Thus, Virchow, Schüppel and others declared that the tubercles in cattle were lympho-sarcomata. Leisering considered them simply as sarcomata. Spinola and Haubner maintained that human and bovine tuberculosis were identical.

In 1865, Villemin showed that tuberculosis was due to a specific infection. He produced the disease in rabbits by inoculating them with tuberculous material from human subjects. He also produced the disease by feeding experimental animals and by causing them to inhale tuberculous material. Chauveau, in the same year, produced the disease in cows. These results were soon confirmed by Klebs, Cohnheim and Gerlach. These experiments, in which the disease was produced in one species with tuberculous material from another, followed by the discovery by Koch of the specific bacterium of the disease, led to the view that tuberculosis in all species of mammals was identical. This generally accepted belief caused sanitarians to look upon tuberculosis in cattle as a great menace to public health. The result was that during the closing decade of the last century this disease in cattle was treated more vigorously as a menace to the human species than as a destructive disease of animals.

In 1896, Dr. Theobald Smith pointed out that for certain animals the tubercle bacteria from cattle were more virulent than those from man and further that there were certain morphological and cultural differences existing between them. In 1898, he published the results of a more extended series of investigations. Since that time a number of investigators have arrived at the same conclusion. The fact has come to be well known that certain differences exist between the bacteria of tuberculosis found in the human and in the bovine species. Koch's experiments reported at the tuberculosis congress in London in July, 1901, give additional evidence of a difference in virulence for experimental animals of the bacteria of human and of bovine

tuberculosis. To what extent man becomes infected from the bovine variety cannot be stated, but the accumulating evidence tends to the conclusion that bovine tuberculosis is of less significance in its influence upon public health than was formerly thought, and of more importance as a rapidly spreading and destructive disease among cattle. Concerning its transmission, the conclusion seems to be warranted, that the virus of tuberculosis spreads very largely among men and cattle from individual to individual of the same species rather than from one species to the other. Swine are often infected from cattle.

Geographical distribution. Tuberculosis is an exceedingly widespread disease. In earlier times it was quite prevalent among cattle in Central Europe. It seems to have existed in Western Asia and Northern Africa at an early date. From these centers it has spread to nearly every cattle raising country of the world. Its rapid spread during the last fifty years is attributed to the increase in cattle exchange resulting in the introduction of tuberculous animals into healthy herds. It is stated that in many countries, and in large districts within others, tuberculosis did not exist until it was introduced within recent years by the importation of diseased animals.

In countries where there has been little or no importation of cattle, and in which the native breeds still exist unchanged, as in many parts of Russia, Austria and Spain, in the northern part of Sweden and Norway, and in parts of Africa, tuberculosis is practically unknown. This is true of the cattle on the island of Jersey, where for more than a hundred years foreign cattle have not been introduced.

In the United States, the disease is very widely distributed. It is found to a considerable extent in certain localities where the climatic conditions seem to be beneficial for tuberculous people. The explanation for this seems to be that tuberculous animals have been introduced into these districts. There are, however, large areas in which it is practically unknown. The Western steers that are killed in the large slaughter houses are practically free from this disease except those that come from a few infected regions.

Etiology. Tuberculosis is caused by a rod-shaped organism known as *Bacterium tuberculosis*. It was discovered by Robert Koch in 1882. Schüller and Toussaint had previously studied growths which seem, from the results of their inoculation experiments, to have been due to this organism. The bacterium of tuberculosis is a slender, rod-
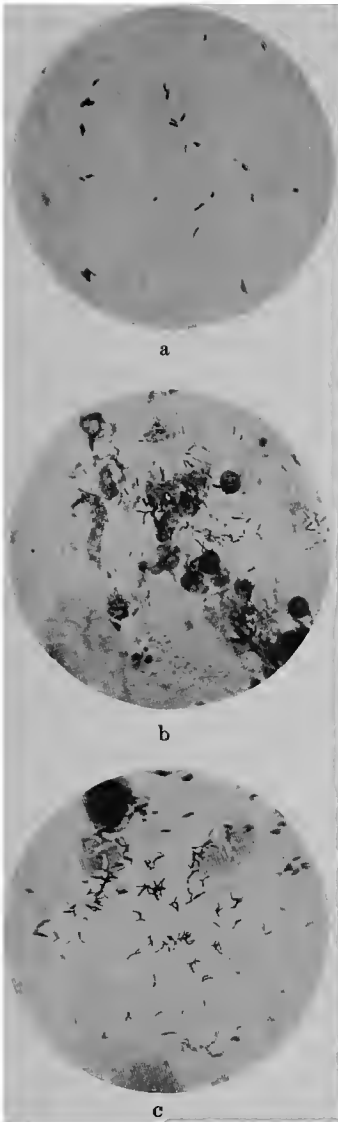
FIG. 25. TUBERCLE BACTERIA. (a) BOVINE VARIETY FROM A YOUNG CULTURE ON GLYCERIN AGAR. (b) BOVINE VARIETY FROM THE MOUTH OF A COW HAVING ADVANCED PULMONARY TUBERCULOSIS. (c) AVIAN TUBERCLE BACTERIA FROM A GLYCERIN AGAR CULTURE. (X ABOUT 1000.)

shaped organism with rounded ends, from 2 to 5 μ in length and from 0.3 to 0.5μ broad. The rods are straight or slightly curved, and occur singly, in pairs or in small bundles. They do not produce spores, but vacuoles are often observed and branching forms have been described.

The bacterium of tuberculosis is readily cultivated on artificial media such as blood serum, glycerinated agar and bouillon after it has been adapted to such artificial conditions.* It is, however, not easy to cultivate it directly from ordinary tuberculous lesions. Although at the time of their discovery, tubercle bacteria from man and from animals were believed to be identical, they have been found to possess slightly different characters and properties. Smith pointed out in 1898, that morphologically tubercle bacteria from cattle were shorter and thicker than those from man, that they grow slightly different on blood serum, and that they were much more virulent for cattle and rabbits than those from the human species. Since that time his conclusions have been confirmed by a number of investigators. Koch

obtained like results. At present, therefore, we must look upon the tubercle bacteria coming from these different species as possessing racial or varietal differences which perhaps are the result of different life conditions.   The investigations which have been made with the different forms of this organism found in tuberculosis of fowls and of fish have led a few experimenters to believe that they are simply varieties of the organism first described by Koch.   Further inquiries are necessary to fully satisfy bacteriologists that all of these forms are thus related to the one species.   There seems to be no reason for doubting that the bovine and human forms are varieties or races of the same species.   The difference in the conditions of life under which   they   exist   in   the bodies of   men   and of cattle is quite enough to explain resulting differences in the bacteria.   There seems to be a tendency for some workers on this subject to consider the varietal differences between the human and bovine to be of less significance than heretofore thought.   Malm found experimentally that the difference between the bovine and human tubercle bacteria is not constant.   He states that "there are human tubercle bacteria that are very virulent for cattle and rabbits.   There are also of the bovine type varieties that show a weak virulence for cattle, rabbits and guinea pigs."   He believes that no distinction should be made between human and bovine varieties.   Findlay and Martin found that the bovine type was more readily destroyed by daylight and drying than the human type.   They explain in part by this fact the more frequent aerial infection in man.

Symptoms.   The symptoms vary according to the course of the disease and the location of the lesions.   There is a chronic form, which is most common, and an acute form or miliary tuberculosis.

The symptoms of chronic tuberculosis depend upon the location and extent of the lesions.   When they are situated deeply and are not of great extent, they may not exhibit visible evidence of their presence.   In such cases, the infected animal may present the picture of perfect health and show no disturbance of function.   Indeed some animals, in which the lesions are both extensive and widely distributed and which have never presented noticeable signs of the disease, are slaughtered for beef without a suspicion of the presence of tuberculosis until they are examined post-mortem.

Since the lesions of tuberculosis vary so much in different cases, it is not possible to give a description of what can be designated the characteristic or even the usual symptoms of this disease.   In the

beginning they are largely referable to the organ affected. There are, however, certain general manifestations that appear in most of the advanced cases, such as emaciation while the appetite continues good. This is always a suspicious indication and especially if accompanied by cough, rough coat and tight, harsh skin. Rough or loud respiratory sounds are suspicious, and, in advanced cases, it is often found that the animal groans when pressure is brought to bear upon the chest wall. Many cases bloat habitually due to presence of enlarged glands upon the esophagus. Enlarged superficial lymph glands are suspicious but there are other causes for enlargement of these glands.

In tuberculosis of the lungs, it may be said that coughing is the most noticeable symptom. It is most common after feeding, drinking, or after rapid moving following a period of repose, but sometimes it occurs without any apparent cause. The cough is usually strong, dry and frequently of a high pitch. Sometimes it is very violent, accompanied by protrusion of the tongue. Auscultation re-

FIG. 26. RIGHT LATERAL ASPECT OF POSTERIOR HALF OF STEER'S HEAD. (a) LOWER JAW, (b) EAR PASSAGE, (c) HORN, (d) STYLOID PROCESS OF OCCIPITAL BONE, (e) PAROTID GLAND, (f) SUBMAXILLARY GLAND. A. RIGHT PAROTID LYMPH GLAND. B. RIGHT POST MAXILLARY LYMPH GLAND. C. RIGHT SUBMAXILLARY LYMPH GLAND. THESE GLANDS ARE OFTEN THE SEAT OF TUBERCULAR DEPOSITS (*Smith*).

veals modified and abnormal sounds of different kinds in the lungs; sibilant, sonorous and mucous rales are most common. A dull sound is often detected on percussion. It is also to be noted that this condition is of slow development and long duration, thus aiding one to distinguish it, in many cases, from bronchitis or pneumonia.

Where the mediastinal lymphatic glands are enlarged and press upon the esophagus the animal bloats more or less. Chronic or habitual bloating accompanied by a good appetite and no other evidence of disease of the digestive tract, especially if there is shortness of

breath and cough, may be looked upon as strongly indicative of tuberculosis with enlarged mediastinal lymphatic glands. Enlarged tubercular glands along the eosphagus may also press upon that organ causing obstruction and preventing the escape of gases from the stomach.

Sometimes large tuberculous masses develop on the pleura. In such cases the principal symptom is a friction sound that is heard most distinctly during inspiration. If the masses are large enough they give rise to a dull sound upon percussion. In tuberculosis of the stomach and intestines, digestion is interfered with. This gives rise to poor appetite, frequently to diarrhea and sometimes to alternation of diarrhea and constipation. In tuberculosis of the peritoneum or of the lining of the abdominal cavity, the lymphatic glands of the flank are often enlarged and hard. Sometimes this condition can be diagnosed positively by a rectal examination and the discovery of the hard, nodular masses. Tuberculosis of the liver does not give rise to symptoms unless the disease is far advanced.

In animals in which the post-pharyngeal lymphatic glands are enlarged, the breathing is harsh and noisy. In this condition there is sometimes difficulty in swallowing, and particles of chewed up food are occasionally expelled from the mouth, either voluntarily when it is found that they cannot be swallowed conveniently, or by the coughing they occasion upon reaching the pharynx. These enlarged glands may sometimes be detected by

FIG. 27. DORSAL ASPECT OF BOVINE LUNGS. (a–a^1) RIGHT AND LEFT CAUDAL LOBES, (b–b^1) R. AND L. VENTRAL LOBES, (c–c^1) FIRST AND SECOND RIGHT CEPHALIC LOBES, (c^2) LEFT CEPHALIC LOBE, (e) TRACHEA, (x–x) REGION MOST FREQUENTLY INVOLVED IN THE EARLIEST STAGES OF PULMONARY TUBERCULOSIS. THE LESIONS AT THIS STAGE ARE USUALLY EMBEDDED IN THE LUNG TISSUE (Smith).

palpation accomplished by placing one hand on each side of the throat above the larynx and then pressing from opposite sides.

Tuberculosis of the udder is detected by an enlargement and hardening of the affected part, usually by the absence of pain and the fact that the secretion is not altered until the part has been diseased for some time. In advanced cases, instead of milk, the udder secretes a yellowish, cloudy and sometimes flocculent liquid. In acute, rapidly developing cases, there may be pain and edema of the skin. In nearly all cases of udder tuberculosis the supramammary lymphatic glands, situated above the udder in the middle of the escutcheon, are enlarged and hard. If there is doubt as to the character of the disease of the udder, the milk, or possibly a piece of excised udder tissue, may be examined bacteriologically.

In tuberculosis of the brain, the animal is unsteady and uncertain in its movements. It lies down much of the time, is usually subject to occasional cramps and is apt to carry the head in an unusual position. Such cases are inclined to advance rapidly and terminate in death following coma or convulsions.

In tuberculous disease of the bones and joints, the parts are enlarged, there is loss of motion, pain and usually abscess formation followed by the discharge of thick yellow pus. In tuberculosis of the uterus or ovaries and sometimes in peritoneal tuberculosis of the cow, the subject is almost continually in heat. In tuberculosis of the uterus
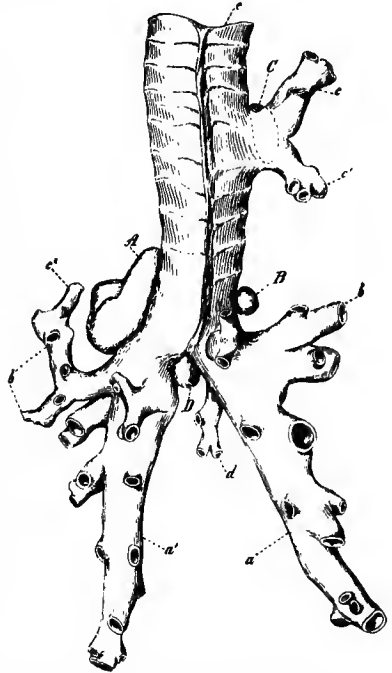
Fig. 28. Trachea and bronchial tubes of bovine lungs showing attached bronchial glands. (a–a¹) supply right and left caudal lobes, (b–b¹) supply R. and L. ventral lobes. (c–c¹) branches of the right supernumerary bronchus, (c²) supply left cephalic lobe, (d) branch to azygous lobe, (e) trachea. A. left bronchial lymph gland. B. right bronchial lymph gland. C. lymph gland base of supernumerary bronchus. D. gland often between bronchi. The glands a to d are often involved (*Smith*).

there is sometimes a discharge of thick, yellowish material mixed with mucus. In tuberculosis of the testicles the organs become enlarged and hard.

In all advanced cases, the nutrition of the animal is interfered with and, sooner or later, the "tuberculous cachexia" appears. It is, however, in many cases remarkable to note the extent of lesions in animals that are well nourished and present no external signs of disease. Animals killed in prime condition by the butcher are sometimes found to contain extensive and widely distributed lesions of tuberculosis. In generalized tuberculosis, many of the symptoms described above may occur simultaneously. The symptoms of acute miliary tuberculosis are rapid loss of flesh, depression, poor appetite, cough, weakness, rapid breathing, harsh respiratory sounds, some elevation in temperature, i n c r e a s e d pulse rate and, sometimes, enlarged lymphatic glands. The course of this form of tuberculosis is always rapid and terminates in death. Acute miliary tuberculosis occurs when large numbers of tubercle bacteria are discharged into the blood or lymph currents. They are then carried to other parts of the body, filtered out in the capillaries of the lungs, liver, spleen, kidneys and elsewhere, causing tubercular lesions in each of these localities. The lesion from which the infectious material entered the circulation may have been a comparatively small nodule. This form of the disease is more likely to appear in

Fig. 29. Dorsal aspect of bovine lungs showing position of the posterior mediastinal glands; (a, b, c, c′) caudal, ventral, cephalic lobes, (f) esophagus, (g) muscular pillars of diaphragm, (h) posterior aorta, (i) caudal margin of the ligament of the lung. A. left bronchial gland. Mediastinal glands are shown, most of them resting on the esophagus. The large caudal gland resting on the pillars of the diaphragm is most frequently diseased and often attains an enormous size. The remaining mediastinal glands are arranged in two sets on the right and left margins of the esophagus (Smith).

young animals than in adults, and is more common among swine than in cattle.

Morbid anatomy. The usual direct anatomical change following the invasion of tubercle bacteria is the formation of nodules or tubercles. A tubercle has been defined as "a small nonvascular nodule composed of cells varying in form and size with some basement substance between them and with an inherent tendency to undergo central necrosis." In a large number of cases the individual tubercles are distinct and easily recognizable, while in others they are coalesced, forming a mass of necrotic tissue. The lesions vary, therefore, from
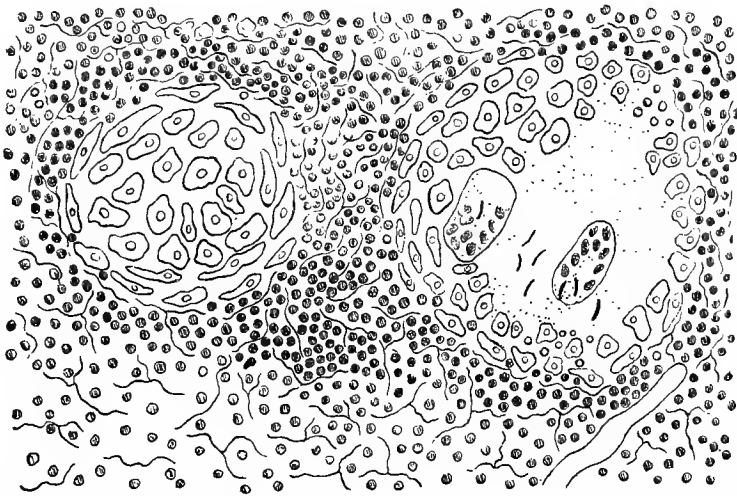
FIG. 30.   A DRAWING OF A SECTION OF VERY YOUNG TUBERCLES IN SPLEEN (*Thoma*).

well isolated minute or larger nodules to masses or cavities containing a purulent, caseous, or calcified substance.

The location of the primary lesion depends upon the channel of infection. If the specific organisms are lodged in the oral cavity or pharynx they may pass through the mucosa and be taken to some of the lymphatic glands about the head; if they are taken directly through the respiratory passages into the lungs they either develop nodules in the lung tissue proper, or they are carried through the lymphatic system to the lymph glands draining the lungs where the lesions first appear. If the specific bacteria are first lodged in the intestinal mucosa, primary tuberculous ulcers may develop or they may pass
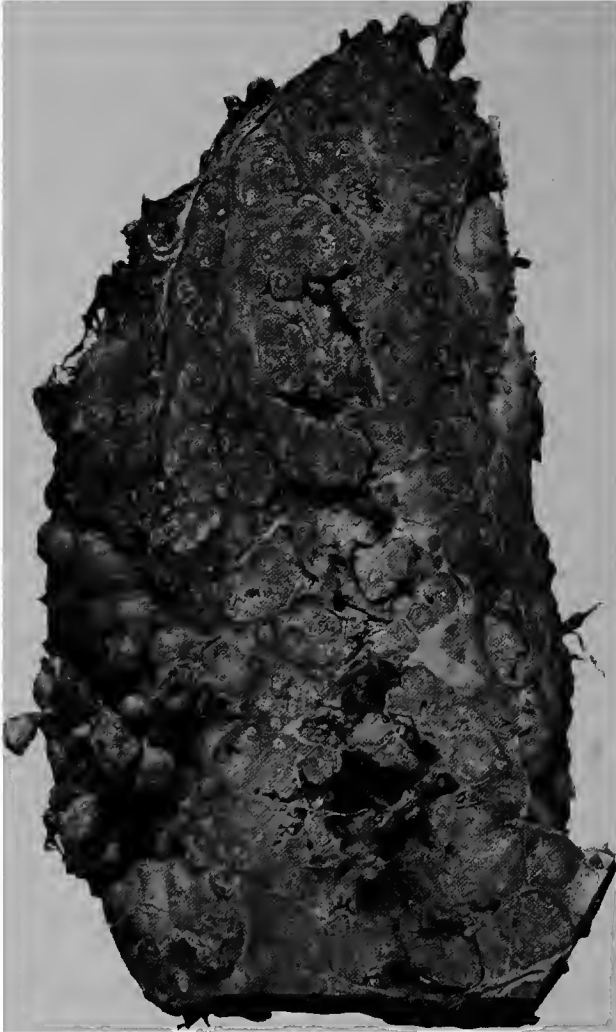
FIG. 31.   A PHOTOGRAPH OF A SECTION OF THE ANTERIOR LOBE OF A COW'S LUNG
ADVANCED IN TUBERCULOSIS.   THE ENTIRE LUNG TISSUE IS INVOLVED.   THE TUBER-
CULOUS MASSES ARE SURROUNDED IN SOME INSTANCES BY A QUITE THICK BAND OF
CONNECTIVE TISSUE.   A LARGE PART OF THE TISSUE IS CALCIFIED.   THIS IS SHOWN
BY THE LIGHT OR WHITISH POINTS.   (NATURAL SIZE.)

into the mesenteric lymphatics or the portal vein.   It may happen
that the bacteria may be carried by means of the lymph or blood

stream and lodged in any part of the body, such as the brain, kidneys, spleen, testes, ovaries, bones, joints, and subcutaneous and intermuscular glands and serous membranes. The evidence at hand, however, seems to show that in a large majority of cases the primary lesions are located in one of the following organs: (1) in the lungs or the lymphatic glands draining them; (2) in the lymphatic glands about the head; (3) in the mesenteric glands and intestines; (4) in the portal glands or liver substance itself; and (5) in the generative organs and udder.

It not infrequently happens that the apparent primary lesions occur on the pleura, peritoneum, meninges or synovial membranes while the organs remain free from disease. In such cases the lesions consist of many tubercles varying from one to ten or more millimeters in diameter or of bunches of closely set tubercles which are more or less flattened or irregular in shape, owing to their mutual pressure. Sometimes these tubercles are attached to the serous membrane by a small, tough, fibrous pedicle; frequently, however, this is absent and the nodules rest bodily upon the membrane.

The structure of the tubercle consists in the beginning of a few cells surrounding the invading specific organisms. These are soon encased by a zone of epithelioid cells and giant cells which is soon surrounded by an outer layer of round or lymphoid cells. The central portion becomes necrosed and as the nodule enlarges the central necrotic portion becomes correspondingly large.

The histological structure of the tubercle is typically illustrated in the beginning avian tubercle. In cattle there is a strong tendency for the necrotic tissue to become infiltrated with lime salts. In certain species a deposit of fibrous tissue in the outer zone of the tubercle has been observed. In the smaller and more susceptible experimental animals such as the guinea pig and rabbit and frequently in swine, the lesions are of a more diffuse nature infiltrating the interstitial tissue with the tuberculous mass and gradually encroaching upon the parenchyma. Circumscribed tubercles may also be present.

In secondary or generalized tuberculosis one or more of the organs, such as the omentum, serous membranes, or lymphatic system, may become more or less thickly sprinkled with minute grayish nodules about the size of a millet seed. These tubercles are at first almost the color of mother-of-pearl but later as the central caseous degeneration begins they become grayish. Giant cells are usually numerous.

FIG. 32.   A PHOTOGRAPH OF THE TUBERCULOUS PROMINENCES OR NODULES THAT HAVE
DEVELOPED ON THE PLEURA COVERING THE RIBS.   (NATURAL SIZE).

In studying the lesions in a fatal case of tuberculosis one may find
with varying modifications one or more of the following conditions:

The primary lesion may be found in any one of the organs or mem-
branes.   Its comparative age is determined by the character of the

anatomical changes. It may be entirely encysted, caseous or calcareous and dead. In addition to the primary focus, there may be a succession of tubercles of various ages distributed in one or more organs.

The lesions may be restricted to one organ, as the liver, in which the primary focus has spread by continuity due to its infiltrating nature until the destruction of the tissues of the organ has become so extensive that death results. Such cases do not seem to be common.
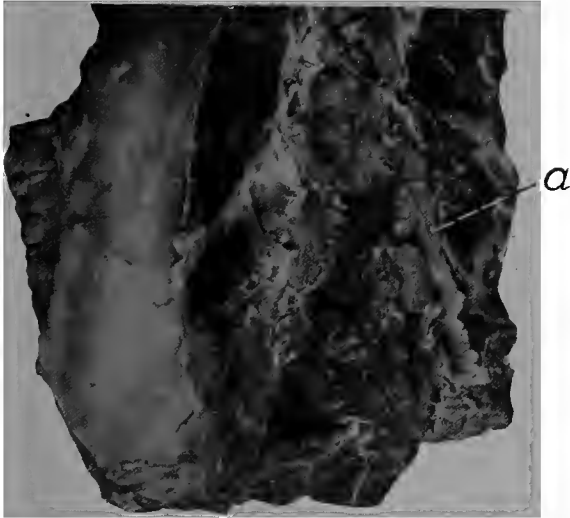
FIG. 33.   TUBERCLE DISCHARGING INTO BRONCHUS.   THIS SHOWS A SECTION THROUGH A BRONCHUS WHERE AT POINT (a) THE TUBERCULOUS TISSUE HAS EXTENDED INTO THE BRONCHUS MAKING IT POSSIBLE FOR THE TUBERCLE BACTERIA FROM THE TUBERCULOUS AREA TO PASS INTO THE BRONCHUS AND THROUGH IT TO THE MOUTH.   FROM THE MOUTH THEY ARE DISSEMINATED WITH THE DROOLINGS OR THEY ARE SWALLOWED AND APPEAR IN THE INTESTINAL CONTENTS.   (NATURAL SIZE).

The primary lesion may be well marked and accompanied by miliary tubercles sprinkled extensively throughout the organs and tissues of the entire body.

The lesions throughout the body may resemble each other very closely, so that difficulty may be experienced in determining the primary focus.

In the lungs, two distinct forms of lesions are observed. (1) The air cells may be infiltrated with the tuberculous mass spreading

directly from the primary focus. This may be purulent, caseous or calcareous. The color may be whitish, gray or of a yellowish tinge. (2) The lesions may consist of miliary tubercles. In later stages these nodules, more or less translucent, may become yellowish, caseated and calcareous in their centers. Large tubercular nodules are frequently formed by the massing of several of these minute tubercles.

Fig. 34. Tuberculosis of the mucous membrane of the trachea, cow.

When the lungs are primarily attacked the caudal (principal) lobes are most frequently involved. Smith considers the seeming predilection for the larger lobes to be due to mechanical conditions. The writer has found, however, that in certain herds which have been killed after the tuberculin test, the primary and only lung lesions were in the

ventral and cephalic lobes. It is important to note that usually the bronchial glands are also involved. When the pleuræ are affected the lesions consist of nodules varying in size from that of a millet seed to a large pea, sprinkled more or less thickly on one or both of the visceral or parietal surfaces. These form the "pearl disease," *Perl-sucht*, of the German and the "grape disease" of the English writers. If they become confluent, large masses are found. Jœst and Mar-janen found that in serous tuberculosis in cattle, there are produced non-specific inflammatory new formations which become infected with tubercle bacteria and result in the formation of the "pearl" nodules.

Tuberculosis of the thoracic glands is very common and usually accompanies lesions in the lungs; but the lungs may be healthy and the glands involved. (See figures for location of glands.) The primary lesions may be and often are found in the lymphatic glands about the head.

In rare cases the lesions are found in the mucous membrane of the trachea. In these cases the mucosa is often wrinkled. In some cases very small lesions are found discharging into a bronchus.

In the abdominal cavity the organs most frequently involved are the peritoneum, mesenteric lymph glands, portal lymph glands and liver. The kidneys, spleen, ovaries and uterus are more rarely the seat of tuberculous lesions. Ulcers in the intestine have not been common in the writer's observation. The ulcers in the cases observed have been isolated with elevated borders and a depressed center. Sections show that the tuberculous infiltration extends outward and to a certain extent undermines the mucosa. Tuberculosis of the testes is sometimes found. The udder becomes the seat of tuberculous deposits in a small percentage of cases. It is more often affected in cases of generalized tuberculosis. M'Fadyean finds udder tuberculosis in from 1 to 2 % of the cows; Poels 0.9%; Vallée and Villejean 5.3 to 6.5%; and Bergman 3.5%.

When the primary infection is restricted to a single focus the disease is said to be localized. When the specific bacteria are spread from the primary lesions through the agency of the lymph and blood streams, sprinkling other organs with the infecting bacteria, each of which becomes the starting point for the development of a new tubercle, the disease has become generalized.*

*The Federal meat inspection regulations state that animals affected with "extensive or generalized tuberculosis" are to be condemned.

FIG. 35.   A PHOTOGRAPH OF A PART OF THE OMENTUM SHOWING A LARGE NUMBER OF
SMALL MORE OR LESS FLATTENED TUBERCLES SCATTERED OVER THE SURFACE.
(SLIGHTLY REDUCED IN SIZE).

It was formerly considered that when the lesions existed in both
of the large (abdominal and thoracic) cavities of the body the disease
was generalized.   It is possible, however, for it to be generalized
when the lesions are restricted to the organs of one cavity, as the

secondary seeding with the bacteria that have escaped from a primary focus through the circulation may be restricted to the cavity in which the first lesions developed. It seems better, therefore, to accept Ostertag's views and classify local and generalized tuberculosis in accordance with the nature of the lesions rather than their distribution in the body.

The fact is worthy of consideration, that very often cattle killed after reacting to tuberculin do not show extensive distribution of lesions. Frequently animals are killed soon after infection has taken place, in which case the lesions are restricted to a single lymphatic gland or other organs. In other cases old lesions of considerable proportion are found.

Diagnosis. Tuberculosis is diagnosed by the symptoms, lesions, by finding the tubercle bacterium, and by the use of tuberculin. The sera tests thus far have not been satisfactory.

Lesions. The lesions in tuberculosis are usually sufficiently characteristic to enable a diagnosis to be made from the gross examination of the affected organs. There are cases, however, where they are atypical and a diagnosis cannot be made from their appearance.

Etiology. In many cases the specific bacterium may be found by staining films made from the lesions with the ordinary tubercle stain.*

FIG. 36. A PHOTOGRAPH OF A SHORT STRIP OF THE SMALL INTESTINE OF A COW SHOWING SEVERAL SMALL AND ONE LARGE TUBERCULOUS ULCERS (NATURAL SIZE).

In young lesions this is usually not difficult but in old ones it is often

A method for staining tubercle bacteria. Stain the preparation with fresh carbol

impossible. Sections of the tissues containing young lesions properly stained are often helpful. In case the tubercle bacteria are not found in smears or sections, guinea pigs should be inoculated.* If the inoculated guinea pigs are chloroformed in about 25 days young tubercles can usually be detected in the adjacent lymph glands from which cultures may be made. The tubercle bacteria are usually readily demonstrated by making film preparations from the lesions.

---

fuchsin. Place a few drops of the stain on the film side of the cover-glass preparation and hold it over a flame with forceps until steam is given off. Allow the hot stain to act for from 3 to 5 minutes, or the preparation may be floated on the carbol fuchsin in a watch glass without heat. In this case it is allowed to act for from 10 to 15 minutes. The preparation is then rinsed in water and decolorized by treating it with a 10% solution of nitric or sulphuric acid for from $\frac{1}{4}$ to 1 minute. It is again rinsed in water, when it is ready for examination. It can be dried and mounted permanently in balsam. The tubercle bacteria should be stained a deep reddish color. All other bacteria or animal tissue in the preparation should be nearly or quite decolorized. If desired, a counter-stain, such as alkaline methylene blue, may be used after decolorizing; that is, the preparation should again be stained for about 1 minute in alkaline methylene blue, rinsed in water, and examined as before. In these preparations the tubercle bacteria are red and the other organisms and cells are blue. A counter-stain is of little value in preparations made for simple diagnostic purposes. When a counter-stain is desired Gabbett's decolorizing and counter-staining solution is very convenient.

GABBETT'S SOLUTION

Methylene blue (powder) .... 2 grams
10% sulphuric acid .... 100 cc.

After staining with the carbol fuchsin treat the preparations with this mixture until the film has a faintly bluish tint. This solution decolorizes and counter-stains at the same time. Care must be taken not to confuse the other acid fast bacteria with those of tuberculosis. The acid fast bacteria other than tubercle are decolorized with acidulated alcohol (3 per cent hydrochloric acid in 95 per cent. alcohol).

*In animal inoculation for the purpose of diagnosis guinea pigs are preferable. Rabbits rarely develop the disease when inoculated with the human variety but usually do when infected with the bovine variety. With tuberculous tissue either of the two methods described below may be employed.

A small piece (about the size of a pea or bean) of the tissue may be inserted under the skin by first making an incision with a sharp scalpel through the skin and superficial fascia, and then with a pair of fine forceps insert the bit of tissue well under the skin and close the opening with one or more sutures.

The tissue may be crushed in a mortar and thoroughly mixed with a few cubic centimeters of sterile water or bouillon and then injected with a hypodermic syringe. The needle should be of large calibre. If it is suspected milk, it may be injected into the abdominal cavity. If the material is tuberculous and contains living tubercle bacteria, the death of the animal follows in from three weeks to four months. Usually the lymphatic glands in the groin and axilla are enlarged and often caseous. If a guinea pig is used, the liver, spleen, lungs and kidneys are liable to be affected, in the order named; if a rabbit, the lungs are often the first of the viscera to be attacked.

In avian tuberculosis it is necessary to use chickens instead of guinea pigs. They may be inoculated subcutaneously or into the abdominal cavity. Several weeks may be necessary for the lesions to develop sufficiently to enable one to distinguish the disease.

FIG. 37. A PHOTOGRAPH OF A CROSS SECTION THROUGH THE PORTAL GLAND OF A TUBER-
CULOUS LIVER OF A COW. IT SHOWS SEVERAL TUBERCULOUS MASSES (a) WITHIN THE
LIVER TISSUE. IN SOME OF THESE THE DEAD TISSUE IS BEGINNING TO CALCIFY. THE
PORTAL GLAND (b) IS VERY MUCH ENLARGED AND TUBERCULOUS THROUGHOUT.
(REDUCED IN SIZE).

In case of poultry the bacteria are usually present in very large num-
bers in the original lesions.

It is important to differentiate tubercle bacteria from other acid fast organisms that may be present in the saliva, excreta or milk. When but one or at most but a few acid fast bacteria are present in microscopic preparations from any of these substances, it is necessary to establish the identity of the organism. For this certain micro-chemical procedures, such as decolorization with acidulated alcohol, are recommended. It is safer, however, to resort to guinea pig inoculation. Formerly the finding of acid fast bacteria in the excreta was considered conclusive evidence of tuberculous infection. Now, the large number of saprophytic acid fast bacteria that are known to exist and which may readily be found in saliva, excreta and milk necessitate careful differentiation of these organisms before a positive diagnosis can be made.

There are two other recognized methods for securing material from the lungs for examination for tubercle bacteria.

Trachea method. This method was introduced by Scharr and Opalka* who emphasized the importance of securing mucus from the lower part of the trachea and upper bronchi. The method of proce-dure, briefly stated, is as follows: Render the field of operation over the trachea surgically clean. A short incision is made following the median line down upon the trachea at a place located about level with the point of the shoulder. A small sharp tracheal tube is inserted between two of the cartilaginous rings. Through this tube is passed a long wire in which is an eye armed with a small piece of sterile gauze. The wire with the gauze is passed well down the trachea where it usually induces a cough. After swabbing out the trachea the gauze is withdrawn and placed in a sterile retainer until examined. Smears may be made for direct microscopic examination but the most successful procedure is to inoculate guinea pigs. This can be done by agitating the gauze in a small quantity of bouillon or sterile normal salt solution and injecting the washings hypodermically.

Esophagus method. This requires a small thick-walled cup attached to the end of a curved wire. The head of the cow is elevated and the cup is passed directly into the esophagus. The cup is withdrawn and its contents placed in a sterile retainer. It is examined microscopi-cally and by guinea pig inoculations.

Udall and Birch adopted the esophageal method after testing both. They found by this method in the examination of four reacting herds

*Their report was translated by Dr. A. T. Peters and published in Bulletin No. 5, State Board of Livestock Commissioners of Ill., 1912.

that from 6 to 29% of the animals that failed to show physical symptoms were eliminating tubercle bacteria.

Tuberculin. The tuberculin test is the best, and, in a large majority of tuberculous cases among animals and in man, the only means of positively detecting the disease in the living individual. Tuberculin* is the concentrated liquid, usually glycerinated bouillon, on which tubercle bacteria have grown until the products resulting from their multiplication including the disintegrated bodies of dead tubercle bacteria have become imparted to the medium in sufficient quantity to inhibit their further development.

Tuberculin in the dose necessary to bring out its diagnostic effect is harmless for healthy animals. In the tuberculous animal it produces a rise of temperature which, within certain limits, follows a definite course usually terminating in from 18 to 24 hours after the injection. Occasionally the temperature remains above the normal for a longer time. The temperature usually begins to rise in from six

---

*Tuberculin is prepared as follows:

"The preparation of the culture medium (glycerinated bouillon), distributing it in suitable flasks (500 cc. Erlenmeyer putting but 100 cc. of liquid in each) and inoculating it with the growth from a pure culture of tubercle bacteria that will produce a good tuberculin. Both the human and bovine varieties of the organisms are used for this purpose.

"The flasks are placed in an incubator at a temperature of 37° C. where they remain until the growth ceases. The length of time necessary to accomplish this depends upon the age and condition of the culture from which the inoculations were made. From six to ten weeks are required.

"After the maximum growth is attained, the cultures are sterilized by heat, either by boiling in a closed water bath or heating to a higher temperature in an autoclav.

"After sterilization, the cultures are filtered to remove the bacteria, and the filtrate is evaporated, over a water bath, to the desired degree of concentration.

"The concentrated liquid is passed through a Pasteur or Berkefeld filter, standardized, bottled and labeled for distribution. It should be perfectly clear although its color may vary. If it is cloudy it should be rejected."

It will be seen from the method of preparation that tuberculin cannot possibly contain living tubercle bacteria. It is heated on two occasions to a temperature and for a length of time far in excess of that required to destroy them, besides being passed through a filter capable of removing all bacteria.

The original tuberculin or lymph of Koch was concentrated to one-tenth of the volume of the saturated culture. This gave a thick, syrupy liquid owing to the presence of the glycerin. The diagnostic dose which came to be recommended for cattle of medium weight was 0.25 cc. On account of its consistency as well as the minuteness of the dose, it was found to be practicable to dilute this quantity with seven parts of a diluent. A weak solution of carbolic acid was ordinarily used. The difficulties and the danger of contamination involved in making the dilutions in the field led to the method of diluting the tuberculin in the laboratory before sending it out. This has been the practice of the Bureau of Animal Industry for a number of years. Equally as good results are obtained by concentrating the saturated culture to the point where 2 cc. contains an equivalent of the 0.25 cc. of the highly concentrated lymph. This process avoids the necessity of dilutions and, with the addition of a few drops of carbolic acid, the weaker solution keeps perfectly.

to eight hours, giving a steady but quite rapid elevation for from 1 to 3 hours, a continuous high elevation for from 2 to 4 hours, possibly longer, and a gradual decline. This is practically constant, be the raise moderate or extreme. In rare cases the elevation of the temperature does not begin for 18 or more hours after the injection. In addition to the elevation in temperature there is sometimes a marked nervous chill or muscular trembling. This has been referred to as the "Organic reaction." It is often overlooked by those making the test.

Subcutaneous use of tuberculin. In brief, this method of applying tuberculin is as follows:

"The normal temperature of the animal to be tested must be determined. It is recommended that it be taken hourly or every two hours for the day preceding the test.

"The tuberculin is injected subcutaneously, usually in the side of the neck. Care must be taken that the syringe is sterile and the site of injection should be disinfected. The size of the dose depends upon the preparation of tuberculin used, that is, the degree of concentration.

"Beginning 6 hours after the injection, the temperature should be taken hourly, or at least every two hours, for fully twenty-four hours after injection. If at that time the temperature is rising it should be taken thereafter regularly until it returns to the normal.

"During the time of testing, the cattle should be kept quiet and free from all exposure, and fed normally.

"If a reaction occurs there is a gradual rise of temperature, beginning usually in from 4 to 10 hours after the injection of tuberculin, and continuing for some hours. The rise of temperature forms a curve with a maximum varying from $1.5°$ C. to $5°$ or $6°$ F. above the normal temperature of the previous day. There may also be an organic reaction. If there is a maximum rise of temperature of $1.5°$ F. with a definite curve it is usually safe to consider it a positive reaction. Erratic elevations of short duration are to be excluded. In all cases where the rise of temperature with a curve is under $103.5°$ F. there may be doubt as to the diagnosis. All cases that give a temperature of $103°$ F., if appreciably above the preinjection temperatures, should be considered suspicious. These cases should be retested a few months later using a double dose of tuberculin.

"Animals advanced in pregnancy and those known to be suffering from any other disease or in œstrum should not be tested. All

methods of treatment, including exposure to cold, or kind of food and drink which would tend to modify the temperature, should be avoided. Animals in which the disease is far advanced sometimes fail to react.

"The dose should vary to correspond with the weight of the animal. The dose for an adult cow of average weight is 0.25 cc. of the concentrated Koch tuberculin. In cases of a second test within a few weeks the quantity of tuberculin injected should be larger than for the first test."

In cattle, there is a marked variation in the normal daily temperature. A fluctuation of two or even three degrees within 24 hours is reported. Cold water when drunk in considerable quantities lowers the temperature from one to four degrees. A temporary excitement usually causes an elevation of from 1 to 1.5° F. There are also marked variations in the temperature of the same animal on consecutive days. The temperature at 12 noon and 12 midnight are often the same. In some cases the maximum elevation for the day occurs near midnight and on the following day the minimum temperature appears at that time. It is not uncommon for the maximum temperature to occur twice in the same day and occasionally several times within the twenty-four hours. There are marked individual variations in the effect of ordinary conditions upon the temperature, such as food, excitement or temperature of the air. A hot spell often causes a rise of one or more degrees. The average temperature taken hourly for two weeks of the animals in three herds taken by Howe and Ryder were 102.5°, 102.6°, and 101° F. respectively.

In a well kept Government herd that was tested with tuberculin, the temperature of part of the animals was taken hourly for 24 and part of them for 16 hours preceding the injection. An examination of the records* shows the average daily variation of 20 animals in which the temperature was taken for 24 hours to be 2.31° F. The maximum individual variation in a single day was 4.3° F., the minimum 0.5° F. In 25 other animals where the temperature was taken for 16 hours, the average variation was 1.79° F. In these the maximum variation was 3.2° F., the minimum 0.6° F. Ten healthy animals (did not react to tuberculin) in the same herd gave an average variation of 2.08° F. In these the maximum daily variation was 4.1° F., the minimum 1° F. The lowest temperature was usually, but not

*Bulletin No. 7, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C. The tests were made by Drs. F. L. Kilborne and E. C. Schrœder, under the direction of Dr. Theobald Smith.

invariably, in the morning and the highest in the afternoon or evening.

In view of these normal temperature variations, which often exceed the thermal tuberculin reaction, it is obvious that before applying the test the normal temperature of the animals should be approximately determined and that when they are being subjected to the test they should be cautiously protected, otherwise the comparatively slight elevation necessary to detect the disease may be disguised.

As the reaction seems to be the result of an affinity existing between the tuberculin and the products of the tissues stimulated by tubercle bacteria, it is natural to suppose that when the two are brought together in the same animal the reaction would invariably take place. Experience has shown that it almost always does.   It is important to understand, however, that under certain conditions a reaction will not occur.   Tuberculin fails to give a reaction or to indicate the pressure of infection under the following conditions:

"Where the tuberculin itself has not been properly made.

"Where the temperatures are not taken long enough after the injection of tuberculin to detect the late reactions.

"When infected animals are tested during the period of incubation before lesions have developed.

"When the lesions are arrested, encapsulated or healed or when the lesions are very extensive, a reaction may not follow the injection of tuberculin."

Ophthalmic use of tuberculin.   Wolff-Eisner and Calmette applied tuberculin to the conjunctiva for the purpose of detecting tuberculosis. The method consists in applying a few drops of tuberculin to the conjunctival sac.   The reaction usually begins in cattle in from 5 to 10 hours.   The first evidence is watering of the eyes, reddening of the conjunctiva and edematous swellings.   This is followed by a purulent exudate which tends to accumulate, usually in the inner canthus, and finally to drop from the eye.   It may dry forming a crust which eventually drops off.   The reaction continues for some time (1 to 2 days or longer).   The severity of the conjunctivitis is no indication of the extent of the disease.   It is reported that the subcutaneous injection of tuberculin either previously or at the same time does not affect the eye reaction.   The eye test does not give rise to symptoms other than those on the conjunctiva.

This method has not given uniformly satisfactory results.   It was found by Foth to give a reaction in tuberculous animals that had

ceased to react to the subcutaneous injection, because of too many tests.

The tuberculin to be used for the eye test should not contain substances that will irritate the conjunctiva. Foth recommends a 5% solution of dry tuberculin. A 50% solution of the concentrated tuberculin of Koch has been used, also the tuberculin of the Bureau of Animal Industry.

Intradermal test. Moussu and Mantoux reported the intradermal use of tuberculin as a means of diagnosing tuberculosis in cattle. The method consists in injecting the tuberculin into the deeper layers of the skin. The most suitable place to make this injection is in one of the folds of the skin on the under side of the base of the tail. The skin on the eyelid has been used. The skin at the base of the tail is soft and pliable and also free from hair. A hypodermic syringe with a short needle point should be used. Haring recommends a syringe with a 25 or 26 gauge needle with a point from 3 to 5 mm. in length. Dentists employ such needles for injecting local anæsthetics. The method of injecting tuberculin is described by Haring as follows: "The subcaudal fold is grasped between the thumb and the first two fingers of the left hand and the needle inserted horizontally into the thickness of the skin grasped between the thumb and finger. The 0.1 to 0.2 cc. dose, if properly placed, can be felt in the layers of the skin as it is expelled from the syringe, where it remains as a small lump in the skin after the needle has been removed. In our first test we made the mistake of trying to inject as near the surface of the skin as possible. It is difficult to inject into the layers of the epidermis and an injection into this part of the skin is of little diagnostic value. In case the needle is of the proper length, namely, one-quarter of an inch, there is little danger of going completely through the skin. When the proper point in the subcaudal fold is selected, it makes little difference whether the point of the needle is in the derma or in the subdermal connective tissue. With the proper syringe an expert operator can inject in the dark as accurately as in a good light. We have found that characteristic reactions occur with the injections from a long needle, placed completely through and beneath the skin layers of the subcaudal fold. It is well to inject at a point on the fold about two and one-half or three inches down the tail from the anus, since elsewhere reactions are not so easily perceived and at this point the bone and solid tissue of the tail form a background which renders the local reactions more prominent than those of the skin of the neck."

Haring recommends at least a 5% solution of alcoholic precipitated tuberculin in normal salt solution. He injects from 0.1 to 0.2 cc. Koch's Old Tuberculin in 10% solution has also given good results. A number of chemical firms put out intradermal tuberculin but they do not seem to be uniform in their preparation. Hutyra and Marek state that a 50% solution of Koch's Old Tuberculin gave good results. Some in this country use the Bureau of Animal Industry tuberculin direct in doses of from three to five drops and do not believe it is necessary to use the purified or precipitated tuberculin.

A positive reaction from the intradermal use of tuberculin is indicated by a thickening of the subcaudal fold or by the appearance at the point of injection of a characteristic sensitive swelling varying in size from that of a small pea to that of an orange. The swelling may be either edematous or hard and inflamed. The swelling can be recognized in some cases by the sixth hour. Generally it is not clear until about the twelfth hour. The swelling continues to increase in size for two or three days. In some cases the swelling does not appear so early. If only one observation can be made after the injection, it is recommended by Haring that it should be on the 72d hour. He states: "Small indurations at the point of inoculation about the size of the head of a parlor match frequently occur in normal non-reacting cattle, but anything larger than this which persists to the seventy-second hour should be considered a positive reaction."

In recording the reactions, a convenient method for comparison is to describe the size of the swelling as pea size, hazelnut, walnut or hen's egg size. The exact size, if desired, may be measured by means of calipers. Romer and Joseph consider that a thickening of the skin fold more than three millimeters larger than the thickness previous to injection should be considered a positive reaction. Cases in which the increase in thickness amounts to only three millimeters, they consider doubtful and subject to a retest. In our work, however, we have found the measurement of the swelling was not of much assistance. Experience and practice will enable the operator to judge of the size of the swellings without measuring, although for the sake of accurate records we have made a practice of taking careful measurements.

"In judging a local swelling, the observer should depend more upon the shape, appearance, tenseness, sensitiveness and location with respect to the exact point of injection, than upon the actual measurements. Only experience can teach an operator how to be certain of

a positive reaction when the local swelling is small. This is especially true when the tuberculin containing glycerin has been used."

Haring and Bell found that there was a thermal reaction following the intradermal injection but Moussu and Mantou state that they had no rise of temperature following this test.

This method of using tuberculin is recommended especially for testing range cattle or those that are not accustomed to being handled. Haring and Bell report excellent results by this method. They have found that in certain cases where the subcutaneous test failed to give
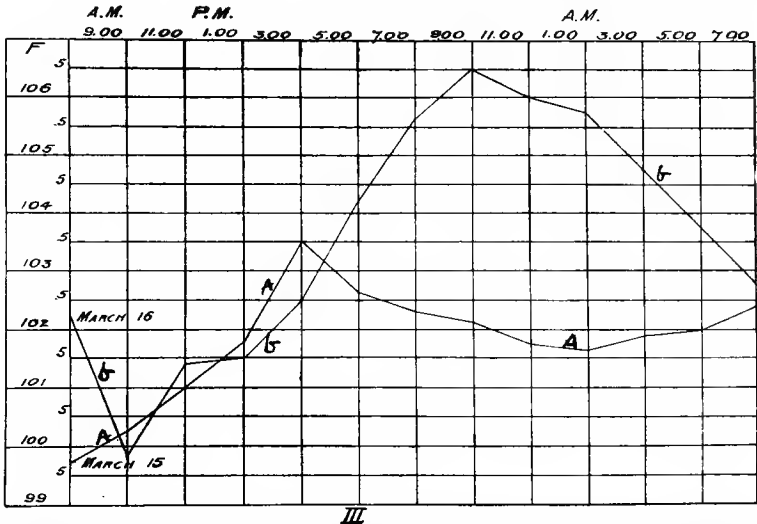
Fig. 38. TEMPERATURE CURVE OF A TUBERCULOUS COW FOR 48 HOURS. THE LINE A.A. SHOWS TEMPERATURE FOR 24 HOURS PRECEDING THE INJECTION OF TUBERCULIN, WHICH WAS INJECTED AT 9 A. M., MARCH 16; b, b, b, SHOWS THE TEMPERATURE FOR THE 24 HOURS AFTER THE TUBERCULIN INJECTION.

a reaction the intradermal test was positive and on post mortem the animals were found to contain tuberculous lesions. This method of using tuberculin is recommended by a number of veterinarians and it is now the official method recognized for using tuberculin in certain states.

Recently the lower eyelid has been selected as a more suitable place to inject the tuberculin. Nörgaard, Mohler and others have reported upon it, generally with favor.

Differential diagnosis. Tuberculosis in cattle is to be differentiated from actinomycosis, traumatic pericarditis, Johne's disease, certain

forms of pneumonia, and parasitic nodules. In each of these the
diagnosis is made, either microscopically or by guinea pig inoculation.
The use of tuberculin is of great value in the living animal.

Tuberculosis in swine must be differentiated from parasitic lesions
and certain forms of broncho-pneumonia. The histological study of
the tissues and a bacteriological examination for tubercle bacteria will
determine the nature of the lesion.

Tuberculosis in fowls is to be differentiated from leukæmia lympha-
denoma, sarcoma of the liver, asthenia, nodular tæniasis of the intes-
tine, and excessive infestation with the air sac mite (*Cytodites nudus*).
The diagnosis in these cases can be made largely from a histological
study of the tissues. In asthenia the obvious lesion is emaciation.
The same is true in case of the air sac mite.* The nodular tæniasis
can be determined by serial sections of the smaller nodules in which
will be found the heads of the flat worm causing the trouble. The
nodular tæniasis in fowls was described in Circular No. 3, Bureau of
Animal Industry, 1895. It has been mistaken for tuberculosis a
number of times. It is caused by a small flat worm described by
Piana (1881) as *Taenia bathrioplitis* which is probably synonymous
with *Dovainea Tetragona Molin,* 1858. In fowls, tuberculin does not
give satisfactory results.

Tuberculosis in sheep is to be differentiated from the nodular
intestinal disease caused by *Oesophagostoma columbianum Curtice.* It
is very rare in this species.

Tuberculosis in horses is to be distinguished from glanders and
parasitic lesions.

The control of tuberculosis in cattle. Several methods have been
proposed to eliminate tuberculosis from cattle. The preventing of
the spread of the virus from the diseased to healthy animals is the
most important precaution. The feeding of calves with infected milk
and the introduction of tuberculous animals into a herd are two of the
most important means of spreading the disease and therefore two that
should be most carefully avoided. The system introduced by Prof.
Bang of Copenhagen, Denmark, and generally known as the Bang
method, has proven to be very successful in certain herds. It con-
sists in the slaughter of the advanced cases and the isolation of the

---

*In 1903 the writer examined a number of fowls with Dr. Powers, Health Officer of
Los Angeles, that were thought to be tuberculous because of the extreme emaciation.
The examination showed an excessive invasion of air sac mites with no lesions in the
visceral organs.

reacting animals, which are kept for breeding purposes. The calves are separated from their dams immediately after birth and fed upon the milk of healthy cows or the sterilized milk of the reacting ones. This method has enabled many owners of infected animals to replenish their herds in from four to six years. In countries where it has been generally applied the percentage of tuberculous cattle has been wonderfully reduced.

The conservative method, recommended by Ostertag and employed in Germany, consists in a repeated careful physical examination and the elimination of all animals exhibiting evidence of tuberculosis and the raising of calves on pasteurized milk or that from tuberculous free cows. Ostertag states that this method, if carefully carried out, will be effective in combating the disease and he believes will eventually eradicate it.

The method, used largely in England, of examining the milk for tubercle bacteria and if any are found making a careful examination of the herd producing it and finding the spreader of the bacteria and eliminating her from the dairy, was reported to have greatly reduced the amount of infected milk in the market.

In the United States the method of testing the herd with tuberculin, slaughtering the reactors under inspection and the state paying an indemnity to the owner has been in operation for many years. It has been successful in many localities and a large number of herds have been freed of the disease by this procedure. It requires, however, that the barns be thoroughly disinfected, the test repeated at intervals and only sound animals introduced. In herds where the disease has been of long standing it requires several years before the infection is entirely removed. Owners of infected herds should follow some definite procedure, which will prevent further spread of the virus and conserve the value of the animals as much as possible. Many cows are as valuable for beef as for milk and others, where the breeding is of a high quality, can be isolated under the Bang method. There seems to be no method of procedure that can be followed in all places. The principle of preventing the spread of the virus from the infected to the well is the only one to strictly adhere to. The application of this principle can be made by different methods. The method to be followed should be the one best adapted to the existing conditions.

Immunization. The immunization of cattle against tuberculosis has been very carefully studied by a large number of investigators.

Koch pointed out that it was necessary to produce a bacterial as well as a toxic immunity. Tubercle bacteria of all varieties have been tried. Von Behring thought he had succeeded with his "bovo-vaccine" but its use was not satisfactory. Pearson and Gilliland did much work on this subject but a practical method for immunizing cattle has not been formulated although experimentally a certain amount of resistance can be produced.

The conclusions drawn from the recently reported extended investigations of M'Fadyean, Sheather, Edwards and Minett in the production of immunity against tuberculosis in cattle with the avian and human varieties of the organism are as follows:

"By the intravenous inoculation of avian tubercle bacilli it is possible to confer on healthy calves a markedly increased power of resistance to infection with bacilli of the bovine type.

"Such a method of vaccinating young cattle against tuberculosis involves little or no risk to the animals.

"When the vaccination of young cattle against tuberculosis is considered advisable avian bacilli should be preferred to human, in order to avoid the danger of infecting human beings with bacilli persisting in the bodies of the vaccinated animals and passed out with their milk."

Specific biological treatment. There is as yet no method of successfully treating tuberculosis by any serum or bacterin. Certain physicians report good results from the use of tuberculin in selected cases. This does not hold for cattle although a good many infected animals that reacted to tuberculin seem to have had the disease arrested or to have recovered and thereafter fail to react.

TUBERCULOSIS IN SWINE

Channels of infection. In most cases infection takes place by ingestion. The pig easily becomes tuberculous when fed on material rich in tubercle bacteria. Many illustrations of this are found in pigs fed on the refuse from dairies and cheese manufactories in districts where there is much tuberculosis in cattle or on tuberculous viscera in slaughter houses. Mohler found that when hogs were fed on tuberculous milk for three days, and killed and examined 107 days later 83.3 per cent. were tuberculous. Hogs that received infected milk for 30 days and were allowed to live fifty days thereafter were all

affected. Infection through the respiratory tract seems to be rare. The piggeries where the separated milk from creameries and whey from cheese factories are fed and those which join abattoirs supply the majority of swine found on post-mortem to be tuberculous.

Ostertag has called special attention to this disease among swine in certain parts of northern Denmark and Germany, where there was much tuberculosis in cattle, and where the swine were fed the slime from creamery separators. In the cases which have come to our notice there is very strong evidence that the swine were infected by being fed the milk from tuberculous cows. There are statements that swine tuberculosis is due, in certain cases at least, to the avian variety of the organism. Christiansen found that pigs were highly suscepti-ble to this variety of tubercle bacteria.

Symptoms. In most cases tuberculosis of the pig is first recognized at the abattoir. Sometimes, however, it causes local and general 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 fatten-ing and the progressive wasting of the subject. The mucous mem-branes become pale, the hide becomes dirty and there is usually either constipation or diarrhea. The animal is depressed, the corkscrew of its tail is straightened, the abdomen is pendulous and the eyes are sunken. Palpation of the abdomen is painful and may reveal more or less voluminous masses, due to the changes in the mesenteric glands. It is common to find the nodular tumors in the submaxillary region or at the thoracic inlet. In this form, the malady may last several months, but death supervenes rapidly if the lesions become generalized by the scattering of the bacteria through the blood stream. Primary pulmonary tuberculosis is very rare but sooner or later lung lesions complicate abdominal tuberculosis. They betray themselves at the outset by a short, dry, abortive cough and by difficult respira-tion. The cough soon becomes paroxysmal and painful and is often followed 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 reveals to be caused by the subjacent glands, these being enlarged, indurated, still fairly mobile and free from heat or tenderness. The retropharyngeal, superior cervical and sublingual glands are usually affected, forming a
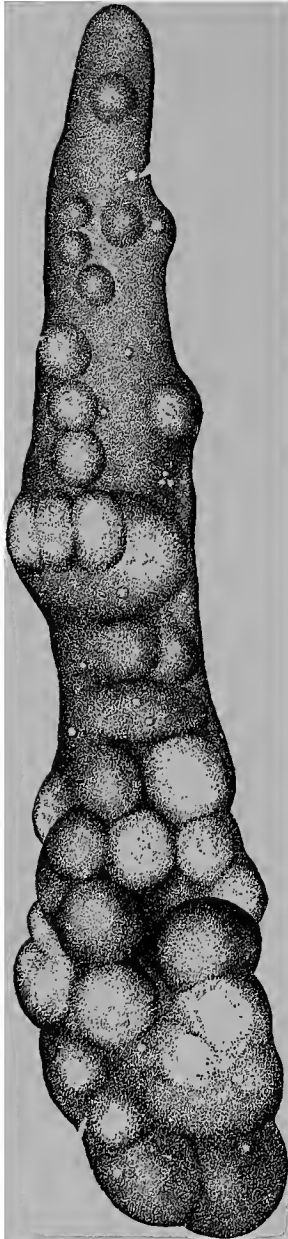
FIG. 39.   TUBERCULOUS SPLEEN
FROM A PIG.

kind of necklace of unequal and knotty tumors, reaching from ear to ear and becoming larger under the neck between the 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 may suppurate and discharge a small quantity of a 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.

There may be swellings of the bones, causing a true tuberculous arthritis when the lesions happen to be situated at the level of an epiphysis. Persistent lameness, fistulous wounds suppurating indefinitely, necrosis and caries, are the complications of the lesions of the bone, the development of which is always extremely slow.

Morbid anatomy. The manifestations of tuberculosis in swine are exceedingly interesting. Nocard found 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 lympha-

tic glands (submaxillary, parotid, pharyngeal, superior cervical, mesenteric, sublumbar, etc.) may be decidedly diseased, while the other organs remain practically intact. Lesions of the small intestine and of the cecum· 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 nodules which are yellowish white in color, varying in size from that of a pea to a hazel nut and of a tough consistency. On section they appear sometimes to be firm, homogeneous and fibrous; sometimes softened in the center, and often 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 number of minute, translucent, gray granulations, caused by the dissemination of tubercle bacteria through the blood stream, in which case the liver, the spleen, the kidneys, the medulla of the bones, and the mammæ may be infiltrated with similar growths.

Mohler has reported the results of the examination of 120,000 infected hogs of which 93.3 per cent. had tuberculous glands.

It is common to find lesions localized in one or several lymphatic glands. The pharyngeal and submaxillary glands are most often affected. They become enlarged, hard and knotty due to development of fibrous tissue. In section they have the appearance of old fibrous tissue; here and there small yellow foci are seen of a softer consistency, sometimes 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 bacteria are not usually found. The bacterium, however, is present and if this substance is inoculated into guinea pigs it 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 were also assigned the tuberculous lesions of bones (ribs, vertebræ, articulations, shoulder blades, hip bones) 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 or even the relationship of the two affections.

The generalization of the disease, especially in the muscular tissue, is reported by several observers. Moulé called attention to this peculiarity of the disease. Stockman shows that while the disease is ordinarily generalized, muscular lesions may exist in swine in the
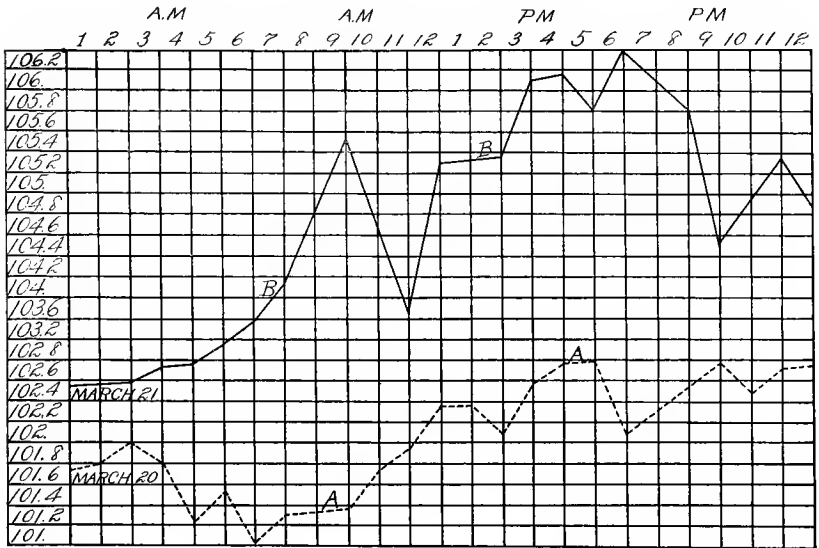
F_{IG}. 40.   TEMPERATURE CURVE OF A HOG.   DOTTED LINE A REPRESENTS TEMPERATURE
OF A HOG FOR 24 HOURS BEFORE THE INJECTION OF TUBERCULIN.   THE FULL LINE
A REPRESENTS THE TEMPERATURE OF THE HOG FOR 24 HOURS AFTER THE
INJECTION OF TUBERCULIN (*Schroeder*).

absence of generalization. Zschokke has called special attention to the localization of tuberculous lesions in the head of swine, especially in the nares and brain.

Diagnosis. Tuberculosis is diagnosed in swine by the same methods as in cattle although several of the special tests are not applicable to swine. The most usual means are (*a*) by the lesions; (*b*) finding the specific bacterium; and (*c*) the use of tuberculin either subcutaneously or by the intradermal test.

TUBERCULOSIS IN OTHER MAMMALS

Genera affected. It is stated that all species are sometimes attacked. Tuberculosis in the horse is rare, although a total of many cases have been reported. Bang has collected twenty-nine cases. In Saxony .08 per cent. of the horses (3,500) that were slaughtered were tuberculous. In this and most countries there are no reliable

FIG. 41.   LUNG OF HORSE CONTAINING TUBERCLES (a).

statistics respecting the extent of the disease in this species. M'Fadyean has pointed out the fact that in a considerable number of cases of equine tuberculosis, where the horses have been fed milk from tuberculous cows, the morbid anatomy differs but slightly from that in tuberculous cattle. Recently several authors have reported isolated cases in Europe. In this country horses are practically free from it. We have seen but one case.

Sheep and other domestic animals are reported to suffer more or less extensively from this disease. All of the so-called tuberculosis in

sheep that I have examined proved not to be tuberculosis but the "nodular disease" caused by an animal parasite (*Oesophagostoma columbianum*).   A few cases, however, have been reported.

Tuberculosis in dogs and cats is quite rare but several cases in each genus are on record.   Schlesinger reports a case of miliary tuberculosis in a dog with ulcerative endocarditis.   Blair has reviewed the literature on this subject and given the results of his investigations.
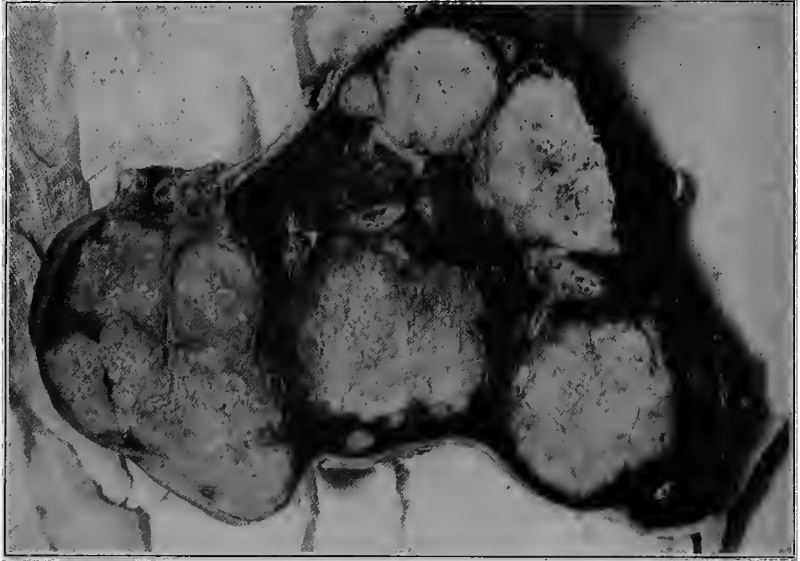
FIG. 42.   SPLEEN HORSE SHOWING TUBERCLES NATURAL SIZE

AVIAN TUBERCULOSIS

History.   In America, tuberculosis in fowls was described in 1900 by Pernot in Oregon and Burnett in northern New York.   In 1903 Moore and Ward found the disease in California, where in certain flocks it was very destructive.   It was recognized by the owners as "spotted liver," going light, and rheumatism.   In Europe it has been known for many years.   There is an extensive literature on this subject.

Symptoms.   The symptoms that are quite constant are emaciation, which in advanced cases becomes extreme, and anemia.   The comb, skin, and visible mucosa about the head are usually pale.   As the course of the disease advances the feathers become ruffled and the

fowls are weak, dumpish and move about very little. The eyes are bright in most cases until the end is near. The appetite is good, and the fowls eat ravenously until a few days before death. The temperature is in most cases within the normal limits, rarely it is subnormal. The blood is pale. The hemoglobin varies from thirty-five to seventy per cent. as tested with Gowers' hemoglobinometer. The red blood corpuscles vary from 1,010,000 to 2,600,000 per cubic millimeter. There appears to be a slight increase in the number of white corpuscles especially of the eosinophiles.

FIG. 43. A PHOTOGRAPH OF A TUBERCU-LOUS LIVER FROM A FOWL.

Tuberculous fowls are often lame. Pernot mentions this as one of the important symptoms in the cases he observed. It is due to joint lesions in some cases. In others it appears to be due to extensive lesions in the viscera.

The avian variety of tubercle bacteria resembles quite closely those of the human and bovine varieties in size and general morphology as they are found in the tissues of the fowl. A measurement of over two hundred individual organisms in cover glass preparations made directly from organs of fowls gave the following: In the liver the length varied from 1.2 to 3.5μ in the spleen and in the skin they varied from 1 to 4μ in length. A general average gave a length of 2.7μ. They often appear in these preparations in dense masses. Chains made up of a number of short elements are rarely present. Granules are occasionally observed. In the preparations from the skin a considerable number of them contain polar granules and not infrequently three such bodies were noticed in a single individual. Perhaps the most striking feature concerning these organisms in the tissues is their enormous numbers. Sibley has called attention to the similarity of avian tubercle bacteria to those of leprosy in that they multiply to

such enormous numbers without a pronounced breaking down of the tissues.

This variety can be obtained in pure cultures in about 20 per cent. of serum tubes inoculated directly from tuberculous lesions in fowls (Moore). It grows readily in glycerin agar, Dorset's egg medium, in glycerin bouillon and on potato.

Fowls inoculated in the abdominal cavity or subcutaneously with from one-half to one cubic centimeter of a glycerin bouillon culture develop either localized or generalized tuberculosis in from six weeks to three months, but a longer time is ordinarily necessary to kill them.

Rabbits and guinea pigs are not readily infected by the inoculation of pure culture. Moore and Ward failed to produce any tuberculous lesions in these species by this method.

Morbid anatomy. The lesions are widely distributed, and vary much in their location in different individuals. The liver is most frequently involved. The spleen, intestines, mesentery, kidneys, lungs and skin are affected in order mentioned. The appended table gives the distribution of the lesions in 17 cases observed by Moore.

THE DISTRIBUTION OF LESIONS IN TUBERCULOUS FOWLS

Fowl No.	Died or killed	Temp. F. before killing	ORGANS INVOLVED.*								
			Liver	Spleen	Intestine	Mesentery	Kidney	Ovary	Lungs	Bones	Skin
1	K	......	XX	.....	.....	.....	.....	.....	.....	.....	.....
2	D	......	.....	.....	.....	.....	.....	.....	.....	.....	XXX
3	K	106.5	XXX	.....	.....	.....	.....	.....	.....	.....	.....
4	K	107	XX	XX	X	.....	.....	.....	.....	.....	.....
5	K	105.2	XXX	.....	...X	.....	.....	.....	.....	.....	.....
6	K	107.4	XXX	XX	.....	.....	.....	.....	.....	.....	.....
7	K	107.2	.....	.....	XXX	XXX	.....	XX	.....	.....	.....
8	D	......	XX	X	X	X	X	.....	.....	.....	.....
9	D	......	.....	.....	.....	.....	X	.....	.....	XX	.....
10	K	108.4	XXX	X	.....	X	.....	.....	.....	.....	.....
11	K	106.4	XXX	X	X	.....	.....	.....	.....	.....	.....
12	K	107.4	XXX	XX	X	.....	XXX	.....	XXX	.....	.....
13	K	107	XXX	.....	.....	.....	.....	.....	.....	.....	.....
14	K	107.6	XXX	.....	.....	.....	.....	.....	.....	.....	.....
15	K	106.8	XXX	XX	.....	.....	.....	.....	.....	.....	.....
16	D	......	.....	.....	.....	XXX	.....	.....	.....	.....	.....
17	K	105	XXX	XX	.....	XXX	.....	.....	.....	.....	.....

*The relative numbers of tubercles are indicated by the number of Xs. XXX indicates an extensive invasion, XX a less number of tubercles, and X very few. Figures 43 and 44 show extent of lesions represented by XXX.

The tubercles in the earlier stages of the disease, especially in the liver, are small greyish points varying from 0.25 to 1.0 millimeter in

diameter. In advanced cases they are larger. They have a cheesy consistency, and are easily removed from the surrounding tissue. The removed, necrotic nodules have a roughened surface. The color is greyish or whitish in the early stages, but in the later ones it changes to a yellowish tint. Occasionally there are two distinct crops of tubercles, one consisting of nodules 4 to 6 millimeters in diameter and separated by a centimeter or more, and the other of closely set grayish

FIG. 44. A PHOTOGRAPH OF A SECTION OF TUBERCLE FROM
A FOWL, SHOWING THE NECROTIC CENTER AND
SURROUNDING ZONES. ENLARGED.

tubercles 0.25 to 0.5 mm. in diameter. In some cases the tubercles are few in number but larger in size. The liver cells between the tubercles are usually in a state of more or less degeneration, and frequently fat globules are numerous. The blood spaces are more than normally distended with blood. The lesions in the spleen, like those in the liver, consist of minute or larger tubercles of a grayish or of a yellowish tint. The central portions of the larger tubercles are often homogeneous, darker in color and more or less hyaline in appearance and consistency.

The tubercular growths in the intestine start in the walls of the intestine. They present a glistening appearance, grayish in color

and firm to the touch.   Frequently they are confluent.   When single
they vary from 1 to 10 mm. in diameter.   They are usually sessile on
the intestine but on the mesentery they are frequently peduncula-
ted, varying from 2 to 5 mm. in length.   On section the young
tubercles exhibit a grayish, glistening surface, but the more advanced
nodules contain recognizable necrotic centers.   In the larger tuber-
cles on the intestines the necrotic centers frequently open into the
lumen.

FIG. 45.   A PHOTOGRAPH OF A TUBERCULOUS MESENTERY OF A FOWL.   THERE ARE A
FEW SMALL TUBERCLES ON THE INTESTINE.

The skin lesions consist of a cellular infiltration usually about the root of the feathers. Frequently the nodules become confluent. They may or may not involve the subcutaneous connective tissue. The microscopic examination of the tubercles of the liver shows them to consist of a necrotic center surrounded by an irregular zone of epithelioid and giant cells. This is surrounded by a band of tissue consisting for the greater part of liver cells, more or less disintegrated free nuclei and a few infiltrated round cells. This zone is circumscribed by a narrow reactionary band consisting very largely of round cells. The structure is constant in both small and large tubercles, and not strikingly different from the structure of tubercles in certain of the mammals. The larger nodules seem in some instances to be the result of a continuous growth of a single tubercle, and in others to have resulted from the coalescence of a number of small ones. The necrotic center and reactionary zone of round cells are beautifully demonstrated by their reaction to nuclear stains.

REFERENCES

1. ADAMI. On the significance of bovine tuberculosis and its eradication and prevention in Canada. *Canadian Jour. of Medicine and Surgery*, Dec., 1899.

2. ASSMANN. Vergleichende Untersuchungen über die thermische Tuberkulinprobe und die Phymatin-Opthalmoreaktion. *Berliner Tierärztlich. Wochensch.*, Bd. XXVII (1911), S. 449.

3. BLAIR. Tuberculosis in the dog and cat. *Proceedings of the N. Y. State Vet. Med. Soc.*, 1915.

4. CURTICE. The detection of tuberculosis in cattle. *Annual Report, Bureau of Animal Industry, U. S. Dept. Agric.*, 1895–96.

5. DORSET. Experiments concerning tuberculosis. *Bulletin 52. Bureau of Animal Industry*, 1904.

6. EBER. Suggestions for a uniform system of interpreting the tuberculin reaction in cattle. *The Jour. Compr. Path. and Therap.*, Vol. XVIII (1905), p. 224.

7. FINDLAY AND MARTIN. The effect of daylight and drying on the human and bovine types of tubercle bacilli. *British Med. Jour.*, 1915, p. 110.

8. FOTH. Tuberkulipnroben nach Moussu und Mantoux. *Berliner tierärztlich. Wochensch.*, Bd. XXV, (1909), S. 727.

9. HARING. Bovine tuberculosis investigations at the University of California Farm. *Proceedings of the A. V. M. A.*, 1910, p. 306.

10. JOEST AND MARJANEN. Histologische Studien über die Serosentuberkulose des Rindes. *Zeitsch. für infek. Krankheiten*, Bd. XV (1914), S. 1.

11. KOCH. The etiology of tuberculosis. *Mitt. aus. dem. Kaiserl. Gesundheitsamte*, Bd. II (1884). Translated in Vol. CXV, (1886). New Sydenham Society.

12. KOCH. The combating of tuberculosis in the light of the experience that has been gained in the successful combating of other infectious diseases. *Amer. Vet. Rev.*, Vol. XXV (1901), p. 441.

13. LUCKEY. The intradermal tuberculin test. *Amer. Vet. Rev.*, Vol. XLI (1912), p. 316.

14. M'FADYEAN, SHEATHER, EDWARDS AND MINETT. Experiments regarding the vaccination of cattle against tuberculosis by the intravenous injection of tubercle bacilli of the human and avian types. *Journ. of Comp. Path. and Therap.*, Vol. XXVI (1913), p. 337.

15. MALM. Ueber die Typen und Uebergangsformen des Tuberkelbacillus. *Deut. tier. Wochensch.*, Bd. XXI, S. 746.

16. MALM. Ueber die sogenamten bovinen u. humanen Typen des Tuberkelbacillus. *Contralb. f. Bakt.*, Bd. LXV.

17. MEYER. The conjunctival reaction for glanders. *Jour. Infect. Dis.*, Vol. XII (1913), p. 172.

18. MOHLER. Infectiveness of milk of cows which have reacted to the tuberculin test. *Bulletin 44. Bureau of Animal Industry*, 1903.

19. MOHLER. Tuberculosis in hogs, with special reference to its suppression. *Amer. Vet. Rev.*, Vol. XXXII (1907), p. 176.

20. MOHLER AND WASHBURN. A comparative study of tubercle bacilli from varied sources. *Bulletin 96. Bureau of Animal Industry*, 1907.

21. MOORE AND DAWSON. Tuberculosis in swine, the nature of the disease with a report of three cases. *Annual Report, Bureau of Animal Industry, U. S. Dept. Agric.*, 1895–96.

22. MOORE. The preparation of tuberculin, its value as a diagnostic agent, and remarks on the human and bovine tubercle bacilli. *Trans. of the Med. Society of the State of N Y.*, 1900.

23. MOORE. A report on bovine tuberculosis. *New York State Dept. of Agric.*, 1903.

24. MOUSSU AND MANTOUX. On the intradermal reaction to tuberculin in animals. *Comp. Rend. Acad. Sci.*, Paris, Vol. XVII (1908), p. 502. *Abs. in Expt. Sta. Record*, Vol. XXI (1909), p. 582.

25. MOUSSU AND MANTOUX. Sur l'intra-dermo-reaction à la tuberculine chez les animaux. *Proceedings 6th International Congress on Tuberculosis*, Vol. IV, p. 821.

26. NOCARD. The animal tuberculosis. New York.

27. PEARSON. The Pennsylvania plan for controlling tuberculosis of cattle. *Proc. Amer. Vet. Med. Asso.*, 1899.

28. PEARSON. Tuberculosis in cattle and the Penn. plan of its repression. *Bulletin 75. Penn. Dept. of Agric.*, 1901.

29. PEARSON. The repression of tuberculosis in cattle by sanitation. *Bulletin 74. Penn. Dept. of Agric.*, 1901.

30. PEARSON. The artificial immunization of cattle against tuberculosis. *Amer. Vet. Rev.*, Vol. XXIX (1905), p. 543.

31. RAVENEL. The dissemination of tubercle bacilli by cows in coughing a possible source of contagion. *Univ. of Penn. Med. Magazine*, Nov., 1900.

32. RAVENEL. The comparative virulence of the tubercle bacillus from human and bovine sources. *Univ. of Penn. Med. Bulletin*, Sept., 1901.

33. RAVENEL. The intercommunicability of human and bovine tuberculosis. *The Univ. of Penn. Med. Bulletin*, May, 1902.

34. REPP. Transmission of tuberculosis through meat and milk. *American Medicine*, Oct. 6, Nov. 2, 1901.

35. SALMON. Legislation with reference to bovine tuberculosis. *Bulletin 28. Bureau of Animal Industry, U. S. Dept. of Agric.*, 1901.

36. SALMON. The tuberculin test of imported cattle. *Bulletin 32. Bureau of Animal Industry, U. S. Dept. of Agric.*, 1901.

37. SALMON. Bovine and human tuberculosis. *Proceedings Amer. Vet. Med. Asso.*, 1903, p. 436.

38. SALMON. Tuberculosis of the food-producing animals. *Bulletin 38. Bureau of Animal Industry*, 1906.

39. SCHROEDER AND COTTON. The relation of tuberculous lesions to the mode of infection. *Bulletin 93. Bureau of Animal Industry*, 1906.

40. SCHROEDER AND COTTON. Experiments with milk artificially infected with tubercle bacilli. *Bulletin 86. Bureau of Animal Industry*, 1906.

41. SCHROEDER AND MOHLER. The tuberculin test of hogs. *Bulletin 88. Bureau of Animal Industry*, 1906.

42. SMITH. Investigations concerning bovine tuberculosis with special reference to diagnosis and prevention. (Pathological part.) *Bulletin No. 7, Bureau of Animal Industry, U. S. Dept. of Agric.*, 1894.

43. SMITH. A comparative study of bovine tubercle bacilli and of human bacilli from sputum. *The Jour. of Exper. Med.*, Vol. III (1898).

44. SMITH. The thermal death point of tubercle bacilli in milk and some other fluids. *The Jour. of Exper. Med.*, Vol. IV (1899), p. 217.

45. SMITH. The channels of infection in tuberculosis, together with some remarks on the outlook concerning a specific therapy. *Trans. Mass. Med. Soc.*, 1907.

46. UDALL AND BIRCH. The diagnosis of open cases of tuberculosis. *Report N. Y. State Vet. College at Cornell Univ.*, 1913–1914, p. 55.

47. WARD AND BAKER. Experiments with the intradermal test for tuberculosis in cattle. *Proceedings Am. Vet. Med. Asso.*, 1910.

48. WILLS AND LINCH. Delayed reactions following injection of tuberculin. *Report of U. S. Live Stock Sanitary Association*, 1913.

49. WILLS AND LINCH. Delayed reactions following injection of tuberculin. *Cornell Veterinarian*, Vol. IV (1914), p. 16.

REFERENCES AVIAN TUBERCULOSIS

1. BRAY. Tuberculosis in chickens. *Jour. Compar. Med. and Vet. Archives*, Vol. XVII (1896), p. 461.

2. BURNETT. Tuberculosis in chickens positively identified in New York. *Am. Vet. Review*, Vol. XXX (1907), p 312.

3. CADIOT. Sur la tuberculose du cygne. *Bul. de la Soc. Cen. et Méd. Vét.*, Vol. XLIX (1895), p. 570.

4. CADIOT, GILBERT AND ROGER. Inoculation of the tuberculosis of gallinaceous to mammalia. *Amer. Vet. Review*, Vol. XX (1896–7), p. 225.

5. CADIOT, GILBERT ET ROGER. Note sur la tuberculose des volailles. *Recueil de Méd. Vét. Série VII*, Vol. VIII (1891), p. 22.

6. CADIOT, GILBERT AND ROGER. A contribution to the study of avian tuberculosis. Studies in clinical veterinary medicine and surgery. (1900). (Translated by Dollar.)

7. CHRISTIANSEN. Über die Bedeutung der Geflügeltuberkulose für das Schwein. *Zeitsch. für. Infek. Krankheiten*, Bd. XIV (1913), p. 323.

8. EBERLEIN. Die Tuberculose der Papageien. *Monatshefte für praktische Thierheilkunde*, Bd. V (1894), S. 248.

9. HASTINGS, HALPIN AND BEACH. Avian tuberculosis. *Journ. of Infect. Dis.*, Vol. XIII (1913), p. 1.

10. LUCET. Sur un symptome de la tuberculose chez la poule. *Recueil de Méd. Vét., Série VII*, Vol. VIII (1891), p. 172.

11. MAFFUCCI. Die Hühnertuberculose. *Zeitschr. für Hygiene*, Bd. XI (1892), p. 445.

12. MOORE AND WARD. Avian tuberculosis. *Proc. Amer. Vet. Med. Asso.*, 1903, p. 169.

13. MOORE. The morbid anatomy and etiology of avian tuberculosis. *Jour. of Med. Research*, Vol. XI (1904), p. 521 (Bibliography).

14. NOCARD. Sur une tuberculose zoogléique des oiseaux de bassecour. *Bul. et Memoires de la Soc. Centrale et Méd. Vét.*, 1885, p. 207.

15. NOCARD. Transmission de la tuberculose des poules et l'homme. *Veterinaire*, Vol. III (1886), p. 658.

16. PERNOT. Investigations of diseases of poultry. *Bulletin No. 64. Oregon Agric. Expt. Sta.*, 1900.

17. SIBLEY. Tuberculosis in birds. *Jour. of Compar. Med. and Vet. Arch.*, Vol. XI (1890), p. 317.

18. STRAUS ET GAMALEIA.   La tuberculose humaine, sa distinction de la tuberculose des oiseaux.   *Archiv. de Méd. Exper.*, Bd. III (1891), p. 457.

19. STRAUS ET WURTZ.   Sur la resistance des poules à la tuberculose par ingestion. *Congress pour l' étude de la tuberculose*, 1888, p. 328.

20. WARD.   Tuberculosis in fowls.   *Bulletin No. 161. California Agr. Exper. Station*, 1904.

21. WEBER.   Review of the avian tuberculosis.   *Jour. of Compar. Med. and Vet. Arch.*, Vol. XIII (1892), p. 429.

THE FOLLOWING BULLETINS ON TUBERCULOSIS HAVE BEEN ISSUED FROM THE VARIOUS
STATE AGRICULTURAL EXPERIMENT STATIONS

1. BANG.   The application of tuberculin in the suppression of bovine tuberculosis. *Bulletin 41*. Massachusetts. 1896. (A translation).

2. BEACH.   The history of a tuberculous herd of cows. *Bulletin 24*. Storrs, Conn. 1902.

3. BITTING.   Bovine tuberculosis in Indiana.   *Bulletin 63*. Ind. 1896.

4. BREWER.   Tuberculosis.   *Bulletin 41*. Utah. 1895.

5. BRISCOE AND MACNEAL.   Tuberculosis of farm animals. *Bulletin 149*. Illinois. 1911.

6. CARY.   Bovine tuberculosis.   *Bulletin 67*. Alabama. 1895.

7. CONN.   The relation of bovine tuberculosis to that of man and its significance in the dairy herd.   *Bulletin 23*. Storrs, Conn. 1902.

8. DINWIDDIE.   The relative virulence for the domestic animals of human and bovine tuberculosis.   *Bulletin 57*. Kansas. 1899.

9. DINWIDDIE.   The relative susceptibility of the domestic animals to the contagia of human and bovine tuberculosis.   *Bulletin 63*. Kansas. 1900.

10. FARMER'S BULLETIN 473.   *U. S. Department of Agriculture*, 1911.

11. FISCHER.   Bovine tuberculosis.   *Bulletin 79*. Kansas. 1898.

12. GLOVER.   Relation of bovine to human tuberculosis.   *Bulletin 66*. Colorado. 1901.

13. GRANGE.   Tuberculosis.   *Bulletin 133*. Michigan. 1896.

14. HARDING, SMITH AND MOORE.   The Bang method etc. *Bulletin*. Geneva, N. Y. 1906.

15. HARING AND BELL.   The intradermal test for tuberculosis in cattle and hogs. *Bulletin 243*. California. 1914.

16. HILL AND RICH.   Bovine tuberculosis.   *Bulletin 42*. Vermont. 1894.

17. LAW.   Tuberculosis in relation to animal industry and public health.   *Bulletin 65*. (Cornell), New York. 1894.

18. LAW.   Experiments with tuberculin on non-tuberculous cows. *Bulletin 82*. (Cornell), New York. 1894.

19. LAW.   Tuberculosis in cattle and its control.   *Bulletin 150*. (Cornell), New York. 1898.

20. MARSHALL.   A study of normal temperatures and the tuberculin test.   *Bulletin 159*. Michigan. 1898.

21. MARSHALL.   Killing the tubercle bacilli in milk.   *Bulletin 173*. Michigan. 1899.

22. MAYO.   Some diseases of cattle, Texas itch, blackleg, tuberculosis, Texas fever.   *Bulletin 60*. Kansas. 1897.

23. MAYO AND KERR.   Treatment of bovine tuberculosis.   *Bulletin 199*. Virginia. 1912.

24. MOORE.   The elimination of tubercle bacilli from infected cattle, and the control of bovine tuberculosis and infected milk.   *Bulletin 299*. (Cornell), New York. 1911.

25. MOORE. Bovine tuberculosis. *Bulletin 225.* (Cornell), N. Y. 1905.

26. NELSON. On the use of Koch's lymph in the diagnosis of tuberculosis. Report of the biologist. New Jersey. 1893.

27. NELSON. Experimental studies of the Koch test for tuberculosis. New Jersey, 1895.

28. NELSON. The suppression and prevention of tuberculosis of cattle and its relation to human consumption. *Bulletin 118.* New Jersey. 1896.

29. NESOM. Tuberculosis of cattle. *Bulletin 50.* S. C. 1900.

30. PAIGE. History of tuberculosis in a college herd. Use of tuberculin in diagnosis. *Bulletin 27.* Massachusetts. 1894.

31. PEARSON. Tuberculosis of cattle. *Bulletin 29.* Penn. 1894.

32. REYNOLDS. Bovine tuberculosis. *Bulletin 51.* Minn. 1896.

33. RUSSELL. Tuberculosis and the tuberculin test. *Bulletin 40.* Wisconsin. 1894.

34. RUSSELL. The history of a tuberculous herd of cows. *Bulletin 78.* Wisconsin. 1899.

35. RUSSELL. A lesson in bovine tuberculosis. *Bulletin 114.* Wisconsin. 1904.

36. RUSSELL. Two ways of treating tuberculosis in herds. *Bulletin 126.* Wisconsin. 1905.

37. RUSSELL. The spread of tuberculosis through factory skim milk with suggestions as to its control. *Bulletin 143.* Wisconsin. 1907.

38. RUSSELL AND HASTINGS. Bovine tuberculosis in Wisconsin. *Bulletin 84.* Wisconsin. 1901.

39. STALKER AND NILES. Investigation of bovine tuberculosis with special reference to its existence in Iowa. *Bulletin 39.* Iowa. 1895.

40. THORNE. Bovine tuberculosis. *Bulletin 108.* Ohio. 1899.

41. VAN ES. Bovine tuberculosis. *Bulletin 77.* North Dakota Agricultural Experiment Station. 1907.

42. WILLIAMSON AND EMERY. Tuberculosis and its prevention. *Bulletin 117.* N. C. 1895.

———

1. Report of the International Commission on the Control of Bovine Tuberculosis. *Proceedings Am. Vet. Med. Assò.,* 1910, p. 90.

2. Report of the Royal Commission on Tuberculosis (Human and Bovine). London. 1907–1911.

JOHNE'S DISEASE

Synonyms. Specific paratuberculous enteritis; pseudo-tuberculosis; chronic bovine pseudo-tuberculous enteritis; specific paratuberculosis of cattle; enteritis paratuberculosis; bovis specifica; *la diarrhée chronique du boeuf.*

Characterization. Johne's disease is an intestinal disorder caused by an acid-fast bacterium. It is characterized by a diarrhea, gradual emaciation and the presence of large numbers of acid-fast bacteria in the mucous membrane of the affected portions of the intestine. The small and large intestines and associated lymph glands are

involved. In addition to cattle, sheep, goats, deer, buffalo and possibly the horse are susceptible.

History. Johne and Frothingham described a disease in 1895 in which the intestinal mucosa contained large numbers of acid-fast bacteria. They thought it was a case of tuberculosis in a cow due to the avian tubercle bacterium. In 1903 Markus called attention to its frequent occurrence in Holland. Since that time it has been recognized in Belgium, Switzerland, Denmark and England. A few cases have been observed in this country. Sir John M'Fadyean has proposed the name Johne's disease for this very serious affection. It seems to be prevalent in many localities.*

Etiology. The cause of this disease is an acid-fast bacterium (*B. paratuberculosis*) which is found in large numbers in the affected mucosa, and also in the mesenteric and colic lymphatic glands. Morphologically it closely resembles the tubercle bacterium. It varies in size from 1 to 2μ in length and a few are said to attain to 4μ. It stains uniformly with the acid-fast stains. Occasionally the longer forms show alternating stained and unstained segments.

According to M'Fadyean this organism is not inoculable to either guinea pigs or rabbits. It has been cultivated on media containing the dead bodies or extracts of a number of other acid-fast bacteria. From these cultures it has been grown on media not containing acid-fast organisms.

This bacterium apparently does not form a strong cell poison, which may account for the absence of necrosis. On the other hand,

---

*Stockman in his article on Johne's disease in sheep refers to a malady of sheep on the Eastern border of England and Scotland known locally as "scrapie." In the intestines he found acid-fast bacilli indistinguishable from those of Johne's disease. M'Gowan (Investigation into the disease of sheep called "scrapie," 1914) considers the all important symptom to be pruritis. He believes it to be the one distinguishing sign by which it may be recognized. His conclusions, after a careful investigation, are that the cause of "scrapie" is a heavy infestation of the sheep with a Sarcosporidium. His final conclusions are:

"The sarcocyst is always present in the skeletal muscles of scrapie sheep in large numbers; and the more advanced the case the larger is the number of the sarcocyst present.

"Pruritis (or itching), the chief symptom in scrapie, can be reproduced in rabbits by the injection into them of sarcosporidial emulsions.

"Careful clinical examination of typical cases makes it highly probable that the paretic phenomena of the disease are due to a primary muscle lesion.

"There is an absence of any condition post-mortem, except extensive sarcosporidiosis, sufficient to or of a nature likely to cause the phenomena observed in the disease. In this connection one would specially note Cassirer's findings in cases of the Traberkrankheit in Germany

"No single view can explain so well the symptomatology and the epizoötiology, etc. of the disease as this."

the tissues appear to be almost powerless to restrain its multiplication and invasion.

The period of incubation is not determined but it is known to be long, possibly a year or more.

Symptoms. The first symptom to be observed is a loss of flesh, although the appetite remains normal. The hair becomes roughened
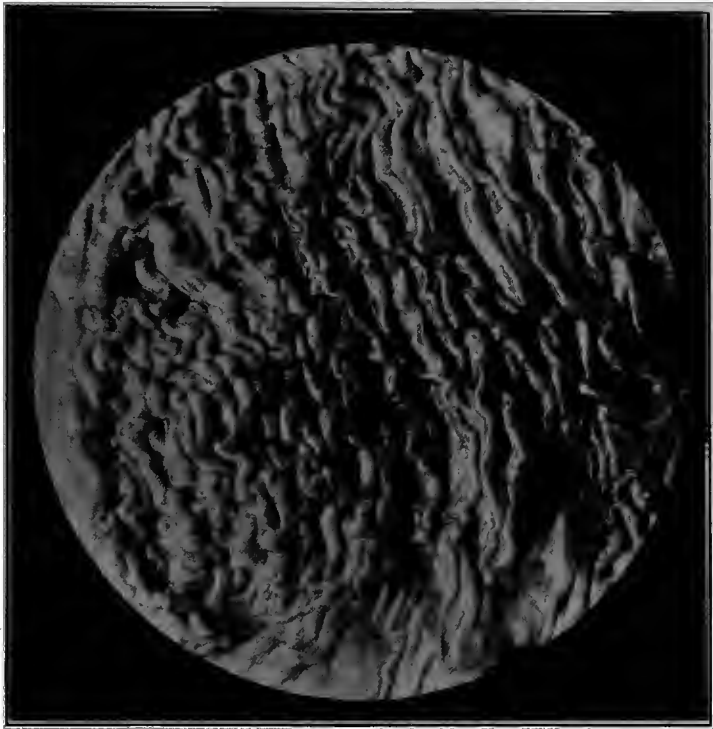
FIG. 46. A PHOTOGRAPH OF A PORTION OF THE SMALL INTESTINE OF A COW DEAD OF JOHNE'S DISEASE. NATURAL SIZE.

and the animal presents an unthrifty appearance. Diarrhea sets in early and usually it is profuse and persistent from the time it begins, although it may sometimes be checked temporarily by giving dry food and by the administration of astringents. Emaciation becomes a prominent symptom.

Morbid anatomy. The lesions are primarily in the large and small intestines and associated lymph glands. The distal part of the small

intestine is most often involved. The lesion is in the mucous and submucous tissue. In some cases the acid fast bacteria invade the submucosa in which case the wall of the intestine becomes thickened. According to Sheather a variable amount of new cellular tissue composed of cells of the epithelioid type, and containing an occasional giant cell, is developed beneath the epithelium and often also in the submucous layer. In proportion to its thickening the mucosa shows more or less coarse wrinkling. Ulceration is not observed. There is little congestion on the tops of the folds of the mucous tissue.

The mesenteric lymphatic glands may be enlarged. They may contain collections of epithelioid and giant cells. Meyer found more or less edema of the abomasum in about 40 per cent. of his cases. The atrophy of the spleen with a brownish dry appearance of the pulp was always present and he found clumps of pigment often in large amounts. This condition was first mentioned by Bang. When cut an appreciable amount of water-like liquid exudes from the surface. The absence of congestion has been noted.

The most striking feature of the disease is the slight tissue changes even when the bacteria are exceedingly numerous. In sections made at right angles to the mucous surface of the intestine an irregularity in the size and outline of the villi can be observed. Some of the villi may be partially denuded of epithelium. In the glandular layer the interstitial tissue between the tubular glands may be increased in amount and the glands may show evidence of atrophy. In sections stained by the Ziehl-Neelsen method, with Pappenheim's stain for contrast, M'Fadyean states that those parts in which the bacteria are numerous have an appearance very similar to that of a genuine tuberculosis lesion just before the onset of necrosis and caseation, that is, they appear to be mainly made up of the so-called epithelioid cells, with occasionally a well formed giant cell. Sometimes the outlines of these epithelioid cells are distinct, but, as a rule, wherever the bacilli are numerous there appears to have been a partial fusion of the cell bodies, and the appearance is that of a sort of matrix substance with imbedded nuclei. The majority of these nuclei are shrivelled or distorted in appearance, and they stain lightly as compared with any of the nuclei in the surrounding normal tissue. According to M'Fadyean, the important points to notice are that the diseased tissue is never sharply circumscribed and that there is no actual necrosis, although the appearance of the new tissue may be interpreted as indicating that the cells are on the point of losing their vitality.

Within the parts which contain large numbers of the bacteria there are also sometimes recognizable small round compact nuclei, apparently belonging to cells of the lymphocyte type, and at their margins there are numerous cells whose bodies stain red with Pappenheim's stain.

The bacteria do not appear to be specially intra-cellular; many of them seem to be lying free, and others appear to be situated within the fine reticulum of the villi. The structural alterations are everywhere proportional to the number of bacteria which indicates that, contrary to what is the case in tuberculosis, the bacteria have little or no tendency to degenerate and disappear from the older lesions. The bacteria when numerous are generally arranged in clumps or groups, and these often form a very large part of the epithelioid areas.

The lesions in the lymphatic glands have a similar histology. They may be present in either the cortex or the medulla, but they are not in the anatomical sense like those of tubercles. A small number of giant cells may be present.

The duration of the disease varies from a few weeks to several months and often a year or more. It seems to be fatal in most cases.

Diagnosis. Johne's disease is to be diagnosed by the symptoms, lesions, etiology and specific reactions. The diarrhea with the loss of flesh and a generally good appetite is very suggestive of this disease. The positive diagnosis, however, requires an examination of the lesions or the finding of the specific organism.

Etiology. Meyer found that the bacterioscopic examination of the feces and rectal scrapings is of diagnostic value in only about 40 per per cent. of cases in the advanced stages of the disease. M'Fadyean, Sheather and Edwards diagnosed the disease in 20 per cent. by this method. In a total of 15 cases, they made a positive diagnosis in 5 from rectal scrapings. The difficulty with this method is not restricted to the small number of individuals in which it is applicable. It is necessary to differentiate the acid-fast bacteria found from those of tuberculosis and purely saprophytic forms that may be present. The differentiation from tubercle bacteria can be made by guinea-pig inoculation. With the development of culture methods the identification of the organism may be simplified. The presence of large numbers of acid-fast bacteria in the feces or in the mucous membrane of the intestine is very significant especially if symptoms are present.

Tuberculin reaction. O. Bang called attention to the value of tuberculin made with the avian variety of tubercle bacteria in diagnosing

this affection. He reported very good results and recommended its subcutaneous use as a diagnostic agent. The results from the ophthalmic application were not satisfactory. From 4 to 8 cc. of avian tuberculin is used. The reaction is not so pronounced as that of tuberculin in tuberculous cattle. Our experience has not been satisfactory in the cases tested although in a few of them a distinct temperature curve was obtained. Meyer states that we are not justified in recommending the avian tuberculin as a diagnostic agent for paratuberculosis, and that the ophthalmic test, as recommended by Howe, is also unreliable. Holth, also Twort and Ingram, have prepared a tuberculin (paratuberculin) from cultures of the Johne's bacterium, which they report to be successful. They obtained maximum temperatures in three cases of 105°, 106.1° and 104.8° F. respectively.

Sera tests. Both the agglutination and complement fixation methods have been tried. The results are not satisfactory and the experimental work with these methods is too meager to warrant their recommendation at present.

The diagnosis depends upon a combination of the physical conditions, histological and bacteriological findings together with the reactions obtained with the specially prepared tuberculin. There is no reaction in animals affected with this disease to tuberculin prepared from the human or bovine varieties of tubercle bacteria.

Johne's disease is to be differentiated from tuberculosis and parasitic enteritis. Miessner and Trapp have mentioned a case of sarcomatosis of the bowel in which the animal gave every symptom of Johne's disease. Twort and Ingram point out coccidioses as a condition to be differentiated. It is accomplished by finding the coccidium cyst in the feces. The diagnosis is made on the accumulation of evidence of Johne's disease and the absence of causes to otherwise explain the physical condition. It is presumed that ere long more definite methods of diagnosis will be formulated.

Prevention. M'Fadyean states that in all the cases which have come under his observation there was a history of similar cases on the farm, in several instances extending back over a period of many years. During the advanced stages of the disease large numbers of the bacteria are voided with the feces, and under all ordinary circumstances there are ample opportunities for infection from this source. In this way both pasture and other materials as well as drinking water may become seriously contaminated. At present there is no

definite knowledge of the resistance of the specific bacteria outside the animal body, or the length of time that a contaminated pasture may be dangerous.

In the present state of knowledge, the formulation of a method for prevention is an extremely difficult task on farms where the disease has existed for a number of years. The isolation or destruction of diseased and suspected animals should be practiced. The feces passed by diseased or suspected animals ought to be burned. Healthy cattle should be kept off from pastures in which such animals have run. As the time during which the bacteria remain alive outside of the body is not known, it is impossible to indicate the period during which infected pastures are dangerous to other cattle. It is stated by O. Bang that cattle imported from countries known to be affected with this disease should be tested with avian tuberculin to detect the infected individuals.

REFERENCES

1. BANG. Chronische pseudotuberculöse Darmentzündung beim Rinde. *Berliner Tierärztliche Wochenschrift*, 1906, S. 759.

2. BANG. Johne's Disease (Enteritis chronica paratuberculosa bovis). *Proceedings Tenth International Veterinary Congress*, London, 1914.

3. BEEBE. Johne's disease in cattle. *American Veterinary Review*, Vol. XXXIII (1908), p. 708.

4. JOHNE AND FROTHINGHAM. Ein eigenthümlicher Fall von Tuberkulose beim Rind. *Zeitschrift für Thiermedicin*, Bd. XXI (1895), S. 438.

5. LIENAUX AND ECKHOUT. Contribution à l' étude d'une entérite tuberculeuse spéciale et de la diarrhée chronique du boeuf. *Annales de Médécine Vetérinaire*, Vol. LIV (1905), p. 65.

6. M'FADYEAN. Johne's disease; a chronic bacterial enteritis of cattle. *Jour. of Compar. Path. and Therap.*, Vol. XX (1907), p. 48.

7. M'FADYEAN, SHEATHER AND EDWARDS. Johne's Disease: (1) records of examinations of natural cases; (2) cultural characters of the bacilli. *Jour. of Comp. Path. and Therap.*, Vol. XXV (1912), p. 217.

8. MARKUS. Eine spezifische Darmentzündung des Rindes wahrscheinlich tuberkulöser Natur., *Zeitschr. für Tiermedizin*, Bd. VIII (1904), S. 68.

9. MATHIS. Lésions de la diarrhée chronique des bovidés, *Bulletin de la Soc. des Scien. Vét. de Lyon*, 1906.

10. MEYER. The specific paratuberculous enteritis of cattle in America. *Proceedings of the Am. Vet. Med. Asso.*, 1913, p. 877.

11. MIESSNER. Der infektiöse Darmkatarrh des Rindes, *Ninth International Veterinary Congress*, Hague, 1909.

12. MIESSNER AND TRAPP. Der Chronische Infektiöse Darmkatarrh des Rindes, *Mitt. d. Kaiser Wilh. Institut für Landwirtsch. in Bromberg*, Bd. II, (1910), S. 219.

13. SHEATHER. Johne's Disease. *Tenth International Veterinary Congress*. London, 1914.

14. STOCKMAN. Johne's Disease in Sheep, *Jour. of Comp. Path. and Therap.*, Vol. XXIV (1911), p. 66.

15. TWORT. The agglutination and complement-fixation reactions in animals experimentally inoculated with Johne's bacillus, etc., *Centralblatt für Bakt. Originale,* Bd. LXVI (1912), S. 316.

16. TWORT AND CRAIG. The Pathogenicity of Johne's bacillus compared with that of Other Acid-Fast Bacilli for some of the laboratory animals, *Centralbl. für Bakt. Originale,* Bd. LXXVIII (1913), S. 455.

17. TWORT AND INGRAM. A Method for isolating and cultivating the *Mycobacterium enteritidis chronicæ pseudotuberculosæ bovis Johne,* and some Experiments on the Preparation of a Diagnostic Vaccine for Pseudotuberculous Enteritis of Bovines, *Proceedings Royal Society,* B., Vol. LXXXIV (1912).

18. TWORT and INGRAM. Further Experiments with the *Mycobacterium enteritidis chronicæ pseudotuberculosæ bovis Johne,* and with Vaccines prepared from this Micro-Organism, *Centralblatt für Bakt.,* Bd. LXVII (1912), S. 126.

19. TWORT AND INGRAM. Johne's Disease. London, 1913.

INFECTIOUS ABORTION IN CATTLE

Synonyms. Cattle abortion; contagious abortion; "picking," "slipping," "casting."

Characterization. The disease known as infectious abortion consists in the expulsion of the immature fetus, usually before it has sufficiently developed to live after birth, by a large proportion of pregnant animals that are kept together. Usually the abortion occurs in cattle between the fifth and eighth month of gestation. It is characterized by certain morbid changes in the uterine mucosa and fetal membranes. It usually affects the young cows. Williams defines infectious abortion in cattle as a widespread and highly destructive chronic infection having its chief seat in the genital organs of cattle and expressing itself by a variety of symptoms, four of the most prominent of which are sterility, expulsion of fetus, premature birth and metritis with retained fetal membranes.

Other domesticated animals such as mares, swine and sheep suffer from infectious abortion but the cause seems to be different. Surface has described an epizoötic in guinea pigs caused by the Bang organism.

History. Abortion in epizoötic form has been recorded from very early times. Mascal, in 1859, gives directions in his work on cattle, "How to keep cows which are great bellied with calf." In Germany the disease seems to have existed in a somewhat severe form in the latter part of the eighteenth century.

A number of important scientific investigations have been made into its nature in France by Nocard, 1885; in Great Britain, by a committee appointed in 1886 by the Highland Agricultural Society of Scotland consisting of Drs. Woodhead, Aitken, M'Fadyean and

Campbell; and more recently by another committee headed by Sir John M'Fadyean; in Denmark, by Bang and Stribolt; by the U. S. Bureau of Animal Industry; by the New York State Veterinary College; and by a number of the State Experiment Stations.

Geographical distribution. This affection exists with more or less constancy in all countries where cattle raising is an industry. It seems to be world wide.

Etiology. A considerable number of bacteria have been found to be associated with abortion. Bang and Stribolt found an anærobic bacterium which they believed to be the specific cause of the disease. They found it in a number of cases and reported positive results from inoculation experiments. Its cultivation required a medium composed of agar, gelatin and blood serum. Bang considered this bacillus to be a pathogenic organism which has no saprophytic existence. The fact that when he injected it into the blood stream it seemed to grow only in the pregnant uterus and in the fetus was considered evidence of this. The fact seems to be clearly established that the specific cause is the organism isolated by Bang and Stribolt. Its morphology and cultural characters as well as the technique for its cultivation have been carefully studied by Bang and Stribolt, M'Fadyean and Stockman, MacNeal and Kerr, Nowak, Zwick and Wedemann, Holth, Fabyan, Schroeder, Cotton, Mohler and Traum and others. While slight differences appear in the descriptions of the organism, they are not greater than might be expected, or than actually exist with different varieties of many species of bacteria. The organism can be cultivated on a number of media, such as the agar-gelatin-serum of Bang and Stribolt, and on potato, glycerin agar and certain liquid media, such as glycerin-broth-serum, glycerin bouillon, milk and others. It has been shown that this organism can be cultivated in a variety of artificial media, that it is not an obligatory anærobe and that after a few generations it can usually be cultivated under ærobic conditions. Its cultural characters seem to be quite constant when it becomes adapted to artificial media.

Its natural distribution or habitat has not been fully determined. As it is a pathogenic organism the question is whether or not it exists in nature outside of the infected animals. The observations, as recorded in the literature, indicate that it is able to live for a considerable length of time on litter or other articles contaminated with it. Thus far is does not seem to have been isolated from any source

where its presence could not be accounted for on the theory that it came from an infected animal.*

The natural channels of infection in dairy cattle seem to be either through the *genital* tract or the *digestive* tract. Authors are not agreed as to which is the more common. Williams considers the genital tract the principal, if not the only, channel of infection. He finds, however, that calves fed on infected milk abort in their first pregnancy.

The period of incubation according to Bang is about 10 weeks.

Morbid anatomy. The investigations and clinical observations reported in this country have not ca led attention to any definite

---

*The finding of the bacterium of abortion in milk has occasioned considerable alarm as to its possible effect upon calves. In their earlier work, M'Fadyean and Stockman called attention to the possible "upkeep of the abortion bacillus in the milk of infected cows." Schrœder and Cotton have found it in the milk of a large number of cattle. Fabyan and Smith found it in the milk of cows that had aborted. Cotton has published interesting facts on the presence of the organism in the udders of three cows at the Bureau of Animal Industry Experiment Station that were not known to have aborted. It should be stated, however, that these animals were in a herd in which abortion had occurred.

The resistance of the Bang organism to disinfectants and heat has been studied by several. Zwick and Wedemann found that it was killed by a 2.5 per cent. kresol solution in 2.5 minutes when a one day old culture was used and in 15 minutes when a three day culture was employed and 40 minutes with a seven day culture. They found a 3 per cent. solution of carbolic acid, or a 2.5 per cent. dilution of formalin able to destroy it in from ten to twenty minutes. Old cultures were more resistant than those that were but two days old. Preisz found that it was destroyed by a 1-1000 corrosive sublimate solution in 15 seconds.

Rich found in laboratory tests that one part of corrosive sublimate in 10,000 parts of water destroys the organism in from one to three minutes. For stable disinfection, he recommends a dilution of 1 to 1,000. He found that a solution of lysol, 1 part in 1,000, killed the organism in from three to five minutes. Liquor cresolis compositus was quite as effective as the lysol solution.

Zwick and Wedemann found that a temperature of 55° C. in a water bath would kill the organisms in from twenty-five to thirty minutes. A 60° C. temperature destroyed them in from ten to fifteen minutes. M'Fadyean and Stockman found that a temperature of from 55° to 60° C. was fatal in ten minutes. Fabyan found the thermal death point to be 59° C. for ten minutes.

The virulence of the Bang organism varies in a marked degree in different cultures as indicated by their effect on pregnant cattle and guinea pigs. The inoculation of pregnant cattle either intravenously or by feeding produces abortion in certain cases after a variable length of time. A fibrino-purulent inflammation of the fetal and maternal placenta has been reported. A like inflammation has been observed in the stomach and intestines of aborted fetuses.

In guinea pigs the lesions resemble somewhat closely on microscopic examination those produced by the bacterium of tuberculosis not only in their general appearance but also in their distribution in the spleen, liver and lymph nodes. They usually appear between the third and sixth week, the acute changes extending over a period of ten to twenty weeks after which reparation processes begin. The disease is accompanied by fever and tends toward a final recovery although the animal may die from rupture of the spleen, emaciation and exhaustion. All the tissues may be attacked with the exception of the muscles. Jensen traced the cause of white scours in calves to the abortion organism in one case out of 208.

lesions in the uterine mucosa or fetal membranes that differ materially from those found in the affected uterus by Bang. Bang's description of the lesions is as follows:

"The external surface of the uterus was normal. The os uteri was firm'y closed and the cervical canal was filled with the normal thick mucus. After disinfection of the serous covering of the uterus by burning, I made a section through the uterine walls: when the mucous membrane was divided we saw *between that and the fetal envelope an abundant odorless exudate*—a dirty yellow, somewhat thin, pultaceous material of a slimy, somewhat lumpy character. At some places where the fluid constituents had run out the exudate was of a semi-solid nature; its reaction was alkaline. When it was allowed to stand in a glass it separated into two strata, namely, superiorly a reddish-yellow cloudy serum, and at the bottom a thick greyish-yellow precipitate.

"On cutting through the chorion we saw under that a thin, clear, apparently gelatinous substance, with very fine membranes running through it: closer examination showed that this was the fine connective tissue lying between the chorion and allantois, saturated with edematous exudates. This was present over the entire extent of the fetal envelopes and formed a layer one and one-half centimeters thick. The allantoic fluid was natural in appearance, thin, yellowish, and containing fine flocculi. Nothing abnormal was observable in connection with the amniotic fluid. The umbilical cord was edematous. The size of the fetus and degree of development of the hair on it indicated an age of seven months. It was quite fresh and on section it showed no striking alteration. The pericardium contained a little reddish fluid; the intestinal mucous membrane was, perhaps, rather redder than ordinary; the spleen was in very slight degree swollen and the blood was fluid.

"The examination of a cover-glass preparation made from the yellowish exudate and stained with Lœffler's methylene blue immediately showed the presence of a very small bacterium, apparently in pure culture. This organism was present in very considerable numbers; many individuals lay free, but most striking were the large dense clumps of bacteria. Closer examination showed that these heaps were included within cells whose bodies were often in this way greatly distended. Sometimes the body of the cell was very indistinct, but as a rule one could still recognize external to the heap a part of the cell body and often also the cell nucleus. Not seldom the cell body had assumed a peculiar homogeneous appearance.

"In the dense heaps the bacteria mostly had the appearance of cocci, but some of the free-lying individuals were of a longer shape, and these were at first regarded as short oval structures; closer examination, however, under very high magnification showed that we had in fact to deal with a small bacillus whose body contained one, two, or more rarely three, roundish or elongated granules. These granules most readily took up the stain. The length of the bacillus is very variable; the longest examples are about as long as tubercle bacilli. . . . . The granules may occur a little distance from the extremities but frequently they are at the end of the bacillus. They stain with the ordinary aniline dyes, but not by the method of Gram The bacilli are non-motile. In the subchorial œdema I found no bacteria. In the heart blood of the fœtus there were a few, and in the intestinal contents there were many staining granules; but it was not possible to say with certainty whether these were bacteria or not."

Bang states that the discovery of this particular organism indicates that epizoötic abortion ought to be regarded as a specific uterine catarrh, determined by a definite species of bacteria. While the uterine mucous membrane was not strikingly altered he affirms that chronic catarrh is not necessarily associated with striking anatomical alterations. He states further, "In my opinion the very abundant exudate which contained a quantity of shed epithelial cells, pus cells and detritus must necessarily have been furnished by the uterine mucous membrane and not by the thin chorion, and consequently the disease must be regarded as a uterine catarrh."

Williams recognizes as a part of the morbid anatomy of infectious abortion retained afterbirth which he considers to be uniformly metritis or "placentitis" or "cotyledonitis." That is, retained afterbirth is due primarily to the presence of an infection within the uterine cavity which causes an inflammation, especially of the placentæ, cotyledons or "buttons," inducing swelling of these tissues and causing the tufts of the afterbirth, which extend deeply into the cotyledons, to become caught and the afterbirth retained. This infectious inflammation must exist in the uterus prior to abortion, premature birth, or birth at full term, or retained afterbirth cannot and does not occur. If the uterus is normal at time of birth, the afterbirth is expelled so quickly (one to two hours) that no cause of retention can arise during or after the birth act. The cause is of earlier date and exists in the uterine cavity weeks, perhaps months, before the expulsion of the fetus.

In the few recorded cases where cows with retained afterbirth have been promptly slaughtered, there has been found in the uterus, between it and the afterbirth, the "exudate of contagious abortion" containing the Bang organism. It has not been shown that any other organism may exist in the pregnant uterus for an extended period of time. No other cause for retained afterbirth has been demonstrated.

Diagnosis. With infectious abortion, as with tuberculosis, the animals may be infected and yet give no physical evidence of that fact. The premonitory symptoms are usually brief in duration and there are no lesions in evidence until the abortion occurs. It is impossible, therefore, to pick out by means of physical examination cows that are infected. The specific organism cannot be detected in the uterine discharge until just before and following the expulsion of the fetus. The diagnosis, therefore, must be made from some specific reaction or test of which four have been described, namely: abortin, precipitation, complement fixation and agglutination.

Abortin is analogous to tuberculin, made from bouillon cultures of the abortion organism. It resembles tuberculin in the method of its preparation and use. It has not been satisfactory. The precipitation test is likewise unsatisfactory. The complement fixation was applied first by M'Fadyean who was enthusiastic in its favor. The time required to make the test and the dangers of error, especially where one is not making it constantly, are so great that it is not entirely satisfactory as a practical diagnostic method. The agglutination test is very satisfactory. A positive reaction to complement-fixation or agglutination test does not determine whether the animal has aborted or is going to abort, but it does indicate that the animal is at present or has been infected with *Bacterium abortionis*.

Prevention. Dairyman have come to believe that if they keep animals that abort away from their sound cattle the trouble does not appear among them. As it affects young cows, it is the practice in some places to keep the young animals separated from the others until they have become free from the infection or at least until they have passed the period when it is apt to occur after which they are admitted to the herd of older cows with impunity.

Williams has made the following statement concerning the control of abortion:

"We are in great need of a logical and comprehensive plan for combating abortion. Up to the present time, little definite progress has

been made. It appears, however, that enough is known of the fundamental character of the disease, the source of infection and the avenue of invasion, that a comparatively safe plan may be formulated. Recent investigations apparently show that there are two great points of danger—contaminated milk fed to the calf, causing an infection through the alimentary tract, which eventually finds its way to the genital organs, and copulation.

"The quarantining of cows which have aborted has failed. Admittedly, abortion is only one symptom of the presence of the infection, and we cannot control an infection by quarantine upon the basis of a single symptom. If we attempt a more delicate diagnosis as a basis for quarantine, we find it impossible at present to draw a clear line of demarcation between the infected and the non-infected, and the number of infected is so great that quarantine is rendered impracticable. Moreover, it appears now that an animal which has aborted or suffered other disaster is not a great direct menace to neighboring pregnant animals.

"Stable disinfection has been advised for controlling abortion, but it is to be remembered that the infection which is doing harm is within the animal. The gutter may be disinfected thoroughly and an infected animal standing over it may reinfect it thoroughly within an hour.

"The administration of disinfectants, such as carbolic acid and methylene blue, has not proven efficient. The principal area in which active harm is being done is in the utero-chorionic space of the pregnant cow, into which sealed cavity, so far as we are aware, no drug is carried by the circulatory system, and consequently no disinfectant action can be brought about.

"In the light of our present knowledge, the only constructive program that we can offer is to guard the new-born calf against infection through the milk and to guard the animal of breeding age against infection by copulation.

"When a cow is well advanced in pregnancy, generally at about 270 days, she should be given a thorough bath with warm water and soap, lathering the skin until it is thoroughly clean. The soap should then be washed away with a reliable disinfecting solution. The cow should then be placed in a thoroughly clean stall, and in order to maintain the highest state of cleanliness her tail, thighs and udder should be carefully disinfected once a day and the vagina douched daily with a 0.25 per cent. Lugol's solution or with some other equally good disinfecting solution.

"When the calf is born, it should be taken immediately from the cow and not permitted to suck. It should be rubbed dry under antiseptic or aseptic precautions and placed in a clean isolated stall.

"In feeding the new-born calf, select the milk of a healthy cow which has calved promptly and naturally in thirty minutes or less, has expelled her afterbirth quickly and naturally within two hours, and has no discharge from the uterus. The milk from cows which have aborted or have had retained afterbirth should not be used. The milk for the calf should be drawn under strict application of the rules for the production of certified milk and fed to the calf from a sterile pail. When the calf has reached eight to ten days of age, the milk fed may come from any source if it is boiled in a water bath.

"The control of the infection by copulation must be based upon thorough disinfection of all available genital organs. The systematic douching of the sheath of the bull and the vagina of the cow is highly valuable. It is of still greater value to disinfect thoroughly the uterine cavity of a cow which has aborted or has had retained afterbirth or metritis with a discharge. In such cases, the disinfection should be prompt and vigorous and the uterus should be brought to its natural state as early as possible.

"If the disease is to be suppressed to the greatest possible degree, it is desirable to establish as a rule of practice the douching of the uteri of all cows before breeding, beginning three to four weeks prior to the time it is desired to breed and repeating at least once a week up to within a day or two of the time when it is desired to breed. The uterus may be douched with two per cent. Lugol's solution or its equivalent. Just prior to breeding, the vagina may be douched with a 0.25 per cent. Lugol's solution or with a normal salt solution."

The immunization of cattle against infectious abortion has been the subject of much experimental work. Observations pointed to an acquired immunity after from one to three abortions. Williams attributes the phenomenon that older cows suffer less than young ones from abortion to a natural age immunity. M'Fadyean and Stockman were unable to secure any evidence that natural immunity to the abortion organism is possessed by any individuals of the bovine species. They carried out several experiments for the purpose of producing immunity. Their results were encouraging and with a few animals they seemed to be positive.

Schreiber reports good results in the production of immunity with an extract of the Bang organism. He points out also the necessity for great care in cleanliness and thorough disinfection.

Mohler and Traum found that cows rarely abort more than twice or three times, after which they develop a tolerance or immunity to the infection.

Bang carried out many experiments for immunizing cattle against abortion with both living and killed cultures. His results were in part favorable and in part unsatisfactory. He considers the best method to combat the disease is isolation and disinfection.

Olaf Bang reports a large number of immunizing experiments with various serums and vaccines made by veterinarians, in Denmark, not reported in our literature. The results were variable, some being favorable and others not. The use of vaccines has thus far been unsatisfactory.

In Norway infectious abortion is scheduled under the so-called "milder contagious diseases." The owner is obliged to report when such a disease appears in his herd, and he is not allowed to bring such animals to fairs or cattle shows.

•

REFERENCES

1. Ascoli. Uber die Reinzüchtung des Bangschen Bacillus. *Zeit. f. hyg. u. infek:*, Bd. LXXV (1913), S. 172.

2. Bail. Uber die Agglutinationswirkung des normalen Rinderserums. *Cent. f. bakt. paras. u. infek.*. Bd. LI (1909), S. 170.

3. Bang, B. Das seuchenhafte Verwerfen der Rinder. *Arch. f. wiss. u. prak. Tier.*, Bd. XXXIII (1907), S. 312.

4. Bang. The etiology of epizoötic abortion. *The Jour. of Comp. Path. and Therap.*, Vol. X (1897), p. 125.

5. Bang. Infectious abortion in cattle. *The Jour. of Comp. Path. and Therap.*, Vol. XIX (1906), p. 191.

6. Belfanti. Uber den Wert einiger neuer Diagnosemittel beim infektiösen Abortus. *Zeit. f. Infek. para. Kran. u. Hyg. d. Haust.*, Bd. XII (1912), S. 1.

7. Chester. *7th Annual report, Del. Agric. Exp. Station.*

8. Cotton. The persistence of the bacillus of infectious abortion in the tissues of animals. *Amer. Vet. Rev.*, Vol. XLIV (1913), p. 307.

9. Dalrymple. *Bulletin No. 10, 2d Series, La. Agr. Exp. Station, 1891.*

10. Desmond. An outbreak of epizoötic abortion in cattle. *Amer. Vet. Rev.*, Vol. XLIII (1913), p. 604.

11. Fabyan. A contribution to the pathogenesis of B. abortus, Bang. *Jour. of Med. Research*, Vol. XXI (1912), p. 441.

12. Fabyan. The persistence of B. abortus, Bang, in the tissues of inoculated animals. *Jour. of Med. Research*, Vol. XXVIII (1913), p. 81.

13. Fabyan. A note on the presence of B. Abortus in cow's milk. *Jour. of Med. Research*, Vol. XXVIII (1913), p. 85.

14. GARDINER. Contagious abortion in Montana. *Bulletin No. 49, Mont. Agr. Exp. Station*, 1903.

15. GILTNER. Infectious abortion in cattle. *Amer. Vet. Rev.*, Vol. XLII (1912–13), p. 145.

16. GILTNER. Infectious abortion and sterility in cattle. *Tech. Bull. No. 14, Mich. Ag. Coll.* (1912).

17. GOOD. The etiology of infectious abortion in live stock. *Amer. Vet. Rev.*, Vol. XL (1911–12), p. 473.

18. HADLEY AND BEACH. Results with the complement fixation test in the diagnosis of contagious abortion of cattle. *Amer. Vet. Rev.*, Vol. XLII (1912–13), p. 43.

19. HADLEY AND BEACH. The diagnosis of contagious abortion in cattle by means of the complement fixation test. *Research Bull. No. 24, Univ. Wis. Ag. Ex. Sta.*

20. HESSE. Der Bakterien-Extrakt gegen seuchenhaftes Verwerfen der Deutschen. Schütz und Heil-Serum Gesellschaft Berlin. *Berl. tier. Woch.*, Jr. 26 (1910), S. 280.

21. HOLTH. Die Agglutination und die Komplementbindungsmethode in der Diagnose des seuchenhaften Verwerfens der Kühe. *Berlin. tier. Woch.*, Jr. 25 (1909) S. 686.

22. HOLTH. Untersuchungen über die Biologie des Abortusbacillus und die Immunitätverhältnisse des infektiösen Abortus der Rinder. *Zeit. f. infek. para. Kran. u. Hyg. d. Haust.*, Bd. X (1911), S. 207.

23. LARSON. The complement fixation reaction in the diagnosis of contagious abortion of cattle. *Jour. of Infec. Dis.*, Vol. X (1912), p. 178.

24. LAW. Contagious abortion in cows. *Report of the N. Y. State Commissioner of Agriculture*, 1897.

25. MacNAL AND KERR. Bacillus Abortus of Bang, the cause of contagious abortion in cattle. *Jour. of Infec. Dise.*, Vol. VII (1910), p. 469.

26. MacNEAL AND MUMFORD. Contagious abortion of cows. *Bull. No. 152 (1911), University of Illinois, Ag. Ex. Sta.*

27. MELVIN. The bacterium of contagious abortion of cattle demonstrated to occur in milk. *Vet. Jour.*, Vol. N. S. XIX (1912), p. 526.

28. MELVIN and others. Infectious abortion of cattle. B. A. I. Cir. 216.

29. M'FADYEAN AND STOCKMAN. The report of the departmental committee on epizoötic abortion (1909).

30. M'FADYEAN AND STOCKMAN. The agglutination test in the diagnosis of bovine contagious abortion. *Jour. of Comp. Path. a. Therap.*, Vol. XXV (1912), p. 22.

31. M'FADYEAN, SHEATHER AND MINETT. Researches regarding epizoötic abortion of cattle. *Jour. of Comp. Path. a. Therap.*, Vol. XXVI (1913), p. 142.

32. MOHLER AND TRAUM. Infectious abortion of cattle. *Ann. Rept. B. A. I.*, 1911, p. 147.

33. MEYER AND HARDENBURGH. On the value of the "Abortin" as a diagnostic agent for infectious abortion. *Jour. of Infec. Dis.*, Vol. XIII (1913), p. 351.

34. MOORE AND FITCH. A study of infectious abortion in cattle. *Rep. of the N. Y. S. Vet. College*, (1912–13), p. 82.

35. NOCARD. Recherches sur l' avortment epizootique des vaches. *Recueil de Méd. Vétér.*, Vol. III (1886), p. 669.

36. NOWAK. Le bacille de Bang et sa biologie. *Ann. de l'Inst. Past.*, Vol. XXII (1908), p. 541.

37. NEUSCH-FLAWYL. Zum infektiösen Abortus des Rindes. *Schw. arch. f. tier.*, Bd. L (1908), S. 323.

38. PALMER. Pathology and etiology of epizoötic abortion. *Amer. Vet. Rev.* Vol. XXXVI (1909–10), p. 360.

39. PEKAR. Epizootisches Verwerfen. *Berl. Tier. Woch.*, Jr. 25 (1909), S. 277.

40. PEMBERTHY. Epizoötic abortion. *Jour. of Comp. Path. and Therap.*, Vol. VIII (1895), p. 95. (A good summary of the history and a very complete list of references to the earlier literature is given.)

41. PIORKOWSKI. Lymphe gegen seuchenhaftes Verwerfen. *Berlin. tier. Woch.*, Jr. 26 (1910), S. 279.

42. PREISZ. Der Bacillus des seuchenhaften Verwerfens. *Cent. f. Bakt. Paras. u. Infek.*, 1 Abt., Bd. XXXIII, S. 190.

43. RICH. Methylene blue. A remedy for infectious abortion. *Bull. No. 174, Vermont Ag. Ex. Sta.*, 1913.

44. REYNOLDS. Infectious abortion. Press Bull. No. 38 (1912), University of Minnesota, Ag. Ex. Sta.

45. SCHREIBER. Studien über den infektiösen Abortus der Rinder und seine Bekämpfung mittels Impfung. *Deut. Tier. Wochs.*, 1913, S. 33.

46. SCHROEDER AND COTTON. An undescribed pathogenic bacterium in milk. *Proc. Amer. Vet. Med. Assn.*, 1911, p. 442.

47. SCHROEDER AND COTTON. The bacillus of contagious abortion found in milk. *Ann. Rept. B. A. I.*, 1911, p. 139.

48. SMITH U. FABYAN. Uber die pathogene Wirkung des Bacillus Abortus Bang. *Cent. f. Bakt. Paras. u. Infek.*, Bd. LXI (1911–12), S. 549.

49. SURFACE. The diagnosis of infectious abortion in cattle. *Bull. No. 166, Ky. Ag. Ex. Sta.*, 1912.

51. SURFACE. The inhibiting effect of excess cow serum in complement fixation with infectious abortion. *Zeit, f. Immu. u. Exp. Ther.*, Bd. XVII (1913), S. 487.

52. SURFACE. The artificial inoculation of cattle with the bacillus of contagious abortion. *Amer. Vet. Rev.*, Vol. XLIII (1913), p. 624.

53. SZYMANOWSKI. Uber die Anwendung der Prözipitationsmethode zur Diagnostik des ansteckenden Verkalbens. *Arbeit aus dem Kaiserlichen Gesundheitsamte*, Bd. XLIII (1912), S. 145.

54. TAYLOR. The internal use of carbolic acid for the prevention of contagious abortion in cattle. Bull. No. 90, Mont. Ag. Ex. Sta.

55. VAN ES. Abortion in cattle. *Bulletin No. 54, North Dakota Agricultural College and Experiment Station*, 1902.

56. WALL. Uber die Feststellung des seuchenhaften Abortus beim Rinde durch Agglutination und Komplementbindung. *Zeit. f. infek. para. Kran. u. Hyg. d. Haus.*, Bd. X (1911), S. 23.

57. WILLIAMS. Abortion and sterility in cattle. *Rept. N. Y. State Veterinary College*, 1911–12, p.79.

58. WILLIAMS. Contagious abortion. *Rept. Sixteenth Annual Meeting United States Live Stock Sanitary Association*, 1912, p. 111.

59. WILLIAMS. Suggestions for the repression of abortion, sterility and mammitis in cows and of white scours in calves. *Report of the N. Y. State Vet. College*, 1913–14, p. 163.

60. WILSON. Contagious granular vaginitis in cattle and its relation to sterility and abortion. *Vet. Jour.*, Vol. XVII N. S. (1911), p. 460.

61. WOODHEAD AND OTHERS. First Report of the Committee of the Highland and Agricultural Society of Scotland. Transactions of the Society for 1887. Second Report of Committee. *Jour. of Comp. Path. and Therap.*, Vol. XI (1889), p. 97.

62. ZWICK U. WEDEMANN. Biologische Untersuchungen über den Abortus-Bazillus. *Arbeiten aus dem Kaiserlichen Gesundheitsamte*, Bd. XLIII (1912), S. 130.

63. ZWICK U. ZELLER. Uber den infektiösen Abortus des Rindes. *Arbeiten aus dem Kaiserlichen Gesundheitsamte*, Bd. XLIII (1912), S. 1.

OVINE CASEOUS LYMPH-ADENITIS (PSEUDO-TUBERCULOSIS IN SHEEP)

Synonyms. Pseudo-tuberculosis of sheep; caseous lymph adenitis; cheesy broncho-pneumonia.

Characterization. Caseous lymph-adenitis is a disease of adult sheep which until recently was designated as pseudo-tuberculosis. It has been characterized by an enlargement of one or more lymphatic glands, which contain foci of a greenish-yellow, caseous or purulent substance. It is rarely found in young animals. The mortality is very low, due perhaps to the fact that the sheep are slaughtered before the disease runs its course. It does not occur in epizoötic form although it is more prevalent in certain localities than in others.

History. The name "ovine caseous lymph-adenitis" was proposed by Nörgaard and Mohler in 1899. These writers found the lesions and the accompanying microörganism to correspond with those described by Preisz and Guinard in 1891 as pseudo-tuberculosis. The bacterium was fully described by Preisz in 1894. It appears that at least many of the cases of lymphatic gland enlargement in sheep heretofore called pseudo-tuberculosis belong to this disease. Gilruth prefers the name pseudo-tuberculosis. Cherry and Bull describe it as caseous lymphatic glands and Sivori as caseous broncho-pneumonia, the bacterium of Preisz being found as the probable cause in each case.

Geographical distribution. In the United States this disease is quite common in certain districts in the western and southwestern states. It exists in South America, New Zealand, Australia and Europe.

Sivori found that 10 per cent. of the old sheep killed in Buenos Ayres were affected. The prevalence of the disease in the United States is indicated by the reports of the federal meat inspectors, which show that of 16,000,000 sheep slaughtered in Chicago, Kansas City and South Omaha 3,236 were condemned for caseous lymph-adenitis or lesions which might be confounded with it. It is reported by an inspector from Los Angeles that of 950 sheep coming from a certain district, 82 were suffering from lymph-adenitis.

Etiology. Caseous lymph-adenitis is caused by a specific microörganism first described by Preisz as the bacillus of pseudo-tuberculosis. Its description shows it to vary in size to such a degree that its polymorphism is said to be characteristic. It is non-motile and hence belongs to the genus *Bacterium*. It is aërobic, facultative anaërobic,

and does not produce spores.   It develops readily on agar when this medium is inoculated from the caseous material from the affected glands.   It is pathogenic for mice, guinea pigs, rabbits and swine. The organism isolated by Gilruth seems to have been more virulent than the one described by Nörgaard and Mohler.   According to Carri and Bigoteau it produces a strong toxin.

Nocard and Glasser found that lambs could be infected by both inhalation and ingestion of the organism.   The natural mode of infection is not known but it is believed that the channels of infection are the digestive and respiratory tracts and the umbilicus.

Symptoms.   In the majority of cases no symptoms of any import- ance are observed in the affected animals during life.   The course

FIG. 47.   THE LEG OF A RABBIT SHOWING ENLARGED GLANDS AFTER INOCULATION WITH THE BACTERIUM OF PREISZ (*Nörgaard and Mohler*).

of the disease is that of a chronic affection and the pathological changes develop so slowly that no general or local interference with the health is observed in sheep that are bred and raised for mutton and are marketed before they are two years old.   Only in older sheep does it advance to a degree to be clinically recognizable without the aid of manipulation.   The superficial glands most often involved are the precrural and the subscapular which in some cases become so enlarged as to interfere with locomotion.   The animals thus affected may appear to be in perfect health although they may show a certain degree of unthriftiness or emaciation.   In advanced cases the lesions may be disseminated through metastasis to the principal organs of the body.   In such cases the disease may assume the appearance of chronic broncho-pneumonia or pleurisy, with occasional cough, slight dyspnea, emaciation and anemia.   The course of the disease is very slow and fatal results are rare.

Morbid anatomy.   The principal lesions are confined, according to the various descriptions, to the lymphatic glands.   In many cases

only a single gland is affected. The relative frequency with which the various glands become the seat of the lesions may be given as follows: prescapular, precrural, superficial inguinal, bronchial, mediastinal, sublumbar, deep inguinal and scrotal. Rarely the suprasternal and mesenteric glands are affected. Sivori mentions the mesenteric glands among those frequently affected. He fails, however, to mention the mesenteric glands as the seat of lesions in the detailed description of twelve typical cases of caseous broncho-pneumonia caused by the bacillus of Preisz.
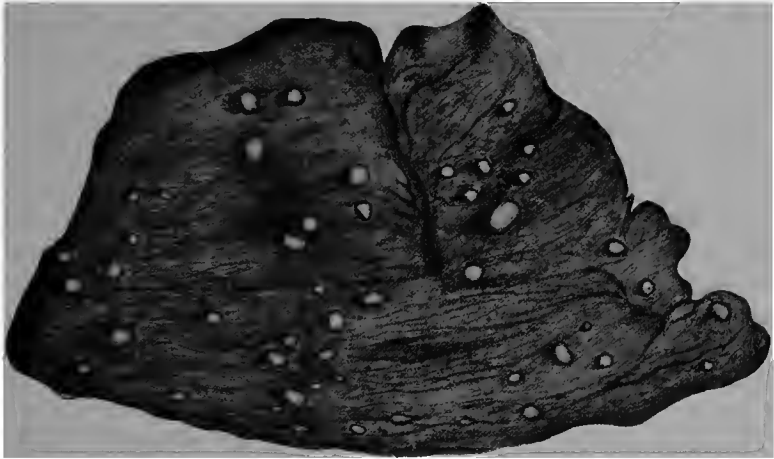
FIG. 48. LUNG OF SHEEP STUDDED WITH NODULES (*Gilruth*).

When first invaded by the bacterium, the adenoid tissue becomes hyperplastic and the gland enlarges to several times its original size. On section the surface is found to be edematous but otherwise normal in appearance. This is followed by the formation of various centers of degeneration which show concentric layers and gradually become confluent. Finally, the total volume of the gland is transformed into a homogeneous, caseous mass. At the same time the distended capsule increases in thickness and forms a sac which confines the semi-fluid, grumous mass. In rare instances the sac ruptures and when close to the surface the contents will be discharged. Under ordinary circumstances, the caseous contents become cohesive and sticky and of the consistency of putty. In very old cases the mass becomes dry and mealy, with little or no tendency to calcification. The greenish-yellow color of the caseous mass, which is stated to be most

characteristic, closely resembles the contents of the intestinal nodules produced by *Oesophagostoma columbianum*. In very advanced cases the internal organs may contain lesions which macroscopically resemble those of tuberculosis. The lungs may be studded with small nodules the size of a pea, the spleen, liver and in rare instances the kidneys also may contain one or more foci of the same character, namely, a mass of greenish-yellow material, surrounded by a firm, fibrous wall. The bronchial and the mediastinal glands may be affected without lesions being found in the lungs. In some cases the lungs contain nodules varying in size from that of a millet seed to that of a walnut. This condition is, as a rule, accompanied by a chronic pleurisy with extensive adhesions and also effusions into the pleural cavities.

The liver may contain nodules similar to those in the lymphatic glands. Cases have been reported, however, where the entire organ was thickly sprinkled with miliary nodules.

The kidneys are rarely affected, but when they are the lesions assume the same characteristic appearance of a firm walled abscess protruding on the surface of the organ. As a rule, only one to two such foci are observed in each case.

A histological examination of tissues containing miliary nodules shows them to be composed chiefly of leucocytes and round cells. Toward the center they are degenerated into a granular detritus. Giant cells are not observed. Among the cells are seen the short bacteria arranged singly or in clumps. They stain irregularly. The bacteria are frequently seen within the degenerated leucocytes, the destruction of which is due, according to Preisz, to the specific chemical products elaborated by the microörganisms.

When a miliary nodule from the liver of an experimental animal, which has been destroyed three weeks after inoculation, is examined microscopically the following picture is observed: A caseous center composed of an amorphous material that does not take any of the ordinary stains. Surrounding the center may be seen numerous leucocytes more or less degenerated and frequently containing one or more bacteria, while clumps of these organisms are scattered among them. External to this is a dense round cell infiltration, the peripheral zone of which is undergoing connective-tissue formation, thus serving as a line of demarcation between the atrophied liver cells and the central cell mass. The process then repeats itself until a connective-tissue barrier strong enough to encapsulate the central part of

the nodule and prevent its further growth is obtained. The nodules in the kidneys and lungs present a similar microscopic appearance, excepting that the foci in the lungs are more regular·on account of the catarrhal inflammation that accompanies the reaction of the surrounding tissue. The center contains a dense mass of disintegrated cell structures, composed of the desquamated and proliferated epithelial cells, degenerated leucocytes and round cells. In experimental animals which succumb quickly to an intravenous injection of virulent material, the lung tissue immediately surrounding the nodules is frequently hepatized.

According to Gilruth the lesion commences by the arrest of the specific bacterium, generally in a lymph gland, where one or more are surrounded by and included within the phagocytes. The microorganisms multiply within the cell and ultimately cause the degeneration and death of the latter. Simultaneously a slow chronic inflammation occurs around the focus of attack; there is proliferation of connective tissue cells and the formation of more or less new fibrous tissue. As the process spreads outwardly the centre degenerates, and the protecting wall increases in thickness. In fact, all the phenomena of the pathology of true tuberculosis in a gland occurs, with the exception of the formation of giant cells. The degenerated · centre of the nodule assumes a greenish tint, especially distinct at the time of exposure by the knife, but becoming gradually grayer afterwards. In the centre of the older purulent or caseous mass (for the consistence varies from that of cream to that of cheese in different tumors) there are usually present no bacilli which can be demonstrated by the microscope or by cultural methods.

Diagnosis. The diagnosis is made by the symptoms, lesions and the finding of the specific bacterium. There are no specific reactions for diagnostic purposes. This disease is to be differentiated from infections of various kinds, not recognized as specific, which may cause enlargement or suppuration of lymph glands. The specific infectious diseases, such as tuberculosis. Lymphadenoma and in case the lesions are restricted to the lungs or organs other than lymph glands, from parasitic lesions. In cases of parasitic lung or liver nodules the intestinal wall is usually affected.

If the diagnosis cannot be made from the gross appearance of the lesions a bacteriological examination will be necessary. The fact should be kept in mind that tuberculosis in sheep is very rare. In

lymphadenitis, cultures in ordinary media will give a growth of the bacterium of Preisz. With tuberculosis the results would be negative (see tuberculosis). Lymphadenoma would be determined by a histological examination.

Prevention. This disease is not communicable directly from infected to healthy sheep. The virus seems to be wide spread and consequently good hygiene is all that can be done in a precautionary way. In localities where it is prevalent, the careful disinfection of the navel as soon as the cord is severed is indicated. Carré reported good results with the use of a vaccine which seemed to cause immunity.

REFERENCES

1. EBERTH. Bacilläre Nekrose der Leber. *Virchow's Archiv.*, Bd. C (1885), S. 23.

2. GILRUTH. Pseudo-tuberculosis in sheep. (Lymph-adenitis). *Jour. Compar. Path. and Therap.*, Vol. XV (1902), p. 324.

3. GILRUTH. Pseudo-tuberculosis in sheep. (Lymph-adenitis). *Bulletin No. 1, New Zealand Dept. of Agriculture.*

4. NÖRGAARD AND MOHLER. The nature, cause, and economic importance of ovine caseous lymph-adenitis. *Sixteenth Annual Report, Bureau of Animal Industry,* 1899, p. 638. (Full bibliography).

5. PREISZ AND GUINARD. Pseudo-tuberculose chez le mouton. *Jour. de méd. vét. et de zoötech,* ser. 3, Vol. XVI (1891), p. 563.

6. PREISZ. Recherches comparatives sur les pseudo-tuberculoses bacillaires et une nouvelle espéce de pseudo-tuberculose. *Ann. de l' Inst. Pasteur,* Vol. VIII (1894), p. 231.

7. SIVORI. Sur une broncho-pneumonie caseouse du mouton, causée par le bacille de Nocard-Preisz. *Recueil de méd. vét.,* ser. 8, Vol. VI (1899), p. 657.

ASTHENIA IN FOWLS AND PIGEONS

Characterization. This is a disease especially of chickens and pigeons in which there is marked emaciation and a failure to take on flesh even when fed on the most nourishing food. Because of this, the disease has received the popular name of "going light."

History. Although this condition or disease has been recognized for a long time, it seems to have been first described in 1898 by Dawson. He gives a brief account of the symptoms, morbid anatomy, etiology and a somewhat extended description of the specific organism which he isolated from the diseased chickens. The writer has studied this affection in pigeons but did not succeed in finding the organism isolated by Dawson.

Etiology. Dawson found this disease to be due to the presence of a certain species of bacterium which he obtained in pure culture from the duodenal contents. He described it as *Bacterium astheniæ.* This organism varies from 1 to 1.3μ in length and about 0.5μ in width with rounded ends. It is reported to possess the peculiarity of multiplying in temperatures varying from 50 to 120° F. It is fatal to rabbits within 24 hours when inoculated into the abdominal cavity with 0.5 cc. of a bouillon culture. Chickens inoculated with this organism remained well.

Symptoms. The only symptoms which seem to be in evidence are the gradual loss of flesh and an exceedingly good appetite. It is reported by certain pigeon fanciers concerning pigeons, and the fact is reiterated by Dawson, that the disease is a very chronic one, often extending over a period of several months but usually terminating in death. In the cases reported, the fowls were well kept and given an abundance of nourishing food. There seems to be an inability on the part of the affected animal to assimilate nourishment.

Morbid anatomy. The most conspicuous lesion is extreme emaciation. According to Dawson the mucosa of the duodenum contains areas in which the walls are deeply reddened and in which the contents are of a mucoid substance. The writer made a number of postmortems on pigeons suffering from this disease without finding any gross tissue changes other than emaciation.

The disease needs further investigation, but the fact that an organism has been found in the duodenum in large numbers, where it multiplies and apparently produces by-products that are absorbed and which interfere with the normal metabolism of the body, is of sufficient interest to call attention to the preliminary findings herein mentioned. It is not unlikely that if the present hypothesis concerning the nature of this disease is verified, a number of disorders now attributed to general causes may be traced to some form of intestinal infection.

REFERENCES

1. DAWSON. Asthenia (going light) in fowls. *Annual Report of the Bureau of Animal Industry, U. S. Department of Agriculture,* 1898, p. 329.

BACILLARY WHITE DIARRHEA OF FOWLS

Synonym.   Fatal septicemia in young chicks.

Characterization.   Bacillary white diarrhea is a disease of young chickens characterized by depression, loss of appetite, labored breathing, and the presence in the tissues of *Bacterium pullorum*.   This disease usually affects chickens from 1 to 4 days old but adults are sometimes attacked.

History.   In 1908 Rettger and Harvey published a description of a disease called fatal septicemia in young chickens or white diarrhea, in which they found the cause to be a bacterium which they named *Bacterium pullorum*.   Rettger and his co-workers published during the next two or three years several articles on this subject, giving detailed accounts of the various phases of the disease.   Morse in 1908 published an article on "White diarrhea in chicks with notes on the coccidiosis of birds."   He claimed that much of this so-called trouble in chickens is due to coccidiosis, identifying the parasite as *Coccidium tenellum*.   In the same year Milks described the disease from Louisiana.   In 1909 Cushing described a white diarrhea under the title of Aspergillosis.   In 1910, Jones confirmed the findings of Rettger concerning its etiology.   In 1911, Gage pointed out the ovarian infection in fowls with *Bact. pullorum*.   He isolated the organism from the ovaries of three hens in two of which he states there was gangrene.   Gage also found *Bact. pullorum* in the ovaries of a number of adult hens dying from a then unknown disease.   The same year, Jones demonstrated that a number of adult fowls that had been infected when young chicks and had recovered carried the organisms in their ovaries.   He found the ovaries to be abnormal.   He also showed that the localization of the lesions in the ovaries of adult hens could be produced by the intravenous injection of 1.5 cc. of a bouillon culture of the organism.   Later, Jones investigated an outbreak among adult fowls from which he obtained the organism from the ovaries of the dead hens.   The ovaries in practically all of these cases were abnormal.

Geographical distribution.   This disease of chickens seems to be widely distributed in the United States.

Etiology.   The cause of bacillary white diarrhea is *Bact. pullorum*. It is present in the organs of the infected chickens.   It is from 2µ to 3.5µ in length and about 0.5µ in width.   The ends are rounded.   In

the tissues, it appears singly and in clumps, stains readily with the ordinary bacterial stains and can be cultivated on the usual media. It has a somewhat close resemblance in certain of its cultural characters to the bacterium of fowl typhoid. It is fatal to young guinea pigs when inoculated subcutaneously in pure culture. Chickens are most susceptible within the first 48 hours after hatching and they seem to be quite resistant after they are four days old. Inoculation with pure cultures in adult fowls rarely if ever produces the disease although outbreaks of it sometimes occur.
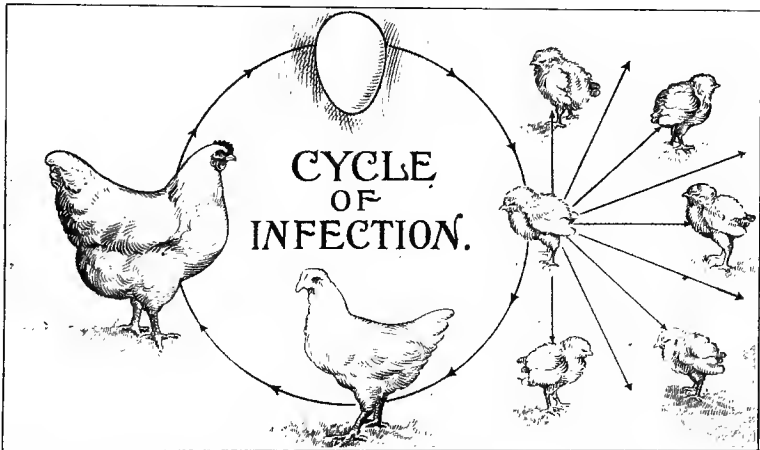
FIG. 49. DIAGRAM SHOWING HOW BACILLARY WHITE DIARRHEA PERPETUATES ITSELF
IN THE BREEDING STOCK. (BULLETIN NO. 68, STORRS AGRIC. EXP. STATION, 1911).

The period of incubation varies in young chicks but it is very short.

Mode of infection. Infection takes place in three ways, namely; through the infected egg, by young chicks coming in contact with those that have acquired the disease through egg infection and by young chicks being housed in contaminated brooders or pens that were recently occupied by infected chicks.

Symptoms. The symptoms of this disease are not characteristic but resemble closely those of other intestinal troubles in young chickens. The first indication of disorder is the desire for more heat, indicated by the young chicks seeking the warmer places in the brooder. This is followed by general depression, loss of appetite and labored breathing. The chick stands with feet apart, the eyes are

dull, the head is drawn down and the respirations are increased and labored. Diarrhea is usually present and in the cases of longer standing the feces gum up the tail feathers. If the disease becomes chronic, lasting from one to three weeks, emaciation is marked and the gait is uncertain. Both Rettger and Jones have called attention to the fact that in those that live the legs seem to grow but the body is stunted, giving rise to the expression "short backed chickens" among poultrymen. In adults there is paleness of the comb and visible mucous membranes. The comb seems to be shrunken, scaley and grey in color. The fowls are listless, depressed, head drawn down and the wings sag. There is loss of appetite and diarrhea.

The duration of the disease is, in young chickens, from 1 to 4 days and in adults from 4 to 5 days, although in some cases the fowls live for a much longer time.

Morbid anatomy. The lesions in the young chick are those of septicemia. There is marked congestion of the liver, spleen and kidneys. The unabsorbed yolk is, according to Jones, the most characteristic lesion of the disease. In adults, minute necrotic foci appear in the liver, spleen, pancreas and sometimes larger areas in the heart muscle. Jones described a fibrinous exudate as a somewhat constant lesion on the capsule of the liver, spleen and pericardium. In the carriers the ovaries are usually cystic.

Diagnosis. Bacillary white diarrhea in young chickens is diagnosed by the symptoms and the finding of *Bact. pullorum* in the organs of the dead chicks. In adult fowls that die of the disease it is diagnosed in the same manner. Infected fowls, that harbor the organism in the ovaries, can be detected by the agglutination test or, if killed, by the bacteriological examination of the ovaries. Jones found that the macroscopic agglutination test is of great assistance in picking out fowls that are harboring this organism. The agglutination takes place in dilutions of 1.50, 1.100, and 1.200 which dilutions are recommended for practical purposes. In his investigations, Jones found that all the infected fowls agglutinated at 1–100. 91% agglutinated in a dilution of 1–200 and 82% in a dilution of 1–500. A few agglutinated in still higher (1–800 to 1–2000) dilutions. He obtained the best results with a test fluid made from several freshly isolated strains of *Bact. pullorum.*

Bacillary white diarrhea is to be differentiated from dietary troubles, chicken cholera, fowl typhoid, aspergillosis, and coccidiosis.

Prevention. Bacillary white diarrhea is introduced into new districts by the indiscriminate buying of eggs for hatching and the purchase of day old chicks from infected farms. Female chicks that survive the attack and become adults often become spreaders of the organism through their eggs. Investigations by Jones and Rettger show that not all the eggs of such fowls are infected but that now and then they are and chicks hatched from such eggs are not only infected themselves but also spread the infection to others in the same brood. The prevention of the disease consists, therefore, in the detection and removal of adults that are spreaders, the segregation of the chicks until they have reached the age of four days and the thorough disinfection of the brooders, runways and feeding and drinking utensils. Jones suggested the very practical method of segregating the eggs near the close of the incubation period into isolated groups of two or three so that if any egg was infected and a chick hatched from it the germs could not spread to others. This is a very serious disease but one that can be readily controlled if precautionary measures are taken in time.

Specific treatment. No satisfactory biological treatment has been discovered.

REFERENCES

1. CUSHING. Aspergillosis. *Canadian Poultry Journal*, 1909, p. 404.

2. GAGE. Notes on Ovarian Infection with Bacterium pullorum (Rettger) in the domestic fowl. *Journ. Med. Res.*, Vol. XXIV, N. S., Vol. XIX (1911), p. 491.

3. GAGE, PAIGE AND HYLAND. On the diagnosis of infection with Bacterium pullorum in the domestic fowl. *Bulletin 148. Mass. Agric. Exp. Station*, 1914.

4. HADLEY. Studies in Avian Coccidiosis. *Centralbl. f. Bakt.*, Bd. L (1909), S. 348.

5. JONES. Fatal septicemia or bacillary white diarrhea in young chickens. *Report N. Y. S. Vet. College*, 1909–10, p. 111.

6. JONES. Further studies on bacillary white diarrhea in young chickens. *Report N. Y. S. Vet. College*, 1910–11, p. 69.

7. JONES. An outbreak of an acute disease in adult fowls due to *Bact. pullorum*. *Report N. Y. S. Vet. College*, 1911–12, p. 140.

8. JONES. The value of the macroscopic agglutination test in detecting fowls that are harboring *Bact. pullorum*. *Report N. Y. S. Vet. College*, 1911–12, p. 149.

9. JONES. Bacillary white diarrhea in chickens. *Proceedings of the Amer. Vet. Med. Asso.*, (1912), p. 379.

10. MILKS. A preliminary report on some diseases of chickens. *Bulletin 108 Agric. Exp. Sta. of La. State Univ. and A. and M. College.*

11. MORSE. White Diarrhea of chicks with notes on the coccidiosis of Birds. *Circular 128, U. S. Dept. of Agric. B. A. I.*

12. RETTGER. Further studies on fatal septicemia in young chickens, or "White Diarrhea." *Journ. Med. Res.*, Vol. XXI (1909), p. 115.

13. RETTGER. Septicemia among young chickens. *N. Y. Med. Journ.*, Vol. LXXI (1900), p. 803.

14. RETTGER. Septicemia in young chickens. *N. Y. Med. Journ.*, Vol. LXXIV (1901), p. 267.

15. RETTGER AND HARVEY. Fatal septicemia in young chickens, or white diarrhea. *Journ. Med. Res.*, Vol. XVIII (1908), p. 277.

16. RETTGER AND STONEBURN. Bacillary white diarrhea of young chicks. *Bulletin 60, Storrs Agric. Exp. Sta.*, 1909.

17. RETTGER AND STONEBURN. Bacillary white diarrhea of young chicks. *Bulletin No. 68, Storrs Agric. Exp. Sta.*, 1911.

CHAPTER V

DISEASES CAUSED BY BACTERIA
GENUS BACILLUS

General discussion of the genus bacillus. The genus *Bacillus* in Migula's classification includes all rod-shaped *motile* bacteria. In the older classifications it includes both non-motile and motile forms. The fixing upon motility as an essential generic character, and thus restricting the genus *Bacillus* to motile forms, is the occasion of some confusion between the genera *Bacterium* and *Bacillus* as applied to a number of important disease-producing bacteria. It is customary to speak of the *Bacillus* of anthrax, of tuberculosis and of glanders rather than of the *Bacterium* of these affections. There are a number of species of bacilli that are widely separated from each other. The diseases which they produce give very different pictures both clinically and in their morbid anatomy.

There are a few species that are especially important from the rather non-specific nature of the infections they produce. The colon bacilli, including the bacilli belonging to the intermediate or Gærtner group, furnish good illustrations of this. They are found in a great variety of lesions but they do not seem to produce a constant, clearly defined reaction on the part of the infected tissues. There are other species that are significant because of the constant character of the lesions they produce regardless of the location in the body or the species of animals attacked. This is particularly true of the bacillus of necrosis. These two groups of bacilli (colon and necrophorus) are, for domesticated animals, the most important etiologically outside of those that produce specific infectious diseases such as tetanus and symptomatic anthrax.

SALMONELLOSIS

Characterization. The term *Salmonellosis* has been introduced to designate the disease caused by *Bacillus suipestifer* (*Bacillus cholerae suis*) described in 1885 by Salmon and Smith. It is usually, if not always, possessed of a low degree of virulence for swine. It was believed at the time to be the cause of hog cholera. The discovery of

a filterable virus as the cause of that disease removes the greater part of the original significance attached to *B. suipestifer* as a pathogenic organism. However, inoculation experiments with this organism by Salmon and Smith, as well as the findings of Uhlenhuth and others relative to its distribution and pathogenesis, indicate that it is a factor of greater or less importance in the production of disease among swine. The symptoms and lesions of the disease produced by natural infection with this organism have not been determined with certainty. The description of the cases that were supposed to have been caused by it in the earlier work on hog cholera shows that they may have been due to the filterable virus or a mixed infection with it.

Salmon and Smith found that subcutaneous inoculation with *B. suipestifer* from cultures is successful in only a small percentage of inoculated swine except when the germs are unusually virulent. In the report of the Bureau of Animal Industry for 1885 several cases of successful inoculations are recorded. Two of these are quoted:

"Two pigs (Nos. 112, 114) were inoculated subcutaneously into the thigh with three cubic centimeters each of a pure liquid culture. No. 114 died nine days after inoculation The superficial inguinal glands were swollen, with hemorrhagic points in medulla. Spleen enlarged, dark. Extravasations on auricular appendages of heart. Lungs oedematous; bronchial glands enlarged, dark red throughout (hemorrhagic). Glands cf abdomen in general hemorrhagic, except those of mesentery; petechiae under serosa of caecum. Kidneys with glomeruli appearing as blood-red points, the entire organ congested. Mucosa of fundus of stomach, lowest portion of ileum, and of the caecum and colon deeply reddened with slight extravasation. Cover-glass preparations as well as cultivations in gelatine and beef infusion revealed hog cholera bacilli, and these only.

"No. 112 died on the 15th day. Diarrhea appeared two days before death. The lesions of this animal resembled those of No. 114, with the following differences; Spleen very large, dark, friable. Kidneys less congested. Lungs with minute hemorrhages throughout the parenchyma. Ecchymoses beneath the endocardium of left ventricle. Lymphatics and digestive tract even more congested and hemorrhagic than in No. 114."

This organism possesses very definite pathogenic power for the rabbit and to a slightly less degree for the guinea pig. There is a large literature on its pathogenesis.

INFECTIONS WITH THE COLON AND GAERTNER BACILLI

Grouse disease. In 1887 Klein described a disease of grouse characterized by congestion of the lungs, liver and kidneys with small necrotic areas in the liver and areas of redness in the intestines. The disease was found to be due to a bacillus which has been found to

belong to the colon group. [The author has studied the bacillus of the grouse disease a culture of which was obtained from Krahl and found it to be *B. coli communis*.] Migula designates it *B. scotius*.

Cobbold also describes a disease of grouse. Leslie* who mentions a large number of diseases, many of which are parasitic, concludes that Klein and Cobbold worked with two distinct diseases, Klein with an acute infectious pneumonia and Cobbold with emaciated birds that had probably died as a result of extreme parasitism.

Quail disease (*Colibacillosis tetraonidarum*). In May, 1907, Morse (*circular No. 109, Bureau of Animal Industry*) described a disease of quail characterized by congestion of the lungs, focal necrosis of the liver, and intestinal ulceration. He found the cause to be a member of the colon group of bacteria. This seems to have several centers of infection in this country. Several species of quail and grouse are susceptible. While there are strong resemblances between this affection and the grouse disease described by Klein, Morse assumes that they are not identical.

Enzoötic in cattle caused by a bacillus of the enteritidis group. In 1902, Mohler and Buckley described an outbreak caused by *B. enteritidis* among cattle in a stable of 21 animals of which eight contracted the disease and died. Three others exhibited the early symptoms.

The symptoms were first refusal of food, suspension of urination and diminution in lactation. There was excessive salivation in some cases. The temperature varied from 102.7° to 104.1° F. The visible mucosæ were congested. There was a wild expression in the eyes and the animals were very excitable. The gait was irregular. Convulsions set in prior to death.

The duration of the disease varied from two days to several weeks.

Morbid anatomy. In the acute cases the anatomical changes were very slight. The most noticeable and characteristic lesion observed consisted of petechial hemorrhages under the endocardium. These were present in every case. There were occasional blood extravasations in the intestinal mucosa. There was marked injection of the blood vessels of the meninges and blood tinted fluid in the cavity. The chronic cases presented a wider range of lesions in the organs.

Cultures of the bacillus were obtained from the different organs. The bacilli were found in small numbers in cover-glass preparations

*Leslie and Shipley. The Grouse in health and in disease. London, 1912.

made from the organs. The organism was fatal to experimental animals and to calves. It was more virulent than the bacillus of hog cholera obtained from hogs dead of that disease.

Since Gærtner first discovered this organism in 1888 in the meat of a diseased cow, it has been isolated by others from both animals and man. It has been found to be pathogenic for animals, and several people have been reported to have become ill from eating broth made from meat containing this organism. During the last few years several bacilli differing slightly from Gærtner's bacillus have been isolated from cases of meat poisoning.

Bacilli of this group, or at least of closely related groups, have frequently been found to stand in a causal relation to the lesions with which they were associated. The more important of these are *Bacillus typhi murium* obtained by Lœffler in 1890 from an enzoötic among mice, the bacillus isolated by Mereshkowsky in 1895 from the ground squirrel, and *Bacillus psittacosis* isolated by Nocard in 1893 from the organs of parrots.

Bacteria belonging to the "Intermediate group" (Gærtner and colon bacilli) have been isolated from diseases in ground squirrels, field mice, ferrets, pigeons, parrots, yellow fever in man, septicemia in calves and still other morbid conditions.

REFERENCES

1. BASENAU. Ueber eine im Fleisch gefundene infectiöse Bacterie. *Archiv. f. Hygiene*, Bd. XX (1894), S. 242.

2. EBERTH AND SCHIMMELBUSCH. Der Bacillus der Frettchenseuche. *Virchow's Archiv.*, Bd. CXV (1889), S. 282.

3. GÄRTNER. Ueber die Fleischvergiftung in Frankenhausen a. Kyffh. und den Erreger derselben. *Correspond. d. allg. ärztl. Vereines von Thüringen*, 1888, S. 573.

4. LASER. Ein neuer, für Versuchsthiere pathogener Bacillus aus der Gruppe der Frettchen-Schweineseuche. *Centralbl. f. Bakteriol.*, Bd. XI (1892), S. 184.

5. LOEFFLER. Ueber Epidemieen unter den in hygienischen Institute zu Greifswald gehaltenen Mäusen und über die Bekämpfung der Feldmausplague. *Centralbl. f. Bakteriol.*, Bd. XI (1892), S. 129.

6. MERESHKOWSKY. Ein aus Zieselmäusen ausgeschiedener und zur Vertilgung von Feld. resp. Hausmäusen geeigneter Bacillus. *Centralbl. f. Bakteriol.*, Bd. XVII (1895), S. 742.

7. MOHLER AND BUCKLEY. Report on an enzoötic among cattle caused by a bacillus of the enteritidis group. (Illustrated and bibliography). *Annual Report of the Bureau of Animal Industry*, 1902.

8. MOORE. On a pathogenic bacillus of the hog-cholera group associated with a fatal disease in pigeons. *B. A. I. Bul. No. 8*, 1895, p. 71.

9. REED AND CARROLL. A comparative study of the biological characters and pathogenesis of bac. X (Sternberg), Bac. icteroides (Sanarelli), and the hog-cholera bacillus (Salmon and Smith). *Jour. of Exper. Med.*, Vol. V (1900), p. 215.

10. REED, CARROLL AND AGRAMONTE. Etiology of yellow fever. *Journ. of the Am. Med. Asso.*, Vol. XXXVI (1901), p. 431.

11. Report of the committee on the Nomenclature of Swine Diseases. *Report of the U. S. Live Stock Sanitary Association*, 1911, p. 142. (The name Salmonellosis was recommended and accepted by the Association.)

12. SALMON. Special report on hog cholera, its history, nature and treatment. *U. S. Bureau of Animal Industry*, 1889.

13. SMITH. On a pathogenic bacillus from the vagina of a mare after abortion. *B. A. I. Bul. No. 3*, 1893, p. 53.

14. SMITH. Other Bacilli not found in outbreaks of Hog Cholera, which belong to the same group. *B. A. I. Bul. No. 6*, 1894, p. 17.

15. SMITH AND MOORE. Experiments on the production of immunity in rabbits and guinea pigs with reference to hog cholera and swine-plague bacteria. *B. A. I. Bul. No. 6*, 1894, p. 41.

16. THOMASSEN. Une nouvelle septicémie des veaux. *Ann. de l' Inst. Pasteur*, Vol. II (1897), p. 523.

17. TURNER. A new cattle disease. *The Veterinary Journal*, Vol. XXXVI (1893), p. 239.

NECROBACILLOSIS

Characterization. Necrobacillosis is a name given to the various lesions caused by *Bacillus necrophorus* in different species of animals. It is characterized by more or less sharply circumscribed areas of necrosis or by a progressive destruction of tissue. It may occur in any organ or tissue of the body and nearly if not all species of animals are susceptible to it. In the internal organs the necrotic areas usually remain quite firm and on microscopic examination *B. necrophorus* may be found in greater or less numbers.

History. This organism was first observed by Koch in 1881. It was later isolated and studied by Lœffler who found it to be the probable cause of the condition designated by Dammann as calf diphtheria. Bang called attention to its ability to produce a coagulation necrosis which led him to give it the name of *Necrobacillus*. In 1876 Dammann published the results of his investigation of caseonecrotic inflammation of the mouth, throat and upper air passages of young calves. The appearance of the lesions was similar to that of diphtheria in man. Schütz working with Bang on a severe epizoötic generally known at that time in Denmark as swine diphtheria, found in the caseous inflammations of the intestines long thread-like bacilli which he considered to be the cause of the deeply penetrating intestinal necrosis. He called it *Bacillus filiformis* and thought it was identical with the microörganism of calf diphtheria. He believed it to be the cause of all the caseous necrotic lesions occurring in the intestine in cases of hog cholera. In his work on hog cholera, Smith refers to "large bacilli following the course of the blood vessels in the

embryonic tissue under the sloughs." Schmorl reports a fatal
epizoötic among rabbits due to this organism. M'Fadyean, Kitt,
Olt, Jensen, Johne, Mohler and others have found this organism in
numerous necrotic lesions in both domesticated and wild animals.
The organism has been carefully studied in its relation to necrosis
by Bang, Schmorl and Ernst. Many of the lesions that have been
found to be due to this organism are known as separate diseases such
as calf diphtheria, lip and leg ulceration, foot rot in cattle and sheep
and multiple necrosis in the liver. In addition to these, many
lesions of a more or less local nature have been attributed to it.
These have been grouped together under the general heading of
Necrobacillosis.*

Geographical distribution. The lesions or diseases caused by
B. necrophorus are widely distributed in Europe and America. It is
believed that the organism is an inhabitant of the normal intestine of
swine and possibly of other animals. Apparently it is widely distrib-
uted.

Morbid anatomy. Broadly stated, the lesion is a coagulation
necrosis with subsequent caseation. There is a slight tendency of the
organisms to invade the surrounding tissue by a progressive advance,
or by invading distant parts of the body by metastasis. There may
be a general intoxication. The lesions are described as sharply
circumscribed patches of yellowish or dull brown color, sometimes
greenish-white, homogeneous, rather dry, friable tissue. It usually
emits a characteristic odor resembling somewhat that of old cheese
and glue. The line of demarcation between the necrotic and healthy
tissue is sharp and consists in a narrow zone of hyperemia. The
organisms are usually found in the outer zone of the necrotic area
rather than in the central portion. Infection takes place through
injuries to the tissues and on that account it is an inoculation disease.

---

*It has been stated by Mohler that *B. necrophorus* is essentially a pleomorphic
organism. It varies in size from short coccoid forms to filaments of 100μ or more
in length and varying from 0.75μ to 1.5μ in width. The longer forms usually appear
as slender and more or less beaded filaments. In the necrotic tissues they exist in
large numbers. Frequently one end of the filament may be broad and club shaped
and the other quite tapering. Involution forms may be present in cultures formed
in media. The short forms can easily be mistaken for other organisms. Most ob-
servers have failed to detect motility in this organism but Schmorl found motile
forms. It is anaerobic and will grow but feebly on the ordinary media. More satis-
factory results are obtained when it is cultivated on agar-bouillon, agar-gelatin,
serum-agar, or various combinations of these. It is quite readily destroyed by disin-
fectants such as five per cent carbolic acid and 1 to 1000 bichloride. Because of the
character of the death of the tissue it is supposed to produce an extra cellular toxin.

The metabolic products are exceedingly poisonous. At the point of entrance the organisms multiply, producing a marked reaction of congestion, followed by an exudation rich in fibrin-forming substances and a defensive immigration of leucocytes. The products of the organism acting upon these defensive processes of the body cause their necrosis. With the death of the tissues the bacilli invade the deeper
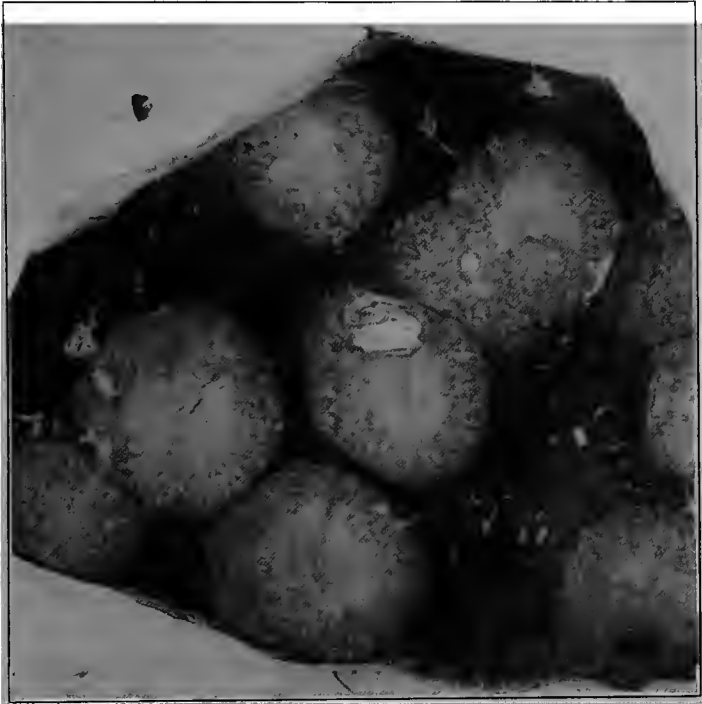
Fig. 50. AREAS OF NECROSIS IN THE LIVER OF A PIG DUE TO *B. necrophorus.*

layers. The lesions due to this organism are liable to be found, as already stated, in any organ of the body or on any mucous membrane or the skin.

Necrobacillosis of the skin. Necrotic inflammation of the skin or dermatitis gangrenosa forms a group of destructive inflammations of the skin following various causes such as burning, freezing, slight trauma or pressure during which *B. necrophorus* may gain entrance to the tissues. Fröhner describes an enzoötic of this disease in horses.

In that case the lesions took the form of multiple necroses of the skin, the parts involved being those spots exposed to irritation. The infection was quite malignant showing a tendency in spite of disinfectants to spread and involve subjacent tissues. The necrotic areas were characterized by the presence of a greyish, yellow, greasy, foulsmelling secretion. A number of varieties of skin necrosis, due to this organism, are described.

Necrotic scratches in the horse have been attributed to *B. necrophorus* in those cases where there is a gangrenous grease, called by the Germans, *Brandmaucke*. In this form the lesions begin as an erythema. The abraded surface becomes coated with the exudate which by mixing with the dirt forms an air-tight covering under which *B. necrophorus* is able to multiply, invade the tissues and produce a caseo-necrotic mass. In this form only the skin and the subcutaneous tissues are involved.

The lesions in the skin due to sheep pox, cow pox and the like, in certain cases, become necrotic. This occurs in the severer varieties in which the lesions become gangrenous and which are spoken of by the Germans as variola diphtheritica, *Brandpocke* or, on account of the odor, putrid pox. From these gangrenous lesions in cattle and swine, Bang isolated *B. necrophorus* which he regarded as the cause of the necrosis. Jensen isolated the organism from necrotic dermatitis of the muzzle, on the outside of the lips, and on the feet of swine. Leclainche and Vallée regarded enzoötic necrosis of the lips and nose of sheep to be due to the same cause and Bang demonstrated this organism in the deep necrosis of the skin of hogs in hog cholera. His results were confirmed by Lindqvist and Zschokke.

Foot rot in cattle. Cattle and sheep occasionally suffer from a contagious foot rot. In these cases there is swelling, the hoof is hot and painful, the toes are usually separated because of the swelling and frequently the tissue of the interdigital space is ulcerated. The swelling may extend up the leg and fistulous tracts occur in the deeper tissue. In certain of these cases, *B. necrophorus* has been isolated. Bang, Hess and others have reported outbreaks of the disease caused by the entrance of this bacillus in some primary lesion. It is to be differentiated from infections with pyogenic organisms which cause inflammation leading to suppuration. If the disease is not promptly treated with disinfectants, the condition may become serious, the animals being unable to stand, lose their appetite, lose flesh, have a

rise of temperature and finally die from absorption of the toxic products.

Foot rot in sheep. Foot rot in sheep is a condition somewhat resembling the same disease in cattle and found to be caused by the same organism. The work of Mohler has shown that long thread-like bacilli which resemble *B. necrophorus* are constantly present and when brought into contact with the feet of healthy sheep produce lesions like those found in the cattle outbreaks. Whenever this occurs in the feet there is a progressive degeneration and destruction of the tissue with a tendency to spread in every direction from the point of infection leaving behind a soft, dead, caseous mass as a result of its action upon the tissues. As in cattle, the sheep are lame, fistulous tracts extend under the horny layer as well as in the deeper tissue of the leg, sometimes causing necrosis of the tendons and ligaments. The prognosis is favorable when promptly treated with disinfectants. This may be differentiated from suppurative lesions due to infection with pyogenic bacteria.

Necrotic quittor. A number of observations have been made by Eberlein, Fröhner, Jensen, M'Fadyean and others that frequently serious consequences follow apparently very slight injuries, as a nail prick in the foot, due to infection with *B. necrophorus*. The first manifestations are an inflammation of the tissues which results in a hot, painful swelling of the coronet over the infected quarter and marked lameness. Finally the sinuses are formed which discharge a sanious fetid fluid and which connect with the necrotic cartilage. In other animals the necrosis of cartilage such as that of the larynx and nasal septum are attacked and destroyed by this organism. Even bone tissue has been found to yield to the action of this organism. Usually pyogenic streptococci and micrococci are associated with *B. necrophorus*.

Lip-and-leg ulceration of sheep. In 1910, Mohler described lip-and-leg ulceration which existed in sheep in various sections in the West. He reviews somewhat at length the literature on this subject. He points out that the lesion may occur anywhere on the exterior of the body where the bacillus may gain entrance but cuts, bruises and other injuries occur less frequently on parts covered with wool than they do about the mouth and lower limbs. The lesions occur, therefore, either on the lips or legs or both. In these necrotic areas Mohler found *B. necrophorus*. The infection is transmitted from one sheep to

another and also it has been found to pass from one species of animals to another.

The disease can be prevented and in a large number of cases satisfactorily treated. The prevention consists first in the removal of the healthy animals; second, close examination of the exposed sheep; and third, thorough disinfection of corrals and sheds where the infected sheep have been kept. This is necessary as the necrosis bacilli retain their virulence under favorable conditions for a very long time. The treatment consists in a local application of disinfectants, several of which are reported to be efficient. These are the pure strength coal tar dip, peroxide of hydrogen, tincture of iodine, or better an application of mutton tallow or vaselin containing 5% of a recognized sheep dip when thoroughly rubbed into the necrotic area.

Caseonecrosis of the digestive tract. Necrosis of circumscribed areas of the mucous membrane of the mouth, the esophagus and other parts of the digestive tract has been observed by a number of investigators and found to be due to *B. necrophorus.* Necrobacillosis in the intestines of calves and pigs has been described. Olt has described cases of such necrosis in the three stomachs of the calf which by extension gave rise to a peritonitis. He has also found it in the first stomach of the deer and Jensen has described it in the antelope. Necrobacillosis of the colon of horses has also been described. The wide distribution of this organism makes it possible for lesions of this kind to occur in the digestive tract quite as readily as in lesions of the skin.

Necrotic stomatitis. This is an acute, specific, highly contagious inflammation of the mouth occurring enzoötically in many species of animals especially in calves, lambs and pigs. It is characterized by the formation of ulcers and caseonecrotic patches on the mucosa and general symptoms caused by the toxins elaborated by the necrosis bacillus. This form of stomatitis is frequently referred to as diphtheria, gangrenous stomatitis, malignant stomatitis, canker and ulcerative stomatitis. It is quite common in pigs.

The period of incubation is said to be from three to five days. It has been found in very young animals. Local lesions are soon followed by general manifestations of intoxication. There is elevation of the temperature. Because of the soreness of the mouth the animals do not eat. The drooling from the mouth becomes profuse, swallowing difficult and even opening of the mouth is painful. An offensive

odor is exhaled. The tongue becomes swollen and sometimes the mouth remains open and the large tongue protrudes. This condition is followed by weakness, loss of flesh and a tendency to lie down. The caseous processes may extend into the larynx, trachea and even into the nasal passages. If the disease continues, the lungs may also become involved. The condition may extend to the intestinal tract in which case there is diarrhea. The lesions may be treated locally with disinfectants. There seems to be a tendency to self-limitation of the infection but treatment enhances recovery very much. When left untreated the animals may die or become permanently stunted in growth. The duration of the disease is from ten to fifteen days. This is to be differentiated from foot-and-mouth disease and also from inflammations due to fungi.

Necrobacillosis in rabbits and guinea pigs. Mazzanti and Schmorl have described necrobacillosis in rabbits. The lesions begin on the lower lip and gradually involve the lower portions of the head, neck and breast, death ensuing in about eight days. They also found lesions due to this organism in the thigh, flank and abdomen. They consisted of fibrous sacs which upon section contained a creamy, homogeneous mass. Somewhat rapid emaciation and death follow. In other forms they found necrotic dermatitis in other regions of the body, especially on the nose and upper lip. There was a marked progressive invasion and destruction of tissues extending to the gums and nasal cavities. Horne describes multiple necroses of the lungs due to this organism. Eberth describes a bacillary necrosis of the liver occurring spontaneously in guinea pigs due to this bacillus. As a rule, however, guinea pigs are less susceptible than rabbits.

Diphtheria in calves. Diphtheria of calves is an infectious disease of young calves characterized by the formation of a diphtheritic membrane (necrosis) on a greater or less portion of the mucous membrane of the mouth and throat. It often leads to death. It is caused by the bacterium of necrosis, described by Bang. This affection is quite common in Europe but it does not seem to be as well known in this country.

Diagnosis. Inoculations for the purpose of diagnosis can be most successfully made in the rabbit and mouse. The rabbit is to be preferred as it is not so susceptible although the white mouse is a very good animal in which to keep up the virulence of pure cultures. Rabbits may be inoculated under the skin with a suspension of the

ground-up tissue from a necrotic lesion. The tissues should be taken at the edge rather than from the center of the necrotic mass. When about 0.5 cc. of this is injected, death usually occurs within a week. The characteristic lesions will be found at the point of inoculation where lying beneath the skin and extending for a greater or less depth in the muscular tissue is an irregular area from 2 cm. to 5 cm. in diameter, consisting of a soft, putty-like appearance of necrosed tissue. It also has a penetrating odor resembling one between cheese and glue. In order to secure a pure culture of the necrosis bacillus, it is often necessary to inoculate a second rabbit and sometimes a third in a series. In later inoculations in which there are few organisms other than the one in question, much longer time is required for the death of the rabbit. In these cases, from eight to twenty or more days are required for the death of the rabbit and lesions may be found in the lungs, liver, kidneys and possibly other organs. From these secondary areas of necrosis in the organs pure cultures may be obtained by the use of anærobic methods. *B. necrophorus* is an obligatory anærobe.

REFERENCES

1. CAUDWELL. Case of equine bacillary necrosis, with metastatic lung lesions. *Journ. Comp. Path. and Therap.*, Vol. XVII (1904), p. 65.

2. DAMMANN. Die Diptherie der Kälber, eine neue, auf den Menschen übertragbare Zoonose. *Deut. Zeitschr. f. Thiermed.*, Bd. III (1877), S. 1.

3. DAVIS. Gangrenous dermatitis. *Vet. Journ.*, Vol. XLIV (1897), p. 99.

4. DAVIS. Gangrenous dermatitis. *Vet. Journ.*, N. S., Vol. 1 (1900), p. 162.

5. EBERLEIN. Die Nekrose der Huflederhaut des Pferdes und ihre Behandlung. *Monatsh. f. prakt. Thierheilk.*, Bd. VII (1896), S. 529.

6. EBERTH. Zwei Mykosen des Meerschweinchens. *Virchow's Arch.*, Vol. C (1885), p. 15.

7. ERNST. Ueber Nekroses und den Nekrosebacillus (Streptothrix necrophora). *Monatsheft f. prakt. Tierheilk.*, Bd. XIV. (1903), S. 193.

8. FROHNER. Dermatitis gangraenosa. *In Bayer and Frohner's Handbuch der tierärztlichen Chirurgie und Geburtshilfe*, Bd. II, S. 284.

9. HESS. Die Klauenkrankheiten des Rindes. *Landwirthschaftl. jahrb. der Schweiz.*, Bd. VI (1893), S. 333.

10. LIGNIÈRES AND SPITZ. Contribution à l'étude à la classification et à la nomenclature des affections connues sous le nom d'Actinomycose. *Rec. d. méd. vét.*, Vol. LXXXII (1905), p. 64.

11. MELVIN AND MOHLER. Lip-and-Leg-ulceration of sheep. *Cir. 160. B. A. I.*, 1910.

12. MOHLER AND MORSE. Bacillus necrophorus and its economic importance. *Ann. Report of B. A. I.*, 1904, p. 76.

13. MOHLER AND MORSE. Necrotic stomatitis with special reference to its occurrence in calves (calf diphtheria) and pigs (sore mouth). *Bull. No. 67, B. A. I.*, 1904, p. 48.

14. MOHLER AND WASHBURN. Foot-rot of sheep; its nature, cause and treatment. *Bull. No. 63, B. A. I.*, 1904, p. 39.

15. OLT. Ueber die progrediente Gewebsnekrose bei Thieren. *Deut. Med. Woch. Jahrg.*, Bd. XXVIII (1902), S. 287.

16. REPP. External ulcerative ano-vulvitis of cattle; a preliminary report. *Am. Vet. Rev.*, Vol. XXVI (1902), p. 595. Also *Proc. Am. Vet. Med. Assn.*, 1902, p. 159.

17. SCHMORL. Ueber ein pathogenes Fadenbacterium (streptothrix cuniculi) *Deut. Ztschr. f. Thiermed.*, Bd. XVII (1891), S.375, also, *Cent. f. Bakt.*, Bd. 1 (1895), S. 666.

18. STEDDOM. A cattle disease in Marshall County, Kansas. *15th Ann. Rept. B. A. I.*, 1898, p. 382.

TETANUS

Synonyms. Lockjaw; trismus.

Characterization. Tetanus is an infectious disease in which the specific organisms remain at the place of introduction. The disease is an intoxication with tetanus toxin. It is characterized by spasmodic contractions of the muscles referable to the nervous system and by the absence of obvious tissue changes. It is caused by *B. tetani*. All mammalia including man are susceptible. It occurs most frequently in horses, asses and mules; next to them in the smaller ruminants such as the sheep and goat; it appears least often in the dog. It is reported to occur rarely in birds. Pigeons and fowls are said to be naturally immune. The human species is very susceptible.

History. Tetanus is one of the diseases that was recognized and described before the Christian era. It was not clearly differentiated until the discovery of its specific cause in 1884.

Geographical distribution. Tetanus is reported to be more prevalent in the hot climates than in the temperate ones, while in the very cold latitudes it is rarely if ever encountered. It is more frequently met with in some districts than in others. Although very common in certain localities, it is, on the whole, a somewhat rare disease. There seem to be no statistics by which its frequency can be determined in this country, but in certain of the European armies this has been noted. In the Prussian army, it is reported to occur once in a thousand cases of sickness among horses. At Würtemberg, Hering reports it once in 3000 cases of disease among the horses in the cavalry. It has also been noted that in some veterinary hospitals it does not occur for long periods, while at other times several cases may appear in rapid succession. It is, however, a wide-spread disease.
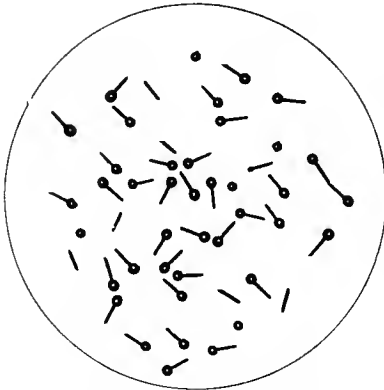
FIG. 51.   BACILLUS TETANI.

Etiology. Tetanus is caused by a slender bacillus (*B. tetani*) 2 to 4μ in length. It forms spores which are at the end of the organism giving it somewhat the appearance of a pin. On this account it has been designated the "pin bacillus." It is anaërobic. It was first observed by Nicolaier in 1884. Carle and Rattone showed that the disease could be transmitted from man to animals by inoculation with the pus from the local lesion. In 1889, Kitasato isolated the bacillus and studied it in pure culture. *B. tetani* stains readily with the aniline dyes, especially with carbol-fuchsin. It takes the Gram stain. It grows well in nutrient gelatin, agar or bouillon and on blood serum at the temperature of the body and in an atmosphere of hydrogen or in the *absence of air* as in deep agar cultures. The addition of a little grape sugar facilitates its growth. It has the distinction of producing the most powerful (poisonous) toxin of any known bacteria, 0.23 of a milligram being estimated as a fatal dose for a man of 175 pounds weight. In the infected animal the bacilli remain at the point of inoculation,

Tetanus bacilli are found in the soil and in the larger intestine of horses and cattle. They have been found in hay dust, in the mortar of old masonry, in the dust in rooms, barracks and hospitals. It is reported that certain savages in Africa destroy their enemies by putting bits of broken glass mixed with certain soils in their shoes. The cause of death is tetanus. Mold rich in horse manure seems to be the most favorable abode for them.

The tetanus bacillus is very resistant, especially in its spore form, to destructive agents such as drying and the ordinary disinfectants. Kitasato found that a 5 per cent. solution of carbolic acid applied for ten hours failed to kill the spores. Tizzoni and Cattani found that mineral and organic acids produced no effect upon the dried spores. Von Behring found that iodine trichloride possesses a strong antiseptic effect upon them. They are not affected by the gastric fluids. It has been noted by Kitt that the dried spores in pus have retained their virulence for sixteen months. They are destroyed

when subjected to a temperature of 100° C. in water or steam for ten
minutes. The bacilli in the vegetative state are readily destroyed
by disinfectants, such as 5 per cent. carbolic acid.

A number of bacilli closely resembling *B. tetani* have been described.
This renders a careful study of the suspected organism necessary, as
it is difficult in some cases to determine *B. tetani* microscopically.
The guinea pig inoculation affords a ready means of differentiation
whenever fresh material is available.

Mode of infection. As the bacillus of tetanus is widely distributed
in the soil and consequently on articles contaminated with it, the
most common modes of infection are punctures, scratches, and pricks
made by splinters, nails or infected instruments (traumatic tetanus).
It is stated by Hutyra and Marek that in these cases it is the other
bacteria introduced that disturb the tissues thereby enabling the
spores of the tetanus bacillus to multiply. Infection may follow
slight abrasions of the skin where infected earth comes in contact
with the lacerated epidermis. Infection through wounds in the
intestinal mucosa does not seem to have been clearly demonstrated.
The most usual method seems to be by pricks and punctures, where
the virus may be carried well into the tissues and there is little or no
bleeding to wash it out. Infection often occurs in young foals and
lambs through the freshly broken umbilical cord (tetanus neona-
torum).

The period of incubation. The shortest period which seems to be
reported is a few hours and the longest is six weeks. In horses the
period of incubation is usually from four to twenty days. After
inoculation with pure cultures it is from four to five days and in sheep
from two to four days. In guinea pigs inoculated with infected soil
the incubation period is usually not over forty-eight hours and often
less than that.

Park has found that mice, guinea pigs, rabbits, rats, horses, goats
and a few other animals inoculated with pure culture have a period
of incubation of from one to three days. In man it varies from one
to twenty days. There are, however, a few exceptionally long periods
reported. It has been noted by Richter and others that the shorter
the period of incubation the more severe the disease, the mortality
being over 90 per cent. in the first and about 50 per cent. when the
symptoms are slow in appearing.

Symptoms. The first symptoms are often obscure and may be
overlooked or they may be ushered in suddenly with violent and exten-

sive tonic spasms. The tetanic spasms usually begin in the muscles of
the head and neck, extending from these to the muscles of the throat,
trunk and extremities. It often happens that the spasms first appear
in the hind quarters and extend forward. There is stiffness of the
parts affected. If in the head, the muscles of mastication are first
attacked with spasms, while if the hind quarters are first attacked,
there are usually spasms of the muscles of the tail. The muscles at
the site of inoculation are frequently the first to show spasms and,
if the disease is of a mild type, they may be the only ones to exhibit
symptoms. Friedberger and Fröhner have grouped the muscles
which are attacked with the more obvious effects upon the appearance
of the animal. They are as follows:

The muscles of mastication. The contraction of these muscles is
called trismus or "lockjaw." According to the degree of contraction,
the jaws remain in more or less close contact, rendering prehension or
mastication difficult or impossible.

The other muscles of the head. These are spasmodically contracted
in different degrees. Spasms of the muscles of the ears cause the ears
to be "pricked" and their tips to be drawn together; of the recti
muscles of the eyes, cause the eyes to be retracted in the orbit with
protrusion of the nictitating membrane; of the nose, produce dilata-
tion of the nostrils; of the dilator of the upper lip, give an abnormal
shape to the opening of the mouth. The muscles of the tongue, of
deglutition and of the larynx are also usually attacked by spasms.

The extensor muscles of the neck. Contraction of these muscles
causes a stiff, outstretched carriage of the head and "ewe neck." The
muscles of the neck become hard and tense to the touch.

The extensor muscles of the back. Spasms of these muscles are
manifested by an extremely hard condition of the muscles of the
back, loins and croup. Several conditions may arise: orthotonous
in which the neck is stretched out and the back and croup are
carried horizontally, or opisthotonous in which the head is raised or
drawn back and the vertebral column slightly depressed. This is
the most common occurrence. There may be a lateral curvature of
the cervical vertebræ which is uncommon and also a convex curva-
ture of the vertebræ which is very rarely observed. The tail, espec-
ially in horses, is often raised and occasionally said to be straight
with the back.

The muscles of the limbs. The spasms in these muscles make the
limbs stiff and cause the animal to assume an attitude in which the

fore legs are extended forward and laterally and the hind ones back-
ward and laterally. They are bent at the joints only with difficulty.
The contraction of the muscles of the abdomen gives the animal
a tucked up appearance and the spasms of the muscles of respiration
render breathing difficult.

Besides the spasms the animal shows an increased reflex irritability
and heightened sensibility. These manifest themselves in excitement,
timidity and intensified muscular contractions if irritated. Sweating
is common, especially in severe cases. In mild cases it may be absent.
There is usually little or no change in the internal temperature. In
fatal cases the temperature is usually constantly high toward the last.
The high temperature (104° to 106° F.) usually continues for some
time after death. Bayer has observed in a horse, 24 hours before
death, a temperature of 102° F.; one and a half hours before death,
105° F.; at the moment of death, 111° F.; and fifty minutes after
death, 113° F. There is frequently no increase in the number of pulse
beats until severe exacerbation sets in. The frequency of the pulse
is much greater in animals which continue recumbent than in those
which keep upon their feet. The pulse is often hard and small and the
walls of the arteries are spasmodically tense. In many cases, how-
ever, it is full, soft and easily compressible. There is, as a rule, an
increase in the number of respirations, which may become very high
if the respiratory muscles are attacked. The number varies accord-
ing to the excited condition of the animal. The respirations may
increase four fold without a corresponding increase in the pulse beat.
The breathing may reach from 80 to 100 per minute. In character
the respirations are shallow on account of the fixed condition of the
ribs and the spasms of the muscles which compress the abdomen.
There may be cyanosis and catarrh of the nasal mucosa, coughing
and in fatal cases symptoms of hyperemia and edema of the lungs and
often pneumonia (usually aspiration in nature). There is constipa-
tion due to lack of peristalsis and the rigid condition of the muscles
which compress the abdomen. Micturition becomes less frequent
and more difficult. Complete retention of urine is said to occur in
some cases. The urine has a high specific gravity and occasionally
contains albumen. Some animals can eat readily while others eat,
if at all, with great difficulty. They like to play with drink set
before them and often try to satisfy their thirst, which seems to
increase as the disease advances. In fatal cases the animals seem to

be perfectly conscious to the last. They seem to be possessed of a feeling of terror.

The duration of the disease varies in different species and in different individuals of the same species. In the horse it may last for two or three days only or it may continue for several weeks. In cattle the course is usually less rapid, but it rarely runs longer than two weeks. In sheep it usually terminates fatally within a week and often in two or three days.

Morbid anatomy. The gross examination of the tissues at post-mortem of animals dead from tetanus is usually negative. It has been pointed out by Goldscheider and Flatau that in experimental animals there are certain characteristic changes in the *motor cells of the anterior horns of the spinal cord* which in the order of their development depend upon the concentration of the toxin or virulence of the bacteria injected and upon the duration of the disease. The changes are primarily an enlargement of the nuclei, which at the same time become more distinct; then follows *an enlargement and disintegration of Nissl's cell-granules with an enlargement of all of the nerve cells.* These investigators also found that where antitoxin had been used it had a distinct retarding influence upon these changes. They found like lesions in the spinal cord of a human subject dead of tetanus. Very similar results have been obtained by Matthes, Westphal, Gœbel and others. The lesions point to the anterior horns of the spinal cord as the primary seat or origin of the tetanic contractions. The changes pointed out above are said by Moschowitz to be characteristic of tetanus and constantly present. The motor ganglia cells of the anterior horns of the spinal cord seem at present, therefore, to be the most likely source of the spasms, due apparently to a specific affinity between those cells and the tetanus toxin. It is possible to explain also the local spasms on this hypothesis as the toxin elaborated by the bacilli acts on the nerves terminating in the affected region. The experiments of Tizzoni and Cattani suggest the possibility of such a theory. There is, however, need for further investigation on this subject.

A considerable number of lesions may be found elsewhere in the body, none of which can be considered as characteristic of the disease, but which are secondary to the tonic contractions. The blood, owing to lack of oxidation, may be dark colored. There may be numerous ecchymoses and sanious exudates in the subserous and mucous membranes. The lungs may be variously affected according to the extent

of the trouble with the respiratory muscles. Thus congestion, edema, hemorrhages, pneumonia, emphysema and hypostatic congestions have been described. In the heart there are usually epi- and endocardial hemorrhages. The fibers of the muscles may show cloudy swelling and the various organs may be more or less affected. The specific lesions are those in the nerve cells. It is believed that the toxin elaborated at the point of infection reaches the cells of the central nervous system through the nerves themselves. The toxin is diffused very slightly through the blood. Tetanus toxin as demonstrated by Wasserman and Takaki has a special affinity for the nerve cells. The toxin becomes fixed, after the lapse of some hours, by the cells of the central nervous system and it is then that the symptoms begin. It is because of this affinity that the therapeutic value of tetanus antitoxin is not greater. The antitoxin neutralizes the toxin circulating in the blood but it is ineffective against existing lesions.

Diagnosis. Tetanus is to be diagnosed very largely by the symptoms. The microscopic examination of the local lesion, if it can be found, might reveal the tetanus bacillus. There are no specific tests that can be relied upon. The injection of guinea pigs with a small quantity of the blood of the suspected animal can be resorted to if desired. It would, in case of tetanus, produce symptoms. Courmount has shown that normal horse serum agglutinates tetanus bacilli feebly (1 to 50 to 1 to 100). The serum from highly immunized horses agglutinates in dilutions of 1 to 2000 and sometimes higher.

Tetanus is to be differentiated from rabies, acute muscular rheumatism, eclampsia, catalepsy, strychnine poisoning, convulsions in the newly born and pyemic polyarthritis in lambs and foals. It must be distinguished from tetany due to the traction of certain nerves adhered in a cicatrix.

The symptoms of tetanus that are the most diagnostic are the continuous tonic spasms of different groups of muscles, the apparent clearness of mind (if we may attribute such a quality to animals), and the absence of fever in the beginning of the symptoms. The general attitude of the animal is also of much value. Poisoning with strychnine usually occurs in dogs where tetanus is rare, and again in strychnine poisoning the suddenness of the attack, the rapidity of the course and the increased reflex irritability are valuable diagnostic features. In differentiating tetanus from other affections in the newly born, the microscopic examination of cover-glass preparations made from the end of the umbilicus may be of assistance.

Prevention. Owing to the wide distribution of *B. tetani* or their spores precautions consist in the thorough disinfection of all wounds and in localities where tetanus is prevalent the injection of tetanus antitoxin. With animals at pasture, it is impossible often to know of the wounds until it is too late. In stables where the disease becomes prevalent, the floors and siding should be thoroughly disinfected and special watchfulness exercised to find at the earliest moment any injury by which infection could occur. The practitioner should learn as soon as possible the tetanus infected lands and stables in his community and, knowing these, give instruction to his clients to take such precautions as are possible. In case operations are to be performed on animals in such localities an immunizing dose of tetanus antitoxin should be administered. This practice is followed in many places in Europe.

Tetanus antitoxin. The antitoxin is prepared by injecting horses with the filtrate of bouillon cultures, either alone or with a quantity of antitoxin. After the first dose the animal becomes tolerant to a certain degree so that by repeated and constantly increasing doses complete resistance to the toxin is acquired. When this point is reached the serum usually possesses a strong antitoxic power. As a practical remedy for the disease in animals the recorded results from the use of this antitoxin are somewhat contradictory. In human practice the results are similar. Moschcowitz has collected 290 cases in man where it has been used subcutaneously, with 173 recoveries and 177 deaths or a mortality of 40.33 per cent. In a total of 48 cases where the antitoxin was injected intracerebrally 23 recovered and 25 died, a mortality of 52.08 per cent.

Specific biologic treatment. The tetanus antitoxin has been recommended but its value as a prophylactic seems to be greater than as a remedy after symptoms have appeared.

Some interesting experiments suggested by Krokiewitz directed toward the finding of a specific treatment consist in the injection of an emulsion of brain substance. Primarily this method of treatment is based upon the hypothesis, set up by Goldscheider and Flatau, who, as a result of their research came to the conclusion that "the morphological changes in the nerve cells are the expression of a chemical process, i. e., of the chemical combination of the toxins with the nerve cells. Every nerve cell possesses atom groups which have a certain affinity for the atom groups of the tetanus toxin and are able to com-

bine with them." Wassermann and Takaki substantiated this hypothesis experimentally. These observers injected into experimentally tetanized animals an emulsion of spinal cord, obtained from a freshly killed animal, to test, if possible, whether the nerve cells of the dead animal also have this affinity for the tetanus toxin, like the nerve cells of the living animal. By this experiment, they have come to the conclusion that every part of the nervous system, particularly the brain of the examined animals including man, has a definite and positive tetanus antitoxic power; and that the injection of normal brain substance into experimentally tetanized animals has the power to save life. Further work in this direction is necessary to fully demonstrate the efficiency of this procedure.

REFERENCES

1. VON BEHRING UND KITASATO. Ueber das Zustandekommen der Diptherie-Immunität und der Tetanus-Immunität bei Thieren. *Deutsche Med. Wochenschrift,* Jr. 1890, S. 1113.

2. KITASATO. Uber den Tetanusbacillus. *Zeit. f. Hygiene,* Bd. VII (1889), S. 225.

3. KITASATO. Experimentelle Untersuchungen über das Tetanusgift. *Zeit. f. Hygiene,* Bd. X (1891), S. 267.

4. MCFARLAND. Tetanus and vaccination. *The Journal of Medical Research,* Vol. VII (1902), p. 474. (New series Vol. II).

5. MOSCHCOWITZ. Tetanus, a study of the nature, excitant, lesions, symptomatology and treatment of the disease, with a critical summary of the results of serum therapy. *Studies from the Department of Pathology of the College of Physicians and Surgeons, Columbia University,* Vol. VII (1899–1900). (M. gives pathology and antitoxin treatment, summary of cases and full bibliography.)

6. NICOLAIER. Beiträge zur Aetiologie des Wundstarrkrampfer. *Inaug. Diss.,* Göttingen, 1885.

BLACK LEG

Synonyms. Black quarter; symptomatic anthrax; emphysematous anthrax; quarter ill; quarter evil; gangrenous emphysema; struck; merylen; *charbon symptomatique; Rauschbrand.*

Characterization. Black quarter is an acute infectious disease of cattle characterized by the development of an emphysematous swelling of the subcutaneous tissues and muscles. These lesions are usually located upon and ordinarily extend over the greater part of a hind quarter or of a shoulder. The disease does not spread from animal to animal by simple contact but the infection takes place apparently from a common source, the soil. The virus exists in the soil in certain localities.

Symptomatic anthrax is a disease of cattle, sheep and goats, although the latter two species are rarely attacked. Swine can be

infected artifically and they have been observed affected with it about packing houses. Guinea pigs are very susceptible to inoculation. It is reported that horses, asses and white rats develop local lesions when inoculated subcutaneously with the virus. Other animals seem to be immune. In cattle, it rarely occurs in the very young, under six months, and in adults after the fourth year. Katona reports its appearance in calves three to four weeks of age and in cattle seven and eight years old.

Infection takes place through wounds in the skin, and presumably the digestive tract and trachea. The disease occurs usually in the summer months. The influence of the site of inoculation is said to be very marked. According to Besson, the dose of bacilli that will kill a cow inoculated in the cellular tissues of the body will cause merely a benign swelling if injected into the connective tissue of the neck. The intravenous inoculation leads to a slight rise of temperature only. In rabbits the disease can be produced if the inoculation is accompanied by *M. prodigiosus* or the site of injection traumatized.

History. It is supposed that black quarter has existed for hundreds of years, although it was not until late in the last century that it was positively differentiated and recognized as a distinct and specific disease. The descriptions given to many of the earlier epizoötics designated as anthrax correspond more exactly with the present knowledge of black quarter than they do of anthrax.

In 1782, Charbert classified the various anthracoid diseases recognized at that time into three groups, (1) anthrax fever, where the disease manifested itself without external swelling, (2) true anthrax, where the lesions consisted at first of small, hard and very painful swellings followed or accompanied by fever and other general symptoms, and (3) symptomatic anthrax, where the swelling was preceded by a rise of temperature, loss of appetite and symptoms of general depression. This classification was held for nearly a century. Boutrolle, in 1797, refers to a disease which he called *mal de cuisse* (quarter evil) because it affected the animal in the thigh. Viborg described the disease in Denmark, where it has long been known to the laymen and designated by them as *raslesyge* ("rattle disease"). Its clinical features were very accurately described by Walraff in 1856. In 1879, Arloing, Cornevin and Thomas proved the causal relation of a certain microörganism to this disease and thus established its specific nature. A year later (1880) they described the specific microörganism

and demonstrated that the disease could be produced by inoculating susceptible animals with it. Since that time both the organism and the disease have been studied by many investigators. The most extended investigations in this country have been made by the Bureau of Animal Industry.

Geographical distribution. Black quarter exists to some extent in nearly every country in the world. It is reported as occurring in the most northern latitudes in which cattle are kept, as well as in the temperate and tropical zones.

In Europe, it occurs on the pastures on the Alps, where for five months in the year the ground is covered with snow and ice and in America it is quite common in certain northern districts. It has been reported from Asia and from Northern and Southern Africa.

In the United States, it prevails to a greater extent than is generally supposed. The states and territories which, according to the reports of the Bureau of Animal Industry, suffer most from it are Texas, Oklahoma, Kansas, Nebraska, Colorado, North and South Dakota and New Mexico; but a number of the other Western States are badly infected. Many of the states east of the Mississippi river have infected localities, but in a few of the Eastern and Southern states it seems not to exist. During the last few years infected localities have been found in New York where there has been an annual loss from this disease, but where, prior to recent investigations, the cause of death has been attributed to poisoning.

Etiology. Black leg is caused by *Bacillus Chauvaei*. This organism varies from 3 to 6μ in length and from 0.5 to 1.μ in width. The ends are rounded and it produces spores. It stains readily with ordinary aniline dyes and also after the gram method. In cultures long involution forms are often observed. It is anaërobic. In suitable culture media under anaërobic conditions or in animal tissues (other than blood) it multiplies rapidly with the evolution of gas. The presence of spores renders it very resistant to natural destructive agents and to common disinfectants. The bacillus of black leg or its spores are supposed to gain entrance to the tissues of animals through abrasions of the skin or, possibly, the mucous membranes of the mouth or intestine.

The period of incubation is not positively known in cattle but it is very short (three to five days). Guinea pigs inoculated with a culture of the organism die in from one to three days.

Symptoms. The symptoms generally are those of a bacteriemia. Arloing, Cornevin and Thomas state that general symptoms always precede the local manifestations. Nörgaard reports finding cattle with marked local lesions but few of them suffering from general symptoms, although the latter may have preceded the former and have subsided.

There is elevation of temperature, reaching in some cases 107° F. It usually falls to the normal or even subnormal before death. There is loss of appetite, loss of rumination and pronounced depression. Respiration is accelerated. The animal moves with difficulty and lies` down frequently. At first the visible mucous membranes are congested and within twelve to fifteen hours they have a dirty leaden or purplish color.

The local symptoms may appear on different parts of the body except below the knee or hock joints and on the tail. They are less frequently found about the head, although they may appear in angles of the jaws and throat. They are more frequent on the thighs, neck, shoulders and lower region of the chest. The swelling is at first small and painful. It spreads rapidly and may in a few hours attain to a large size, when it becomes characterized by a crackling and a gurgling sound when the hand is passed over it. On percussion it gives a clear, tympanic sound due to the collection of gas in the affected tissues. At the center of the larger swellings the skin becomes dry and parchment-like, cool to the touch and painless upon pressure. If lanced, a dark, reddish, frothy fluid flows from the wound. It emits a disagreeable odor. In some cases there is but one swelling but usually there are two or more which may become confluent. The lymph glands adjacent to the swellings are much enlarged. There is usually trembling of the muscles, which, as death approaches, may develop into violent convulsions.

Hun has pointed out the interesting fact that in a very large percentage of cases the swellings appear on the right side. There seems to be no explanation given for this localization. In this country, records are wanting of observations on this point. Hoopen has called attention to the characteristic sour odor from the mouth.

Arloing has called attention to a mild form of this disease in which the symptoms are slight debility, loss of appetite and slight local swelling.

The duration of the disease is from several hours to three days. Occasionally it is longer. *The prognosis* is grave.

Morbid anatomy. After death the carcass soon becomes distended with gas. This is due in part to the fermentation in the digestive tract and in part to the formation of gas in the subcutaneous tissues due to the presence of the specific bacillus. The subcutaneous distension is especially marked in the region of the swellings but it extends for a considerable distance from these foci in the direction of least resistance. The tympanitic condition often causes the two legs on the upper side of the carcass to extend out straight without touching the ground. A dark, blood-colored, frothy discharge flows from the nostrils and anus. Decomposition takes place very rapidly except in the affected muscles, which retain a sweetish-sour odor for a considerable time after other parts of the carcass have become putrid.

The skin covering the swelling is often affected with dry gangrene. The subcutaneous connective tissue is yellow, gelatinous, infiltrated with blood and bubbles of gas which escape if the tumor is incised. The muscles underneath the tumors are of a dirty brown or of a blackish color. At other places they are dark red or dark yellow and, when exposed for some time to the air, they may have a golden lustre. They are brittle, putrid and very rich in fluids. They crackle on palpation. When incisions are made into them, blood of a frothy, greasy, tarry appearance and of a sickish, fetid odor issues from them when they are squeezed. The fibres of the muscles show extremely varied degenerative changes. The gases that are present in the muscles are inflammable and burn with a bluish flame on being ignited. They are said to have but little odor, on which account it is assumed that they consist of carburetted hydrogen. A complete chemical analysis of these gases seems not to have been made. The lymph glands near the tumors are enlarged and full of blood. They contain hemorrhages and are infiltrated with a serous fluid. The afferent lymph vessels are sometimes distended with gases, giving them the appearance of strings of beads. Changes similar to those of the external muscles appear in the muscles of the tongue and pharynx when the disease, as may happen in rare cases, is localized on the oral and pharyngeal mucous membranes.

A large amount of blood-red exudate is frequently found in the abdominal cavity. In other cases only a small quantity of a serous fluid is present. In still others no changes at all appear. The abdominal changes seem to be determined by the swelling of the muscles, that is, whether it has or has not spread to the peritoneum. Yellow gelatinous and hemorrhagic infiltrations are often met with on

the omentum, mesentery and in the neighborhood of the kidneys. The mucous membrane of the stomach and small intestines is frequently swollen, congested and infiltrated with hemorrhages, in which case the contents of the intestine are bloody.   The liver is hyperemic, but the spleen is usually normal.

In the thoracic cavity, the pleuræ in the neighborhood of the swollen parts of the skin and mediastinum are sometimes hemorrhagic.   The pleuræ may also show large ecchymoses, in which case the thoracic cavity contains a sero-sanious exudate.   Hemorrhages are sometimes present in the lungs, pericardium, myocardium and under the endocardium.   The muscular tissue of the heart is very soft, but the other muscles show only slight changes.   The mucous membrane of the bronchi is sometimes hyperemic and sprinkled with hemorrhages.

The blood is of a normal color and coagulates readily,   The fluids of the muscles have, according to Feser, an acid reaction, and the flesh becomes rapidly putrid.   The bacilli of black leg are found only in small numbers if at all in the blood during life, but abundantly a few hours after death.   They are numerous in the local lesions.   In swine, it is reported that the lesions are restricted to a severe inflammation of the throat.

Diagnosis.   Black leg is diagnosed by the symptoms, especially the local swellings, the lesions found on post mortem and the finding of *B. Chauvaei.* This can be done by making cultures in deep agar or in the fermentation tube, from the local lesions or by the inoculation of guinea pigs.   The organisms are also found in microscopic preparations from the lesions.   There are no satisfactory specific tests for diagnosing this disease.   The serum of a cow affected with black leg is reported to agglutinate the bacilli in dilution of about 1 to 300. The serum from a healthy cow agglutinated in dilutions of less than 1 to 12.

Black quarter is to be differentiated from anthrax, septicemia hemorrhagica, malignant edema and various forms of poisoning. Usually the localized subcutaneous lesions are sufficient to differentiate black quarter from these other affections.   It often happens, however, that post-mortem changes have so modified the carcass before it can be examined that the diagnosis is questionable.   In this and all doubtful cases it is necessary to resort to a more definite method or methods, such as the microscopic examination, cultures and animal inoculation.   These methods are described under the respective diseases.

It is more difficult to differentiate between black leg and malignant edema as both produce crepitant edematous swellings. The bacillus of malignant edema forms long chains which are never met in cultures of black leg. It is also more actively motile, longer and more wavy than that of black leg.

Animal inoculation. In guinea pigs inoculated in the deeper sub-cutaneous tissues with pure cultures of black leg bacteria or with bits of tissues from the affected area of an animal dead from the disease, death ensues in from one to two days. It is preceded by a rise of temperature, loss of appetite and general indisposition. The site of inoculation is swollen and painful and drops of bloody serum may sometimes be seen exuding from it. At autopsy the subcutaneous cellular tissues and underlying muscles present a condition of emphy-sema and extreme edema. The edematous fluid is often blood stained and the muscles are of a blackish or blackish-brown color. The lymphatic glands are markedly hyperemic. The internal viscera present but little alteration visible to the naked eye. In the blood stained serous fluid about the point of inoculation short bacilli are present in large numbers. These often present slight swellings at the middle or near the end. They are not seen as threads but lie singly in the tissues. If the autopsy is made immediately after death, these organisms may not be detected in the internal organs, but if not made until after a few hours, they will be found there also. In early autop-sies only the vegetative forms of the bacilli may be found, but a few hours later spore-bearing rods may be detected.

Prevention. In checking the spread of the disease it is very import-ant wherever it is possible to remove the well animals from the infected field. The swellings should not be opened and the discharge scattered over the field. The dead animals should be burned if possible, other-wise buried deeply and covered well with lime as soon as possible after death. Birds and other animals should not be allowed to feed upon the carcasses and the skin should not be removed. Every precaution to restrict the spread of the bacteria of this disease should be taken. It is very desirable to thoroughly disinfect the ground where the animal lay at the time of death. The spores are very resistant both to disinfectants and the natural destructive agencies such as sunlight and drying. It is not wise to use land upon which animals have contracted the disease for grazing purposes for susceptible species.

According to Jensen, it is possible to reduce the infectiousness of the soil in the localities where black leg exists by draining the soil,

planting trees and cultivation.   In Denmark the disease has decreased
considerably on account of such treatment.

Vaccination. Several methods of fortifying exposed animals
against the infection of symptomatic anthrax virus have been pro-
posed.   In 1880, Arloing, Cornevin and Thomas demonstrated at
Chaumont that animals injected with the filtrate of cultures of this
virus into the jugular vein were protected against inoculation with the
strong virus.   It was found, however, that this method was difficult,
as the vein had to be exposed and the greatest care was necessary to
prevent infection of extra vascular tissue in introducing and with-
drawing the syringe.   Later these investigators attenuated the virus
by heating it to a temperature of 100 to 104° C. and injecting it into
the subcutis of the shoulder.   This gave a partial immunity which
was reinforced after eight or ten days by a second inoculation of a virus
that had been heated from 90 to 94° C. for six hours.   They injected
the virus where the subcutis is quite dense, such as at the end of the
tail where only local swellings would occur.   This process is known as
"the French method," Arloing's or the "Lyons method."

In 1888 Kitt proposed a single injection method using a virus
attenuated by heating at a temperature of from 85 to 90° C. for six
hours.   A single injection of this vaccine would further confer
immunity.   He further modified Arloing's method by making the
injections in the shoulder regions where the skin is looser and the
operation easier.   Later, Kitt made further important investigations
concerning preventive vaccines for this disease.

In the fall of 1896, investigations looking to the preparation of a
black quarter vaccine were begun in the Bureau of Animal Industry
at Washington by Dr. Nörgaard under the direction of Dr. Salmon.
The various European methods were tried.   The one finally adopted
consists of a single vaccine, the Arloing principal with Kitt's modifica-
tion.

The material used for the vaccine is obtained from a fresh blackleg
tumor, by pounding the muscle tissue in a mortar with the addition
of a little water and squeezing the pulp through linen cloth.   The
· juice is spread in layers on plates and dried quickly at a temperature
of about 35° C.   This temperature does not in the least affect the
bacteria, and the dry virus obtained in this way retains a high degree
of virulence for two years or longer.

When vaccine is to be prepared, the dried material is pulverized
and mixed in a mortar with two parts water until it forms a semifluid

homogeneous mass. This is spread in a thin layer on a suitable glass dish and placed in an oven, the temperature of which may be regulated with exactness. The reason for mixing the dried muscle with water is to insure a quicker and more uniform attenuation. The temperature of the oven is previously brought up to $95°$ to $99°$ C., and the virus is allowed to remain in it for six hours. When removed it appears as a brownish scale, which is easily detached from the dish. This scale is pulverized and put up in packages containing 10 doses each. Before it is used, it is mixed with 10 cc. water, filtered and the filtrate injected in doses of one cubic centimeter.*

Specific biologic treatment. Arloing immunized a heifer by inoculating it with increasing doses of the virus over a period of six months. Its serum possessed prophylactic, therapeutic and antitoxic properties. In doses of 10 cc., it protected sheep against a fatal dose of the virus. Leclainche and Vallée report an antiserum from hyperimmunized goats and horses.

Roux, Dünschmann and Leclainche and Vallée have all demonstrated that a toxin is produced in cultures of *B. Chauvaei*. A satisfactory specific treatment, however, does not seem to have been produced.

REFERENCES

1. FISCHER AND KINSLEY. Blackleg in Kansas, and protective inoculation. *Bulletin No. 105, Kan. State Agric. Exper. Station*, 1901.

2. HOOPEN. Rauschbrand Diagnose. *Tijdsch. Veearts.*, Bd. XL. (Ref. *Ellenberger's Jahresbericht*, 1914. S. 32.)

3. KATONA. Beiträge zur pathologischen Anatomie des Rauschbrand. (*Ref. Ellenbergers Jahresbericht*, 1911, S. 39.)

4. LEWIS. Symptomatic anthrax. *Bulletin No. 27. Oklahoma State Agric. Exper. Station*, 1897.

5. MAYO. Blackleg. *Bulletin No. 69. Kan. State Agric. Exper. Station*, 1897, p. 108.

6. NÖRGAARD. Blackleg in the United States and the distribution of vaccine by the Bureau of Animal Industry. *Annual Report of the Bureau of Animal Industry, U. S. Dept. Agric.*, 1898.

7. NÖRGAARD. Blackleg. Its nature, cause and prevention. *Circular No. 23. U. S. Bureau of Animal Industry*, 1898.

8. PAQUIN. Blackleg. *Bulletin No. 12. Mo. Agric. Exper. Station*, 1890.

9. PETERS. Blackleg. Its nature, cause and prevention. *Bulletin No. 65. Neb. State Agric. Exper. Station*, 1900.

10. SALMON. Black quarter. *Annual Report Bureau of Animal Industry, U. S. Dept. of Agric.*, 1893–4.

---

*Leclainche and Vallée have described a method of making a vaccine from pure cultures.

MALIGNANT EDEMA

Synonyms. Septicæmia gangrænosa; œdema malin; traumatic spreading gangrene.

Characterization. Malignant edema is an acute, wound infection disease of domesticated animals, which is especially characterized by edematous, later crepitating tumors at the place of infection due to *Bacillus oedematis maligni.* Nearly all species of animals including man are susceptible.

History. Crepitating tumors following wounds, withdrawing of setons, castration, and the like have long been known. It was pointed out by Girard that crepitating tumors in sheep could be caused by subcutaneous injection of animal tissue taken from putrefying tissue. Chauveau (1873) showed by experiments performed on goats that the development of the disease stood in a close relation to living organisms present in injected putrescent blood. Pasteur, in 1887, studied more closely the organism and named it *Vibrion septique.* He obtained it in pure culture. Later Koch and Gaffky (1881) studied exhaustively the disease caused through contamination by contact with the ground, which they named malignant edema. Since then Kitt, Jensen, Sand and Leclainche have studied its bacteriology, while Jensen and Sand, v. Rátz, Fröhner, Carl and others have made valuable contributions to the appearance of the malady among domesticated animals. Malignant edema is a wide spread but not a common disease.

Etiology. Malignant edema is caused by *Bacillus oedematis maligni* Koch (*Vibrion septique* Pasteur). This was the first anaerobic organism to be isolated or studied. It is found in the soil and also in the intestinal tract of certain animals. It is described as a bacillus resembling that of anthrax, but somewhat more slender with rounded ends, and spore bearing. It is a strict anaerobe. In artificial cultures as well as in the animal body, after the death of the latter the bacilli grow into long filaments. Exceptionally, living animals contain the spore bearing bacilli in the edematous fluid (Jensen and Sand). The bacilli stain very readily with aqueous aniline dyes. In culture media containing sugar, gases are formed which have a characteristic disagreeable odor. The inoculation of a pure culture into the deep connective tissue of mammals and birds produces a tumor quickly spreading from the place of inoculation and later crepitating on account of the formation of gas. Several varieties of its bacillus

have been described. It is possible that they are modified forms of the one species.

Of the domestic animals, the horse is the most susceptible to natural infection; the cow, the sheep and the goat are less susceptible. The pig, dog and cat are rarely attacked.

Infection sometimes follows contusions or lacerations and incisions and sometimes delivery, when help is given with hands or instruments not clean. It also occurs from injuries such as castration, shearing, bleeding, drawing setons, and subcutaneous injections.
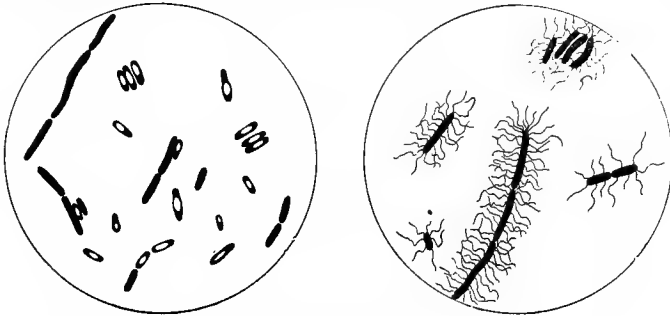
FIG. 52. BACILLUS OEDEMATIS MALIGNI WITH FLAGELLA AND SPORES (*Hutyra*).

This organism produces a toxin. Besson found that if spores freed from the toxin were injected they were destroyed by the phagocytes and no lesions were obtained.

Morbid anatomy. On the expansion of a tumor appearing in any part of the body, the connective tissue is distended and infiltrated with yellow or reddish fluid containing many small gas bubbles, which emits a characteristic odor. These gelatinous infiltrations of the connective tissue follow between the deeper layers of the muscle. The muscle itself is often sallow or dark red in color and is very brittle. The connective tissue in places is often sprinkled with larger or smaller hemorrhages. In the intestinal cavity there is a little reddish, serous fluid; the peritoneum is deeply injected. In the cases in which malignant edema has developed in connection with parturition the uterus is insufficiently contracted, the subserous connective tissue of the true pelvis and the walls of the uterus are edematously infiltrated.

The spleen is usually not affected. Occasionally acute tumors with gas formation are found in the pulp (Fröhner). The liver may show

tumefaction. The mucous membrane of the intestinal canal may show signs of acute catarrh. The lymph glands are swollen. The lungs are hyperemic and for the most part edematous. The muscular system of the heart shows usually a high degree of parenchymatous degeneration. The blood clots very little. The body decomposes quickly. The fluid pressed out of the crepitating tumor contains edema bacilli in great number, associated possibly with other bacteria.

The presence of the bacilli of malignant edema in the tissues of a dead animal does not of itself prove that it died of this disease. These bacilli appear very often in the intestinal contents of animals which feed on plants. They often pass through mucosa of the intestine into the blood stream at the time of death. They may multiply in the tissues, especially when the blood remains fluid for some time.

Diagnosis. Malignant edema is diagnosed by the symptoms, lesions and bacteriological examination. The blood of immune animals agglutinates the bacilli in dilutions of from 1 to 30 to 1 to 3000. There are no satisfactory sera or other specific reactions. It is to be differentiated from black leg, anthrax and inflammatory edemas.

As distinguishing malignant edema are the occurrence of the disease in a region where symptomatic anthrax is not native. Often the advanced age of the patient excludes black leg. The occasional localization of the tumor on parts of the body poor in muscle, and the insignificant affection of the muscular system in comparison to the severe affection of the connective tissue are observed.

In horses and sheep the development of a crepitating tumor with a fever indicates malignant edema. The crepitation distinguishes the disease from anthrax edema as well as from the inflammatory edemas often following wounds and caused by streptococci. In simple subcutaneous emphysema, following skin wounds, fever rarely occurs. Finally care must be taken that on post mortem a simple emphysema caused by putrefaction is not confused with a crepitating tumor formed during life.

Prevention. In the prophylaxis of the disease the wounds of the skin and the mucous membrane are to be kept from infection from the soil. Wounds that are already infected are to be disinfected. After difficult parturition in which the genital passages have been injured a thorough cleansing of the latter and of the uterus will usually check the development of the disease. Experimental animals can be immunized against the virulent infection by injection of tissue juices

containing spores after heating it to 92° C. for seven hours (Leclainche and Vallée). These inoculations protect only against malignant edema and not at the same time against symptomatic anthrax, as Leclainche and Vallée have proved in contradiction to the earlier experiences of Roux and Dünschmann. There seems to be no successful method for immunizing animals against this infection.

REFERENCES

1. BRESS. Malignes Oedem bei einer Kuh. *Münch. tier. Woch.*, Bd. LVII (1913), S. 620.

2. OPPERMANN. Malignes Oedem beim Schaf und Schwein. *Deutsch. tier. Woch.*, 1913, S. 81.

3. THUM. Zur Diagnose des malignen Oedemes und sogenannten Geburtsrauschbrandes beim Rinde. *Monatsh. f. prak. Tierheilk.*, Bd. XXIII (1911–12), S. 389.

4. WORNER. Malignes Odem der Zunge eines Pferdes. *Münch. tierärzt. Wochenschr.*, Bd. LVI (1912), S. 471.

CHAPTER VI

DISEASES CAUSED BY HIGHER BACTERIA
GENUS ACTINOMYCES

General discussion of the genus. There is considerable confusion regarding the genus Actinomyces. The higher bacteria having filaments showing true branching and that form the so-called rosettes in the tissues are placed here in the genus Actinomyces. There seem to be good reasons for retaining both the generic and specific name of *Actinomyces bovis* given by Bollinger and Harz to the organism, which forms clubs and which causes the lesions in cattle known as actinomycosis. The forms that have filaments with true branching but do not form rosettes in the tissue are placed in the genus *Nocardia* (Streptothrix).

Wright has questioned whether the "actinomyces colony" is an essential product of the microörganism itself, that is, a product analogous to capsule formation among lower bacteria, or a deposit upon the microörganism from the surrounding tissue and fluids. The animal fluids seem to be essential for their production. The function of the clubs or hyalin envelope surrounding the peripheral filaments seemed, according to Wright, to be to protect the mass of the colony from the destructive action of the juices and cells of the tissue. Bostrœm and others have pointed out that the clubs are formed only when there is evidence of resistance on the part of the tissue to the organism. In rapidly spreading cases of actinomycosis, it has been shown that there is little or no club formation.

ACTINOMYCOSIS

Synonyms. Lumpy jaw; wooden tongue; big head.

Characterization. Actinomycosis is a chronic disease determined by the presence of a specific cause—the ray fungus—which by irritation stimulates the formation of new growths consisting of round cells, epithelioid cells, giant cells and fibrous tissue. The neoplasms appear as tumors having either a tendency to develop into large and hard masses or to suppurate. Wright restricts the meaning of actinomy-

cosis to a suppurative process combined with granulation tissue formation, the pus of which contains the characteristic granules composed of the dense aggregation of the organisms.

Cattle (genus *Bos*) are most often attacked. Horses, dogs, cats, sheep, elephants, bears and the human family are susceptible and cases have been reported in each. Other species seem to be immune.

History. The early history of actinomycosis is quite obscure. Prior to the discovery of its specific cause, it was much confused with other diseases resembling it more or less closely in certain gross appearances. It was designated by a variety of names, the more common of which are swelled head, lumpy jaw, big head, fibroma, sarcoma and osteosarcoma. The popular names were probably suggested by the character of the lesions, which differ to a marked degree. It was recognized as a specific disease by Rivolta in 1868, by Perroncito in 1875 and by Bollinger in 1877. The ray fungus was undoubtedly observed prior to this by Lebert and Robin, both of whom failed to recognize it as a vegetable parasite.

The fungus was carefully described by Dr. Harz, a botanist, who gave it the name *Actinomyces* or ray fungus. Bollinger was the first to carefully study the disease in cattle and to demonstrate the power of the ray fungus to produce disease. With this discovery of Bollinger in 1877, actinomycosis became recognized as a definite, specific disease which could in most cases at least be differentiated from the other affections with which it had hitherto been confused. In 1845 von Langenbeck of Kiel observed and made drawings of peculiar bodies in a case of vertebral caries in man which it is now believed were rosettes of the ray fungus. In 1878, Israel demonstrated the disease in man. Since that time it has been carefully studied and described by a number of investigators.

Geographical distribution. Actinomycosis is quite widely distributed throughout North and South America and Europe. It is much more prevalent in certain countries and districts than in others. The observation has been made that animals pastured upon low lands and in river valleys are more liable to contract it than those feeding upon high and dry ground. It has also been noted that cattle fed upon rough or coarse forage are more prone to the disease on account of abrasions of the buccal mucosa than those kept upon less harsh food.

It is very difficult to procure reliable statistics concerning the extent to which it occurs. The observations which have been made at the

union stock yards, Chicago, show one case of actinomycosis in from
1600 to 1700 cattle.   The statistics from the abattoirs in Berlin show
one case to 4150 cattle and one in 8000 pigs.   These figures do not,
however, indicate the extent of the disease among the farm animals,
as they are collected from those animals presented for slaughter only.
At the clinic of the New York State Veterinary College there are pre-
sented for treatment a very few cases each year.   In the Mississippi
Valley and in the Southwest it seems to be more prevalent than it is
east of the Alleghany Mountains.
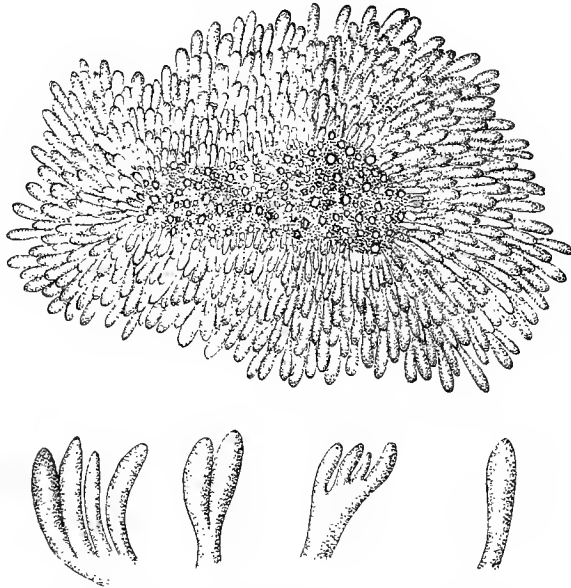
FIG. 53.   A ROSETTE OF THE RAY FUNGUS TOGETHER WITH DIFFERENT FORMS OF THE
SINGLE CLUB ENDS.   X 3500.   (AFTER CROOKSHANK).

Etiology.   Actinomycosis is produced by a higher bacterium
(*Actinomyces bovis* Harz, *Streptothrix actinomyces* Rossi Doria), com-
monly known as the "ray fungus."   The disease is the result of its
multiplying in the tissues and not from the elaboration of a toxin.
Undoubtedly the lower bacteria often associated with it in suppurat-
ing lesions are of some significance.   Wright states that he believes
they play an important part in the extension of the disease.   The
organisms appear as minute, yellowish granules in the lesions.
When examined microscopically, these granules are found to be made

up of rosettes varying in size from 10 to 200μ in diameter, the average size ranging from 30 to 40μ. This fungus can be cultivated on artificial media. It stains somewhat feebly with the aniline dyes.

The rosettes are composed of a number of club-shaped structures (rays), radiating from the central mass which is composed of the mycelial part of the organism. The club-shaped bodies vary in size but usually they are from 1 to 10μ long. The rays are connected with the central portion by fine thread-like structures which are not

FIG. 54. PHOTOGRAPH OF A YOUNG ACTINOMYCOTIC GROWTH UNDER LOW POWER, SHOWING CLUMPS OF THE ACTINOMYCES.

readily demonstrated. In tearing or crushing the rosette, the clubs break off at or near their junction with the mycelial threads. Some investigators have mentioned a polymorphous form of actinomyces in which coccoid and rod-shaped structures are found. These are doubtless the ends of the clubs which first appear in focusing on a rosette.

The natural habitat of this organism is said to be certain plants. According to Brazzola, they vegetate on the grasses, chiefly on *Hordeum murinum*. He discovered quantities of it between the vegetable fibres of barley which were imbedded in the gums of ani-

mals.   Johne, Piana, Bostrœm and others have found it on the awns
of grain which were imbedded in the tonsils of pigs and in the tongues
of cattle.   Mayo, after making a careful study of this disease, states
that the actinomyces are probably a degenerative form of some fungus
which grows naturally upon food stuffs or grain.   Bostrœm enter-
tains the view that they develop exclusively on grains, particularly on
the awns of barley.

The period of incubation is not known.
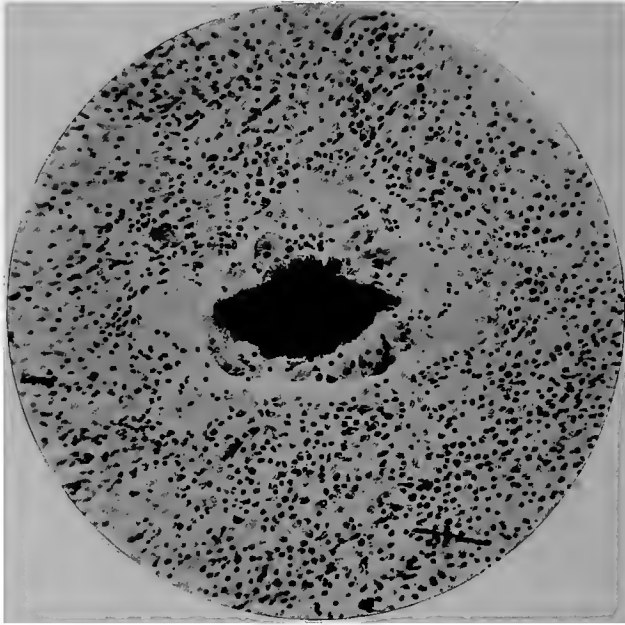
FIG. 55.   PHOTOGRAPH UNDER HIGHER MAGNIFICATION OF ONE OF THE CLUMPS OF
ACTINOMYCES SHOWING GIANT CELLS.

Infection.   While actinomycosis is an infectious disease it does not
seem to be transmitted directly from one animal to another.   Numer-
ous investigators have tried to produce it by inoculating cattle, calves,
sheep, goats, pigs, dogs, cats, rabbits and guinea pigs with actinomyco-
tic lesions.   The results have almost invariably been negative when
pus was used, but the disease has developed after inoculating cattle
with pieces of tissue containing the organism in its vegetating state.

The supposition is that the parasite gains entrance to the living
tissues through slight wounds of the mucous membrane of the mouth
or throat and perhaps the alveoli of diseased teeth or during the shed-

ding of the milk teeth. It multiplies and extends from the points of entrance. After the infected awns once gain lodgment, especially between the teeth, they are removed only with difficulty. The favorite points for the actinomyces to enter the tongue are on the upper surface midway between the dorsum and the tip. The lungs may be the seat of primary infection due to the inhalation of the fungus. The disease rarely has been observed primarily in the udder but requently in the subcutaneous tissue about the head.

FIG. 56. HEAD OF A STEER WITH ACTINOMYCOSIS OF THE LOWER JAW. (PHOTOGRAPH BY HOPKINS).

In man the source of infection is more obscure. Many cases have been reported where the individuals did not come in contact with diseased animals and were not occupied in agricultural pursuits or in handling grain but were glaziers, tailors and various shop tenders. There are a few cases reported, however, where the circumstantial evidence points to direct infection from diseased animals.

The present knowledge indicates that the organisms must attain to a certain stage or period in their development before they will live and multiply in living animal tissues. It has been observed that, as a rule, cattle become infected when they are kept upon dry food.

Symptoms. Actinomycosis is manifested by a firm swelling or tumor usually situated in the region of the head or throat.   It is first recognized as a slight swelling of the affected part resembling somewhat the result of a bruise.   It is stated that many cases of actinomycosis seem to be caused by blows or injuries received while struggling in stanchions.   The enlargement gradually increases in size.   It is ordinarily sharply defined from the surrounding tissues.

Upon manipulation the tumor feels hard and dense. After a variable length of time, the tumor-like mass may soften in one or more places, rupture and discharge a rather thick, yellowish and more or less sticky, purulent substance.   The discharge may continue or, as often happens, the opening heals temporarily only to rupture again. The discharge often takes place into the cavity of the mouth or throat. Sometimes the neoplastic tissue increases in amount until it gradually forces its way through the opening, resembling somewhat a cauliflower in appearance.   The actinomycotic growth frequently increases rapidly in size after it has discharged.   In later stages the teeth may become ulcerated and loose.

When the tongue is affected the animal finds it difficult to eat. The organ is swollen and in advanced cases hangs from the mouth. There is, in these cases, profuse salivation.   When the pharynx is affected there is difficulty in swallowing and when the larynx is attacked there is difficulty in breathing.   In this country the tumor is most frequently seen on the external surface of the jaw.   It is stated by Salmon that it usually begins in the connective tissue beneath the skin but soon extends to the bone, which it penetrates. Actinomycosis of the cervical vertebræ may cause spinal paralysis. When the lungs are attacked the animal may present the appearance of one suffering from a chronic pulmonary disease such as tuberculosis.   There are no symptoms characteristic of pulmonary actinomycosis.

Actinomycosis is not a rapidly fatal disease. Animals rarely if ever die from its immediate effects.   The length of time during which they survive depends very largely upon the location of the tumor and the rapidity of its development.   If it is situated where it does not interfere seriously with prehension, mastication or swallowing of food or where it does not occlude or press upon the respiratory passages the animal usually survives for several years.   When death occurs it is usually due to inanition, the animal being unable to take sufficient food, although the drain upon the system by the long continued dis-

charge of pus must be severe.   Mayo reports several cases where the
disease was watched for five or six years and where it would possibly
have continued several years more had not the animals been destroyed.
Most animals which become affected with actinomycosis are either
destroyed, treated or slaughtered for beef.

  Morbid anatomy.   The new actinomycotic growths have in or near
their centers rosettes of the ray fungus surrounded usually by giant
cells.   These in turn are surrounded by tissue consisting principally

FIG. 57.   A DRAWING OF AN ACTINOMYCOTIC JAW.

of epithelioid and spindle shaped, connective tissue cells, among which
giant cells may appear.   As these cells increase in number they press
against the surrounding tissues, thus producing the hard and dense
tumor-like growths.   This is especially true when they are located in
the connective tissue.   In certain other positions, such as the liver,
the inflammatory cells are surrounded by a fibrous tissue frame-work
which gives to the lesion a honeycomb appearance.   On section a dis-
agreeable "nutty" odor is given off which Mayo considers to be quite
characteristic of the disease.   The outside of the tumor is usually
composed of a dense layer of fibrous connective tissue.   Extending
from the periphery toward the center, the tissue becomes less dense
and is composed largely of epithelioid cells.   In the softer tissue
there are often cavities of greater or less size filled with a viscid,
purulent substance in which the small, yellowish granules, "rosettes,"

can be found.   If the pus is spread in a thin layer on a smooth surface granules composed of the organism can often be seen with the unaided eye.   These pus cavities are usually connected with each other by small sinuses but sometimes they are separated by bands of fibrous tissue.

If the disease is in the bone as it is when the specific organism gains entrance and begins to grow in the interior of the jaw, the bone tissue about the organism becomes in places disintegrated and absorbed and pockets are formed containing the granules.   While the interior of the
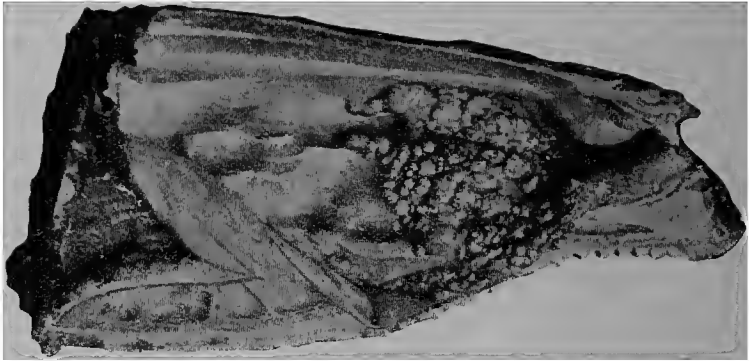
FIG. 58.   ACTINOMYCOTIC NODULES IN THE NASAL CAVITY OF A SIX YEAR OLD COW. (AFTER JOEST).

bone is being broken down and absorbed by the action of the actino-mycotic growth within, its diameter is being increased by the invasion and deposition of new material until it may become several times its normal size.

The lesions spread in most cases by gradual invasion of the tissues surrounding the infected point.   At the seat of infection, minute, inflammatory points appear, which extend at their periphery and unite to form larger areas of diseased tissue.   These masses tend to extend in one direction and to heal in another, leaving behind bands of cicatricial tissue.   The process usually differs widely from that of a simple inflammation.   In its progress the disease shows no preference for structures but invades one tissue after another so that all may be involved alike.

The lymphatics show no constant tendency to become involved. Metastasis occurs in a very small proportion of the cases. When it does, as reported by Ponfick, large areas may be simultaneously affected. He reports a case in the human subject in which the left jugular vein was perforated by a mass of the organisms resulting in the formation of actinomycotic infarcts in the lungs, spleen, brain, and heart.

In cattle, actinomycosis usually appears in one or more of the following locations.

In the maxillary bones. Here it generally results in large tumor-like growths. Actinomycosis of the jaw usually commences with
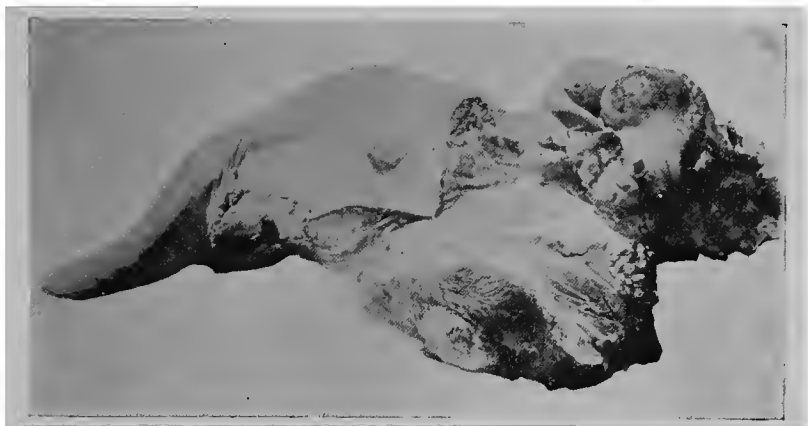
Fig. 59. ACTINOMYCOSIS OF THE TONGUE, "WOODEN TONGUE" (PHOTOGRAPHED BY HOPKINS).

flat granulation of the gums and mucous membranes in the neighborhood of the teeth and spreads finally to the medullary tissues of the bone and to the periosteum, soon giving rise to the osseous tumor. From the maxillary bone the disease may advance either to the subcutaneous connective tissue and the skin or to the oral cavity in the direction of the molar teeth, which become displaced.

In the tongue. When the lesions appear in this organ the disease takes the form of an indurating glossitis. The tongue becomes thickly sprinkled with round or oval, hard, fibrous nodules which finally become purulent or chalky at the center. Around these there is a considerable increase of connective tissue which leads to the atrophy of the muscle fibers. Upon section the tongue is found to be

hard and often gritty.   The indurated tongue may be eroded from
friction and various deformities of this organ are reported.   Jœst has
given a detailed account of the histological changes that take place in
actinomycosis of the tongue and mucous membrane.

In the pharynx.   Here the disease usually takes the form of soft
polypoid or fungoid nodules or lumps with a smooth surface and short
peduncle.   These nodules vary in size often reaching that of a goose's
egg.   These polypoid growths may cause great difficulty in swallow-
ing and likewise interfere with respiration.   Tumors of this kind may
form in the esophagus or trachea.   Rarely actinomycotic growths
occur at other places in the alimentary tract.   There are some cases
in which the lesions are not restricted to the digestive tract.   The
lesions sometimes occur on the nasal mucosa.
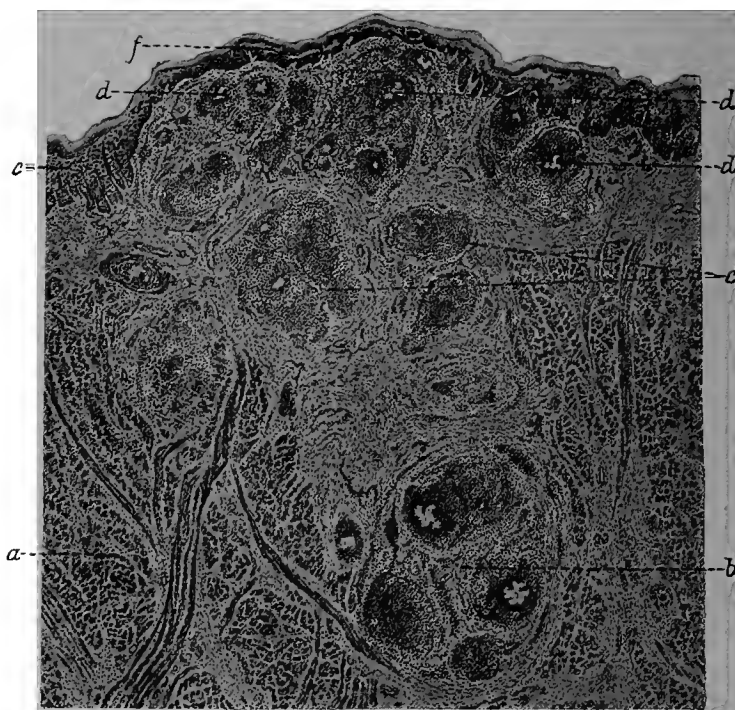
FIG. 60.   ACTINOMYCOSIS OF THE TONGUE.   (*a*) NORMAL TONGUE MUSCLE; (*b*) ACTINO-
MYCOTIC NODULES; (*c*) ACTINOMYCOTIC NODULES IN SUBMUCOUS TISSUE; (*d*) IN
THE PROPRIA MUCOSAE; (*e*) NORMAL MUCOUS MEMBRANE; (*f*) GREATLY THINNED
MUCOUS MEMBRANE.   (AFTER JOEST).

In the skin and subcutaneous tissue. The lesions of the skin and subcutis are found chiefly on the head and neck. They usually consist of firm nodules from the size of a hazel nut to that of a man's fist or even larger. Sometimes these nodules are pedunculated and at others they are attached to the skin by a broad base. Instead of the hard tumor there may occur soft granular fungoid proliferations

Fig. 61. ORDINARY FORM OF SINGLE ACTINOMYCOTIC NODULE IN THE TONGUE. (*a*) ACTINOMYCES; (*b*) CENTRAL ZONE; (*c*) MIDDLE OR INTERMEDIATE ZONE; AND (*d*) PERIPHERY OR OUTER ZONE OF THE NODULE. (AFTER JOEST).

covered with a brown crust or with a purulent secretion. At other times minute nodules appear in these proliferations and the skin becomes thickened and indurated. However, the skin lesions may become very large. In this organ, the disease may be either primary or secondary.

In the lymph glands. Actinomycosis often appears in the lymph glands of the head, larynx and pharynx. The parotid and submaxillary glands are sometimes involved as secondary infections. It is reported that the sub-parotid glands are most frequently affected.

In the lungs. The lesions in the lungs vary. They may consist of firm, somewhat yellowish nodules which eventually become calcareous in their center and vary in size from mere specks to that of a pea. This form is spoken of as miliary actinomycosis. In the second form the actinomycotic foci soften and become filled with a gray muco-purulent fluid. The lesion may spread to the pleuræ and even reach to the surface of the body by penetrating through the thoracic wall. The b r o n c h i a l glands and the mucosa of the air passages may also become affected. The trachea may be the seat of actinomycotic growths.

FIG. 62.   ACTINOMYCOTIC GROWTH (*a*) IN THE TRACHEA OF A COW.

In other organs. Actinomycosis has been reported rarely as attacking the udder, spermatic cord, brain, spleen, liver, muscle, diaphragm, peritoneum, inguinal glands, vagina, uterus and cervical vertebræ. Jœst has reported a case in the kidneys of swine. Actinomycotic peritonitis has been f o u n d i n c a t t l e. This consists of nodules scattered more or less thickly over the serous membrane.

Assmann has recently summarized the literature on the dissemination of the lesions and has concluded that generalized actinomycosis is not rare in occurrence. He gives a detailed description of eleven cases in cattle and hogs.

It is stated by Salmon that in England the disease appears most often in the tongue, in Denmark the soft parts of the head are affected most frequently, while in some parts of Germany it is more commonly found in the pharynx. In the United States it usually appears in the lower jaw. In man as in cattle, the appearance of the lesions varies according to the part infected. In some cases the lesions closely resemble those of chronic inflammation but in others, such as the liver or skin, they are often characteristic. In the lungs the lesions have frequently been mistaken for tubercle. Usually the disease affects the head and if the maxillary bones are attacked the teeth are usually lost.

Actinomycosis in swine. Actinomycosis appears in this species in the lower jaw, larynx, lungs, wounds caused by castration, in the mammary gland, muscles and bones. The character of the lesions does not differ to any marked extent in swine from those in cattle or man. In case of bone infection purulent cavities and sinuses are formed in which the yellow granules of the fungus occur. It is reported that occasionally pigs suffer from generalized actinomycosis. Duncker has found in the muscles of the pig a variety of the ray fungus which has been called *Actinomyces muscolorum suis*, to distinguish it if possible from the bovine species. Its relation to the *Actinomyces bovis* has not been clearly determined. It is reported to have been found frequently.

Actinomycosis in horses and sheep. In the horse, actinomycosis of the bones, tongue, trachea, spermatic cord and submaxillary glands has been observed. The disease is reported to have been mistaken for glanders. The affection known as scirrhous cord seems to be due in rare cases to an infection by the ray fungus. One such case has occurred in the clinic of this institution. A very few cases of this disease have been reported in sheep.

Diagnosis. Actinomycosis is to be diagnosed by the symptoms, lesions and the finding of the specific organism in the affected tissues or in the discharges from them. The presence of the organism in the discharges can often be determined by the use of a good hand lens. At other times a regular microscopic examination is necessary.* In the tissues it is necessary in most cases to make sections for the examination.

---

*In preparing the purulent discharges for a microscopic examination it is usually sufficient to crush one or more of the yellow granules between a slide and cover-glass. It is of advantage to wash it with a dilute solution of caustic soda to clear away the pus cells. The rosettes are easily recognized with a low magnification.

In cattle, actinomycosis is to be differentiated from tuberculosis, especially of the lungs, glands of the throat, head and of the udder and from various forms of glossitis, polypoid growths in the pharynx, fibroma, sarcoma and osteosarcoma of the jaw, parotitis and cellulitis. Occasionally bacterial infection of the maxillary glands gives rise to the formation of tissue which becomes caseous, and causes swelling and firmness of the part suggestive of actinomycotic tumors. Foreign bodies may be wedged in beside the jaw causing an appearance suggestive of actinomycosis.*

In making a positive diagnosis of actinomycosis it is necessary to make a microscopic examination of some of the diseased tissue or of the discharged pus in which the "rosettes" may be found if the disease is actinomycosis. It is impossible to obtain this positive proof from the living animal when the lesion is situated in the internal organs.

Specific treatment. The investigations of Thomassen, Nocard, Nörgaard and the experience of a large number of veterinarians have shown the specific, curative effect of iodide of potassium. According to Salmon, iodide of potassium is given in doses of from 1.5 to 2.5 drams dissolved in water and administered in a drench, once a day. The dose should vary somewhat with the size of the animal and with the effects that are produced. If the dose is sufficiently large there appear signs of iodism in the course of a week or ten days. The skin becomes scurvy, and the eyes moistened. There is nasal catarrh and loss of appetite. When these symptoms appear the medicine may be suspended for a few days and afterwards resumed in the same dose. The cure requires from three to six weeks' treatment. Some animals do not improve with the administration of this drug and these are generally the ones which show no signs of iodism.

If there is no sign of improvement after the animals have been treated four or five weeks and the medicine has been given in as large doses as appear desirable, it is an indication that the particular animal is not susceptible to the curative effects of the drug and the treatment should be abandoned.

It is not, however, advisable to administer iodide of potassium to milch cows, as it will considerably reduce the milk secretion or stop

---

*In one instance a specimen reported to be actinomycosis was sent to our laboratory for examination. It was found to contain a piece of bone about three inches long which had become wedged between the teeth and cheek and surrounded by inflammatory tissues.

it altogether. Furthermore, a great part of the drug is eliminated with the milk making it unfit for use.

There is no diagnostic specific test or biological treatment for this disease.

Sanitary significance. The literature upon this subject is largely to the effect that actinomycosis is rarely if ever either contagious or infectious in the sense that it can be transmitted from one animal to another or from one of the lower animals to man. There seems to be no indisputable case on record of such a transmission, although a few cases are very suggestive. It is the opinion of most pathologists that, when the disease is restricted to small tumors and these are localized, the affected parts should be destroyed but the remainder of the carcass may be used for human consumption.*

REFERENCES

1. ASCHOFF. Ein Fall von primärer Lungenaktinomykose. *Berliner klinische Wochensch,* 1895, S. 738, 765 *u.* 788.

2. BOLLINGER. Über eine neue Pilzkrankheit beim Rinde. *Deutsche Zeits. für Tiermedizin,* Bd. III (1877), S. 334.

3. BOSTROEM. Untersuchungen über das Aktinomykose des Menschen. [*Zieglers Beitrag. zur path. Anat. u. zur allgem. Pathologie,* Bd. IX (1891), S. 1.

4. HARZ. Actinomyces bovis, ein neuer Schimmel in den Geweben des Rindes. *Jahresbericht der Kgl. Zentral-Tierärzneischule in München,* 1877–88, S. 125.

5. ISRAEL. Neue Beobachtungen auf dem Gebilde der Mykosen des Menschen. *Virchow's Archiv.* Bd. LXXIV (1878), S. 15.

6. JOHNE. Die Aktinomykose oder Strahlenpilzerkrankung, eine neue Infektionskrankheit. *Deutsche Zeits. für Tiermedizin,* Bd. VII (1882), S. 141.

7. KITT. Die Aetiologie und pathologische Anatomie der Actinomykose. *Monatshefte für praktische Tierheilkunde,* Bd. II (1891), S. 466.

8. MAYO. Actinomycosis bovis or "lumpy-jaw." *Bulletin No. 35, Kansas State Agric. Exp. Station,* 1892.

9. MOORE. Actinomycosis mistaken for tuberculosis at postmortem following the tuberculin test. *Am. Vet. Review,* Vol. XXX (1906), p. 181.

10. NOCARD. Notes sur l'actinomycose des animaux. *Bul. de la. Soc. Cent. de Méd. Vétér.,* Vol. XLVI (1892), p. 167.

*In Bulletin No. 2, of the Board of Live Stock Commissioners of Illinois, published in 1891, is the report of the somewhat famous trial in the Peoria county circuit court of the case of J. B. Greenhut *et al. vs.* John M. Pearson *et al.* to recover damages for the rejection and destruction of certain actinomycotic cattle, in which is given the testimony of a large number of distinguished veterinarians and sanitarians concerning the wholesomeness of the meat of cattle affected with this disease. Although at that time there was a strong popular sentiment against the use of such animals, the jury after a forty hours' consideration reported their inability to agree and were discharged by the court. The most conspicuous feature of this evidence was the inability of the plaintiff to produce satisfactory evidence of the communicability of the disease from animal to man. This evidence did much to show that the danger from this disease in eating meat of affected animals is after all a matter of opinion, fear or sentiment rather than a demonstrated fact. Mayo states that there is no danger of persons contracting this disease from eating the flesh of affected animals provided the visibly diseased portions are removed.

11. PERRONCITO. Uber den Actinomyces bovis und die Sarkome der Rinder. *Deutsche Zeitschrift für Tiermedizin*, Bd. V (1879), S. 33.

12. PUSCH. Beiträge zur Kenntnis der Lungenaktinomykose. *Archiv. für wissenschaftl. und prakt. Tierheilkunde*, Bd. IX (1883), S. 447.

13. SALMON. Report upon investigations relating to the treatment of lumpy-jaw, or actinomycosis, in cattle. *Bulletin No. 2, U. S. Bureau of Animal Industry*, 1893.

14. SALMON. Actinomycosis or lumpy-jaw. *Annual report, Bureau of Animal Industry*, 1893–4, p. 88.

15. STOLPE. Uber Aktinomykose der Lymphdrüsen bei amerikanischen Rindern. *Zeitschrift für Fleisch- und Milchhygiene*, Bd. XVII (1907), S. 339.

16. WRIGHT. The biology of the microörganism of actinomycosis. *The Jour. of Med. Research*, Vol. XIII (1905), p. 349.

ACTINOBACILLOSIS

Synonym. Streptotrichose.

Characterization. Actinobacillosis is described as an infectious disease of cattle caused by an organism which "resembles, in a marked degree, the bacterium of fowl cholera," characterized by its clinical resemblance to actinomycosis in forming "rosette" or ray-like forms in the tissue. Some workers consider it a variety of actinomycosis while others classify it as a distinct disease.

History. Lignières and Spitz described, in 1902, a disease in cattle resembling actinomycosis but which was caused by a bacterium. Until 1900–01 this affection was not differentiated from actinomycosis. Nocard, in 1902, identified the disease in France. In 1904, Higgins described four cases in Canada. A number of European writers refer to it as *Aktinobacillose*, an atypical actinomycosis.

Geographical distribution. It is reported by Lignières and Spitz to be epizoötic in Argentine Republic. It has been described in France, and in Canada.

Etiology. This disease is caused, according to its investigators, by a bacterium which arranges itself in the tissues in a rosette or ray-like appearance.* It is aërobic, facultative anaërobic, non-motile and of a variable size, ranging between 1.0 and 1.8μ in length and from 0.4 to 0.6μ in breadth. According to Higgins, it has a distinct polar arrangement of the protoplasm as observed in the hanging drop

---

*The original description of the cause of this disease suggests the possibility of a mixed infection. The non-motile organism or form fatal to experimental animals and the "rosette" arrangement as found in later lesions, which resembles the actinomyces rays, give strength to the assumption that there may have been present a septic bacterium and actinomyces or nocardia growing together.

preparation.   It stains with the ordinary aniline dyes.*   It does not take the Gram stain.

In the fresh tissues or in sections it appears in granules the same as in actinomycosis.

The actinobacillose is virulent for guinea pigs and rabbits.   When inoculated into the abdominal cavity with pure cultures guinea pigs die in from nineteen to thirty-one days (Higgins) of generalized actinobacillosis.   These are reported to be characteristic and different from those of any of the other observed infective agents.   According
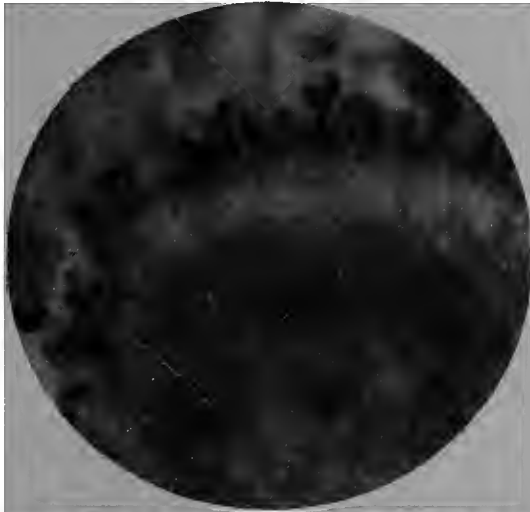
FIG. 63.   A PHOTOGRAPH OF A SECTION OF A TUMOR STAINED BY GRAM'S METHOD BUT NOT FULLY DECOLORIZED.   X ABOUT 1,000.   (AFTER HIGGINS).

to Higgins, "Small pearly-white nodules appear just beneath the peritoneal and pleural membranes, varying from 1.0 to 5.0 mm. in diame-

*Higgins recommends the Romanowsky stain as modified by Dutton and Todd for sections or preparations from pus.   The formula for the stain and method for its use are as follows:

Stain: Eosin, aqueous solution.................................... 1 part
       Borrell's Blue .... 1 "
       Water .... 8 parts

Mix just before using and filter.   Suspend the preparations (sections fixed to the slide) upside down on the stain to saturate them from below, to avoid percipitate. Stain in this solution for thirty minutes.   Wash thoroughly in water, then in a 10% solution of tannic acid, which will brighten the color, and again wash in water.   Dehydrate in alcohol, clear and mount on xylol balsam.   The stain as above prepared spoils quickly.

ter. The liver presents lesions throughout its substance, the surface being mottled. The spleen shows, usually, a varying number of nodules. The great mesenteric fold of the omentum has in every instance been the seat of extensive lesions. The kidneys present nodules beneath their serous covering, but none have been observed in the substance of the organ. The stomach and intestines usually present nodules on their serous surfaces, varying from 1.0 to 0.5 mm. in diameter." There are other lesions such as ulcers in the stomach, nodules in the heart and pericardium.

Rabbits are said to react the same as guinea pigs. Nocard found an intravenous injection fatal to dogs in 24 hours. In horses a local abscess resulted which healed rapidly.

The method of infection has not been fully explained, but it is supposed that the organisms are taken with food as in cases of actinomycosis.

The organisms are said to agglutinate in serum of animals affected with actinobacillosis.

It is destroyed in 10 minutes at 62° C. It grows best at incubator temperature (37° C.). It is not rapidly destroyed by freezing.

Symptoms. They do not appear, in cattle, to be differentiated from those of actinomycosis.

Morbid anatomy. The lesions are very similar to those of actinomycosis. The location of the affected parts varies. Lignières describes it as attacking the skin, lymphatic glands, tongue, pharynx, mammary glands, the viscera, and bones. The tissue changes appear to be an infiltration of purulent material and the new formation of connective tissue. The skin is often affected. In the single specimen which the writer has had an opportunity to study, the ray-like arrangement of the organism and the tissue immediately surrounding it could not be easily distinguished from a section of actinomycosis. The possibility of a double infection with the bacterium and the ray-fungus does not seem to have been carefully investigated.

Diagnosis. Actinobacillosis is to be diagnosed by finding the bacterium and the ray-fungus or the ray-fungus-like organisms. It is to be differentiated from actinomycosis, tuberculosis, perhaps certain parasitic diseases of the skin, and localized bacterial infections.

The diagnosis is assisted by the fact that the lesions are most often found in the skin. The examination of the fresh pus does not reveal the yellow granules as observed in actinomycosis but when squeezed

between two cover-glasses they are reported to be distinct. The organism does not take the gram stain, it is infectious for guinea pigs and rabbits, and is readily cultivated on artificial media. It appears to be transmitted more often in cattle by cohabitation than actinomycosis.

Prevention. As the natural habitat of the cause of *actinobacillosis* is not known, the source of infection is undetermined and consequently effective preventive measures can not be formulated. The fact that it seems to spread from diseased to healthy cattle when placed together necessitates the isolation of the infected.

REFERENCES

1. HANER. Un cas d'actinobacillose chez la vache. *L' hyg. de la viande et du lait* (Ref. Ellenberger Jahresb., 1912).

2. HIGGINS. Actinobacillosis. *Bulletin No. 1, Biological Laboratory, Department of Agriculture, Dominion of Canada*, 1904. Also *Proceedings Amer. Vet. Med. Asso.*, 1904, p. 131.

3. LIGNIÈRES AND SPITZ. Actinobacillose. *Contribution à l'étude des affections connues sous le nom d'actinomycose.* Buenos Aires, 1902.

4. LIGNIÈRES AND SPITZ. L'Actinobacillose. *Recueil de Médecine Vétérinaire*, 1902, p. 546.

BOVINE FARCY

Synonyms. Farcin du boeuf; farcin de France; arboulets.

Characterization. This is a chronic disease of cattle characterized by a suppurative inflammation of the superficial lymphatic vessels and glands and, according to Nocard and Leclainche, due to a *streptothrix*. It is pathogenic for cattle, sheep and guinea pigs but rabbits, dogs, cats and horses are said to be refractory.

History. This disease is reported to have been prevalent in France in former years but at present it is rarely if ever found in that country. It is said to have been studied by Sorillon in 1829 and later by Mousis (1837), Lafosse (1853) and Cruzel (1869).

Geographical distribution. According to Nocard and Leclainche this disease does not exist to any great extent at least in any country except Guadeloupe. It does not seem to have been described from this country.

Symptoms. It attacks cattle of all ages. It manifests itself in the form of circumscribed tumors or cordlike projections which follow the course of the superficial veins on the limbs, more especially on the

internal surface of the metacarpal and metatarsal regions, the forearms and thighs and rarely the neck. In chronic cases the lymphatic glands become swollen and painful.

Morbid anatomy. The nodule swellings or cords rarely form circumscribed abscesses but they frequently become pasty and fluctuate in certain places. It is said not to be common for swellings and suppurative processes to extend to the skin or to cause ulceration. When the swellings are opened they are found to contain a whitish, odorless, sebaceous appearing material of a creamy consistency. The openings rarely suppurate but heal quite promptly. In some cases the lungs, liver, spleen and internal glands are sprinkled with nodules the central part of which have undergone caseous or purulent changes.

Diagnosis. This disease must not be confused with epizoötic lymphangitis, skin tuberculosis or skin actinomycosis.

REFERENCES

1. CRUZEL. Traité des maladies de l'espèce bovine. 1869.
2. MAUSIS. Mémoire sur le farcin. 1869.
3. NOCARD. Note sur la maladie des boeufs connue a la Guadeloupe sous le nom de farcin. *Ann. de l'Inst. Pasteur*, Vol. II (1888), p. 293.
4. SORILLON. Exemples de farcin dans le boeuf. *Recueil de méd. vétér.*, Vol. LXXIX (1829), p. 651.

NOCARDIOSIS

History. In 1910 Burnett described a pneumonia in cattle due to a new species of Nocardia (Streptothrix) which he designated *Actinomyces pulmonalis*. It was found in two animals that had been slaughtered under suspicion of tuberculosis but neither of them had reacted to tuberculin. The lesions found were unlike those of tuberculosis and upon more careful examination were found to be due to an organism resembling Sanfelici's second group, type *Streptothrix flava*, though differing from it in the color of its growth on agar.

Etiology. *Nocardia pulmonalis* appears in the tissues as short and longer filaments, resembling the mycelium of some fungi except that they are more slender. They exist in the tissue as thread-like filaments which become more or less matted or clumped together. They grow in bouillon and milk, forming colonies on the sides of the tube. The growth on agar after two days resembles that of boiled sago and adheres to the agar surface. On potato the growth is more luxuriant. On gelatin it develops a whitish growth with branched filaments

growing out into the medium giving an arborescent, translucent zone about each colony. Gelatin is liquefied.

Morbid anatomy. The knowledge of the morbid anatomy is confined to the cases described by Burnett. For assistance in differentiating lung lesions in cattle the following quotation is taken from Burnett's report.

"Principal lobes of the lungs are not collapsed, ventral and cephalic ones are partially collapsed. On the surface of the principal lobes of both lungs are several depressed scars with radiating depressions. The surface of the lungs is mottled with areas of a greyish color
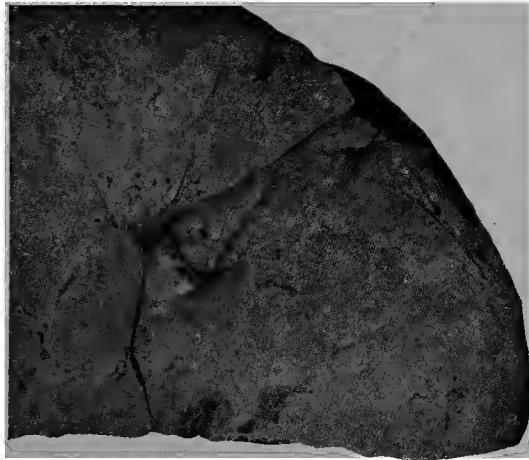
FIG. 64. SURFACE OF LUNG, COW, SHOWING DEPRESSED SCARS, AND RADIATING LINES. ABOUT ONE-HALF NATURAL SIZE (*Burnett*).

involving from one to several (6–12) lobules, with areas of normal lung in which the interlobular tissue is emphysematous, and with areas varying from 2.5 to 7 cm. in thickness in which the interlobular tissue is very much thickened as indicated by streaks of greyish fibrous tissue.

"The lungs throughout are sprinkled with firm nodules varying from 2 to 15 mm. in diameter. There are several areas in which these nodular masses are coalescent, giving firm masses from 3 to 7 cm. in diameter. These exist near the surface and within the tissue of the lung. Upon section the smaller nodules are greyish in color and

apparently homogeneous; the older nodules have a yellowish tinge and upon pressure a yellowish pasty substance is expressed from the surface in many places, giving the appearance of vegetables passing

FIG. 65. SECTION OF LUNG, COW, SHOWING NODULES AND LARGER CONSOLIDATED AREA AND INCREASED INTERLOBULAR CONNECTIVE TISSUE. (a) SCAR ON PLEURA, (b) NODULE (*Burnett*).

through a colander. In the old nodules the center is caseous, soft and yellowish in color. The larger masses, composed of several of these smaller nodules, show on section yellowish areas varying from 2 to 10 mm., separated by bands of fibrous connective tissue varying from .5 to 2 mm. in thickness. Radiating from these areas the interlobular tissue is very much thickened, the lung tissue being more or less collapsed. In a few of these nodules there are hemorrhages.

"The lymphatic glands are very much pigmented and contain areas that are indurated and very firm to the touch. No calcification was found in any of the nodules or in the lymph glands."

|, *Histological examination.* A section of a small (young) nodule shows that it is composed of alveoli separated by a pronounced stroma of connective tissue. In most of the alveoli are desquamated and proliferated epithelial cells, which show parenchymatous degeneration, some showing clear drops (mucus). In some alveoli are polynuclear leucocytes, mostly showing degeneration, and a few eosinophiles, some red corpuscles and fibrin. Some alveoli contain irregular felted masses of threads of bacteria. These masses do not stain well. Individual threads may be seen projecting from the masses, but the main body is a tangle of filaments in which one can not follow individual threads for any distance. Between the alveoli the connective tissue is much increased, in places as wide bands, in others as narrower bands. The appearance under low magnification resembles that of an adenoma. The connective tissue is largely fibro-elastic. Many polynuclear leucocytes are found around the peripheral part of the nodule. The smaller bronchioles contain polynuclear leucocytes, masses of bacteria and desquamated epithelial cells. The bacterial masses in the young nodules are most abundant in the peripheral part of the nodules.

"In larger (older) nodules and in scars the alveoli contain proliferated and desquamated epithelial cells, some contain fibrin, red corpuscles, leucocytes and, in some are masses of bacterial filaments. The interlobular tissue is thickened and consists mostly of elastic tissue. The interalveolar tissue is widened, consisting of elastic tissue with some polynuclear leucocytes and fibrin."

Diagnosis. The diagnosis is made by finding the organism in the nodules or pneumonic areas.

REFERENCES

1. BERESTNEW. Ueber Pseudoaktinomykose. *Zeitschr. f. Hyg.*, Bd. XXIX (1898), S. 94.

2. BIRT AND LEISHMAN. A new acid-fast streptothrix, pathogenic to man and animals. *Journ. of Hyg.*, Vol. II (1902), p. 120.

3. BUCHHOLTZ. Ueber menschenpathogene Streptothrix. *Zeitschr. f. Hyg.* Bd. XXIV (1897), S. 470.

4. BURNETT. A preliminary report on a pneumonia in cattle due to a new species of actinomyces (Streptothrix). *Report N. Y. State Vet. College*, 1909–10, p. 167.

5. CAMINITI. Ueber eine neue Streptothrixspecies und die Streptothriceen im allegemeinen. *Centralbl. f. Bakt.*, Bd. XLIV (1907), S. 193.

6. FLEXNER. Pseudo-tuberculosis hominis Streptothricha. *Journ. of Exper. Med.*, Vol. III (1898), p. 435.

7. FUCHS. Ueber Färbbarkeit der Streptotriceen nach Methoden der Tuberkelbacillenfärbung. *Centralbl. f. Bakt.*, Bd. XXXIII (1902–03), S. 649.

8. GASPERINI. Versuche über das Genus "Actinomyces." *Centralbl. f. Bakt.*, Bd. XV (1894), S. 684.

9. LEVY. Die Wachstums und Dauerformen der Strahlenpilze (Aktinomyceten) und ihre Beziehungen zu den Bakterien. *Centralbl. f. Bakt.*, Bd. XXXIII (1902–03) S. 18.

10. LIGNIÈRES ET SPITZ. Contribution à l'étude à la classification et à la nomenclature des affections connues sous le nom d'actinomycoses. *Centralbl. f. Bakt.*, Bd. XXXV (1903–04), S. 294.

11. LUBARSCH. Zur Kentniss der Strahlenpilze. *Zeitschr. f. Hyg.*, Bd. XXXI (1899), S. 187.

12. MacCALLUM. On the life history of Actinomyces asteroides. *Centralbl. f. Bakt.*, Bd. XXX (1902), S. 529.

13. NESCHCZADIMENKO. Ueber eine besondere Streptothrixart bei der chronischen Eiterung des Menschen. *Centralbl. f. Bakt.*, Bd. XLVI (1908), S. 573.

14. NOCARD. Note sur la maladie des boeufs de la Guadeloupe, connue sous le nom de Farcin. *Ann. de l'Inst..Pasteur*, Vol. II (1888), p. 293.

15. NORRIS AND LARKIN. Two cases of necrotic broncho-pneumonia with Streptothrix. *Journ. of Exper. Med.*, Vol. V (1900–01), p. 155.

16. RABE. Ueber einen neu entdeckten, pathogenen Mikroorganismus beim Hunde. *Berlin. tierärtztl. Woch.*, 1888, Nrs. 43 u. 44.

17. SANFELICE. Ueber die pathogene Wirkung einiger Streptothrix (Actinomyces) Arten. *Centralbl. f. Bakt.*, Bd. XXXVI (1904), S. 355.

18. SCHMORL. Ueber ein pathogenes Fadenbacterium (Streptothrix cuniculi). *Deutsche Zeitschr. f. Thiermed.*, Bd. XVII (1891), S. 375.

19. SCHULZE. Untersuchungen über die Strahlenpilzformen des Tuberculoseerregers. *Zeitschr. f. Hyg.*, Bd. XXXI (1899), S. 153.

20. SILBERSCHMIDT. Sur un nouveau Streptothrix pathogène. *Ann. d. l'Inst. Pasteur*, Vol. XIII (1899), p. 841.

CHAPTER VII

DISEASES CAUSED BY FUNGI

General statement. There is a large literature on the relation of fungi to disease in man and animals. The number of fungi that grow in living animal tissues, however, is not large and those that are thus facultatively parasitic are not often encountered. For these reasons, the more common morbid conditions produced by them will be briefly described without special reference to their classification.

ASPERGILLOSIS

Characterization. The term Aspergillosis has been given to the morbid changes or diseases in different species of mammals and birds caused by the genus *Aspergillus*. This genus of fungi has been found to grow in the tissues of a number of species of animals. The literature contains a considerable number of reports of cases of *Aspergillosis*. The lesions encountered, as a result of the growth of this fungus, are largely restricted to the respiratory tract. Cadeac, Schneidemühl, Friedberger and Fröhner, Ostertag, Kitt and others have called attention to mycotic pneumonia or pneumomycosis. Rénon considered the lesions resulting from aspergillus infection as a pseudo-tuberculosis which he thought should be designated as *Aspergillar tuberculosis*.

The genus *Aspergillus* belongs to the mucedinæ or moles. Although there are several species in the genus, *A. fumigatus* is the one most commonly found in animal tissues. *A. malignus* has been reported to occur in the ear of man and to be pathogenic for rabbits. The infection takes place through ingestion or inhalation of the spores only. It has been observed in tissues other than the lungs and it is exceedingly rare for aspergillosis to pass directly from one animal to another. Although it is widely distributed on forage it does not appear to cause any trouble until some favorable condition enables it to multiply in the animal body. It is said to become more virulent when passed from one animal to another. In birds infection commonly follows the ingestion of grain charged with spores and the

entrance of the latter into the air passages is favored by dryness of the grain. Neumann states that aspergillosis is common in Italian pigeons sold in Paris. Many of them show large mycotic swellings. Its localization in the beak of the pigeon gives it special prominence.

It is thought by many authors that horses become infected when they are obliged to live on mouldy oats or forage. The extent to which the so-called "forage poisoning" is due to this fungus is not known.

Description and method of cultivation. *A. fumigatus* grows on most of the ordinary culture media used in bacteriology if the reaction is acid; it develops poorly in alkaline media. The well-known Raulin's fluid is reported to be the best medium for its cultivation, especially where the aspergillus must be isolated from mixed growths, as in the examination of sputum. Sabourand's* solution of maltose also gives good results.

For ordinary use potato, with or without glycerin, gives excellent results. A paste made by rubbing up crumbs of stale bread in water is also a good medium. Growth is said to be more rapid, however, in Raulin's fluid than in any other medium, the mycelium appearing in from five to twelve hours and spores forming in from twelve to fifteen hours. The growth is first a velvety white, soon becoming a delicate bluish-green, which grows darker. On Raulin's fluid it changes after some days to a dark brown. Cultures on potato retain the green color for a long time, while those on bread paste become brown.

The fungus retains its vitality in cultures for many months unimpaired. Its development has been reported when inoculated from cultures three or four years old. Spores do not form in a temperature below 20° C. and like the mycelium they require fresh access to oxygen for their best development. They measure 2.5 to 3μ in diameter. In nature the spores are widely distributed but seem to be especially abundant in grain and vegetable matter. They have considerable power of resistance to heat and to chemical agents. They are killed by a temperature of 60° C. in five and one-half hours. In moist heat

---

*The formula recommended by Ravenel is as follows:

Maltose,	3.70 grams.
Peptone,	0.75 grams.
Distilled water,	100.00 c. c.

To this may be added gelatin or agar to solidify it, the latter being preferable, as the aspergillus grows best and forms fruit best at 37° to 39° C.

and in solution of bichloride of mercury 1 to 1,000 they are destroyed in fifteen minutes.

Aspergillus fumigatus is differentiated from other species by its color in cultures, the high temperature at which it grows, the size of the spores and by its pathogenesis. *Aspergillus glaucus* is the one most likely to be confounded with it. It may be differentiated from *A. fumigatus* by its ability to grow at low temperature, its delicate green color, the large diameter of its spores—9 to 15µ —and its lack of pathogenic power.

The mode of infection is through the respiratory tract. Only a small number of the spores inspired are able to reach the alveoli, the greater number of them being arrested in the tracheal and bronchial secretions. Hildebrant has shown that, having reached the alveoli, they penetrate the epithelial lining without difficulty. Both animals and man seem to possess immunity to intestinal infection.

The aspergillus does not form toxins. Its pathogenic power is due entirely to lesions produced by the masses of mycelium which cause a necrosis of the cells and a leucocytic reaction which diminishes the functions of the organs, the final result being an enfeebled condition of the animal and a lessened resistance to hurtful influences. When fruit hyphæ can form, the myriads of spores given off by them may be carried to other parts of the organ. In this way the foci rapidly multiply and practically the entire organ becomes invaded. The opinion held by some authors that in the mould mycoses there is "no fructification or actual multiplication" of the infected agent and that the "number of the diseased foci corresponds exactly with the number of spores introduced," is erroneous both for the disease naturally contracted as well as for the experimental form. In the produced lesions, fruit formation of the fungus is exceptional. It has been observed by Renon to take place only where there is full communication with the air.

Aspergillosis in cattle. Aspergillosis in cattle was described by Zürn in 1876. In 1900, Pearson and Ravenel described an interesting case of pneumomycosis in a six year old cow due to *A. fumigatus*. This seems to be the only carefully described case of this infection in cattle reported in American literature.

The cow had been in poor condition for six months prior to bringing her to the Veterinary Hospjtal where she was tested with tuberculin with no reaction. At this time she did not eat, was weak and depressed, respiration labored and from 40 to 90 per minute. Pulse rapid. Percussion of the chest walls gave a sound that, if anything,

was clearer and louder than the normal percussion sound.   Upon auscultation it was found that the vesicular and bronchial murmurs were considerably increased in intensity and accompanied here and there by sibilant râles.   She coughed violently at times.   Six days after she came to the hospital the breathing became more rapid and difficult and the pulse very much accelerated.   The animal did not eat, grew weak rapidly and died four days later, or ten days after admission to the hospital.

FIG. 66.   COMPOSITE DRAWING OF SECTION OF LUNG THROUGH NODULE OF ASPERGILLUS ORIGIN.   F, FIBRIN IN ALVEOLI.   S, FRUIT HYPHAE AND SPORES OF FUNGUS (*Ravenel*).

The anatomical changes given here are restricted to the case of Pearson and Ravenel. The most striking feature on external examination was the extreme amount of emphysema.   The lobules were separated from each other by 3 to 5 mm. and even at some

distance from the borders one could see through the crevices by transmitted light. On the surface, the sub-pleural connective tissue was distended by large blebs. Upon palpation the lung crackled and numerous hard nodules could be felt. On section numerous dark red nodules appeared in the surrounding normal tissue. In each lung there were from fifty to sixty of these nodules, from 5 to 12 mm. in diameter. Most of them were dark red and closely resembled partially organized blood clots. However, on crushing a portion in glycerin between two slides and examining it under the microscope, they were found to be made up almost entirely of a felted network of mycelial threads. Between these large nodules there were numberless smaller areas of much the same color, 1 to 2 mm. in diameter, not perceptible to the touch as nodules but which were of the same character and were no doubt foci of recent origin. These were seen especially well in portions of the lungs which were preserved by Pick's method, the slight bleaching of the tissue bringing them into relief. On opening some of the interlobular emphysematous spaces, small, whitish, mouldy looking patches were noticed which bordered the cavity. Scrapings of these patches were made up entirely of perfect fruit hyphae, with myriads of spores. The diagnosis of a mould mycosis was in this way made at once and confirmed by cultures and examination of sections. Cultures were obtained on glycerinated potato, bouillon and plain agar, by opening a nodule with sterile instruments and tearing out a small portion of the center, which was transferred to the culture tubes and placed in an incubator at 39° C. A rabbit was inoculated in the aural vein with one-half cubic centimeter of a suspension of the spores. The animal died in forty-four hours and from the liver and kidneys cultures were recovered. All of the organs were examined in sections, but the mycelium was detected in two only.

The histology was studied largely in sections stained with hematoxylin and eosin. The bronchial epithelium was normal in places, but, for the most part, the columnar cells had been replaced by a sort of membrane, which appears to be made up almost entirely of a felt-work of mycelial threads. From this membrane hyphæ grew out into the lumen of the bronchus, and here, owing no doubt to the supply of air, fruit hyphae arose, with perfect sterigmata and spores. There were no cellular nor other exudate and very little debris. The under surface of this membrane was of looser texture and contained some cellular infiltration made up of round cells, leucocytes, proliferated connective tissue cells and red blood corpuscles. The adjacent structures were closely filled with a cellular infiltration with a quantity of mycelium of the same description, this extended to the neighboring alveoli, which under low power appeared to have preserved their outline but with greater amplification were seen to have lost all their normal structure, showing clumps of homogeneous, irregular masses which stained faintly with eosin and were probably of connective tissue origin.

In these areas the mycelium followed the alveolar wall as a trellis, the tissue seeming to afford no obstacle to its advance. Within the alveoli was a finely granular debris, with some coarser particles, probably the remains of cells. In sections stained with carbol-thionine large numbers of mast cells were seen in the alveolar walls. Bordering these degenerated areas were alveoli which had retained their normal structure and were filled with a network of fibrin holding in its meshes a few cells. In other parts of the sections were areas resembling those just described, but in which all anatomical landmarks had been destroyed, so that it was impossible to tell whether or not the spaces seen were bronchi.

Some sections showed a widespread interstitial and alveolar hemorrhage, the blood showing a considerable increase in the number of leucocytes. The capillaries were congested and areas of edema with thickening of the alveolar walls were not uncommon.

There were peribronchitis and arteritis, while in some sections arterial thrombosis was seen, the thrombus being penetrated by mycelium, though no fruit was found. Areas were also found in which the alveoli were filled with a cellular exudate producing consolidation and thickening of the alveolar walls.

Emphysema, both interstitial and vesicular, were marked and often extreme. Around the borders of the interstitial cavities was a distinct zone made up of red blood cells, leucocytes and homogeneous material, which was yellowish in fresh as well as stained sections. These areas contained very little mycelium. All sections showed a small amount of anthracosis. The appearance of sections varied in different nodules as they were taken further and further away from the center. In general the fungus was thickest at the center, so thick in many instances that the lung tissue was hidden entirely, and grew less as we went outward. The tissue changes noted took place in a zone beyond the greatest growth of the fungus. In other nodules the fungus was evenly distributed throughout, following the alveolar walls. In these the· tissue changes were slight. At times the fungus grew in dense, brush-like clusters, closely resembling actinomycosis under low amplification.

This form was considered to show a marked reaction and resistance on the part of the animal and a lowered vitality in the fungus. When found it indicated that the aspergillosis was a primary and not a secondary or terminal affection. No giant cells were found in any section.

Fruit formation was not observed in the substance of the tissues at any time. It was observed most frequently in bronchi, which were for the most part denuded of their epithelium, and next in emphysematous cavities, where it could be detected in clusters by the naked eye. Fruit was found in sections, in spaces the nature of which it was impossible to determine accurately. Whenever the formation of fruit was seen, there were innumerable free spores as well as those still attached to the sterigmata, but in no case were spores detected in the substance of the tissues.

In many sections, especially those from near the center of the nodules, the mass of mycelium was so dense that the structure of the tissues was obscured. Besides the dense growths resembling actinomycosis already described, other brush-like clusters not unlike them were frequently seen. These differ from the former in being somewhat less compact, and that from their periphery numerous hyphae run out into the surrounding tissues, whereas in the actinomycotic form the masses are sharply defined and only here and there a few threads grow out beyond the clusters. Their appearance suggests that they may be actinomycotic forms which have finally overcome the resistance of the tissues. Emphysema is less marked in the neighborhood of the latter.

Diagnosis. Pulmonary aspergillosis in cattle is diagnosed by finding the fungus in the tissues or the detection of the fungus in the discharges. The symptoms suggest those of pulmonary tuberculosis. There is thus far no specific reaction for detecting the presence of this infection. The course of the disease is chronic.

Aspergillosis in sheep. Mazzanti, in 1891, found in the lung of a lamb a number of nodules. These varied in size from a poppy seed to that of a hemp seed and were scattered throughout the lung. They were surrounded by a zone of hepatization. Upon examination he found the nodules to contain a purulent substance in which were spores and mycelia radiating outward from the air cells. He did not positively identify the fungus.

Aspergillosis in horses. Rivolta, in 1856, mentioned the invasion of the sinuses of the head and lungs of a horse with a fungus. Later he described other cases and gave the name *Gutturomycosis* to the disease and *Gutturomyces equi* to the fungus. Mazzanti, in 1902, reported a case of this infection. Pneumomycoses have been reported in horses by Martin. Varnell and Mitchell reported an outbreak among horses due to eating oats covered with an *Aspergillus*. They obtained fatal results from feeding aspergillosed oats to healthy horses.

Symptoms. The symptoms depending upon the location of the lesions are those of sore-throat, stomatitis, bronchitis, or pneumonia due to foreign bodies. Unless there is inflammation of the lungs there is usually no rise of temperature. There is depression and disinclination to move. Because of the soreness of the throat little food is taken and loss of flesh follows. If the lungs are involved the symptoms of foreign body or gangrenous pneumonia are exhibited.

Lesions. The lesions vary according to the seat of infection. If on the mucous membranes of the pharynx or guttural pouches, the membranes are swollen, contain muco-pus and are either light greyish or dark in color. Slightly raised patches may appear which resemble those of diphtheritic false membranes. They are composed of mycelia and spores of *A. fumigatus*, bacteria and inflammatory products. If the lungs are involved there are scattered throughout them areas of consolidated lung tissue, varying in size from 0.5 to 10 or more centimeters in diameter. The center of the nodules may be purulent or caseous depending upon their age. Sections of the nodules show the fungus on microscopic examination. Hoare described nodules in the liver and kidney containing the fungus with parenchymatous degeneration of the organs.

Diagnosis. Aspergillosis is to be diagnosed by finding the fungus or the spores in swabs from the throat and from the symptoms and

lesions. The pulmonary form may be diagnosed by finding the fungus in the tracheal discharges and by finding the fungus in the lesions after post mortem. It is to be differentiated from strangles, influenza and other forms of pharyngeal trouble.

Aspergillosis in the dog. Rivolta, in 1885, observed in the organs of a dog what he called *"Encephaloid sarcomata"* containing filaments interlaced in various directions. He called the fungus *Muromyces canis familiaris*. Stazzi found *A. fumigatus* in a dog which suffered from epileptiform convulsions and which on post mortem showed brownish patches in the inferior turbinated bone which consisted of fungi and inflammatory products.

Aspergillosis is liable to be found in any species of mammals. The work of Lucet on this fungus in domestic animals indicates that it is of much more etiological significance than is generally believed. Any lesions of an unknown, undetermined nature should be carefully examined for the presence of this fungus.

Aspergillosis in birds. Aspergillosis in birds was first observed by Mayer in 1815, who found the bronchi and air sacs of a jay filled with this fungus and inflammatory products resulting from it. Since that time a large number of workers have described lesions in birds and fowls due to a fungus. Lucet gives a list of over 40 recorded observations on the pathogenesis of aspergillosis in birds. The disease is usually local and its manifestations vary according to the location and extent of the infection. In many cases it runs a very short and apparently harmless course while in others it is more chronic. If the lesions are extensive, the fowls become anemic, emaciated and die as a result of mechanical interference such as an occlusion of the trachea or from exhaustion. It is more fatal to the young than adult and older birds. The lesions are sometimes found in the nasal passages, air sinuses of the long bones, in the connective tissue about the joints, in the mouth, trachea, esophagus and internal organs. They appear either as a membraneous growth on the surfaces or in the form of nodules. The nodules vary considerably in size and contain a caseous center, surrounded by a reactionary zone. The fungus is found within the nodules.

Mohler and Buckley described a case of this affection in the lung of a flamingo which died at the National Zoölogical Park in Washington. In this case the bird was very much emaciated. The lungs presented lesions suggesting those of tuberculosis in their general

appearance. The other organs (liver, spleen and kidneys) appeared to be normal. From the lung *A. fumigatus* was obtained.

In 1913, Archibald describes a case of aspergillosis in the Sudan ostrich. The lesions were in the lungs and bronchioles. The upper portion of the bronchus was lined with dark, granular-looking masses which projected into the lumen. They were friable but firmly adherent to the wall. There were several plaques raised from the surface. These consisted, for the most part, of dense fibrous tissue with the granular amorphous areas devoid of cellular elements. Within these were mycelial filaments. Many of them showed branching and club-shaped dilations at their extremities. Morphologically it was not unlike *Aspergillus fumigatus*. Jowett describes a pulmonary mycosis in an ostrich in which the lung was thickly sprinkled with nodules that upon section exhibited three zones, a necrotic center, a deeply stained infiltrated zone and surrounding this an area in which the air cells were still more or less pervious. These areas contained spores (conidia).

Aspergillosis is somewhat prevalent in poultry and pigeons, especially those that are forcibly fed. This fungus sometimes finds its way into the eggs and causes them to decay promptly on incubation. In infected incubated eggs more or less developed dead embryo chicks may be found. The infection of the egg is supposed to be by means of the spores passing through the pores of the shell. Hoare states that "the microscopical examination of sections of the decalcified shell shows an abundance of branched fertile mycelia on the free face of the shell membrane, and a compact felted mass of luxurious mycelia infiltrating the whole thickness of this membrane, and penetrating the interstices of the albuminoid meshwork of the shell."

REFERENCES

1. ARCHIBALD. Aspergillosis in the Sudan ostrich. *Journ. of Com. Path. and Therap.*, Vol. XXVI (1913), p. 171.

2. ARWINE AND LAMB. A fifth case of "fungous foot" in America. *The Amer. Jour. of Med. Sciences*, Vol. CXVIII (1899), p. 393.

3. BALFOUR. Aspergillary pneumokoniosis in the lung of a turkey. *Fourth Report, Wellcome Tropical Research Laboratories*, 1911.

4. BODIN ET GAUTIER. Note sur une toxine produite par l'Aspergillus fumigatus. *Annales de l'Inst. Pasteur*, 1906, p. 209.

5. DINWIDDIE. On the toxic properties of moulds. *Bulletin No. 40, Arkansas Agric. Exp. Sta.*, May, 1896.

6. DUBREUILH. Moisissures parasitaires de l'homme et des animaux superieurs. *Archives de Médécine Expérimentales et d'Anatomie Pathologique*, 1891, p. 428.

7. FLEXNER. Pseudo-tuberculosis hominis streptothricha. *The Journal of Experimental Medicine*, Vol. III (1898), p. 435. (Bibliography).

8. GRAY. Aspirgillosis. *Hoare's System of Veterinary Medicine*, Vol. I (1913), p. 1206.

9. JOWETT. Pulmonary mycosis in the ostrich. *Journ. of Comp. Path. and Therap.*, Vol. XXVI (1913), p. 253.

10. LIGNIÈRES ET PETIT. Péritonite aspergillaire des dindon. *Rec. Méd. Vét.*, Vol. LXXV (1898), p. 145.

11. LUCET. De l'Aspergillus fumigatus chez les animaux domestiques et dans les oeufs en incubation. With bibliography. Paris, 1897.

12. MÉGNIN. Médicine des oiseaux. Fourth edition, Vol. I, p. 209.

13. MOHLER AND BUCKLEY. Pulmonary mycosis in birds—with a report of a case in a flamingo. *Annual Report of the Bureau of Animal Industry, 1903.* (*Also issued as circular No. 58.*)

14. NEUMANN. Aspergillosis in domesticated birds. *Journ. of Comp. Path. and Therap.*, Vol. XXI (1908), p. 260.

15. OPHÜLS AND MOFFITT. A new pathogenic mould. *The Philadelphia Med. Journal*, Vol. VI (1900), p. 1471.

16. PEARSON AND RAVENEL. A case of pneumonomycosis due to the Aspergillus fumigatus. *The University Medical Magazine*, Aug. 1900. *The Vet. Journal, New Series*, Vol. II (1900), p. 229.

17. RÉNON. L'étude sur l'aspergillose chez les animaux et chez l'homme. 1897.

18. RIES. Gutturomycosis in the horse. *Journ. of Comp. Path. and Therap.*, Vol. XVI (1903), p. 383.

19. ROQUET. Aspergillose broncho-pulmonaire du faisin. *Soc. des Sciences Vétérinaires de Lyon*, 1912.

20. STAZZI. Nasal aspergillosis in the dog. *La Clinica Veterinaria*, 1905, Nos. 34, 36 and 38.

21. VARNELL AND MITCHELL. Death of several horses from feeding on oats affected with fungi. *Veterinarian*, 1862, p. 65.

22. WEIS. Four pathogenic tourlæ (Blastomycetes). *The Jour. of Med. Research*, Vol. VII (1902), p. 280.

EPIZOÖTIC LYMPHANGITIS

Synonyms. Japanese farcy; pseudo-farcy; equine pox; equine syphilis; inundation fever.

Characterization. Epizoötic lymphangitis is described as a virulent infectious disease characterized by suppuration of the superficial lymphatic vessels, due to the presence of a specific organism. It is a disease of the solipeds, although Tokishige reports finding it in cattle in Japan.

History. This affection seems to have been known for a very long time and to have been confused with cutaneous glanders (farcy). French veterinarians have recognized the disease as river farcy, *farcin en cul de poule,* curable or benign farcy. In France these various forms were acknowledged to be identical, the "river farcy" being considered as an attenuated form of glanders (farcy) until 1873, when Rivolta discovered the specific organism (*Saccharomyces farcimino-*

sus). This affection has been recognized at different times in Japan, China and India. It has been known in Algiers for many years and during the war in South Africa it seems to have been introduced there. From South Africa it has been imported into England and Ireland by government horses returning from the Cape. The first case in England appears to have been detected in 1902. In 1907, Pearson discovered it in western Pennsylvania. Gasparini and, later, Ducloux in 1908 described the cryptococcus in every detail, as it has been observed by all writers on this subject, but avoided any discussion as to the possibility that the organisms belong to the yeasts, and they created, therefore, a new genus and species: *Lymphosporidium equi* (Gasparini) and *Leucocytozoon piroplasmoides* (Ducloux), respectively. Galli-Valerio in 1909 expressed the opinion that the cryptococcus has remarkable similarity to the Leishmanioses and he therefore proposes the name of "*Leishmania farciminosa*" (Rivolta).

Meyer, who studied this disease in South Africa and in the United States, concludes that the disease which was diagnosed as "epizoötic lymphangitis" in 1907 in Pennsylvania was sporotrichosis. "Epizoötic lymphangitis" apparently does not exist in this country. Morphologically, by cultures and serum tests, the two diseases can easily be separated. In horses the parasite of sporotrichosis is very rare in the pus and can rarely be demonstrated microscopically. Page, Frothingham and Paige made a careful study of material from two cases in the Pennsylvania outbreak and found that the organism was a sporothrix which they cultivated in pure culture. They found it to be pathogenic for horses, mice and rats.

Etiology. This disease is caused by an organism described by Rivolta as *Saccharomyces farciminosus*. It is also called a cryptococcus. According to Pallin, it is found in large numbers in the diseased tissues and products, partly free and partly enclosed in pus corpuscles, which often contain from ten to thirty or more of them. It is characterized by its clearly defined contour and its very refractile double outline. It measures from 3 to 4μ in diameter, and in the unstained preparations it is said to be best seen with an oil immersion and Abbé condenser, under a magnification of not less than 800 diameters. In stained preparations it can be recognized with a much lower magnification.

The classification of this organism has been much discussed by several workers. Canalis places it with the coccidia, Piana and Galli-

Valerio consider it as belonging to the protozoa, and Formi and Aruch as a blastomycete. Tokishige and Marconi believe that it belongs with the saccharomyces. It is not easily stained by the aniline dyes, although Mettam has shown that by the Gram method, Nicolle's violet, Nicolle's thionine and others it is readily colored. It is cultivated with difficulty. Tokishige obtained cultures in bouillon, agar, gelatin and on potato. In bouillon it required seventeen days to obtain a growth.

The infectious material may be transported by contact between the diseased and well horses, by stall bedding, by stable utensils and harnesses and possibly by insects.

The period of incubation is placed at from three weeks to three months and in certain cases it may extend to even eight or ten months. In experimental cases symptoms have appeared after 32 days.

Symptoms. An infection takes place in wounds, the first symptom usually appearing at the seat of a pre-existing wound. The lesions usually appear in the skin, but they may occur on a mucous membrane. They consist of swelling and suppuration of the lymph vessels and glands. These break and discharge a thick, yellow pus, stained with blood. Pearson states that the horses do not, as a rule, show any general disturbance except in very advanced cases. Pallin describes the opened sores as follows:

"The buds, ulcers, or sores, by all of which names they are known, are characterized by their bright red exuberant granulations and their fungoid appearance, as well as by their indurated base and well-defined edges; the adjoining skin, which is partially inverted, has a peculiar shiny appearance; an opening exists in the center of the bud, from which the pus, at first creamy, and afterwards yellowish, oily, and curdled, is continually discharging."

These buds are quite different from those of glanders. The lesions are commonest in the limbs. The most usual location is on the fore-leg generally extending up along the fore-arm to the brachial region and point of the shoulder.

The sores vary in size from that of a pea to a hen's egg. Pallin reports lesions on the mucous membranes in from 7 to 10 per cent of the cases. When these occur on the nasal mucosae they are liable to be confounded with those of glanders.

Usually the general symptoms are not conspicuous. The temperature remains normal and the appetite good. The disease seems to thrive best on animals in good condition.

Pearson describes its symptoms as follows: "The most common manifestation consists in the presence of small, chronic, discharging ulcers in the vicinity of the hock joint of a thickened hind leg. In such a case, one may also find small scars showing where ulcers have healed and there will probably be some firm nodules beneath the skin and, perhaps, one or more nodules that have softened, forming fluctuating abscesses. The regional lymphatic ducts are corded and the glands inside the thigh are hardened and nodulated.

"The earliest observed symptom may be the occurrence of a firm nodule, from the size of a pea to that of a walnut, beneath the skin, anywhere on the body. Corded lymphatics extend from this lesion. In time, the nodule will soften and, at length, its purulent contents will break through the skin. The time required for these developments is most variable and may reach several weeks. Such pus is thick yellowish or greyish yellow and often is mixed with blood. Sometimes it contains flakes.

"In other cases, the first symptom observed is an indolent sore, covered with pus and scab, surrounded by a slightly swollen zone and from which one or more firm cords extend beneath the skin toward the lymphatic glands. This condition is, no doubt, the first to develop, but such a sore often escapes special notice until nodules occur.

"Fresh ulcers may be surrounded by a slightly raised zone of bright red granulation tissues ('proud flesh'). The ulcers occur irregularly and they disappear slowly; some heal in a fortnight, others continue to discharge for months, and, after healing, may break out again. From this long continued irritation and from the formation of scar tissue, the skin thickens and the affected parts become indurated.

"Ulceration sometimes occurs upon the conjunctiva and on the mucous membrane of the nostrils and upper respiratory tract."

Morbid anatomy. The lesions are an inflammation of the lymphatics. On section the walls of the vessels are thickened, their internal membrane is congested, and the ducts filled with thick-clotted lymph mixed with pus, which is followed by the formation of the abscesses (pustules) and granulating sores. The affected parts become indurated as the result of the formation of fibrous tissue due to the inflammation set up by the disease. On the mucosa, the ulcers have a round, well-defined raised border. They are at first

isolated but later they become confluent. Nodules are occasionally found in the liver and spleen. A few horses appear to recover spontaneously. A few are apparently benefited by proper treatment. It is said to be fatal in from 10 to 15 per cent of all cases.

Diagnosis. This affection is to be diagnosed by the symptoms, lesions and by finding the specific organism in the discharges from the lesions.

In 1910, Bridré and Nègre demonstrated that the complement fixation test could be used to prove that epizoötic lymphangitis is due to a blastomyces and not to protozoa. The serum of horses suffering from epizoötic lymphangitis gave positive fixation with the *Cryptococcus farciminosus*, or yeasts in general—but not with bacteria or *Leishmania infantum* parasites, etc. A yeast immune serum gave fixation with the organisms of lymphangitis, but not with bacteria or protozoa. Meyer confirmed their findings. He states that the sera from sporotrichotic infections give complement fixation with the *Cryptococcus farciminosus*, indicating a relation of the *Sporothrix* Schencki-Beurmanni to the cryptococcus. This observation is further proof of the vegetable nature of the parasite of "epizoötic lymphangitis."

It is to be differentiated from sporotrichosis, glanders, ulcerative lymphangitis, tubercular lymphangitis, bursatti, and the so-called botryomycosis. The finding of the specific organism in case of epizoötic lymphangitis affords a positive means for its diagnosis.

REFERENCES

1. DUCLAUX. Sur un protozoaire dans la lymphangite epizootique du mulet en Tunisie. *Compt. rend. Soc. de biol.*, Vol. LXIV (1908), p. 593.

2. METTAM. The staining of the organism of epizoötic lymphangitis. *The Vet. Record*, Vol. XVI (1904), p. 834.

3. MEYER. Epizoötic lymphangitis and sporotrichosis. *Am. Journ. of Trop. Dis. and Preven. Med.*, Vol. III (1915), p. 144.

4. MEYER. The relation of animal to human sporotrichosis. *Journ. of the Am. Med. Asso.*, Vol. LXV. (1915), p. 579.

5. MOHLER. Mycotic lymphangitis of horses. *U. S. Dept. of Agr. B. A. I., Circular 155.*

6. PAGE, FROTHINGHAM AND PAIGE. Sporothrix and epizoötic lymphangitis. *Jour. Med. Research*, Vol. XXIII (1910), p. 137.

7. PALLIN. A treatise on epizoötic lymphangitis. London, 1904.

8. PEARSON. Epizoötic lymphangitis of horses and mules. *Circular No. 8, Pennsylvania State Livestock Sanitary Board*, 1907.

9. SANFELICE. Uber die pathogene Wirkung der Blastomyceten. *Zeitsch. f. Hyg. u. Infek.*, Bd. LIV (1906), S. 299.

10. TOKISHIGE. Uher pathogene blastomyceten. *Centralbl. f. Bakteriol.*, Bd. XIX (1896), S. 105.

LEECHES

Synonyms. Summer sore; leeching; *barsati, barsáti, barsattee, barsatti, bausette, bursati, bursatie, burusattee, bursatti, bursautee.* (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).

Characterization. "Leeches" or "leeching" is an infectious disease quite prevalent among the horse kind in Florida with lesions localized on the skin or the mucosa of the head. It is thought by many that this affection is identical with the disease known as bursattee* in India.

History. Neal of Florida first described this disease as affecting horses and cattle in the south. He believed it to be peculiar to that section, where he states it is 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 a belt of grassy prairie, water grass and water lilies. Into these grassy places the horses, mules and cows often go during the summer and feed all day in the water. He adds, "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. This rapidly grows in size till in a few weeks there is a raw surface from four inches to a foot square. This oozes blood and serum but no pus. An examination will usually show a mass of yellow, gritty growth, coral-like in shape, embedded in a mass of bruised, bloody tissue, dark in color with the edges roughened, elevated above the skin, and the skin decaying at the outside of the ulcer. The leech invades almost 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 and the teeth destroyed."

---

*It seems to be true that an entirely different affection is known by the same name in the northern portion of the United States. The term "Leeches" is also applied to the condition following the invasion of the liver fluke (*Fasciola hepatica*).

In this country the disease has not attracted very much attention, nor has it been considered of much economic importance. An explanation for this may be offered on account of its seemingly non-contagious character and because it has been thought to be confined to comparatively limited areas, and because the animals, although infected, may be utilized for some purposes. On account of the chronic course of the disease the affected animals are often killed from a sentiment of mercy before the disease can terminate fatally.

Although this affection presents many points of similarity to the one found in India, the question of their identity ought to be held in abeyance until a more thorough investigation can be made.

Dawson, of the Florida Experiment Station, states "that 'leeches' or bursattee is a common disease in Florida, which manifests itself in the formation of tumor-growths which have some of the characters of actinomycotic tumors. Its structure is fibrous, and contains many sinuses, which discharge a bloody, 'honey-like' fluid. It is a fatal, infectious disease, which has its origin in the skin and finally penetrates all the tissues. Here and there in the tumor tissue yellow bodies with root-like projections may be found. These bodies are called 'leeches' by the natives. They consist of the mycelia of the fungus which causes the disease. The only remedy is the complete removal of the tumor and adjacent tissue at once. In Florida the disease affects only the genus equinus."

In 1896, some of the diseased tissues from cases of this affection in Florida, were sent to the Bureau of Animal Industry for investigation. They were studied by Fish who made an extended report on the results of his findings. He also gave a very complete review of the literature.

Hodgson, in 1853, referred to the sores as cancerous ulcers and Hart, in 1872, was strongly inclined to pronounce it cancer, although he could not confirm this view structurally by microscopic examination of the tissue. 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.

In India, native as well as foreign bred horses are susceptible, but according to some writers, none of the other equine species is affected.

In the United States mules and cattle are said to develop it, but not so readily as the horse. Outbreaks among cattle are compara-

tively rare. Thin-skinned animals are more susceptible than thick-skinned ones. Neal states that Cuban and Texan ponies are as a rule exempt. Anderson found 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 contract it.

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, 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 reported except in the United States, unless upon further investigation certain mycotic diseases which have been described in Europe should prove to be identical with it.

Lyford reported it in Minnesota, Anderson in Kansas and Alabama, and Neal and Bitting in Florida. The latter writer states, 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." A few cases of summer sore have been presented for treatment in the clinic of the New York State Veterinary College. It is not definitely determined whether or not this is identical in its etiology with the "Summer Sores" found in various parts of the country.

Etiology. A summary of the literature shows that among the old theories "leeches" was believed to be a blood disease in many ways not unlike syphilis, scrofula and farcy. The "fly theory" of the causation and dissemination of bursattee was entertained by the natives of India as early as 1820. Jackson, in 1842, seems to have been the first to believe that there was any connection between the disease and a fungus.

He suggested, in 1842, that the disease might be caused by a fungus or a vegetable parasite. Collins, in 1874, expressed a similar belief. F. Smith, in 1879 and 1884, seems to have been the first to have worked along this line. He was able to find fungi in every fresh specimen of the sores that he examined. Steel, in 1881, also found fungal elements in these sores. T. Smith, in 1893, examined some alcoholic material and gave expression to the belief that the disease was caused by a fungus. Fish, in 1896, found a fungus embedded in the lesions. He did not name it neither did he obtain

it in pure culture, but his illustrations are very clear in showing the existence of the fungus.*   He also gives in detail the methods he employed. It is to his work that we are indebted for the more careful description of the morbid changes.

Morbid anatomy.   As a rule the lesions are near the surface.   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.   This nodular or "kunker" growth may vary in its density according to the stage of its development. During the early 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 kunker evenly and to have it retain its proper relations with the other parts.   Even if successful in cutting, the nodule drops out after some of the other 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 leuococytes, 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 are 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.

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.

FIG. 67.   A SECTION OF THE LIP OF AN AFFECTED HORSE, SHOWING SEVERAL DISEASED FOCI (*Fish*).

---

*There is considerable evidence that the "Summer Sore" in the northern states is due to a *Streptothrix*.

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. In most cases the filament remains unstained, or, as observed in a 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 are 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. The free ends of many of the clubs point toward the periphery of the nodule, but this is not a constant feature.

As a result of the treatment of the nodules with a 10 per cent. cold solution of caustic potash, a very profuse and intricately branched fungus becomes 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 of varying size.

In certain of the teased preparations (Biondi-Ehrlich stain) the wall of the filament, instead of being smooth and homogeneous, appears roughened, as if covered with very minute but numerous spinous processes.

In the sections of the tissue in which the fungus appears the substance of the filament is not uniform. In places it is drawn together in an irregular manner, with intervening clear spaces of greater or less area.

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. Scattered among and coiled around the ordinary filaments there have been observed much more slender ones apparently devoid of any external sheath.

There have also been observed numerous small circular bodies of inconstant size. They have been seen lying free 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 arrange-ment. With possibly one exception, no trace of blood vessels has been found in the nodules.

There is an infiltration of the connective tissue with a great number of wandering cells. In some places there are well-defined nests in the stroma of the connective tissue, 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 1 to 12 in a cell.

In some preparations numerous leucocytes, of the mononuclear and polynuclear varieties, 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 eosinophiles, which took a deep orange color with the Biondi-Ehrlich stain. In places adjacent to these leucocytes there were frequently noticed a number of these small bodies apparently lying free in the tissue.

The vacuolated cells are present in greater numbers than the heavily laden leucocytes. In the former nuclei are present and present various phases of change. In some there is 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 are 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 eight microns. In one specimen there 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 is some appear-ance of vacuolated cells, each with a single nucleus and fairly well-defined cell boundary. 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.

The connective-tissue cells surrounding the nodule show marked signs of degeneration, their cytoplasm in most cases being extremely vacuolated. 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 methylene 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.

Bitting has figured the jaw bone of a horse quite extensively affected with this disease. He believes that the lesions about the mouth result from the animal biting the affected areas on the body.

Neyrick reports finding the inflammatory growths in the lungs of an affected subject, and Burke has reported them in the liver. There are no other lesions described in the internal organs although Neal writes that any tissue may be invaded.

Diagnosis. The diagnosis is made by the lesions and the finding of the fungus in the tissues.

Treatment. On the ground that the fungus supposed to be the cause of this disease may be closely related to the cause of actinomycosis, the use of iodide of potassium has been recommended. It is reported to be fairly successful. The efficiency of this drug as a specific needs further confirmation.

REFERENCES

1. Bitting. Leeches or leeching. *Bulletin No. 25, Florida Agricultural Experiment Station*, 1894.

2. Fish. A histological investigation of two cases of an equine mycosis, with a historical account of a supposed similar disease called bursattee occurring in India. *Annual Report, Bureau of Animal Industry, U. S. Dept. of Agriculture*, 1895–6, p. 229. (This report contains a bibliography on Bursattee.)

3. Neal. "Leeching" of horses and cattle. *Annual Report, Bureau of Animal Industry, U. S. Dept. of Agriculture*, 1887–8, p. 489.

4. Roadhouse. Observations on bursatti. *Amer. Vet. Review*, Vol. XXXVIII (1910–11), p. 376.

MISCELLANEOUS FUNGUS INFECTIONS

A considerable number of lesions have been attributed to different fungi. Some of these have been carefully described while others have not. These infections are liable to be encountered at any time. Because of the indefinite character of most of the descriptions, but one of them is mentioned here.

Blastomycetes infection in horses. Fermi and Aruch described a disease in horses resembling glanders except that it did not affect the lungs, which was caused by a blastomycete. It is known as *Farcin d'Afrique.*

Frothingham has described a tumor-like lesion in the lungs of horses caused by a blastomycete torula. The growth was about ten inches in diameter and on section resembled in appearance a myxosarcoma. The central portion was easily removed, the outer zone forming a firm border wall composed of fibrous tissue. A microscopic examination of the central portion showed it to consist of a fine meshwork of fibrous tissue, in the meshes of which were many cells and blastomycetes. These were fatal to rabbits and guinea pigs. He cultivated the organism on artificial media.

REFERENCES

1. FROTHINGHAM. A tumor-like lesion in the lung of a horse caused by a blastomyces (Torula). *Jour. of Med. Research,* Vol. VIII (1902), p. 31.

2. NICHOLS. The relation of blastomycetes to cancer. *Journ. of Med. Research,* Vol. VII (1902), p. 312.

3. RICKETTS. Oidiomycosis (blastomycosis) of the skin and its fungi, *Journ. of Med. Research,* Vol. VI, p. 377.

4. WEIS. Four pathogenic torulae. *Journ of Med. Research,* Vol. VII (1902), p. 280.

CHAPTER VIII

DISEASES CAUSED BY PROTOZOA
GENUS SPIROCHAETA

General consideration of the Spirochæta. The genus *Spirochaeta* is given by Migula as one of the *Spirillaceae*. The investigations that have been made, since this classification was published, on the mode of reproduction and other biological phases of these organisms have led systematists to look upon the genus as belonging to the protozoa rather than to bacteria. The genera *Spirochaeta* and *Spirillum* were first described by Ehrenberg. The essential point of difference between them was the inflexibility of the spirillum and the flexibility of the spirochæta. Schaudinn described an undulating membrane on the spirochæta. The constancy of this morphological element has been questioned by equally eminent authority.

Calkins has pointed out the relation of spirochæta to bacteria and protozoa in the following paragraph:

"From the foregoing review of the structures and life histories of the spirochætes there is little that is definite to determine the natural affinities of these spirilliform organisms. The plastic nature of the body and polymorphism are protozoan characters. The structure of the so-called flagellum is a point in favor of the bacterial nature, but the highly kinetic membrane is an equally strong point in favor of the protozoa. The nucleus or its equivalent is more like that of the bacteria than like the morphological nucleus of the protozoa; but there are protozoa with distributed nuclei, so that this character is not distinctive. The physiological characteristics are quite as typical of protozoa as they are of bacteria; division, so often a subject of acrimonious and contradictory statements, is not decisive, for many protozoa divide transversely (all ciliates and *Oxyrrhis* and *Polykrikos* among flagellates), while some bacteria are said to divide longitudinally. Cultivation on artificial media, thus far unsuccessful with spirochetes, is now, thanks to the excellent work of Novy and MacNeal and their followers, no longer a distinctive feature, for trypanosomes, like most bacteria, may be so cultivated. The results of plasmolysis, urged by Novy and Knapp ('06) as an argument in favor of the bacterial nature of spirochetes, have but little value, for the time factor necessary to plasmolyse is a purely relative matter dependent upon the nature and resistance of the cell membrane. Differences among the bacteria themselves, in this respect, as Prowazek, Siebert, and many others have pointed out, are quite as marked as the differences between undoubted protozoa and spirochætes. The periodicity of symptoms in the hosts of disease-causing forms is more characteristic of protozoa than of bacteria, but the formation of toxins and the installation of immunity give no light on either side.

So, too, the passive carriage or active multiplication within the insect host, which Stiles ('06) regarded as a sufficient test of the plant or animal nature of spirochetes, only pushes the problem a step farther back, for some spirochetes, at least, multiply in the insect host and some trypanosomes are apparently carried and transmitted in a passive state.

"On the whole, therefore,while again repeating that the controversy now has only an academic importance, the weight of evidence favors the view that spirochætes as a group are structurally (ectoplasmic) more complex and more plastic and variable in form than bacteria, while functionally they have a more complicated life history.   On the other hand, their structures (endoplasmic especially) are much less complex than in protozoa, and their life history, so far as it is known, more simple than that of the known protozoa. Until further observations on the life histories of different species are made we are justified in doing no more than to place the spirochetes as an intermediate group between the bacteria and the protozoa, but leaning more toward the latter, and in this sense they are included under the name spirochætida in our classification."

A.   TYPE GENUS SPIROCHAETA

With undulating membrane; without flagella.

Spirochæta plicatilis.   Ehrenberg, 1838.   Free living.   Length up to 200^μ.

Sp. balbianii.   Certes, 1882.   In oysters, clams, etc.   Length up to 150^μ.

Sp. anodontoe.   Keysselitz, 1906.   Mussel (anodon).   Length up to 60^μ.

Sp. vincenti.   Blanchard, 1906.   Human ulcers.

Sp. pyogenes.   Mezincescu, 1904.   Tuberculous cattle.

Sp. refrigens.   Schaudinn, 1905.   Human syphilitic lesions (external).

Sp. pseudopallida.   Kiolemenoglou and von Cube.   Ulcerating carcinoma.

Sp. eberthi.   Kent, 1880.   Bird intestine.

Sp. gigantea.   Warming, 1874.

Sp. buccalis.   Steinberg, 1862.   Probably same as dentium.   Same habitat.

B.   GENUS TREPONEMA

Without undulating membrane; with flagella.

Treponema pallidum.   Schaudinn, 1905.   In human and ape syphilitic lesions.

Tr. pertenuis.   Castellani, 1905.   In lesions of frambesia or yaws.

Tr. anserinum.   Sacharoff, 1890.   Blood of geese.

Tr. gallinarum.   March and Salimbeni, 1903.   Blood of chickens.

Tr. theileri.   Laver and Vallée, 1904.   Blood of cattle.

Tr. muris.   Wenyon (Tr. Laverani, Breinl and Kinghorn).   Blood of mice.

C.   UNDETERMINED FORMS REFERRED TO GENERA SPIROCHAETA AND SPIRILLUM

Spirochæta dentium.   Koch, 1877.   Human mouth and teeth.

Sp. vaccinæ.   Bonhof, 1905.   Pustules of calf.

Sp. recurrentis (Sp. obermeieri).   Lehert, 1874.   Cause of relapsing fever.

Sp. duttoni.   Novy and Knapp, 1906.   Cause of tick fever in man.

Sp. microgyrata.   Löwenthal, 1906.   Ulcerating human carcinoma.

Sp. microgyrata.   Löw. var. Gaylordi.   In non-ulcerating mouse tumors.

Sp. of dysentery.   Le Dantec.

Sp. ovis.   Novy and Knapp.   Blood of sheep.

Sp. equi. Novy and Knapp, 1906. Blood of horses.
Sp. vespertilionis. Novy and Knapp, 1906. Blood of bat.
Sp. muris, variety Virginiana. MacNeal, 1907. Blood of rat.

Spirochætosis. The term spirochætosis is given to the diseases caused by various species of the type genus spirochæta. The term spirilloses should be considered as a synonym until a more definite classification of the spirochætæ can be made. Some of the spirochætæ seem to be obligatory parasites and others capable of living on certain mucosæ of the body without in any known way affecting it. Infections with spirochæta have been described as follows:

Spirochætosis of the fowl.
      "   of the goose.
      "   of the ox and calf.
      "   of the sheep.
      "   of the pig.
      "   of the horse.
      "   of mice.
      "   of man.

The study of the type genus *Spirochæta* has resulted, as shown in Calkin's classification, in placing the spirochæta of fowls, geese and cattle in the genus *Treponema*, to which the spirochæte of syphilis and the germ of yaws belong. The distinguishing character of each is that *Spirochæta* has an undulating membrane and no flagella. The genus *Treponema* has flagella but no undulating membrane.

SPIROCHAETOSIS IN FOWLS

Synonyms. Spirillosis of fowls; *spirillose des poules*; *Hühner-spirillose.*

Characterization. An infectious disease of fowls caused by spirochæta of which there are several species described. The disease affects the blood and is a septicemia due to spirochætes and transmitted by the fowl tick, *Argas miniatus, A. persicus,* and *A. victoriensis* and possibly by other extozoa. It is a disease of fowls but it has been transmitted experimentally to geese, pigeons and other birds. Rabbits, guinea pigs and rats are said to be susceptible.

History. This disease was described in Rio de Janeiro in 1903 by Marchoux and Salembini. Since that time it has been found in Algiers, Australia, Bulgaria, India, Rhodesia and other parts of South Africa, Tunis and the United States. In the various countries the

spirochete has been assigned different names but whether they are different species is not clearly determined. It is possible they are identical or varieties of the same species.

Bevan has described a serious disease of chickens in Southern Rhodesia due to a spirochete. He has associated it with the common fowl tick, *Argas persicus.* Lounsbury maintained that the fowls died "entirely from the loss of blood and the inflammation produced by the excessive parasitism." Blanchard demonstrated that *Spirochaeta gallinarum* is transmitted by the bite of an argas. In Rhodesia the disease is most prevalent during the spring months, that is, when the first rains set in. Ordinarily the fowls that apparently are healthy at night are found dead in the morning. There are chronic cases in which the disease manifests itself by ruffled feathers and lessened power of the use of their limbs. There is intense thirst and they drink large quantities of water. There is diarrhea and sometimes the fowl will eat almost till the time of death. The essential lesion is anemia. At the point where the tick attached itself there may be a small subcutaneous hemorrhage.

Geographical distribution. Spirochaetosis of fowls is, as stated above, widely distributed. It is possible that it exists in the United States to a much greater extent than has heretofore been supposed.

Etiology. Spirochætosis in fowls as originally described is caused by *Sp. gallinarum.* According to Jowett it is found free in the blood of the fowl naturally infected and also has rounded bodies in the interior of the erythrocytes. The free spirochete is an extremely thin, thread-like, spirally formed organism. It is of uniform thickness except at the extremities, which are tapered. In the fresh blood they are actively motile. In stained preparations they are found to vary in size, measuring from 8 to 10μ in length. Occasionally much longer individuals are observed. Frequently two spirochetes are joined end to end. They are regularly formed spirals but they may be looped in the figure of a pattern. The intra-corpuscular bodies may be found before or after the free spirochetes or they may coexist with them. They are bodies generally round or spherical in form and invariably situated in the extra-nuclear portion of the erythrocyte. Jowett found they were more frequently located at the end of the carpus than in any other place. They are sometimes situated close to the edge of the nucleus and at others close to the periphery of the cell. In size these bodies vary from one to four microns. Dodd did not find the

"after-phase" bodies in fowl spirochætosis in Queensland and Gilruth did not observe endoglobular forms in the Victoria outbreak. According to Brumpt the spirochetes in fowls differ according to locality. He subdivides them into *Sp. gallinarum* vel Marchouxi (Brazil), *Sp. Neveuxi* (Senegal) and *Sp. nicollei* (Tunis). For a more detailed description of the spirochetes and their classification it is necessary to look up references made to the original articles on this subject.

The ticks that transmit the spirochetes are nocturnal parasites which feed during the night and hide away between crevices, cracks and behind objects during the day. At night they leave their hiding places and crawl on the fowls which they bite and extract blood and at the same time inoculate their host with other spirochetes which they have obtained from an infected fowl. It is believed that the organism can pass through the mucous membrane of the alimentary tract. The red mite of fowls is not a carrier. Fowl lice are said to be capable of transmitting *Sp. granulose penetrans balfour.* Levaditi has cultivated the spirochetes. He used the collodion sacs filled with fowl serum previously heated to a temperature of 72° C. and placed them in the peritoneal cavity of the rabbit.

Symptoms. There are two distinct types of the disease, acute and chronic. In the acute form a rise of temperature and intense thirst are the first manifestations. These are followed by a loss of appetite, roughening of feathers, loss of flesh, dullness, diarrhea and drowsiness. The crisis occurs in one or two days after somnolence starts in. At this time the temperature falls, the comb becomes bluish red in color and death follows, usually in convulsions, or convalescence begins. Recovery may be apparent when the legs become paralyzed and in some cases the wings also. The chronic form may follow the crisis or arise independently. The fowls become paralyzed in the legs and sometimes in the wings and neck so that walking becomes difficult and finally impossible. The paresis is rather slow in development being first detected by the fowl having difficulty in using its feet. In some cases recovery follows the paralysis but in others they become gradually emaciated and finally death follows.

In the chronic form the spirochetes are not found in the blood. In the acute stage the spirochetes may be detected early in the crisis when they rapidly disappear. According to Nuttall and also Levaditi the organisms are destroyed by lysis. Gabritschewsky and Uhlenhuth found parasiticidal substances in the blood of infected fowls before the crises was reached.

The duration of the disease in the acute form is usually from four to six days and in the chronic form from eight to fifteen or more days.

The period of incubation in chickens is from seven to nine days after placing infected ticks upon them.

Morbid anatomy. Wasted muscles and extreme anemia are the most pronounced lesions. In the acute cases the liver and spleen are enlarged. The liver contains numerous small areas of necrosis. The intestines are congested and at times show punctiform hemorrhages. There may be fatty degeneration of the heart and liver but this lesion is not considered specific for this disease. The spirocheta disappear from the blood soon after death and consequently are not usually detected in examinations made after death. In the chronic cases anemia and emaciation are very pronounced and the spleen may be atrophied. In fowls killed before the crisis, especially in the Brazilian form, the spirochetes are found widely distributed in the body.

Prevention. As the spirochetes are transmitted from one fowl to another through the agency of the fowl tick the most effective way of prevention is to keep the premises thoroughly cleaned and the fowls freed from vermin. This can be done by dipping in a bath of sulphurated lime solution. A 3% creolin solution has also been recommended.

Immunity. The blood drawn from infected fowls is said not to be infectious after 48 hours but if it is injected subcutaneously into healthy chickens it produces an immunity. Marchoux and Salimbeni found that the fresh blood from infected fowls heated for five minutes at 55° C. has an immunizing effect but if heated for 10 minutes it loses that property. Salvarsan or "606" has been found to be a strong spirochetacide. Balfour found that if this was given in sufficient doses it eliminated the spirochetes from the peripheral blood and in certain cases cured the fowl. It has little or no effect upon the granular phase of the parasite. Abrin will destroy *S. gallinarum.* Atoxyl in 5 centimeter doses was found by Uhlenhuth, Gross and Bickel capable of preventing infection when given one or two days after exposure and also to be of therapeutic value but the blood of fowls thus treated remains infective.

SPIROCHAETOSIS OF GEESE

Synonyms. Spirillosis of geese; *spirillose des oies; Gänsespirillose.*

Characterization. Spirochætosis in geese is characterized by emaciation, loss of appetite and the presence of *Treponema anserina* in the blood. It is said that the disease may be transmitted to turkeys, canaries, crows, magpies, sparrows and larks but as a rule it is not fatal. Pigeons are said to be refractory. Very young chickens are slightly susceptible and rarely die from inoculation.

History. This disease was discovered by Sacharow in the Trans-caucasus in 1890. He described the organism causing it which was present in large numbers in the blood and named it *S. anserina* (according to Calkins, *T. anserina*). In 1898 Gabritchewsky made an exhaustive study of the relationship between the spirochætosis of geese and recurrent fever in man. He also obtained an immunizing serum. In 1899 Cantacuzène determined the method of destruction of the parasite in the blood. Duschunkowsky and Luhs in 1907 reported the disease to be quite prevalent in the Caucasus.
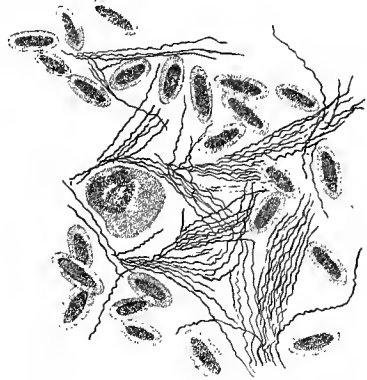
FIG. 68. T. ANSERINA FROM THE BLOOD OF A GOOSE (*after Cantscuzène*).

Geographical distribution. This disease has been found in Southern Russia and in Tunis. Spirochætosis has been reported from several other localities. It is a disease, however, that has not been fully investigated and the extent of its distribution and frequency is not known.

Etiology. It is caused by *T. anserina.* It is from 6 to 15µ in length and about 0.21µ in width. It has from 2 to 7 or more spirals. The spirochete has at each extremity a cilium of variable length.*

*The cilia are clearly demonstrated by making smears of the blood on clean slides, treating them with hydrochloric acid and absolute alcohol, passing them through the flame and then allowing them to dry in the air. The slide is then washed with a jet of blue distilled water. It is dried by means of blotting paper and a few drops of Löffler's or Bunge's mordant solution are poured on the slide which is then heated until boiling over Bunsen flame. It is then thoroughly washed and dried with blotting paper. The preparation is then treated with a few drops of aqueous solution of Gentian violet, heated to a boiling point for a few seconds, thoroughly washed in a stream of distilled water and immediately examined while moist. The preparation may be dried and mounted in balsam.

Sometimes one or both of these are absent. The organism is actively motile and moves like a corkscrew on its long axis by spiral undulations. The spirochæta are visible in the blood without previous staining, if examined early in the stage of infection. They frequently run together forming loose bundles of radiating organisms. They stain with more or less difficulty.

Symptoms. The infected geese become weak, lose their appetite, become dull in appearance and assume a sitting position. Diarrhea appears and the visible mucous membranes are pale. This condition continues for from 6 to 10 days when they die apparently from exhaustion. Their temperature reaches 108° to 110° F. It is reported by some that the geese have very painful swellings on the joints of the feet. This condition does not appear to be constantly present.

Morbid anatomy. The geese become very much emaciated. The liver is hyperemic, swollen and of a brownish red color. The spleen may be much enlarged. It is of a dark purplish color, soft and friable. If the disease has lasted for a number of days the liver is found to be undergoing fatty degeneration and to contain numerous foci of necrotic tissue. The spleen may also contain such areas. The intestines are very much congested or inflamed. The spirochæta disappear very soon after the death of the goose.

Prevention. As the life history of the spirochæta is not fully known it is not always possible to state what precautionary measures are necessary other than isolating. It is reported that the organs of the dead geese in which the spirochetes have died or disappeared can be used in the preparation of a vaccine which confers a high degree of permanent immunity. The serum of immune geese is said to be both protective and therapeutic. Atoxyl is reported to be a specific therapeutic agent given in doses of 0.10 to 0.15 per 1000 grams of the body weight. It is stated that the serum can be prepared by infecting the geese and then treating them with this specific.

SPIROCHAETOSIS IN MAMMALS

Spirochaeta in mammals. A number of spirochetes have been found in different species of mammals but their disease producing power does not seem to be definitely understood. Experiments in the production of disease with spirochetes have not been altogether successful. We are unable at this time to look upon this genus as being of as much etiological significance in mammals as in fowls.

Spirochaetosis of cattle. In 1902, Theiler found spirochæta (spirillum) in a few cattle in South Africa. The animals were suffering with "red water" with possibly one exception. He made a number of inoculations with the blood of animals infected with spirochæta with negative results. He believed, however, that they were the cause of the illness in cattle. The organism was named by Laveran *S. Theileri* after its discoverer. The following description of the organism is quoted from Theiler's report:

"The microörganism in question is a typical spirillum, and varies considerably in its length. The longest microbes measure from 20 to 30μ. They are somewhat thinner at both ends, otherwise the thickness is about the same throughout the whole length—viz., about .25 to .40μ. The corkscrew-like forms are, as a rule, predominant, but there are other forms which are atypical in appearance and also shorter. For instance, the parasite may show itself as a simple curved line without any spiral curves, representing sometimes the shape of S; it may also be completely doubled up, both ends may meet and take the shape of a ring, or both ends may cross each other and also form a loop. These loops may be of different shapes and sizes. Double loops may also be found. It is somewhat difficult to describe all the various forms, but they can be easily imagined, considering that the spirillum is a very flexible and agile parasite.

"In preparations made by placing a cover-glass on a drop of fresh blood the microörganisms are easily detected. Some of the red corpuscles show a slight irregular motion, and when closely watched it may be noticed that this disturbance may proceed in a certain direction, or again whirl round in the same place. When examined under a high power (1–12th inch obj.) the agile spirillum is usually seen attached to one or more red corpuscles. Sometimes it is curled all round a red or white corpuscle. As soon as the organism becomes free and begins to travel through a clear space, a characteristic undulating movement is visible, which continues until it finally attaches itself to some other blood corpuscle. This motion may be noticed for some time; it then becomes relaxed, and finally slackens down completely. I have observed the movements in preparations which were twenty-four hours old.

"Staining of the organism may be obtained with any of the aniline dyes used in bacteriology, viz., methylene-blue, fuchsin, thionin. Good preparations were obtained with Laveran's modification of Romanowsky's stain, and also with Azur II. In using the last two

mentioned methods no chromatic body could be traced, such as is present in parasites belonging to the protozoa.

"Cultivations on the usual artificial media were repeatedly tried, but always with negative results."

The disease produced by this organism is of a benign character and the symptoms are as a rule said to be slight. If infective blood is inoculated a febrile reaction takes place in from three to four days and about a day later the organisms may be found in the blood, occasionally a little earlier. There may also be a slight anemia with a considerable loss of red blood corpuscles.

Theiler in 1904 found that the spirochete was transmitted from diseased to susceptible cattle by means of both adults and larvæ of the blue tick *Boophilus decoloratus* and by *Rhipicephalus evertsi*. This means of transmission was confirmed by Laveran and Valée. Theiler found the spirochætal infection to be associated with piroplasma infection. Heanley found a few spirochetes in the buffalo, principally in the spleen and blood. They resemble those described by Theiler in the ox. Mezincescu studied a spirochete found in tuberculous cattle which he named *S. pyogenes*. Bonhoff found a spiral shaped organism in the vaccinial pustules of the calf and which he designated provisionally, *S. vaccinae*.

Spirochætosis in sheep. In 1902, Theiler described spirochetes in the blood of sheep in the Transvaal and in 1904 Martoglio and Carpano observed them in the blood of Abyssinian sheep. It has from three to ten spirals and is from 10 to 20μ in length and from 0.2 to 0.4μ in breadth. The extremities are tapered and they do not take the Gram stain. This organism is found in the blood plasma while the red blood corpuscles contain an unrecognized body. Blanchard designated is at *S. ovina*. The fact should be noted that Dodd transmitted *S. theileri* from the ox and horse to the sheep and it is possible that the spirochetes found in the three species are identical. Susceptible sheep inoculated subcutaneously with defibrinated blood from the infected horse have a rise of temperature on the third to fifth day which continues intermittently for some days.

Spirochætosis in the horse. Theiler also found spirochetes in horses in South Africa. Martin found them in the blood of horses. They were joined together around the red blood corpuscles. They were 12 to 15μ in length and 0.25μ in breadth. Some of them were undergoing transverse division. The inoculation of the blood of the horse

to the fowl did not transmit the disease. In 1906, Stordy observed the spirochete in the Abyssinian pony. The animal was dull, hanging head, large swellings over the orbits and acute edema of the neck. The temperature was 97° F. The next day the edema had disappeared about the head, the temperature had fallen to 95° F, while on the third day rapid emaciation had begun, edema between the legs and under the abdomen became persistent, and the temperature rose to 99° F. On the fourth day spirochetes were found in the blood and edematous fluid. The tick *Rhipicephallus pulchellus* was found on the animal as it was common to cattle, horses, dogs and game in the district. There was suspicion of its being a carrier. He does not give evidence to prove that this was the case.

In 1905, Jowett found a spirochete varying from 7 to 20μ or more in length by 0.25μ in breadth in cases of cancre and grease of the horse.
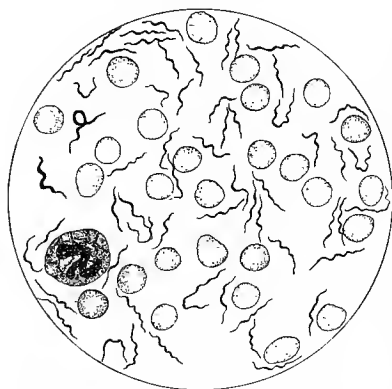
Fig. 69   SPIROCHETES FROM THE LESIONS IN THE SKIN OF A PIG *(after Dodd)*.

In this connection it should be stated that spirochetes have been found in ulcerous processes in man, in the venereal granulomata of the bitch, the carcinomata of the mouse and in granular and ulcerating suppurating wounds in other animals. But the relation of these organisms to the lesions with which they are associated has not been determined. Jowett, however, found the spirochetes in practically every case that he examined.

Spirochætosis in swine. In 1906, Dodd described a spirochete which he found in a pig sent to the Government laboratory in Pretoria. The examination showed very few morbid changes in the tissues but the skin was sprinkled with dark hemorrhagic-like spots. From scrapings of these lesions he was able to find the organism. It was not discovered in its blood. The spirochetes disappear from the lesions very soon after death.

The spirocheta is described as long and very slender, its length varying from 9 to 26μ. Both extremities pointed. The spiral forms predominate but in the same preparation curved or simply long straight threads were observed. They appeared singly, in pairs and

in clumps. It was difficult to distinguish it in the unstained preparation. It stains well with any of the ordinary aniline dyes but does not retain the coloring matter after being treated by the Gram method.

The lesions are ancmia and a large number of circumscribed superficial ulcers on the skin ranging from one to two centimeters in diameter. The ulcers after definite periods become healed. The cicatrisation begins under a scab which forms over the lesions. Later the scab drops off leaving a glistening cicatrix. Infected pigs were found not to regain their former condition after cicatrisation was complete but continued to emaciate until the time of death. The disease is transmitted by cohabitation. The spirochetes have been found in various lesions in the pig by Cleland and also by Gilruth. The etiological relation between the spirochetes and the disease in the pig is not fully established. Szante found spirochetes resembling those described by Dodd in what is known as "pig variola" in Hungary and Bucharest.

Other spirochetes. Bonhoff (1905) described a spirochete found in the pustules of vaccinia of the calf. According to Bizzozero the stomach of the dog always contains numbers of extremely slender spirochetes formed of from three to seven turns and from 3 to 8µ in length lodged in the interior of the gastric cells. These were found in the Norway rat, cat and dog. In the latter they were found to be constantly present.

The spirocheta (*S. plicatilis*) was first described by Ehrenberg, in 1838. It is common in stagnant water and often attains a length of from 100 to 200µ. In 1875, Cohn described *S. buccalis* which is tapered at both extremities and common in the dental tartar and in the saliva. In the same year *S. Obermeieri* was named by Cohn after its discoverer as the cause of recurrent fever in man. This organism varies from 15 to 30µ in length, is very thin and tapered at both extremities. In 1905, Schaudinn and Hoffmann described a spirochete which is the cause of syphilis. It is known as *Treponema pallidum*. A number of other spirochetes have been found, some of which are pathogenic for man.

REFERENCES

1. BEEBE AND EWING. A study of the so-called infectious lymphosarcoma of dogs. *Journ. of Med. Research*, Vol. XV (1906), p. 209 and *Journ. Comp. Path. and Therap.*, Vol. XIX (1906), p. 331.

2. BEVAN. Spirochetosis of fowls in Southern Rhodesia. *Journ. Comp. Path. and Therap.*, Vol. XXI (1908), p. 43.

3. BLANC.   Les spirochetes leur évolution chez les Ixodidæ.   Paris, 1911.

4. BLANCHARD.   Spirilles, spirochètes, et autres micro-organismes à corps spiralé. *Revue Vétérinaire*, Vol. LXIII (1906), p. 86.   Also *Journ. Comp. Path. and Therap.*, Vol. XIX (1906), p. 68.

5. BRUMPT ET FOLEY.   Existence d'une spirochétose des poules à spiroehæta gallinarum, R. Bl. dans le Sud-Oranais, transmission cette maladie, par *Argas persicus*.   Société de Biologie, 1908.

6. CANTACUZÈNE.   Recherches sur la spirillose des oies.   *Ann. de l' Inst. Pasteur*, Vol. XIII (1899), p. 529.

7. DODD.   The Identity of the spirochætæ found in the horse, ox, and sheep. *Journ. Comp. Path. and Therap.*, Vol. XIX (1906), p. 318.

8. DODD.   A Disease of the pig due to a spirochæta.   *Journ. Comp. Path. and Therap.*, Vol. XIX (1906), p. 216.

9. DSCHUNKOWSKY AND LUHS.   Untersuchungen über die Gänsespirillose.   *Ninth Report of the (Hague) International Veterinary Congress*, Sept., 1909.

10. GABRITSCHEWSKY.   Beiträge zur Pathologie und Serotherapie der Spirochäten-Infektionen.   *Centralb. f. Bakter.*, Bd. XXIII (1898), S. 365, und 778.

11. GALLI-VALERIO.   Recherches sur la spirochétiase des poules de Tuniste et sur son agent de transmission: Argas persicus Fischer.   *Centralbl. f. Bakt.*, Bd. LXI (1912), S. 529.

12. GILRUTH.   Spirochætæ in lesions affecting the pig.   *Vet. Journ.*, Vol. XVII (1910), p. 528.

13. GILRUTH.   Note on the existence of spirochætosis affecting fowls in Victoria, Australia.   *Vet. Journ.*, Vol. XVII (1910), p. 533.

14. HEANLEY.   The presence of a spirochæta in Chinese buffaloes.   *Journ. Comp. Path. and Therap.*, Vol. XIX (1906), p. 322.

15. JOWETT.   Fowl spirochætosis at Cape Town.   *Vet. Journ.*, Vol. XVIII (1911), p. 240.

16. JOWETT.   Notes on the occurrence of spirilla in "equine canker" and "grease." *Vet. Record*, Vol. XVIII (1905–1906), p. 375.

17. LAVERAN.   Sur la spirillose des bovidés.   *Comptes Rendus de l' Académie des Sciences*, Vol. CXXXVI (1903), p. 939.   Also *Revue Générale de Médecine Vétérinaire*, Vol. 1 (1903), p. 575.

18. LEVADITI ET MANOUELIAN.   Nouvelles recherches sur la spirillose des poules. *Ann. de l' Inst. Pasteur*, Vol. XX (1906), p. 592.

19. MARCHOUX ET SALIMBENI.   La spirillose des poules.   *Ann. de l'Inst. Pasteur*, Vol. XVII (1903), p. 570.

20. MARTIN.   Sur un cas de spirillose du cheval observé en Guinée Française. *Société de Biologie*, 1906, p. 124.

21. MATHIS ET LEGER.   Spirochète du lapin.   *Société de Biologie*, 1911, p. 212.

22. METTAM.   Spirochætes in an infective sarcoma of the vagina of the bitch. *Vet. Journ.*, Vol. XIV (1907), p. 80.

23. POENARU.   Variola in pigs.   *Archiva Vet.*, 1907, p. 67 and *Journ. Comp. Path. and Therap.*, Vol. XX (1907), p. 158.

24. PONSELLE.   Contribution à la physiologie du Spirillum gallinarum, assimilation du glucose.   *Société de Biologie*, 1910.

25. SAKHAROFF.   Spirochæta anserina et la septicémie des oies.   *Ann. de l' Inst. Pasteur.* Vol. V (1891), p. 564.

26. STORDY.   Spirillosis in the horse.   *Journ. Comp. Path. and Therap.*, Vol. XIX (1906), p. 226.

27. THEILER.   Spirillosis of cattle.   *Jour. Compar. Path. and Therap.*, Vol. XVII (1904), p. 47.

28. TRAUTMANN.   Etude expérimentale sur l' association du spirille de la Tick-fever et de divers trypanosomes.   *Ann. de l' Inst. Pasteur*, Vol. XXI, (1907).

CHAPTER IX

DISEASES CAUSED BY PROTOZOA
GENUS AMEBA

General discussion of Ameba. The ameba belong to the *Phylum protozoa, Subphylum sarcodina*. They are protozoa usually with simple structure and characterized mainly by motile organs in the form of changeable protoplasmic processes called pseudopodia. The ameba are a sub-class of the sarcodina which include the more common forms of rhizopods with blunt, lobose pseudopodia which do not anastomose on touching one another. The genus contains a few species that have become parasitic in their habits. These parasites do not produce noxious products like bacterial toxins but whatever damage they may cause is due to the mechanical disturbances set up by their presence and multiplication.

The genus ameba contains very few pathogenic species. Those best known are *Ameba coli* (*Entamoeba coli*), the cause of a dysentery in man, and *Ameba meleagridis* Smith, the cause of an infectious disease of turkeys. Laveran and Lucet gave it the name *Haemamaeba Smithi*. Musgrave and Klegg introduced the term *Amebaisis* to denote infections caused by ameba.

INFECTIOUS ENTERO-HEPATITIS

Synonyms. Blackhead; amebaisis of turkeys and fowls.

Characterization. This disease is characterized by thickening of areas or of the entire walls of the ceca and areas of tissue degeneration and necrosis in the liver. It affects turkeys largely but hens and young chickens are sometimes attacked.

History. In the fall of 1893, Prof. Samuel Cushman of the Rhode Island State Experiment Station sent a few specimens of the affected organs of turkeys which had died of "blackhead" to the Bureau of Animal Industry, where they were carefully examined by Dr. Theobald Smith. In the summer of 1894, Smith made a careful study of this disease at the Rhode Island Experiment Station. He found that it was caused by one of the protozoa (*Ameba meleagridis* Smith).

He published a full description of the disease which, in accordance with the lesions, he designated *infectious entero-hepatitis.*

In 1895, the disease was further investigated respecting the mode of transmission of the infecting protozoa. The results showed that it could be transmitted directly from diseased to healthy turkeys without the intervention of an intermediate host. These results were published in 1896. The place and the time of the first appearance of this diesase are not clearly stated, but it seems that New England was the first to suffer from it.

Chester of the Delaware Agricultural Experiment Station has shown that a very similar disease attacks chickens and Milks at the Louisiana Station found it in fowls.

Geographical distribution. The available data bearing upon the geographical distribution of this disease indicate that it is widely distributed. The New England states, particularly Rhode Island and certain districts in the Middle and Western states, are affected. It has been reported from Louisiana. For want of statistics the amount of loss to the poultry industry occasioned by this disease can not be accurately estimated, but the fact that it has caused many farmers and poultry men in New England to discontinue the raising of turkeys shows that it is of much economic importance. It is stated in the report of the Rhode Island Experiment Station for 1894 that "the eradication of this disease would be worth hundreds of thousands of dollars to the eastern farmers alone." These heavy losses in the East, together with the accumulating evidence that the entire country is sprinkled with infected districts from which the disease is spreading, indicate that this malady is of more than ordinary significance to those engaged in the turkey industry. Jowett states that it has been introduced into South Africa with turkeys brought from the United States. Its existence in several other countries seems to be established.

Etiology. In 1895, Smith described a microörganism belonging to the protozoa which he found to be associated directly with the disease process. He designated it *Amœba meleagridis.* The parasites were very numerous in recently affected tissues, while in those in which the disease process was far advanced and associated with degenerative or regenerative changes, the parasites were found with difficulty. Curtice finds that chickens are hosts for the parasites, and together with the adult turkeys spread them broad-cast through the droppings.

He has shown that the ameba are not transmitted through the eggs. The young turkeys become infected soon after being exposed to contaminated surroundings. The young turkeys are much more susceptible than the older ones.

The most frequent appearance presented by the parasites is that of round homogeneous bodies with a sharply defined, single-contoured outline. Within these bodies and situated somewhat eccentrically is a group of very minute granules, probably representing a nuclear structure. They vary somewhat in size, measuring from 8 to 10µ in diameter in some cases, from 12 to 14µ in others. In the fresh tissues they are distinctly larger than the parasites within the tissues which have undergone the hardening process. The latter are from 6 to 10µ in diameter. The difference may be due to shrinkage, on the one hand, and on the other to a slight flattening of the bodies by pressure in the fresh preparations. These peculiar homogeneous bodies are found, as a rule, free in the crushed preparations, although occasionally giant cells are detected which contained a number of them. The cell nuclei of the giant cells are not visible in the fresh condition. Numerous coarse granules, less frequently fat globules, are embedded in its protoplasm.

Fig. 70. AMEBA MELEAGRIDIS.   (1) ISOLATED ORGANISMS, (2) SINGLE PARASITES, (3) GROUPS OF THE PARASITE (a) OF THE AMEBA IN THE MUCOUS MEMBRANE OF A TURKEY'S CECUM (Smith).

Smith believed from the results of his investigations that the parasite lived in the interstices and lymph spaces of the tissue, but not within cells. This seems certainly true of the cecum. The liver cells become necrotic or else disappear so rapidly that it is impossible to determine just where the parasites begin to multiply. They do not live within the blood vessels, as they are not found within them excepting perhaps in a thrombosed vessel. They must, therefore, occupy the place of the liver cells. It is probable that they begin to multiply in the connective tissue adjoining the blood vessel and simply crowd

out the liver cells, leaving the connective tissue stroma of the lobules in whose meshes they are found.

Their presence within giant cells is seen in almost every infected organ. This intracellular condition is, however, a purely passive one so far as the parasite is concerned.

The microparasites within the tissue of the host seem to tend toward destruction. Both the death of the tissue itself and the repair seem to lead to the disappearance of the parasites. In most cases there may be seen in the same section a partial dissolution of some of the bodies, while others are still in good preservation. Evidently their life within the tissues is not very long.

A discharge of the microparasites which escape destruction probably takes place from the walls of the ceca, when these break down into the contents with which they are carried outward. A similar discharge may take place from the liver through the bile ducts into the intestine. Another way of dissemination is in the death of the diseased turkey and the dissolution of its body, whereby the organisms are set free.

The occurrence of amebæ in intestinal affections of man was noticed by Lösch in 1875. Since that time it has been the subject of many investigations.

The points of analogy between the avian and the human disease are that in both there is an affection of the intestine (large intestine in man, ceca in turkeys) associated with liver disease due to amebæ.

The intestinal wall in amebic dysentery (human) is greatly thickened, owing to an edematous condition. It is also thickened in circumscribed areas and contains cavities filled with gelatinous-looking pus. The amebæ vary much in size and contain vacuoles. They are found in variable numbers in the bottom of the ulcers and in the discharges. The large numbers of amebæ found in the intestinal contents led Councilman and Lafleur to infer an active multiplication therein. The presence of the parasites within the submucosa is described by these authors in one case only.

In the turkey, the parasites are always present in the connective tissue spaces of the mucous and submucous membrane. Their presence in the contents of the cecum is highly probable.

It differs from the *Ameba dysenteriæ* in being quite uniform in appearance, varying but slightly in size (from 6 to 10µ in diameter) and in being free from vacuoles. Movements characterized as ameboid have not yet been demonstrated.

The liver affection in man appears usually as an abscess. In turkeys it appears as a variable number of foci in which the microparasites may be present in great numbers. The difference in the nature of the lesions must be largely attributed to the different reaction of the tissues of birds toward injuries.

Hadley believed the cause to be a coccidium and states that the ameba described by Smith is identical with certain forms in the life cycle of the coccidium. His findings have not been confirmed.

Symptoms. Diarrhea is the symptom which sooner or later may be expected to appear. It probably occupies the most prominent place among the objective manifestations. The disease of the ceca is presumably responsible for this. Diarrhea occurs with at least one other disease of the ceca and with the presence of tapeworms. Emaciation is pronounced in very chronic cases but it is not constantly present. As it may accompany other wasting diseases, it can not be depended upon as an indication of this affection. As the disease progresses the turkeys become less active, lag behind their flock or do not go out with it. Later the comb, wattles and even the skin of the head become dark colored, hence the popular name "blackhead."

The period of incubation is not known in the naturally infected turkeys. In those artificially infected by feeding lesions from diseased turkeys, the period is short until lesions appear in the ceca and liver. In Moore's experiment with four turkeys fed on infected viscera, November 28 and the following days, two died of the disease December 20 and 25 respectively. The other two were apparently well but upon examination one showed extensive lesions and the other was not affected.

Morbid anatomy. Turkeys are attacked quite young. Smith found a turkey about three weeks old in which the disease had already made considerable progress. It seems, moreover, as if the disease is contracted only by the young, because in the examination of turkeys of different ages the oldest show lesions of the longest standing; that is, such as had undergone the most extensive transformation. In general it may be said that the age of the disease process corresponds with the age of the turkey. The most serious and extensive destruction of tissue occurs in the turkeys in the fall. In midsummer the disease is making most progress and the microparasites are present in greater numbers. It is probable that the

delicate tissues of the young are best adapted for the temporary habitat and rapid multiplication of this parasite.*

The primary seat of the disease is the ceca. From these the liver is secondarily infested through metastasis. Other organs have not been found to be attacked. Hadley refers to lesions in the spleen, kidneys, pancreas, lungs, heart and oviduct.

The lesions of the ceca are in substance a thickening of the wall, followed in most cases by a destruction of the epithelium and deeper portions of the mucous membrane. This destruction results in the outpouring of a coagulable fluid into the tube. The thickening of the wall may vary considerably in extent from case to case.

It may be uniform over the greater portion of the tube or it may be limited to circumscribed patches. The commonest seat of these lesions is near the blind end of the tube where it evidently starts and whence it spreads to other portions. Not infrequently only one cecum is diseased, the other remaining normal.

The affection of the cecum is due primarily to the multiplication of the micro-organism which may take place chiefly either in the mucous membrane, or in the submucous tissue; it may, though rarely, extend into the muscular coat. The thickening of the wall is the result of several processes—the multiplication of the parasites, the increase of the normal tissue elements and later on the accumulation of masses of small cells and some giant cells.

FIG. 71. CECA OF A TURKEY; (a) AND (b) ARE DISEASED AREAS, (c) A SECTION OF TRE THICKENED WALL

In the early stages of the invasion, the adenoid tissue between the tubules and in the submucosa becomes greatly increased, owing to the presence of large numbers of micro-parasites of round or slightly oval outline and from 6 to 10μ in diameter which stimu-

*In this regard it simply follows the rule observed by large numbers of parasites whose most destructive action is visited upon the young.

late the proliferation of the tissue cells. Numerous mitoses have been seen in this stage. The parasites seem to occupy the meshes of the adenoid tissue either singly or in groups or nests. In these meshes they are soon inclosed in cells acting as phagocytes, so that the appearance of an intracellular habitat of the parasites is suggested.

The presence of the parasites in this reticulum probably stimulates also the accumulation of lymph cells within the spaces, by virtue of which the mucous membrane is thickened. In this early stage of invasion the epithelium, both of the tubules and of the surface, remains unaffected. The parasites do not invade the epithelium at any time.

As the disease progresses there is a continued increase in cellular elements of the mucous and submucous coats and a gradual invasion of the muscular coat. Here the bundles of fibers of the circular coat are thrust apart by masses of cells, so that this coat also becomes greatly thickened. The inflammation finally extends to the serous covering, where the blood vessels become greatly dilated and give the cecum a congested appearance. In cases of ordinary severity the wall of the cecum which is not more than 0.2 to 0.5 mm. thick normally becomes 2 to 3 mm. thick.

With the progress of the disease the mucous membrane may be shed and a coagulable fluid poured out into the cecum. In some cases it appears in isolated masses, which adhere to certain spots of the mucous membrane. In others, this exudate fills the entire tube with a yellowish-white mass, built up in concentric layers consisting of a mixture of blood corpuscles, fibrin and small round cells in variable proportion.

In the further progress of the local disease it is not improbable that bacteria are also concerned. The exudate contains immense numbers of them and the denuded mucosa furnishes a favorable place of entry. It is otherwise difficult to explain the continued increase in thickness of the walls of the cecum after the mucous membrane has been shed. This continued increase in thickness is due to an extensive infiltration of small round cells and the presence of some giant cells. Parasites in this advanced stage are scarce and usually recognizable only as vacuole-like bodies within the giant cells.

The thickening of the wall is associated in some cases with an extension of the inflammation to the contiguous wall of the intestine, which becomes firmly attached to the cecum. Yellowish exudates are sometimes found outside of the diseased cecum on its serous covering

and they bind it to the other cecum or to the intestine or attach it to the abdominal wall. In these stages, the microparasite is not found.

It seems to have done its work by destroying the mucous membrane and to have left the field for miscellaneous bacteria.

Other portions of the digestive tract are not affected. The secondary lesions are found in the liver, although in some cases they do not appear. The organ itself is enlarged to probably twice the normal size. Over the surface are distributed roundish, discolored spots, distinctly demarcated from the surrounding tissue. These may be distributed uniformly over the whole surface of the liver or they may be limited in number to a few. They vary from 3 to 15 mm. in diameter. Several types of these spots appear corresponding to different conditions of the diseased tissue. We have in the early, most active disease process sharply defined circular areas of a lemon yellow, or a neutral gray or of an ochre yellow color. The spot is not homogeneous in structure, but made up of a delicate network of grayish yellow, dead tissue.

FIG. 72. DISEASED CECUM
SHOWING THE ULCER-
ATED MUCOSA.

In nother class of spots there is a mottled brownish color which contrasts only slightly with the surrounding liver tissue by its darker color. It may contain a central yellow nucleus of dead tissue and a narrow outer border of the same character, or the border may be a dark brownish circular line. The entire spot has an indistinct appearance and is flattened or even slightly depressed below the surface. In some cases they are uniformly whitish and shade off somewhat gradually into
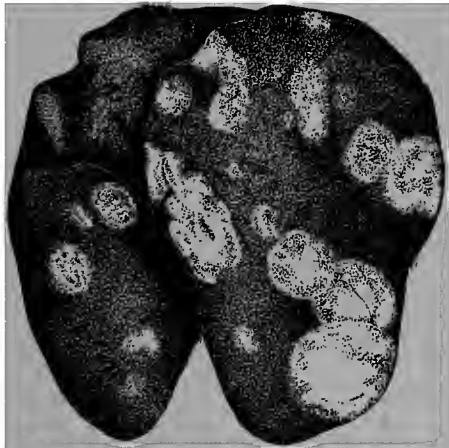
FIG. 73. LIVER SHOWING NECROTIC FOCI.

the surrounding tissue. In sections of the affected organ it will be found that the surface spots represent masses of liver tissue in the same condition, the spots being simply the places where these diseased foci intersect the surface. Some are found

deeply imbedded in the liver tissue, and therefore not visible on the surface. The lesion of the liver is thus represented by few or many foci of disease having in general a spherical form and appearing on the surface of the organ as round spots. Occasionally the lesions become more extensive and the death of large portions of liver tissue follows.

The changes in the liver are most easily explained by assuming that the microparasites are conveyed by the blood directly from the diseased ceca into the liver and there deposited in different places, where they begin to multiply and spread in all directions. In this way they form the spherical foci of disease which appear as circles on the surface of the liver. This theory is borne out by the results of the microscopic examination.

FIG. 74. A DRAWING OF A BEGINNING NECROTIC MASS. (*a*) GIANT CELLS, (*c*) FREE NUCLEI, AND (*b*) DISINTEGRATED NECROTIC TISSUE.

In sections of hardened tissue from the liver in which the disease has but recently begun, the affected regions are invaded by large numbers of protozoa which occupy a kind of reticulum formed probably

FIG. 75. AREAS OF NECROSIS IN LIVER.

FIG. 76. CROSS SECTION OF CECUM.

from the connective tissue stroma. The liver cells have partially or wholly disappeared from these foci. The border of the necrotic tissue is surrounded by a zone of giant cells. The parasites occupy the meshes of the tissue either singly or in groups. The reticulum is

provided with a small number of nuclei, some of which are closely applied to and curved partly around the parasite. The blood vessels are usually much dilated and filled with red corpuscles.

The yellow masses observed with the naked eye in the surface spots are shown to be patches of an amorphous substance which take nuclear stains very feebly, the aniline colors not at all. This may be described as a coarse network in the meshes of which small cells, and very rarely parasites, are seen. This substance is assumed to be the result of coagulation necrosis of the liver cells by which they have lost their nuclei and have become fused into a formless mass. It is probable that the plugging of blood vessels in the liver by parasites carried from the cecum is the cause of the necrosis, since such plugs or thrombi are not uncommon in sections of the diseased areas.

With the appearance of the microparasites regenerative changes begin at once which complicate the process. We have at the outset an active multiplication of the microparasites which take the place of the original liver tissue and a process of coagulation necrosis going on at the same time. Soon multinucleated (giant) cells appear. Not infrequently they are grouped around what appears to be a plugged vessel or else they occupy the lumen of the vessel itself.

In still older cases the diseased areas are found more or less filled with small round cells which may have passed into the dead regions from the blood vessels. In all cases the latter are more or less enlarged and they seem to encroach upon the liver tissue, thus filling in part the void produced by the cell death and giving the surface of the liver a brownish, mottled appearance wherever the disease spots are. The processes of advancing disease and necrosis or death of tissue on the one hand and of repair on the other seem to go on side by side, now one, now the other, predominating.

The results of the investigations thus far made indicate that the disease may follow several courses.

After a certain period of disease, regenerative processes begin which tend toward a permanent recovery.

The disease may proceed so rapidly from the very start that the affected turkeys die early in life.

The disease may come to a standstill but the amount of dead tissue in the ceca and liver may be so great as to favor the entrance of bacteria which are directly responsible for the death of the bird late in the summer or fall.

The description of the lesions of a turkey dead of this disease is appended. It is quoted from Smith's report.

"*Turkey No. 14.*—About 5 months old. Taken from a flock August 8 because of lack of strength to keep up with the rest when driven. Indications of diarrhœa. Placed in a coop, where it died during the night. Examined next morning.

"Slight odor of decomposition. A few small warts on skin of neck. The various organs were found normal, with the following exceptions:

"Mucosa of duodenum almost blackish, from intense injection and pigmentation of villi.

"Both cæca diseased. The left is slightly distended. On serous aspect two yellowish spots, with markedly injected borders, corresponding to thickenings of the walls near the blind end of tube. The mucous surface of one is smooth; to the other an exudate is attached. Besides the thickening of these spots, the free half of this cæcum is somewhat thickened uniformly.

"The right cæcum is very much distended over two-thirds of its length. From the serous surface local thickenings are recognizable, which have a yellowish, mottled appearance. The small intestine is firmly attached to one of these. ¯The disease has, however, not invaded the wall of the latter. The border of these spots is intensely hyperæmic. When the cæcum is slit open its width is three to four times that of the undisturbed tube, and the thickness of the wall varies from one-eighth to one-half of an inch, being not less than one-eighth of an inch over three-fourths of the entire length. When the brownish feces were washed away the increased local thickenings were found covered with firm exudates, usually attached in but one spot.

"Sections were examined of that portion of the caecal walls which was very much thickened, and to which the contiguous small intestine was inseparably attached by the new growth.

"The mucosa of the cæcal portion had sloughed away, while that of the imbedded small intestine was intact. The neoplastic tissue between cæcum and intestine was fully 1 cm. (two-fifths inch) thick. Inasmuch as the infiltration probably followed the narrow mesentery between cæcum and intestine the original boundary lines of the cæcal wall are no longer recognizable. The muscular coat of the cæcum may be traced for only a short distance into the neoplasm, when it appears. Microparasites were not seen distinctly in the diseased tissue.

"The liver is very much enlarged, and dotted everywhere with roundish spots of varying appearance. The majority are from 5 to 12 mm. in diameter, round or slightly oval. The center of each is usually occupied by a group of yellowish dots and the circle is bounded by a narrow yellowish ring. The space of the circle is mottled brownish. Among these spots there are also circles of a completely yellowish color. On the convex surface of the left lobe there is a very firm, ring-like, yellowish mass, cutting like firm cheese.

"In crushed preparations of fresh liver tissue from within the brownish circles many giant cells are seen. They consist of a meshwork of protoplasm of a rather coarsely granular character inclosing spheres which appear homogeneous. The giant cells are up to 30^{μ} in diameter.

"Sections of liver tissue hardened in alcohol and in Foa's solution were also examined. The foci of disease contain necrotic areas in which are numerous giant cells each inclosing a number of micro-parasites. In some portions there is much cell infiltration in the interlobular tissue around the portal vessels. Among the cells the protozoa are recognizable."

Diagnosis. This disease is to be diagnosed by the lesions and finding the specific organism in sections of the diseased ceca and liver. It is to be differentiated from certain local affections of the cecum, not especially uncommon in turkeys,* and coccidiosis. The lesions in the liver and the presence of the microparasite, as previously described, are sufficient to differentiate this disease.

Prevention. The present knowledge of this disease shows that the parasite is transmitted directly from diseased to healthy turkeys. This suggests that the first precaution is to avoid the transfer of diseased or seemingly healthy turkeys from a diseased flockin to a healthy one. The discovery of Chester indicates that a like precaution must be taken with reference to fowls. If the disease exists the best, although most radical, method as suggested by Smith is the total destruction of the affected flock, thorough disinfection of the roosts and droppings under the same, and the introduction of healthy turkeys. Curtice found the eggs do not carry the protozoa and that the specific organism is easily killed by drying.

REFERENCES

1. CHESTER. *Report of the bacteriologist of the Del. College Agric. Exp. Station,* 1899–1900. (C. reports disease in chickens).

2. CURTICE. Notes on experiments with blackhead of turkeys. *Circular No. 119. Bureau of Animal Industry,* 1907.

3. CUSHMAN. Nature of blackhead in turkeys. *Report R. Island Agric. Exp. Station,* 1894, p. 199.

4. HADLEY. Blackhead in turkeys: a study in avian coccidiosis. *Bulletin 141. Agric. Exp. Station, R. I. State College,* Kingston, 1910.

5. JOWETT. Epizoötic pneumo-pericarditis in the turkey. *Journ. Comp. Path. and Therap.,* Vol. XXI (1908), p. 324.

6. LAVERAN AND LUCET. Deux hématozoaires de la perdrix et du dindon. *Académie des Sciences,* 1905.

7. MILKS. A preliminary report on some diseases of chickens. *Bulletin No. 108, Agric. Exp. Station of La.,* Baton Rouge, 1908.

8. MOORE. The direct transmission of infectious entero-hepatitis in turkeys. *Circular No. 5, Bureau of Animal Industry,* 1896.

9. MUSGRAVE AND CLEGG. Amebas: Their Cultivation and Etiologic Significance. *Bulletin No. 18. Bureau of Government Laboratories, Manila,* 1903–4.

10. PERNOT. Disease of turkeys (Infectious entero-hepatitis). *Bulletin No. 95. Oregon Agric. Exp. Station,* 1907.

11. SMITH. Infectious entero-hepatitis in turkeys. *Bulletin No. 8, U. S. Bureau of Animal Industry,* 1895.

12. WOOLLEY. The pathology of intestinal amoebasis. *Pub. Bureau of Gov. Laboratories, No. 32.* Manila, P. I.

---

*Zürn (*Deutsche Zeit. f. Thiermed., Bd. X* (1883), p. 189) has described a cecal disease in water fowls and turkeys and von Ratz has described a cecal disease in turkeys in which the liver lesions seem to be absent.

CHAPTER X

DISEASES CAUSED BY PROTOZOA
GENUS PIROPLASMA

General discussion of piroplasma. The genus *Piroplasma* belongs to the order *Hemosporidia* or blood dwelling sporozoa, cytozoic in mode of life in the blood constituents and with or without alternation of hosts. In 1888, Babes first described a blood parasite in European cattle which he designated *Hematococcus bovis*. In 1893, Starcovici gave it the generic name *Babesia*.*

In 1893, Smith described the parasite that causes "Texas Fever" in cattle which he named *Pyrosoma bigeminum*. The generic name, having been previously used for a genus of tunicates, was changed, in 1895, by Patton to *Piroplasma*. Since that time a number of these parasites have been found in different animals. The organism causing East Coast fever in Africa was named by Theiler, in 1904, *P. parvum*. Babes described in 1892 a blood parasite in Roumanian sheep which is known as *P. ovis*. Piana and Galli-Valerio found in 1895 a similar parasite in dogs which is known as *P. canis*. In 1899, Gugliemi found a similar organism in the blood of horses which is known as *P. equi*. *P. mutans* is a widely distributed species found in the blood of cattle in South Africa. *P. parvum* is the cause of East Coast fever. Its generic name was changed to *Theileria* by Bettencourt. A number of other species have been described but they are of little etiological significance.

Theiler describes a small body found in the marginal zone of the red blood corpuscle in certain cases of Texas Fever as *Anaplasma marginalis*. He differentiates this organism from *P. bigeminum*, *P. mutans* and *P. parvum*.

The life history of these parasites is not known. In the infected animal they live within the blood, often entering into and destroying the red blood corpuscles. They are transmitted from the infected to

---

*Most writers on protozoology accept the generic name *Babesia* on the assumption that the organism found by Smith and Kilborne is identical with the one described by Babes. On the other hand, most of the writers on the diseases caused by this parasite retain the name *Piroplasma* for the genus and Nocard's term Piroplasmoses for the diseases produced by the different species.

the uninfected animal by means of ticks. The known species of this genus are parasitic and most of them at least are pathogenic for different species of animals. The term *Piroplasmoses* was introduced by Nocard to designate the diseases caused by the various species of the genus *Piroplasma*.

The only species of pathogenic *Piroplasma* that is of special interest to Americans is the one that causes Texas fever in cattle. Piroplasmosis has been reported once in sheep but the organism was not found. It has likewise been reported in dogs but as yet Texas fever is the only serious disease in this country caused by these organisms.

TEXAS FEVER

Synonyms. Piroplasmosis; bovine malaria; red water; Spanish fever; splenic fever; bloody murrain; southern cattle fever; tick fever.

Characterization. Texas fever is an infectious disease of cattle, characterized by rise of temperature, hemoglobinuria, destruction of the red blood corpuscles and the presence in the blood of a protozoan parasite which is transmitted from animal to animal by means of the cattle tick.

It is believed to be identical with the hemoglobinuria in Roumania, tick fever in Australia, and "La Tristeza" in South America. It has been named *malaria des bovidés* by Celli and Sentori and *Malaria bovine* by Lignières. Although it differs in many ways from human malaria, the analogy is so close respecting the specific cause and means of its transmission, that bovine malaria seems to be a very suitable name for this affection. At least it has the advantage of not stigmatizing any locality.

The peculiar and interesting feature of this affection is the fact that cattle raised in the infected districts become immunized so that they do not suffer from the disease but they still carry, in some stage, its specific organism in their blood. When such animals are imported into non-infected districts, they transmit the virus, by means of the cattle tick, to susceptible cattle, but remain themselves perfectly well.

History. There is little knowledge concerning the early history of this disease. In 1868 it seems to have made its first impression upon the American people. In June of that year, Texas cattle were shipped up the Mississippi river to Cairo and thence by rail into the states of Illinois and Indiana, where they caused, during the summer, enormous

losses from this disease. Cattle from these states shipped east brought the disease with them. The cattle commissioners of New York and the Board of Health of New York City endeavored to check the importation of such animals. It was carefully investigated at that time but nothing beyond a very accurate description of the gross lesions was accomplished. Later, Salmon determined the boundary line between the non-infected and the permanently infected districts, or what is now known as the Texas fever line. In 1889, the United States Bureau of Animal Industry undertook a systematic investigation as to the nature of this disease, which resulted in the discovery of its specific cause by Dr. Theobald Smith and the demonstration of the fact that the cause of the disease is transmitted from southern to northern cattle through the medium of the cattle tick. Prior to this (1888), v. Babes had found an intraglobular parasite in the blood of cattle suffering from an epizoötic disease (hemoglobinuria) in Roumania. While at first these diseases were thought to be different in their etiology they are now believed to be identical.

Geographical distribution. In the United States the distribution of Texas fever corresponds with that of the blue cattle tick (*Margaropus (Boöphilus) annulatus*). This includes, with possibly a few small exceptional areas, that portion of the country south of the "Texas fever line." It has been identified with the tick fever of Australia which has become a source of great loss to the cattle industry of that country. Lignières has identified the disease in South America (Argentine Republic). It is also reported to be identical with a malady affecting cattle along the Danube river, in the Balkan provinces and in South Africa. It is restricted, however, to those countries where the climate is not sufficiently severe to destroy the cattle tick during the winter season. Cattle (genus *Bos*) are the only animals which suffer from it.

Etiology. Texas fever is caused by a microörganism belonging in the protozoa discovered by Smith and named by him, *Pyrosoma bigeminum.** The generic name Piroplasma given it by Patton is generally accepted.

It seems that Dr. Stiles, in 1868, observed this organism but failed to recognize its significance. It is found in the blood in cases of Texas

---

*The genus of the parasite has been changed to *Piroplasma* by Patton, to *Apiosoma* by Wandelleck, to *Aœmbosporidies* by Bonome, and *Proteus virulentissimus* by Perroncito. Starcovici has named the organism described by Babes as Hematococcus, *Babesia bigeminum bocvis.*

fever and it also exists in the blood of immune animals in the tick infested districts. The life history of this parasite has not been determined. In the blood of the diseased animals they appear in the unstained, fresh preparation, as minute or larger bright bodies which may be from 0.5 to 4.0μ in diameter according to the form of the disease. In the acute types certain of the red corpuscles contain pale or brighter pyriform shaped bodies. One end of each body is broad and rounded, the other tapering and pointed. Usually there are two of these bodies, both of the same size, in a corpuscle. More rarely there is but one, although four are occasionally observed (Fig. 78). When two are present the tapering ends approach each other and usually they are joined while the other ends may point in any direction. Several forms have been noted varying from a round to a pyramidal outline. The small and often the larger bodies have been observed to change their position within the red corpuscle. Smith has noted that the ameboid bodies observed were apparently single within the corpuscle. In dried and heated cover-glass preparations, stained with alkaline methylene blue, these parasites are distinctly colored. They are also stained with carbol fuchsin and with hemotoxylin. As a rule they stain more deeply in preparations made from parenchymatous organs than they do in those from the living blood.
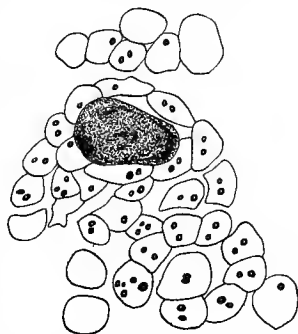
FIG. 77. COVER-GLASS PREPARATION FROM KIDNEY. CORPUSCLES SHOWING PIROPLASMA, COCCUS FORM (*Smith*).

In the capillaries of the congested organs, the blood corpuscles contain many more parasites. Smith has noted in one case from 2 to 3 per cent. of infected corpuscles in the circulating blood but in cover-glass preparations made at the autopsy from the skeletal muscles, blood of the right heart, and blood from the bone marrow (sixth rib) very few infected corpuscles were found. In the preparations of blood from the left heart and lung tissue from 2 to 3 per cent. of the corpuscles were infected; those from the spleen 5 per cent.; from the liver and kidney tissue from 10 to 20 per cent.; and in the hyperemic fringes of the omentum and the heart muscle 50 per cent. of the corpuscles were infected. In other cases the blood corpuscles in the capillaries were more and in still others less extensively invaded. In

the living blood the parasites were pyriform, but in the post-mortem specimens they were more nearly round. In the mild type of the disease from 5 to 50 per cent. of the red corpuscles in the circulation are infected for a period of from one to five weeks. The parasite is round (coccus form). In the fresh preparations it is seldom seen; rarely it can be detected as a pale spot about 0.5µ in diameter at the periphery of the corpuscle. In stained (alkaline methylene blue) preparations, the parasites appear as round coccus-like bodies from 0.2 to 0.5µ in diameter. They are situated within the corpuscle on its border. As a rule only one is found in a corpuscle. Sometimes a division is evident separating the parasite into two parts. They must be differentiated from somewhat similar looking bright bodies
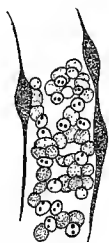
FIG. 78. BLOOD
IN CAPILLARY
OF HEART
SHOWING
PIROPLASMA
(Smith).
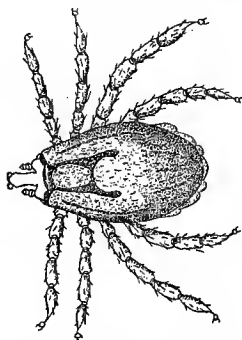
FIG. 79. SEXUALLY MATURE
MALE TICK AFTER THE LAST
MOULT, DORSAL VIEW
(Smith).

which are seen in the corpuscles of healthy blood during different seasons of the year.

Concerning the life history of this parasite, Smith considered the intraglobular stage hypothetically the swarming stage, which precedes the peripheral coccus-like bodies and the pyriform and spindle shaped bodies which develop from the divided coccus-like peripheral forms. The free bodies are the parasites set free after they have reached the preceding stage by disintegration of the infected corpuscles. They are most commonly found in the kidney. The reproductive stage has not been recognized.

Infection. Although practical stockmen had long looked upon the tick as a source of infection, it remained for Smith and Kilborne to

experimentally demonstrate that so far as known the cattle tick (*Boöphilus annulatus*)* is the sole carrier of the parasite.† In Germany and Finland it is said to be transmitted by *Ixodes reduvius*; in Africa, *Rhipicephalus decoloratus*, *R. appendiculatus* and *R. evertsi*; and in Australia by *R. Australis*. It was pointed out by them that when southern cattle were freed from ticks they would not when kept together in small enclosures transfer the disease to susceptible animals, but that when susceptible cattle became infested with the ticks

FIG. 80. PHOTOGRAPH OF ANIMAL SICK WITH TEXAS FEVER.
(*Photographed by Connaway*)

either by grazing in infested pastures or by having placed upon them young ticks hatched in the laboratory the disease appeared.

The infection of northern cattle with Texas fever by southern

---

*This tick was first described by C. V. Riley in 1868 as *Ixodes bovis*. Later, Cooper Curtice investigated this parasite (Biology of the Cattle Tick, Journ. Comp. Med. and Veterinary Archives, July, 1891, Jan., 1892) and gave it the generic name of *Boöphilus* (ox loving). This seems to be the only genus of cattle ticks which transmits the parasite of Texas fever. Recently Karsch's genus *Margoropus* has been proposed as the correct name instead of Boöphilus.

†Crawley has recently found what appears to be a parasitic protozon in the smears made from female cattle ticks and from crushed eggs which they had deposited. He believes it to be a stage in the life history of *P. bigeminum*. *The Journal of Parasitology*, Vol. II (1915), p. 87.

animals consists therefore in first infesting them with the cattle tick.* The number of ticks necessary to carry the disease is small so that frequently they will not be observed unless the sick animals are carefully examined. The life cycle of the tick will, therefore, explain the variation in the time elapsing between the exposure of northern to southern cattle and the appearance of the disease. Starting with tick infested animals placed with native cattle in a northern pasture the adult female ticks drop to the ground almost daily, so that the following life cycle may be assumed to begin at once.

Adult ticks drop to the ground in from 1 to 3 days after the infested cattle are placed in the field.

Adult ticks lay their eggs in about 7 days after dropping to the ground.

Eggs are hatched in about 20 days after they are laid.†

Young ticks crawl upon cattle from 1 to several days after they are hatched.

FIG. 81.   CATTLE TICKS ON AN INFESTED ANIMAL
(after Mohler).

In about ten days from the time the young ticks crawl upon the susceptible cattle the first symptom, a rise of temperature, appears.

The length of time that must elapse (period of incubation) from the

---

*It is interesting to add the results of an experiment conducted by Dr. Cooper Curtice in the Bureau of Animal Industry which shows the necessity of the tick in inoculating cattle.   In a tick infested district in the south, a field was cleared of ticks by fencing and keeping cattle off for a year. Susceptible northern cattle were transported to and placed in this field where they thrived for a season.   The second year they were placed in a second cleared pasture where they kept well for another year.   The third year they were placed in a tick infested pasture where they died promptly of Texas fever.

†Mohler states that the time required for the hatching varies from 13 days to six weeks, depending upon temperature, moisture, soil, etc.   He states further that the eggs may remain dormant for several months.

exposure of susceptible cattle to the development of the disease depends on whether or not the whole life cycle of the tick must be passed or part of it has already gone by. If susceptible animals are placed in a pasture where the young ticks are just ready to crawl upon them the infection of the cattle is accomplished at once and the high fever appears in about ten days, practically the minimum time. It has been experimentally demonstrated that the young ticks are able to travel for a considerable distance in a pasture. In pastures where tick infested cattle are grazing, young ticks are very liable to be on the ground continuously. In estimating the time to elapse after the exposure to the tick infested animals or field, before the disease will appear, it is necessary to know in what stage in the life cycle the ticks are at the time when the susceptible animals come in contact with them. Cattle have been exposed in feeding pens to young ticks that had hatched from eggs laid by adult ticks left in the yards some time before by infested cattle and developed the disease ten days after exposure.

Small quantities of the blood from immunized cattle in the tick infested district, when injected into susceptible animals either intravenously or beneath the skin, will produce the disease. While this mode of infection rarely if ever occurs in the natural order of events, it may happen that in cases of certain operations bits of blood may be carried directly from a southern to a northern animal thus inoculating the latter with the disease.*

Symptoms. In the acute type of the disease which occurs during the hot summer months, the onset is sudden and usually all animals exposed to the same infection together come down at the same time. The first indication of the disease is a rise of temperature, at first higher in the afternoon than morning, but later in the course of the disease the temperature remains high. It rarely rises above 107° F. The rise of temperature occurs two or more days before there are other symptoms. The respiration may rise to between 60 and 100 and the pulse may range between 80 and 100 per minute. Late in its course

---

*In the fall of 1898 two cases occurred in the practice of Dr. Ambler of Chatham, N. Y. The owner had his animals dehorned in December and soon afterward two fatal cases of Texas fever developed. The *Piroplasma* and the characteristic lesions were present. Inquiry revealed the interesting fact that the two animals which sickened and died were dehorned immediately after two imported southern cattle. The owner was not aware of the fact at the time that these were southern bred cattle, as he had bought them of a dealer in Vermont. More recently another case of this disease produced in the same way has been reported.

there may be hemoglobinuria.   Smith and Kilborne found it in 33
out of 46 fatal cases in which urine was found in the bladder.   The
passing of the colored urine before death was noted in but four of their
cases.   In one animal which showed hemoglobinuria four days before
death, the urine in the bladder was clear at post-mortem.   As this
condition seems to depend upon the rapidity of the destruction of the
red blood corpuscles, a slow disintegration may enable other organs to
dispose of the coloring matter, while in rapid destruction of the blood
much of it may be thrown into the urine.   The urine contains small
quantities of albumin.   At first the specific gravity may be high but
later it ranges from 1010 to 1020 and fails to effervesce with acids.
The color varies according to the quantity of hemoglobin.   As a rule
there is marked constipation, loss of appetite and usually cessation
of rumination during the high fever.   The blood is thin and pale.

The high temperature,
hemoglobinuria and thin-
ness of the blood are quite
diagnostic symptoms of
the acute type.

The course may vary,
but the continuous high
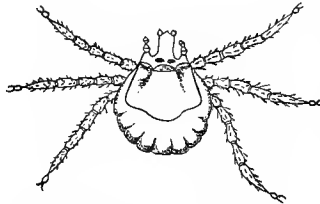
FIG. 82.   EGGS AND YOUNG TICK, JUST HATCHED.   temperature does not us-
(*Smith*).   ually last for more than

ten days.   Death often intervenes in from five to eight days.
In the mild, nonfatal or chronic type which was first pointed
out by Smith and Kilborne and which occurs in the late sum-
mer or autumn, the general symptoms are similar to those of
the acute type except that they are not so severe and are prolonged
for a greater length of time.   The parasite is of spherical or coccus
form.   The general symptoms are not manifested unless the tempera-
ture goes above 103° F.   Hemoglobinuria is not observed in this type.
Cattle which have passed through an acute type owing to the heated
season may have a relapse in the form of a mild type in the fall.   The
essential difference between the two types is found in the different
stages of the parasite circulating in the blood.*   Unless the tempera-

---

*Theiler has differentiated this form of the parasite from *P. bigeminum* and placed
it in a new genus *Anaplasma* of which he has two species, *A. centrale* and *A. marginale*.
He also holds *Anaplasma* to be an organism reduced by parasitism, having lost its
"originelles" and its plasma so that phylogenetically it represents one of the oldest of

ture is taken and the blood carefully examined, mild types of Texas fever would be either overlooked or mistaken for any one of a variety of disorders common among cattle.

Morbid anatomy. Cattle which die of Texas fever undergo post-mortem changes very rapidly. For this reason the description of lesions made some hours after death may be misleading.

Externally the animal presents nothing abnormal or characteristic of the disease. Rarely dried bits of blood may be found and also some small slightly elevated areas of a bluish color. The skin between the thighs, upon and about the udder and possibly elsewhere may have cattle ticks attached. It is important under ordinary circumstances to look for this parasite. The subcutaneous tissue may be more or less yellow in color. Edema of the subcutis over the ventral portion of the body has been observed. The muscles are usually normal in appearance although frequently they are pale.

Very slight if any lesions have been recorded in the central nervous system and lungs. Blood extravasations usually occur beneath the skin and endocardium, especially of the left ventricle. On the external surface the petechiæ occur for the greater part along the intraventricular groove near the base. The capillaries of the heart muscle are packed with corpuscles. Parenchymatous and fatty degeneration of the muscular fibers sometimes exist. The right ventricle is distended with blood either fluid or clotted and the left one firmly contracted.

In the abdominal cavity there are frequent edematous areas about the kidneys and in the portal regions between the duodenum and liver. The omentum is often sprinkled with peculiar hyperemic patches con-

---

the *Piroplasmata*. It is transmitted by the tick *Boophilus decoloratus*. Theiler further states that *Anaplasmosis* is the cause of the South African Gall Sickness.

"Two forms of anaplasmoses are recognized—a mild one which never leads to death, although the anemia may be strongly pronounced, and a malignant one, in which about 50 per cent. of the cases end in death, with the symptoms of anemia and icterus. The mild form is characterized by the presence of *A. centrale*, the malignant by *A. marginale*. The former is generally situated within the corpuscles, and is smaller than the latter, which is mainly placed on the margin. There is a difference of opinion about the protozoic nature of these anaplasma, and some authors are inclined to identify them with Jolly's bodies, but from this view we differ, basing our opinion not exclusively on the morphological appearance of the bodies, but rather on their biological behavior in connection with the disease."

According to Balfour, Jolly was the first to describe the small rounded chromatine bodies in the red blood corpuscles in the blood of rodents and referred to as Jolly bodies. Dodd points out the possibility of confusion between anaplasma and these bodies.

sisting of delicate shreds of vascular tissue.   This condition, however, is not characteristic of Texas fever.

Usually the most conspicuous changes are in the spleen.   This organ is much enlarged and its normal weight increased from two to four times.   The normal markings, Malpighian bodies and trabeculæ are hidden in the dark brownish-red, glistening pulp which distends the capsule.   The pulp may be firm or it may be in a semi-fluid condition so that it oozes out if the surface is cut.   The enlargement and color of the spleen are due to an engorgement of red blood cells. There may also be present a greater or less number of large cells containing granules, red corpuscles or clumps of yellowish pigment.   Free pigment is much more abundant than it is in healthy spleens.

The liver is extensively affected.   It is enlarged, congested, edges rounded, the bile ducts more or less distended and the parenchyma is usually in a state of fatty degeneration.   The color is paler than normal and usually the surface is somewhat mottled.   On section the color of the cut surface is brownish-yellow or it may be mottled like the surface.   The mottling is due to a discoloration owing to degeneration of a zone bordering the intralobular vein.   This zone varies in width and its peculiar color seems to be due to a tendency to necrosis. It is characterized by parenchymatous degeneration and the loss of nuclei.   It may involve a third or more of the lobule.   This portion stains very feebly or not at all.   The explanation for the necrosis of the liver cells is suggested by Smith as being due to the plugging up of the ultimate bile canals with solid bile which may interfere in some way with the nutrition of the adjacent liver cells.   The bile stasis he considers as a result of the breaking up in the capillaries of the liver of enormous numbers of infected corpuscles.   This results in an abnormal fluid containing an excess of solids which the bile ducts are unable to carry away.   When examined in fresh condition or in sections of tissue fixed in Müller's fluid the engorgement of the bile canaliculi is seen.   The bile stasis may occur over a portion or a whole of the lobule.   The gall bladder contains usually an abnormal quantity of changed bile.   It is thick and often semi-solid in consistency, holding in suspension many flocculi.   It imparts a deep yellowish tinge to all articles coming in contact with it.   Owing to the mucus which is present it is quite viscid.

The changes in the kidneys vary.   If death occurs early they are usually enlarged and uniformly darker than normal throughout. The capillaries are distended with red corpuscles.   Parenchymatous

and fatty degenerations are not common, although occasionally present in the epithelium of the tubules. The pelvis is often sprinkled with ecchymoses.

The bladder may contain ecchymoses.

It is important to note that throughout the kidneys, liver and spleen pigment may be more or less abundant.

In the digestive tract the lesions of this disease consist largely of congestion of the mucosa especially in lines corresponding to the summits of the folds of the mucous membrane. It is more marked in the cecum and rectum than in the colon. The cecum and less often the rectum contain dry, hard fecal balls. In some cases in the intestines lesions are not observable. In the older writings much emphasis is placed on certain lesions, mostly nodular or abrasions, in the digestive tract. · Smith has shown, however, that most of these are due to animal parasites and have no relation to Texas fever.

As already stated, Texas fever is a disease of the blood and consequently it is in this fluid that the most constant morbid changes occur. They are characterized by the blood becoming thin and watery with a destruction of the red blood corpuscles. In some of the cases the loss of corpuscles is rapid and continuous until death or convalescence, while in others there is a marked oscillation between destruction and regeneration. In some animals the loss is not continuous, but the course of the disease is marked by the periods of rapid blood destruction and periods of rest, or, as it were, where the blood destruction was holding its own. These points are best illustrated from actual cases, three of which are taken from the report by Smith and Kilborne.

(No. 129)		(No. 142)		(No. 56)	
Date	No. of Corpuscles	Date	No. of Corpuscles	Date	No. of Corpuscles
Aug. 11,	6,125,000	Sept. 16,	6,890,000	Sept. 20,	6,844,000
13,	7,171,000	22,	5,430,900	22,	5,640,000
16,	5,370,000	24,	4,562,000	29,	2,307,000
27,	3,310,000	29,	5,274,000	Oct. 9,	5,436,000
29,	1,675,000	Oct. 4,	3,902,000	22,	4,666,000
30,	Died 8 P. M.	8,	5,983,000	25,	2,754,000
		22,	4,333,000	30,	2,720,000
First high A. M. temp.		Nov. 4,	5,586,000	Nov. 6,	2,344,000
Aug. 24.				8,	1,984,000
				13,	1,183,000

As evidence of the diminution of the number of corpuscles within the body these authors point (1) to the loss of hemoglobin through the kidneys, (2) to the overproduction of bile which is abnormal in the abundance of pigment and (3) to the actual observation of their destruction by the micro-parasite under the microscope.

The regeneration of blood corpuscles is indicated perhaps by the count, but more surely by the forms of the corpuscles themselves. The abnormal forms are the very large corpuscles, "punctate" forms and lastly the diffuse stained or "tinted" forms and the erythroblasts. The first of these may appear when the blood count reaches 3,000,000 and the other forms when it is still lower. These various forms, however, are probably embryonic or immature corpuscles, which are forced prematurely into the circulation by the blood producing organs in trying to overcome the rapid destruction of corpuscles by the parasite. The action of the disease upon the leucocytes or the defensive activities of the white corpuscles in combating the parasite of Texas fever have not been determined. In the work thus far performed and reported, they have received little attention. Suffice it to say that they have not been in evidence in this conflict and probably take little or no part in the morbid changes of Texas fever.

Diagnosis. Texas fever is easily diagnosed by the presence of its specific parasite. Its symptoms and lesions are sufficient for diagnostic purposes in the typical cases. Texas fever is to be differentiated from anthrax and poisoning. The possible existence of *Anaplasmosis* may necessitate its differentiation from that infection.

The lesions are not simulated by any other disease of cattle, although the enlarged, dark spleen may suggest anthrax and in a hasty diagnosis the two may be confused. From the fact that all animals exposed together usually come down with the disease together poisoning may be suggested, but here again a study of the symptoms and lesions are sufficient to eliminate toxic disorders.

Prevention. The discovery of the specific cause of Texas fever and of the cattle tick as the common means of its transmission has reduced the preventive measures to a direct warfare against the tick. The National government has determined the territory in which the tick naturally exists and from which cattle, on account of the parasite, cannot be shipped to uninfected districts, except under certain very restricted conditions. (See regulations of the Bureau of Animal Industry for shipping cattle). Likewise susceptible northern cattle

cannot be transported to the infested districts (south of Texas fever line) unless they can be placed in fields that have been freed from ticks. The elimination of the disease depends therefore upon the elimination of *Boöphilus annulatus*. The government, in coöperation with the States, has undertaken to eradicate the ticks. Thus far the reports are encouraging. There seems to be no reason why their eradication cannot be accomplished.

Immunizing susceptible cattle. A number of investigations have been undertaken directed toward the development of methods for immunizing northern cattle against Texas fever in order to enable the shipment, especially of breeding stock, into the South. In 1895, the writer, in conjunction with Schrœder, began an immunizing experiment which was continued and reported by Schrœder in 1898.

Young animals were selected and injected with blood taken directly from the jugular veins of southern animals. The injections were made in the fall and winter and in the spring the animals were placed in a highly infected field at Manchester, Va., where they remained for the summer. During this time they were under the immediate observation of Curtice, who made a careful study of the blood, temperature and extent of tick invasion. The results of this experiment are shown in Schrœder's tabulation, which is appended. The animals were again exposed the following season without the development of Texas fever.

Animal No.	Effect of blood injection	Effect of the exposure to cattle ticks
1	Severe reaction.	Very mild disease.
2	" "	Well marked but mild.
3	Very severe reaction.	Very mild.
4	" " "	No disease.
5	Mild reaction.	" "
6	" "	Very mild.
7	Severe reaction.	No disease.
8	" "	" "
9	" "	" "
	Control Animals.	
10		Died June 20.
11		" July 9.
12		" June 26.
14		" July 9.
14		Very severe disease, recovered.

The inoculation disease appears in from eight to ten days after the injection of the blood. It lasts from one to two weeks. The symptoms are occasionally of a still shorter duration, but the altered condition of the blood persists in some cases for a much longer period.

Dalrymple, Dodson and Morgan, of the Louisiana Experiment Station, conducted experiments along this line. They showed that immunity against a fatal attack of Texas fever can be conferred on susceptible cattle by inoculation with the blood of a native Southern animal or one which has recently been rendered immune.

Lignières reports good results in the vaccination of cattle against piroplasmoses in Argentina. He also reports that animals that are resistant to *P. bigeminum* may be very susceptible to *P. argentinum*. He used, in South America, two vaccines: the first, blood taken from diseased cattle at the time it contained the greatest number of parasites and kept at $5°$ to $8°$ C. for 30 days, when it was injected intravenously in doses of 10 cc. each. This produced a mild form of disease followed by immunity. Ten days after the first injection, he injects 1 cc. of blood equally rich in piroplasma but which had been kept for 15 days only at temperature of $5°$ to $8°$ C. This acted as a test noculation.

Lignières found that while blood taken from diseased animals retains its virulence for several days, especially at a temperature of $5°$ to $8°$ C., he also states that if the vaccine be injected under the skin the toxin to which its vaccination properties are due is destroyed before it reaches the blood stream. The preparation of vaccine from blood containing *P bigeminum* by desiccation or freezing, he states, does not apply to *P. argentinum* because this organism remains alive and virulent after freezing.

In Missouri, Connaway has immunized a few animals with the blood serum from immune (southern) cattle. In Mississippi, Robert has tried the serum for both prevention and treatment with somewhat similar results. In Virginia and Oklahoma the disease and its prevention have been studied. In Texas, Francis immunized cattle with most excellent results. He has pointed out the value of immunizing young (2 to 6 weeks old) calves by artificially infesting them with ticks. In Australia the problem of immunizing cattle against "tick fever" has become a matter of serious consideration. Certain European writers claim that immunity can not be permanently induced by artificial injections. Schrœder has shown that the parasite remains

virulent in the blood of naturally immunized cattle for from 10 to 12 years.

The very limited knowledge of the life cycle of the parasite of Texas fever precludes a satisfactory explanation of the *modus operandi* in the production of immunity by these various procedures. On this point, there is need for much additional investigation.

REFERENCES

1. BABES. Die Aetiologie der seuchenhaften Hämoglobinurie des Rindes. *Virchow's Archiv*, Bd. CXV (1889), S. 81

2. CONNAWAY. Texas fever or acclimation fever. *Bulletin No. 37. Mo. State Board of Agriculture*, 1897.

3. CONNAWAY AND FRANCIS. Texas fever. *Bulletin No. 48. Agric. Exp. Station, Mo.* 1899.

4. CURTICE. Progress and prospects of tick eradications. *Twenty-seventh Annual Report, B. A. I.*, 1910, p. 255.

5. DALRYMPLE, STAPLES, MORGAN AND DODSON. Texas or southern cattle fever. *Bulletin 51. Louisiana Agric. Expt. Station*, 1898.

6. DAWSON. Cattle tick eradication. *Publication 103 State Board of Health oj Florida*, 1913.

7. DINWIDDIE Some Texas fever experiments. *Bulletin No. 20. Ark. Agric. Exper. Station*, 1893.

8. DODD. Anaplasm or Jolly bodies. *Jour. of Comp. Path. and Therap.*, Vol. XXVI (1913), p. 97.

9. FRANCIS AND CONNAWAY. Texas fever. *Bulletin No. 53. Texas Agric. Expt. Station*, 1899.

10. GAMGEE, DODGE, BILLINGS AND CURTIS. Diseases of cattle in the United States. *Report of the Commissioner of Agriculture, Washington, D. C.*, 1871.

11. HUGHES. The fight against Texas fever. A review of the work being attempted in the south for the control and eradication of the disease. *Am. Vet. Review*, Vol. XXIX (1906), p. 1309.

12. HUNT AND COLLINS. Report on tick fever. Brisbane. Queensland, Australia, 1899.

13. KLEIN. Methods of eradicating cattle ticks. *Circular No. 110. B. A. I.*, 1907.

14. KOCH. Vorläufiger Bericht über das Rhodesische Rotwasser oder "Afrikanisch Küstenfieber." *Archiv. für wiss. u. praktische Tierheilkunde*, Bd. XXX (1904), S. 281.

15. LEWIS. Texas fever. *Bulletin No. 39. Oklahoma Agric. Exper. Station*, 1899.

16. LIGNIÈRES. La "Tristeza" ou Malarie Bovine dans la République Argentina Buenos Aires, 1890. (Full bibliography),

17. LIGNIÈRES. Vaccination against bovine Piroplasmosis. *Rev. Général de Méd. Vét.*, 1911, p. 489. Reviewed *Jour. Comp. Path. and Therap.*, Vol. XXV (1912), p. 59.

18. MAYO. Texas fever. *Bulletin No. 69. Kan. Agric. Exper. Station*, 1897.

19. McCULLOCH. The prevention of Texas fever and the amended laws controlling contagious disease. *Bulletin No. 104. Virginia Agric. Exper. Station*, 1899.

20. MOHLER. Texas fever. *Bulletin No. 78. Bureau of Animal Industry*, 1905.

21. NILES. Splenetic or Texas cattle fever. *Bulletin No. 61, Virginia Agric. Exper. Station*, 1896.

22. NORGAARD. Dipping cattle for the destruction of ticks. *Annual Report, Bureau of Animal Industry*, 1895–6, p. 109.

23. PAQUIN. Texas fever. *Bulletin No. 11. Mo. Agric. Exper. Station*, 1890.

24. SALMON. Contagious diseases of animals. *Special report No. 22, Washington, D. C.*

25. SALMON. *Report Commissioner of Agriculture*, 1881–2.

26. SALMON. *Annual Reports, Bureau of Animal Industry*, 1884–5.

27. SCHROEDER. Inoculation to produce immunity from Texas fever in Northern cattle. *Ibid.* 1898, p. 273.

28. SCHROEDER. Notes on cattle tick and Texas fever. *Annual Report of the Bureau of Animal Industry*, 1905, p. 49.

29. SCHROEDER AND COTTON. The Persistence of Texas fever organism in blood of southern cattle. *Ibid.*, p. 71.

30. SMITH. Preliminary observations on the microörganism of Texas fever. *The Medical News*, 1889.

31. SMITH AND KILBORNE. Texas fever. *Bulletin No. 1, Bureau of Animal Industry, U. S. Dept. Agriculture*, 1893.

32. SMITH AND KILBORNE. *Annual Report, Bureau of Animal Industry*, 1891–2. (*Issued 1893*).

33. STILES. *Report New York State Board of Health*, 1868.

34. STOCKMAN. Some points to be considered in connection with Rhodesian red-water. *Jour. Comp. Path. ond Therap.*, Vol. XVIII (1905), p. 64.

35. THEILER, GRAY AND POWER. Diseases transmitted by ticks; their classification, treatment and eradication. *Tenth International Vet. Cong.*, London, 1914.

36. THEILER AND STOCKMAN. Some observations and experiments in connection with Tropical bovine piroplasmosis (East-coast-fever or Rhodesian Redwater). *Jour. Comp. Path. and Therap.*, Vol. XVII (1904), p. 193.

37. VINCENHELLER. Cattle tick eradication in northwestern Arkansas. *Bulletin No. 93, Ark. Agr. Expr. Station*, 1907.

CANINE MALARIA

Synonyms: Piroplasmosis of dogs; malignant malarial jaundice; malignant jaundice in the dog; tick fever.

Characterization. This disease is characterized by a high temperature, rapid course, jaundice and anemia. These are due to the invasion of the blood with *Piroplasma canis*.

History. In 1895, Piana and Galli-Valerio found the piroplasma in the blood of dogs in Italy. In 1899, Hutcheon described a malarial fever in dogs that could be transmitted by inoculation subcutaneously with the infected material. Koch found the disease in Africa, and Celli mentions a modified form of the affection in Lombardy. In 1901 Nocard and Almy reported several cases of piroplasmosis in dogs presented at the clinic of the Alfort Veterinary school. Robertson described the disease under the name of malignant jaundice and pointed out its transmission by the dog tick (*Haemaphysalis leachi*). In 1902, Nocard and Motas reported an experimental study of the specific parasite. More recently Nuttall and Graham-Smith have investigated this affection.

Geographical distribution. This disease has been found in several places in Africa, India, Italy and France.

In 1908 Phillips and McCampbell reported an outbreak of piroplasmosis in dogs from Ohio.

Etiology. The *Piroplasma canis* is the cause of this affection. Like *Piroplasma bigeminum* it appears in two forms, round and pear-shaped. They nearly always occur in the red blood corpuscles. It varies in its morphology. According to Marchoux *Piroplasma canis* in France is larger than *Piroplasma bigeminum* and single parasites occur more frequently. They vary in size from 2 to 4μ. At the beginning of the disease usually single large round forms appear. It is said by Nocard and Motas not to be pathogenic for other species of animals. The parasite is said to be transmitted by means of certain insects. Lounsbury has demonstrated that *Pulex serra liceps*, which is very abundant in certain localities, plays a rôle in its transmission. In Italy *Piroplasma canis* is supposed to be transmitted by *Ixodes ricinus*. In France it is supposed to be conveyed by *Dermacentor reticulatus*. In South Africa it is conveyed by *Haemaphysalis leachi*. The parasite appears in the blood in from two to four days after artificial infection.

Graham-Smith did not find the parasites in films from the peripheral circulation when stained by Leishman's method until several days after infection. The earliest day on which they appeared was the sixth day and the latest the thirty-sixth day. In most cases they were observed between the eighth and twelfth days after infection. In a few cases he worked out the percentage of red blood corpuscles that were infected. He found that it varied from .3 to 1.4%. Free parasites were seldom encountered in the earlier stages but later they became more numerous. The day before death he found one free parasite to 23 infected corpuscles, and the day of death the free parasites were still more numerous. He found little evidence of phagocytosis in the cases examined. Nucleated red corpuscles appeared in films from three dogs the day before death, and in three other dogs they appeared six days before death. Blood pigment was present in nearly all samples of urine. Young dogs are more susceptible than old ones.

It is possible that the piroplasma of dogs is of different species in different countries. M'Fadyean and Nuttall found dogs that had recovered from piroplasmosis in India to be susceptible to the African infection.

The period of incubation after direct inoculation is about three days. When it follows exposure to the infected tick it is reported to be between ten and twenty days.

Symptoms. Two distinct forms have been described: the acute which is nearly always fatal, and the chronic which often terminates in recovery. In the acute form the dog is dull, drowsy and refuses food. It may be thirsty. The temperature is high (104° F.) but after two or three days it drops to subnormal. Icterus is not constant and the hemoglobinuria is not always present. The blood is pale and it coagulates slowly. The red corpuscles are reduced to 2,000,000 per cubic millimeter or below. The polynuclear leucocytes are increased in number. In the acute cases death nearly always occurs in from three to six days.

In the chronic cases the fever remains high for from 36 to 48 hours, when it returns to normal. The anemia is the most constant manifestation. The mucosæ become pale and the appetite is poor. The symptoms persist for from three to six weeks. Recovery frequently takes place.

The duration in fatal cases is from 3 to 6 days. In chronic cases it may be several weeks.

Morbid anatomy. The mucous membranes are pale and the subcutaneous tissue and fat more yellow than normal. The spleen is 3 or 4 times the normal size. The liver is engorged with blood which is heavily charged with parasites. The gall bladder is distended with greenish bile. The mucosæ of the digestive tract are slightly, if at all, changed. The kidneys are congested, often sprinkled with petechiæ or ecchymoses. The capsule is easily removed. The parasites are found in the blood in the early stages of the disease. They are more numerous in the capillaries than in the heart blood.

The lungs are in some cases edematous. In all young animals one finds edema of the lungs with blood stained mucoserous substance in the trachea and bronchioles. The lymphatic glands are rarely if ever altered in appearance.

The central nervous system presents nothing of note, except a slight congestion of the meninges.

The histological study of the lesions shows that the tissue changes start from greatly engorged capillaries. In these vessels containing masses of blood, a large part of the red corpuscles contain the parasite.

The histological examination of the organs when hardened in Müller's fluid shows, according to Graham-Smith, the capillaries of the alveoli of the lungs to be dilated and in some instances a proliferation of the lining cells some of which are seen lying free in the alveoli. In other alveoli the process is more advanced and proliferated cells, leucocytes, and in some cases red corpuscles are present in them. The lumen of many of the bronchioles frequently contains desquamated epithelium, leucocytes and mucus. There is no evidence of any increase in the connective tissue. No pigmentary or fatty changes were observed in the heart or skeletal muscles. The liver showed the most marked changes. The central vein of the lobule and the capillaries lying between the liver cells were dilated. The protoplasm of the liver cells stains feebly but the nuclei take the stain fairly well. The cells are distorted between the dilated blood vessels and in many cases almost destroyed, especially those in the central zone. The vessels in the interlobular spaces are dilated but the bile ducts are normal. There is no increase of fibrous tissue and the capsule is normal. The capillaries contain a large number of red blood corpuscles and the proportion of leucocytes is high. The latter are also very numerous in the larger vessels. In these vessels about 10% of the red corpuscles are infected. The parasites usually appear in small groups in the cells. The proportion of leucocytes to red corpuscles is about 1 to 10. In the capillaries from 23 to 53% of the red corpuscles are infected and in some instances the proportion of leucocytes to red corpuscles is as high as 1 to 3. In but one dog did he find fatty changes. The smear preparations showed that 98.9% of all infected corpuscles contained one to four parasites and the balance more than four parasites. Free parasites were found in the proportion of 1 to $2\frac{1}{2}$ infected corpuscles. The spleen pulp contains in most cases a large quantity of blood and the vessels in the trabeculæ are dilated and contain numerous leucocytes. The proportion of infected corpuscles in the pulp is small (3 to 12%) but in the small trabecular veins it is high (48%). In the kidney there were no changes that were constantly present except dilatation of the blood vessels. In some cases about 46% of the red corpuscles in the vessels of the glomeruli were infected.

Diagnosis. The positive diagnosis is made by finding the parasite in the red blood corpuscles. The symptoms are not very conclusive. Piroplasmosis is to be differentiated from distemper and from an anemia and disintegration of red corpuscles, hyperleucocytosis,

albuminuria and hemoglobinuria, said to sometimes occur from the ingestion of decomposed meat.

REFERENCES

1. GRAHAM-SMITH. Canine piroplasmosis. Morbid anatomy. *Jour. of Hygiene*, Vol. V (1905), p. 250.

2. HUTCHEON. Malignant malarial fever of the dog. *The Veterinary Journal*, Vol. XLIX (1899), p. 398.

3. MARCHOUX. Piroplasma canis (Lav.), chez les chiens du Sénégal. *Comp. R. de la Société de Biologie*, Vol. LII (1900), p. 97.

4. NOCARD ET ALMY. Une observation de piroplasmose canine. *Bulletin de la Société cent. de Méd. Vétér.*, 1901, p. 192.

5. NOCARD ET MOTAS. Contribution à l' étude de la piroplasmose canine. *Ann. de l' Inst. Pasteur*, Vol. XVI (1902), p. 257.

6. NUTTALL. Canine piroplasmosis. *Jour. of Hygiene*, Vol. IV (1904), p. 219.

7. NUTTALL AND GRAHAM-SMITH. Canine piroplasmosis. *Jour. of Hygiene*, Vol. V (1905), p. 237.

8. PHILLIPS AND McCAMPBELL. Infectious jaundice due to piroplasma commune. *Centralbl. f. Bakt.*, Bd. XLVII (1908), p. 592.

9. ROBERTSON. Malignant jaundice in the dog. *The Jour. of Comp. Path. and Therap.*, Vol. XIV (1901), p. 327.

10. WEBB. Piroplasmosis in Fox-hounds in India. *The Jour. of Comp. Path. and Therap.*, Vol. XIX (1906), p. 1.

11. WETZL. Ueber die Piroplasmose der Hunde. *Zeitschrift für Thiermed.*, Bd. X (1906), S. 369.

12. WRIGHT. Canine piroplasmosis (on certain changes in the blood). *Jour. of Hygiene*, Vol. V (1905), p. 268.

EQUINE MALARIA

Synonyms: Piroplasmosis of horses; South African horse sickness; "biliary fever."

Characterization. This affection of horses is characterized by a high temperature and a yellowish tint of the mucous membranes. The spleen is enlarged and the blood contains *Piroplasma equi*.

History. The disease appears to have been first described by Wiltshire in 1883. Guglielmi discovered the parasite of this affection in 1899, and Rickmann found it in a large number of horses that died of "horse sickness." A good description of this affection was given by Theiler in 1901.

Geographical distribution. This disease appears to be very largely restricted to Southern Africa and Europe. The disease known as malarial fever in the United States has not been demonstrated to be due to a *piroplasma*. Peters reported malaria in horses in the West. The diagnosis was made from the symptoms and lesions as he did not find the *Piroplasma*.

Etiology. Laveran who has studied this affection states that its cause is *Piroplasma equi*. It is closely related to *P. bigeminum*. It measures from 0.5 to 2.0μ. During the invasion and multiplication of the parasites there is a high temperature. In the blood corpuscle the parasite is single, in pairs or in rosette form. The disease was believed not to be transmitted directly with the blood containing the parasite. Theiler, however, succeeded in proving that equine piroplasmosis is inoculable with the blood of immune horses into susceptible ones. The natural method of infection is not known, but it is believed to be by means of a tick. Berlitzer mentions *Dermacentor reticulatus* as the accepted carrier of the Russian tick fever of horses but he also suspects the biting flies.

Theiler's conclusions relative to the transmissibility of this parasite are as follows:

The piroplasma found in the mule and the donkey is identical with *Piroplasma equi* first found in the horse.

The disease caused by this piroplasma is inoculable with blood of immune animals into susceptible ones belonging to the domesticated species of the genus *equus*.

The horse shows the greatest susceptibility for this piroplasma; the donkey is less, and the mule the least, susceptible.

The possibility of a practicable inoculation against the piroplasmosis stands in the reverse order of the susceptibility. The mule may be safely inoculated with immune blood of any of the three respective equines; the immune horse-blood produces the severest reaction, the immune mule-blood causes little reaction, and so does the immune donkey-blood.

The period of incubation is stated by Theiler to be 21 days. Marzinovski found the period of incubation from tick infection to be 12 days. Nuttall places it at 16 to 17 days in the horse.

Symptoms. An acute and chronic type are recognized. The acute type begins with a high temperature. There is jaundice, appearing first in the eyes. Death follows rapidly, often at the time of the maximum temperature. In the chronic cases the symptoms are prolonged.

The duration of the disease is from a few to several days.

Morbid anatomy. The animal is emaciated. There is a rapid destruction of the red blood corpuscles. The blood is said to be thin and watery. The conjunctival subcutaneous tissue is of a yellowish

color. The muscles are reddish brown. All of the tissues are *anemic.*
The spleen is very large. Theiler has found the spleen to be twice the
normal size. The lymphatic glands, especially those of the spleen,
liver and kidneys, are tumefied and often hemorrhagic. The liver is
yellowish in color and engorged with blood. The bile capillaries are
distended. The mucosa of the digestive tract is pale, or sprinkled
with reddish areas. The glands in the thorax are enlarged and
infiltrated with a gelatinous substance. Occasionally there are
ecchymoses on the lungs and heart. The heart muscle, pericardium
and valves are frequently infiltrated with a gelatinous substance. The
blood clots are soft. The exuded serum is of a yellowish brown color.
The parasites are found in all parts. Death usually follows an acute
attack.

Diagnosis. Equine malaria is to be diagnosed by its symptoms,
lesions and the finding of its specific organism. It is to be differen-
tiated from *Brustseuche,* nagana, and anthrax. In the last two
named diseases the finding of the specific organisms will determine the
diagnosis. The finding of the *piroplasma* will distinguish it from
Brustseuche. It may be mistaken for influenza.

REFERENCES

1. BOWHILL. Equine piroplasmosis or "biliary fever." *The Jour. of Hygiene,*
Vol. V (1905), p. 7.
2. DALE. Piroplasmosis of the donkey. *Jour. of Comp. Path. and Therap.,* Vol.
XVI (1903), p. 312.
3. GUGLIELMI. Un caso di malaria del cavallo. *Clinica Veterinaria,* 1899, p. 220.
4. LAVERAN. Contribution à l'étude de Piroplasma equi. *Comp. R. de la
Société de Biologie,* 1901, p. 385.
5. RICKMANN. Südafrikanische Pferdesterbe. *Berliner thierarztl. Wochenschrift,*
1902, S. 4.
6. THEILER. Die Pferde-malaria. *Thèse de Berne. Schweizer-Archiv für Thier-
heilkunde,* 1901, S. 253.
7. THEILER. Further notes on piroplasmosis of the horse, mule and donkey.
Jour. Comp. Path. and Therap., Vol. XVIII (1905), p. 229.

ICTERO-HEMATURIA IN SHEEP

Synonyms. Piroplasmosis of sheep; carceag; hemoglobinuria in
sheep; Babesiosis.

Characterization. This is an enzoötic disease characterized by a
rise of temperature with chill, and later icterus and marked changes
in the blood due to a specific parasite (*Piroplasma ovis*) invading its
red blood corpuscles. Goats are said to suffer from this infection.

History. Mazureano observed this disease in 1884. In 1892, Babes pointed out the constant presence of an intraglobular parasite in the blood of sheep suffering from an enzoötic hemoglobinuria in Roumania. Bonome studied the same affection in Italy in 1895. Williams described this disease in Montana in the same year but he did not report finding the parasite.

Geographical distribution. The piroplasma infection of sheep has been found in several places in Europe, especially in the flats of the Danube where it is reported 20% of its sheep may die. Pause found similar parasites in the blood of sheep in East Africa. Its existence in the United States is in question, although the description given by Williams of the disease he found suggests very strongly a *Piroplasma* origin.

Etiology. *Piroplasma ovis* is the specific cause. This organism is very closely related to *Piroplasma bigeminum* of Texas fever. It is transmitted by *Rhipicephalus bursa* which as larvæ or nymphs have sucked the blood of affected sheep.

The period of incubation is from 8 to 10 days.

Symptoms. In the beginning, the symptoms are said to be severe. There is a rise of temperature usually accompanied with a chill. After one or two days icterus appears. The urine is occasionally of a reddish brown color, due to the presence of hemoglobin. Death is preceded by a collapse in which the temperature is subnormal. The duration of the disease is usually but a few days.

Morbid anatomy. The subcutaneous tissues are infiltrated with a yellowish colored liquid. The blood is thin and watery. The muscles are pale and edematous. The mucous membranes of the pharynx and intestines are often hemorrhagic. The liver is small, soft and yellowish in color. The spleen is usually slightly enlarged. The kidneys are soft and friable. There usually occurs a parenchymatous nephritis.

Diagnosis. It is positively diagnosed by finding the piroplasma in the blood. It is to be differentiated from anthrax.

REFERENCES

1. BABES. L'étiologie d' une enzootie des moutons dénommée Carceag en Roumanie. *Comp. R. de l' Acad. des Sciences*, Vol. CXV (1892), p. 359.

2. BONOME. Uber parasitäre Ictero-Hämaturie der Schafe. Beitrag zum Studium der Amoebo-Sporidien. *Archiv. für path. Anatomie*, Bd. CXXXIX (1895), S. 1.

3. WILLIAMS. The parasitic Ictero-Hæmaturia of Sheep. *Bulletin No. 8, Mon. Agric. Exp. Station, 1895*, also *Amer. Vet. Review*, Vol. XXI (1897), p. 377.

EAST AFRICAN COAST FEVER

Synonyms: East Coast fever; Rhodesian red water; Rhodesian tick fever.

Characterization. This is a disease of cattle characterized by the presence of a piroplasma. It is distinguished from the other piroplasmoses of cattle in that it cannot be transmitted from one animal to another by the direct inoculation of the blood.

History. This disease was differentiated from the other piroplasmic diseases by the investigations of Theiler in the Transvaal and Koch and Buluwayo. In 1905, it is stated that 500 farms in the Transvaal were infected and that within a year 50,000 cattle had died of it.

Geographical distribution. This disease seems to be restricted to the Eastern coast of Africa. In 1900 it was introduced into the Portuguese territory from whence it spread into the interior of Africa, especially to the German East Africa, Natal, the Transvaal and Rhodesia.

Etiology. This disease is caused by *Piroplasma* (Theileria)* *parvum.*

It is transmitted by the ticks (*R. appendiculatus* and *R. simus* and possibly other species of the same genus. In the blood of infected cattle three forms of the blood parasite are found. In the acute form of the disease the parasites assume a ring or bacillary form and not infrequently the one may be seen to change into the other; they exhibit amœboid movement, and a small mass of chromatin can be made out. In the chronic form of the disease the parasite appears as a nonmotile punctiform mass of chromatin. It is stated that the parasites are very abundant in the blood. In the acute form as many as 90% of the red blood corpuscles are affected. It is also stated that *P. mutans* is often present in the blood of animals suffering from this infection. As already stated, the disease cannot be produced artificially by the inoculation of the blood containing the parasite. Meyer obtained positive results by introducing large pieces of spleen

---

*França points out that Bettencourt and Borges proposed that the genus Piroplasma should include only those organisms which at one stage are pear shaped—twin parasites appearing in the single corpuscle and which multiply by budding as described by Nuttall. The characteristic features of the two genera suggested are as follows:

Genus Theileria Bettencourt, França and Borges, 1907. Rod-like parasites which when dividing give rise to cross-forms. The individual elements of the cross-forms are very small, rounded, and composed, practically, exclusively of chromatin.

Genus Nuttallia, França, 1909. Parasites oval or pyriform, No rod-like bodies. Cross multiplication, the protoplasm of these forms being relatively abundant.

from infected animals into the abdominal cavity. Twelve days after this inoculation the parasites appeared in the blood and simultaneously there was a rise in temperature. Stockman produced the disease in cattle in England with the nymphs of the brown ticks, sent there from Africa. The infection always takes place while the cattle are at pasture. It seems to be more prevalent after the rainy season where the grass is high as the ticks, which are present in large numbers, easily get upon the cattle. Healthy cattle become infected by being pastured with animals from infected localities or even by driving them over infected pastures.

A few writers consider the possibility of this disease being due to an ultra-microscopic organism. They consider *P. mutans* and *P. parvum* to be identical. They point out that animals which have recovered from East Coast fever are no longer infectious for ticks.

The period of incubation is according to Theiler from 10 to 12 days after exposure to infected pastures.

Symptoms. The symptoms are a high fever, difficult respiration, salivation, passing of dry or bloody tar-like feces, swelling of the sub-maxillary lymph glands, emaciation and weakness, especially of the hind quarters. The appetite remains normal for some days. Anemia, icterus and hemoglobinuria are rare. According to Theiler, there may be an acute, rapidly fatal form and a chronic form, characterized by transitory fever and jaundice. At the height of the fever, the parasites are present in a large percentage of the red blood corpuscles but destruction of the corpuscles is slight. Cattle that have recovered from Texas fever or *P. bigeminum* infection, if exposed to East Coast fever, develop that disease and in the blood of the animal the parasites of both diseases are found.

The duration is not long. Most of the infected animals die.

Morbid anatomy. The lesions are punctiform hemorrhages in the subcutaneous and subserous connective tissue, enlargement of the lymph glands and edema of the lungs. There is congestion or ulcerative inflammation of the intestinal mucous membrane and true stomach. The spleen is not enlarged. Peculiar wedge-shaped infarcts are described in the cortex of the kidney. Koch has described specific bodies, so-called plasma spheres, in the swollen lymph glands, in the spleen and in the diseased foci of the liver and in the infarcts of the kidneys as well as in the borders of the ulcers of the mucous membrane and also in the blood. When stained after Giemsa they appear

as sharply circumscribed blue colored spheres containing point or comma-shaped chromatin granules or they are covered by such.

Diagnosis. This disease is to be diagnosed by its symptoms, lesions and finding its specific organism. It is to be differentiated from the other piroplasmoses, especially those caused by *P. mutans* and *P. bigeminum*.

Lichtenfeld describes a pseudo-coast fever, a disease in African cattle due to *P. mutans*.

Prevention. The disease can be kept out of uninfected territory by eliminating the tick by means of dipping or spraying. The infected pastures are rendered safe in eighteen months, during which time the cattle are to be kept entirely off. Horses, goats or sheep may pasture upon it.

REFERENCES

1. França. Quelques considérations sur le genre Theileria et description d' un nouvelle espèce de ce genre (Theileria stordii). *Centralbl. f. Bakt.*, Bd. LXVII (1913), p. 17.

2. Gonder. The life cycle of Theileria parva: The cause of East Coast fever in cattle in South Africa. *Journ. of Comp. Path. and Therap.*, Vol. XXIII (1910), p. 328.

RED WATER (BRITISH)

Synonyms. Bovine piroplasmosis; black water; red murrain; May disease; moor-ill; bovine hemoglobinuria.

Characterization. Red Water (British) is an enzoötic hemoglobinuria of cattle due to *Piroplasma bovis* (Craig). It affects cattle on the British Isles.

Geographical distribution. This disease is described from Southwest England, and many parts of Scotland and in Ireland. It is said to be most common in low-lying, rough pastures such as moors (hence the local name, moor-ill) and on lands in the neighborhood of woods. If the farms on which the disease exists are subjected to tillage, the trouble disappears.

Etiology. According to Craig, this disease is due to *Piroplasma bovis* of which he distinguishes two forms according to their size, the large form being identical with *P. bigeminum*, the small form occupying only a very small portion of the red corpuscle. This is by far the more common in Ireland. It is transmitted by inoculation and the tick. The ticks are *Ixodes ricinus* the common species, and *Haemophysalis punctata*, found in the south of England. M'Fadyean and

Stockman have described a new piroplasma which they obtained from a bull inoculated with blood taken from a case of clinical red water and which they designate *Piroplasma divergens*. They were able to transmit this parasite direct by inoculation.

The period of incubation, after inoculation, is said to vary from 4 to 12 days. When cattle are placed in infected pastures the symptoms appear in about two weeks, when the exposure is made in the spring and autumn. Sometimes the symptoms do not appear for months.

Symptoms. The symptoms are said to resemble somewhat closely those of Texas fever. The disease occurs chiefly in animals at pasture. Young calves appear to be more resistant. It rarely attacks animals under three months of age.

The duration of the disease varies. In acute cases death may occur in from 3 to 4 days. In the more chronic forms it is protracted for one or two weeks. If recovery sets in the bowels act freely, the urine clears up and the animal begins to eat and ruminate. The parasites decrease rapidly in number. It is not a very fatal disease.

Morbid anatomy. The post mortem appearances are not unlike those found in Texas fever. It is diagnosed by finding the specific organism. It is prevented by keeping the animals away from tick infested pastures.

Inoculation against red water. In Germany, Kossel, Schütz, Weber and Miessner recommended for the inoculation against piroplasmosis, the employment of sterile, defibrinated blood from calves that had recovered from the disease at least 50 days and the protective inoculation should be carried out from four to six weeks before the animals are turned out to pasture.

"The directions issued with the inoculation material (which is prepared at the pathological institute of the veterinary school in Berlin) direct that the process should be carried out by veterinary surgeons, and previous to the animals being sent to grass, that is, about the month of March. Cows in an advanced stage of pregnancy or diseased animals should not be injected. The defibrinated blood constituting the inoculation material retains its properties for eight days, and should be kept in a cool, dark place. Before use it should be cautiously shaken; bottles when once opened should be used up on the same day. In carrying out the operation an area on the side of the neck about the size of a cheese plate should be shaven, cleansed, and dried. All syringes must be boiled before each injection. Disin-

fecting materials should not be brought in contact with the syringes
or with the material."

REFERENCES

1. CRAIG. British Redwater. *Hoare's System of Veterinary Medicine*, Vol. I
(1913), p. 1005.

2. M'FADYEAN AND STOCKMAN. A new species of piroplasm found in the blood of
British cattle. *Journ. of Comp. Path. and Therap.*, Vol. XXIV (1911), p. 340.

3. STOCKMAN. The treatment of redwater in cattle (bovine piroplasmosis) with
trypanblue. *Journ. Comp. Path. and Therap.*, Vol. XXII (1909), p. 321.

GALL SICKNESS OF CATTLE

Characterization. Gall sickness of cattle is a name applied to a
variety of diseases in South Africa, characterized by anemia and
icterus and the finding on post mortem of an abnormal liver or gall
bladder. According to Theiler and Hutcheon, it is a name given by
the natives to almost any obscure complaint affecting the digestive
tract. A number of organisms have been found associated with it.
Theiler was of the opinion that this infection was due to *P. mutans.*
Still more recently he associated it with anaplasma. The disease
named anaplasmosis by Theiler attacks cattle exclusively, especially
when they are brought from high lands into lowlands or from less
infected localities into more infected districts. The parasite may be
transmitted to the healthy with the blood of the infected and recovered
animals. Mixed infections seem to be very numerous in that terri-
tory.

Theiler found that the incubation period after a pure *P. mutans*
infection varies between 13 and 42 days. The blood lesions consist
in anisocytosis, polychromasia, metachromasia, basophile granula-
tions and microcytes. In his experiments on 25 animals there were
no fatal cases.

Dodd found, in his investigations in Queensland, Australia, at
least two distinct diseases caused by piroplasmata. One is due to
P. bigeminum and is known as tick fever, red water or Texas fever.
The other is due to small parasites whose prevailing form is that of a
rod. There is as yet no popular name for this latter disease.

REFERENCES

1. THEILER. Further notes on *Piroplasma mutans*. *Journ. of Comp. Path. and
Therap.*, Vol. XXII (1909), p. 115.

2. THEILER. Gall sickness of South Africa. (Anaplasmosis of cattle). *Journ.
of Comp. Path. and Therap.*, Vol. XXIII (1910), p. 98.

CHAPTER XI

DISEASES CAUSED BY PROTOZOA
GENUS TRYPANOSOMA

Classification of trypanosoma. The trypanosomata belong to the protozoa, but their species diagnosis is not satisfactorily determined. A number of classifications of these organisms have been proposed. The one suggested by Salmon and Stiles is appended.

Protozoa, class *Mastigophora*, subclass *Flagellata*, order *Monadida*, family *Trypanosomidae*, genus *Trypanosoma* Gruby.

According to Stiles the family contains at present two genera, which are distinguished as follows:

1. One flagellum present, extending from the centrosome along the undulating
    membrane and becoming free at the anterior extremity..........*Trypanosoma*
2. Two flagella, one extending anteriorly, the other posteriorly....*Trypanoplasma*
    Generic diagnosis of Trypanosoma. "Body fusiform, presenting a lateral, longitu-
    dinal, undulating membrane, the thickened border of which terminates posteriorly, in
    the posterior half of the body in a 'centrosome,' and is prolonged anteriorly in a free
    major flagellum; nucleus generally anterior; there is a tendency to agglomeration by
    the posterior extremity; divisions longitudinal and unequal. Parasitic in the blood of
    vertebrates."

In order that a somewhat definite idea of the structure of these organisms may be obtained the specific characters of Tr. Lewisi are quoted:

Trypanosoma: "8 to 10u long, 2 to 3µ broad, 24 to 34µ long by 1 to 44µ broad(Laveran
and Mesnil, 1901); a very refringent granule (near centrosome) in place of which a
clear vacuole is seen in stained preparations. Animalcules exceedingly minute, alter-
nate and vermicular under normal conditions, but highly polymorphic and capable of
assuming a variety of contours; flagellum single, terminal, two or three times the length
of the extended body. No contractible vesicle . . . as yet detected. Habitat,
blood of the rat and hamster."

Schaudinn distinguished several genera* of the Trypanosomidæ and his classification has been adopted by many writers.

*The genus *Trypanosoma:* having an undulating membrane and one flagellum.
   The genus *Trypanoplasma:* having in addition to an undulating membrane and
an anterior flagellum, a second free flagellum inserted into the posterior part of the body.
   The genus *Herpetomonas:* having a free flagellum but no undulating membrane.
   The genus *Spirochæta:* elongated, sinuous organisms having an undulating mem-
brane, but no flagellum.
   The genus *Treponema:* elongated, sinuous organisms having a short flagellum at
each end but no undulatory membrane.

General morphology of trypanosoma. Trypanosomata of all species are in general very similar. The morphology is said to vary greatly in the same species and to a greater extent in different species. In general the trypanosoma may be said to measure from 1 to 5μ in thickness and from 15 to 45μ in length, including flagellum. They all show very active eel-like movements and some motility. The nature and extent of the motility varies. The fact as stated, that variations are occasionally found in one species, often, indeed, in a single preparation, which are nearly as great as those observed between different species, renders the specific determination difficult. The flagellum at the anterior end of the parasite varies greatly in length. It is actively motile, pointed and continuous with the thickened margin of the undulating membrane ending at or near the centrosome. The undulating membrane extends along the border of the organism from near the centrosome in the posterior portion to the anterior end of the organism, from whence it continues as the free flagellum. Its breadth and folds vary considerably.

The nucleus is usually situated in the anterior half of the parasite and varies both in size and shape. It is generally oval or round, and assumes other contours with the different stages of division. The centrosome as a rule is in the posterior and more blunt end, and it appears to have an intimate association with the flagellum and undulating membrane. Its location has been used as a diagnostic point in determining species.

FIG. 84. TRYPAN-OSOMA BRUCEI: c, CENTROSOME; f, FLAGELLUM; m, UNDULATING MEMBRANE; n, NUCLEUS. × ABOUT 2,000. (After Laveran and Mesnil).
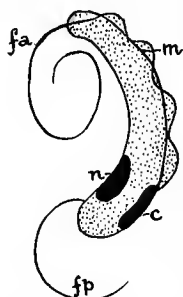
FIG. 83. TRYPAN-OPLASMA BORRELLI: c, CENTROSOME; fa, ANTERIOR FLAGELLUM; fp, POSTERIOR FLAGELLUM; m, UNDULATING MEMBRANE; n, NUCLEUS; × ABOUT 1,800. (After Laveran and Mesnil).

The protoplasm is homogeneous or granular, depending upon the age of the parasite, its environment and, perhaps to a certain degree, upon the species. Few or many fine or coarser granules may be found scattered throughout the protoplasm.

Multiplication. Voges gives three forms of multiplication, namely, longitudinal, transverse fission, and segmentation. He did not

observe conjugation. The chromatin divides into from 3 to 10 seg-
ments, which assume irregular shapes and locations, some of which are
often found well up in the flagellum. The nucleus usually divides
into equal parts, but may break into several segments. After the
nuclear division the protoplasm may assume various irregular forms.
The young nuclei arrange themselves in groups, and the parasite
twists and splits by longitudinal or more often by transverse
fission. The new forms resulting from the division soon assume
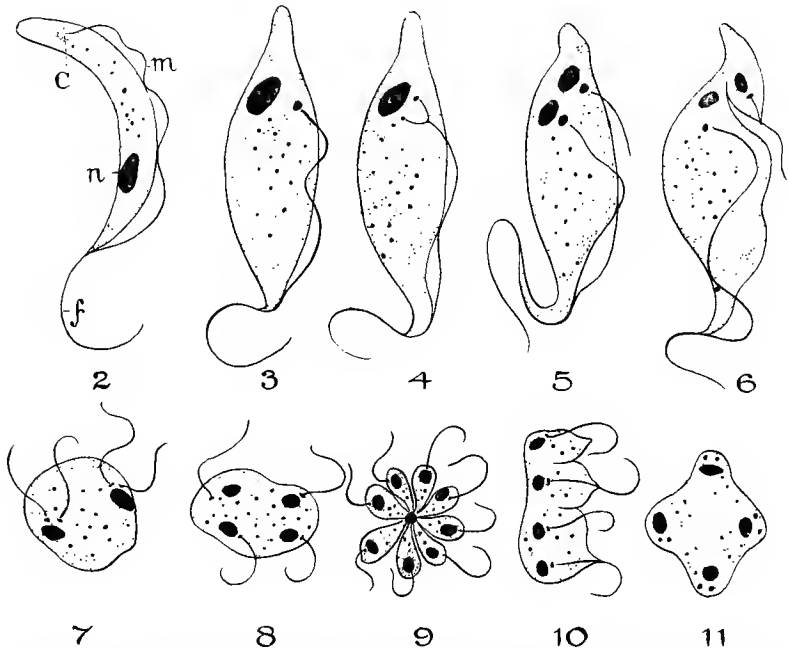
FIG. 85. *2*, TRYPANOSOMA LEWISI COMPLETELY DEVELOPED; *n*, NUCLEUS: *c*, CENTRO-
SOME; *m*, UNDULATING MEMBRANE; *f*, FLAGELLUM. 3–6, TRYPANOSOMA IN PROCESS
OF DIVISION. 7–10, OTHER FORMS OF MULTIPLICATION OF TR. LEWISI, 11, FLAGELLA
NOT STAINED. × ABOUT 2,000 DIAMETERS (*after Laveran and Mesnil*).

the regular shape. Plimmer and Bradford consider longitudinal and
transverse division the more frequent modes of reproduction. They
observed conjugation, which consisted in the fusion of the micronuclei,
followed by an amœboid stage and division by segmentation. The
order of division appears to be (1) centrosome, (2) flagellum, and (3)
nucleus and protoplasm. Other forms of reproduction have been
described by Martini, Laveran and Mesnil and others. Involution

forms have been observed by a number of workers. Rodet and Vallet state that *Tr. brucei* multiply principally in the blood and lymph. There is a difference of opinion concerning the agglutination of trypanosomata. Musgrave and Clegg state as a result of their researches that the so-called phenomenon of agglutination is of no value from a diagnostic point of view, and it is too uncertain, if it is a reaction, to serve as an index of immunity or susceptibility.

Distribution in the body. It is the opinion of most students of the trypanosomiases that in the infected animal the parasites are found in all of the body juices, and are not present at the same time in great numbers in one part, with but few in another. Animals having many parasites in the blood when killed show them also in the organs; and if they are not demonstrable in a microscopic examination of the one they will not appear in the other. The blood of animals suffering from the disease is nearly always infectious by animal inoculation, although the parasites may not be found microscopically at the time. Martini, however, regards the spleen, lymphatics, bone marrow, and to a less extent, the liver and kidneys, as the places for the destruction of trypanosoma. It has been found that trypanosomata injected into the peritoneal cavity multiply considerably before they enter the blood.

Disappearance after death. Trypanosomata disappear very suddenly after the death of the host. Within two hours signs of degeneration begin; the parasites shrink, assume irregular shapes and then disappear. Motile forms are rarely found after two hours.

Distribution in nature. Trypanosomata are not known to exist in nature outside of the bodies of living animals. They have been found in the blood of a number of species of mammals, birds, fish and amphibians, where for the most part they seem to lead a harmless life. A few species, however, are pathogenic for animals and man. They have been kept alive in blood or salt solution for a few hours. Novy has succeeded in cultivating them, *i. e.*, getting them to multiply in an artificial culture medium.

Historical sketch. There is a voluminous literature on the trypanosoma from which the following brief summary was taken. In 1841, Valentin discovered hematozoa in trout (*Salmo fario*) and in 1842, Glugge found them in the blood of frogs. In 1843, Gruby observed a flagellate infusorium in frogs which he named *Tr. sanguinis*. Gruby has generally been credited with the discovery of these forms. From

1843 to 1879 the organisms were found by many observers, not only in frogs but in birds as well.

In 1879–80, Lewis described trypanosoma found in rats in India. Later he states that they are identical with *Tr. evansi*.

In 1880, G. Evans discovered trypanosoma in the blood of horses suffering with surra, the well known disease of India. He proved their causal relation to the affection. In 1885, Steele confirmed Evans' work, and named the parasite *Spirocheta Evansi*.

The work of Evans and Steele was followed by many interesting discoveries of trypanosoma especially in fish and in man.
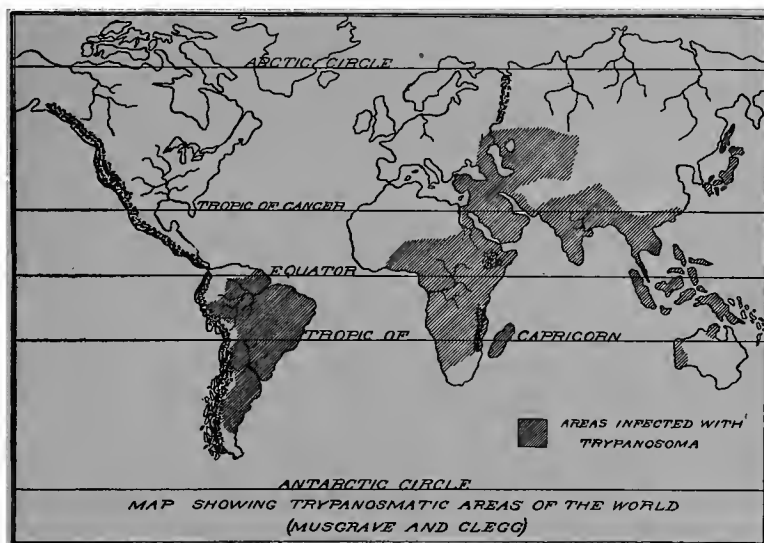
Fig. 86. A MAP SHOWING THE GEOGRAPHICAL DISTRIBUTION OF TRYPANOSOMA DISEASE.

In 1896, Rouget described *Trypanosoma* found in the blood of a horse suffering from dourine. Wasilewsky and Senn, in 1899, confirmed Rouget's work and determined the pathogenic action of this parasite for the horse. Laveran and Mesnil proposed the name *Tr. rougeti* for the parasite of dourine. Doflein (July, 1901) named it *Tr. equiperdum*, which term was adopted by Salmon and Stiles.

In 1901, according to Voges, Elmassian first differentiated the trypanosoma of *Mal de caderas* in South America. Voges described it the following year, demonstrated its pathogenic action, and named it *Tr. equinum*.

In 1902, Bruce and Laveran independently published articles in which they credit Theiler with the discovery of a new trypanosoma of cattle in South Africa. They each proposed the name *Tr. theileri*.

In 1901, Smith and Kinyoun described a parasite which had been observed by Jobling in the blood of a sick horse in Manila. Later in the year Smith described it as *Tr. Evansi*.

Trypanosomiasis. Salmon and Stiles introduced the term trypanosomiasis to describe an infection with parasites belonging to the flagellate family *Trypanosomidæ*. The term is analogous to *teniasis* and *coccidiosis*. There are a number of different trypanoso-miases now recognized, being caused by different species of trypano-soma. Among these the following may be mentioned:

Surra. A disease of equines, camels, elephants and certain other animals in India, caused by *Tr. evansi*. Rats, mice, dogs, monkeys and cattle are susceptible to inoculations with the blood of animals suffer-ing with surra. It occurs naturally in the horsekind, cattle and camels.

Nagana, nygana or *tsétsé-fly disease* of Africa. A disease affecting cattle, horses, mules, and according to Laveran and Mesnil, with few exceptions, all mammals are susceptible. It is caused by *Trypano-soma brucei*.

Dourine or *mal du coit* of Algiers, France and Spain. It attacks the horse and the ass in particular, but may be transmitted to the dog, rabbit and in certain cases to the rat and mouse by inoculation. It is caused by *Trypanosoma equiperdum*.

Mal de caderas of South America. This is a serious disease of horses, asses, cattle, hogs and certain other animals. It is caused by *T. equinum*.

Rat trypanosomiasis caused by *T. lewisi*. By some authors this parasite is alleged to be identical with the horse surra organism, but it is quite certain that rats may harbor a distinct species. Until the results of further investigations are recorded it is deemed best to consider these as distinct infections. Musgrave and Clegg conclude that proof sufficient to establish the individuality of the *Trypanosoma* causing trypanosomiases in domestic animals has not yet been ad-vanced. These authors consider the trypanosoma found in domesti-cated animals in the Philippine Islands as *Tr. evansi*.

It is important to note the observation of Musgrave and Clegg that "in all the forms of trypanosomiasis the infection seems to involve particularly the genitalia, the skin, and the organs of special sense.

The skin symptoms consist of roughening of the hair, which also falls out in places; a thickening of the epidermis, often with exfoliation, and in some stages of the disease, various skin eruptions. There may be simple erythema, and more rarely they may assume the severer forms, as urticaria, or in extreme cases a distinct localized ulceration may occur. The scrotum and penis in the male and the vulva in the female are often swollen, and ulcerations of the penis or vulva are frequent symptoms especially in dourine."

The geographical distribution of the trypanosomiases is shown in Figure 88.

REFERENCES

1. ARCHIBALD. A trypanosome of cattle in the Southern Sudan. *Journ. of Comp. Path. and Therap.*, Vol. XXV (1912), p. 292.

2. BEVAN AND MILLINGTON. Notes on a strain of human trypanosomiasis and a review of the present knowledge of the human trypanosomiasis of Northern Rhodesia and Nyasaland. *Journ. of Comp. Path. and Therap.*, Vol. XXV (1912), p. 298.

3. CRAWLEY. Trypanosoma Americanum new species: A trypanosome which appears in cultures made from the blood of American cattle. (Preliminary notice). *Journ. of Comp. Path. and Therap.*, Vol. XXIII (1910), p. 17.

4. DARLING. The infection of mules by *Trypanosoma hippicum* through mucous membranes. *Journ. of Exp. Med.*, Vol. XV (1912), p. 367. Ref. *Journ. of Comp. Path. and Therap.*, Vol. XXV (1912), p. 164.

5. DURRANT AND HOLMES. A trypanosoma found in blood of cattle in India. *Journ. of Comp. Path. and Therap.*, Vol. XVII (1904), p. 209.

6. DUTTON AND TODD. First report of the Trypanosomiasis Expedition to Senegambia (1902). *Thomson Yates and Johnson Laboratories Report*, Vol. V.

7. FRANK. Uber den Befund von Trypanosomen bei einem in Stein-Wingert Westerwald, Regierungsbezirk Wiesbaden verendeten Rinde. *Zeitsch. f. Infektionskr. parasit. Krankh. u. Hyg. d. Haust.*, Bd. V (1909), p. 313. Ref. *Deutsch. Tierärtz. Wochensch.*, Vol. XVII (1909), p. 565.

8. GIBSON. Two cases of trypanosomiasis. *Jour. of Comp. Path. and Therap.*, Vol. XVIII (1905), p. 79.

9. HART. Transmission of trypanosomiasis in Northeastern Rhodesia. *Journ. of Comp. Path. and Therap.*, Vol. XXIV (1911), p. 354.

10. HEANLEY. A note on the occurrence of a large trypanosome in the blood of native cattle in South China. *Journ. of Comp. Path. and Therap.*, Vol. XXI (1908), p. 178.

11. HOLMES. A reply to Capt. Martin on trypanosomiasis. *Jour. of Comp. Path. and Therap.*, Vol. XVIII (1905), p. 223.

12. JOWETT. Further Note on a cattle trypanosomiasis of Portuguese East Africa. *Journ. of Comp. Path. and Therap.*, Vol. XXIV (1911), p. 21.

13. LAVERAN AND MESNIL. Recherches morphologiques et expérimentales sur le Trypanosome des rats (*Tr. Lewisi*, Kent). *Ann. de l' Inst. Pasteur*, Vol. XV (1901), p. 673.

14. LAVERAN ET MESNIL. *Trypanosomes et Trypanosomiases*, Paris, 1904.

15. MARTIN. Trypanosomiasis in cattle of India. *Jour. of Comp. Path. and Therap.*, Vol. XVIII (1905), p. 144.

16. MASON. Equine trypanosomiasis in Egypt. *Journ. of Comp. Path. and Therap.*, Vol. XXV (1912), p. 93.

17. MASON.   Note on the camel trypanosomiasis of Egypt, and results of first series of experimental drug treatment.   *Journ. of Comp. Path. and Therap.*, Vol. XXIV (1911), p. 47.

18. NOVY AND MACNEAL. On the Cultivation of Tr. Brucei. *The Jour. of Infectious Diseases*, Vol. 1 (1904), p. 1.

19. PETRIE. Observations relating to the structure and geographical distribution of certain trypanosomes. *Jour. of Hygiene*, Vol. V (1905), p. 191.

20. RODET ET VALLET. Contribution à l' étude des Trypanosomiases. *Arch. de Méd. Expér.*, Vol. XVIII (1906), p. 450.

21. SMEDLEY. The Cultivation of Trypanosomata. *The Jour. of Hygiene*, Vol. V (1905), p. 24.

22. STOCKMAN. Preliminary note on a trypanosome of British Cattle. *Journ. of Comp. Path. and Therap.*, Vol. XXIII (1910), p. 189.

23. THIROUX. Recherches morphologiques et expérimentales sur Trypanosoma Paddæ. *Ann. de l' Inst. Pasteur*, Vol. XIX (1905), p. 65.

24. TODD. Note on immunity in cattle trypanosomiasis. *Jour. of Comp. Path. and Therap.*, Vol. XXIII (1910), p. 276.

DOURINE

Synonyms.   Venereal disease of solipeds; equine syphilis; chancerous epizoötic; breeding paralysis; epizoötic paraplegia; *mal du coit*; *Beschälkrankheit.*

Characterization.   A contagious affection of solipeds, transmitted by copulation and attended by specific lesions of the generative organs and nervous system, such as local venereal swellings, chancerous ulcers and cicatrices, dementia and paralysis.

The disease is essentially an equine one, although the following species are susceptible to experimental inoculation, namely: dogs, rabbits, rats, mice and asses.   While the horse shows the greatest susceptibility, the ass is comparatively resistant to the infection.

History.   Dourine seems to have first been recognized in Algeria. It was described in 1796 by Ammon who found it in the royal stud at Trakehnen in Northern Prussia.   It was found in Bomberg in 1817 to 1820, in Austria and Bohemia in 1821–8, in Syria in 1821, in Switzerland in 1830, in France in 1830–32, in Siberia in 1833–40, in Italy in 1836, in Russia in 1843, in Poland in 1830–40, in Algiers in 1847–55. In Syria and Asia generally it is reported to appear perennially.   It would seem that the original home of this disease was Asia and Northern Africa.

It was found in De Witt County, Ill., by Williams.   It was traced to a percheron stallion imported from France* in 1882.   In this

---

*This animal had a brand mark under the mane resembling very much the letter D. Mohler states that it is supposed the disease had been detected and that he had been branded because of it before leaving France.

locality, it spread to a considerable number of breeding mares and stallions. The disease was very largely stamped out of that region by a rigid quarantine of diseased and exposed animals. Some exposed animals had, however, been taken from the district. In 1892, it appeared among the breeding horses in northwestern Nebraska. Five years later it reappeared in the same locality. In 1901, it occurred in South Dakota and Nebraska. In 1903, it was reported in Iowa and again in 1911. It was discovered in Canada by Burnett in 1904 and the trypanosome was demonstrated by Watson in 1907. In 1912 it was found in Montana.

Etiology. Rouget, in 1896, described a *Trypanosoma* found in the blood of a horse suffering from dourine, and for over two years continued the study of this organism in susceptible animals. In 1899 Schneider and Buffard demonstrated its causal relation to the disease. Wasileswky and Senn also confirmed Rouget's work and determined the pathogenic action of this *Trypanosoma* for the horse, passing it through other animals and back to the horse, reproducing the disease. Laveran and Mesnil (1901) proposed the name *Tr. rougeti* for the parasite of dourine but Doflein had previously named it *Tr. equiperdum.*

FIG. 87. TRYPANOSOMA OF DOURINE IN THE PROCESS OF DIVISION (*after Lignières*).

In its morphology and evolutionary forms, the trypanosoma of dourine has not been shown to differ from that of surra by any striking character. The granule form, the spherical, the club shaped or pyriform bodies, the fusiform with more or less stellate groupings seem to be generic characteristics. Baldrey states that it is smaller than the trypanosoma of surra. The specific distinction is found in the pathogenesis as shown by the two diseases, surra and dourine.

In the active stages, the parasite is usually found abundantly in edematous fluid, the blood, semen, milk, vaginal secretions and the erosions of the vaginal mucosa and penis. During intermissions, however, and in the absence of local lesions, the parasites are not found in the blood on microscopic examination, yet the inoculation of the blood into a dog will usually produce the disease. The parasite disappears from the blood and tissues very rapidly after death.

Schneider and Buffard, Nocard and others found the trypanosoma in the blood and exudates of horses, asses and dogs suffering from dourine but failed to find them in the same localities in animals of the same species which were free from dourine. The infected blood preserved for 24 hours in sealed glass tubes, and then inoculated into dogs produced characteristic symptoms and lesions with many trypanosomes in the blood. Inoculation into two other dogs, with the same material, but at the end of 48 hours, produced a slight transient hyperemia only, without local lesions or propagation of the parasite in the blood. The blood from the same animal inoculated after fifteen days gave negative results.

Baldrey found Romanowsky's and Wright's modifications of Leishman's method the best methods for staining the trypanosoma; the latter is very useful and handy, as no mixing of solutions is necessary and no fixing required.

Romanowsky's Stains:

STOCK SOLUTION NO. 1

Höchst's medicinal methylene blue .... 1 part
Sodium carbonate pure .... 0.5 parts
Distilled water .... 100 parts

Place this solution in an incubator at 37° C. for two or three days, when a purple color will be noticed at the edges of the liquid; this depends upon the formation of a new red color—methylene red—which combined with eosin forms the active principle of the stain and has a particular affinity for chromatin. Unless this polychromatophylic change takes place the solution is useless.

STOCK SOLUTION NO. 2

Eosin .... 1 part
Water .... 1000 parts

For staining, the stock solutions are separately diluted with water, 5 parts of stock solution to 100 parts of water.

In order to obtain a smear, prick the center of the plaque and take a drop of blood on a slide, which should be chemically clean, having been taken from an alcohol bottle and dried with a clean piece of art muslin. Then, either in the ordinary way with a piece of cigarette paper, make a thin even smear over the slide, or, as an easier and equally efficient method, take a perfectly clean flat large-sized needle and place it edgeways on the drop, when the capillary attraction of the needle will cause the blood to stream right across the slide; then evenly and gently draw the needle down the length of the slide, and a very even smear may be obtained. Quickly dry the smear in the air by waving rapidly about, which prevents the red corpuscles from crenating; the slide can then be kept indefinitely or used at once. The advantages of using a slide instead of a cover-glass are that you get a much larger field on which to work, it is much more easily manipulated and it can be kept without any mounting. Place the film in absolute alcohol or alcohol and ether, for fifteen minutes to half an hour to fix.

This coagulates the albumen and makes a permanent film in which the corpuscles and organisms are retained. Remove from the alcohol, wash in water, and then apply the stain. This is made by mixing equal parts of the above two solutions, freshly prepared, in a small glass measure or porcelain dish. It is important that the admixture should be as fresh as possible. Apply the stain to the whole of the film and let it remain for seven to ten minutes; wash in water and dry in the air, no heat being applied. The red corpuscles may have a bluish tinge, which may be removed by further washing. If the blood platelets appear bluish the film requires further staining; they should appear as ruby-red granular bodies. By continuous application of water the stain may be washed out, but the film may always be stained over again.

By this stain the protoplasm of the trypanosome is stained blue, the nuclear chromatin a carmine violet, and the flagellum and centrosome a brilliant red. The red corpuscles will be a pinkish color, and the various forms of leucocytes will be well differentiated. In examining the smear, time may be saved by looking along the edge of the film, as it is here that the parasites will be most numerous if they are present, as they, being like the leucocytes of less density than the rest of the blood, tend to run to the periphery when the smear is made.

A film made in this way requires no cover-glass, but if the cedar oil is left on it tends to withdraw the color; however, if it be carefully blotted and wiped off with a soft rag after use the film may be kept indefinitely.

The period of incubation in naturally infected animals is placed by different authors at from 2 to 60 days. Cases are reported, however, where symptoms did not appear for 12 months.

Symptoms. The first symptom in the stallion consists in the swelling of the glans penis. Reddish spots, vesicles and ulcers on the outer surface of the organ. The meatus urinarius is reddened and swollen with a mucous discharge. Later small yellowish-red nodules develop singly or in groups on the mucosa at the opening of the urethra or on the glans itself, which may ulcerate and, healing, leave whitish areas. The animal has a continuous desire to micturate and frequently manifests sexual excitement. The swelling also spreads from the penis to the sheath and scrotum in which case the testicles become inflamed. Finally, the inguinal glands and lymph vessels become involved. These local affections may, as the disease advances, almost entirely disappear. In some cases the external changes are absent, as the mucous membrane of the urethra is first affected, the only visible symptoms being strangury and a mucous discharge from the urethra.

In mares, the disease begins with a doughy or tense swelling of the pudendum, which frequently spreads to the udder and inner surface of the thighs. The vulva may be sprinkled with non-pigmented spots. The mucous membrane of the vagina is red in spots and

swollen, sometimes thickened by gelatinous elevations and covered with a turbid and orange colored secretion. The local manifestations are sometimes insignificant. The mucous membrane in the neighborhood of the clitoris is more congested than at other points and the clitoris itself may be swollen and erect. It is devoid of pigment and unnaturally dry. Williams states that this depigmentation is "peculiar and characteristic." The spots of discoloration are not caused by previous ulcers. There is excessive sexual excitement. Frequently mares suffer from strangury and, after considerable straining, urine is discharged in small jets. In his report on the Illinois outbreak, Williams states that in mares, "The open vulva and enlarged, protruding, unnaturally dry clitoris, especially in young and otherwise healthy mares is quite pathognomonic."

Instead of urine there may be discharged small quantities of sticky, discolored mucus. The mucous follicles are swollen and appear as nodules. These may develop into small ulcers with sharp borders and yellowish bases. There may be vesicles on the vagina and vulva which leave ulcers. These usually heal leaving whitish spots. The animals shake their tails and open and close the vagina in rapid succession, showing the clitoris as mares do in œstrum. The discharge often exerts a corrosive action on the tail and legs. In severe cases the neighboring lymph glands become inflamed and swollen as well as the udder, in which abscesses may appear. The swelling may even extend to the hypogastrium. There are many cases in which the early symptoms are slight and easily overlooked but the later ones are pronounced.

The general symptoms follow the local ones or develop after weeks or even months; their appearance is often delayed until the local symptoms have entirely disappeared. At first the animals are depressed and weak, they frequently continue to lift up their hind feet, alternately, so as to try to avoid putting weight upon them, knuckle on their fetlock joints and lose control over the movements of their hind legs while walking. The temperature is not so high as in other forms of trypanosma infection.

The second group of symptoms suggests affection in part of the skin, peripheral nerves and intervertebral ganglia. The animals suffer from an urticaria in the form of sharply defined, round, flat eminences which may be raised the breadth of a finger above the surface and which may vary in size from two to four centimeters or more in diameter. These have been referred to as "dollar spots."

They are caused by a serous infiltration of the papillary layer of the skin in the neighborhood of a small artery and are evidently of a baso-neurotic character. They often appear and disappear very rapidly and may shift their position. Usually they persist for several days during which time they become moderately hard and then slowly disappear. Their favorite sites are the croup, neck, shoulders, chest and abdomen.

Later in the course of the disease, a progressive paralysis of the hind quarters combines with excessive emaciation. The paralysis may last to the end, or apparent improvement for a time may occur. The animal has a staggering gait and often gives way on the pasterns and at the knees, can raise itself from the gound only with difficulty, and sometimes falls down unexpectedly. Some animals exhibit permanent tremblings over the whole body or local paralysis as for instance, of the lips, ears and eyelids. Hyperesthesia of the skin is observed particularly in stallions and with it is extensive pruritis, so that the animal continually rubs itself, bites the affected parts and thus produces extensive sores on the skin. The patient becomes extremely emaciated especially in the hind quarters so that the outlines of the pelvic bones and ribs become prominent. The skin becomes dry, the hair is ruffled and loses its gloss. Some animals manifest pain when the lumbar region is pressed. The senses become more and more blunted and the eyes assume a staring and expressionless appearance. As the end approaches the patient persistently maintains a recumbent position and finally dies from the effect of secondary lesions such as hypostatic inflammation of the lungs, septicemia or perhaps general cachexia. There is often a continued fever at the end, due to secondary infection. Sometimes in the final stage the patient suffers from nasal catarrh with swelling of the submaxillary glands and conjunctivitis. Severe internal inflammation of the eyes has been observed. The appetite continues longer than any of the other normal functions.

Baldrey has divided the symptoms into three different stages, as suggested by Nocard. These stages are more or less distinct, and may, if the case is carefully watched, be easily recognized. They are:

Primary. In which occur the local manifestations of discharge and urethral irritation, and ulceration of the penis and sheath.

Secondary. In which the exanthematous eruptions appear in the skin—the so-called "plaques."

Tertiary. Characterized by the formation of lesions in the central nervous system, and by nervous disturbance with ultimate paraplegia.

It was probably on account of these three periods that the older writers confounded the disease with syphilis, and it is also possible that the ulcerations and chronic enlargements of the sheath and penis gave rise to the idea that it was a form of localized glanders.

The duration of the disease is stated to be from three months to several years. *The prognosis* is unfavorable.

Morbid anatomy. In the early stages there are phlegmonous or edematous swellings of the sheath, scrotum, penis and inguinal glands and a yellowish liquid effusion into the scrotal cavity. The skin covering the parts may show a papular or vesicular eruption or if this has passed a mottling with white spots shows where these lesions have been. Later, the inguinal glands shrink and become firm, owing to the development of fibrous tissue. The testicles, which are either swollen or shrunken, contain in some cases foci of suppuration or caseation. The connective tissue of the epididymis and the cord is the seat of a gelatinous exudate. The walls of the scrotum may be greatly thickened and be the seat of abscesses or of caseous degenerations. In advanced cases the testicles are usually abnormally small, even if the scrotal mass is enormously distended. The sheath and penis may be the seat of more or less numerous ulcers and swellings. Contraction and contortions of the penis are not uncommon. It may, however, retain its normal dimensions.

The walls of the lymphatics in the inguinal region may be the seat of hyperplasia; the thickening causes them to stand out like cords as in glanders. In the advanced stages the muscles, especially those of the hind limbs, become pale and atrophied.

The nerve centers undergo profound changes which have been studied by Thanhoffer. The pia mater in the affected part of the spinal cord may be the seat of active congestion and thickening. The central canal of the spinal cord is irregularly dilated, contains more than the normal amount of liquid and the neuroglia around it is thickened and fibrous. The substances of the cord, both white and gray, may show congestion, blood staining, and points of softening and of hyperplasia of the neuroglia. The nerve cells are modified in various ways, some being granular, some discolored by fine granular pigment, some having enlarged and multiple nuclei and some show vacuoles. Marek found in chronic cases cellular infiltration, degeneration and atrophy of some of its nerve fibers, especially of its posterior

extremities, but sometimes in the cerebral nerves as well. In the affected portion of the cord, leucocytes are numerous and there is often hyperplasia. The neuroglia tends to increase, and apart from the foci of softening, tends to give a special firmness to the substance. The subarachnoid and subdural fluid is increased and there may be at the roots of the spinal nerves, especially in the dorsal and lumbar regions, a gelatinoid exudate investing the nerve, distending the connective tissue beneath the neurilemma and even occupying the interval between the nerve filaments. Sometimes large corpuscular bodies are found between the nerve fibers.

Weber and Nocard state that sections show cachexia and hemorrhagic softening of the spinal marrow. The parasites found in these areas and in the serous effusions resemble those of surra and nagana.

The cerebral meninges are congested and opaque. Foci of softening are by no means uncommon and the cerebral ventricles contain an abnormal quantity of fluid. Hutyra states that changes in the nervous system visible to the unaided eye are often absent in fatal cases.

The bony tissue generally has lost its consistency and the medullary matter may be unduly reddened. The large joints contain an excess of synovia of a somewhat pinkish color. The ligaments of the hip joint are often congested, thickened and softened. The articular cartilages may even show areas of blood staining.

The intestines are usually nearly empty, soft, pale and flaccid. Ruthe has in one case observed rounded ulcers on the mucosa.

The internal organs are usually anemic with edematous infiltration. There may be a suggestion of septic infections. In acute, rapid cases there may be an enlargement of the spleen which is usually small, and the lymph glands may be swollen. Fröhner has called attention to occasional accumulation of leucocytes in the peripheral arteries.

The kidneys are usually large, pale and blackened. The thoracic organs may show little change, though hypostatic congestion may be present. The blood is light colored and forms a loose, pale clot. There is a diminution in the number of red blood cells and a relatively large increase in the number of leucocytes.

In the mare, in addition to the lesions in the internal organs and blood, the following may be noted in connection with the generative system. Phlegmons or edematous swellings, or ulcers on the lips of the vulva and on the vulvar and vaginal mucosæ. The parts become variously distorted. The mammary glands are sometimes inflamed,

edematous and tender, with suppurative or necrotic foci. The adjacent lymph glands are enlarged by infiltration or contracted by sclerosis.

In the dog the symptoms and the lesions resemble those in the horse.

Diagnosis. Dourine is diagnosed by the symptoms, lesions, finding the specific trypanosome and by the complement fixation test. The "dollar spots" or cutaneous plaques are quite characteristic in northern latitudes. The trypanosome may be found in the urethral and vaginal discharges in the early stages of the disease by a careful microscopic examination. They are not present in the blood except for short periods. In chronic cases they are difficult to find.

The complement fixation method is reported by Mohler to be very satisfactory.*

It is being used to identify infected horses in districts where the disease exists.

Dourine is to be differentiated from the other forms of trypanosoma disease, and also from "benign venereal disease."

Pearl found a disease simulating dourine caused by *filaria*.

Williams describes a benign venereal disease of mares all of which had been bred to an imported French draft horse. He states that "the margins of the vulva retain their natural color in this disease, except at the seat of eruptions, when the color quickly returns. The

*The method for the Complement Fixation Test for Dourine used and recommended by Mohler, Eichhorn and Buck (see Journ. of Agricultural Research, November, 1913), is as follows:

"The test proper for the diagnosis of dourine is carried out in a manner similar to that practiced for the diagnosis of glanders.

"The hemolytic system consists of sensitized rabbit serum, serum from a guinea pig, and a 5 per cent. suspension of washed sheep corpuscles.

"The serum to be tested is, of course, inactivated for one-half hour at 56° C. and is used in the tests in quantities of 0.15 cc. since it has been found that fixation in this quantity is obtained only with sera of horses affected with dourine. Tests to determine the smallest quantity of serum of horses having dourine which will give a fixation showed that in several instances even 0.02 cc. of serum was sufficient to give a complete fixation.

"The complement from the guinea pig is always titered previous to the test, as it is absolutely necessary to use the exact amount of the complement to obtain the best results, since a deficiency or an excess of the complement would interfere greatly with the reaction."

The antigen used consists of the ground-up spleen of rats that have died from surra. The spleens are ground with a small amount of salt solution and the suspension thus obtained is filtered twice through double gauze into a test tube. The quantity of suspension from each spleen is made up to 40 cc. by dilution with salt solution.

The complement fixation for dourine using the spleen of surra infected rats for antigen is a group reaction. As dourine is the only trypanosomic disease of horses in this country, such a group reaction is practically specific for its diagnosis.

vulva remains naturally closed, and does not gape, as in *mal du coit.*
The clitoris retains its natural color, size and appearance." The dura-
tion of this affection is stated to be from two to six weeks, but may
persist for a longer time if neglected. The cause is not known. He
reports that when it becomes established it is highly contagious. It
appears to be spread by copulation.

Prevention. The prevention of dourine seems to rest in the isola-
tion of all affected animals. It is important, therefore, that its
diagnosis be made at the earliest possible moment. As it is not
spread except by copulation, it is a comparatively easy disease to con-
trol if taken in time.

Prophylaxis. In Austria where the disease has existed for a long
time, the following rules are observed. Baldrey states that they sum
up the necessary preventive measures. It is understood that they
apply to a country in which the disease is prevalent:

"Even when there is nothing to lead to the supposition that
the disease exists, every mare about to be put to the horse shall be
carefully inspected, and refusal made to old and weakly mares, or to
those which have a discharge from the vulva, or have that organ
enlarged or swollen, or which do not present the ordinary manifesta-
tions of œstrum. It is also suggested that an edematous swelling, no
matter where situated, should negative covering.

"The stallion's penis to be carefully and frequently examined, and
on no account is the animal to be used if there is the slightest lesion
upon it. He is to be kept secluded until all doubt as to the nature of
the lesion has passed away.

"Give all information possible to breeders as to character of the
disease.

"Immediate information is to be given of all stallions in which the
disease is suspected, and the necessary steps to be taken.

"To prevent extension, the sale of all mares in the affected areas
to be stopped during the prevalence of the disease.

"If the malady has spread in a district, all breeding stallions to be
stopped employment, whether Government or private property.
Those already diseased to be sequestrated under police supervision,
whether private or Government property.

"Affected animals to be separated from healthy, to have their own
attendants, and no interchange of clothing, utensils, etc., to take
place. Those deemed curable to be treated, those incurable to be
destroyed.

"All horses attacked to be castrated, as well as those which, not-withstanding their apparent good health, have transmitted it to mares they have served, and also those which have been put to infected mares.

"Mares that have been in the least affected and apparently cured not to be covered the following year, or until certified by a veterinary surgeon, as cured. It is even better to exclude all such mares entirely, and brand them as having had the disease."

Eradication. In this country the Government has undertaken the eradication of the disease by preventing its further spread from any and all places in which it is found. All diseased mares are slaughtered, infected stallions slaughtered or castrated, exposed stallions castrated or quarantined and the exposed mares frequently inspected. The inspections at short intervals of all horses in the infected territory is important.

REFERENCES

1. BALDREY. Dourine. *Jour. Comp. Path. and Therap.*, Vol. XVIII (1905), p. 1.

2. FAVILLE. Extirpation of maladie du coit. *Annual Report, Bureau of Animal Industry*, 1893–4, p 13.

3. HIGGINS. Maladie du coït or dourine. *Special Report. Dept. of Agric.*, Ottawa, 1907.

4. MAREK. Untersuchungen über die Beschälseuche. *Deutsche Tierarzt. Wochens.*, Bd. XVII (1909), S. 121. Ref. *Journ. Comp. Path. and Therap.*, Vol. XXII (1909), p. 169.

5. MOHLER. Cultivation of *Trypanosoma equiperdum*. *Proceedings Am. Vet. Med. Asso.*, 1905, p. 363.

6. MOHLER. Dourine of horses: its cause and suppression. *Bul. 142. U. S. Dept. of Agric., B. A. I.*, 1911.

7. PEASE. DOURINE AND SURRA. *Vet. Journ.*, N. S., Vol. X (1904), p. 297.

8. PEASE. A disease simulating dourine caused by filaria. *Jour. Trop. Vet. Sci.*, Vol. I (1906), p. 414.

9. RENNES. Traitement de la dourine expérimentale des équidés. *Bull. de la. Soc. Cen. de Méd. Vét.*, Vol. LXIII (1909), p. 135. Ref. Treatment of experimental dourine in horses. *Journ. Comp. Path. and Therap.*, Vol. XXII (1909), p. 160.

10. ROUGET. Contribution à l' étude du trypanosome des mammifères. *Ann. de l' Inst. Pasteur*, Vol. X (1896), p. 716.

11. THANHOFFER. Über Züchilähme. Wien, 1888.

12. WATSON. Dourine and the complement fixation test. *Parasitology*, Vol. VIII (1915), p. 156.

13. WATSON. Dourine—its pathogenicity, and a practical test of the efficacy of drug treatment, with especial reference to the action of atoxyl and arsenophenylglycin. *Journ. Comp. Path. and Therap.*, Vol. XXV (1912), p. 39.

14. WILLIAMS. Maladie-du-Coit, or equine syphilis. *Annual Report of the Board of Live Stock Commissioners, for the State of Illinois, Fiscal Year Ending* Oct. 31, 1887. (A full report of the disease and its eradication in Illinois.)

15. WILLIAMS. Benign venereal disease—equine chancroid. *Ibid.* p. 84.

16. WILSON-BARKER. Maladie du coït in Nebraska. *Vet. Jour.*, Vol. XXXV (1892).

SURRA

Synonyms. Relapsing fever of equines; pernicious anemia of horses.

Characterization. Surra is an infect'ous disease of solipeds, camels and cattle caused by a flagellate protozoan. It is determined by a continuous fever with alternate paroxysms and intermissions, with a general or localized eruption of the skin, petechiæ of the mucosæ and more or less subcutaneous edema. There is rapid emaciation and great weakness. It is usually fatal. It can be inoculated into other animals such as dogs, rats, rabbits, guinea pigs and mice. From an economic point of view it is reported to be essentially a disease of horses.

In India, cattle are said to be infected with the trypanosomes of surra but they are not appreciably affected by them. It is reported that horses become infected by insects that have first bitten such cattle.

History. This disease appears to have been known for many years to the natives of the low lands on both sides of the Indus on the northwest frontier of India. Haig appears to have observed it in Persia in 1876. In 1880, Evans found several cases of it in the Dera Ismael Khan county. He was the first to describe it and attribute its cause to an animal parasite which he discovered in the blood. In 1885, Steel met with a disease among mules in Burma which he regarded as identical with Evans' surra, and which he believed to be relapsing fever. In 1888, there was an outbreak among the Bombay Tramway Company's horses. Since then surra has become epizoötic in Bombay; Lingard reports that thousands of ponies, horses, camels and asses died from it during the rains of 1893 and 1894. Its ravages in the Punjab and Northwest Provinces during 1895 are reported to be appalling.

Geographical distribution. It is a disease of Asia and Africa. It is reported that "the distribution of this malady seems to be entirely influenced by the physical aspect of this country; being far more prevalent in those parts where floods and inundations occur than in the higher and dryer portions" (Pease). If the identity of surra with tsétsé-fly disease proves to be true, which is still questioned, it has a wide distribution in Central Africa.

Surra does not exist in the United States, but because of its prevalence and long standing in the Philippines it is liable to be introduced

at any time. In 1906, it was brought to New York with the importation of cattle from India but was suppressed by the slaughter of the animals at quarantine by the Federal Government. It was introduced into Australia with infected camels. For these reasons its nature should be understood by American veterinarians.

Etiology. Surra is due to the presence in the blood of *Trypanosoma evansi*.

"A motile trypanosoma 20 to 30μ in length to 1 to 2μ in breadth, somewhat blunt at the posterior end and gradually tapering at the anterior end. The undulating membrane is well defined, beginning at or near a small body (centrosome) in the posterior portion of the parasite and extending forward as a free flagellum. It is provided with a nucleus and a granular protoplasm."

This organism is invariably found during the paroxysms of the disease in the blood of animals which have acquired surra either naturally or experimentally. Although blood containing these infusoria readily communicates the disease to susceptible animals it entirely loses its virulence when it is filtered through porcelain, so as to free it from the parasite. The disease can be transmitted to healthy, susceptible animals even of different species with the unfiltered blood of a diseased animal. The microscope reveals the trypanosomes in vast numbers moving with great activity in the blood. When this acute stage has passed the organisms disappear, the temperature falls, the severity of the symptoms abates, and there is an intermission, during which, at the beginning of the attack, the patient may appear in good health. Although the blood during an intermission may appear under the microscope to be absolutely free from the parasites, its inoculation into susceptible animals will, as a rule, produce the disease. The blood of surra affected horses loses its power of transmitting the disease by inoculation in about eighteen hours after death.

Under the microscope, these parasites are detected in a drop of blood by an irregularly intermittent and characteristic quivering of some of the red blood corpuscles, which become much altered in form. The leucocytes remain unchanged in appearance. After a further and careful examination of this slightly quivering blood a minute thread-like organism with eel-like movements emerges from the mass of corpuscles. It may be seen apparently tugging with all its might at a red corpuscle endeavoring to detach it from its *rouleau*. The question of the manner in which these parasites interfere with the health of the affected animal has not yet been settled. When they

are outside the animal body and in a dry state, they are killed or rendered inert by prolonged atmospheric action.

The period of incubation seems to be liable to great variation. It may be put from four to eight days after inoculation or ingestion of blood taken from an animal suffering from surra. It appears from Lingard's investigation that it may be prolonged to thirteen days, if the blood used for inoculation has been taken from a dead animal.

Means of transmission. It was formerly believed that surra was transmitted by ingestion of infected food or water. Lingard stands almost alone in the belief that infection can take place through healthy mucosæ. It is reported that carnivora have contracted the disease by eating blood and meat from infected animals.

The demonstrated natural methods of transferring the virus from infected to non-infected animals is by means of insects, especially the biting flies. Of the flies, the tsétsé-fly (*Glossina morsitans*) is reported to be the most important. Musgrave and Clegg conclude concerning the rôle played by flies in transmitting this disease that "it has thus far been conclusively shown that the tsétsé-fly (*Glossina morsitans*), at least one other variety of *Glossini, Stomoxys calcitrans, Musca brava* (?), *Taon*, and at least one variety of *Tabani* transmit the virus. All other biting insects have been looked upon with suspicion, but absolute proof of transmission by them has not been furnished." The theory as to the method of transferring the parasite is that it is purely mechanical, although some have thought the trypanosome passed through one phase of its life cycle in the fly. Mohler and Thompson state that the breeze fly, *Labanus atratus*, was the carrier of the trypanosome in the quarantined India cattle in New York in 1906.

In 1901, surra was introduced into the Island of Mauritius by an importation of Indian cattle and from 70 to 80 per cent. of the native cattle died of it. Mitzmain found *Tabanus striatus* to be the carrier in the Philippine Islands.

The spread of the disease from one locality to another is caused by the introduction of cattle carrying the parasite, although themselves but slightly susceptible.

Symptoms. The symptoms as given by Lingard are as follows: "The chief symptoms are the occasional appearance of an urticarial eruption, generalized or localized, closely following the first rise of temperature, but which may make its appearance at any time during

the course of the disease; then the presence of petechiæ on the mucous membranes, chiefly that covering the membrana nictitans, lachrymation and the exudation of a semi-gelatinous material into the subcutaneous and other connective tissues. There is rapid wasting and great weakness, although in the majority of cases the appetite remains good throughout, no matter how high the fever. There is extreme pallor of the visible mucous membranes, and this is followed at a later period by yellowness. From first to last there is progressive anemia; the blood at first presents a normal character, but after a varying period of time it undergoes marked changes. The white corpuscles are increased in number and the red corpuscles usually cease to form normal rouleaux, lose their individuality and run together forming irregular masses. They are at first dark, but gradually, as the disease advances, almost entirely lose their coloring matter and become pale."

It is stated that there are edematous swellings of the extremities, the submaxillary region and the genital organs.

The duration of the paroxysms and intermissions is very irregular. Lingard puts it down as from one to six days. He states that in a few experimental horses the paroxysms lasted from eighteen to twenty-two days.

The duration of the disease is, according to Gunn, about fifty-two days. Others place it at from one to eight weeks. In the Philippine Islands the duration in horses is from fourteen days to three months. The prognosis is always unfavorable, the mortality in most species of animals is very high, although in cattle a large percentage recover.

Morbid anatomy. As described by those having an opportunity to study the disease, there is great emaciation, enlargement of the liver and spleen, and petechiæ on various internal organs. A yellow or amber-colored jelly-like exudation occurs in the connective tissue of the throat, chest and abdomen, about the muscles and other tissues, and especially around the base of the heart. The lungs often show signs of inflammation. The mucous membranes and other tissues are frequently tinged yellow by the coloring matter of the bile.

It has been stated that the hide is often removed with difficulty. In the areas corresponding to the edema during life are found yellowish-tinged, gelatinous infiltrations. The serous membranes, especially the peritoneum and pleura, often show flakes of plastic, fibrous material. These are especially numerous over the liver. All of the organs have a dry, pale appearance. There are numerous sub-serous

hemorrhages, particularly on the right side of the heart and over the lower portion of the lungs. The lymphatics are in general somewhat enlarged, often markedly so. The heart muscle shows parenchymatous changes, in degree depending somewhat on the duration of the disease.

In some of the lower animals the scrotum and even the testicles in the male and the vulva in the female are greatly swollen, and in the male rabbit the tension may be so great as to rupture the scrotum. Small preputial or labial ulcers are not uncommon.

Steel noticed ulceration of the stomach in about two-thirds of his
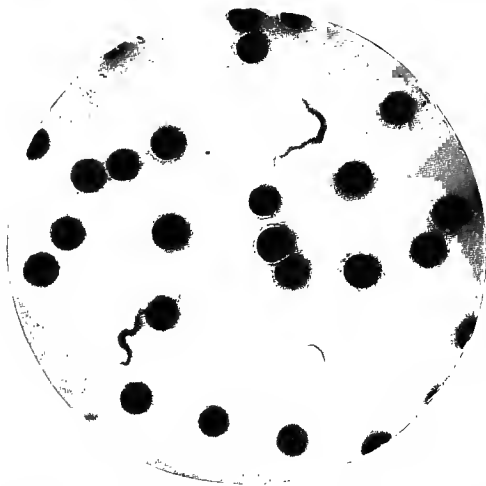
FIG. 88. PHOTOGRAPH OF BLOOD OF HORSE CONTAINING TRYPANOSOMA.
(*Taken by Smith and Kinyoun.*)

cases among mules in Burma. In India this ulceration has not been observed among horses as a sequence of surra. In the Philippines, changes in the intestine due to anemia with occasional ulcers are reported.

The clinical aspect of surra is essentially one of progressive anemia, accompanied by paroxysms and intermissions, during both of which there is a natural decrease in the number of the red blood corpuscles and in the amount of hemoglobin in the blood, with consequent anemia of the visible mucous membranes.

Smith and Kinyoun found the blood changes to be as follows:

"The pathological changes caused by this parasite is a rapid destruction of the red blood cells, causing an acute anæmia. The changes occur in the blood coincident with the

invasion of the parasite. In one horse which had been ill seven days, the red blood cells numbered 3,500,900, the white 14,500. In another, ill six weeks, the red blood cells were 3,200,000, and the white were 13,900. The blood of a healthy horse, taken as a comparison, gave red blood cells 6,900,000, white 9,800. There is also a slight diminution in the amount of hæmoglobin, about 85 per cent."

Diagnosis.   Surra is to be diagnosed by the symptoms, lesions and finding the specific organism.   In case of infected cattle, it is usually necessary to resort to animal inoculations with the blood of the suspected animal and finding the trypanosomes in the blood of the inoculated animal.   For this purpose the rabbit or dog is very satisfactory.   In from five to ten days after inoculation with from one to two cubic centimeters of its blood the temperature of the animal goes up and its blood reveals the parasite on microscopic examination. The surra organisms agglutinate.   Complement fixation test is used for diagnosis.

Surra is to be differentiated from anthrax as well as other trypanosoma diseases.   A history of the case or outbreak together with the chronic course and intermittent temperature in surra will usually suffice to determine the nature of the disease.   A positive diagnosis can easily be made in the horse, in a majority of cases, by a microscopic examination of the blood.   The trypanosomes are readily observed, and usually they are in sufficient numbers to be quickly seen.   In a suspicious case, where the organisms are not found, the examination should be repeated daily or small animals inoculated with the blood.   Although this requires some time the importance of a positive diagnosis demands that it should be done.

Prevention.   The importation of animals from infected countries should be prohibited.   If the disease gains entrance, the infected animals should be destroyed.   In framing regulations for quarantine particular attention should be paid to the danger of infected wild animals and of circus animals bringing the virus to this country where it may be transmitted to native stock by means of flies.

Lingard found that arsenic has a decided effect in diminishing the number of surra organisms in the blood of affected animals.   Thus far serum therapy is not successful.

REFERENCES

1.   BURKE.   Surra or progressive pernicious anæmia.   *Vet. Jour.*, Vol. XXV (1887).
2.   DURHAM.   Tsetse disease.   *Veterinarian*, Vol. LXXI (1898), p. 535.
3.   DURRANT.   A trypanosoma found in blood of cattle in India.   *Jour. Comp. Path. and Therap.*, Vol. XVII (1904), p. 209.

4. Evans. On a horse disease in India known as "surra," probably due to a Hæmatozoon. *Vet. Jour.*, Vol. XIII (1881).

5. Hassall. Bibliography of surra and allied trypanosomatic diseases. *Bulletin No. 42, U. S. Bureau of Animal Industry*, 1902, p. 131.

6. Holmes. Evolution of the trypanosoma evansi. *Journ. of Comp. Path. and Therap.*, Vol. XVII (1904), p. 210.

7. Kanthack, Durham and Blandford. On nagana or tsetse fly disease. Report made to the tsetse fly committee of the Royal society, etc. *Proc. Royal Soc., London*, Vol. LXIV, p. 100.

8. Lingard. Report on horse surra. 1893. (Bombay).

9. Metzmain. The mechanical transmission of surra by *Tabanus striatus*. *Bulletin No. 28, Bur. of Agric.*, P. I., 1913.

10. Mohler and Thompson. A study of surra found in an importation of cattle, followed by prompt eradication. *Cir. 169, U. S. Dept. of Agric., B. A. I.* 1911. (See also Bulletin No. 42 or 18th Ann. Rept. of the Bureau).

11. Musgrave and Clegg. Trypanosoma and Trypanosomiasis, with special reference to surra in the Philippine Islands. *No. 5, Bureau of Government Laboratories, Manila*, 1903.

12. Nocard. Sur les rapports qui existent entre la dourine et le surra ou le nagana. *Comp. rend. Soc. de Biol.*, Vol. LIII (1901), p. 465.

13 Pease. Surra trypanosoma in cattle. *Jour. of Comp. Path. and Therap.*, Vol. XVIII (1905), p. 222.

14. Ranking. A preliminary note on the nature and pathology of the disease known as "surra" affecting horses and mules in India. *Vet. Jour.*, Vol. XXXII (1891).

15. Salmon and Stiles. Emergency report on surra. *Bulletin No. 42, U. S. Bureau of Animal Industry*, 1892.

16. Schilling. Bericht über die Surra-Krankheit der Pferde. *Centralblatt f. Bakteriologie u. Parasit.*, Bd. XXX (1902), p. 545.

17. Smith and Kinyoun. A preliminary note on a parasitic disease of horses. *Army Pathological Laboratory, Manila*, Oct. 17, 1901.

18. Steel. On relapsing fever of equines. *Vet. Jour.*, Vol. XXII (1886).

19. Steel. Report upon an obscure and fatal disease among transport mules in British Burma. 1885.

MAL DE CADERAS

Synonyms: Caderas; flagellosis of equidæ.

Characterization. Mal de caderas (disease of the rump) is a disease essentially of the horse kind, characterized by an intermittent fever, a progressive paralysis of the posterior parts, rapid emaciation and death. It is a "wet weather" disease, as it is reported to almost entirely disappear in the dry season. Horses, mules, and asses suffer from natural infection. It is inoculable into many species.

History. Rebourgeon studied this disease in 1889. He made a bacteriological investigation into its cause without success. Leclerc described it clinically in 1899. He believed that he had found its pathogenic bacterium. In 1901, Elmassian showed that this disease was caused by one of the trypanosomata. Voges and Lignières confirmed his discovery.

Geographical distribution. Mal de caderas is a disease of tropical South America.

Etiology. Elmassian differentiated the trypanosoma of this affection in 1901. Voges pointed out its pathogenic action and named it *Tr. equinum*. In length it is 3 or 4 times the diameter of the red blood corpuscles. Its width is one-third to one-half the diameter of a red blood cell. Its free flagellum is about 5μ in length.

Its motion resembles that of an eel, but its actual motility is not great, the whole body taking part in an excessively active wriggling motion with the flagellum and beak ends moving in opposite directions. The nucleus is toward the anterior end, a very small centrosome near the posterior end, and there is a granular protoplasm.

It is found in the blood of horses, mules, asses, hogs and water hogs suffering from *mal de caderas*. It is transmissible to white and gray rats and mice, rabbits, dogs, goats, sheep, and certain monkeys found in South America. Cattle are said to be more immune. They acquire infections but do not suffer from the disease.

The parasites are most numerous in the circulating blood during the rise of temperature. Upon its reaching 40–41° C. they gradually disappear, but reappear with the next rise of temperature.

Mode of infection. Unlike dourine, the virus of mal de caderas is not transmitted by copulation. It has been proven that animals are infected with it by means of certain insects. *Stomoxys calcitrans*, *Tabanidae* and several other insects are under suspicion. It is also stated that a common rodent of Paraguary (*Hydrochœrus Capybara*) suffers naturally from this disease and that when it dies with it in pastures the horses are attacked.

Symptoms. The first symptom is an elevation of temperature which rises irregularly but suddenly falls to normal. Emaciation is rapid. The urine is dark colored and usually contains albumin, and perhaps blood. The blood changes so that it gives the picture, on microscopic examination, of pernicious anemia. There is an increase in the lymphocytes and in the eosinophiles. The most obvious symptom is said to be a symmetrical or asymmetrical paresis of the hind legs. Defecation and urination are difficult (coinciding with paralysis of the sphincters). The paralysis gradually extends to other parts of the body. Edema is often present. The appetite remains good until near the end when there is extreme thirst.

The duration of the disease is variable. Some animals die after a month, others live for a year or longer. Stiles states that it lasts from two to five months in horses and from six to twelve months in mules and asses. Horses are reported to never recover.

Morbid anatomy. The muscles are pale and atrophied in the posterior part of the body. The intermuscular tissue is infiltrated with a gelatinous serous-like substance. Hemorrhagic foci appear in the muscles of the rump.

The spleen and lymphatic glands are enlarged. The liver is enlarged and congested. The heart muscle is soft and flabby. The lungs often contain ecchymoses and subpleural emphysematous areas. There is a serofibrinous exudate in the body cavities, especially in the pericardial sac and pleural spaces. There are conflicting statements concerning the morbid anatomy and it is difficult to select statements relative to the lesions that are not contradicted. The pathological histology and the lesions in the nervous system do not appear to have been described.

Diagnosis. The diagnosis is made from the intermittent fever, emaciation, progressive paresis, anemia, and the finding of the parasite. The inoculation of experimental animals (mice or dogs) with the blood is of value when the parasite is not found on microscopic examination in the blood of the horse. This affection is to be differentiated from the other forms of trypanosoma infections. There seems to be no other specific disease with which it would be confused.

Morphologically the trypanosome of caderas is distinguished from the parasites of nagana, surra, and dourine by the small size of its centrosome. Caderas is not propagated in the same way as dourine, and moreover, most mammals are susceptible to caderas, whereas the number of species susceptible to dourine is very limited. Finally, animals which have acquired immunity against nagana, surra, or dourine are as susceptible to caderas as normal animals, and *vice versa*.

REFERENCES

1. LECLERC. El mal de Caderas. *Buenos Ayres*, 1899.
2. LIGNIÈRES. Contribución al estudio de la trypanisomiasis de los Equideos Sud Américanos. *Buletin de agricultura y yanaderia (Republic Argentina)*, 1902, p. 843.
3. REBOURGEON. Note sur le mal de Cadeiras. *Recueil de méd. vét.*, 1889, p. 85.
4. VOGES. Das mal de Caderas .*Zeitschrift f. Hygiene*, Bd. XXXIX (1902), S. 323.

See also literature on other trypanosomata.

NAGANA

Synonym: Tsétsé-fly disease.

Characterization. Nagana is a disease characterized by anemia and rapid emaciation caused by *Trypanosoma brucei*. It attacks horses, mules, zebras, cattle, and sheep. A number of the smaller animals are susceptible. It is known to all dialects as the tsétsé-fly disease.

History. Livingston pointed out the existence of this disease in Central Africa. In 1886, Bruce studied it in the Zulu Land. He found constantly in the blood of the sick animals a trypanosoma similar to that found in surra. It has been carefully studied by Kanthack, Durham and Blandford, Koel, Plimmer and Bradford, Theiler, Schilling, Laveran and Mesnil.

Geographical distribution. This disease is found in central and southern parts of Africa. There seems to be some doubt about its identity with the disease of a similar nature in the Transvaal.

Etiology. This affection is caused by *Trypanosoma brucei*. It is from 28 to 33μ in length with the flagellum and about 1.5μ in width. It is closely related to *Tr. equiperdum*. Some investigators have been unable to satisfactorily differentiate the two species, while others recognize them as distinct.

The trypanosomes are transmitted from the diseased to the healthy animals by means of the tsétsé-fly (*Glossina morsitans*), which exists in certain parts of Africa. It is suspected that other species of this genus transmit the trypanosoma. The affection is extended into uninfected areas by the introduction of diseased animals.

The period of incubation, in artificially produced cases, is reported to be about 4 days in the horse.

Symptoms. The first indication of the disease is a rise in temperature which lasts for 3 or 4 days, when it suddenly drops. After this time the temperature oscillates between 35° and 41° C. Emaciation is rapid, the hair becomes rough and may fall out. There is a tendency to diarrhea. There is edema of the abdominal walls. In cattle the symptoms are not usually so acute as in the horse.

The duration of the disease is said to vary from a week to six months or more. The appetite remains good until near the end. According to Bruce recovery is rare.

Morbid anatomy. There appear to be no distinctive anatomical changes for this affection. The tissues generally are reported to be anemic and infiltrated with a serous exudate. If the edematous portions are incised a clear amber or citron colored fluid escapes. The spleen may be enlarged but the color and consistency are normal. The liver and kidneys are said to be slightly affected.

Diagnosis. The diagnosis is made clinically from the progressive anemia and edema, coincident with a good appetite. The finding of the parasite in the blood is positive evidence. This disease is to be differentiated from the other affections caused by trypanosoma.

REFERENCES

1. KANTHACK, DURHAM AND BLANDFORD. On nagana or tsétsé-fly disease. *Proceedings of the Royal Society*, Vol. LXIV (1898), p. 100.

2. KLEINE. Positive Infektionsversuche mit *Trypanosoma Brucei* durch *Glossina palpalis*. *Berl. Tierärzt. Wochensch.*, Bd. XXV (1909), S. 244. Ref. *Journ. of Comp. Path. and Therap.*, Vol. XXII (1909), p. 256.

3. LAVERAN AND MESNIL. Recherches morphologiques et expérimentales sur le trypanosome du nagana ou maladie de la mouche tsétsé. *Ann. de l' Inst. Pasteur*, Vol. XVI (1902), p. 1.

4. PLIMMER AND BRADFORD. A preliminary note on the morphology and distribution of the organism found in the tsétsé-fly disease. *The Veterinarian*, Vol. LXXII (1899), p. 648.

5. THEILER. Die Tsétsé-Krankheit. *Schweizer-Archiv für Thierheilk.*, 1901, S. 97.

Differentiation of surra, dourine, mal de caderas and nagana. The divergence of opinion concerning the nature of these affections and the specific identity of the exciting cause, renders a differentiation or unification of these most interesting diseases exceedingly difficult. Very few investigators have had the opportunity of studying all of them in their natural environment. The conclusion of Musgrave and Clegg in their recent report is worthy of consideration. They say: "In summing up the whole matter it appears to us, when we take into consideration the work done by others and add our own results, that we are justified in believing surra, nagana, mal de caderas, and probably dourine, the same disease, and that all are caused by Tr. Evansii."

Koch, who worked with *surra* and *nagana*, considered the parasites and the resulting infections identical. Many others have formed similar conclusions. Other investigators such as Voges, Laveran and Mesnil and others maintain that certain differences exist. The evidence is convincing that dourine and mal de caderas are different in some respects from the other two. Voges' reasons for this are:

"Dourine and mal de caderas can not be transmitted to cattle.

"In regions where mal de caderas exists cattle do not die of surra.

"We have no reason to believe that trypanosoma show the same irregularities of virulence as bacteria, so that the different forms of the disease may be said to be produced by different degrees of virulence in the same trypanosoma. On the contrary, during our four years of experimentations, the latter have shown a constant virulence."

The fourth reason which he considers decisive is based on the morphological differences in the parasites.

Voges concludes by saying, "I think these four proofs are entirely sufficient to establish for all time the difference between *surra* and *dourine* as well as between *surra* and *mal de caderas*." Laveran and Mesnil give extensive consideration to the differences between surra and nagana. A summary of their considerations is appended.

"The same animals are susceptible to both diseases—the horse, ass, mule, goat, sheep, cow, camel, dog, cat, monkey (long-tailed macayo), rabbit, guinea-pig, and rat.

"In the horse the course of the disease is the same, whether surra or nagana. The animal dies at the same time, in 30 days on the average. In inoculation cases the period of incubation is the same, and the same symptoms and lesions supervene.

"The other equides, the goat, sheep and dog, die of the two diseases in the same length of time and with similar symptoms and lesions.

"Rabbits, guinea-pigs and rats succumb to the infection in a like manner.

"Cows rarely survive *nagana*, and they rarely die from *surra*. They become emaciated with surra but recover in health and a subsequent inoculation does no harm. This is a marked difference between them but it may be explained when further experiments are made."

Laveran and Mesnil believe that the paralysis of the posterior extremities, a marked symptom in *mal de caderas*, is less marked in surra and nagana, although they believe the three affections very closely related. Dourine differs from the other three in two distinct points: (1) The morphology of the parasite is different. (2) In dourine contagion by coition seems to be the only natural mode of infection.

Much additional investigation will be necessary before the question of either the identity or the non-identity of these affections can be positively determined. As these diseases are not liable to become of great economic importance in this country, further discussion of the voluminous literature seems unnecessary.

CHAPTER XII

DISEASES CAUSED BY PROTOZOA
SUB ORDER MICROSPORIDIA

RABIES

Synonyms: Hydrophobia; canine madness; lyssa; *rage*; *Toll-wut*; *Wutkrankheit.*

Characterization. Rabies is an acute infectious disease transmitted from animal to animal or from animal to man by the bite of the rabid individual or by direct inoculation. It is not known to be contracted or transmitted in any other manner. It is characterized by a long and variable period of incubation, followed by symptoms referable to the nervous system, lasting from one to ten days, and ending in paralysis and death, without recognizable gross anatomical changes.

The dog is the animal most commonly affected, although all of the canine and feline races seem to suffer from rabies more than other species. All warm blooded animals appear to be susceptible. It is a serious disease in man, cattle, sheep, horses and swine. An explanation for its greater frequency among dogs is found in their tendency to bite. A very large percentage, in fact nearly all, of the cases in man and in the domesticated animals are caused by inoculation from the bites of rabid dogs.

History. Rabies was described by Aristotle in the fourth century B. C. He wrote,"Dogs suffer from madness that puts them in a state of fury, and all the animals that they bite, when in this condition, become also attacked by rabies." Cornelius Celsius, who lived in the first part of the Christian era, seems to have been the first to refer to human rabies and to employ the term "hydrophobia." It is referred to in the writings of Hippocrates.

The transmission of the disease by wolves to man was recorded in 1591. In 1803, and for a number of years following, it was epizoötic among foxes in Southern Germany and Switzerland.

The first outbreak in this country was reported from Boston in 1768. In 1770 and 1771 it was observed in dogs and foxes in the vicinity of Boston; in the year 1779 it appeared in Philadelphia and

in the state of Maryland; in 1785 it was prevalent throughout the Northern States and soon after it spread to the South. It has caused heavy losses among farm animals throughout Europe and America.

It has called forth careful study from many of the ablest men in the medical professions. Among them may be mentioned John Hunter in England, Viborg in Copenhagen, Waldinger in Vienna, Hertwig in Germany and Pasteur in France. Without detracting in the least from the great work of other investigators, we may say that to Pasteur and his co-laborers, Nocard and Roux, we owe much of the knowledge of the nature of rabies which we possess at the present time.
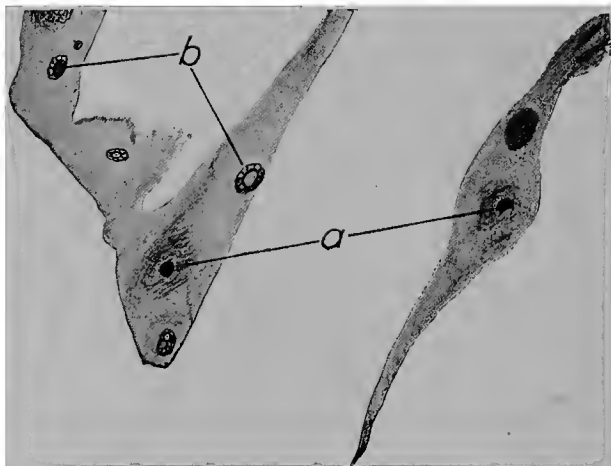
FIG. 89. NEGRI BODIES IN NERVE CELLS: (a) NUCLEI OF CELLS; (b) NEGRI BODIES
(after Frothingham).

Geographical distribution. Rabies is known to exist or at some time to have existed in almost every country on the globe. Australia is the largest area which is said to be absolutely free from it. This exemption is the gratifying result of a rigid quarantine enforced against dogs imported on that island. It is wide spread in the United States.

Etiology. In 1903, Negri described small bodies or cell inclusions, since called Negri bodies, which he found in the Purkingie cells of the cerebellum and in the large ganglion cells of the Ammons horn. Negri believed these bodies to be the etiological factor of the disease and classified them among the protozoa.

He believed the bodies were specific microörganisms characteristic of the disease, found only in animals affected with rabies. They appear early in the course. They occur in larger numbers and are of greater size as the disease progresses. They are most numerous and largest at the time of death. Smears or sections of the brain are stained in saturated alcoholic solution of eosin for from 10 to 30 minutes after which they are counter-stained in alkaline methylene blue. No special technique is necessary to demonstrate these bodies. Their constant appearance in cases of rabies forms a basis for a positive diagnosis and they are affected very little by beginning decomposition of the surrounding nervous tissue.

Williams and Lowden state concerning the channels of infection that "in whatever way the virus enters the body, so far as we know, there is no development of the organism, or none, to any appreciable extent, until it reaches the central nervous system, and not until after a certain amount of development there does it infect the peripheral organs. Before the disease was well studied it was thought that the salivary glands were the chief site of the infection. But it has been shown that these glands are not always infective, and when they are, not until comparatively late in the disease and that when the virus is inoculated into them, the animal seldom comes down with the disease and probably never if the centripetal nerves are cut (Bertarelli). This means that the parasite does not grow in the salivary glands, that it is only carried there incidentally by its spread from the central nervous system along the nerve branches. That the organisms escape into the blood and are carried in this way in small numbers is shown by the fact that the blood in large quantities has been found infective (Marie). Principally by the nerve channels, secondarily by the blood and lymph channels, the organisms are carried in small numbers to all parts of the body. With other investigators, we have found the suprarenal capsules infective." Their conclusions relative to the nature and diagnostic value of the Negri bodies are as follows:

"The smear method of examining the Negri bodies is superior to any other method so far published for the following reasons: (a) it is simpler, shorter and less expensive; (b) the Negri bodies appear much more distinct and characteristic. For this reason and the preceding one, its value in diagnostic work is great; (c) the minute structure of the Negri bodies can be demonstrated more clearly; (d) and characteristic staining reactions are brought out.

"The Negri bodies as shown by the smears as well as by the section are specific to hydrophobia.

"Numerous 'bodies' are found in fixed virus.

"'Bodies' are found before the beginning of visible symptoms—i.e., on the fourth day in fixed virus, on the seventh day in street virus, and evidence is given that they may be found early enough to account for the appearance of infectivity in the host tissues.

"Forms similar in structure and staining qualities to the others, but just within the limits of visible structure at (1,500 diam. magnification) have been seen. Such tiny forms, considering the evidence they give of plasticity, might be able to pass the coarser Berkefeld filters.

"The Negri bodies are organisms belonging to the class Protozoa. The reasons for this conclusion are: (a) They have a definite, characteristic morphology; (b) This morphology is constantly cyclic, i. e., certain forms always predominate in certain stages of the disease, and a definite series of forms indicating growth and multiplication can be demonstrated; (c) The structure and staining qualities, as shown especially by the smear method of examination, resemble that of certain known Protozoa, notably of those belonging to the sub-order *Microsporidia.*"

The proof that the "Negri bodies" are living organisms is sufficient proof that they are the cause of hydrophobia; a single variety of living organisms found in such large numbers in every case of a disease, and only in that disease, appearing at the time the host tissue becomes infected in regions that are infective, and increasing in these infective areas with the course of the disease, can be no other, according to our present views, than the cause of that disease.

The Negri bodies or cell inclusions vary in shape. The most common forms are round or oval. The round bodies are from 0.5 to 23μ in diameter, while the oblong ones vary from 0.5 to 1.5μ to 5 to 27μ. The round or oval forms are by far the most common. Irregular forms are occasionally seen. In preparations stained with eosin and methylene blue they appear as bright red bodies containing one or two nuclear-like structures which are surrounded by a number of other small circular, regular bodies. They are said to preserve their form even when the brain tissue has undergone marked degenerative changes, after prolonged immersion in glycerin and after several days' drying. A number of workers report finding them quite uniformly. Schüder does not consider them as the cause, largely because his virus passed through a filter that retained the cholera spirillum.

Remlinger and Riffat-Bey found that the virus would pass through a Berkefeld filter "V," but its passage was not constant, as rabbits inoculated with the filtrate did not all die of rabies. The filter which he used held back the organism of chicken cholera, which was used as a check on the filter. Remlinger more recently stated that the virus will pass through the more porous Berkefeld filters only.

Babes states that the Negri bodies are not always present. He seems to consider them as a product of the reaction of the cells to the cause of the disease.

Resistance of the virus. The virus is destroyed by drying and by the action of light. In dry air, protected from light and putrefaction, the virulence of the spinal cord of rabbits is destroyed in fourteen to fifteen days. When spread in thin layers it is killed by drying in from four to five days and by sunlight in about forty hours. The loss of virulence by drying is gradual but quite regular, which fact was taken advantage of by Pasteur in the preparation of his vaccine.

The virus may be preserved in neutral glycerin at ordinary temperature for a long time. Roux found that after four weeks in glycerin at 30° C., the virus in a rabid brain had the same power as when perfectly fresh. Moore found that rabbits inoculated with rabid brains that had been kept in glycerin from three to four weeks did not develop the disease as quickly as those that were inoculated with the freshly removed organ.

Rabies virus is quite resistant to putrefaction. Galtier found the virus active in the central nervous system of rabbits that had been buried for twenty-three days, of sheep buried thirty-one days and of dogs buried forty-four days. Other observers have found it still active in animals buried for twenty-four days.

It is destroyed completely by a temperature of 50° C. in one hour or 60° C. in one-half hour. It is uninjured by exposure to extreme cold, resisting the prolonged application of a temperature from 10 to 20° C. below zero.

Its activity is destroyed in one hour by a 5 per cent. solution of carbolic acid, or by a 1 to 1,000 solution of corrosive sublimate. Water saturated with iodine destroys it in ten minutes.

Method of invasion. When introduced into an animal either experimentally or by the bite of a rabid dog, the virus remains for a time without producing either local or general symptoms. The virus penetrates the nervous system by following the nerve trunks from the site of infection to the spinal cord, then through the spinal cord to

the brain. It seems to be necessary to lacerate the nerves in order to cause infection. This has been proven by inoculating an animal in one of the legs with virulent material. After a suitable time, but before the symptoms of rabies appear, the virus will be found, on killing the animal, in the nerves of the limb, and even in the part of the spinal cord into which the nerve enters, while the upper part of the cord and the brain are still uninfected. This explains why the earliest symptoms, both in man and animals, such as itching, tingling, numbness and other nervous sensations, often, if not always, appear in the part of the body which received the virus. Inoculation into the large nerve of the leg is almost as certain to produce the disease, as inoculation directly into the sub-dural space. In the case of a bite about the face and head the route along the nerve to the central nervous system is much shorter. While the nerves seem to form the main route by which the virus travels, the circulation may at times assist, especially in small animals. Nicholl finds that the virus passes from the point of infection to the brain exclusively through the nerves. He also finds that it is rapidly destroyed in the blood.

Nocard and Roux, also Rabieaux and Guinard showed that the saliva was virulent two days before symptoms appeared. Williams and Lowden found "bodies" in the cells before symptoms developed.

Noguchi has succeeded in keeping the virus of rabies alive on artificial media. Further reports on its cultivation are awaited with interest.

Period of incubation. The period of incubation is quite variable depending on the site and character of the wound, which is almost always a bite, the amount of virus introduced and its virulence. In general it may be said for all animals that the period of incubation seldom exceeds sixty days, although in man and in some larger animals, it sometimes, though very rarely, reaches a year. A few cases of a longer period have been reported. The average period as given by Ravenel is as follows:

In man, 40 days; in dogs, 21 to 40 days; in horses, 28 to 56 days; in cattle, 14 to 80 days; in cats, 14 to 28 days; in pigs, 14 to 21 days; in goats and sheep, 21 to 28 days; in birds, 14 to 40 days.

In rabbits inoculated subdurally with the brain from rabid animals, Moore found the period of incubation to vary from 12 to 62 days and the duration of the disease to range from a few hours to three days. Westbrook reports a period of incubation in rabbits to extend in one case over a hundred days. In the disease as it is naturally con-

tracted from the bites of rabid animals, the period of incubation varies with reference to the location and extent of the bites. If the individual is bitten about the head the period of incubation is much shorter than if the injuries are on the extremities.

In the dog, the period of incubation in 144 cases was clearly determined by Peuch. His table with the addition of percentages is appended.

PERIOD OF INCUBATION OF RABIES IN THE DOG

Number of days of incubation	Number of cases	Per cent
5 to 10	3	2.08
10 to 15	8	5.55
15 to 20	13	9.03
20 to 25	25	17.36
25 to 30	13	9.03
30 to 35	25	17.36
35 to 40	6	4.17
40 to 45	11	7.64
45 to 50	9	6.25
50 to 55	4	2.78
55 to 60	2	1.39
60 to 65	7	4.86
65 to 70	1	.69
70 to 75	5	3.47
80 to 90	7	4.86
100 to 120	4	2.78
365	1	.69
Total	144	

The somewhat popular opinion that most of the cases of rabies occur in the summer, especially in "dog days," is not well founded. Rabid dogs are nearly, if not quite, as numerous in spring, fall and winter as in summer. Salmon collected 14,066 cases of rabies in dogs with the months in which the disease occurred. The results are exceedingly interesting as the appended table shows.

Symptoms. Rabies is generally divided into two forms, furious and dumb. In the first the animal is irritable, aggressive and bites nearly every object which comes in its way; in the second the muscles of its jaws are paralyzed almost from the beginning and being unable to bite the animal remains more quiet and tranquil. Essentially the two forms of the disease are the same, but, probably owing to the

parts of the brain infected or the acuteness of the attack or both, paralysis appears much sooner in the dumb form than in the other. The saliva from a case of dumb rabies is just as virulent as that from a case of the furious form. Dogs affected with dumb rabies are less dangerous simply because they are unable to bite and thus to infect others.

Dumb and furious rabies do not always represent two distinct types of disease. The typical cases belong to the two extremes of symptoms and there are always gradations between them. In fact, almost

CASES OF RABIES IN DOGS, BY MONTHS

Source	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Bourrel	36	31	26	32	32	42	32	30	35	41	24	32	393
St. Cyr	12	15	6	15	13	7	4	9	1	3	....	2	87
Hōgyes	309	310	314	367	450	502	580	537	455	438	303	396	4961
Leblanc	103	97	121	192	155	138	147	123	104	117	95	100	1492
France													
1895	89	155	153	184	181	129	157	147	133	110	105	149	1692
1896	124	138	151	150	147	199	138	117	131	125	103	164	1687
1897	131	151	189	202	225	172	192	154	136	131	150	140	1973
1898	139	148	....	181	216	278	185	177	150	....	153	154	1781
Total	943	1045	960	1323	1419	1467	1435	1294	1145	965	933	1137	14066

every case of furious rabies sooner or later changes to the dumb form, that is, the final stage of the disease is almost invariably paralytic. In the typical development of the dumb form, the paralysis occurs on the first day of the disease. It may not appear, however, until the second or third or even later.

Again, a dog does not necessarily bite everything about it even if it has rabies and its jaws are not paralyzed. It may be combative and furious all of the time or only part of the time, or not at all. There is perhaps no other disease in which the symptoms may vary more than in rabies of the dog.

Furious rabies. The symptoms appear very gradually. The animal's habits and behavior are changed. It may be more restless or affectionate than usual, seeking to be near its master, fawning, licking the hand or face, and apparently, seeking sympathy and assistance. Such caresses are, however, extremely dangerous, for the animal's tongue, moist with virulent saliva, coming in contact with a part

where the skin is thin, abraded or wounded, may fatally infect the person to whom it is endeavoring to demonstrate its affection. The reported cases in which rabies have developed from such inoculations are quite numerous.

In most cases dogs first become dull, gloomy, morose, seeking solitude and isolation in out-of-the-way places or retiring under pieces of furniture. But in their retirement they cannot rest, they are uneasy and agitated, they lie down and assume the attitude of repose, but in a few minutes they are up walking about "seeking rest, but finding none." Occasionally this restlessness may disappear for a time and the animal becomes lively and affectionate; oftener it sinks into a sullen gloominess from which even its master's voice rouses it but temporarily. At this period dogs may have aberrations of the senses which cause hallucinations and lead them to think they are being annoyed by something or that some animal or person is endeavoring to injure them. They crouch ready to spring upon the enemy; they rush forward and snap at the air; they throw themselves, howling and furious, against the wall as though they heard sounds beyond it.

While at first the affected dog may not be disposed to bite, it becomes more dangerous as its hallucinations and delirium increase.

Food is taken at first if it is something that can be swallowed without mastication, otherwise it is soon dropped. Difficulty in swallowing is an early symptom. Mad dogs have no fear or dread of water, they continue to drink until paralysis prevents them from swallowing.

When the furious symptoms appear, the dog may leave his home and start upon a long chase with no apparent object in view other than to be traveling. He trots at a rapid pace, eyes haggard and tail depressed. He is indifferent to the surroundings. He often flies at and bites persons whom he meets, but usually he does not search for them or even notice them if they remain quiet. Dogs in this condition may travel many miles and finally drop from exhaustion and die. Often after an absence of a day or two they return to their homes, exhausted and emaciated, presenting a most forlorn and miserable appearance. Those who have pity for such an animal and try to make it clean and comfortable are in great danger of being bitten, as the disease has advanced to a point where the delirium or insanity is most marked and where a treacherous bite is most common.

If the animal, instead of being allowed to escape, is kept confined, the paroxysms of fury are seen to occur intermittently or, in the

absence of provocation, they may be entirely wanting. If excited it howls, rushes upon objects that are thrust toward it or throws itself against the bars of its cage and bites with great fury.

As death approaches the animal becomes exhausted and is scarcely able to stand. The eyes are dull and sunken and the expression is that of pain and despair. Paralysis appears in the jaws or in the posterior extremities and extends rapidly to other parts of the body. The animal, being unable to stand, lies extended upon its side, the respiration becoming more and more difficult. There are spasmodic contractions of certain groups of muscles, complete prostration and finally death.

Dumb rabies. When this form of the disease is typical, it comes on with restlessness, depression, a tendency to lick objects and paralysis of the muscles which close the jaws. As a consequence of the paralysis, the lower jaw drops, the animal is unable to close the mouth, the tongue hangs out and an abundance of saliva escapes. The mucous membrane of the mouth becomes dry, discolored and covered with dust. The animal remains quiet, it does not respond to calls and appears to understand its helplessness. Bouley states that the animal cannot bite and does not desire to bite. When dumb rabies follows the furious form, the desire and tendency to bite may be retained even after the jaw is paralyzed.

The urine usually contains sugar. During the period of excitement the temperature is raised. Later it becomes subnormal. The contraction of the pupil has been reported to be a very constant symptom.

The symptoms in horses and cattle vary but in the main they do not differ materially from those in the dog. There is more tendency to irritate the local wound than in dogs. Cattle are also more liable to rub their nose on the ground or against fences. A great variety of symptoms have been described.

The duration of the disease varies. It may be 4 or 5 days or it may be as short as 2 or as long as 13 days. It is usually shorter in its dumb form.

The prognosis is unfavorable. Most animals die unless they have had prophylactic treatment. Pasteur and others have, however, noted recovery in a few dogs.

Morbid anatomy. One of the striking characteristics of rabies is the absence of constant, recognizable lesions. The mucosa of the pharynx and larynx are congested. In dogs the stomach sometimes contains a variety of foreign matter such as earth, stones, bits of

leather, wood, etc. Axe reports finding such foreign susbstances present in 90 per cent. of 200 cases he examined. Galtier reports such findings in from 50 to 70 per cent. In experimental animals and cattle the writer has rarely found them. The obvious lesions are not constant and it is probable that the pronounced changes occasionally found in a single organ are accidental or secondary rather than primarily related to the disease. The lesions in the brain and spinal cord are likewise variable. In some cases there is a marked hyperemia, while in others the brain appears to be normal.

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 were 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.

In 1887 Babes described the following changes:

"In animals dead from street rabies there are found usually a hypermia and an acute generalized œdema of the cerebral meninges, acute hemorrhages localized around certain vessels, as well as inflammatory lesions. On microscope examinations we find an increase of the plasma cells, augmentation of the reticular substance, fibrinous in character, between the several layers of the meninges.

"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 tunic. 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.

"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.

"The most constant lesions are microscopic in character; they are found more 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.

"The lesion of the nervous elements of the parts indicated is quite characteristic; it consists of 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 lymphatic origin often invade the protoplasm even of the

cell and fill out the dilated pericellular lymphatic spaces by a multiplication of small nuclei.

"The lesion of medullary substance is less pronounced, it consists chiefly of an edema of the medullary sheath of the nerve fibers.

"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 ameboid granules about 1μ in diameter, pigmented or stainable by aniline dyes, and which in part seem to possess the power of movement."

Golgi draws attention to the following morbid changes in rabies:

(a) Changes in the structure of the nucleus, all the various phases of karyokinesis may be simulated, yet no true nuclear division may take place. (b) 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. (c) 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 occur rence and mutual interdependence are taken into consideration.

The morbid process is 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 Germano and Capocianco state that in their researches they have been able to confirm the statements made by Golgi, that instances of the complete disappearance of nerve cells have been observed, while other cells show fatty degeneration, and partial destruction of their entirety representing 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 are noted marked changes in the axis cylinders. In some cases they appear uniformly swollen for their whole length, while in others there are varicose enlargements. In the swollen portions there were frequently observed small vacuoles which interrupted the continuity of the axis cylinder.

In 1900, the discovery of changes distinctive of rabies was announced by Van Gehuchten and Nélis. These were found in the peripheral ganglia of the cerebrospinal and sympathetic systems and are especially marked in the plexiform ganglion of the pneumogastric nerve and the Gasserian ganglion. Normally these ganglia are composed of a supporting tissue holding in its meshes the nerve cells, each one of which is enclosed in a capsule, made up of a single layer of endothelial cells. The action of the rabic virus seems to exercise its effect on these cells particularly, bringing about an abundant multiplication of the cells forming this capsule, leading finally to the complete destruction of the normal ganglion cell and leaving in its place a collection of round cells. Ordinarily a considerable number of ganglion cells will be found which have undergone only a slight change, but under certain conditions the process is so widespread that all the ganglia cells are destroyed. The intensity of these changes varies in different animals; they are perhaps most pronounced in the dog, less marked in man and still less in the rabbit.

Diagnosis. Rabies is diagnosed by the symptoms, presence of Negri bodies, lesions in the ganglia, animal inoculation and by the complement fixation method.

Diagnosis by the presence of Negri bodies. The presence or absence of Negri bodies has come to be used as a means of diagnosis in most if not all laboratories. These bodies, which are often quite large, are readily brought out by proper staining. Whether the cause or a specific degeneration, they are of much value in making a rapid diagnosis. Unlike the ganglion changes they appear early in the course of the disease, and consequently they are of value in making an early diagnosis when the animal is killed soon after the appearance of the first symptoms. Thus far these bodies have not been found in the brains of animals dying from other diseases except one report of their possible presence in a case of tetanus. They are found prior to the appearance of symptoms.

Bohne uses for diagnosis of rabies a piece $\frac{1}{2}$–$\frac{3}{4}$ mm. thick from the Ammons horn which after 30 to 40 minutes of fixing and hardening in acetone for 60 to 75 minutes he puts in paraffin. In this way it is possible for one by the aid of a short staining after the Mann method to get stained sections in the course of three hours. In his 170 investigations (157 dogs, 6 cows, 4 people, 3 cats) he confirmed the later discoveries of Negri and Volpino relative to the presence of the Negri bodies and their finer structure. He examined 50 dogs attacked

with other diseases without finding Negri bodies or similar forms. He therefore holds these bodies as specific for rabies and the diagnosis as assured if they are found.   Later writers agree with this conclusion.

Examination for Negri bodies.   At least four preparations from each of the hippocampi, four from each of the cerebra, and four from the cerebellum, should be carefully examined before pronouncing the case negative.   In making the preparations for examination any one of the following three methods may be employed:

(a) Smear preparations as described by Dr. Williams, of the New York City Board of Health Research Laboratory.

(b) Impression preparations as described by Dr. Frothingham, of Harvard University.

(c) Fixing the tissue and making sections.

Either of the first two methods is preferable to the last, although occasionally the Negri bodies seem to be more clearly differentiated in the section.   Zenker's fluid is recommended for fixing the tissue.   It does not seem to be necessary to fix for more than from four to six hours when the tissues are cut in thin pieces.

The stain to be employed for smear or impression preparations should be either Van Giessen's, or eosin and methylene blue.   If the tissue is fixed in acetone and sectioned, the staining method recommended by Mann or that proposed by Lentz gives excellent results.   If the tissue is fixed in Zenker's fluid, eosin and methylene blue should be used.

Diagnosis by histological examination of the ganglia (specific tissue changes).   The rapid diagnosis by means of the histological changes pointed out by Van Gehuchten and Nélis has been very successful in the experience of a number of workers.

We have found the plexiform ganglion, which is situated just outside of the cranial cavity near the *foramen lacerum basis cranii*, on the pneumogastric nerve, the most convenient and the most desirable for study.   The removal of this ganglion is comparatively easy and simple.

There are two ways by which this ganglion can be easily found:

Take up the pneumogastric nerve and trace it anteriorly to the point where it enters the cranium.   Near this point a slight enlargement, the ganglion of the trunk of the vagus, will be found.

Cut through the skin from the mandibular symphysis posteriorly along the neck and reflect it back.   An incision is then made through the mylohyoid muscle near the inner face of the mandible posteriorly past the digastric muscle and superiorly until the lingual nerve going to the tongue is exposed.   Trace this posteriorly until the point where it enters the cranium together with the vagus is reached.   In this way it is easy to locate the vagus nerve and the plexiform ganglion.   We have found either one of these methods or a combination of the two very convenient, and with a knowledge of the location of these parts there is no reason why the ganglion should not be removed quickly and easily.

After the ganglion is removed there are a variety of methods which may be used in fixation and staining.   The following we have found to be very satisfactory.   As soon

as the ganglion is removed it is placed in Flemming's fluid or in a standard aqueous solution of mercuric chloride for a few hours, washed in water, carried through the alcohols and sectioned by the paraffin method. With this method of fixation it is almost imperative that the sections be stained with iron or Delafield's hematoxylin, of which we have found the latter the most convenient. Alcohol, either 95 per cent. or absolute, may be used as a fixer, in which case other staining methods may be used. However, the fixation by this method is not as good, but it admits of a trifle more haste.

Normally this ganglion is composed of a fibrous capsule from which a supporting fibrous tissue extends into the interior, holding in its meshes the nerve cells, each of which is enclosed in an endothelial capsule. The changes characteristic of rabies consist in the atrophy, the invasion and the destruction of the ganglion cell as a result of new formed cells, evidently from the endothelial capsule. These cells appear first between the nerve cell and its capsule. These changes are quite uniform through the entire ganglion and in advanced cases of the disease nearly all of the nerve cells are oftentimes destroyed.

The fact must be kept in mind that this is a method for rapid diagnosis in case the animal dies and not a means for an early diagnosis. This method is not trustworthy if the animal was killed in the early stages of the disease as the changes do not appear until later in its course.

Diagnosis by animal inoculation. The method experience has shown to be the best is the subdural inoculation of rabbits or guinea-pigs 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 injection.

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 sterile water or bouillon. This forms the suspension to be injected.

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 anesthetic* has passed off

*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.

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 inocula-
tion 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.

The symptoms following the inoculation are quite uniform.   There is, however, a
marked difference in the length of time the rabbits live 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 hyperesthesia.   Usually the first indication
of the disease is a partial paralysis of one or both hind limbs.   This gradually advances
until the rabbit is completely prostrated, the only evidence of life being a slight respira-
tory movement.   The head occupies different positions.   In some it is drawn back-
ward as in tetanus;   in others it is drawn down with the nose near the fore legs;   and in
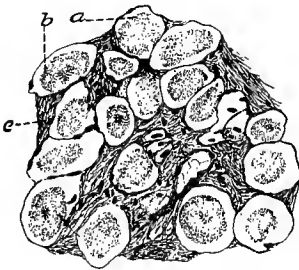
FIG. 90.   SECTION OF A NORMAL
PLEXIFORM GANGLION (a), AND
(b) GANGLION CELLS, (c) INTER-
CELLULAR SUBSTANCE.
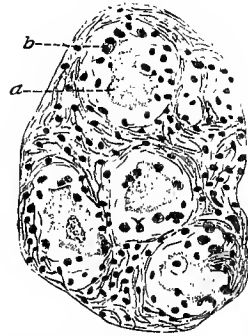
FIG. 91.   SECTION OF PLEXIFORM
GANGLION FROM A CASE OF
RABIES;   (a) GANGLION CELL,
(b) CELLS INFILTRATING THE
GANGLION CELL AND SPACE.

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 does not exceed twenty-
four hours.   Although these animals are unable to move voluntarily, there is a reflex
action of the limbs until a very short time before death.

During the period of incubation the temperature of the rabbit is normal.   As the
time approaches for the first symptoms to appear there is an elevation of temperature
of from 1 to 2 degrees, which continues for a variable length of time, but rarely longer
than two days.   This is followed by a gradual or usually a more rapid drop to the sub-
normal, which continues to the end.

This method of diagnosing rabies requires that the inoculated animals remain ap-
parently well for a considerable length of time after the subdural inoculation and before
the paralytic symptoms appear.   If the death is 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 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.

Animals other than rabbits have been used and a number of other methods of inoculation have been proposed.

Diagnosis by complement fixation method. Zell applied the complement fixation test to rabies, following the Wasserman technique. He used for an antigen a preparation of the salivary glands following the method of Poor and Steinhardt.*

The serum of animals infected with street rabies was found by Zell to give a positive reaction, usually several days before clinical symptoms appeared. He points out that by this method it is possible to make diagnoses without destroying the suspected animal and therefore that it furnishes a test after the completion of the Pasteur treatment to determine whether or not immune bodies are present in the patient's serum. In a later publication he recommends the use of the serum of the immunized animal as a prophylactic agent. He considers the serum from the immunized animal as efficient a prophylactic as tetanus antitoxin.

The employment of sera has not been generally accepted as a means of diagnosis, but Zell's report suggests that it may be of much value.

Differential diagnosis. Rabies is to be differentiated from morbid conditions caused by certain parasites, paralytic forms of other diseases, and disorders manifested by nervous symptoms such as meningitis. Besson states that dogs are susceptible to *Ps. pyocyaneus* which develops symptoms resembling those of rabies.

Prevention. The only means of preventing rabies after infection has taken place is to employ some one of the prophylactic treatments. The method formulated by Pasteur has proven to be most successful in the past. The serum immunizing method has been recently reported to be giving satisfactory results.

"The Pasteur method of treatment is based upon the fact that the rabic virus in the spinal cord of rabbits loses strength at a fairly regular and even rate when the cord is removed from the body after death and carefully dried. In the preparation of material for the preventive treatment rabbits are inoculated with "fixed virus"—a term given

---

*The method consists first, in the application of collodion sacs for the dialysis of glycerin. Second, the use of aspiration for obtaining the virus of rabies from the submaxillary glands of rabid dogs. This aspiration extract is virulent when filtered through the Berkefeld filter.

by Pasteur to virus that is so exalted in virulence by successive passages through rabbits that it will produce the death of these animals in six or seven days. Beyond this point no increase of virulence can be obtained; hence the name, fixed virus. The spinal cord is removed aseptically from rabbits killed by the inoculation of fixed virus, cut into three pieces and suspended over a solution of caustic potash in a drying chamber. Here the cords are kept in the dark and at a constant temperature of 23° C. for fourteen days. Emulsions of the dried cord are prepared in sterile salt solution or broth and injected every day, or sometimes more frequently, during a period of fifteen to twenty-one days, the interspacing of doses and duration of treatment being determined by the nature of the case. As a rule, the most attenuated material (fourteen-day cord) is injected first, and this is followed by virus of gradually increasing strength. The method is essentially one of active immunization, and involves a race between the action of the attenuated virus and the virulent virus introduced by the bite of a rabid animal. It follows that the preventive treatment must always be begun at the earliest possible moment after the bite. In a certain proportion of cases a spinal cord lesion seems to follow the treatment.

"The serum of animals immunized against hydrophobia possesses considerable protective power, and according to some investigators, has also a marked curative effect. Favorable results have been reported from the use of immune serum, especially in cases of severe bites about the head, or in persons who have delayed beginning the Pasteur treatment.

"Högyer produced immunity in dogs by injecting them subcutaneously with emulsions of virulent spinal cords in normal salt solution in high dilutions. Galtier immunized sheep and goats by injecting saliva and also medulla oblongata into the jugular vein. Babes injected sheep subcutaneously with normal cerebral tissue and found them resistant to subsequent infection with virulent cord."

Semple's antirabic vaccine. This method of anti-rabic vaccination was introduced by Lieutenant-Colonel Sir D. Semple. An eight per cent. dilution of rabies virus is made in normal saline, and one per cent. carbolic acid is added; the resulting mixture is then kept at a temperature of 37° C. for twenty-four hours. By this means the virus is killed. The mixture, diluted with an equal bulk of saline solution, gives a vaccine containing four per cent. virus (dead) and 0.5 per cent. carbolic acid. This conferred a high degree of immunity in monkeys, dogs, and rabbits, and the serum obtained from these animals had a well-marked rabicidal action on living virulent virus. Its efficacy was apparently as great as that obtained by the use of living virus, and it retains its power for some time."

Eradication. The eradication of rabies infection resolves itself into two procedures. (a) The destruction of all ownerless and vagrant dogs, and (b) the muzzling of all dogs that appear upon the streets or in public places. In thus preventing the propagation of the virus, as shown by the results obtained in Germany and Great Britain, the disease will be practically exterminated.

REFERENCES

1. BABES. Sur certains caractères des lesions histologiques de la rage. *Ann. de 'l Institut Pasteur*, Vol. VI (1892), p. 209.

2. BABES. Untersuchungen über die Negrischen Körper und ihre Beziehung zu dem Virus der Wutkrankheit. *Zeitschr. f. Hygiene*, Bd. LVI (1907), S. 435.

3. BOHNE. Beitrag zur diagnostischen Verwerthbarkeit der Negrischen Körperchen. *Zeitschr. f. Hygiene*, Bd. LII (1905–6), S. 87.

4. FLEMING. Rabies and hydrophobia.

5. LAW. Rabies. *A System of Practical Medicine by American Authors*, Vol. III (1898).

6. MARTIN. Rabies in India. *Journ. of Comp. Path. and Therap.*, Vol. VIII (1895), p. 207.

7. MOHLER. Pathological report on a case of rabies in a woman. *Annual Report, U. S. Bureau of Animal Industry*, 1903, p. 54.

8. MOORE AND FISH. A report on rabies in Washington, D. C. *Annual Report, U. S. Bureau of Animal Industry*, 1895–6, p. 267.

9. MOORE AND WAY. A rapid method for the diagnosis of rabies. *American Veterinary Review*, Vol. XXVIII (1904), p. 658.

10. NEGRI. Beitrag zum Studium der Aetiologie der Tollwnth. *Zeit. f. Hygiene*, Bd. XLIII (1903), S. 507.

11. POOR AND STEINHARDT. Two methods for obtaining a virus of rabies free from the cells of the host, etc. *Journ. of Infect. Dis.*, Vol. XII (1913), p. 202.

12. PUBLIC HEALTH COMMISSION, DISTRICT OF COLUMBIA. Rabies. *Bul. 25, U. S. Bureau of Animal Industry*, 1900.

13. RAVENEL. Rabies. *Bul. 79, Dept. of Agr., State of Penn.*, 1901.

14. RAVENEL AND MCCARTHY. The rapid diagnosis of rabies. *Univ. of Penn. Med. Magazine*, 1901.

15. REMLINGER. Le passage du virus rabique à travers les filtres. *Ann. de l' Inst. Pasteur*, Vol. XVII (1903), p. 834.

16. REMLINGER ET RIFFAT-BEY. Sur la perméahilité de la bougie Berkefeld au virus rahique. *C. R. Soc, de Biol.*, Vol. LV (1903), p. 974.

17. SALMON. Rabies, its cause, frequency and treatment. *Year Book, Dept. of Agriculture*, Washington, D. C. 1900, p. 122.

18. SALMON. Rabies in the District of Columbia. *Circular No. 30, U. S. Bureau of Animal Industry*, 1900.

19. SCHÜDER. Der Negrische Erreger der Tollwuth. *Deut. Med. Wochenschrift*, 1903, S. 700.

20. SUZOR. Hydrophobia. An account of M. Pasteur's system. 1887.

21. VAN GEHUCHTEN AND NÉLIS. Diagnostic histologique de la rage. *Annales de Méd. Vét.*, Vol. XLIX (1900), p. 243.

22. WAY. The Negri bodies and the diagnosis of rabies. *Amer. Vet. Review*, Vol. XXIX (1905), p. 937.

23. WILLIAMS AND LOWDEN. The etiology and diagnosis of hydrophobia. *Jour. of Infectious Diseases*, Vol. III (1906), p. 452.

24. WILLIAMS AND LOWDEN. The etiology and diagnosis of hydrophobia. *Studies from the Research Lab., Dept. of Health*, N. Y. City. Vol. II (1906), p. 13.

25. ZELL. Sero-diagnosis of rabies. *Am. Journ. of Vet. Med.*, Vol. VIII (1913), p. 637.

26. ZELL. Rabies, diagnosis and treatment. *Am. Journ. of Vet. Med.*, Vol. X (1915), p. 835.

CHAPTER XIII

INFECTIOUS DISEASES FOR WHICH THE SPECIFIC CAUSE IS NOT DETERMINED

General consideration. The study of microbiology has revealed the specific cause of a large number of the infectious diseases but there still remain several well recognized maladies for which specific micro-organisms have not been demonstrated. The methods that have been effective in finding the cause of other specific diseases have failed here. Their study, however, has revealed much concerning the nature of the virus and its location in the body of the infected animal, so that measures for their prevention have been formulated which are quite as efficient as they are for the diseases of known etiology. The viruses of this group of diseases are spoken of as ultramicroscopic. As many of them will pass through filters that hold back known bacteria they are often called "filterable viruses."

RINDERPEST

Synonyms: Contagious typhus; steppe murrain; cattle plague.

Characterization. Rinderpest is the most fatal disease affecting cattle. It is a specific eruptive fever, occurring both sporadically and in epizoötics. It is characterized by a more or less typhoid condition, with lesions largely located in the mucosa of the digestive tract and skin, and by the infectious nature of all the tissues, secreta and excreta. It is a disease peculiar to cattle, although other ruminants are suscep-tible to it.

History. Rinderpest seems to have been brought to western Europe by the importation of cattle from central Asia. It is supposed that it had long existed on the steppes of central Asia and eastern Europe. The first great epizoötic, of which there seem to be records, occurred about 1709 and spread over nearly all of the countries of Europe. It is reported that 1,500,000 cattle died from its effects during the years from 1711 to 1714. Ramazzini seems to have been the first (1711) to give a description of the symptoms and lesions.

404

It was in connection with this disease that the first veterinary police regulations were instituted, and it is stated that because of the ravages of this affection Veterinary Colleges were first established with government aid. In the latter half of the eighteenth century, rinderpest was prevalent in nearly all of the countries of Europe. During the years from 1740 to 1750 it was estimated that three million cattle died. In Italy alone, during the year 1792, from three to four million cattle are reported to have died from its effects. Nearly all the countries of Europe have from time to time lost heavily from it.

Geographical distribution. Rinderpest is a well-known cattle plague in Russia and the steppes of central Asia. It has extended from time to time from its home in Russia and Asia to nearly every country in Continental Europe and Asia. More recently it has occurred in southern Africa. In 1882,* it appears to have been introduced into the Philippine Islands. It has not appeared in the United States or other American countries.

Etiology. The specific etiological factor of rinderpest is not known. Koch makes the following statement concerning the etiology of rinderpest in the second report of his investigations in South Africa in 1897: "All efforts to find by means of the microscope, as well as through cultivation, a specific microörganism in the blood have as yet been fruitless. I also did not succeed in finding any specific microörganism amongst the microbes which the mucus from the nose, the secretions from mucous membranes, and the contents of the intestines naturally contain in large numbers." Jobling reports that 0.1 cc. of blood taken from a sick animal and injected under the skin of a healthy one will produce the disease.

Nicoll and Adil-Bey found that the virus would usually pass through the porous Berkefeld cylinders, but not through the denser form or the Chamberland bougie.

The virus may be transmitted from the sick to the healthy individuals in a variety of ways, both direct and indirect. It is said to be present in the various excreta of the diseased animal, such as the discharge from the nose, the saliva, the urine and the feces.

Ward, Wood and Boynton found the virus not to survive beyond 24 hours in corrals bare of vegetation but containing water. Animals became infected in such corrals seventeen and one-half hours after removal of the sick. They found that infected animals were capable

---

*No authoritative evidence of its occurrence there prior to that time has been found.

of transmitting the disease to susceptible ones in close contact with them only during the febrile period and most certainly during the time the temperature was declining. The infection was not transmitted during the convalescent stage. The virus in the urine, diluted with water and sprinkled on grass, survived not longer than 36 hours. They did not find any recovered cases that transmitted the virus.

Persons may carry the virus on their shoes, clothing and farm implements. Even small animals such as cats and rats, which frequent barns and stables, have been looked upon as carriers of the infection. The hides of animals dead of the disease may transmit the infection. The virus is destroyed, according to Hutcheon, by complete desiccation. Kraiewsky found that the virus was destroyed in hides soaked in corrosive sublimate 1–1000 for 24 hours, or in 2.4 per cent. carbolic acid for the same time.

Boynton found that the large water leech (*Hirudo Boyntoni* Wharton) can retain the virus of rinderpest alive in its body for at least twenty-five days in a virulent condition. The large leech held the virus alive much longer than the smaller ones. His studies of the leech in connection with the transmission of rinderpest have shown very conclusively that the leech is a factor in the propagation and spread of rinderpest.

The period of incubation varies from 3 to 6 days. In animals inoculated with virulent blood it is from 60 to 90 hours. Boynton found that the period of incubation in cattle infected with virus from a leech was 10 days.

Symptoms. The first symptom of rinderpest is a rise of temperature* (105° F. to 106° F.) which continues near that point with but slight variations until near the end when in fatal cases it drops to subnormal and in those that recover it gradually returns to normal. At first the animal is more lively than normal, showing evidence of being under a nervous strain. This is stated by Boynton to be more prevalent in cattle than in carabaos. About the third day the nervousness begins to disappear, the hair is roughened, the ears lop forward and the conjunctiva is congested. The eyes become sensitive to light. Usually there is a profuse discharge of tears from the inner canthus. Later this may become sero-purulent. The pulse is small, beating from 120 to 160 per minute. There is debility, decrease in

---

*Boynton reports a case of rinderpest without a rise of temperature.

the yield of milk and loss of appetite; rumination becomes disturbed and the animal may have slight attacks of shivering.

After these preliminary symptoms, respiration becomes accelerated and the visible mucous membranes assume a scarlet color. There is thirst, loss of appetite, and rumination ceases. The nostrils become congested and often discharge a sero-purulent substance. The muzzle is usually dry and hot.

The feces gradually become thinner until violent diarrhea accompanied by colic sets in. The evacuations become fetid, viscid and sometimes mixed with blood. There may be straining and groans due to pain in defecating. The sphincter muscle loses its tension and becomes paralyzed. The animal becomes rapidly emaciated, staggers when walking, is very sensitive to pressure on the loins and lies down a great deal. In exceptional cases the patient manifests nervous symptoms. Others exhibit violent dyspnea and symptoms of severe inflammation of the lungs.

As the disease advances characteristic changes are to be seen in the mucous membranes. Red patches which may be flat or in the form of wheals and which quickly become covered with a grayish white, loose crust appear on the mucous membrane of the lips, tongue, cheeks, gums, nostrils and vagina. The uppermost epithelial layer consequently becomes opaque and yellowish gray spots develop on it. Less frequently crusts are formed from the tumors by their caseous disintegration. The crusts on being shed leave dark red hollow places, the so-called erosion ulcers, which bleed readily. In slight cases of the disease there may be no crusts or erosions. It is stated that sometimes an eruption in the form of very minute pimples and pustules occurs on the abdomen, inner surface of the thighs, perineum and udder. In these cases it may be confused with that of variola. Pregnant animals frequently abort. In the later stages the animal lies down most of the time.

Boynton has succeeded in keeping the virus alive on artificial media for some generations. His full report has not been published.

In sheep and goats the disease is milder, and its infectiousness is said to be less than in cattle, although the symptoms are essentially the same.

The duration of the disease varies, in fatal cases, from 3 to 10 days after the rise of temperature. Boynton states that the average duration in the Philippines is about 6 days. Occasionally there is a relapse

after the animal seems to be improving. These cases usually die in from 3 to 4 weeks.

The prognosis is not favorable. *The mortality* ranges from 60 to 90 per cent.

Morbid anatomy. Authors differ somewhat on the lesions characteristic of this disease. Walley has pointed out the fact that none of the morbid changes are constant and consequently they vary with the stages of the epizoötic and the condition of the animal. There is always emaciation, the muscles are dark and the capillary congestion is marked. All the tissues of the body may be the seat of effusions, exudations and blood extravasations. If symptoms referable to the nervous system have occurred, brain lesions will be found at postmortem.

The muscle of the heart is pale and relaxed. Blood extravasations may be found beneath the pericardium especially along its coronary grooves. The kidneys are usually the seat of congestion or ecchymoses either beneath the capsule or in the cortical structure. There are also parenchymatous changes. The liver is congested, often giving a mottled bile stained appearance. The lymphatic glands are usually enlarged and the mesenteric glands are often hyperemic or even hemorrhagic.

The structures most often affected are the mucous membranes of the digestive, respiratory and genito-urinary tracts and the skin.

Nodules and pustules are sometimes found on the skin, especially of the udder and abdomen. The mucous membrane of the mouth and pharynx is congested in spots, swollen and exhibits rounded, yellowish gray, caseous plates or deposits. The removal of plates discloses erosions and highly congested depressions in the mucous membrane, the so-called erosion ulcers. These changes are most marked on the inner surface of the lips, palate, walls of the pharynx and base of the tongue. Congestion and erosions are sometimes found in both the trachea and esophagus.

In the first three stomachs the mucous membrane is usually normal or slightly congested in spots, and the epithelium is so loose that it can be easily detached. The contents of the rumen and reticulum are soft and those of the omasum are often dried, rarely they are fluid. Generally the abomasum is empty or contains a small quantity of foul smelling liquid and mucus or sanious material. Its mucous membrane is highly congested, especially in the neighborhood of the pyloric orifice. The congestion is partly diffuse, partly in spots, in the form

of points or streaks. Erosion ulcers are quite constant. They are more numerous when the congestion is marked. They occur most frequently on the edge of the folds of the mucous membrane.

The glands (peptic and mucous) of the stomach are swollen and show considerable cellular hypertrophy. Like changes are present in the small intestine, where there is a good deal of swelling and congestion of the mucous membrane with isolated scab-like caseous deposits and erosions. In very severe cases these deposits form tube-like casts of the intestinal canal. At the same time there may be considerable infiltration of the solitary glands and of Peyer's patches, which become enlarged. Jobling states that he never saw them ulcerated. Frequently the walls of the stomach and duodenum are swollen and edematous.

In the large intestine the inflammatory changes are much less pronounced. The cecum is usually congested and often hemorrhages are present. Congestion and erosions may be found about the ileocecal valve. The congestion occurs in blotches or in streaks and may extend throughout the large intestine. Boynton states that peritonitis is constantly present, the exudate usually being of a fibrous nature. It is more prevalent over the region of the small intestines and fourth stomach.

The nasal mucous membrane is of a dark red color and covered with grayish-yellow, soft scabs. After they are removed, the true tissues of the mucous membrane lie bare. Similar changes are found in the larynx and trachea, where the deposited masses are frequently purulent and of a creamy consistence. The lungs are sometimes hyperemic, sometimes edematous, hepatized or emphysematous. Pneumothorax and subcutaneous emphysema may be present.

Diagnosis. It is very difficult to diagnose rinderpest from the first cases that occur, especially if there is no history of infection. The diagnosis is based upon the symptoms, morbid anatomy, progress of the epizoötic and the history. The most characteristic diagnostic symptoms are the rise in temperature (which often occurs some days before other symptoms), formation of red spots and a yellow coating on the visible mucous membranes and later the development of erosion ulcers. A mucous discharge from the mouth, nares, eyes and vagina with symptoms of severe intestinal disturbances and excessive emaciation are of diagnostic value.

Rinderpest is to be differentiated from "foot-and-mouth-disease," and malignant catarrh if complicated with emphysema of the lungs.

Anthrax, Texas fever, and other affections such as contagious pleuro-pneumonia and enteritis may be mistaken for it.   The differentiation may be made from the specific nature of each disease.

Immunizing cattle.   Koch found that the serum of an animal that had suffered from rinderpest and recovered possessed immunizing powers.   This fact being demonstrated, its utility has been availed of in immunizing animals for procuring the serum.

Ward and Wood have carefully tested the immunity induced by serum in the Philippine Islands where it was extensively tried.   Their conclusions were as follows: "The experiments seem to show that antirinderpest serum does not prevent infection with rinderpest.   On the contrary, animals injected with serum and exposed to rinderpest soon contract the disease and pass through a more or less modified attack.   We have shown that the blood of animals is infective during this attack.   If by passive immunity is meant an artificial condition by means of which the severity of an attack is lessened, we grant that such exists, but deny that there is a passive immunity of a kind that prevents invasion by the virus of rinderpest."

Control.   The simultaneous method formerly employed extensively seems to be less successful.   It does not seem to be used regularly except in Russia.   Theiler points out the danger from the various blood parasites that may be transmitted by this process.

Arloing, in describing its use in Egypt, states that simultaneous inoculation had to be abandoned in Egypt because a panic took possession of the cattle owners.

In India the application of simultaneous inoculation to a number of susceptible Australian cattle led to disastrous results.   A committee of investigation brought out the fact that this method is not generally employed there outside of laboratories.   They recommended that as an act of grace the cattle owners be compensated for the loss of their stock.

Writers treating of rinderpest in Africa attribute to serum the property of conferring a lengthy passive immunity to natural infections, a belief that must be taken into consideration in estimating the part played by serum in its eradication.

In the Philippines serum was formerly undoubtedly used with the idea that it would absolutely protect.   However, quarantine and general sanitation were enforced to some degree.

REFERENCES

1. BOYNTON. An atypical case of rinderpest in a carabao. *Bull. No. 31, Bur. of Agric. P. I.*, 1914.

2. BOYNTON. Symptoms and lesions presented by cattle and carabaos suffering from rinderpest in the Philippine Islands. *The Philippine Agricultural Review*, Vol. V (1912), p. 644.

3. BOYNTON AND WARD. Duration of the infectiveness of virulent rinderpest blood in the water leech, Hirudo Boyntoni Wharton. *Bulletin 29, Bur. of Agric. P. I.*, 1914.

4. DANYSZ, BORDET AND THEILER. *The Vet. Journal*, Vol. XLVI (1898), p. 298.

5. GAMGEE. The cattle plague. London, 1866.

6. HEAD. Cattle plague in the Anglo-Egyptian Sudan. *Journ. of Comp. Path. and Therap.*, Vol. XIX (1906), p. 12.

7. HOLMES. Some diseases complicating rinderpest among cattle of India. *Jour. Comp. Path. and Therap.*, Vol. XVII (1904), p. 317.

8. HUTCHEON. Rinderpest in South Africa. *Journ. of Comp. Path. and Therap.*, Vol. XV (1902), p. 300.

9. HUTCHEON, EDINGTON, KOLLE AND TURNER. Report of rinderpest investigation. *The Veterinary Journal*, Vol. XLVI (1898), p. 64.

10. JOBLING. Report of the director of the serum laboratory. *Fourth annual report of the Philippine Islands*. 1903. Ibid. *Bulletin No. 4, Bureau of Government Laboratories*. Manila, 1903.

11. KOCH. Report. *The Veterinary Journal*, Vol. XLV (1897), p. 462.

12. KOLLE UND TURNER. Über Schutzimpfungen und Heilserum bei Rinderpest. *Zeit. f. Hygiene*, Bd. XXIX (1898), S. 309.

13. LINGARD. Report on the preparation of rinderpest protective serum. Calcutta, Office of the Supt. of Printing, India, 1904.

14. LITTLEWOOD. Cattle plague in Egypt in 1903–04–05. *Journ. Comp. Path. and Therap.*, Vol. XVIII (1905), p. 312.

15. RAMAZZINI. Dissertatio de Contagiosa Epidemica. Padua, 1711.

16. REFIK-BEY AND REFIK-BEY. La peste bovine en Turque. *Ann. de l'Inst. Pasteur*, Vol. XIII (1899), p. 596.

17. RUEDIGER. Observations on cattle plague in the Philippine Islands and the methods employed in combating it. *Philip. Journ. of Science*, Vol. IV (1909), p. 381.

18. RUEDIGER. The difference in susceptibility to cattle plague encountered among cattle and carabao. *Philip. Journ. of Science*, Vol. IV (1909), p. 425.

19. THEILER. Symptoms and pathological changes observed in rinderpest complicated with redwater. *Transvaal Dept. of Agric. Ann. Report*, 1903-4, p. 169.

20. TODD AND WHITE. Experiments on cattle plague. *Government Press*, Cairo, Egypt, 1914.

21. TURNER AND KOLLE. Report of investigations. *The Vet. Jour.*, Vol. XLV (1897), p. 462.

22. WALKER. The prophylactic treatment of rinderpest by means of preventive inoculation, more especially considered in regard to the condition prevailing in India. *Journ. of Comp. Path. and Therap.*, Vol. XVII (1904), p. 326.

23. WALLEY. The four bovine scourges. London, 1879.

24. WARD. The rinderpest problem. *Philip. Agric. Review*, Vol. IV (1911).

25. WARD AND WOOD. Experiments on the efficiency of antirinderpest serum. *Bull. No. 19, Bur. of Agric. P. I.*, 1912.

26. WARD, WOOD AND BOYNTON. Experiments upon the transmission of rinderpest. *Bull. No. 30, Bur. of Agric., P. I.* 1914.

CONTAGIOUS PLEURO-PNEUMONIA IN CATTLE

Synonyms: Lung plague; pleuro-pneumonia zymotica; *peripneumonie contagieuse; Lungenseuche der Rinder.*

Characterization. Contagious pleuro-pneumonia of cattle is a specific epizoötic disease which affects bovine animals and from which other species are exempt. When the disease results from exposure in the usual manner, it is characterized by an exudative inflammation. of the lungs, especially of the interlobular lymph vessels and pleura, which is generally extensive and which has a tendency to invade portions of these organs not primarily affected and to cause death of the diseased portion of the lung. It is reported that range cattle resist infection more than high bred stable kept animals.

History. It is stated that contagious pleuro-pneumonia was first observed in Hesse in 1693, and that its earliest appearance as an epizootic was in 1713 and 1714, when it prevailed chiefly in Switzerland and the neighboring countries of Würtemberg, Balden and Alsace. In 1773, Haller published an essay on this disease. He described its symptoms, anatomical characters and the protective measures laid down by the sanitary police. In 1735, the disease appeared in England and in 1765 in France. It was described in detail that year by Bourgelat. From 1790 it spread over the whole of Germany, France and Italy. At the commencement of the nineteenth century it visited all the countries of Western Europe.

South Africa was infected by a bull brought from Holland in 1854, and Australia received the contagion with an English cow in 1858. It is also reported as existing in various parts of the continent of Asia; but the time of its first appearance and the extent of its distribution are very uncertain.

Some countries which have been infected for only a short time, such as Norway, Sweden and Denmark, have succeeded in eradicating the disease without much difficulty by slaughtering all affected and exposed animals. Through energetic efforts it has been eradicated from a large part of Europe.

Contagious pleuro-pneumonia has been brought to the United States several times. Its first introduction was in a diseased cow sold in Brooklyn, N. Y., in 1843. It was brought to New Jersey by importing affected animals in 1847. Massachusetts was infected in the same way in 1859. Massachusetts eradicated pleuro-pneumonia during the period from 1860 to 1866. New York and New Jersey

made an attempt to eradicate it in 1879 but were not successful. Late in 1883 the contagion was carried to Ohio, probably by cattle purchased in the vicinity of Baltimore, Md., to which place it had extended previous to 1868. From the herd then infected it was spread by the sale of cattle during 1884 to a limited number of herds in Illinois, to one herd in Missouri and two herds in Kentucky. By coöperation between the United States Department of Agriculture and the authorities of the states, it was found possible to prevent its further spread and to completely eradicate it after a few months.*

In 1886, pleuro-pneumonia was discovered in some of the large distillery stables of Chicago and among cows on neighboring lots. This led to renewed efforts to secure its eradication. Congress, in 1887, enlarged the appropriation available for this purpose and gave more extended authority to the Bureau of Animal Industry. During the same year the disease was stamped out of Chicago and has not since appeared in any district west of the Alleghany Mountains.

The work of eradication was at the same time commenced in all of the infected States. Before the end of the year 1889 Pennsylvania, Delaware, Maryland, the District of Columbia, and Virginia had been freed from the disease. More difficulties, however, were encountered in the States of New York and New Jersey on account of the larger territory infected and the density of the population. The last animal in which the disease appeared in the State of New York was slaughtered early in 1891 and the last one affected in New Jersey met the same fate early in the spring of 1892.

On the 26th day of September, 1892, the following proclamation was issued, declaring the United States to be free from this disease.

PROCLAMATION—ERADICATION OF PLEURO-PNEUMONIA

U. S. Department of Agriculture,
Office of the Secretary.

To all whom it may concern:

Notice is hereby given that the quarantines heretofore existing in the counties of Kings and Queens, State of New York, and the counties of Essex and Hudson, State of New Jersey, for the suppression of contagious pleuro-pneumonia among cattle, are this day removed.

The removal of the aforesaid quarantines completes the dissolving of all quarantines established by this Department in the several sections of the United States for the suppression of the above-named disease.

---

*It was the presence of this disease with its menace to the cattle industry of the United States that led Dr. D. E. Salmon to urge the establishment of the Bureau of Animal Industry in 1884.

No case of this disease has occurred in the state of Illinois since December 29, 1887, a period of more than four years and eight months.

No case has occurred in the state of Pennsylvania since September 29, 1888, a period of four years within a few days.

No case has occurred in the state of Maryland since September 18, 1889, a period of three years.

No case has occurred in the state of New York since April 30, 1891, a period of more than one year and four months.

No case has occurred in the state of New Jersey since March 25, 1892, a period of six months, and no case has occurred in any other portion of the United States within the past five years.

I do therefore hereby officially declare that the United States is free from the disease known as contagious pleuro-pneumonia.

J. M. RUSK, *Secretary.*

Done at the City of Washington, D. C., this 26th day of September, A.D., 1892.

The time required for its eradication was about five years and the total expenditure was a little over $1,500,000.

Etiology. All attempts to discover the etiology of contagious pleuro-pneumonia failed until 1898 when Nocard and Roux succeeded in obtaining a very feeble growth of an exceedingly minute organism in bouillon, containing cow or rabbit serum in proportion of one part serum to 25 parts bouillon, when cultivated in collodion sacs within the abdominal cavity of rabbits. The rabbits that received the inoculated capsules became emaciated, and some of them died. Those receiving the uninoculated capsules remained well. With the cultures obtained in the collodion sacs the disease was produced in cattle. The virus passed through a Berkefeld filter and Chamberland F-candle but it was held back by a Chamberland filter B. Under a magnification of 1500 diameters and strong light the "bodies" appear as minute refracting dots. They are said to appear in several shapes, some of which appeared to be branched. The virus is present in the affected lung tissue, pleural exudate, lymph glands, bronchial secretions and nasal discharges. The virus is transmitted by direct contact. Stables in which diseased animals are kept remain infectious for a long time. Affected animals transmit the disease during all stages but more surely during the period of acute symptoms. Recovered animals are reported by Walley to transmit the disease for 15 months and Minette traced outbreaks in herds of cattle introduced two and three years previously. The virus is supposed to enter the body through the respiratory tract and develops first in the lymph spaces of the interlobular tissue.

The period of incubation is, after the subcutaneous injection of the virus, from 6 to 27 days and after inhaling it from 12 to 16 days, according to Nocard and Roux.

Symptoms.   The symptoms are such as would be expected with inflammation of the lungs and pleura, but they vary considerably according to the course which the disease runs.   If the attack is an acute one, the symptoms appear suddenly.   The breathing becomes rapid and difficult, the animal grunts or moans with each expiration, the shoulders stand out from the chest, the head is extended on the neck, the back is arched, the temperature ranges from 104 to 107° F., the milk secretion is suspended, there is loss of appetite, rumination is stopped, the animal may bloat and later be affected with a severe diarrhea.

Very often the attack comes on slowly and the symptoms are more obscure.   In the milder cases there is a cough for a week or two but no appreciable loss of appetite or elevation of temperature.   The lungs are but slightly affected and recovery soon follows.   Such animals may disseminate the virus for a long time without being suspected and for that reason are very dangerous.

In more severe cases the cough is frequently more or less painful, the back somewhat arched and the milk secretion diminished.   The inflammation of the lung does not, as a rule, subside and the organ does not return to normal condition as in ordinary pneumonia, but with this disease the affected tissue dies and a fibrous wall is formed around it.   These sequestra may later break and discharge into bronchi, thereby eliminating the virus.   The tissue, thus encysted, gradually softens and becomes disintegrated into a purulent-like substance.   The recovery, therefore, is only partial.

The urine is dark in color and acid in reaction.   It is passed at long intervals.

Seriously affected animals remain standing if they have sufficient strength, but those which lie down are said to always lie on the affected side.

The proportion of animals that become affected after being exposed varies according to the virulence, the susceptibility of the animals and the length of time during which exposure is continued.   Sometimes not over 15, 20 or 30 per cent. of the animals exposed will contract the disease, while at other times 80 or 90 per cent. may be infected. Some of the earlier writers supposed about one animal in four was immune.   The mortality may not exceed 10 per cent. and it may

reach 50 per cent. In general it may be said that about 40 per cent. of the exposed animals will contract the disease and about one-half of these will prove fatal. Pregnant cows usually abort.

The duration of the disease in acute cases is usually from 7 to 20 days. In chronic ones the time of death or recovery is uncertain.

Morbid anatomy. There is a progressive interstitial pneumonia with secondary hepatization of the lungs and exudative pleuritis. Usually only one lung, the left, is affected. The anatomical changes vary according to the duration of the disease.

The otherwise healthy lung shows, in the initial stage, small, circumscribed, inflammatory centers from the size of a hazelnut to that of a walnut. The interlobular tissue in it is hyperemic, permeated by single hemorrhages and infiltrated with serum. The reddened lobules of the lungs are surrounded by bright margins, which are 1 to 2 mm. broad and which are filled with a serous or lymphatic fluid. When the deposits are superficial, the pluræ become opaque and covered with slight clots.

At the height of the disease there is a lobular pneumonia with pleuritis which is usually spread over the greater part of one lobe of the lung. The exudate may be soft, membranous, fibrinous, lumpy and easily detached. The lung is considerably enlarged, of firm consistency, very heavy (weighing up to one hundred pounds), sinks in water and does not crackle when cut. Its section appears marbled, in consequence of the interstitial connective tissue having become thickened into broad lines which vary in color from orange to dirty white and which surround the dark colored lobules of the lung. The larger lobules have a thickness of from 0.2 to 5 cm.; and the smaller ones of from 0.25 to 0.50 cm. The color of the enclosed lobules of the lungs depends on the duration of the process and varies from brown-red to dirty yellow. The recently infected lobules have a blood-red, reddish-brown or dark brown color (stage of red hepatization). The color of the older ones varies from orange to yellow (yellow hepatization) and that of a still older date is gray (gray hepatization). The central foci, because they are the oldest, are usually in a stage of yellow or gray hepatization. Some of the enclosed lobules of the lungs are normal or only compressed, while others are merely hyperemic. If we closely examine the bright interstitial lines, we find that they consist at first of an edematous infiltration, which later on becomes plastofibrinous, gelatinous, indurated and finally tends to the formation of adventitious connective tissue. The lymph-spaces

in the lines are dilated like lacunæ and filled with a serous or fibrinous fluid.   In robust animals, the exudate in the alveoli is firm; but is of a more serous character in animals of a weak constitution.   In the former case, a section made through the lung will be found to be granular.   Besides these changes, the other lymph vessels of the lungs are dilated, their walls are infiltrated with cells and their lumen is in a state of thrombosis.   The blood vessels frequently show thrombi and small hemorrhagic infarcts.   The contents of the finer bronchi are often infiltrated with numerous white corpuscles.   The bronchial glands and frequently the mediastinal glands are inflamed and swollen.

After the disease has existed for some time, the affected parts of the lungs undergo induration, cicatrization, caseation, calcification, necrosis or suppuration.   At first the interstitial infiltration becomes dense, solid and dry and changes into firm connective tissue which makes a crunching noise while it is being cut with the knife.   In other places we have fatty degeneration, caseation, calcification or suppuration, in which the enclosed lobules of the lungs, in consequence of the existing suppuration, become gangrenous, and form sequestra surrounded by sequestral cavities which have smooth walls.   The dead portions of the lungs may remain unchanged in these cavities for a long time.   Frequently they become softened to the consistency of a greasy, yeast-like paste.   Sometimes, when they are comparatively small, they become absorbed, and a scar is formed.   The hepatized lobules of the lungs rarely regain their normal condition after the absorption of the exudate.   More frequently they atrophy or collapse, undergo atelectasis, calcification or softening, become necrotic or suppurating or form cavities.   On the pleuræ we find thick and wart-like hypertrophies of connective tissue, which frequently cause the lungs to adhere to the sides of the chest.

The changes in the lungs and pleuræ are the most important general lesions in pleuro-pneumonia.   It is stated that we may sometimes meet with an interstitial fibrinous exudate on the liver with atrophy of the liver cells; serofibrinous effusions into the articulations, tendon sheaths, subcutis, dewlap and brisket; intestinal catarrh; erosions of Peyer's patches and ulcers on the gastro-intestinal mucous membrane.

Meyer and also Boynton have called attention to the changes brought about in the muscle by the intermuscular injection of lymph from the thoracic cavity of an infected animal.   Boynton concludes that:

"From all appearances the contagious pleuropneumonia virus seems to have a specific action upon muscle and connective tissue, affecting chiefly the connective tissue elements.

"The appearances suggest that the virus multiplies in the lymph spaces of the connective tissue and blood vessels, gradually working its way through the walls of the blood vessels, causing an inflammation of the intima and thus giving rise to thrombus formations.

"The virus having invaded the tissue gives rise to a sero-fibrinous exudate, intermingled with groups of leucocytes leading to thrombosis of both lymph and blood vessels.

"The muscle lesions correspond with the lung lesions of contagious pleuro-pneumonia in the following respects:

(a)   Thrombus formation in the veins in both tissues.

(b)   The inflammatory areas around the blood vessels are similar.

(c)   The connective tissue is chiefly affected in both tissues.

(d)   The abundant serofibrinous exudate is present in both.

(e)   The deep staining line of leucocytes along the edge of the connective tissue is characteristic in both tissues.

(f)   The tendency toward a chronic productive inflammation is present in both.

"Thus in summing up all the lesions one finds the lung and muscle lesions corresponding in practically every respect."

One of the most conspicuous features in a microscopic examination of a lung affected with acute contagious pleuro-pneumonia is the presence of intensely stained foci and lines. These lines, to which Welch seems to have been the first to call attention, are visible to the naked eye and when viewed with a hand lens suggest by their peculiar curves the contour lines of 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 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 the cause of the disease itself. The space between the lines is filled with fibrin, in which very few leucocytes are found.

Diagnosis. It is extremely difficult to make an accurate diagnosis of the disease during its development, because the symptoms which are present are few in number and by no means characteristic. The slight fever and cough are the only symptoms of diagnostic importance in the prodromal stage. In the second or acute stage a positive diagnosis can be made only when cases of pleuro-pneumonia have previously occurred or when several cases occur simultaneously. As a rule, a correct diagnosis can be made only by a post-mortem examination. It is to be differentiated from: non-infectious inflammation of the lungs; tuberculosis; traumatic pneumonia or pneumonia due to foreign bodies; broncho or interstitial pneumonia; and pulmonary septicemia hemorrhagica.

Preventive inoculation and eradication. In Europe inoculation was practiced as early as the beginning of the last century.

The advocates of inoculation, especially Hausmann, Wilhelms, Haubner, Bouley, Schütz and others, start from the well known fact that one attack of pleuro-pneumonia successfully passed through confers immunity for the remainder of the animal's life. By inoculation, a local specific, inflammatory process which is analogous to that in the lungs, is produced and is followed by subsequent immunity of the whole body. Haubner calculated that the mortality from the inoculation is from 1 to 2 per cent. and that the tips of the tails are lost in from 5 to 10 per cent. of the cases. Since the work of Nocard and Roux in 1899, cattle have been immunized in France by the inoculation of pure cultures of the virus. Serum for hyperimmunized cattle gives, on repeated large doses, additional resistance.

The opponents of inoculation assert that up to the present no positive case of immunity has been proved to have been obtained from inoculation. They also point to the fact that even the advocates of inoculation are unable to give the exact duration of the immunity and consequently make several inoculations. The best procedure seems to be the stamping out of the disease by means of slaughter of all infected and exposed cattle, thorough disinfection or destruction by fire of all infected sheds and barns. The success of this method is illustrated by the eradication of the disease from the United States.

REFERENCES

1. Boynton. Notes on the muscular changes brought about by intermuscular injection of calves with the virus of contagious pleuro-pneumonia. *Bull. No. 20, Bur. of Agric.* P. I., 1912.

2. Dujardin-Beaumetz. Le microbe de la péripneumonie et la culture. *Thésis*, Paris, 1900.

3. MEYER. Notes on the pathological anatomy of pleuro-pneumonia contagiosa bovum. *The Vet. Bact. Laboratories of the Transvaal*, Pretoria, 1909, p. 135.

4. NOCARD ET ROUX. Le microbe de la péripneumonie. *Trans. Veterinary Journal, London*, Vol. XLVII (1898), p. 147. *Ann. de l' Inst. Pasteur*, Vol. XII (1898), p. 240.

5. SALMON. *Annual Reports of the Bureau of Animal Industry*, 1884–1892.

6. SMITH. *Annual Report of the Bureau of Animal Industry*, 1895–6, p. 143.

7. WALLEY. The four bovine scourges. 1879.

8. WILHELMS. Memoire sur la péripneumonie epizoötique du bétail. 1852.

HOG CHOLERA

Synonyms: Swine fever; pneumo-enteritis; pig typhoid; *Svinpest*.

Characterization. The distinguishing features of this disease are a continuous fever, ulceration of the intestines, petechial hemorrhages, especially in the kidneys, and more or less discoloration of the skin, especially over the ventral surface. It affects swine only.

History. The earliest recorded outbreak in this country of a disease supposed to be hog cholera, occurred in the state of Ohio in 1833. It is presumed that it was brought from Europe with some of the animals imported from there for breeding purposes. After being introduced, it spread at first slowly, but later with increasing rapidity along the lines of commerce, until it invaded every part of this country where swine raising had become an industry. The disease was investigated and very carefully described by Dr. C. Sutton, of Aurora, Ind., from 1850 to 1858. In 1861, Dr. Edwin M. Snow, of Providence, R. I., contributed an important paper on this disease to the U. S. Department of Agriculture. In 1875, Dr. James Law, of Ithaca, N. Y., furnished to the same Department a valuable paper setting forth the symptoms and morbid anatomy of this disease. He believed it to be contagious although the specific organism had not been found. The U. S. Commissioner of Agriculture appointed, in 1878, nine men for a period of two months each to investigate the disease in various localities. In their report the symptoms and morbid anatomy formerly described were confirmed and two additional features set forth. Law showed that it was transmissible by inoculation to other animals, and Dr. Detmers described a microörganism which he called *Bacillus suis*, and which he believed to be the specific cause of the trouble. Later, Detmers described his organism as a micrococcus. The work of investigation was continued under the direction of the Commissioner of Agriculture. In 1885, Salmon and Smith described an organism thought at the time to be the specific cause. It was

called Bacterium of swine plague. In 1865 Dr. Budd of England published a very exhaustive description of pig typhoid which corresponds to the lesions of hog cholera. There are references to a disease among swine in France in 1822 that appears from the description given to have been hog cholera. Fleming refers to an epizooty among swine in Ireland in 1040. There are also numerous references to infectious diseases among swine in Germany and other European countries prior to 1833. The positive diagnosis of those earlier outbreaks is impossible but the inference is strong that hog cholera existed at that time.

In 1886, Dr. Theobald Smith discovered another bacterial disease among swine. It was found to be similar to the German *Schweineseuche* both in the morbid anatomy and in the morphology and properties of its specific organism. In naming this disease the Bureau of Animal Industry called it, on account of its similarity to the German *Schweineseuche*, swine plague and its organism the bacillus of swine plague, and changed the name of the disease described in 1885 to hog cholera and its organism to the bacterium* of hog cholera.†

In 1903, de Schweinitz and Dorset discovered what they called a disease identical with hog cholera but which they produced with virus that passed through the finest porcelain filters. Subsequent investigations by Dorset, Bolton, McBryde and Niles showed that the organism known as the bacillus of hog cholera was not the cause of that disease but when present it was a secondary invader. They did not, however, deny that it possessed pathogenic properties for swine.

Soon after the discovery of the filterable virus, it was found that the serum of hogs that had recovered from cholera possessed a certain amount of immunizing power against the disease and that when they were hyperimmunized their blood serum would produce a temporary passive immunity against the virus in hogs injected with it. It was

---

*In 1888 the genus *Bacterium* was changed to *Bacillus* and this organism is spoken of since that time as the hog-cholera bacillus.

†Billings, of the Nebraska State Experiment Station, opposed this nomenclature. He not only refused to accept the change but continued to write about hog cholera under the title of swine plague. He also denied the existence of swine plague, as described in the reports of the Bureau of Animal Industry for 1886. The wide dissemination of his publications on this subject has unquestionably been responsible for much of the haziness concerning the distinguishing features of these maladies.

In 1893, Drs. Welch and Clements read a paper before the International Veterinary Congress in which they gave a very clear history of the nomenclature of these diseases and in which they adhered to the one of the Bureau of Animal Industry.

also pointed out that if the immunizing serum was used in conjunction with the virus, or the simultaneous method, the pigs became immune for a much longer time.   It is this serum, known as the Dorset-Niles serum, together with the use of the virus and serum or the simultaneous method, that are now being employed as prophylactics against hog cholera.

Geographical distribution.   Hog cholera is widely disseminated throughout the central part of the United States.   It exists, however, to a certain extent in practically every state in the Union.   It is known in Great Britain, and it prevails to a greater or less extent on the continent of Europe.

Etiology.   The cause of hog cholera is a filterable virus.   It exists in the blood, urine, lymph and all organs of affected swine.   So far as known, the virus of this disease exists only in the tissues and excretions of infected hogs.   No other species of animals are known to be affected with it, and its propagation outside of the body is unknown. In different outbreaks there seems to be a variation in the degree of virulence.   The virus is quite resistant to heat but rather quickly attenuated or destroyed by decomposition processes in tissues.   A 5% solution of chloride of lime or a solution of from 3 to 6% cresol soap seem to be the most effective disinfectants but they require considerable time when added to virulent blood.   The virus seems to be destroyed rather promptly when exposed to light and drying, but in damp places especially "wallows" it may persist for months. There is much difference of opinion regarding the significance of *B. suipestifer* in hog cholera.   It is frequently found in the tissues of swine suffering from cholera but it is generally believed to be a secondary invader.   Uhlenhuth found it in a large percentage of healthy hogs.

Dorset, Bolton and McBryde state that "it must be admitted that a disease in hogs may exist which is due to *B. suipestifer*, and which has no connection with the filterable virus found by us in the outbreaks we have studied."   They believe, however, that such a disease "would be possessed of a low degree of contagiousness."

Glässer found this bacillus to be a normal inhabitant of the intestines of swine.

The period of incubation varies from 7 to 14 days.   Berry states that it varies from one to three weeks and perhaps longer.

Symptoms.   In uncomplicated cases of hog cholera the first symptom is a rise of temperature.   This occurs before the animal

exhibits other evidence of being infected. In the very acute form this may be the only recognizable symptom. The temperature attains the maximum elevation about the seventh day.

When pigs live long enough to show physical symptoms there is a general depression, appetite poor or entirely gone. The animals usually stand or lie apart from others. Constipation or diarrhea may exist. The ears hang down, the tail is straight, the conjunctiva is congested and there is evidence of general weakness. As the disease advances the diarrhea becomes marked. The stain becomes a bluish or more scarlet red over the abdomen and about the ears. This is due to congestion of the parts. There may be convulsions. There may be a cough. There are sometimes necrotic areas on the mucosa of the mouth. In the chronic cases the animals become emaciated, the abdomen drawn up, the back arched and the gait unsteady. In chronic cases there may be secondary infections which may modify the symptoms.

In still more chronic forms the pigs eat fairly well until the end. There may or may not be diarrhea. Frequently the bowels are costive. It is quite common in these cases to have an active diarrhea during the last few days. The color of the discharge depends largely on the food. The changes in the respiration and the pulse are difficult to determine. There is rarely any cough. Usually the reddening of the skin on the nose, ears, abdomen and on the inside of the thighs and pubic region is very pronounced. It becomes more intense as death approaches. In some cases there is a discharge from the eyes. These symptoms vary to such an extent that it is usually necessary to make a post-mortem examination before a diagnosis can be made. Occasionally it is necessary to examine several animals in an outbreak before characteristic lesions are found. It not infrequently happens that swine suffering from hog cholera have pneumonia.

The duration of the disease varies. In the per-acute form it may not be more than a few hours or a day at the longest. In the acute form it lasts from 5 to 7 days. In the chronic form it lasts from one to two weeks, sometimes longer.

The prognosis is not good. Berry states that recoveries are not rare. Although there are outbreaks where the mortality reaches from 80 to 100 per cent., there are others of a milder type where the fatalities are much less.

Morbid anatomy. The acute type might with equal propriety be called the hemorrhagic or septicemic type, inasmuch as the chief and perhaps the only obvious changes are hemorrhagic in nature. They are more conspicuous when an animal is examined immediately after death. The hyperemia is first noticed in the lymphatic glands and the serous membranes. Later the cortex of the glands appears on section as a hemorrhagic line or band, according to the amount of extravasated blood, or the entire gland may be infiltrated with it. The glands most commonly hemorrhagic are those of the mesocolon, those at the root of the lungs, and on the posterior thoracic aorta. Besides these, the retro-peritoneal and the gastric glands may be

Fig. 92.   PETECHIAL HEMORRHAGES IN THE KIDNEY OF A PIG DEAD OF CHOLERA.

involved. The mesenteric glands may show congestion or slight blood extravasations. Hemorrhages are also quite frequent beneath the serous surfaces of the abdomen and thorax. They are most abundant as petechiæ and larger patches under the mucous membrane of the large and small intestines. They are occasionally found under the peritoneum near the kidneys, the diaphragm and the costal pleura, as ecchymoses or extravasations. The kidneys are usually the seat of petechiæ. The glomeruli may appear as blood red points; larger extravasations may occur in the medullary substance and blood may collect around the apices of the papillæ.

The more usual lesions are petechial hemorrhages in the cortex. They are also frequently present in the mucosa of the urinary bladder. The hemorrhages, according to Meyer, are due to a degeneration of the endothelium of the small blood vessels and capillaries brought about

by the action of the virus upon these cells or that the cement substance holding them together is dissolved or chemically broken down in which case the cells would be indirectly affected. In some cases the petechiae are restricted to one or more organs and in others they are wide-spread. The lungs are occasionally sprinkled with hemorrhages. In addition, there are usually distinct parenchymatous degenerations, especially in the kidney and liver. The extent of these changes depends upon the duration of the disease.

The lungs, in a small percentage of cases, show subpleural ecchymoses. On section small hemorrhagic foci are sometimes observed throughout the lung tissue. In a few cases severe hemorrhages involving one or more lobes have been observed. The subcutaneous tissue over the ventral surface of the body may be dotted with petechiæ and occasionally collections of blood (hematomata) are found in the superficial muscular tissue. Petechiæ have been observed on the cerebellum. The skin over the abdomen, inside of legs and back of ears is usually hyperemic.

The digestive tract is usually the seat of extensive lesions. The fundus of the stomach may be deeply reddened; there may be more or less hemorrhage on the surface, giving rise to larger areas of blood clots. In some cases the small intestine has submucous ecchymoses throughout its entire length. In the large intestines these may be so numerous as to give the membrane a dark red appearance. The intestinal contents are now and then incased in a layer of blood clot.

The chronic form is perhaps the most common, at least in those epizoötics which have been reported. The lesions of the large intestines are diphtheritic and ulcerative in character. The ulcers may be isolated and appear as circular, slightly projecting masses, stained yellowish or blackish or both in alternate rings, or they may be slightly depressed and somewhat ragged in outline. When the superficial slough is scraped away many ulcers show a grayish or white base. A vertical section reveals a rather firm neoplastic growth, extending usually to the inner muscular coat. When the sections are stained with aniline dyes and examined under the microscope, the submucous tissue is very much thickened, infiltrated with round cells and containing a large number of dilated vessels. Resting upon this thickened submucosa, is a line of very deeply stained amorphous matter and upon this is situated the necrotic mass which fails to retain the coloring matter and which is permeated by a very large number of bacteria

of various kinds.   Frequently the eggs of trichocephalus are imbedded
in the slough.

The extent of the submucous infiltration depends upon the age of
the ulcer.   In old ulcers it contains many newly-formed capillaries,
and evidences of the formation of connective tissue are present.   The
capillaries may extend to the very edge of the border where the slough
begins.   The latter may have been partly shed, leaving a smooth
line bounding the cicatricial tissue.   The submucous infiltration
gradually disappears toward the periphery of the ulcer and slightly
outside of the ulcer no inflammation of the membrane exists.   Giant
cells have been observed in the intertubular tissues at the edge of the
ulcer.   The depth to which the infiltration extends is not always
limited to the submucosa; it may extend into the muscular coats and
cause inflammatory thickening and inflammation and the formation of
new vessels in the subjacent serosa.

In some cases the necrosis, instead of appearing in circumscribed
ulcers from one-sixteenth to one-half inch or more across, involves the
whole surface of the mucous membrane, giving it the appearance of a
so-called diphtheritic membrane.   In such cases the walls of the
intestine are very much thickened and so friable as to be easily torn
with the forceps in handling them.   Such necroses are rare in epizo-
otic cholera, but they frequently appear in animals which have been
fed with pure cultures of *B. suipestifer*.   It is not clear, to what extent
the ulceration and inflammatory conditions of the intestines are due
to secondary infection.

The distribution of the ulcers varies but slightly.   They appear
most frequently in the cecum and on the ileo-cecal valve, as well as in
the upper half of the colon.   The lower half is implicated in severe
cases only and then less extensively.   The rectum is rarely ulcerated.
The lower portion of the ileum is ulcerated in a small percentage of
animals, especially when they have been fed with viscera from infected
hogs.   The lymphatic glands of the affected intestine are usually
much enlarged, pale, tough and whitish on section.   The spleen is
rarely enlarged.   The liver shows degenerative changes.   The heart
and lungs are usually normal.   The broncho-pneumonia, frequently
found in young pigs in the winter months, must be ascribed primarily
to exposure rather than to the presence of hog cholera virus.

In some outbreaks the acute and the chronic types are not clearly
separated.   Frequently recent hemorrhagic lesions seem to be

associated with cases presenting extensive ulcerations, which certainly are much older than the extravasations.

Diagnosis. Hog cholera is diagnosed by finding in the various organs of the dead animal the lesions that are at present considered characteristic of the disease; and by inoculating susceptible pigs with the blood from a suspected case and producing hog cholera. If a mixed infection is suspected the blood should first be filtered through a Berkefeld filter before injecting it. The inoculation is sometimes necessary because frequently pigs die of cholera before they show the lesions peculiar to the infection.

The diagnosis must be made in the field by the practitioner excepting in those cases where pig inoculations are to be made in which case blood from the suspected animal should be carefully taken. As experimental animals, such as rabbits and guinea pigs, are not susceptible the only assistance the laboratory can render in making the diagnosis is to inoculate one or more pigs. By this method the diagnosis cannot be made for several days which renders it of little value from the practitioner's point of view.

Hog cholera is to be differentiated from septicemiæ, from death caused by dietary conditions and poisons such as alkali or brine poisoning brought to pigs in their food. The particular diseases from which it is to be differentiated are Salmonellosis, hemorrhagic septicemia, infectious pneumonia, shoat typhoid and swine erysipelas, necrotic laryngitis, anthrax, heat stroke, lightning or sudden deaths from other causes. In the differentiation it is necessary to take into account the history and the general conditions under which the affected animals exist. It is of the greatest importance that practitioners understand that with hog cholera the prompt diagnosis is dependent upon the findings on the post mortem and that the sending of pieces of tissue to a laboratory for assistance is, in most instances, of little or no assistance.

Prevention. As hog cholera is caused by a specific virus, the prevention consists in keeping away from healthy swine any and all agencies that may bring the virus to them. It is known that litter from infected pens that has been brought into enclosures where healthy hogs were kept has caused infection. The virus is found in nearly, if not all, tissues of the body of the diseased hog and this virus remains alive and virulent in the tissues of infected animals for a considerable length of time after death. It is present in the

various organs early in the course of the disease so that when infected hogs are slaughtered for food before they exhibit symptoms, or the lesions are recognizable, their tissues if fed to healthy pigs may produce the disease. It is of the highest importance, therefore, that scraps of fresh pork that come from an unknown source should not be given to healthy swine unless they are thoroughly cooked. It is important that healthy hogs should not be shipped in infected crates or cars or placed in infected pens. Great care should be exercised in exhibiting hogs at fairs or stock shows. It is important that people who have the care of infected hogs should not be allowed to come in contact with healthy ones.

When hog cholera breaks out in a herd of swine it is of first importance that the diagnosis be made at once, that the apparently healthy hogs should be separated from the diseased ones and treated at once with anti-hog-cholera serum. If the diagnosis is promptly made and the prophylactic treatment administered at once, most of the individuals can be saved.

Anti-hog-cholera serum. Anti-hog-cholera serum is the defibrinated blood obtained from hogs that have been hyperimmunized against cholera. In some laboratories the defibrinated blood is centrifuged and the clear serum is used. It is employed to immunize hogs that have been or that are liable to be exposed to the disease. Experience has shown that serum will when properly administered protect healthy hogs against infection. It is not a therapeutic agent but frequently infected pigs carrying a high temperature have recovered after its administration.

In the use of serum care must be exercised to prevent common infections that are liable to result in abscesses. The serum is usually injected into the ham, the jowl or subcutaneously in the flank. There is some difference of opinion as to the application of serum to young pigs. While it is generally not considered with favor a few have used it with success.

Use of hog cholera serum. Three methods are employed in the use of hog cholera serum in the field: serum alone, serum and virus at the same time (simultaneous method), and serum alone followed in a week or more by simultaneous treatment (double method). Serum alone produces a transient immunity in sound herds and a permanent immunity in infected herds. Its chief indications·are for well animals in infected herds, in all cases where immunity lasting a month will meet the requirements, and in cases where the simultaneous method cannot safely be used. The simultaneous method produces a lasting immunity and is indicated in sound herds where all possible sanitary precautions seem unlikely to prevent infection. Contra-indications are:

first, in infected herds; second, where it cannot be used by experienced men; third, where the entire herd cannot receive serum, that is, no susceptible animal in a herd should remain untreated when any of the animals receive simultaneous treatment; fourth, in animals with subnormal resistance due to radical changes of feed and quarters, shipping, weaning, castrating, insanitary surroundings, etc.; fifth, in pregnant sows, especially those near farrowing time; sixth, in sows that are suckling pigs; seventh, in unweaned pigs and those weighing less than 40 pounds; eighth, in herds that cannot be properly isolated during the four or five weeks subsequent to treatment. Double treatment is indicated in cases where permanent immunity is desired with the least possible risk, and where its additional expense seems justified (pure-bred herds). It can also be used with great advantage where sows and suckling pigs are in imminent danger of exposure to cholera. The first dose (serum alone) protects until weaning time is over, and until the pigs are large enough and the sows are in proper physical condition to receive the second dose (simultaneous treatment). Some authorities question whether the passive immunity produced by the first treatment does not in some cases completely inactivate the virus given in the second treatment, so that sometimes only a temporary immunity follows the double treatment. While accumulating evidence indicates that the ultimate effects of double treatment and simultaneous treatment are identical, it is nevertheless prudent to prolong the time between treatments to a reasonable limit (3 to 6 weeks) of safety.

Usual method of serum preparation. A hog is immunized with a dose of virus, usually 2 cc., and sufficient serum to protect. After the resulting reaction is over (in 10–14 days) the animal is hyperimmunized by an intravenous injection of virus—5 cc. for each pound of weight. (The virus used is defibrinated, and preferably cooled, blood from pigs killed while in the height of an attack of acute hog-cholera. The pigs are infected with intramuscular injections of small doses of virus. The essential requirements are that the pigs shall show marked symptoms of acute hog cholera, that they shall be ready to kill in 7–14 days from the date of injection, that they shall show pronounced lesions of acute hog cholera, and no evidence of other infectious diseases). In about two weeks after the serum hog is hyperimmunized it is bled from the tail. Subsequently it is bled each week until the bleedings of a series number three or four. Then the immunity is reinforced by a second injection of virus—usually one-half the original amount. This is followed in about ten days by the first bleeding of a second series. Sometimes there is a third or even a fourth dose of virus each followed by three or four weekly bleedings. The final bleeding takes place from the throat. Each time blood is drawn it is immediately defibrinated, cooled, and preserved by the addition of one part in ten of 5% phenol. It is then stored in a dark cool place until enough is on hand for a test.

When a test is to be made the serum is poured into a large container, mixed thoroughly, a sample is drawn for testing, and the remainder is placed in bottles for shipping. The bottles are sealed and refrigerated. Eight pigs, preferably of the same litter and weighing 50–75 pounds each, are chosen for the test. Each receives 2 cc. of virus. Two receive 15 cc. each of serum, two receive 20 cc. each of serum, two receive 25 cc. each of serum, and the remaining two receive no serum. The pigs are marked for identification, placed together and given like care. Daily temperatures are taken. The requirements are that the pigs receiving virus alone shall sicken and reach a dying condition in 7–14 days from the date of injection, and display on post-mortem marked lesions of acute hog cholera; also that the pigs that receive virus and serum shall recover

showing very slight if any clinical evidence of disease. Usually a moderate elevation of temperature is the only symptom noted. (*Birch.*)

Control. Hog cholera can be checked in a herd by making an early diagnosis and promptly using serum on all of the animals not showing symptoms. The broader question of control, however, requires the enforcement of all sanitary procedures necessary to prevent the spread of the virus. During recent years there has been much progress in checking the losses from this disease but its spread has continued largely because the dissemination of the virus has not been controlled. At present there are in this country important investigations and experiments under way, which, when completed, will presumably give valuable information concerning the details to be observed for its control.

REFERENCES

1. BERRY. Swine-fever. *Jour. Compar. Path. and Therap.*, Vol. XV (1902), p. 1.

2. BILLINGS. *Bulletins Neb. Agric. Expt. Station, 1886–1893.*

3. BIRCH. A study of hog cholera transmission. *Report of the N. Y. State Vet. College*, 1913–14, p. 106.

4. BUDD. Typhus in pigs. *The Veterinarian*, 1865, p. 521.

5. DAWSON. The serum diagnosis of hog cholera. *New York Med. Jour.*, Feb. 20, 1897.

6. DE SCHWEINITZ. The production of immunity in guinea pigs from hog cholera by the use of blood serum from immunized animals. *U. S. Dept. of Ag. B. A. I. Ann. Rept.*, 1898, p. 269.

7. DE SCHWEINITZ AND DORSET. A form of hog cholera not caused by the hog cholera bacillus. *Circular No. 41, U. S. B. A. I.*, 1903.

8. DETMERS. *Report of the U. S. Department of Agriculture*, 1877.

9. DORSET. Hog cholera control investigations of the United States Department of Agriculture. Report of progress. *Proc. of U. S. Live Stock San. Asso.*, 1915, p. 99.

10. DORSET. The control of hog cholera. A review of four months' work by the Bureau of Animal Industry. *Proc. of U. S. Live Stock San. Asso.*, 1913, p. 38.

11. DORSET, BOLTON AND McBRYDE. The etiology of hog cholera. *Bulletin No. 72, B. A. I. U. S. Dept. of Agric.*, 1905.

12. DORSET, McBRYDE AND NILES. Further experiments concerning the production of immunity from hog cholera. *Bulletin No. 102, Bureau of Animal Industry*, 1908.

13. EICHHORN. Preparation of hog cholera serum in Hungary. *Proc. of U. S. Live Stock San. Asso.*, 1910, p. 33.

14. FISCHER. The control of hog cholera with immune serum. *Proc. of U. S. Live Stock San. Asso.*, 1912, p. 79.

15. FLEMING. Animal plagues, their history, nature and prevention. London, 1871.

16. GLASER. Studie über die Aetiologie der Deutschen Schweinepest. *Deutsche Tierärz. Wochens.*, Bd. XV (1907), S. 617.

17. HILTON. The control of hog cholera by slaughter methods. *Proc. of U. S. Live Stock San. Asso.*, 1913, p. 138.

18. KING, BAESLACK AND HOFFMANN. Studies on the virus of hog cholera. *Jour. Infect. Dis.*, Vol. XII (1913), p. 206.

19.   KING AND HOFFMANN. Spirochæta suis, its significance as a pathogenic organism. Studies on hog cholera. *Jour. Infect. Dis.*, Vol. XIII (1913), p. 463.

20.   LAW. *Report of the U. S. Dept of Agriculture*, 1875.

21.   OSTERTAG. Zur Aetiologie der Schweineseuche. *Berliner Tierärtliche Wochenschrift*, 1904.

22.   OSTERTAG. Ist das Virus der Schweineseuche und der Schweinepest filtrierbar. *Berliner Tierärztliche Wochenschrift*, 1906, p. 623.

23.   OSTERTAG. UND STADIE. Weitere Untersuchungen über die Ätiologie der Schweineseuche und Schweinepest. *Zeitsch. f. Infek. d. Haustier*, Bd. II (1907), S. 425.

24.   PETERS. Serum therapy in hog cholera. *Bulletin No. 47, Univ. of Neb. Agric. Exper. Station*, 1897.

25.   PFEILER. Uber die Beziehungen des Bazillus Voldagsen zur Schweinepest. *Berl. Tierärztl. Wochenschr.*, Bd. XXVIII (1912), S. 567.

26.   PFEILER. UND KOHISTOCK. Untersuchungen über Voldagsenpest (Ferkeltyphus). *Arch. f. Wissenich. u. prakt. Tierheilkunde*, Berlin, Bd. XL (1913–14), S. 114.

27.   PFEILER. Uber die Beziehungen des Bazeillus Woldagsen zur Schweinepest. *Berl. Tierärztl. Wochenschr.*, Bd. XXIX (1913), S. 209.

28.   PFEILER UND LENTZ. Die Züchtung des Virus der Schweinepest. *Berliner Tierärztliche Wochenschrift*, Bd. XXIX (1913), S. 689.

29.   REED AND CARROLL. Bacillus icteroides and Bacillus choleræ suis. A preliminary note. *The Medical News*, Apr. 29, 1899.

30.   REICHEL. Fixed hog cholera virus. *Am. Vet. Rev.*, Vol. XLII (1913–14), p. 559.

31.   Report of Committee on Diseases. American Vet. Medical Association for 1915. *Journal of the Am. Vet. Med. Asso.*, Vol. I (1915).

32.   REYNOLDS. Hog cholera and hog cholera vaccination. *Bulletin No. 113, Univ. of Minn. Agric. Exp. Station*, 1908.

33.   REYNOLDS. Virus blood vaccination. *Am. Vet. Rev.*, Vol. XL (1911–12), p. 485.

34.   SALMON. Special report on hog cholera, its history, nature and treatment. *U. S. B. A. I.*, 1889.

35.   SALMON AND SMITH. *Annual Reports of the B. A. I.*, 1885–1895.

36.   SMITH. Zur Kenntniss des Hog-cholera Bacillus. *Centralblatt für Bakter. u. Parositenkunde*, Bd. IX (1891), S. 253.

37.   SMITH. Hog cholera group of bacteria. *Bulletin No. 6, U. S. Bureau of Animal Industry*, 1894, p. 9.

38.   SMITH AND MOORE. Experiments on the production of immunity in rabbits and guinea pigs with reference to hog cholera and swine-plague bacteria. *Ibid.*, p. 41.

39.   STANDFUSS. Schweinepest und Schweinetyphus, ihre kennzichnenden Merkmale und Untershiede. *Mitteil. d. Verein. Deutsch Schweinez.*, 1913, Nr. 14, S. 279.

40.   UHLENHUTH. The Harben Lectures, 1911. Experimental investigations on hog cholera. J. Roy. *Inst. Pub. Health Lond.*, Vol. XIX (1911), p. 577.

41.   UHLENHUTH, HAENDEL, GILDEMEISTER, UND SCHERN. Weitere Untersuchungen über Schweinepest. *Arbeiten aus dem Kaiserlichen Gesundheitsamte*, Bd. XLVII (1914), S. 145.

42.   UHLENHUTH. Experiments concerning the resistance of virus. *Deutsche Tierärztliche Wochenschrift, Ibid.* S. 350.

43.   UHLENHUTH UND HUBENER, XYLANDER UND BOHTZ. Weitere Untersuchungen über das Wesen und die Bekämpfung der Schweinepest mit besonderer Berücksichtigung der Bakteriologie der Hog cholera — (Paratyphus B)—Gruppe sowie ihres Vorkommens in der Aussenwelt. *Arbeiten aus dem Kaiserlichen Gesundheitsamte*, Bd. XX (1909), S. 217.

44. WEIDLICH. Beitrag zur Ferkeltyphusfrage. *Berliner Tierärztliche Wochenschrift*, Bd. XXX (1914), S. 73, also S. 89.

45. WELCH AND CLEMENTS. Remarks on hog cholera and swine plague. *First International Veterinary Congress of America held in Chicago, Ill.*, 1893.

46. WOLBACH. The filterable viruses, a summary. *Jour. Med. Research*, Vol. XXVII (1912–13), p. 1.

FOOT AND MOUTH DISEASE

Synonyms: Aphthous fever; eczema epizoötica; epizoötic aphtha; vesicular fever; vesicular epizoötic; murrain; *cocotte*; *Maul- und Klauenseuche.*

Characterization. Foot and mouth disease is a specific, highly infectious and communicable disease which attacks chiefly ruminating animals and swine. The eruptions occur in the mouth and on the skin of the udder, teats, and interdigital spaces. Its essential characteristics are its definite, though often transient course; the eruption of vesicles; its extreme infectiousness; and its inoculability to practically all species of warm blooded animals. Another point no less characteristic is the appearance of the eruption, especially in cattle, on the three primary points of election, the mucous membrane of the mouth, feet and in cows on the teats.

People may be infected by drinking the unboiled milk of animals suffering from the disease. The mortality is not high.

History. Foot and mouth disease was quite accurately described in the eighteenth century. Very destructive outbreaks are reported in 1809 and in the early part of the nineteenth century it was the source of much loss in southern Europe. In 1883, Great Britain lost heavily from it. In 1892, Germany is reported to have had 1,300,000 cattle, over 2,000,000 sheep and over 400,000 hogs affected. In 1911 that country again suffered severely from it. In the United States there have been six outbreaks. They occurred in 1870, 1880, 1884, 1902, 1908 and 1914. In 1902 the states of Massachusetts, New Hampshire, Vermont and Rhode Island were involved. A total of 4,712 animals in 244 herds were affected. In 1908 the disease appeared in the states of Michigan, New York, Pennsylvania and Maryland. It affected animals on 157 premises. There were 3,636 animals (cattle, hogs and sheep) valued at $90,033.18 slaughtered. In 1914 it occurred again and spread to 21 states and the District of Columbia. A total of 2,245 herds with a total of 111,868 animals were infected.

Geographical distribution. According to Law, it originated in Asia. In 1544, it appeared in Northern Italy, France and England.

It has become firmly established in Europe. From there it has been carried to almost every cattle raising country.

Etiology. The value of the specific cause has not been clearly demonstrated. It has been shown, however, that it is a specific infection and that every outbreak starts from some previous case or cases. The virus is contained in the eruptions and escapes from the broken vesicles. Lœffler and Frosch found that it would pass through the coarser Berkefeld filters but that it was held back by the finer Kitasato bougie.

Animals may be infected directly by coming in contact with the disease, or indirectly by being exposed to the virus in stables, cars or other places recently occupied by infected animals. It is stated that the virus is destroyed in a short time by drying, but some writers maintain that it will persist for several months. Penberthy states that under ordinary circumstances the virus does not retain its infectiousness long. Animals that have passed through the disease may carry the virus for several months. Lœffler and Hess found animals to transmit the virus occasionally seven months after recovery. The infected matter may be carried on the clothing or hands of human beings and thus be transmitted to animals or man. Milk is often the carrier of the virus. In the recent outbreak many animals were infected by being fed unpasteurized milk from creameries.

The period of incubation is usually short, from 24 to 72 hours in inoculated cases and from two to seven days after natural exposure. In the recent outbreak there were cases that developed after a much longer period.

Symptoms. The symptoms of foot-and-mouth disease vary greatly in different epizoötics, sometimes they are quite mild and at others very severe. The first evidence of the disease is a rise of temperature which in cattle rarely exceeds 104° F. The mucous membrane of the mouth becomes reddened, the appetite is diminished, the muzzle dry; coat staring and rumination ceases. The mouth is usually kept closed and the quantity of saliva is increased. A peculiar smacking sound is not infrequently made by the animal. These symptoms are chiefly due to the pain accompanying the lesions in the mouth. After two or three days the eruption appears. This consists of small yellowish-white vesicles or blisters, varying in size from a hemp seed to a centimeter or more in diameter on the gums and inner surface of the lips, the inside of the cheeks, the border and the under surface of

the tongue.   In some cases the back of the tongue may be the seat of large blisters.

The vesicles burst soon after their appearance, sometimes on the first day.   More rarely they may persist for two or three days.   After the vesicles rupture the temperature subsides.   After they have ruptured the grayish-white membrane forming the blister may remain for a day or more or disappear speedily and leave deeply reddened areas or erosions which are very painful. These exposed areas may soon become covered with epithelium or they may be converted into ulcers. In this stage the saliva forms in large quantities and hangs in strings from the mouth.   In eight to fourteen days the disease may have entirely disappeared.

FIG. 93.   PHOTOGRAPH OF SICK COW SHOWING DROOLING.

In addition to the changes in the mouth, one or more feet may become diseased. The skin around the coronet and in the cleft between the toes becomes hot and tender and may swell. Blisters appear as in the mouth, but they are speedily ruptured and the inflamed, exposed areas are covered with a viscid exudate.   The animals move in a stiff manner.   Emaciation progresses rapidly, and in milch cows lactation is greatly diminished.

The udder, more particularly the teats, may be the seat of lesions. Some authorities regard the udder disease merely as the result of infection during milking.   The vesicles are broken by the hands of the milker and the teats become covered with reddened areas deprived of the superficial layer of the skin and are very tender.   The healing, however, goes on quite rapidly.   These are the main symptoms

accompanying the uncomplicated cases of foot-and-mouth disease. In all such cases recovery is usually rapid and complete.

FIG. 94. PHOTOGRAPH SHOWING EROSIONS IN THE INTER-DIGITAL SPACE.

Complications, especially in the absence of suitable quarters and proper treatment, are frequent, and as a rule are responsible for most

FIG. 95. PHOTOGRAPH SHOWING LESIONS ON TEATS IN SPORADIC APHTHOUS STOMATITIS.

cases that do not terminate in recovery. Deep ulcerations due to secondary invasion often appear on the feet. Prolonged lameness,

misshapen feet, laminitis, open joints and even necrosis of the pedal bone and sloughing off of the hoofs are some of the sequelæ.   Diges-
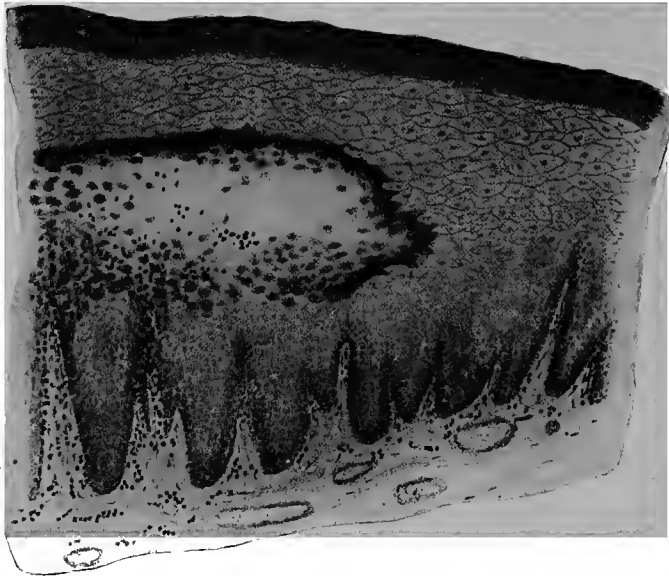
FIG. 96.   MICRO-PHOTOGRAPH OF SECTION SHOWING FORMATION OF APHTHA (ULCER). THE CENTRAL LIGHTER STAINING PORTION SHOWS LIQUEFACTION NECROSIS WITH LEUCOCYTIC INFILTRATION DIRECTLY BENEATH.   THE UPPER DARKER STAINED PORTION IS THE LAYER OF HORNY EPITHELIUM.   (*Zschokke*).

FIG. 97.   MICRO-PHOTOGRAPH SHOWING FORMATION OF APHTHA (ULCER) ON FOOT. (*Zschokke.*)

tive disturbances, pharyngitis and pneumonia are complications referred to mouth lesions. Septicæmia and pyemia sometimes occur.

The duration of the disease in uncomplicated cases varies from 10 to 20 days. When complications occur either with the regular course or as sequelæ the duration becomes indefinite. The mortality varies with the severity of the attacks, the age and condition of the animals and the treatment. Ordinarily the mortality is not high, excepting in very young animals.

FIG. 98.   FOOT AND PASTERN REGION OF PIG SHOWING CHARACTERISTIC LESIONS (*Kitt*).
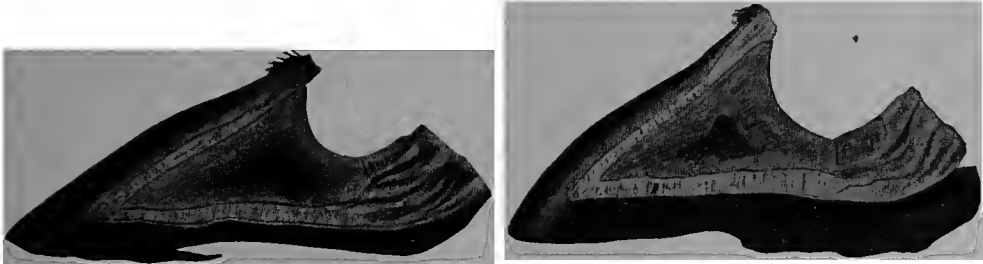
FIG. 99.   SECTION OF HOOFS SHOWING CLEAVAGE BETWEEN THE HORNY AND SENSITIVE SOLE (*Zschokke.*)

Morbid anatomy. The characteristic lesions are the eruptions in the mouth, the interdigital spaces and in cows on the udder and teats.

The eruptions consist of vesicles or blisters, varying in size from one to ten or more millimeters in diameter, containing at first a clear liquid which later becomes grayish in color. The thin layer of epidermis ruptures, sloughs off, leaving a raw, deeply reddened surface. The denuded and slightly depressed surfaces are soon recovered with epithelium. At first they present a brownish color which soon disappears. After the denuded areas are covered with epithelium the characteristics of the disease are lost.

According to Zschokke the lesions on the tongue begin in the *Stratum spinosum* of the malpighian layer of epithelium. Often

large areas of the mucous membrane slough off leaving a raw, painful surface.   Here on section the tissue takes the eosin stain more deeply and the nuclei of the cells do not stain as well with hematoxylin.   The next change noted is liquefaction necrosis with an infiltration of leucocytes and blood plasma.   The necrosis proceeds upward with the consequent liquefaction of the underlying tissue, until the covering epithelium is reached.   A blister or vesicle results which is filled with a serous liquid.

According to Bang the striking characteristic of the skin lesions is their superficiality. It amounts to a simple raising of the epidermis or

FIG. 100.   TONGUE SHOWING ULCERS AND HEALING PROCESSES (*Kitt*).

FIG. 101.   HEART SHOWING YELLOWISH STREAKS OF DEGENERATED HEART MUSCLE. (ALBUMINOID DEGENERATION)(*Hæbiger*).

epithelium of the mucous membrane caused by a serous exudation. There is no deeply rooted inflammation of the mucous membrane or corium; the sore simply consists in the laying bare of the surface of these parts and it has a natural tendency to heal quickly.

The tissue changes reported on post-mortem vary to a marked degree. There are in certain cases hyperemia and edema, catarrh of the nares and mucosa of the lungs and dilatation of the heart. In cases which lead to sudden death, the most pronounced change is usually found in the heart muscle. This condition has been carefully studied in cattle by Jost and Zschokke and in pigs by Hæbiger. They find an extensive myocarditis which can be noted by the yellowish streaks which are shown on the heart. In pigs Hæbiger describes an
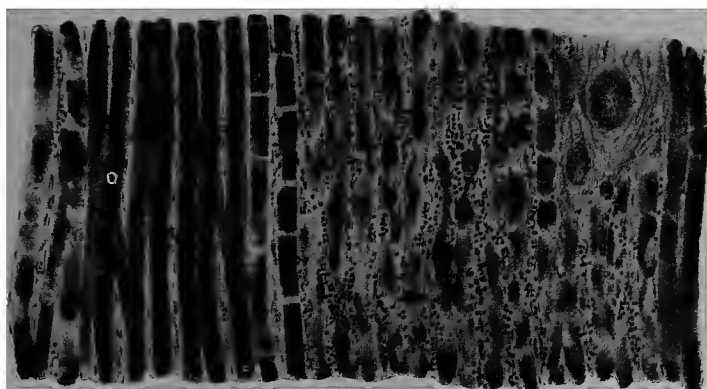
FIG. 102. MICRO-PHOTOGRAPH OF HEART MUSCLE SHOWING DEGENERATION AND LEUCOCYTIC INFILTRATION (*Zschokke*).

"albuminoid" degeneration of the myocardium. These changes in the heart seem to be characteristic of this type of the disease. There may be associated with the primary heart lesions congestion and œdema of the lungs. The mucous membrane of the stomachs and intestines may be dark red and numerous hemorrhages are sometimes found. Ulcers or erosions up to an inch in diameter may be present on the mucous membrane of the pharynx, rumen and abomasum. Ulcers of a nature similar to those found in the stomach occur in the intestines.

On the feet inflammations may follow the simple vesicles about the coronet. These may extend deep into the tissues, pass under the hoof and cause it to slough off, extend into the bone producing necrosis or

permanent arthritis.   It is stated by some authorities that in the mild non-fatal cases the obvious lesions are so slight that frequently they escape notice.   In sheep and swine the lesions are more frequently restricted to the feet.

|~ **Diagnosis.**   Foot and mouth disease is diagnosed by the symptoms, obvious lesions and the inoculation of susceptible animals.   It is to be differentiated from (a) various forms of stomatitis caused by different fungi and often referred to as sporadic aphthæ, (b) from stomatitis due to drugs and injuries, (c) foot rot or suppurative cellulitis in cattle

FIG. 103.   PHOTOGRAPH SHOWING ULCERS ON THE UPPER GUM.

and sheep, (d) from actinomycosis of the tongue and (e) from variola. It must also be differentiated from simple cellulitis, often of streptococcic origin, in the subcutaneous tissue about the coronet and from the sloughing of the hoof resulting from the extension of the inflammatory process, and from eczemas due to dietary causes.   The means of differentiating these must be found largely in the history of the cases and in the study of the nature of the lesions themselves.

The most difficult lesions to distinguish between are certain more or less pigmented depressions due to traumatism and foot-and-mouth disease lesions rather late in their course.

Prevention. Preventive inoculations do not seem to have given satisfactory results. Lœffler produced a serum that possessed some immunizing effect. The isolating of the diseased animals and the placing of the well ones in noninfected fields and stables tend very largely to the checking of the spread of the disease. The milk of the diseased animals should be sterilized before it is used.

Control. In England the slaughter of infected animals has been resorted to in recent years to stamp out the disease. In the recent outbreak in the United States, the prompt destruction of all infected and exposed animals proved to be very satisfactory. The failure in Europe to control the disease by quarantine caused Dammann, Hess, Cope and others to recommend the radical measure of slaughter of infected and exposed animals in order to eliminate the disease. At present there seems to be no other method for eliminating this affection.

REFERENCES

1. BANG. Foot-and-Mouth Disease. *The Jour. of the Board of Agric., London,* Vol. XIX (1912), p. 624.

2. COPE. Foot-and-mouth disease. *Report, 7th International Congress of Vet. Surgeons, Baden Baden,* Vol. 1 (1899), p. 184.

3. CORNELL VETERINARIAN. Special number on Foot-and Mouth Disease. Vol. IV (1915).

4. LECLAINCHE. Foot-and-Mouth Disease. *Tenth Internat. Vet. Cong., London,* 1914.

5. LOEFFLER UND FROSCH. Berichte der Kommission zur Erforschung der Maul- und Klauenseuche bei dem Institut für Infektionskrankheiten in Berlin. *Centralbl. f. Bakt.,* Bd. XXIII (1898), S. 371.

6. LOEFFLER. Ein neues Verfahren der Schutzimpfung gegen Maul und Klauenseushe. *Münch. Med. Wochenschr.,* 1906, S. 1036.

7. MELVIN AND MOHLER. Foot-and-Mouth Disease. *Bulletin No. 2. U. S. Live Stock Sanitary Association,* Series 1915.

8. MOHLER. Foot and Mouth Disease. *Farmers' Bulletin No. 666. U. S. Dept. Agric.,* 1915.

9. NEVERMANN. Foot-and-Mouth Disease. *Tenth Internat. Vet. Cong.,* London, 1914.

10. PEARSON. Report on the outbreaks of aphthous fever in Pennsylvania in 1908–9. *Bulletin No. 211, Dept. of Agric.,* Harrisburg, Pa., 1911.

11. PENBERTHY. Foot-and-mouth disease. *Jour. Comp. Path. and Therap.,* Vol. XIV (1901), p. 16.

12. SALMON. Foot-and-mouth disease. *Year Book U. S. Dept. of Agric.,* 1902. p. 643. *Ibid. Annual Report Bureau of Animal Industry,* 1902, p. 391.

13. WALLEY. The four bovine scourges. 1879, p. 61.

14. WILLS. Foot-and-Mouth Disease in New York State. *Bulletin Dept. Agric., Albany, N. Y.,* 1915.

INFLUENZA IN HORSES

Synonyms: Epizoötic catarrhal fever; epizoötic catarrh; horse distemper; pink eye; mountain fever; shipping fever; typhoid fever; stable pneumonia.

Characterization. Influenza is an acute infectious disease characterized by a rise of temperature and a catarrhal condition of one or more of the mucous membranes, more especially of the head, or by inflammation of the lungs and pleuræ. One or more of the internal organs may become affected. It is a disease of horses, although asses and mules are susceptible and a few cases are reported of its being transmitted to man and to dogs. It appears sometimes in sporadic form and often in epizoötics.

Influenza is a generic term employed to designate a large variety of symptoms. Dieckerhoff designated the pulmonary form contagious pleuro-pneumonia. A somewhat careful analysis of the manifestations suggests that possibly influenza includes a number of etiologically distinct diseases, *i. e.*, morbid conditions brought about by different causative factors. The term has long been employed to designate a considerable variety of equine epizoötics, the independence of which could not be fully established. The disease, as it is seen in the horse, suggests further that possibly it is in its beginning a general affection because of the early rise of temperature and that later in its course it becomes, to a limited extent, localized. At present influenza is restricted to groups of symptoms and lesions in the horse that are analogous to those of *la grippe* in man. As it is not usually fatal, little progress seems to be made in acquiring knowledge concerning the nature of its morbid anatomy. There is much need for further investigation of this very common malady.

History. According to the writings of Falke, influenza was recognized in very early times. There is evidence that it was known in the fourth and fifth centuries. It was described by Löw in 1729 in an equine epizoötic which had spread over Southern Europe. It is also stated that cases of its having been transmitted to man had occurred. Gibson observed it in 1872 in London and in different districts of England. It raged in epizoötics in 1760, 1776 and 1803. The disease was widely disseminated during the last century. The more important epizoötics are reported in the years from 1813 to 1815, 1825 to 1827, 1836 to 1840, 1846, 1851, 1853, 1862, 1870, 1873, 1881 to 1883, and 1890. Anker, who described influenza in Switzerland in 1826,

laid stress on its contagious nature and stated that in his opinion "a volatile infectious matter was the cause."

Influenza spread as an epizoötic in 1872 to 1873 over the greater part of the United States where it received the name of "pink eye" (French, *fièvre typhoide*). It started in Canada and extended south and west, reaching into British Columbia to the north and Mexico at the south. The last great epizoötic raged in Europe from 1881 to 1883 during which time it is said to have spread over nearly the whole continent. In the Prussian army, 3,434 horses became affected in 1890; 2,497 in 1891; and 3,645 in 1892. In Copenhagen, 3,000 horses were affected in 1890 and 1891.

Geographical distribution. Influenza seems to be known in nearly if not all of the countries of Europe and America. In certain sections of the United States it is an almost constant affection. This is especially true of certain cities, owing to the constant introduction of "green" horses.

Etiology. Influenza seems to be produced by a specific infection the nature of which has not yet been determined. A number of bacteria have been described as the probable cause but thus far none of them have been found to be sufficiently constant to warrant their acceptance as the etiological factor. Ferry has recently emphasized the presence of streptococci in the tracheal mucus and in the blood. The infecting agent spreads rapidly among horses. The virus appears to lose its virulence quickly outside of the animal body, but within the body it seems to be preserved for a long time. According to the observation of Jensen and Clark, stallions which have had the disease may transmit it to the mares they serve for months after apparent recovery. Some believe the cause to be a filterable virus. Dieckerhoff succeeded in transmitting the disease to healthy animals by subcutaneous and intravenous injections of the blood of infected horses, but Friedberger and Arloing failed to do so. Horses are most susceptible. Sex, breed, stable management and feeding appear to have little or no influence on their individual susceptibility.

Infection usually takes place from horse to horse. through the secretions and excrements which are especially infectious during the development and at the height of the disease. Convalescent animals often eliminate the virus. The general belief is that the horse becomes infected through the digestive tract. It is not disproven that infection may occur through the respiratory organs. The virus appears

to be carried by infected human beings, litter, harnesses and ther-
mometers. In many cases one attack confers immunity but a second
infection or a relapse frequently occurs. Toward the end of an
epizoötic the disease is usually milder in form, probably due to a
gradual attenuation of the virus. Much work still needs to be done
on the etiology of this affection.

The period of incubation varies from two to seven days. Siedam-
grotzky places it at from four to five days and in rare cases from 12 to
24 hours. In some cases symptoms have not appeared until from two
to five weeks after the latest known exposure.

Symptoms. The disease appears suddenly and may attain its
highest point of intensity within twenty-four hours. The organs of
circulation, nervous centers, digestive and respiratory mucous mem-
branes and conjunctiva are especially affected. There is partial or
entire loss of appetite and depression. The temperature rises sud-
denly from 3 to 4° F. or even more. It remains high with but slight
variations for from three to six days and then falls rather quickly,
often within twenty-four hours, to the normal. At first the frequency
of the pulse is but little increased in comparison to the elevation of
the temperature, but later it rises to from 60 to 70 and in fatal cases
from 80 to 100 or more. It generally continues high for some time
even after the temperature has fallen. The fever is characterized by
unevenness in distribution of the external temperature of the body.
The early rise of temperature, while the affected horses appear to be
healthy, is of much diagnostic value.

Usually the nervous depression coexists with the fever. The
animal may hold its head down and appear to be comatosed. Extreme
muscular debility is frequently associated with this stupor. Tremors
may occur, the hind legs may give way while walking and paralysis
of the hind quarters appears in a few cases.

The oral mucous membrane is greatly congested, hot, dry or covered
with mucus. There is sometimes difficulty in swallowing. The
animal frequently yawns. There is usually constipation in the begin-
ning of the disease which may be accompanied with colic. The feces
are formed into small hard balls and are covered with masses of mucus.
Later diarrhea with considerable tenesmus usually occurs. The feces
are of a thin, pulpy and even fluid consistence and sometimes have a
fetid odor. At the beginning of the attack the urine is alkaline but it
becomes acid when the intestinal lesions are developed. It rarely

contains albumin but desquamated epithelial cells of the bladder are often present in large quantity.

A severe affection of the eyes is a quite constant characteristic symptom of influenza. At first it consists chiefly of a catarrhal and later of a phlegmonous conjunctivitis with considerable swelling of the eyelids, which may be followed by keratitis and possibly by an exudative or hemorrhagic iritis. Usually both eyes are affected. The first indications are the presence of tears, intolerance of light, intense hyperemia of the conjunctiva and contraction of the pupil. The eyelids swell, are hot, painful and kept more or less continually closed. A gray, muco-purulent secretion accumulates between the eyeball and eyelids and the eyeball becomes very sensitive to pressure. The cornea, which at the beginning of the keratitis has a greasy lustre, first becomes irridescent, but later in the course of the disease it may be opaque. It is considerably injected with blood at its edge; the iris becomes swollen and yellowish in color. Often these inflammatory changes of the eye disappear in a strikingly short time.

During the further progress of the disease, swellings may appear on the extremities, sheath, epigastrium and lower part of the chest. It may be concluded that these swellings are due to edema caused by passive congestion. Less frequently the swellings are of an inflammatory nature. The swelling of the extremities causes the gait to be stiff and unwieldly.

The respiratory mucosæ are congested. At first there is a serous and, later on, a muco-purulent discharge from the nose, slight swelling of the submaxillary glands, moderate acceleration of respiration and a cough. As a rule the animal becomes emaciated during the course of the disease. Pregnant mares may abort.

In certain cases complications may arise, such as cardiac debility, grave cerebral symptoms, severe gastro-intestinal inflammation, laminitis and petechial fever, all of which have been described as accompanying complications.

The duration of the disease is from six to ten days, although severe cases may run for two to three weeks and very mild ones may recover in from three to six days.

The mortality varies at different times and in different places. The average appears to be from 0.4 to 4 per cent. Dieckerhoff saw a loss of 4 per cent. among 1,700 horses; Aureggio, one of 3 per cent. among 800 horses; Friedberger, one of 9 per cent.; and Siedamgrotzky, one

of 10 per cent. It is stated that in 1872, in Philadelphia, 7 per cent. of 30,000 infected horses died.

Morbid anatomy. The principal tissue change is the acute hyperemia of the mucous membrane of the digestive tract which may also be swollen and sprinkled with slight hemorrhages. The sub-mucosa is yellowish in color and infiltrated with a gelatinous substance causing the membrane to form thick, somewhat translucent, eleva-tions containing a fluid which coagulates. Peyer's patches are enlarged, especially those in the neighborhood of the ileo-cecal valve. The mucous membrane of the mouth and sometimes that of the pharnyx show similar changes.

The mucous membranes of the upper air passages are hyperemic and swollen. In rare cases, the mucosa of the larynx is inflamed, also the subcutis when inflammatory swellings appear on the skin. Schütz found that in the brain and spinal cord the arachnoid spaces are filled with a fluid which is generally clear, although it may contain leuco-cytes. He reports one case in which the lateral ventricles contained a large quantity (20 c.c.) of fluid. The other lesions which may be found depend upon the extent or localization of the disease. Usually the spleen is slightly enlarged; small hemorrhages in the intestines, under the serous membranes and in the lungs, eyes and brain; gela-tinous infiltration of the renal connective tissue and mesentery; swelling of the lymph glands; yellowish serous exudates in the larger cavities of the body and imperfect coagulation of the blood. One or all of these changes may appear in a single animal. There sometimes occur edematous swellings of the subcutaneous tissue on various parts of the body.

Diagnosis. Influenza is diagnosed by the symptoms and its infec-tious nature. A rise in temperature is an important diagnostic feature in times of an epizoötic. There is no specific test. It is to be differentiated from strangles, contagious pleuro-pneumonia, petechial fever and catarrhal conditions of the head.

Prevention. The removal of uninfected horses from infected stables is important. Newly purchased horses should be kept separate from the others for some days unless their source and non-exposure in sale stables and cars are known. Many efforts to immu-nize horses with vaccines have been made and also with the blood of recovered animals but uniform satisfactory results have not been obtained.

REFERENCES

1. FERRY. Studies on the etiology of equine influenza. *The Veterinary Journal*, Vol. XIX (1912), p. 185.

2. LIGNIÈRES. The etiology of equine influenza or infectious pneumonia. *Jour. of Comp. Path. and Therap.*, Vol. XI (1898), p. 312.

3. M'FADYEAN. Influenza of the horse—what is it? *Jour. of Comp. Path. and Therap.*, Vol. II (1889), p. 105.

4. MARSDEN. Influenza. *The Vet. Jour.*, Vol. II (1900), p. 315.

5. NELSON. Influenza. *Bulletin 22. State Agric. Exper. Station*, Washington, 1896.

EQUINE CONTAGIOUS PLEURO-PNEUMONIA

Synonyms: Pleuro-pneumonia contagiosa equorum; stable pneumonia; bilious pneumonia; pulmonary influenza; epizoötic pneumonia; *pneumoenteritis*; *pasteurellosis*; *Brustseuche;* pectoral influenza.

Characterization. Contagious pneumonia or contagious pleuropneumonia in horses, asses and mules is characterized, in typical cases, by a high temperature, rapid pulse, inflammation of the lungs and pleura with a tendency to gangrene, but in mild cases without definite lung disturbances. Like strangles, both the symptoms and the lesions vary to such a degree that it is difficult to single out diagnostic features.

History. In earlier times, influenza and contagious pleuropneumonia of the horse were not distinguished as separate diseases. Falke differentiated the disease formerly known as influenza into contagious pleuro-pneumonia and influenza. Since his time they have been recognized as distinct diseases.

Geographical distribution. Contagious pneumonia, like strangles, is widely distributed. It appears in epizoötic form, although in certain places it is reported to be almost enzoötic. It prevails most extensively where large numbers of horses are congregated. It has frequently been reported as the cause of much trouble among the horses in the European armies. In the eastern part of the United States, it appears from time to time in more or less serious epizoötics. It is quite common among horses shipped from the West. In these cases, it is designated as "western" or "stable" fever. There is some question, however, as to the identity of *Brustseuche* and the shipping fevers. Until the etiology is more definitely determined authors will doubtless continue to differ on this point.

Etiology. The cause is now believed to be a filterable virus. It is found in the tracheal mucus.

Gaffky and Lührs found that "pieces of diseased lungs removed from killed foals before bacteria were present did not transmit the virus, even when portions were injected directly into the lungs of susceptible horses. This fact, combined with the earlier findings, that the virus is not found in the blood during any stage of the disease, tends to support the conception that it is confined to certain cells. The authors believe these to be the epithelial cells of the air passages. This belief is based on the successful transmission of the disease through the bronchial secretions containing large numbers of these cells. The material was taken from slaughtered horses. In typical cases one finds in horses killed on the third or fourth day that the air passages are more or less filled with a yellowish, glassy, transparent, tenaceous secretion, that may be lifted out of the passages with a pincette. This secretion is coughed up and observed during the course of the disease; many times it is swallowed, and for this reason is not usually seen. In the latter stages of the disease the secretions contain many micro-organisms, when they lose their tenacious condition, and perhaps the greater part of their infectiveness."

In 1887, Schütz described an organism which appeared as a diplococcus in tissues, but in bouillon cultures it grew in flocculi. From the description, it appears that it was a streptococcus, notwithstanding the fact that in the tissues it appeared more often as a diplococcus. According to Schütz, cultures injected directly into the lungs by means of a hypodermic syringe produced the disease. The resulting contagious pleuro-pneumonia exhibited the same symptoms and ran a like course to the disease contracted in the natural manner. The inoculated streptococci were found in the tissues of the artificially produced disease. According to Schütz, the bacteria of contagious pleuro-pneumonia are found most numerously in the lungs or the exudate on the pleuræ. It is quite generally conceded that the streptococcus of Schütz is a secondary invader.

Lignières believed that a "cocco-bacillus" stands in an etiological relation to this disease and that here, as in strangles, the streptococcus is a secondary invader. Because of Lignières' observation the French often refer to this disease as *pasteurellosis*. His theory of the etiology has not been confirmed.

Moore made a bacteriological examination of the organs from five cases of fatal contagious pneumonia of the horse. In each case, the lungs were more or less hepatized, but the other organs were nearly normal in appearance. Without exception, a streptococcus appeared, usually in pure culture, from the lungs. The inoculated

media from the other organs (liver, spleen, and kidney) remained clear. The streptococci isolated from the different cases were identical in their morphology, cultural manifestations and pathogenesis. A microscopic study of the lungs from the different horses showed streptococci singly, in pairs and occasionally in short chains. Distinct capsules were not observed. In bouillon cultures, however, they appeared in long chains, leaving the liquid clear, as described by Schütz.

Recent investigations indicate that the etiological factor will pass through the Berkefeld filter. Gaffky and Lührs found that it was present in the smaller bronchioles and that it was forced out through the nose by coughing. They found that it lost its virulence soon after being expelled from the infected animal. It is spread by infected animals as soon as a rise of temperature begins. Natural infection usually takes place by direct contact. The disease has not been transmitted to horses by insects. According to Gaffky and Lührs the virus is located in the bronchi. They conclude as follows:

The disease as a rule is transmitted only from horse to horse. It is carried from animal to animal by the inhalation of drops of infected material which are expelled from a sick animal when coughing or sneezing.

The virus is not found in the nasal discharge but it lies deep in the bronchi and is forced out through the nose by forcible coughing and sneezing and therefore renders the sick animal dangerous.

The virus soon dies after being expelled from the sick animal. After 24 hours it has been found occasionally in the straw contaminated by sick animals.

An infected animal can spread the disease during the beginning of an attack or as soon as a rise of temperature is noted.

The period of incubation is generally given as varying from one to fourteen days. According to Gaffky the period of incubation varies from 16 to 42 days.

Symptoms. The symptoms vary to a marked degree. The first symptoms usually suggest croupous pneumonia. When pneumonia develops early in its course, the disease may appear suddenly; and in addition to the elevation of temperature there is a cough with difficult breathing. In those cases where the symptoms are restricted to those of a general infection the temperature drops to the normal in a few days. Often the symptoms differ from those of fibrinous pneumonia by the absence of distinct evidences of local lesions which are found in that disease. The first regular symptom is a rapidly rising temperature, frequently accompanied by a chill. The pulse rate is

increased but later becomes slower. There is general depression, usually loss of appetite and muscular weakness; the conjunctivæ and other visible mucous membranes become congested. There may be from the beginning marked indications of localized lesions in the lungs, or the general symptoms may continue without evidence of pronounced lung disturbance. Many symptoms may be exhibited, corresponding to the variations in the morbid processes. If the heart, digestive tract, liver, kidneys or brain become the localized seat of the disease, symptoms referable to impaired functions of these organs are in evidence. The septicemic form has been described as being followed by localized suppurative lesions.

The duration of the disease varies. The high temperature lasts for from five to eight days in the typical cases. The convalescence requires from two to four weeks. If there are complications the course may be much longer.

The mortality is often very high. The literature shows it to vary from one to thirty per cent. It frequently leaves animals practically worthless because of pleural adhesions and other complications.

Morbid anatomy. The morbid changes in the tissues and organs vary according to the course of the disease, which is exceedingly irregular. There may be a regular form of lobar pneumonia, or the disease may run an atypical, complicated, acute, chronic, and not infrequently an abortive course. Further, authorities agree that many complications may arise modifying or changing completely the morbid anatomy of the disease from the conditions found in the more typical cases. In the few cases examined post-mortem by the writer, the gross lesions were *restricted to the lungs.* These were either in a state of congestion, or exhibited changes of fibrinous pneumonia in the cephalic (anterior) portions of one or both lungs. Pneumonia is the most common localized lesion. Several quite distinct forms of lung disturbances are described.

According to Gaffky and Lührs, "the local changes in the lungs begin in the finest branches of the air passages. In the beginning of the disease there is secreted a glassy, transparent, slightly gelatinous, yellowish material. This is surrounded by a thick layer of cells and a serous infiltration. In the region of the affected bronchi the alveoli are filled with a fluid rich in cells.

"In those places where the disease foci are near the pleura there is an infiltration of the subpleural tissue with a transparent, yellow-

ish, gelatinous fluid. The interlobular connective tissue is infiltrated with serum.

"On the fourth or fifth day, not before, bacteria begin to colonize in the diseased parts of the lungs, in the form of cocci arranged in chains. These cause inflammatory, often hemorrhagic, changes that may lead to extensive necrosis."

In the lobular form of pleuro-pneumonia, which it is stated furnishes the largest number of subjects for post-mortem examination, there are frequently hemorrhagic foci in the acute cases and possibly gangrenous pneumonia with secondary pleuritis. Small necrotic areas are scattered through the hepatized portions. Parenchymatous degeneration of other vital organs is reported. The hepatized foci are located more expecially near the base of the lungs and in the lower (ventral) portions. They vary in size from a millimeter to 20 or more centimeters in diameter. In recent lesions, these areas are very small, of a grayish-red color and surrounded by a grayish zone consisting of leucocytes. In more advanced lesions they become yellowish, necrotic and finally cavities are formed varying from the size of a pea to that of a hen's egg. These cavities are surrounded by a smooth capsule. There are other foci which contain greasy, fetid, watery pus (gangrene of the lungs), by reason of the necrotic part of the lung undergoing liquefaction in consequence, it is stated, of the admittance of air. The lungs often contain suppurating foci composed of a whitish pus mixed with necrotic lung tissue. It sometimes happens that the foci just described are absent in the lungs, although during life suggestive symptoms of such a localized affection may have been present. In these cases, it is assumed that absorption of the necrotic tissue has taken place. The remaining tissue of the lungs is more or less hyperemic or edematous.

The pleuræ show signs of a diffuse, exudative inflammation, the starting point of which in the large majority of cases is from necrotic deposits which are situated in the periphery of the lungs. Pleuritis may occur, however, apparently as a primary lesion. The contents of a necrotic deposit in the lungs rarely discharge into the pleural cavity. In some cases, the visceral and costal layers of the pleuræ are congested, diffusely or in spots, and are sprinkled with hemorrhages. Frequently the pleuræ are covered with soft red granulations over which are layers of yellowish exudate which are partly membranous and partly coagulated in a reticular manner, and which can usually be easily removed. The exudate may be less firm and more

purulent in character. The pleural cavities generally contain a considerable quantity of fluid. Dieckerhoff states that from 30–40 liters of a serous fluid are occasionally present. The exudate is usually turbid and of an orange, grayish-red, brownish-red, or dirty-grayish color. It is generally mixed with numerous yellowish colored flakes which form a sediment when the liquid is allowed to stand in a glass. The pleuritic exudate sometimes consists of pure pus and less frequently of blood. The pleuritic exudate when present in large amount compresses the lungs and pushes them away from the thoracic walls. In case of recovery, the pleuritic exudate may become organized, binding the lungs to the costal walls and diaphragm. Various forms of fibrous, villous growths develop on the pleuræ.

The reports show that the other organs of the body are usually in a state of parenchymatous inflammation and fatty degeneration. The muscular tissue of the heart is, as a rule, brownish-gray in color, soft, and suffers from cloudy swelling. In severe cases, it shows well-marked fatty degeneration, is of a clay color, and is occasionally sprinkled with a large number of small, yellowish-white foci. The liver is enlarged, of a clay color or sometimes icteric, and presents signs of fatty degeneration. The spleen is flaccid, its pulp increased and often sprinkled with hemorrhages. The kidneys may be swollen, friable and sometimes show numerous hemorrhagic foci. The lymph glands, especially the bronchial and mediastinal glands, are enlarged, softened, and exhibit on section a grayish-red color. The muscles of the body are soft, and of a yellowish-brown color. Small hemorrhages under the serous membranes are frequently reported. Slight endocarditis may occur. The blood suffers less change than any of the solid organs. It contains an excess of polynuclear leucocytes.

The mucous membranes of the stomach and intestines are frequently hyperemic, swollen, sprinkled with hemorrhages, and sometimes even ulcerated. The bronchial mucous membrane is swollen and inflamed.

In other cases, the lesions are those of lobar pneumonia, in which the stages of hyperemia, red hepatization, gray hepatization and resolution (in favorable cases) follow each other in regular order. In a fatal case post-mortemed by the writer the right lung was entirely involved, the left one being but slightly hyperemic. In non-fatal cases, the crisis is reached on the 5th or 6th day, after which resolution begins.

As already stated the lesions in this disease are so exceedingly variable that, in addition to the more typical pneumonia, almost any

modification can be found. The complication of gastro-enteritis weakens the patient. Cerebral spinal meningitis, hemorrhage into the interior of the eye, and arthritis may occur. The detailed description of many of the variations as described by Dieckerhoff are worthy of careful study.

Diagnosis. Brustseuche is diagnosed by its symptoms. There are no satisfactory specific tests. It is to be differentiated from influenza when the localization of the lesions in the lungs does not occur; bronchial pneumonia which often follows colds; and the pneumonia following the introduction of foreign substances into the lungs, as often happens in giving medicines. The history, course of the disease, and the spread to other animals will do much to settle the diagnosis.

Prevention. The well animals should, if possible, be removed at once to other stables. The stalls occupied by the infected animals should be *disinfected* before being used for other horses. Isolation and disinfection are the important factors in checking the spread of this, as of other infectious diseases.

REFERENCES

1. CADÉAC. Contributions à l' étiologie de la pneumonie contagieuse du cheval. *Compt. rend. de la Soc. de Biol.*, 1889, p. 316.

2. FLEMING. Infectious pneumonia of the horse. *The Veterinary Jour.*, Vol. XXXIII, p. 1.

3. GAFFKY. Bericht über die im Königl. Institut für Infektionskrankheiten ausgeführten Untersuchungen über die Brustseuche der Pferde. *Zeitschrift f. Veterinärkunde*, Jahrg. 24, (1912), S. 65.

4. GAFFKY AND LÜHRS. Further investigations of contagious pleuro-pneumonia in the horse. *Zeit. für Veterinärkunde*, 1913, No. 1. Rev. *Cornell Veterinarian*, Vol. IV (1914), p. 49.

5. LEONHARDT. Betrachtungen über die Brustseuche und ihre Bekämpfung mit Rücksicht auf die Untersuchungsergebnisse von Gaffky und Lührs. *Zeit. f. Veterinärkunde*, Jr. 26, S. 305.

6. SCHÜTZ. Die Ursache der Brustseuche der Pferde. *Virchow's Archiv.*, Bd. CVII (1887), S. 356.

7. SCHÜTZ. Die genuine Lungenentzündung der Pferde. *Archiv. fur wissen. u. prak. Thierheilkunde*, Bd. VIII (1881-2), S. 16.

8. SIEDAMGROTZKY. Ueber infectiöse Pneumonien bei Pferden. *Deutsche med. Wochenschrift*, 1882, S. 668.

9. WILLIAMS. Contagious pleuro-pneumonia of the horse. *Amer. Vet. Review*, Vol. XVI (1892), p. 301.

INFECTIOUS ANEMIA IN HORSES

Synonyms: Equine infectious anæmia; anémie épizoötique; typho-anémie; equine malaria; river-bottom disease; loin distemper; mountain fever; swamp fever.

Characterization. Infectious anemia is a specific disease of the horse and mule due to a filterable virus. It occurs sometimes as an acute and at others as a chronic septicæmic-like disease with rapid loss of flesh and pronounced blood changes. It does not attack other species, but according to Carré and Vallée it may be transmitted to the ass.

History. Infectious anemia was first described by Lignée in 1843. Its infectious nature was pointed out by Anginiard in 1859. In 1883 Zschokke reported several cases in Switzerland. Carré and Vallée, 1904–06, carried on extensive studies of this affection. They established the filterability of the virus and showed that it was present in the urine as well as in the blood. Their findings were later confirmed by Ostertag, Marek and Hempel working independently. In America the disease was probably first mentioned by Torrance in 1882 in the province of Manitoba. In 1902 he described it quite fully. Since that time Van Es, Kinsley, Francis and Marsteller, Mack, Mohler and others have published observations on or experiments with it.

The Seyderhelm's in Germany and the Japanese Commission appointed in 1909 to study this disease have recently reported their findings.

Geographical distribution. Infectious anemiá is apparently a wide-spread disease. In France it occurs in many localities but particularly in the valley of the Meuse. It has been observed in Bavaria. Marek has found it in Hungary in three different counties. It exists in Switzerland and probably in Sweden. It seems to be quite wide-spread in western Canada. Udall and Fitch have recently reported it from northern New York. The disease has been found in Wisconsin, South Dakota, Nebraska, Nevada, Kansas and Texas. Two general features stand out prominently: First, *it is a disease of hot weather, and second, it shows a strong predilection for rich damp soils.* Dr. Law makes the statement that it is because of this latter characteristic that the disease has been so often referred to under the name of swamp fever.

Etiology. The specific cause has not been found. Mack describes a small body which he finds inside of the red blood corpuscles. Van Es
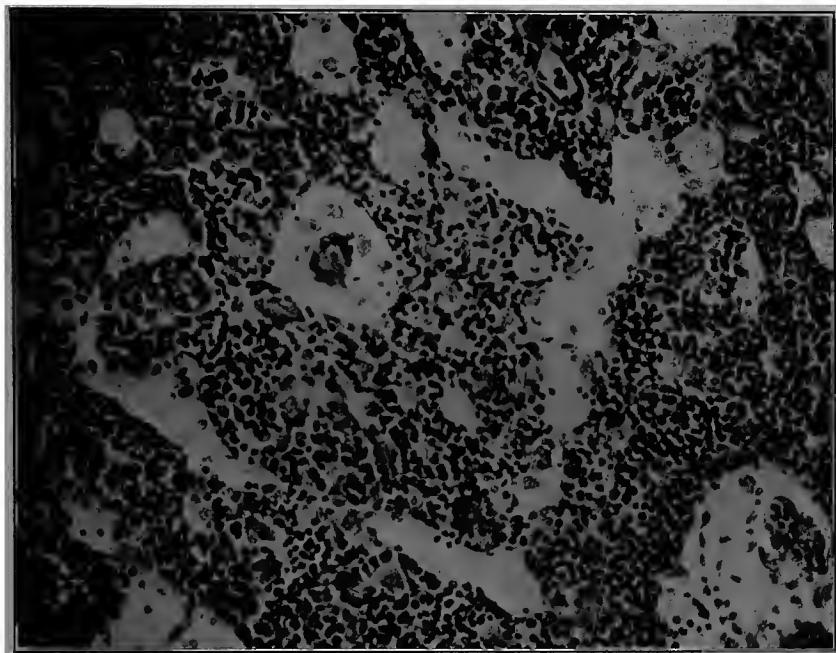
Fig. 104. MICROPHOTOGRAPH OF SECTION OF SPLEEN OF EXPERIMENTAL HORSE SHOW-ING INCREASE IN SIZE OF THE TRABECULAE, AND CONGESTION (*after Udall and Fitch*).
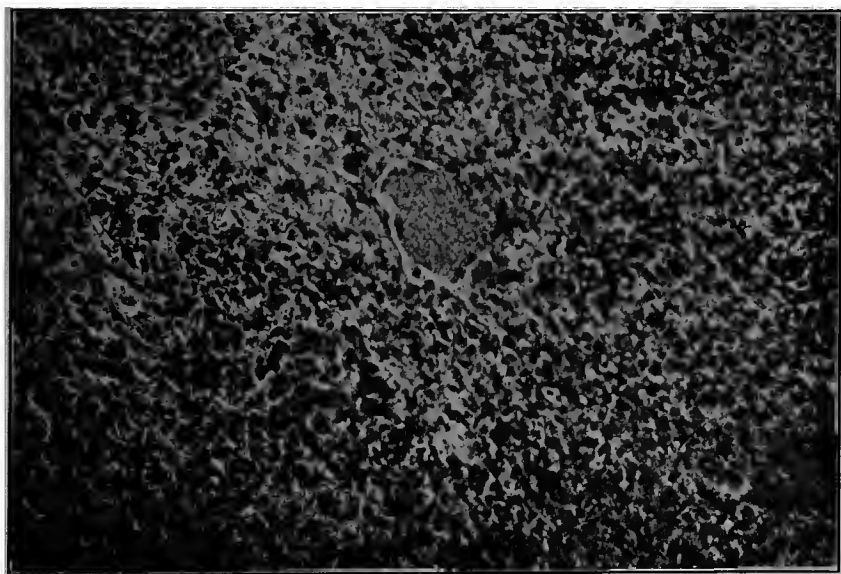
Fig. 105. MICROPHOTOGRAPH OF SECTION OF MESENTERIC LYMPH GLAND OF EXPERI-MENTAL HORSE SHOWING CONGESTION (*after Udall and Fitch*).

mentions several bacteria which he isolated from the cases in Dakota. Udall and Fitch found *B. coli* present in the organs of the animals dead of this disease. Carré and Vallée passed the blood serum of an infected animal through a Pasteur filter and produced the disease by the injection of the filtrate into solipeds. This determined its filterable nature. Francis and Marsteller produced it by the injection of filtrates of blood. This experiment supports the theory that the disease in Europe is the same as that occurring in this country.

The virus is present in the urine as well as in the blood. Carré and Vallée found that it was destroyed by heating at 58° C. for one hour. Drying at room temperature does not alter its virulence. It was only after seven months' drying that the virus was rendered inert. It resists putrefaction for a long time. Van Es states that in natural conditions the virus is able to withstand the climatic influences of our severe northern winters.

The Seyderhelms' and the Japanese Commission found that the disease is produced by the larvæ of bot-flies (Gastrus larva) which occur in large numbers in the stomach and intestines of infected horses. They claim to have produced the disease by the injection of an extract prepared from these larvæ. The Japanese Commission, as a result of their experiments, dismiss "bots" as having nothing to do with the disease. They believe, however, that the biting horse flies (chry-

sopus hematopota and tabanus) are the transmitters of infectious anemia in Japan.

Modes of infection. The disease can be produced artificially in horses by the subcutaneous or intravenous injection of virulent blood, that is, the blood of the sick horse. The size of the dose, as pointed out by Carré and Vallée, does not seem to make any difference. Infection may take place through the digestive tract by contaminated litter and forage. It is important to note that animals which are
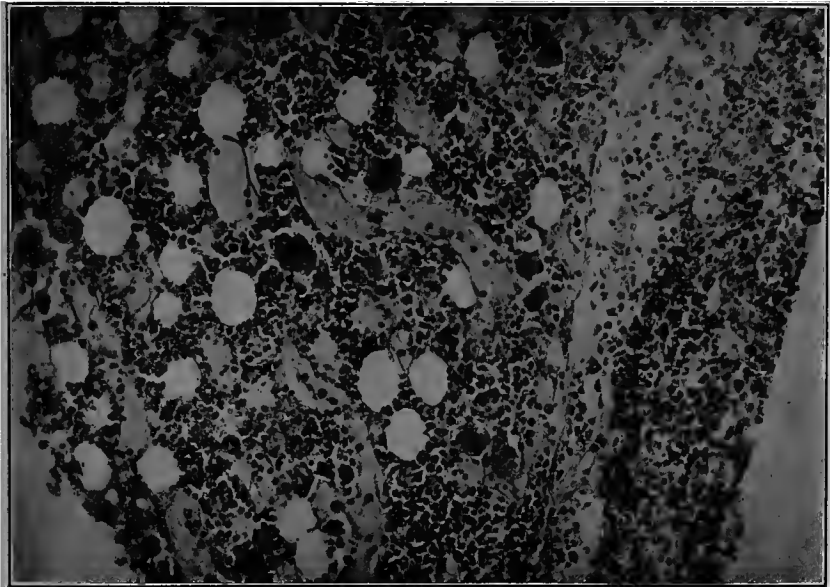
FIG. 107. MICROPHOTOGRAPH OF SECTION OF THE RED BONE MARROW OF THE FEMUR OF A HORSE, SHOWING THE INCREASED ACTIVITY OF THE CELLS OF THE RED BONE MARROW, GIANT CELLS AND CONGESTION (*after Udall and Fitch*).

apparently healthy but which have previously been infected may become "carriers." The disease does not seem to be directly communicable from one horse to another.

Symptoms. In the acute attack there is a high temperature (105° to 107° F.), rapid and weak pulse. Stiffness, swaying gait, usually behind. Udall and Fitch report soreness about the parotid region. Epistaxis is common especially in the later stages. The animals lose weight rapidly. There may be diarrhea with blood stained feces.

These seem to be the most constant symptoms in the acute type which
lasts from a few to fifteen or more days. The mortality is not high
in the first attack. Apparent recovery seems to take place but usually
subsequent attacks occur or a progressive anemia with emaciation
follows. Death follows in from a few weeks to many months. In
some cases years may elapse. The acute stage is usually followed by
the chronic form. The febrile attacks appear at variable intervals
and last for a few days. The mucous membranes become pale.
The urine contains small quantities of albumin. Edematous swellings

FIG. 108. HORSE SUFFERING WITH SWAMP FEVER (*after Udall and Fitch*).

of the extremities may occur. The mortality of the disease is very
high. Few if any infected animals completely recover from it.

Morbid anatomy. The anatomical changes are subject to great
variation. In the acute type the heart shows sub-serous and sub-
pericardial hemorrhages. These may be either single or multiple
and are either petechiæ, ecchymoses or suffusions. The endocardium
also is often studded with petechiæ and the chordæ tendineæ are
enlarged and sometimes edematous. According to Carré and Vallée
this edema may extend to the valves. In the chronic form the myo-

14	15	16	17	18	19	20	21	22
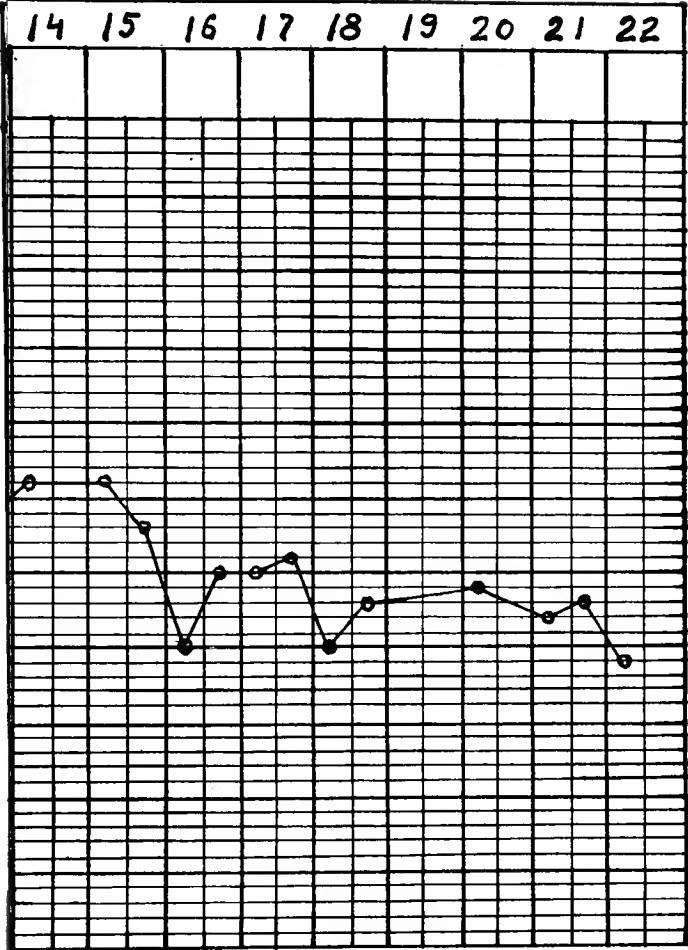
| 42 | 40 | 38 | 40 | 40 | 40 | 44 | 46 | 40 |
| 11 | 10 | 9 | 11 | 10 | 10 | 12 | 15 | 10 |

ter Udall and Fitch).

cardium may be discolored and spotted with rose colored or grayish patches which are the remains of former hemorrhages.

The spleen is usually enlarged. Udall and Fitch observed several where it appeared to be normal in size. In some cases the spleen is harder than normal due undoubtedly to the increase in the connective tissue stroma. The surface of the spleen is sprinkled with petechiæ and ecchymoses. Microscopic examinations of properly stained sections show that the splenic pulp is congested and that in many areas the trabeculæ are apparently enlarged.

The liver is often enlarged and sprinkled with petechiæ on the surface. Carré and Vallée report that it may become so friable that it may rupture if the animal suddenly lies down. In the acute form petechiæ and ecchymoses are present on the surface of the lungs. The kidneys are occasionally congested with petechial hemorrhages. Parenchymatous degeneration is noted especially in the cortical portion. Carré and Vallée state that small abscesses are sometimes found in the cortex. Sub-serous hemorrhages are present in the intestines. The mucous membrane is often hemorrhagic. These changes are present in both the large and small intestines.

The peritoneum is usually congested and may show petechiæ and ecchymoses. The abdominal cavity usually contains varying amounts of more or less sanguineous fluid.

The long bones, particularly the femur and humerus, show the most pronounced lesions. In the proximal ends of these bones the marrow is a brownish or a bright red either wholly or in circumscribed portions. The spongy bone marrow of the ribs and vertebræ show similar changes. These discolored areas are not due to hemorrhages as would be supposed on microscopic examination. But they are as stated by Hutyra and Marek due apparently to the increased activity of the blood forming elements of the red marrow. The spongy bone marrow in some areas shows degeneration and congestion.

The lymph glands, especially the mesenteric, are often enlarged, darker in color than normal and usually congested. There is, according to Mack, Udall and Fitch, an increase in the lymphocytes. The hemoglobin varies.

• Diagnosis. Swamp fever is to be diagnosed by the symptoms which in conjunction with the high mortality, progressive loss in condition and the lesions are fairly conclusive. The positive diagnosis is made by means of a blood inoculation into solipeds. Horses

inoculated with the serum of infected horses will develop the symptoms and usually die in from two to four weeks.

REFERENCES

1. CARRÉ EY VALLEÉ.   Recherches cliniques et expérimentales sur l'anémie pernicieuse du cheval (typho-anémie infectieuse). *Rev. Gén. de Méd. Vét.*, Vol. VIII (1906), p. 593.

2. FRANCIS AND MARSTELLAR.   Infectious anemia of the horse. *Bull. No. 119, Texas Ag. Exp. Sta.*

3. HOARE.   Equine pernicious anemia. *A System of Veterinary Medicine*, Vol. I, p. 927.

4. JAPANESE COMMISSION.   Report on the results obtained by the special committee for investigation of infectious anemia of the horse.   *The Vet. Jour.*, Vol. LXX (1914) p. 604.

5. LIGNÉE.   *Rec. de Méd. Vét.*, 1843, p. 30.

6. MACK.   Equine anemia. *Bul. No. 68, Nevada Ag. Exp. Stat.*

7. MACK.   Intracellular bodies associated with equine anemia. *Proceedings of the Amer. Vet. Med. Asso.*, 1911.

8. MOHLER.   Infectious anemia or swamp fever of horses. *Cir. 138, B. A. I.*, Washington, D. C.

9. SEYDERHELM U. SEYDERHELM.   Wesen, Ursache und Therapie der perniziösen Anämie der Pferde.   *Arch. f. wiss. u. prak. Tier.*, Bd. XLI (1914), S. 50.

10. TORRANCE.   Malarial fever of horses in Manitoba. *Proc. Am.Vet. Med. Asso.*, 1902, p. 282.

11. UDALL AND FITCH.   Preliminary report on the recognition of swamp fever or infectious anemia in New York State. *Cornell Veterinarian*, Vol. V (1915), p. 69.

12. VAN ES, HARRIS AND SCHALK.   Swamp fever in horses. *Bull. 94, N. Dak. Agr. Exp. Sta.*, Fargo, 1911.

ENZOÖTIC CEREBRO-SPINAL MENINGITIS IN HORSES

Synonyms: Crazy disease; Borna disease; "encephalitis."

Characterization.   This is a disease that seems to be infectious, exhibiting symptoms referable to a disturbance in the central nervous system.   Although the literature contains numerous accounts of its seemingly contagious nature, an analysis of the facts fails to bring forth conclusive evidence that it is ever transmitted directly from one horse to another.   In nearly if not all outbreaks, the animals affected have been subjected to like conditions of life.   This disease is, at the present time, peculiar in that its cause is not well defined, that obvious tissue changes are usually absent, and that it has a very high mortality. Jœst described it as an "acute, disseminated, non-suppurative inflammation of the brain and its coverings."

History.   For many years there have appeared from time to time outbreaks among horses of a disease, the symptoms of which suggested brain trouble.   More recent outbreaks have been studied and quite

different results have been obtained. The cause was attributed, at least in many instances, to decomposed fungus infested food and consequently "forage poisoning" became a popular name for these cases. There is a large literature on the subject. The affection in Germany known as "Borna Sickness" has been more carefully studied and its lesions described. They are thought by Friedberger and Fröhner to be different from those of cerebro-spinal meningitis. Johne and Ostertag held the same opinion. Cadéac does not differentiate them. In this country there have been serious outbreaks in which the diagnosis has been in doubt. The work of Jœst and Dengen has clearly defined the Borna disease. Udall considers the disease that occurred in Kansas and Nebraska in 1912, the outbreak of "enzoötic cerebro-spinal meningitis" described by Williams in Idaho, by Milks in Louisiana and by Jœst and Schmidt in Saxony to be the same. He does not think they are identical with the forage poisoning described by Pearson and the anatomical changes are different from those found by McCarthy and Ravenel and designated acute epizoötic leucoencephalitis. There is great need for further investigation into the nature of this class of disorders.

Geographical distribution. This disease seems to be wide-spread. The incomplete differentiation of "forage disorders" from the specific cerebro-spinal meningitis leaves the nature of the disease in many outbreaks still in doubt.

Etiology. The cause has been attributed to a great variety of agents, such as several species of bacteria, fermented food, forage laden with fungi or toxic moulds, various insanitary conditions and other ill-defined agencies. It seems to be true that in most outbreaks all of the animals that suffer have had at least some one thing in common either in surroundings, food or management. The bacteriological examinations have not resulted in finding a specific agent. Micrococci and various bacilli have been isolated from the infected animal. The writer made a careful examination of animals in two outbreaks. In one of them all inoculated media and histological examinations gave negative results, in the other pure cultures of a colon bacillus were obtained from the brain. Jœst found intranuclear bodies in the large cells of the hippocampus and of the ophthalmic tract. They resemble Negri bodies in rabies, but they are located within the nucleus while Negri bodies are extranuclear. Jœst believes the virus gains entrance to the brain through the lymph vessels of the nasal mucous membranes.

Symptoms. A variety of symptoms are reported. Depression with symptoms referable to the nervous system, especially the brain, are reported. The mild attacks may be manifested by paresis or loss of perfect control over the limbs, loss of power over the tail, impairment of appetite and some difficulty in swallowing, together with areas of hyperemia and reddish-brown discoloration of the orbital and nasal mucosæ. In other cases paralysis of one or more limbs may supervene but without marked fever or coma.

The more severe forms are ushered in by violent trembling, or by stupor, apathy and extreme muscular weakness or actual paralysis. In such cases the animal may stagger or fall. The inability to swallow is often a marked symptom, the saliva falling in strings from the lips. Another common phenomenon is the rigid contraction of the muscles of the neck, back and loins, the parts becoming tender to the touch and a more or less prominent opisthotonos setting in. Twitching of the muscles of the shoulders and flanks may be noticed. Trismus is sometimes seen. The breathing is usually rapid and catchy and the temperature ranges from 103° to 106° F. The pulse may be accelerated and hard, soft and weak, or alternating. The eyes are usually violently congested, of a brownish or yellowish-red color, and the eyeballs may be turned to one side. Paroxysms of delirium may set in when the animal will push against the wall or perform any of the disorderly movements following meningo-encephalitis. Sooner or later coma and paralysis supervene and death occurs in from five to forty-eight hours. In the most acute cases the animal falls and dies in convulsions. On an average the disease lasts from eight to fourteen days. In the more favorable cases, improvement may begin on the third or fourth day.

Morbid anatomy. Most writers report lesions of leptomeningitis, hyperemia of the brain and spinal cord, with extensive effusion into the ventricles and subarachnoid spaces. Petechiæ and parenchymatous degeneration of the solid organs of the body are also mentioned. In the cases examined by the writer there has been an absence of macroscopic lesions in the nervous system and other organs that could be detected by a gross examination.

MacCallum and Buckley have found in the brains of horses dying apparently of this disease areas of softening "in the frontal region on each side, anterior to the motor region of the cortex." The neighboring blood vessels were acutely inflamed, with exudation of leucocytes and passage of red corpuscles into the peri-vascular lymph sheath

and adjacent tissue. In a later epizoötic they failed to find the brain lesion but did detect the vascular changes.

McCarthy and Ravenel in a study of fifteen animals found certain lesions in the upper gastro-intestinal tract and in the central nervous system. These were (1) in the intervertebral and Gasserian ganglia where a peri-capsular, small round cell accumulation was present. The cells were all of the same type, the nucleus and protoplasm being about the size of a red corpuscle. There was no evidence that these cells were the result of proliferation of the original layer of capsular cells. (2) Cortical lesions. These consisted of congestion of the cerebellar and cerebral cortex. There were also capillary hemorrhages. The meninges were normal. (3) Changes in the choroid plexus. In three cases the choroid plexus was changed into a triangular, tumor-like mass, of a yellowish red color and of a firm consistency. The increase in size was found to be due to a proliferation of the elastic tissue surrounding the vessels. (4) Changes in the peripheral nerves. There was a distinct degeneration of the nerves supplying the larynx and neck. This was present in the nerve up to the ganglion, but was not found in the posterior roots. Other slight changes were detected.

These authors conclude that this disease is not a true meningitis, but that the evidence goes to show that it is caused by some poisonous substance contained in the forage. They propose the name "forage poisoning" for "cerebro-spinal meningitis" and leucoencephalitis suggested by MacCallum and Buckley. It is highly probable that up to the present time, cases of uncomplicated meningitis and possibly cerebritis have been confused with the disease in question. The entire subject must await the results of further investigation.

Williams investigated an outbreak at Idaho Falls and Milks one in Louisiana. They concluded that the cause was not forage poisoning.

The lesions described by Jœst for Borna disease and found by Udall in the outbreak in Kansas and Nebraska in 1912 were similar and different from those heretofore described. There were no macroscopic characteristic lesions visible on post-mortem. Jœst found: "The principal seat of the lesion is the brain, acute encephalitis being present; slight meningitis is also found, which is probably a secondary condition. The lesions in the spinal cord are less extensive and not so well marked. The vessels of the brain, and to a less extent those of the spinal cord, usually show a marked inflammatory infiltration of their external coats; the perivascular lymph spaces are partly involved. The

substance of the brain and spinal cord shows similar infiltrations widely diffused. The infiltrating cells are chiefly lymphocytes—*i. e.*, they are for the most part derived from the vessels."

Jœst described special bodies termed "nuclear inclusions" which can as a rule be demonstrated "in the olfactory convolution and in the large polymorphic ganglion cells of Ammon's horn." These bodies are not found in any other disease, and are said, when present, to be diagnostic of "Borna sickness." But "in individual cases they
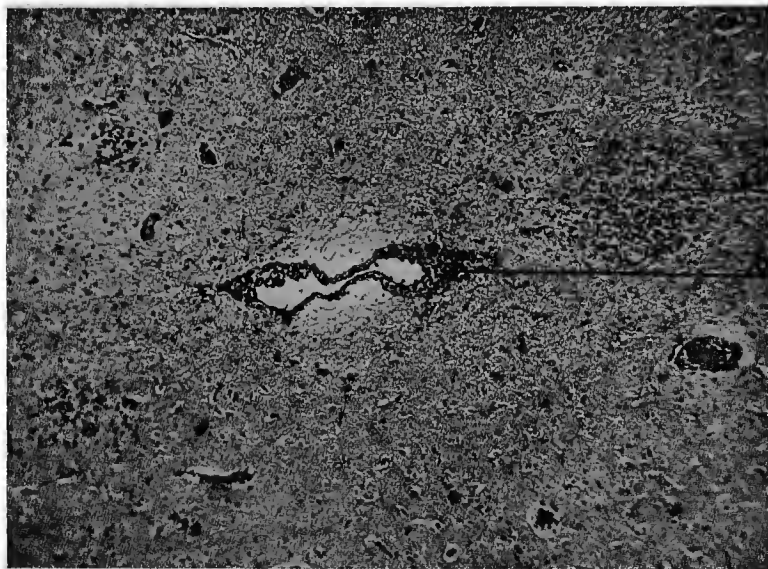
Fig. 109. OLFACTORY TRACT. (*a*) DIFFUSE INTERSTITIAL INFILTRATION, (*b*) PERI-VASCULAR INFILTRATION. x 90 (*after Udall*).

bear no relationship, either as to size or number or as to localization, to the inflammatory infiltration."

The inflammatory process is most marked in the olfactory bulb and olfactory convolution, and next to these in the caudate nucleus and "Ammon's horn." It is believed that it commences in the olfactory lobe, and then extends to other parts, also that the meningitis occurs subsequently to the encephalitis.

Udall found that "the inflammatory infiltrates are undoubtedly due to the local effect of the virus. In considering the possible effect of an actual poison one seeks for the cause in the vicinity of the infiltrates.

The fact that there is no recognizable close local relationship between the inflammatory infiltrates and the intranuclear bodies, in addition to the fact that the ganglion cells containing the bodies are well preserved, does not support the theory that the intranuclear bodies are the cause of the inflammatory changes or of Borna disease.

"The entire histological picture of the disease and the results of the examination of the cerebro-spinal fluid indicate that the genuine infectious process goes on in the nerve substance and its vessels, and that the changes in the meninges are of the nature of an irritation.

"In any case we must regard it as established that the encephalitis (and myelitis) in Borna disease is independent of any disease in the meninges."

Diagnosis. The diagnosis is made by the symptoms. The differential diagnosis is between the various forms of meningitis, rabies and food poisoning. As yet the symptomatology and morbid changes following disorders due to the possible different causes are not determined with the exception of rabies. The evidence seems to be quite conclusive that enzoötic cerebro-spinal meningitis differs from poisoning. The intra-nuclear bodies found by Jœst have not been reported in this country.

Prevention. Until the nature of the virus is more definitely determined preventive measures will remain unsatisfactory. The removal of the well animals from the infected ones may be beneficial. The change of food has been recommended. There is great need for further investigations of this disease.

REFERENCES

1. BUTLER. Notes on a feeding experiment to produce leucoencephalitis in a horse, with positive results. *Am. Vet. Rev.*, Vol. XXVI (1902), p. 748.

2. FAVILLE. So-called spinal meningitis. *Am. Vet. Review*, Vol. XVII (1893–94), p. 9.

3. HARRISON. Cerebro-spinal meningitis. *Am. Vet. Rev.*, Vol. XXVIII (1904), p. 1015.

4. HICKMAN. Epizoötic cerebro-spinal meningitis of horses. *Twenty-third annual report, Bureau of Animal Industry*, 1906.

5. HOARE. Epizoötic cerebro-spinal meningitis in the horse. *A System of Vet. Med.* (Hoare), Vol. I, p. 889.

6. JOEST UND DEGEN. Über eigentümliche Kerneinschlüsse der Ganglienzellen bei der enzoötischen Gehirn-Rückenmarksentzündung der Pferde. *Berl. Tier. Wochens*, Bd. XXVI (1910), S. 300. Abstract, *Journ. of Comp. Path. and Therap.*, Vol. XXIV (1911), p. 369.

7. JOEST UND DEGEN. Untersuchungen über die pathologische Histologie, Pathogenese und postmortale Diagnose der seuchenhaften Gehirn-Ruckenmarksentzündung (Bornaschen Krankheit) des Pferdes. *Zeitsch. fur Infek., parasitare Krankheiten und Hyg. der Haustiere*, Bd. IX (1911), S. 1.

8. JOEST. Enzoötische Gehirn-Rückenmarksentzündung (Bornasche Krankheit) des Pferdes. *Handbuch der pathogenen Mikroorganismen*, Bd. VI (1912).

9. LARGE. Fatal epidemic among horses in America. *The Veterinarian*, Vol. LXVII (1876), p. 655.

10. McCALLUM AND BUCKLEY. Acute epizoötic leucoencephalitis in horses. *Bulletin No. 80, Md. Agric. Exp. Station*, 1902.

11. McCARTHY AND RAVENEL. A pathology for forage poisoning, or the so-called epizoötic cerebro-spinal meningitis of horses. *The Journal of Medical Research*, Vol. X (1903), p. 243.

12. MILKS. A preliminary report on the so-called cerebro-spinal meningitis of horses. *Bul. 106. Ag. Exp. Sta., Baton Rouge, La.* 1908.

13. PEARSON. A preliminary report upon forage posioning of horses (so-called cerebro-spinal meningitis). *Jour. of Comp. Med. and Vet. Archives*, Vol. XXI (1900), p. 654.

14. SCHMIDT. Untersuchungen über das klinische Verhalten der seuchenhaften Gehirnrückenmarksentzündung (Bornaschen Krankheit) des Pferdes nebst Angaben über diese bezügliche therapeutische Versuche. *Berliner Tierarztl. Wochenschr.*, Bd. XXVIII (1912), S. 581.

15. SIEDAMGROTZKY UND SCHLEGEL. Zur Kenntniss der seuchenartigen Cerebrospinalmeningitis der Pferde. *Archiv. f. wiss. u. prakt. Thierheilk.*, Bd. XXII (1896), S. 286.

16. UDALL. A report on the outbreak of "cerebro-spinal meningitis" (encephalitis) in horses in Kansas and Nebraska in 1912. *Cornell Veterinarian*, Vol. III (1913), p. 17.

17. WILLIAMS. Enzoötic cerebro-spinal meningitis in horses, and hog cholera in Idaho. *Rep. B. A. I.*, 1897, p. 179.

CANINE DISTEMPER

Synonyms: Dog plague; dog disease; bench show disease; canine influenza; typhus fever in the dog; typhoid fever in the dog; catarrhal fever.

Characterization. Distemper is an infectious disease appearing in sporadic cases or in epizoötics. It is characterized by a rise of temperature and a catarrh of the mucous membranes. It runs a variable, prolonged course. Frequently there are serious disturbances of the nervous system. It affects carniverous animals. It is caused by a filterable virus.

History. This disease of dogs was known in quite early times. Its history shows that possibly it was introduced into southern Europe from Peru, South America, about the middle of the eighteenth century. It was regarded as being closely allied to a number of diseases of the human species such as the plague and typhus. Trasbot contended that there were at least two, if not three, distinct contagious diseases confounded under the name distemper, namely, true distemper, *la grippe* and a special infectious pneumonia. In 1857, Leblanc described forms of distemper that caused bronchial catarrh, intestinal catarrh, diseases of the nervous system and an eruptive

disease. Urbean Leblanc in 1841 gave it the name of variola. Schantyr stated, in 1891, that canine distemper should be divided into three different diseases and that each is produced by a distinct species of bacteria. In 1899 Jess isolated a bacillus from the catarrhal secretions, blood, serous exudates and organs. He reports having reproduced the disease in dogs with pure cultures of this organism.

Geographical distribution. Distemper in dogs is a wide-spread disease. It is very common throughout America and Europe. It is stated that there is no country or climate in which the dog is exempt from distemper.

Etiology. The specific cause of distemper has not been demonstrated. Ferry and also Torrey and Rahe have studied an organism, *B. bronchisepticus*, which they believe to be the specific cause.

Carré has shown that the serous nasal discharge obtained at the outset of the disease possesses great virulence due to the presence of a virus sufficiently small to pass through certain filters. There is much need for further investigation into the etiology of this malady. Dogs are able to transmit the disease so long as they have any discharge, diarrhea or cough. They are said sometimes to be carriers after all symptoms have disappeared. Gray found that latent cases are often the source of the so-called "spontaneous outbreaks."

The period of incubation varies from 4 to 15 days.* In the so-called latent cases it may be much longer.

Symptoms. The symptoms vary not only in the same epizoötic but at different times to such a degree that it is impossible to refer to all of them. In some cases they suggest a general disorder. In others they are referable to certain parts or organs such as the mucosæ of the digestive and respiratory tracts, the brain or integument. As a rule several organs are implicated.

The initial symptoms such as depression, roughened condition of the coat, loss of appetite and elevation of temperature are suggestive of a general disturbance.

In a large majority of cases, conjunctivitis is the primary ocular symptom. Tears flow from the eyes and photophobia is present. The mucous membrane of the eyelids becomes conjested and a purul-

---

*Krajewski, Mégnin, Laosson, and Babes and Starcovici, compute the period of incubation at 4 to 7 days; Venuta, 4 to 6 days; Trasbot, 5 to 8 days; St.-Yves Menard, 8 to 12 days; Sewell, 4 to 14 days; Nocard and Leclainche, 12 to 15 days; and Hobday, 3 to 4 days to 3 weeks. Jenner states that evidences of illness appear during the second week after exposure to infection.

ent exudate may appear. The exuded matter consists of pasty mucous or dirty yellowish pus. This exudate collects under the lower eyelids, chiefly at the inner canthus of the eye, and soils the edges of the eyelids, upon which it frequently dries and causes the lids to adhere, especially during the night. Ulcers form on the cornea in consequence of the action of the accumulated and decomposing pus and the patient wiping and rubbing the eyes with its paws. The epithelium of the cornea sometimes suffers more or less from shallow flat lesions which give the surface of the cornea a rough and uneven appearance. Frequently smaller and deeper ulcers form especially toward the center of the cornea.

In other cases, there is a diffuse, parenchymatous keratitis which renders the cornea, to a considerable extent, opaque and gives it the appearance of ground glass. These extensive opacities sometimes develop in a comparatively short time. This affection of the cornea, the so-called "distemper of the eyes," is frequently the only evidence of distemper with the exception of the high temperature.

There may be vomiting, well marked congestion and dryness of the oral mucosa. There is usually constipation at first, but later a diarrhea in which the feces, as a rule, are very fetid, often slimy and frothy. Hemorrhagic intestinal catarrh sometimes exists. The urine frequently contains albumin and bile pigment, especially when the patient is weak or in an advanced stage of the disease.

The nasal discharge is serous at first, mucous or purulent later on. It is followed by sneezing, panting and a nasal pruritis, which causes the animal to rub its nose with its paws. The purulent discharge from both nostrils may be very copious. It is sometimes mixed with streaks of blood, and varies in color from a dirty yellow to a dirty green. Later, it may become fetid and even watery. Ulcers may appear on the nasal mucosa.

Laryngeal catarrh usually accompanies the nasal catarrh and manifests itself by a cough, which comes on in paroxysms and which is at first hoarse and dry, but later moist and accompanied by a discharge of phlegm. The cough excites vomiting. The catarrh spreads from the larynx to the trachea and bronchi. The resulting bronchitis is followed by an increased rate of breathing and manifests itself by a cough and hoarse, sharp, vesicular, respiratory murmurs. Frequently there is a catarrh of the mucous membrane of the smaller bronchi. There may be difficulty in breathing and a feeble cough which the patients try to suppress. The cough may be excited by

percussion of the thoracic walls, by the animals getting up and by their being taken out of their kennels. Young and weak animals that cannot remove the accumulated phlegm from the bronchi by coughing become affected with catarrhal pneumonia. The pneumonia can be recognized by the elevation of the temperature.

Distemper often begins, especially in anemic animals, with great depression and dullness. Strong animals, however, more usually exhibit symptoms of acute hyperemia of the brain, such as excitement, restlessness, yelping and even attacks of fury, which give way later on to manifestations of cerebral pressure. Spasms frequently occur either generally or confined to particular limbs which swing backward and forward as if affected by chorea. The animal may fall down as if suffering from epilepsy, bark, become unconscious and exhibit spasms of the muscles generally. The sphincters of the anus and bladder become relaxed and consequently feces and urine are involuntarily passed. There is a gradual return of consciousness which in a short time is complete and the dog manages to get up, although he is very weak. Such an epileptiform attack may pass directly into long continued coma.

Paralysis may follow the convulsions or it may come on simultaneously with them. It seldom occurs at the beginning of the disease. It may be confined to certain groups of muscles, as those of a limb, the whole of the hind quarters or even the entire body in the form of paresis, especially of the motor nerves, combined with excessive muscular weakness. The sick dog staggers and his hind quarters sway from side to side or he becomes incapable of supporting himself on his hind legs. Frequently he knuckles on all four legs and in severe cases is unable to stand. Permanent paresis of the hind quarters with paralysis of the bladder and rectum is a frequent result of distemper. In some cases there may be paralysis of the muscles of the tongue.

A characteristic pustular exanthema is frequently observed on the inner surface of the thighs and abdomen and in abortive cases it may be the only symptom of distemper. It first appears in the form of minute red spots, which after twenty-four hours develop into miliary nodules that are surrounded by a red ring. These nodules change into vesicles and pustules which may become as large as a pea or bean. They dry into a yellowish brown crust, or burst, leaving a raw surface. Healing takes place with desquamation of the epidermis after about eight days, leaving pigmented, pale reddish areas which persist for

some time. Generally, there are only a few pustules present. The exanthema may spread in the form of a scabby eczema over the whole body, to the membrane of the external auditory meatus and less frequently to the mucous membrane of the mouth and eyes. This eruption, contrary to that of sarcoptic mange, is accompanied by only slight pruritis. Intense catarrh of the prepuce may appear simultaneously with the skin eruption.

The temperature, which is usually very irregular, is higher during the initial stage than when local manifestations appear. It often falls with remarkable rapidity below normal towards the approach of death. If the disease runs a protracted course, the patient becomes emaciated and the hair loses its lustre, the body exhalations have a very fetid odor, the eyes are sunken, the mucous membranes become pale and the patient grows weaker, staggers when walking or lies in a state of coma.

The duration of the disease varies. In abortive cases recovery may take place in eight or ten days, although it usually lasts three or four weeks. With severe complications, especially those of the nervous system, distemper assumes a protracted course and is frequently followed by sequelæ, such as paralysis or convulsions at longer or shorter intervals, which may persist for months and even longer. The average mortality appears to be from 50 to 60 per cent.

Morbid anatomy. The variety of symptoms indicates the complicated variation of lesions that may exist. There do not appear to be any tissue changes characteristic of the disease. They vary apparently according to the location of the virus in the body. The anatomical changes in the respiratory system are those of rhinitis, laryngitis, bronchitis and catarrhal pneumonia. The nasal mucous membrane is either very pale or greatly congested, swollen and covered with a thick, purulent, grayish green or dull reddish inflammatory exudate which is mixed with coagulated blood and collects chiefly between the lamellæ of the turbinated bones and in the frontal sinuses. Hemorrhagic ulcers are sometimes present. The mucous membrane of the larynx and bronchi is hyperemic, swollen, often infiltrated with hemorrhages and covered with pus. Sometimes catarrhal ulcers appear. The large bronchial tubes often fail to exhibit changes which might have been expected from the symptoms. The smaller bronchi are on the other hand frequently filled with a dirty gray and even bloody, viscid pus. There are areas of congestion on the surface of the lungs, some parts of which may contain but little

air. There may be areas of collapse or those abnormally filled with air. The inflammatory foci of the lungs are usually consolidated. In very young animals there may be a fibrinous exudate which is very soft and which readily liquefies. The hepatization frequently involves an entire lobe. The hepatized parts are frequently studded with small suppurating foci, or are diffusely infiltrated with pus. The pleura over the affected parts is often inflamed. The bronchial glands are swollen or infiltrated with a serous fluid or with pus.

In the digestive system, the mucous membrane of the stomach and intestines, especially that of the small intestine, is hyperemic and swollen. It may be covered with a tough mucus and is often sprinkled with hemorrhages. In other cases it is very pale, swollen and easily torn. Frequently the contents of the intestine are blood stained and the mesenteric glands enlarged and edematous.

The brain is anemic and often there is a serous effusion into the lateral ventricles and subarachnoid spaces. In a few cases there are signs of a purely venous, cerebral hyperemia, as for instance, great congestion of all the sinuses, venous plexuses and vessels of the pia and the appearance on the cut surfaces of the brain of numerous blood points which can easily be wiped off. Kolesnikoff found microscopically the brain substance, especially the walls of the vessels, infiltrated with leucocytes. Krajewski noticed dilation of the vessels, cellular infiltration of their walls, filling of the perivascular spaces with lymphoid cells and migration of lymphoid cells into the stroma of the brain and into the protoplasm of the ganglionic cells. The changes in the spinal cord, which are not well marked, consist chiefly of anemia and slight edema, especially in the lumbar region. Mazulewitsch states that in acute paralysis there are changes in the walls of vessels, with an exudate along the vessels and in the interstitial tissue of the gray matter of the spinal cord. In chronic distemper, there is a localized interstitial myelitis with partial atrophy of the cord. Hadden found groups of emigrated blood corpuscles in it. In severe cases, according to Trasbot, the spinal cord and its membranes are often considerably injected with a sero-fibrinous exudate in and under the arachnoid and even into the substance of the spinal cord.

Among the other changes which have been described we may mention decrease in the total quantity of the blood of the body, combined with a certain degree of hydremia. There may be fatty degeneration of the liver and kidneys. The muscular tissue of the heart may be

discolored in consequence of cloudy swelling and fatty degeneration of the fibers. The lymph glands may be edematous.

Diagnosis. Distemper is to be diagnosed by the symptoms. There is no specific test for making a positive determination. Distemper is to be differentiated from simple catarrh, rabies, skin diseases, epilepsy, simple coryza, bronchitis, chorea and paralysis.

Until the specific cause is found and can be availed of in making the diagnosis, much doubt will necessarily exist respecting the nature of the disease where many of the symptoms are atypical. It may be found on further investigation and the discovery of the etiological factor that the many symptoms now attributed to distemper may be differentiated into two or more distinct affections.

Prevention. The lack of knowledge concerning the specific cause has rendered it impossible thus far to close all channels of infection, but its spreading can be checked to a considerable degree by isolation and the use of disinfectants.

Care is necessary not to expose dogs either by taking them to infected places or allowing them to mingle with strange canines. Active immunization has been practiced. Dogs were inoculated with the nasal discharge for the purpose of producing a mild form of the disease. Small pox virus has also been used for vaccination against distemper. During recent years, a large number of vaccines have been proposed for immunizing purposes. They do not seem to be satisfactory although great claims have been made for them.*

Infective sarcomata in dogs. Smith and Washburn have described a series of tumors of dogs which spread from animal to animal as the result of coitus. These tumors which varied much in size appeared to start in the mucosa of the vagina. In some cases the walls of the vagina were deeply infiltrated, and the tumors often ulcerated. Death occurred frequently from cachexia or as the result of mechanical obstruction. They were enabled to successfully inoculate the tumors into two dogs. The following are their conclusions:

"The tumors in question are infective round-celled sarcomata occurring in dogs.

"The tumors can be transplanted from the genitals, where they naturally occur, to the subcutaneous tissue of dogs.

---

*Richter has given several of the recent productions a very careful trial. Gray has also tested certain of them with the conclusion that they are useless. M'Fadyean found that a method for bringing about an artificial immunity against distemper has not as yet been discovered. Carré comes to the same conclusion.

"The tumors can be transplanted from subcutaneous tissue to subcutaneous tissue through a series of dogs.

"The tumors after reaching the maximum of growth may disappear spontaneously with or without ulceration.

"The tumors may continue to increase, and may cause death by secondary deposits forming in the viscera.

"If the tumor should disappear the animal is then immune to subsequent inoculation."

Beebe and Ewing have studied these nodular tumors. They have been able to transplant them. From 122 plants by various methods they have produced tumors in 35 cases and there were 12 spontaneous recoveries. The course of the successful implantations is usually quite uniform. When inserted beneath the skin a period of two to three weeks elapsed before enlargement was noticed. Thereafter the nodules grew steadily in size for four or five months producing a well-circumscribed tumor the size of a hen's egg or larger. These authors did not determine the histological classification of the tumor.

REFERENCES

1. BEEBE AND EWING. A study of the so-called infectious lymphosarcoma of dogs. The *Jour. Med. Research*, Vol. XV (1906), p. 209.

2. BLAINE. Canine pathology. 1841.

3. CARRÉ. Sur la "Maladie des Chiens." *Bul. de la Soc. Centr. de Méd. Vét.*, Vol. LIX (1905), p. 335.

4. EDITORIAL. Protective inoculation against distemper. *Journ. of Comp. Path. and Therap.*, Vol. XVII (1904), p. 240.

5. GRAY. Canine distemper. *A System of Vet. Med.*, Edited by Hoare, Vol. I, p. 635.

6. HERTWIG. Krankheiten der Hunde. 1881.

7. JESS. Der Bacillus der Hundestaupe (Febris catarrhalis epizootica canum). *Cent. f. Bak. u. Parasitenk.*, Bd. XXV (1899), S. 541.

8. LIGNIÈRES. Sur la vaccination contre la "Maladie des Chiens." *Bull. Soc. Centr. de Méd. Vét.*, Vol. LIII (1903), p. 340.

9. LIGNIÈRES. La vaccination de la "Maladie des Chiens." Critique des statistiques de MM. Phisalix et Rabieaux. *Bull. Soc. Centr. de Méd. Vét.*, Vol. LIII (1903), p. 377.

10. LIGNIÈRES. La vaccination contre les pasteurelloses. *Comp. Rend. de l' Acad. des Sciences*, Vol. CXXXIV (1902), p. 1169.

11. LIGNIÈRES ET SPITZ. Production d'un sérum polyvalent préventif et curatif contre les pasteurelloses. *Comp. Rend. de l' Acad. des Sciences*, Vol. CXXXIV (1902), p. 1371.

12. NOCKOLDS. Dog distemper. *Am. Vet. Review*, Vol. XXIV (1900), p. 180.

13. PHISALIX. Recherches sur la maladie des chiens. Vaccination du chien contre l'infection expérimentale par la bacille spécifique. *Comp. Rend. des Séances de la Société de Biologie*, Vol. LIII (1901), p. 601.

14. Report of committee formed to carry out experiments with the vaccine of Dr. Phisalix for the prevention of distemper in dogs. *Journ. of Comp. Path. and Therap.*, Vol. XVII (1904), p. 274.

15. TORREY AND RAHE. Studies in canine distemper. *Jour. Med. Research,* Vol. XXVII (1913), p. 291.

16. YOUATT. On vaccination of the dog. *Proc. of the Vet. Med. Asso.,* 1837, p. 60. (Quoted by Gray.)

VARIOLA—POX IN ANIMALS

General consideration. Variola is an acute infectious exanthematous disease affecting man and a number of species of domesticated animals. Variola is used generically, when applied to the disease of lower animals, as the identity of the virus causing the disease in the different species is not positively determined. In man it refers to small pox. Variola presents in old animals a characteristic vesicular, pustular eruption which develops on the skin over the entire body or only over certain parts. In the beginning there is a rise of temperature followed by the eruptions peculiar to the disease.

The papular stage. This commences with the appearance of small red spots resembling flea-bites. These, by proliferation of cells into the papillary layer and *rete mucosum,* are formed into small papules or firm nodules.

The vesicular stage. In this the proliferation of cells increases. They collect in clusters or groups, separated from each other by septa or walls formed chiefly by the epidermic cells. Exudation next takes place, and the lymph collects in these spaces. The vesicle thus differs from that produced by an ordinary vesicant, in which there is but a single sac formed. The summit of the vesicle becomes clear, and in that of cow-pox there is a slight depression or umbilication in the centre. During the next stage, that of suppuration, the septa break down and the umbilication disappears.

The pustular stage. In this the vesicle becomes a pustule, its contents turbid and purulent, and its summit assumes a globular form and a greyish-yellow color. An areola of congestion occurs around the pustules, and the skin between them becomes swollen.

The stage of desiccation. In this the pustule dries up, at first in the form of yellowish scales, which later on become a dark brown crust. Desquamation occurs, and a white shining cicatrix or a brownish-red spot may be left, which is known as a "pit." This results from destruction of the superficial layer of the dermis.

In the development of the tissue changes peculiar to the maturity of the lesions we have the result of the action of the virus itself while the subsequent suppuration of the contents of the vesicles is produced by pyogenic micrococci and streptococci.

Some authors believe that bovine and equine variola are a form of human variola modified by the transmission through animals. Others entertain the opinion that the diseases are separate and independent. Variola affects cattle, sheep, horses, goats, pigs, dogs, buffaloes, camels, monkeys, and man. With the possible exception of sheep pox, the infection of one species can be conveyed to another by accidental infection or by inoculation. Generally speaking, the variola of man and sheep is a serious disease followed by rather high mortality while the pox of other species is usually mild.

Etiology. The cause of variola has not been definitely determined other than that it is known to exist in the contents of the nodules and vesicles and that under sufficient pressure it passes through certain of the porcelain filters, which places it among the ultra-microscopic or filterable viruses. The infectiousness of the blood and of the exhaled air of affected animals during the fever period is doubtful. The secretions and excretions do not seem to contain the virus except where they become mixed with the contents of the vesicles. When inoculated into the skin characteristic pox develops at the point of inoculation in susceptible individuals and in certain instances results in a general eruption. The epithelial layers of the skin and mucous membranes are the most favorable places for the propagation of the pox virus. It seems to be able to enter direct between the epithelial cells and if the virus is injected intravenously or into the trachea it finds its way apparently through the blood stream of the epithelial tissue where it becomes localized.

A large number of microörganisms have been described as having some relation to the cause of variola. Guarnieri believed the cause to be certain enclosures within the epithelial cells. These were called *Cytorhyctes variolae.* There is a large literature on this subject and the conclusions are quite contradictory. Councilman found bodies in the lesions of cow pox and termed them *C. vaccinae.*

While variola in the different species presents like manifestations and while it is believed that their etiological factor is either identical or very closely related the disease in each of the different species should be briefly mentioned.

Immunity. Individuals that recover from attacks of variola are immune to subsequent attacks for several years. This is true whether the infection was naturally or artificially produced. Reciprocal immunization in the different forms of pox exists to a certain extent.

Experiments have shown that horses and cattle may be immunized with variola against cow pox and man with vaccinia against variola but a similar reciprocal action between the diseases of man and sheep does not seem to exist.   .

COW POX

Synonym: Vaccina.

Characterization. Cow pox is an acute infectious disease affecting chiefly milch cows, characterized by slight temperature disturbances and the appearance of an eruption on the skin of the mammary gland and teats which passes through the stages of papule, vesicle and pustule scab. It is transmitted to the horse and man by actual contact or by inoculation. When inoculated into man it produces a more or less permanent protection against small pox. This fact led Jenner in 1796 to introduce vaccination of the human species with the virus of cow pox to protect them against small pox.

Etiology. The virus of cow pox has been passed through the porcelain filters by Negri, Remlinger and others. Proscher reports that he has cultivated the virus on artificial media, prepared especially for this purpose. His results do not seem to have been confirmed. It resists drying for several weeks but is destroyed at a temperature of 57.5° C. in five minutes. It is not affected by freezing. When mixed with glycerin it will remain virulent from 8 to 10 months. Natural infection generally takes place during milking when the virus is usually transmitted from one cow to another on the hands of the milkers.

The period of incubation is from 4 to 7 days when the infection is contracted in the usual manner. After direct inoculation it is from 2 to 4 days.

Symptoms. The first evidence of cow pox is a slight rise in temperature, impaired appetite, irregular rumination and weakness. In many cases these symptoms are exceedingly slight. The udder becomes somewhat sensitive, the milk is said to be thinner and to have a lower specific gravity and to coagulate more quickly. The eruption is described under the morbid lesions.

The duration varies up to 30 or 40 days depending upon the number of successions of vesicles.

Morbid anatomy. On the slightly warmer and swollen teats as well as on the adjoining parts of the udder, nodules ranging from 1 to 5

mm. in diameter appear on the second or third day. These change in from 1 to 2 days into somewhat larger vesicles filled with a clear lymph. The vesicles are reddish or bluish or yellowish white with a mother of pearl or metallic lustre, depending upon the thickness of the skin and its color. On the body of the udder the vesicles are uniformly round while on the teats they are oval, surrounded by a red zone of 1 to 2 mm. in width. From the 8th to the 11th day they show a well defined depression. This may be absent. Following this, the contents of the vesicles become purulent, dry up and form scabs. After the scabs drop off the underlying skin is reddened and slightly swollen but later shallow cicatricial depressions are formed. Usually there are but few vesicles, 15 to 20, and they do not all appear at the same time but at intervals of a few days between the first and last. Sometimes two weeks may elapse before the development of all of the vesicles. Rarely a parenchymatous mastitis may be associated with the skin lesions. Usually the eruptions are restricted to the udder and teats. When cow pox is transmitted to the horse it is said to be liable to produce generalized eruptions. Milk from infected cows is liable to be contaminated from the pustules and capable of transmitting the disease. Milkers of infected cows are liable to be infected unless they have been previously vaccinated.

Diagnosis. The diagnosis of cow pox is to be made on the presence of the roundish or oval vesicles containing a depression in the center and the spreading of the eruption to other animals. It is to be differentiated from foot-and-mouth disease and from other eruptions of the skin. These have been grouped under the name of "false cow pox" by Ceely and Hering. Gamgee described *varicella* or chicken pox as occurring in cows.

Immunization. Efforts have been made to immunize cattle against this disease by vaccinating them with calf lymph used for man against small pox. The results are not uniformly successful.

SHEEP POX

Synonyms: Variola ovina; clavelée; *Schaf-pocke*.

Characterization. Sheep pox is an acute exanthematous disease peculiar to sheep, characterized by a typical variolous eruption on those parts of the body either devoid of wool or scantily covered with it.

History. Sheep pox is supposed to have been introduced from Central Asia. It was extensively studied by Joubert and Rabelais

toward the end of the 16th century. Its infectiousness was established by Bourgelait in 1763 at which time it was very widely distributed in Europe. Its etiology has been studied, especially by Chaveau in 1866 and Borrée in 1902.

Geographical distribution. Sheep pox is quite widely distributed in Europe, Asia and Africa. It is reported to have appeared for the first time in Great Britain in 1847, having been brought there from Denmark. The outbreak lasted for four years and caused heavy losses. It is stated by Hutyra and Marek to have been eradicated from Northern Europe but that it still exists in France and in the countries lying south and east where it is of considerable economic importance.

Etiology. The cause is believed to be a virus which is able to pass through the Berkefeld filter but not through the Chamberland F. As in other variolas, it is contained within the vesicles, pustules and scabs. Nocard found it was restricted to these and that the blood was not virulent. Ostertag and others have found that the disease was transmissible by blood inoculation. According to Ostertag sheep are sometimes capable of transmitting the infection for three months after recovery. He believed the virus was retained in the wool. It is destroyed promptly by direct sunlight, high temperature and putrefaction.

Duclert found that the virus was attenuated when kept in glycerin. A mixture of equal parts of glycerin and lymph from the vesicles became non-virulent in 12 days when kept at a temperature of 25° C. Its virulence is attenuated at a temperature of 48° C.

Natural infection is supposed to take place through the respiratory tract, the virus passing into the circulation and carried by this means to the skin and mucous membrane. According to Nocard the disease can be invariably produced by injecting the virus into the trachea. Metastatic abscesses in the joints and meninges have been reported. Shedding of the hoofs has been observed. Suppuration of the lymphatic glands, subcutaneous abscesses, ulceration of the mucous membranes, septic pneumonia, ulceration and gangrene of the nasal bone and cartilage have been reported.

The period of incubation is reported to be from 2 to 20 days. Ostertag found it to be from 8 to 12 days.

Symptoms. The first symptoms are a rise of temperature, catarrh, dullness, loss of appetite with acceleration of the pulse and respiration.

These are followed in from 1 to 2 days by the appearance of round red spots upon the skin which in some places may be surrounded by moderate serous infiltration manifested by an urticaria-like elevation.

With the development of the eruption, there are more or less severe general symptoms. Frequently atypical symptoms and vesicles occur. At times the eruptions do not develop into vesicles. Hoare divides the symptoms into the discrete, regular or benign; the confluent, irregular or malignant; and variola hemorrhagica.

In addition to the somewhat characteristic skin eruptions there is frequently hemorrhagic inflammation of the air passages and gastrointestinal canal. Vesicles sometimes appear on the mucous membrane of the pharynx and trachea. Changes suggestive of pyemia or bacteriemia may be found. Occasionally there are large hard nodules under the skin giving a homogeneous appearance upon section.

The duration of the disease is from 3 to 4 weeks. The mortality varies from about 7% in the discrete form to 20 or 30% in the malignant. Occasionally the mortality is much higher than this.

Morbid anatomy. The vesicles attain to a size of from 5 to 10 mm. A tenacious fluid oozes from the surface of these nodules. The superficial layers are split and the space filled with a serous fluid which develops into a bluish white vesicle. These continue to develop. It may require several days for all of the vesicles to mature. In the center there is a depression or navel which is said never to be absent. The closer the vesicles are to each other the smaller they are in size. Around the vesicles, the skin becomes hyperemic and the subcutaneous tissue edematous. In from 2 to 3 days, the liquid in the vesicles becomes cloudy, owing to the presence of pus cells. Later they become white and the navel-like depression may disappear or the edema about them may increase. The pustule form lasts about 3 days.

Diagnosis. Sheep pox is to be diagnosed by the symptoms and lesions. The characteristic exanthema renders it not difficult in typical cases. It is to be differentiated from blue tongue occurring in Southern Africa, from scabies and other forms of eczema.

Prevention. Ostertag recommends the slaughter of all infected and suspected animals and the quarantine of farms and areas for a period of three months. The carcasses of the destroyed animals should be burned. The period of quarantine may be reduced by dipping the sheep in a 25% creolin or creosote soap solution. In Algiers a vaccine is prepared from the large pustules.

Soulié and Emery have adopted an intradermic inoculation of diluted virus of which they report good results. Sheep cannot be imported from Algiers into France until they have been inoculated (ovination). Duclert found, in 1896, that blood serum of immune sheep has a protective value in lambs. Borrel recommends the simultaneous injection of immune serum and virulent lymph.

HORSE POX

Synonyms: Variola equina; *vaccine du cheval; Pferde-pocke.*

Characterization. Horse pox is an exanthematous disease of the horse characterized by a slight febrile disturbance and the appearance of typical variolous eruptions.

History. Jenner recognized this disease near the close of the 18th century in the valley of the Severn. It has appeared at various times in France and Germany. In 1877 an outbreak occurred in Montreal and was described by Dr. McEachran in the Veterinary Journal in August, 1877. At that time several hundred horses were affected. It is now considered a rare disease in horses. Many of the French authorities and also Hunting consider equine variola and contagious pustular stomatitis to be identical.

Etiology. The virus of horse pox is contained in the skin lesions. Infection is said to take place by contact and by articles infected from diseased horses.

Symptoms. The first symptom is that of disturbed temperature with an eruption on the mucous membranes of the mouth, lips or nasal mucosa or on the skin of different parts of the body. It usually appears in the hollow of the pastern. The skin becomes swollen and sensitive followed by the development of the papules, vesicles and pustules. It is a mild disease that runs a definite course.

Morbid anatomy. The essential changes consist in the development of the lesions characteristic of variola. The lesions may be restricted to one limb or they may be on two or more. There may be considerable swelling and suppuration of the subcutaneous tissue according to reports. When the lesions are on the mucous membrane of the mouth the vesicles are ruptured by the friction during mastication, and small depressed erosions on the mucous membranes occur. Petechial hemorrhages have been reported.

Diagnosis. Horse pox is to be diagnosed by the symptoms and variolar eruptions. It is to be differentiated from the condition

known as "grease" when on the legs; from glanders when located in the nasal passages; contagious pustular stomatitis when in the mouth; strangles; and other eruptions of the skin.

GOAT POX

Synonyms: Variola caprina; *Variola de chèvre; Pockenkrankheit bei Ziegen.*

Characterization. Variola of the goat is a disease peculiar to this species. It is not transmitted to sheep nor sheep pox to goats. It attacks males, kids and females, especially during the period of lactation.

History. Goat pox has often been encountered in Norway. Hansen has described a number of outbreaks in that country. It has also been found in Algeria, in Spain and in Persia.

Etiology. The virus is transmitted with the saliva of the infected animals. It is transmitted by direct contact. Cutaneous inoculations result in the development of pox eruptions associated with mild general symptoms. In some cases sheep have been successfully inoculated experimentally. The disease spreads rapidly when introduced into a flock.

Symptoms. The manifestations resemble very closely those of sheep pox. The eruptions are characteristic. These appear on the mouth, mammary gland, abdomen, inside of the thighs and occasionally on other parts.

The period of incubation is from 3 to 7 days.

Its period of duration is from 1 to 3 weeks. The mortality is low.

Morbid anatomy. In addition to the vesicles and pustules, already described, pustules sometimes occur on the mucous membrane of the stomach and intestine. Occasionally the pustules are confluent. A phlegmonous mastitis is sometimes present.

Diagnosis. The diagnosis is made by the symptoms and eruptions. It is to be differentiated from foot-and-mouth disease and from other skin eruptions.

SWINE POX

Synonym: Variola sulla.

Characterization. Swine occasionally suffer from a contagious eruptive disease characterized by the variolous eruptions. It has not

been trasmitted to the horse or to the cow but has been transmitted to man, sheep and goats. According to Gerlach and Bollinger, sheep pox may be transmitted to the pig, and Nocard states that human variola may be transmitted to swine. According to Hoare, the eruptions appear on the back, abdomen, chest, neck, head and inner surface of the thighs. The lesions first appear as petechial spots, which soon become papules, and on the 5th or 6th day vesicles and on the 9th or 10th day pustules. The pustules form crusts or scabs which are thrown off in a few days leaving a well defined cicatrix.

DOG POX

Synonym: Variola canina.

Characterization. Youatt has described a disease of dogs characterized by the formation of small red spots irregularly rounded on the abdomen, inside of the groin and forearm. They are sometimes isolated and sometimes clustered together. There is a rise of temperature and on the second day the spots become larger and the integument tumefied at the center of each. The third day the spots are generally larger and the skin more prominent at the center of the spots. This is increased on the 4th day. On the following days the pustules take on their peculiar characteristic appearance. The variation in the period of eruption depends largely upon the age. This was considered by Youatt as rather characteristic of variola.

Dupuis of the Brussels veterinary school made an investigation of vaccine in dogs in 1883–5 from which he drew the following conclusions:

"That vaccine is transmissible to dogs with its specific characters.

"That the first vaccinal inoculation generally preserves against the second.

"That intravenous, subcutaneous and peritoneal injection gives immunity against the vaccine without the development of any experimental manifestations.

"That the vaccine cannot by any means be considered a certain means of preserving the dog against distemper."

Recent investigations on this subject have not been noted.

REFERENCES

1. BERRY. Contagious pustular dermatitis of sheep.
2. BORREL. Étude expérimentale de la clavelée filtration du virus; Sero-clavelisation; Serothérapie. *Ann. de l' Inst. Pasteur*, Vol. XVII (1903), p. 123.

3. COUNCILMAN, MAGRATH AND BRINCKERHOFF. The pathological anatomy and histology of variola. *The Journal of Medical Research*, Vol. VI (1904), p. 12.

4. CRUICKSHANK. A note on cow pox in man. *Brit. Med. Journ.*, Vol. I (1910), p. 984.

5. DUPUIS. Quelques cas de cow-pox: Eruption généralisée. *Annales de Méd. Vét.*, Vol. XXXVIII (1889), p. 183.

6. EBER. Schafpocken mit atypischem Verlauf. *Deutsche Tierärztl. Wochenschr.*, Vol. XIV (1906), S. 4.

7. EILERTS DE HAAN. Vaccine et rétrovaccine à Batavia. *Annales de l' Inst. Pasteur*, Vol. X (1896), p. 169.

8. HIME. Successful transformation of small-pox into cow-pox. *British Medical Journal*, 1892, p. 116.

9. KAEMPFER. Kurze Mittheilung über eine Kuhpockenepidemie met Uebertragung auf den Menschen. *Deutsche Med. Wochenschr.*, 1896.

10. KLEINPAUL. Die Schafpockenseuche im Kreise Johannisburg im Jahre, 1905. *Berl. Tierärztl. Wochenschr.*, 1905, p. 778.

11. MOREL ET VALLÉE. Contribution à l' étude anatomo-pathologique de la clavelée. *Arch. de Méd. Expérim.*, 1900, p. 341.

12. NOCARD. Études expérimentales sur la clavelée. *Bulletin de la Société Cent. Méd. Vét.*, 1899, p. 263.

13. SIMPSON. Vaccinia produced by passing the vaccine of small-pox. *Indian Medical Gazette*, 1896, p. 205.

DIPHTHERIA IN FOWLS

Synonyms. Roup*; pip; canker; swelled head.

Characterization. Diphtheria of birds is an infectious disease the lesions of which first appear on one or more of the mucous membranes of the head and which may extend to the trachea, bronchi, the air sacs, the intestines and possibly to other abdominal organs. The disease is determined by a grayish-yellow, fibrinous exudate or diphtheritic pseudo-membranes which form upon the mucous surface of one or more of the parts mentioned. The exudate may be so abundant as to obstruct the air passages. In some outbreaks, the disease is very acute, progresses with great rapidity and destroys most of the birds attacked. At other times the mortality is not high.

Ratz concludes from his investigation that the etiology of chicken diphtheria and chicken pox is the same and that the lesions are different manifestations of the same disease.**

---

*The origin of this term is somewhat obscure, but it is supposed to be a corruption of croup, and its application explained on account of a peculiar hoarseness accompanying the respiration of the affected birds.

**The disease commonly known as chicken pox consisting of epithelial nodules on the skin especially of the comb and wattles is believed by some workers to be identical etiologically with avian diphtheria. Carnwath produced the diphtheritic lesions on the mucosa with chicken pox material and pox nodules with diphtheritic material. The writer's experience has not confirmed the findings of those who believe in the identity of the two affections. Because of doubt on this point, chicken pox is treated as a separate disease.

Fowls (genus *Gallus*) and pigeons (genus *Columba*) are most commonly attacked and they are the only ones considered in this discussion. Avian diphtheria is reported, however, to attack turkeys, ducks, pea-fowls, pigeons and pheasants. It is presumed that wild birds may be affected.

Avian diphtheria is quite distinct from human diphtheria. There are cases on record, however, which indicate that the diphtheria of fowls may be communicated to children and cause a serious and even fatal sore throat. It is also asserted that diphtheria of children is sometimes communicated to fowls and that the virus may be thus preserved for a considerable time and again be transmitted to children. Concerning this point further investigations are needed.

History. The history of this disease is somewhat obscure. It is evident from the literature, that fowls have always been subject to various affections of the head but the first investigation of this class of maladies seems to have been made by Lœffler* in 1884. Since that time Klemmer†, Babes and Puscarin‡, Eberlin||, Loir and Ducloux¶ and others have studied diseases known as diphtheria in pigeons, fowls and other birds. The disease was investigated by the Bureau of Animal Industry in 1893–4. It has been studied in California by Ward, in New York by Mack, and at Guelph, Ontario, by Harrison and Streit.

Geographical distribution. Chicken diphtheria seems to be more or less common in countries where poultry raising is an industry.

Etiology. In 1884, Lœffler discovered a bacterium which he believed to be the specific cause of diphtheria in fowls and with which he could produce the disease. It differed from the bacterium of diphtheria in man. Loir and Ducloux isolated a still different organism. Moore found in the exudates of the earlier stages of the disease a bacterium belonging to the septicemia hemorrhagica group. It was rapidly fatal to rabbits but the diphtheritic lesions could not be produced by inoculation in fowls. King found a bacterium belonging to this group on the conjunctiva of a healthy fowl. Harrison and Streit described an organism, *Bacillus cacosmus*, which they consider specific.

---

*Mitt. aus dem Kaiserlichen Gesundheitsamte. Bd. II (1884), S. 214.
†Berliner thierärzt. Wochenschrift. 1890, No. 18, S. 138.
‡Zeitschrift f. Hygiene. Bd. VIII (1890), S. 374.
||Monatshefte f. Thierheilkunde. Bd. V. (1894), S. 433.
¶Ann. de l' Inst. Pasteur. Tome VIII (1894), p. 599.

Ps. pyocyaneus has been obtained in pure culture from the exudates. Mack failed to produce the disease with *B. cacosmus*.

Guérin considers it a general disease caused by a coccobacillus which is not unlike the bacterium of septicemia hemorrhagica. He finds it in the blood and organs. Those who believe that chicken pox is identical with roup believe the cause to be a *filterable virus*. It has been suggested that the filterable virus is not very virulent but that the secondary bacterial invaders are largely the cause of the lesions.

Roup is usually introduced into a flock by the exposure of the birds to sick ones at shows or by bringing affected fowls on the premises.

FIG. 110. FOWL SHOWING EYE CLOSED. THE CONJUNCTIVA IS COVERED WITH A THICK EXUDATE (*Ward*).

The contagion may be carried by birds which have the disease in so mild a form that they show no symptoms of it. There is a general belief that the disease may be developed by exposure to draughts or by keeping fowls in damp, filthy and badly-ventilated houses. There seems to be confusion concerning the early symptoms of acute diphtheria and those of all stages of the chronic form, with those of simple colds and catarrhs. Ward was unable to produce the disease by exposing fowls to unfavorable conditions, but when infected fowls were

introduced the disease spread rapidly.   Dampness and lack of ventilation no doubt favor the maintenance of the virus when introduced. Experiments with exudates from affected fowls have been successful in some cases.   The positive results seem ¡to have been where the epithelial nodules were present.

Symptoms.   There is a watery secretion from the nostrils and often from the eyes, with general weakness and prostration greater than would be expected from simple catarrh.   The birds sit with the back arched, the head and neck drawn towards the body, the plumage roughened, respirations rapid and audible, vision impaired, and difficult swallowing.   There is frequent shaking of the head, sneezing

FIG. 111.   FOWL SHOWING THE SUBORBITAL SINUS DISTENDED.   THE EYE IS PARTIALLY CLOSED.

and expulsion of mucous secretions.   At this early period the tongue is pale, while small grayish spots, shaded with black and slightly projecting above the surface, may be seen along the border, the upper surface or at the base.

The following day the condition is aggravated, the temperature is several degrees above normal, the appetite has disappeared and there is diarrhea with greenish or yellowish evacuations.   From the open beak there escapes a thick, stringy, grayish mucus.   The eyes project and possibly the conjunctiva may be covered with a thick exudate which has accumulated between the lids.   The nostrils are obstructed by the thickened and dried exudate.   Walking is irregular and diffi-
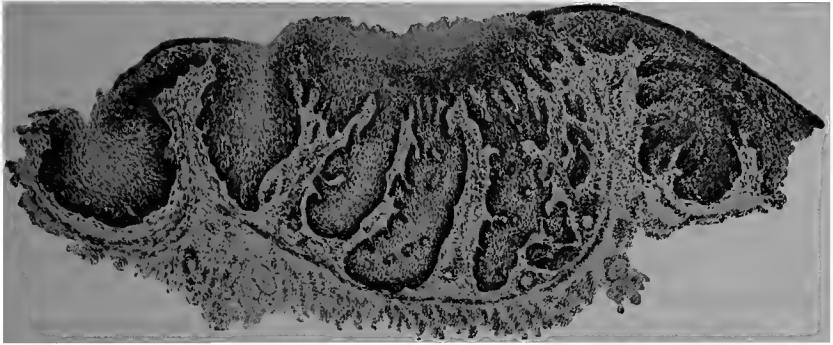
FIG. 112. EARLY STAGES OF DIPHTHERITIC NECROSIS IN THE THROAT OF A PIGEON.

cult. The mucous membranes of the mouth and pharynx are congested and show numerous dark red elevations covered with fibrinous exudate. The patches on the tongue have increased in size, they are gray in color, dried along the edges of the tongue but soft and flattened upon its upper surface. They are covered with a membranous exudate. The voice often fails.

In the acute cases, recovery or death follows in a few days. In chronic ones the fowl may live for weeks or months.

Morbid anatomy. The lesions are largely localized on the mucosa of the head. With the exception of emaciation, there are no general
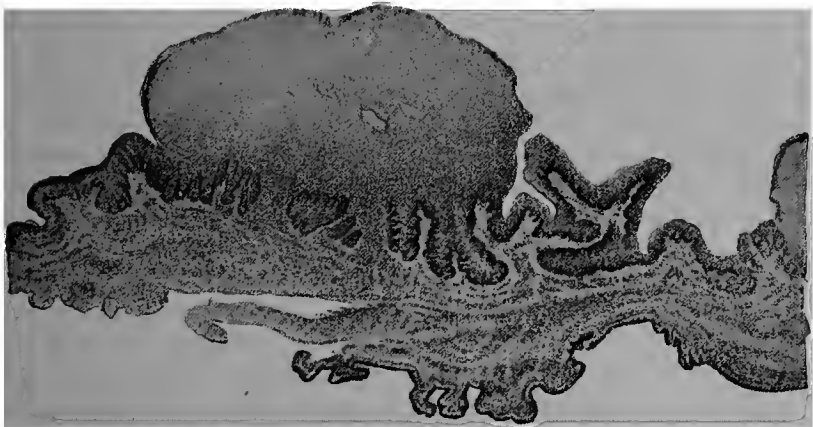
FIG. 113. A SECTION THROUGH A DIPHTHERITIC EXUDATE, LATE IN THE COURSE OF THE DISEASE, FROM A PIGEON'S THROAT.

lesions in most cases. The cause of death and the extreme emaciation are difficult to explain in those cases where the lesions are confined to one eye or to the mucosa of the nares, excepting on the supposition that some poisonous or toxic substance was absorbed. In those cases where the lesions are in both eyes, or in the mouth and throat, difficulty in finding or swallowing food affords a rational explanation.

In some cases the exudate is of a croupous character, in others of a diphtheritic nature. Three stages or varieties of lesions, which represent the types of this disease as encountered in this country, may be more definitely defined as follows:

An exudate of a serous or muco-purulent character in the conjunctiva and nasal cavities. Ordinarily this condition cannot be recognized in the mouth. The mucosa in these cases is apparently but slightly altered.

The mucosa over a small or larger area is covered with a spreading exudate of a grayish or yellowish color. It is firmly attached to the mucous membrane and when removed leaves a raw, bleeding surface. Sections through this exudate and the subjacent tissues show that the epithelial layer is destroyed and the underlying tissue infiltrated with cells. The extent of the infiltration varies in different individuals.

The mucosa is covered with a thick mass of exudate, varying in color from a milky white to a lemon yellow or brown. It is easily removed, leaving a more or less granular and healed surface. This sloughed mass is frequently dried at its margins to the adjacent tissue. It emits a strong putrid odor, due to decomposition. The drying of the margins prevents the fowl from expelling the exudate after it becomes separated from the underlying tissue.

The evidence to support the supposition that the three forms or types of exudate described are different stages in the same morbid process, as gathered from the post-mortem notes and bacteriological study of the cases investigated, may be summarized as follows:

Abnormal conditions, representing the intermediate and connecting links between the types of lesions, are frequently encountered.

Although at the time of examination (post-mortem) but one form of exudate is usually present in a single fowl, there are exceptions, in which two and occasionally the three forms are coincident. Thus the eye is covered with a sloughed exudate, the posterior nares contains a layer of muco-purulent substance and on the mucosa of the mouth are areas of diphtheritic exudate.

In fowls that die, the exudates are for the greater part in the advanced stage, although fatal cases occur in which the lesions are restricted to an abnormal quantity of a serous or muco-purulent, more or less viscid, exudate in the conjunctiva or nasal cavities. The best illustration of the diphtheritic process is found in fowls killed for examination in the second stage of the disease. The distribution of the lesions shows that the conjunctiva is most frequently affected. The exudate in the nasal cavities is in some cases undoubtedly the result of the coagulation of the liquid which has passed during the course of the first stage from the conjunctiva through the lachrymal duct into the nares. In certain cases, how-ever, the lesions appear in the nares primarily. In some cases the exudate appears in the larynx and extends down into the trachea. In these cases the fowls are liable to die from suffo-cation. It occasionally happens that the lesions are restricted to the larynx and as the fowls die suddenly the cause of death is not suspected. Sections of the exudate with sub-jacent tissues from the cornea and the mouth show that there is a cell infiltration into the mucosa which destroys the epithelial layer and frequently the submucous tissues to a considerable depth.

The fact should not be overlooked that the disease in the eye is usually confined to the conjunctiva and the cornea, the posterior por-tion remaining apparently normal.

Fig. 114.   A DRAWING SHOWING AREAS OF DIPH-THERITIC EXUDATE IN THE THROAT OF A PIGEON.

Mack in his work on thirty - three cases found 40 per cent. had lesions in the con-junctiva; in 44 per cent. the nasal mucosa was affected; in 41 per cent. the mouth was involved and in 33 per cent. the suborbital sinuses were distended with exudates.

From the observations thus far made the provisional theory is entertained that the three forms of the exudate—serous or muco-purulent, diphtheritic and sloughed mass—represent three stages in the course of the same disease. It is easily understood that fowls examined in the first stage would be said to be affected with a catarrh of the mucosæ of the eyes or nares. It is highly probable that in many cases the disease never reaches the second stage and if these

cases alone were examined the diphtheritic condition would not be
suspected. It appears, however, that in the majority of cases the
disease runs its course and membranes are formed, slough and recov-
ery follows. It is furthermore presumable that the disease in question
appears sometimes in a virulent and destructive form. Statements
from poultry raisers show that there are occasionally epizoötics
characterized by exudates in the eyes, nose or mouth, which terminate
fatally. It appears that it is such outbreaks that have been reported
in Europe as diphtheria and not the low form of chronic inflammation
which has been studied in this country.
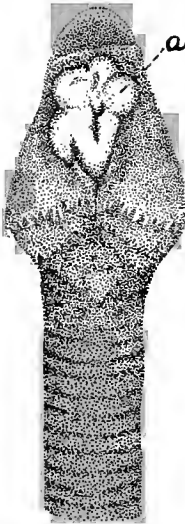
FIG. 115.   DIPHTHERITIC EXU-
DATE IN THE LARYNX OF A
FOWL: (a) THE GRAYISH-
WHITE EXUDATE PROJECTING
FROM THE GLOTTIS.

FIG. 116.   A   LONGITUDINAL
S E C T I O N   THROUGH   THE
LARYNX AND TRACHEA OF A
FOWL (SAME AS 118), SHOW-
ING THE EXUDATE: (a) IN
THE LARYNX AND (b) IN THE
TRACHEA.

Diagnosis. Fowl diphtheria is to be diagnosed by the symptoms
and lesions. There are no specific tests that are satisfactory. It is to
be differentiated from lesions of the head caused by injuries or other
infections, and chicken pox. Manson's eye worm (Oxyspirura
Mansoni) of chickens produces lesions that might be mistaken for
roup. The finding of this worm would determine the diagnosis.

Relation of diphtheria in man to that in fowls. According to a few
writers especially Nocard and Leclainche there are cases of diphtheritic

angina in children traced to this disease in fowls. Although these maladies are shown to be unlike in their etiology and the character of their lesions, the transmission of fowl diphtheria to the human species, and vice versa, is affirmed by several observers.*

Prevention. In order to prevent roup it is evident that many conditions must be strictly complied with. The character of the food and the general sanitary conditions, including cleanliness, ventilation and the temperature of the poultry houses, must be considered. Undoubtedly there is much to be learned in connection with the proper sanitary care of poultry. In addition to the general sanitary methods the following rules should be observed.

Fowls which have an exudate on any of the mucous membranes of the head, or which have come from flocks in which such a disease exists, or has recently existed, should not be placed among healthy poultry.

If the disease appears in one or more fowls of a flock they should be immediately separated from the well ones. If possible, the source of the infection should be determined and removed.

The quite common practice of allowing fowls from different flocks to run together during the day should be discouraged.

Care should be taken to avoid the possibility of bringing the virus from affected flocks in the dirt or excrement which naturally adheres to the shoes in walking through an infected chicken yard. The same care is necessary in the interchange of working implements, such as shovels, hoes and the like.

---

*Gerhardt reports 4 cases of diphtheria in Wesselhausen, Baden, among 6 workmen who had charge of several thousand fowls, many of which died of diphtheria. There were no other cases of diphtheria in the neighborhood and the evidence was quite conclusive that the disease was contracted from the affected fowls.

Debrie (Centralblatt f. Bakteriologie, Bd. XIII (1893), S. 730) reports briefly the transmission of human diphtheria to fowls. He is inclined to the view that human diphtheria is transmissible to fowls and fowl diphtheria to man. Cole (Archives of Pediatrics, XI (1894), p. 381) reports a case of supposed transmission of the disease from a fowl to a child.

The diphtheritic disease of fowls reported by Loir and Ducloux in Tunis, in 1894, spread to the people of that place, resulting in an epidemic of serious proportions. Ménard refers to the fact that men employed to feed young squabs contracted diphtheria by blowing the masticated food into the mouth and crop of squabs suffering with that disease. Schrevens reports several cases of diphtheria in children in which he traces the source of infection to certain poultry.

The non-identity of these diseases has been clearly pointed out by Ménard.

Guérin has pointed out with emphasis that there is no relation between diphtheria in man and in fowls.

·

FIG. 117. SECTIONS OF THE HEADS OF A NORMAL (*1, 2 and 3*) AND OF A DIPHTHERITIC (*4, 5 and 6*) FOWL.

Fig. 117.   Photographs of transections of the heads of two fowls.

1, 2, and 3.   Sections from a normal head.

4, 5 and 6.   Sections from approximately corresponding levels from the heads of fowls suffering with diphtheria.

1.   Cross-section of a chicken's head just posterior to the nasal openings.   *a* Nasal passage, *b* turbinated bone, *c* portion of the wall of the false nostril, *d* sub-orbital sinus, *e* palate.

2.   Cross-section of a chicken's head midway between the nasal openings and the eyes.   *d* Sub-orbital sinus, d^1 superior portion of the sub-orbital sinus, which connects with *d* posterior to the lachrymal duct, *f* lachrymal duct opening into the mouth through the cleft palate.

3.   Cross-section of a chicken's head on a level with the anterior part of the eyes. *d* Sub-orbital sinus and the duct connecting it with the nares.

4.   Cross-section of a chicken's head just posterior to the nasal openings, showing the swollen condition of the nasal mucosa in the first stage of the disease.   The nasal passages are nearly occluded.   *b* Turbinated bone with swollen mucosa, d^1 sub-orbital sinus containing a small amount of exudate.

5.   Cross-section of a chicken's head midway between the nasal openings and the eyes, showing extensive exudate in the left sub-orbital sinus *d* and nasal passage extending into the cleft palate *m*.   The exudate is crowding upon the turbinated bones and nasal septum.

6.   Cross-section of a chicken's head through the eyes, showing exudate in the conjunctival sac, inflammatory thickening of the eyelids and membrana nictitans, and ulcerated cornea.   *g* Eyelid, *h* membrana nictitans, *i* exudate in the conjunctival sac, *k* ulcerated cornea, *l* eye.

All sections are magnified two diameters.

Preventive inoculation has been reported by several but wherever it was successful there is evidence of the epithelial nodules being present.   (See chicken pox.)

The most certain of the known methods of prevention as well as of treatment is the local application of disinfectants.   The dipping of the heads of fowls in a solution of 1 to 2 per cent. of permanganate of potash, or a 3 per cent. solution of creolin, is reported to be very effective in cases where the lesions are external and in the early stages.*   The fact that the lesions are so much exposed renders the disease especially favorable for topical applications.   When the exudates are in the sinuses or in the nares there is less opportunity for treatment.

---

*Practical poultrymen in Petaluma, California, found that where this trouble first appeared if the badly diseased individuals were removed and the other fowls dipped (immersed) in a 3 per cent. creolin solution and the houses disinfected the trouble often disappeared.

REFERENCES

1.   GRATIA ET LIÉNAUX.   Contributions à l' étude bacteriologique de la diphtérie aviaire.   *Annales de Méd. Vét.*, Vol. XLVII (1898), p. 401.

2.   GUÉRIN.   Sur la non-identité de la diphtérie humaine et de la diphtérie aviaire. *Recueil de Méd. Vét.*, Vol. LXXX, (1903), p. 20.

3. HARRISON AND STREIT. Roup. *Am. Vet. Review,* Vol. XXVII (1903), p. 26.

4. HARRISON AND STREIT. Roup: An experimental study. *Bulletin 132. Ontario Agric. Coll. and Exp. Farm,* 1903.

5. HOLMES. An outbreak of diphtheria associated with a similar disease among fowls and a vesicular eruption on the udders of cows. *Jour. of Comp. Path. and Therap.,* Vol. XVII (1904), p. 1.

6. LOEFFLER. Untersuchungen über die Bedeutuug der Mikro-organismen für die Entstehung der Diphtherie beim Menschen, beider Taube und beim Kalbe. *Mittheil. a. d. Kaiserlichen Gesundheitsamte,* Bd. II (1884), S. 421.

7. LOIR ET DUCLOUX. Contributions à l' étude de la diphtérie aviaire en Tunisie. *Ann. de l' Inst. Pasteur,* Vol. VIII (1894), p. 599.

8. MACK. The etiology and morbid anatomy of diphtheria in chickens. *Am. Vet. Rev.,* Vol. XXVIII (1905), p. 919.

9. MOORE. A preliminary investigation of diphtheria in fowls. *Bulletin No. 8. U. S. Bureau of Animal Industry,* 1895.

10. RANSOM. Manson's eye worm of chickens. *Bulletin No. 60. U. S. Bureau of Animal Industry,* 1904.

11. RATZ. Versuche mit dem Virusfiltrate der Vogeldiphtherie und der Geflügel-pocke. *Monatsh. f. praktische Tierheilk.,* Bd. XXV (1914), S. 41.

12. SALMON. The diseases of poultry, 1899, p. 216.

13. WARD. Poultry diseases in California. *Proceedings of the Amer. Vet. Med. Asso.,* 1904, p. 164.

CONTAGIOUS EPITHELIOMA

Synonyms: Chicken pox; avian variola; fowl pox; epitheliosis; pigeon pox; sore head.

Characterization. This is a disease of fowls in which there appear wart-like nodules varying in size from a millet seed to a large pea on the nostrils, openings of the ears, the comb, wattles and eyelids and skin surrounding the eyes. They also appear on the buccal mucous membrane where they are covered with a whitish yellow exudate. Jowett described fowl pox as a highly contagious disease occurring frequently as an epizoötic affecting fowls, pigeons, turkeys, geese and other birds.

When the nodules are limited to the skin the disease is usually benign. When the mucous membrane of the mouth is affected it is more often fatal, death being caused by inanition or asphyxia resulting from the extent of the exudates. This form of the disease closely resembles roup or diphtheria in chickens.

Several writers consider chicken pox and diphtheria to be identical in etiology. Carnwath, Schmid, Uhlenhuth and Manteufel, Ratz, Hutyra and Marek are of this opinion. On the other hand Bordet and Fally believe that chicken pox has nothing in common with avian diphtheria. Jowett states that the identity of the two diseases is an erroneous idea. Haring and Kofoid conclude that there is good evi-

dence to believe that nasal roup and chicken pox are two distinct diseases. They further state that immunity to chicken pox does not confer immunity to roup. Mack concludes that further investigation is necessary to determine the identity or non-identity of these infections.

History. The etiology of chicken pox was found in 1902 by Marx and Stricker to be a filterable virus. Since that time a large number

FIG. 118. A PHOTOGRAPH OF THE HEAD OF A FOWL SHOWING EPITHELIOMA ON THE COMB AND IN FRONT OF THE EYE (*after Ward*).

of investigations have been made in connection with it some of which tend to show, as stated above, that it is identical with avian diphtheria and others that it is an independent disease.

Geographical distribution. This affection seems to be wide-spread existing in practically all countries where fowls are kept. It is stated (by those who believe chicken pox and roup to be identical) that the diphtheritic form is found more frequently in the north and that the

epithelial nodular form occurs more frequently in the south. The statement is quoted by Jowett that chicken pox is one of the most destructive diseases of chickens in Southern Africa.

Etiology. Chicken pox seems to be due to a filterable virus. Rivolte considered the cause to be a protozoan parasite belonging to the *Gregarinidia*.

Symptoms and morbid anatomy. The disease manifests itself by the formation of nodules composed of tissue of an epitheloid nature on the areas of the skin free or nearly so of feathers about the head. The nodules are formed often with a caseous center and usually remain discrete. At other times the contents may be of a flaky or coagulated material. When the lesions are in the mouth they resemble those of diphtheria. When the nodules appear on the eyelids, the eyes may become closed and the fowl die of starvation.

Fig. 119. HEAD OF FOWL SHOWING LESIONS ABOUT THE MOUTH (*Pickens*).

Prevention. Manteufel was the first to immunize fowls against contagious epithelioma by vaccination. He succeeded by injecting into the circulation or subcutaneous tissue a substance prepared from scrapings of epithelial or mucous membranes mixed with salt solution. He also claimed that therapeutic results followed such injections. Later Hadley and Beach, Giltner and Mack reported success in vaccinating fowls with an emulsion prepared from the nodules. Mack grinds this carefully in a mortar with sand in a small quantity of salt solution. It is then filtered through cotton, diluted with normal salt solution until it is moderately turbid and attenuated by heating in a water bath at 55° C. for one hour. Each fowl is then given 1 c.c. of the attenuated virus subcutaneously in an unfeathered area beneath the wing. He reports satisfactory results.

Fig. 120. SHOWING SMALL LESIONS SCATTERED OVER THE COMB AND WATTLES.

REFERENCES

1. Beach. Suggestions to poultrymen concerning chicken pox. *Univ. of Calif. College of Agr.*, Circular No. 145, 1915.

2. Bollinger. Ueber Epithelioma contagiosum beim Haushuhn und die sogenannten Pocken des Geflügels. *Archiv. für. Path. Anat.*, Bd. LVIII (1873), S. 349.

3. Bordet and Fally. Le microbe de la diphtérie des poules. *Annal. de l' Inst. Pasteur*, Vol. XXIV (1910), p. 563.

4. Bordet et Gengou. Le microbe de la coqueluche. *Ann. de l' Inst. Pasteur*, Vol. XX (1906), p. 731.

5. Burnet. L' Épithélioma contagieux des oiseaux. *Annal. de l' Inst. Pasteur*, Vol. XX (1906), p. 742.

6. Gofton. Epithelioma contagiosum. *System of Vét. Méd.*, Edited by Hoare, Vol. I (1913), p. 323.

7. Hadley and Beach. Controlling chicken-pox, sore head or contagious epithelioma by vaccination. *Am. Vet. Review*, Vol. XLIV (1913–14), p. 330.

8. HARING AND KOFOID. Observations concerning the pathology of roup and chicken-pox. *Proceedings of Am. Vet. Med. Asso.*, 1911, p. 413.

9. HARRISON AND STREIT. *Bull. 125, Ontario Agr. Col.*, 1902.

10. JOWETT. Epithelioma contagiosum. *Journ. of Comp. Path. and Therap.*, Vol. XXII (1909), p. 22.

11. JULIUSBERG. Über das Epithelioma contagiosum von Taube und Huhn. *Deutsche Med. Wochenschr.*, Bd. LXVII (1904).

12. MACK AND RECORDS. The control of contagious epithelioma in chickens by vaccination. *Bull. No. 82 Ag. Exp. Sta. Nevada*, 1915.

13. MANTEUFEL. Bieträge zur Kenntnis der Immunitätserscheinungen bei den sogenannten Geflügelpocken. *Arbeit. aus dem Kaiserl. Gesundh.*, Vol. XXXIII (1909–10), S. 305.

14. MARX AND STICKER. Untersuchungen über das Epithelioma contagiosum des Geflügels. *Deutsche Med. Woch.*, 1902.

15. SALMON. *Diseases of Poultry.* 1899, p. 192.

16. SEDGWICK. Chickens and their diseases in Hawaii. *Bulletin No. 1, Hawaii Agric. Expt. Station*, 1901.

17. UHLENHUTH AND MANTEUFEL. Neue Untersuchungen über die ätiologischen Beziehungen zwischen Geflügeldiphtherie (Diphtheria avium) und Geflügelpocken (Epithelioma contagiosum). *Arbeit. aus d. Kaiserl. Gesundh.*, Vol. XXXIII (1909–10), S. 288.

18. WARD. Poultry diseases in California. *Proceedings of the Am. Vet. Med Asso.*, 1904, p. 164.

FOWL PLAGUE

Synonyms: Fowl pest; *Vogelpest;* bird pest; *Hühner-pest;* exudative typhus of birds; Brunswick bird plague; infectious peritonitis of fowls.

Characterization. This is a very acute and rapidly fatal disease of fowls caused by an ultra-microscopic organism that passes through the Berkefeld and Chamberland filters. Pigeons, turkeys, pheasants, geese, ducks, pea fowls, guinea-fowls, parrots, blackbirds, and other wild species are said to be susceptible.

History. In 1880, Rivolta and Delprato differentiated a disease from chicken cholera which they designated "exudative typhus." Perroncito called it a typical fowl cholera. It was investigated and described by Lode and Gruber in 1901. It was found in the Tyrol where during the time from March until July it had attacked 2,300 hens with a mortality of from 80 to 95 per cent. Centanni described this disease and the nature of its virus. He states that it has been recognized as a distinct affection for more than ten years. It is supposed to have been introduced into Germany by a poultry show held in Brunswick in 1901.

Geographical distribution. Fowl plague has been known in Northern Italy since 1894. It has been reported from Germany, Belgium

and France. It has been thought that it came from Soudan and Egypt.

Etiology. This disease is due to an invisible virus. The blood or aqueous suspension of crushed lungs or liver will usually produce the disease when injected in very small doses. It was found by Maggiora that 4 cc. of a dilution of virulent blood in which the blood was present in the proportion of 1 to 125,000,000 destroyed a young hen.

The virus is present in the blood, nervous system, nasal secretions, intestine and exudates in the larger cavities. Landsteiner found serum slightly virulent when the blood was very virulent. The virus retained its virulence in blood kept in sealed glass tubes and in a dark place for three months. The filtrate was virulent for a week only. It was destroyed in thirty minutes at a temperature of 60° C.

The period of incubation is usually from 3 to 5 days. After inoculation death occurs within 36 to 48 hours.

Symptoms. According to Centanni, the chicken acts dumpish the first day and refuses food on the second. The feathers are ruffled, the comb discolored and on the third day it dies. More rapidly fatal cases are mentioned by Lode and Gruber. The temperature is at first high (110° to 112° F.) but it falls to subnormal before death.

Morbid anatomy. The lesions vary. In some cases death follows so rapidly that autopsies reveal no appreciable tissue changes. There may be a slight pericarditis and ecchymoses in the heart muscle. There is an exudative pleuritis and peritonitis in some cases. Punctiform hemorrhages are reported on the inner surface of the breast bone, serous membranes, in the fat about the gizzard and mucosæ of the respiratory passages. In Italy it is reported that a fibrinous exudate often occurs on the pleuræ and peritoneum. The lungs are congested and occasionally there are collapsed areas. The liver, spleen and kidneys are more or less changed.

Diagnosis. The diagnosis is to be made by the symptoms and lesions. According to Freese, "One is entitled to assume a bird has died of avian plague if one finds hemorrhages in the glandular stomach (proventriculus), swelling of the kidneys, marked injection of the bloodvessels in the serosa of the Graafian vesicles, not infrequently accompanied by hæmorrhages, and providing the disease assumes a plaguelike form." It is to be differentiated from fowl cholera which it

closely resembles and fowl typhoid. The diagnosis is made from the bacteriological examinations. As the virus of fowl plague does not affect rabbits, these animals are of value in differentiating it from fowl cholera. The rapid course of the disease cannot be relied upon as a differential character, for both fowl cholera and fowl typhoid often run very rapid courses and likewise have a high mortality.

REFERENCES

1. CENTANNI. Die Vogelpest; Beitrag zu dem durch Kerzen filtrirbaren Virus. *Cent. f. Bakt.*, Bd. XXXI (1902), S. 145.

2. DUBOIS. Une maladie infectieuse des poules à microbes invisibles. *Comp. Rend. Soc. de Biol.*, Vol. LIV (1902), p. 1162.

3. HERTEL. Ueber Geflügelcholera und Hühnerpest. *Arb. a. d. Kais. Ges.-Amte.*, Bd. XX (1904), p. 453.

4. JOEST. Beitrag zur Kenntnis der Bakterienflora des Hühnerdarmes nebst einigen Bemerkungen über eine neue Hühnerseuche. *Berl. Tierärztl. Wochenschr.*, 1902, No. 16.

5. KLEINE UND MOELLERS. Ueber Hühnerpest bei Gänsen. *Centralbl. f. Bakt.*, Bd. XXXIX (1905), S. 545.

6. KRAUS UND SCHIFFMANN. Studien über Immunisierung gegen das Virus der Hühnerpest. *Centralbl. f. Bakt.*, Bd. XLIII (1907), S. 825.

7. LECLAINCHE. La Peste Aviaire. *Revue Générale de Méd. Vétérinaire*, 1904, p. 49. Abstract of article in *Jour. Comp. Path. and Therap.*, Vol. XVII (1904), p. 83.

8. LIPSCHÜTZ. Ueber mikroskopisch sichtbare filtrierbare Virusarten. *Centralbl. f. Bakt.*, Bd. XLVIII, S. 77.

9. LODE UND GRUBER. Bakteriologische Studien über die Aetiologie einer epidemischen Erkrankung der Hühner in Tirol (1901). *Centrallb. für Bakt.*, Bd. XXX (1901), S. 593.

10. M'FADYEAN. The ultravisible viruses. *Journ. of Comp. Path. and Therap.*, Vol. XXI (1908), p. 168.

11. MAGGIORA UND VALENTI. Ueber eine Seuche von exudativem Typhus bei Hühnern. *Zeit. für. Hygiene*, Bd. XLII (1903), S. 185.

12. MAUE. Immunisierungsversuche bei Hühnerpest, *Arb. a. d. Kais. Ges.-Amte.*, Bd. XXI (1904), S. 537.

13. OSTERTAG UND BUGGE. Weitere Untersuchungen über die Hühnerpest. *Zeitschr. f. Infektionskrankh. d. Haustiere*, Bd. II (1906), S. 1.

14. OSTERTAG UND WOLFFHÜGEL. Untersuchungen über die "Hühnerpest," die neue Geflügelseuche. *Monatsh. f. prakt. Tierheilk.*, Bd. XIV (1903), S. 49.

15. RUSS. Beobachtungen über das Virus der Hühnerpest. *Arch. f. Hyg.*, Bd. LIX (1906), S. 286.

16. SCHIFFMANN. Zur Histologie der Hühnerpest. *Centralbl. f. Bakt.*, Bd. XLV (1908), S. 393.

17. ZSCHOKKE. Beobachtung über Hühnerpest. *Schweizer Arch. f. Tierheilk.*, Vol. LIV (1912), S. 282.

CORNSTALK DISEASE IN CATTLE

Characterization. The name "cornstalk disease" was given to a somewhat mysterious affection from which cattle sometimes suffered while feeding in cornstalk fields late in the fall and early winter. The meaning generally accepted and intended to be conveyed by this term

was, that an animal or a number of animals, usually cattle, have died suddenly after feeding in a cornstalk field from four to ten days. From a pathological point of view, therefore, the term is meaningless, but it has served admirably as a general term to designate certain fatalities occurring under a given condition.

History. From a historical point of view, no positive statements can be made concerning it prior to 1868, when the first recorded investigations into its nature and cause were made. At that time Gamgee was employed by the United States Department of Agriculture to investigate this disease. The "smut theory" of its etiology appears to have been the prevailing one at that time and consequently Gamgee's report deals almost exclusively with the effect of improperly prepared food, smuts and the like. He concluded that "smut is not a very active poison in combination with wholesome food."

In 1889, Billings described the cornstalk disease as an "acute extraorganismal septicemia, due to micro-organisms belonging to the class of ovoid-belted germs, to which variety of disease also belongs the swine plague, southern cattle plague, *Wildeseuche*, hog cholera, and yellow fever in man." From the organs of cattle dead from the disease he reported to have invariably isolated a bacillus, which he affirms to be its cause. He identified the bacillus, which he found in the animal tissues, with the one described by Burrill as the cause of a disease in cornstalks. In 1893, Smith identified the bacillus described by Burrill as *Bacillus cloacæ*.

Billings also found pneumonia to be one of the lesions characteristic of this affection and in a subsequent bulletin he places great importance upon this lesion, although he adds very few additional observations to sustain the claim.

In 1890, a few animals from a shipload of American cattle landed at La Villette, France, died of pneumonia. They were examined very carefully by Nocard and other French veterinarians. From the diseased lung Nocard obtained a micro-organism which corresponded very closely to the description of the bacillus of the cornstalk disease of cattle described by Billings in America. The publication of this fact gave rise to a temporary supposition that this American cornstalk disease might be a menace to the cattle of Europe and consequently initial steps were taken to require American cattle to be quarantined against it. The fact was subsequently determined that the bacillus isolated by Nocard belonged to the septicemia hemorrhagica group of

bacteria which is usually found in a form of interstitial pneumonia and the matter was dropped.

A single experiment was made at Champaign, Ill., in 1889, in which the etiological importance of corn smut was tested with negative results. A bacteriological examination of the organs from an animal that died in a cornstalk field, supposedly of this disease, was made with negative results by Burrill in 1889.

In 1892, Moore investigated this disease but was unable to find any definite cause for it. The lesions in the animals that he examined immediately after death correspond very closely to those of septicemia hemorrhagica. The frequent appearance of this disease tends to the conclusion that while cattle pasturing in corn fields late in the fall may now and then die suddenly from various causes, perhaps from eating too many cornstalks, the disease that produces the more serious losses heretofore attributed to cornstalk disease may be septicemia hemorrhagica.

REFERENCES

1. BILLINGS. The cornstalk disease in cattle. *Bulletins No. 7, 8, 9 and 10. Neb. Agric. Exper. Station,* 1886–88.

2. BILLINGS. The corn fodder disease in cattle and other farm animals, with especial relation to contagious pleuro-pneumonia in American beeves in England. *Bulletins No. 22 and 23. Univ. of Neb. Agric. Exper. Station,* 1892.

3. DE SCHWEINITZ. Chemical examination of cornstalks presumably the cause of cornstalk disease in cattle. *Bulletin No. 10, U. S. Bureau of Animal Industry,* 1896.

4. GAMGEE. Diseases of cattle in the United States. *U. S. Department of Agriculture,* 1869.

5. MAYO. Cattle poisoning by nitrate of potash. *Bulletin No. 49, Kansas Agric. Exper. Station,* 1895.

6. MAYO. Cornstalk diseases in cattle. *Ibid, 1896.*

7. MOORE. An investigation into the nature, cause and means of preventing the cornstalk disease (Toxemia maidis) of cattle. *Bulletin No. 10, U. S. Bureau of Animal Industry,* 1896.

8. MOORE. An inquiry into the alleged relation existing between the Burrill disease of corn and the so-called cornstalk disease of cattle. *Proceedings of Society for the Promotion of Agric. Science,* Vol. VIII (1894), p. 368.

CHAPTER XIV

IMMUNITY AND PROTECTIVE INOCULATION

General statement. The application of the principles of immunity is an important factor in the control of infectious diseases of animals. The prevention of these maladies depends upon two procedures, namely, first in keeping the virus of the diseases away from the animals and secondly in immunizing animals against them. Successful control requires both, for it is not possible in all cases to prevent exposure and again a complete immunity cannot be artificially induced in all individuals. The practitioner, therefore, should be familiar with the principles of immunity and should be ready to apply them at any and all times when the checking of an epizoötic disease depends upon accelerating the resisting power of the exposed individuals. For the reasons above stated a short discussion of immunity follows.

Immunity. In a broad sense, immunity is "resistance to disease." The term, however, is usually restricted to the infectious maladies and signifies a condition of the individual which enables it to successfully defend itself against the invasion of its tissues with infecting microorganisms or to resist the toxic effect of the invading organisms should they gain entrance and multiply within the body. It applies to the action of all pathogenic bacteria and protozoa. It will be seen that immunity is only relative; it is neither permanent nor constant but varies with natural and artificial conditions. According to the process by which it is established in the individual, immunity is recognized as natural or acquired.

Natural immunity. The term natural immunity is applied to that condition which enables animals to resist the natural invasion of infecting microörganisms that attack other varieties or species of animals. It is a condition inherent in the nature of the individual, born with it and transmitted to its offspring.

There are a number of very striking examples of natural immunity. The Algerian race of sheep are immune to natural infection of anthrax, whereas other sheep are very susceptible to it. The equine species is

susceptible to glanders but the ruminants are immune.   Black leg, which is very destructive to cattle, does not attack horses, the carnivora or man.   There are, however, instances where a species possesses a general immunity against a disease, but where individuals are occasionally attacked.   Thus, it is not usual to find tuberculosis in the carnivora, but now and then a cat or a dog is found affected wih it.   It sometimes happens that individuals belonging to a susceptible species resist infection.   ·

It has been determined that in cases of marked natural immunity the resistance can be overcome and the animal infected by changing its normal physical condition.   Thus fowls that are naturally immune to anthrax are said to be made susceptible to it by reducing their temperature by immersing them in cold water.   Charrin and Roger found that fatigue would lessen the resistance of white rats to the same disease.   Gibier found that frogs kept at a temperature of 37° C. were susceptible to anthrax.

It has been stated that sewer and other poisonous gases predisposed animals to infection.   Abbott concluded, after a careful experiment in exposing rabbits to sewer gas and gases of putrid meat, that their resistance to infection was not lessened.   Natural immunity usually persists under ordinary conditions throughout life.   It is much more permanent than acquired immunity.

Explanation of natural immunity.   There are a number of explanations of this phenomenon.   In brief they deal with the supposed actions (1) of the cells of the body, (2) of the serums or humors and (3) inability of infecting agents to grow in the animal body.

Those who seek the cause for this condition in the cells find that phagocytosis, so ably described and demonstrated by Metchnikoff, is the source of the individual defence.   Those who find the cause in the humors of the body rely upon the germicidal action of the serum itself or of the substances set free from the cells that are present in the liquids.   Metchnikoff believed that the microcytase elaborated from the leucocytes acts as a solvent directly upon the bacteria.   The split protein theory explains the non-development of invading organisms on the ground that they cannot utilize the tissues for food and consequently they perish.

Acquired immunity.   As the term implies, this is immunity established in the individual after birth.   It is most common in individuals that have survived an attack of an infectious disease.   The

most striking examples of this are cases of recovery from small pox and yellow fever in man and Texas fever in cattle. The exanthematous diseases leave the individual with more or less immunity. In certain other infectious diseases there is little increased power of resistance imparted to the individual that has recovered from the first attack. In such diseases as diphtheria, the duration of the immunity resulting from a natural attack is variable.

The period of duration is variable in artificial immunity.

The fact that individuals that had recovered from certain diseases were rendered immune to a second attack led Pasteur and others to inquire into methods for artificially immunizing animals against the infections most destructive to them. Pasteur found that inoculating animals with attenuated virus* would immunize them against the strong virus or naturally acquired infection. He succeeded with swine erysipelas, chicken cholera, anthrax and later with rabies. Arloing, Cornevin and Thomas introduced a successful method of preventive inoculation with attenuated virus against black leg.

The next procedure was a line of investigations directed toward the production of immunity by the use of heated cultures of the bacteria (bacterins) and the toxins in filtered cultures. The first of these was an immunization of pigeons against *B. suipestifer* with the use of heated bouillon cultures by Salmon and Smith in 1886. This line of investigation led eventually to the immunizing of animals experimentally with the toxins of certain virulent pathogenic bacteria such as those of diphtheria and tetanus.

Another method that has been extensively tried experimentally with the bacterial diseases, but usually without success, is the use of non-lethal doses of virulent virus. It was somewhat successful in contagious pleuro-pneumonia of cattle. With certain of the protozoan diseases, such as Texas fever, this method is more satisfactory.

It has been found that the blood serum of animals that are immune to certain bacterial diseases possesses antitoxic properties by which it is able to impart immunity to healthy susceptible animals, or to act as a therapeutic agent for those already affected with the same disease. Diphtheria antitoxin is a striking illustration of this.

---

*This principle was exemplified centuries before in the far East where inoculation with small pox virus (material from the pustules) was practiced whenever small pox occurred naturally in a very mild form. Lady Mary Wortly Montague is said to have introduced this practice into Europe about 1718. Later, 1796, Jenner, after thirty years of labor, introduced the practice of inoculating human subjects with the virus of cow pox. This is known to-day as vaccination and the vaccine is prepared at the present time from calves. In 1839, Thiele showed that the disease known as cow pox was small pox in cattle.

Methods for producing acquired immunity.   Immunity may be produced in several ways other than by causing the individual to pass through an attack of the disease caused by natural infection. These methods may be summarized as follows:

By inoculating the individual with a non-lethal dose of a strong virus.   This is practiced in immunizing cattle against Texas fever, sheep pox and contagious pleuro-pneumonia.

By inoculating the individual with an attenuated virus.   This is practical in anthrax, blackleg, chicken cholera, *rouget*, rabies and bubonic plague in man.

By inoculating the individual with a vaccine consisting of the virus of the disease modified by passage through another species of animal, as vaccine for small pox.

By the injection of toxins.   In practice this is used for immunizing animals, especially horses, against the virus of the diseases for the purpose of procuring antitoxin from their blood, as in the preparation of diphtheria and tetanus antitoxins.

By the injection of antitoxins.   These are used to immunize animals against toxins, and children against natural infection, as in diphtheria and tetanus.

By the injection of defibrinated blood or serum from immune or hyperimmune animals for prophylactic purposes.   The use of anti-hog-cholera serum to immunize swine against hog cholera is an example of this use of serum.

Active and passive immunity.   Acquired immunity may be active or passive.   If the organism of the disease participates in the process of bringing about increased cell activity the resulting immunity is active.   Such immunity is acquired at the expense and often at the risk of the individual acquiring it.

Passive immunity is produced by the injection of the serum or antitoxin of animals already immune.

It involves no active generation of protective substances on the part of the animal.   The passively immunized animal is simply the recipient of substances formed in the body of other animals and transferred to it.

Active immunity is slow in its development, is more or less dangerous to produce and it always attended with at least some discomfort. It varies in the time it lasts but usually it is quite persistent, continuing from a few weeks or months to several years.   Passive immunity is quite rapidly produced, is attended with little or no danger and

practically no discomfort. It is, however, limited in its period of duration. The most extensive use of passive immunity is in immunizing swine against hog cholera by the serum method and horses and people against tetanus by means of tetanus antitoxin.

Explanation of acquired immunity. A number of ingenious explanations have been offered for acquired immunity. The only ones of the older theories that have withstood the test of their objectors are those of Metchnikoff, representing the cellular theory, and of Ehrlich, representing the humoral theory. Vaughan has advocated the proteolytic enzyme production as an explanation for bacterial immunity. The results of various investigations indicate that the factors involved in securing immunity are multiple in number and varied in character. As pointed out by Meltzer it is difficult to explain immunity as being due to one or even to a few anti-bacterial properties of the animal body. In the struggle against bacteria the defence of the body is carried on by the united action of each and every resisting influence. However, a number of theories have been proposed to explain the phenomenon.

The exhaustion theory. This was suggested in 1880 by Pasteur, who thought that the microörganisms growing in the body used up some substance essential to their further existence and died leaving the body unsuited for future occupation. It applied to active immunity only.

The retention theory. This was proposed by Wernich and Chauveau. It is based on the fact that bacteria elaborate some metabolic product that inhibits their further development and the future invasion in the tissues by the same species. This theory is illustrated by the facts shown in the cultivation of bacteria in artificial media. The bacteria often die apparently from the accumulation of metabolic products long before the nutriment is exhausted.

The phagocytosis theory. Metchnikoff has supposed that acquired immunity is brought about because of the action of the phagocytes upon the invading organisms. He has shown that in cases of infection with the Vibrio Metchnikovi, the phagocytes of unprotected animals do not take up the bacteria, but vaccinated animals do. It appears from the work that has been done on this subject that the phagocytes are active in proportion to the degree of immunity possessed by the individual.

The humoral theory. This is based on the observations of Buchner, Nuttall and others that blood serum has the power of destroying a certain number of bacteria when they are placed in it. Nuttall showed in addition to this that the bacteriolytic power ceased if the blood was heated to 55° C. It is found, however, that the bacteriolytic serums occur only in cases where there is a high degree of forced immunity, their activity being in proportion to the degree of immunity obtained. An explanation for the action of the serums upon bacteria is given by Ehrlich in his lateral side-chain theory.

Ehrlich's side-chain theory. According to Ehrlich, in every living cell there must exist an active central body and a number of other chemical groups or side chains.

These groups have the greater variety of function, especially those of nutrition and assimilation. This theory teaches that immunity depends upon the presence or absence of certain substances which he calls receptors or lateral chains which certain of the cells possess. These receptors are concerned in the normal nutrition of the cells and have affinities for various complex albuminous substances. Among these substances are the molecules of the toxin produced by certain bacteria and possibly other poisons. Every toxin has affinities described as haptophorus and toxophorus, that is, each molecule of the toxin is composed of two different groups of atoms, the one the toxophore or poisonous group, the other the haptophore or combining group of atoms. The haptophorus atoms of the toxin molecule combine or unite with the receptors of those cells for which they have special affinity and through the haptophore group the toxophore part of the molecule is able to act upon the cell. In some cases the cells are destroyed and in others additional receptors are produced because of the stimulation. These receptors may pass out of the cell into the serum, where they act as free receptors or immune bodies to lock up or neutralize the toxin. The free receptors are the active part of the antitoxin.

Ehrlich illustrated his theory by the use of diagrammatic figures to represent the cell throwing off "receptors," the receptors or antitoxin locking up or neutralizing the toxin and the effect of the anti-bodies on the toxin and its complement.

The protein sensitization or proteolytic ferment theory has been advocated by Vaughn an explanation of immunity. In brief, the theory as stated by Vaughn is this: "Protein sensitization consists in developing in the animal body a specific proteolytic ferment which digests the same protein on reinjection." Like the other theories, this one must be proven by the crucial test of experimental work.

Still more recently another theory to explain immunity has been advanced by Williams and Beveridge and called by them the "Proteomorphic theory."

Hemolysins. Belfanti and Carbone, in 1898, pointed out the fact that if horses were injected with the red blood cells of rabbits the serum of these horses would be more or less toxic for rabbits. This result was followed by some interesting experiments by Bordet in which he showed that the blood serum of guinea pigs which had been injected several times with from three to five cubic centimeters of defibrinated rabbit's blood acquires the property of rapidly dissolving in a test tube the red blood cells of a normal rabbit's blood. The serum of an untreated healthy guinea pig will not do this. It was pointed out that this reaction was specific, that is, the serum of animals treated with rabbit's blood (specific serum) dissolves the red corpuscles of the rabbit's blood only. This property that had been demonstrated in the blood serum of guinea pigs treated with rabbit's blood was shown to hold for the sera of other species of animals treated with the blood cells of a different species. Wassermann has formulated this action as follows: "The serum of animals species **A,** after these have been injected either subcutaneously, intraperitoneally, or intravenously with erythrocytes of species **B,** acquires an increased

solvent action for erythrocytes of species **B,** and only for this species. (There are a few exceptions to this general rule). We call this hemolysis, and the substances which affect the solution of the red cells, hemolysins or hemotoxins."

Bordet has been able to show that the solvent power of the specific hemolysins depended on the combined action of two constituents of the specific serum. Thus, when the fresh hemolytic serum was heated for a half hour at 55° C. it lost its power. If to this inactive serum a very small amount of the serum of a normal guinea pig was added, the full hemolytic power was restored to this inactive serum. In other words, it had been reactivated by this addition. This experiment showed that the hemolytic action of the specific hemolytic serum depends upon two substances: (1) the one destroyed by heating to 55° C. and which is contained in the serum of the normal untreated animal as well as in the specific hemolytic serum, and (2) a substance that is able to withstand heating to 55° C. and which is contained in the specific serum only.

The substance which is destroyed at 55° C. and which exists in the blood of the untreated animal and in the specific serum constitutes the alexins of Buchner. The substance which is not destroyed by heating to 55° C. and which is brought about by the action of the blood of one species upon another is known as the immune body, or as termed by Buchner *substance sensibilatrice.*

Agglutinating power of hemolytic serum. It was found by Bordet that another property was increased in the hemolytic serum, namely, the power of clumping red blood corpuscles. This clumping or so-called agglutination of the red cells occurs previous to their solution. This action Bordet considers as a specific one. Wassermann has formulated this reaction thus: "If an animal, species A, be treated with blood of species B, the serum derived from A will have acquired an agglutinating power which differs from that of normal serum of A in one very important particular, namely, in that it is specifically increased with respect to the red cells of species B or its nearest biological relative. This clumping must not be confounded with rouleaux formation in normal blood."

For a more extended discussion of the subject of hemolysis and for cytolysis, cytotoxins agglutinins and precipitins the student is referred to the special literature on these subjects.

Protective inoculation. The practical application of immunity in protective inoculations or vaccination has come to mean the establishment of partial or complete immunity in the individual against the disease in question. The vaccination against small pox introduced by Jenner in 1796 is a well known example of protective inoculation. Following the work of Pasteur there developed many procedures for establishing both active and passive immunity against a number

of diseases of animals. There is some method known, proposed or being investigated for practically every infectious disease. The methods that are now in use, together with the diseases against which they are employed with sufficient success to warrant their recommendation, may be summarized as follows:

Active immunity. This method is employed most extensively in immunizing cattle against Texas fever and bovine contagious pleuropneumonia. It is used in France to immunize against sheep pox (clavelization).

Since the 18th century there has been practiced in France the artificial immunization of sheep by the inoculation of the virus of sheep pox just as the variolization of man was practiced before the discovery of small pox vaccine. In France, the law requires the inoculation (clavelization) of flocks in which sheep pox appears, but it interdicts the practice in unattacked flocks.

The injection of animals with attenuated virus of the disease against which immunity is to be established. This method is used most extensively in anthrax, rabies, symptomatic anthrax and swine erysipelas.

The practical value of vaccination for rabies (Pasteur treatment) over that of most other diseases, is the fact that it is effective if made early in the period of incubation. This method, which takes advantage of the long period of incubation in rabies, constitutes a means of handling an infectious disease intermediate between protective inoculation and a therapeutic treatment.

A large amount of work has been done by many investigators to secure practical means for immunizing cattle against tuberculosis and horses against glanders but thus far satisfactory methods have not been formulated.

Passive immunity. It is employed as a prophylactic in swine erysipelas, tetanus, diphtheria, hog cholera (serum alone method) and also in certain diseases such as rabies and anthrax for which there are also methods for active immunization.

The use of the tetanus antitoxin to immunize horses against tetanus before subjecting them to operations, such as castration, or after receiving punctures of the skin or hoof ("farrier's puncture") is becoming more and more prevalent in those countries and localities where tetanus is common. In France it seems to be used more than elsewhere. The serum is given in two injections from 10 to 12 days apart. Large animals receive 20 c.c. but small ones from 6 to 10 c.c. at each injection.

The anti-hog-cholera serum (while it is called serum it usually consists of the defibrinated blood of the immunized animal) is very extensively employed to protect swine against hog cholera.   If properly used it is very efficient.   The immunity established by the use of the serum alone lasts but a few weeks.

The value of diphtheria antitoxin as an immunizing agent against diphtheria is well known.

The simultaneous method.   This consists in using a strong virus together with an immunizing serum.   It is employed quite extensively against rinderpest, anthrax, and hog cholera.

In case of rinderpest the animals are injected with a protective serum simultaneously with the virulent blood.   The immune serum is obtained from animals that have recovered spontaneously from rinderpest or from cattle that have been immunized to it.   The serum alone of animals that have recovered spontaneously possesses very slight protective properties unless very large doses are given.   Kolle and Turner showed that if animals just recovering from an attack were injected with large quantities of the blood coming from animals suffering with a fatal attack, the protective power of their serum was markedly increased.   This serum may be kept for a long time by adding a small quantity of carbolic acid.

In rabies the method is reported to be most successful.   Its essential advantage over the other process is that it can be used with good results much later in the period of incubation.   It has the additional practical feature that the number of injections is minimized.

In anthrax it is reported to be giving much better results than the double inoculation with a weak and stronger vaccine as followed in the Pasteur method.

The simultaneous method is used extensively in the protection of swine against hog cholera.

The use of bacterins.   Heated (killed) cultures of bacteria have been used both for protection and therapeutic purposes.   They have given as a rule better results when employed as remedies after symptoms have appeared than as purely prophylactic measures.

Difficulties and dangers to be considered in vaccination.   The results of the efforts that have been put forth to obtain control over the infectious diseases of animals show that with the victories there have been some failures.   It is not always possible to know the exact degree of virulence possessed by the attenuated virus or vaccine, and again the resisting forces of animals vary even in different individuals

of the same species. If the virulence is too great or the resistance below the supposed normal the vaccine may produce disease in excess of the amount required to establish immunity and perhaps it may kill the animals it was intended to protect. This has happened on several occasions. On the other side, if the attenuation of the virus is too much, or the natural resistance unusually high, there is not disease (reaction) enough produced to establish immunity. In such cases the results are negative. Where the virulent virus is used in small doses, accidents have happened by way of producing a fatal disease instead of a mild attack that was anticipated. The difficulty rests in the procuring of a vaccine or the quantity of a virus that possesses just the amount of disease-producing power that is necessary to bring about immunity and no more. This is a balance of vital forces that it is exceedingly difficult to strike.

A glance at the diseases for which active immunity has been attained will show that they are acute toxic affections and not those in which the disease consists of extensive tissue destruction. Toxic immunity has been attained in certain diseases but a bacterial immunity is much more difficult to acquire.

The dangers in vaccination, as applied especially to animals at large, may be summarized as follows:

The vaccine may be too much attenuated, resulting in the failure to establish immunity.

The vaccine may be too strong (virulent) so that it will produce more disease than is desired, possibly causing fatal results.

The attenuated virus of which the vaccine consists may regain its virulence. The distribution of living pathogenic microörganisms among animals is of itself not to be recommended. They may be the cause of subsequent outbreaks.

In using virulent virus in non-lethal doses, the danger of producing fatal results because of the susceptibility of the individual treated is always present.

In the simultaneous method the danger of accident resulting from too strong a virus, too weak a serum, or the high resistance or unusual susceptibility of the individual are possible conditions to be kept in mind.

Prevention. The prevention of the specific diseases of animals, when considered from the point of view of etiology, is not so difficult as is often supposed. Infectious diseases are simply parasitisms. If the infecting organisms can be kept away from animals the diseases

they would produce cannot appear, and if the individuals already suffering from the disease are properly isolated, their stables and pens disinfected, and their bodies properly destroyed if they die, the infecting organisms must perish. Pasteur said, "It is within the power of man to make all infectious diseases disappear from the world." The fact that the microörganisms of these diseases are parasites restricts their breeding places, so to speak, to the animal body. If, therefore, the channels through which these microörganisms escape from the infected body are properly guarded, and if the channels through which they enter the healthy body are intelligently protected, the spread of infectious animal diseases will be minimized, if not entirely prevented.

Since the discovery of a specific etiology the life history and possibilities of the virus of the different infectious diseases have been diligently studied and much has been learned concerning them. The result is that frequently by taking advantage of the present knowledge of the infecting organism, preventive measures may be taken that will give equally as good results as vaccine, without its dangers, and often with as little trouble and expense. The prevention of these diseases is well illustrated in many instances where owners of animals have protected their herds against the sources of infection when surrounded with widespread epizoötics. Whole countries have been kept free from certain maladies by enjoining the same methods, as shown in the absence of rabies in Australia. This was accomplished by prohibiting the entrance of dogs until after they had been quarantined for a sufficient time. The eradication of infectious diseases by holding rigidly and simply to the guarding of the channels of dissemination and infection has been accomplished a number of times on a large scale as instanced by the eradication of contagious pleuro-pneumonia in cattle from this country and from Great Britain. The elimination of foot-and-mouth disease in 1902 and again in 1908 are striking illustrations of efforts in this direction. The eradication of infectious diseases from individual herds is being accomplished repeatedly by adhering to the same methods. The Bang method of handling bovine tuberculosis, as already mentioned, enables one to protect the well animals and to save all there is of value in the others.

The isolation of the well from the sick and the thorough disinfection of the houses and yards containing the sick has resulted in stopping many outbreaks of disease. This is followed very generally even where vaccination and serum prevention treatment are resorted to. Several times in the writer's experience success in checking the spread

of the disease by vaccination was not attained until rigid methods of isolation and disinfection were carried out. Just what these methods are to be in each particular case will be indicated by a definite knowledge of the cause and the nature of the disease in question.

The methods for immunization that give the greatest promise in aiding in the solution of the problem of the control of infectious diseases are those productive of a passive immunity. Although these are temporary in duration they are effective immediately and consequently tend to save individuals when they are applied in the period of incubation or before a possible exposure. They protect the animal until the virus can be eliminated and its environment made safe.

The advantages of introducing preventive measures are:

The tendency to eliminate the virus of the disease if it is present, and to keep it away if it has not already appeared.

The animals are free from the virus and there is no danger of their subsequently developing lesions due to the localization of the attenuated microörganisms.

The separating of the well animals from the sick ones and from the infected stables, pens, yards, or fields is not usually an expensive procedure. The stables and yards can be disinfected and the infected fields can be utilized for other purposes until the virus has been destroyed by its own limitations of endurance. In this connection, it is well to call attention to the successful efforts that are being put forth to eliminate the cattle tick from the Southern States and thus prevent Texas fever.

REFERENCES

1. CITRON. Immunity. Philadelphia, 1912.
2. EHRLICH. Studies on immunity. New York, 1906.
3. ERNST. Modern theories of bacterial immunity. Boston, 1903.
4. JOWETT. Notes on blood-serum therapy. London, 1907.
5. KOLMER. Infection, Immunity and Specific Therapy. 1915.
6. KRAUS UND LEVADITI. Handbuch der Technik und Methodik der Immunitätsforschung. 1909.
7. METCHNIKOFF. Immunity in infective diseases. Cambridge, 1905.
8. MUCH. Die Immunitätswissenschaft. Würzburg, 1911.
9. MÜLLER. Vorlesungen über Infektion und Immunität. Jena, 1910.
10. NUTTALL. Blood immunity and blood relationship. Cambridge, 1904.
11. SOBERNHEIM. Deutsche Med. Wochenschrift, 1904, S. 27.
12. STERNBERG. Immunity, protective inoculations in infectious diseases and serum-therapy. New York, 1895.
13. STERNBERG. Infection and immunity. New York, 1903.
14. VAUGHAN. Protein split products in relation to immunity and disease. 1913.

15. VAUGHN AND NOVY.   Cellular toxins or the chemical factors in the causation of disease.

16. WASSERMANN.   Immune sera, hæmolysins, cytotoxins, and precipitins.   New York, 1904.

17. WILLIAMS AND BEVERIDGE.   The mechanism of immunization.   *American Medicine*, N. S. Vol. IX (1914), p. 621.

18. ZINSSER.   Infection and resistance.   New York, 1914.

CHAPTER XV

DISINFECTION

Disinfection. By disinfection is meant the destruction of disease-producing organisms. For this purpose, nature has provided very important agents, such as sunlight and drying, but these are not available or sufficient to destroy all infecting bacteria in all infected places within the necessary time limits. To supplement these natural forces, a large number of chemical substances possessed of germicidal powers have been brought into service. If, however, the results of the test experiments with these different substances are reviewed, one is impressed with the discrepancies, if not contradictory conclusions, recorded concerning their value. In view of these facts the practitioner is often at a loss to know just what chemicals to use, or how to apply them under different conditions and for the destruction of different species of organisms. The failure resulting from the many efforts to disinfect stables, pens, kennels and yards has caused much skepticism concerning the efficiency of many reported disinfectants. In order to rightly understand the reason for the differences in results of the test experiments or the lack of uniformity in the application of the various disinfecting substances, it is well to take into account certain fundamental facts relative to the species themselves, the material with which they are mixed and the chemicals used.

Different species. The bacteria used by different investigators to test the efficiency of certain substances have not been the same. The vital resistance of the various species is very different. The results obtained in testing disinfectants on the spirillum of Asiatic cholera or the bacterium of bubonic plague give but little information relative to the value of the same disinfectants when used for the destruction of the bacteria of glanders, tuberculosis or anthrax. The difficulty in accepting the results of many of the older experiments is that organisms were used which are very unlike those for which these disinfectants are now wanted. The practical value of the more recent experiments is greater because they have dealt with species of bacteria with which most of the work of disinfection has to do.

516

Variability of resistance in the same species. The power of resistance of the same species of bacterium varies greatly under different conditions. For instance, Bear found that a freshly inoculated culture of the bacterium of diphtheria was destroyed with 1-5000 of nitrate of silver, but that a twenty-four hour culture required 1-1000 of the same agent to kill it in the same space of time. In some work done by Esmarch he made use of anthrax spores from seventeen different sources. They were destroyed by steam at $212°$ F. in from one to twelve minutes and by a five per cent. solution of carbolic acid in from two to forty-two days.

The material in which the bacteria exist. The medium in which the bacteria exist influences the results of the disinfectants. The bacterium of tuberculosis from an aqueous suspension dried upon threads may be promptly destroyed by mercuric chloride, but in fresh, purulent, tuberculous discharges it cannot be trusted to destroy them. Again Behring states that sporeless anthrax bacteria in water are killed by corrosive sublimate 1–500,000; in bouillon, by 1–40,000; but in blood serum not with certainty with a solution of 1–2000. Some disinfectants are influenced very much by the character of the material which contains the infectious organism, while other disinfectants are influenced to a comparatively slight degree. The experimental work which does not take into account the influence of the media upon the disinfectant is not of much practical value.

Temperature. The temperature under which the disinfecting agent acts influences very much the rapidity and the certainty of its action. Thus Heider found that anthrax spores which survived the action of a five per cent. solution of carbolic acid thirty-six days at ordinary room temperature were killed in from one to two hours at $131°$ F. Some investigators have failed to state the temperature under which their disinfecting experiments were made.

Interpretation. In many of the results the inhibitory action of the agent in question has been mistaken for its germicidal action. After the bacteria have been subjected to the influence of a disinfectant for a given time, though not killed, their vegetating and pathogenic capabilities may be modified but still able, under favorable conditions, to return to their former vigor.

The rules and recommendations of the various cattle commissions and those having authority in methods for the prevention of infectious diseases of animals do not very clearly define the procedures best

adapted to the various places and conditions requiring disinfection. The many chemicals possessed of germicidal powers and the numerous commercial disinfectants, recommended largely from the results of certain definite tests, which in point of fact may be of no value in determining their efficiency for the conditions in question, render further inquiry into the best methods for disinfecting after animal diseases a matter of much scientific interest and great practical value.

Conditions to be taken into account in practical disinfection. In the effort to destroy the microörganisms in such places as yards, stables, cattle cars and the like, it is necessary to consider before applying a disinfectant the following conditions:

a. The resistance of the particular organism to be destroyed.

b. The medium or material in which it exists.

c. The nature of the place containing the organism to be destroyed.

d. The chemical action of the material surrounding the microorganisms on the disinfectant itself.

If the disinfection is for anthrax a more powerful disinfectant must be employed than would be required in disinfecting for the bacteria of septicemia hemorrhagica. If the infecting organisms are mixed with fecal matter, dirt or fodder, the problem is a different one than where they rest on a comparatively clean surface. It matters again whether the infecting organisms are in the soil (on surface), on a stable floor that is tight and hard or on one containing cracks of various sizes and made up of boards more or less shattered, thus forming deep crevices for the hiding away, as it were, of the specific organisms.

In the disinfection of human dwellings the fumigation with formaldehyde has proven to be one of the cheapest and ordinarily the most efficient procedures, but it requires a tightly closed room. It is evident that such a method cannot be trusted for the disinfection of most barns, stalls or stables which are usually large compared with dwelling rooms, and what is of far more importance, they are too open. In the disinfection for animal diseases the agents used must from the nature of the buildings in most cases be applied in the form of a solution.

Jæger's investigations brought out very clearly the necessity of adapting the disinfecting agent to the specific kind of organism to be destroyed. For instance, while brushing the surface with a 1–3 milk of chloride of lime destroyed anthrax spores, it was untrustworthy as a disinfectant for the bacteria of tuberculosis and of glanders. For

the destruction of the bacterium of tuberculosis he found carbolic acid and the other coal-tar products very efficient, especially when acidulated with hydrochloric acid. For this purpose he recommended especially Laplace's 4 per cent. solution of crude carbolic acid with two per cent. of hydrochloric acid. In the hands of Jæger, the power to destroy anthrax spores with certainty has been shown only by solutions of carbolic acid and the thick chloride of lime mixture.

A thick milk of lime applied once with a brush Jæger found efficient in the destruction of the microörganisms of chicken cholera, swine erysipelas, typhoid fever, glanders, anthrax (without spores) and *Micrococcus pyogenes.*

Giaxa, in a similar line of work to that of Jæger's, found that in the disinfection of walls even a five per cent. lime wash acting forty-eight hours failed to destroy anthrax spores, the bacterium of tuberculosis and the bacillus of tetanus.

A strong solution of the chloride of lime may be classed as one of the rapidly acting disinfectants for most bacteria, but Jæger's report of its failure when applied to the disinfection of tuberculosis and glanders should be borne in mind. For the cleansing of cattle cars Gruber advises scrubbing them out with hot water or washing with a two per cent. solution of soda at $50°$ C. If the cars are infected, he sprays with a 5 to 10 per cent. solution of formaldehyde.

Disinfectants of value in the disinfection of stables and pens. For disinfecting pens, stable floors and the like the following solutions have been recommended and their careful and intelligent use has shown them to be very efficacious.

Corrosive sublimate (mercuric chloride) 1 ounce in 8 gallons of water (one-tenth of 1 per cent). The water should be put into wooden tubs or barrels and the powdered sublimate added to it. The whole must be allowed to stand for some hours, with frequent stirring, so as to give the sublimate an opportunity to become entirely dissolved. Since this solution is very *poisonous,* it should be kept covered and well guarded. It may be applied with a broom or mop and should be used freely on all woodwork. It combines with albuminous substances and consequently when used the floor and sidings should be cleaned by sweeping and scraping. Its very poisonous nature renders it less desirable for general use than some other solutions.

Carbolic acid. A 5 per cent. solution of carbolic acid is one of the best disinfectants for mangers, feed boxes and fixed watering basins.

It should be applied in quantity sufficient to thoroughly wet all parts and soak deep into the cracks and crevices if there are any.

Chlorinated lime. Five ounces of chloride of lime to a gallon of water (4 per cent). This should be applied in the same way as the corrosive sublimate.

Cresol. The commercial cresols guaranteed to contain more than 90 per cent. of cresylic acid are relatively cheap and well suited to the disinfection of cars, barns, and yards. For general disinfection a 1.5 to 2 per cent. solution of cresol in water should be used, allowance being made for the impurities when the cheaper grades are employed. Cresol is not easily soluble in water; therefore, in preparing solutions warm water should be used and care taken to see that all is dissolved before applying the solution. A 2 per cent. solution of cresol is regarded as being a more efficient disinfectant than a 5 per cent. solution of carbolic acid and should be applied in the same way.

Compound solution of cresol. "This preparation, known as liquor cresolis compositus, United States Pharmacopœia, is recognized as official by the last edition of the United States Pharmacopœia, and is a mixture of equal parts of cresol (U. S. P.) with a linseed-oil-potash soap. The mixture is a thick, dark, amber-colored fluid which mixes readily with water in all proportions to form a clear soapy solution. A very efficient disinfectant may be made from the commercial cresols or liquid carbolic acids of known strength by mixing these with the soap described in the United States Pharmacopœia under the heading Liquor cresolis compositus. When other than United States Pharmacopœia cresol is used a sufficient excess must be added to insure 50 per cent. of actual cresylic acid in the mixture. Compound solution of cresol is recommended for use as a general disinfectant in a 3 to 4 per cent. solution in water. In this strength it will accomplish the same results as a 1.5 to 2 per cent. solution of cresol and may be applied in the same manner as a 5 per cent. solution of carbolic acid.

"It may be said in favor of the compound solution of cresol that it possesses all the advantages of cresol, and in addition is far more readily soluble. It is, however, somewhat more expensive, than cresol, owing to a stronger solution being required; this is in great measure compensated for by its ready solubility."

A mixture of crude carbolic and sulphuric acid. The following disinfectant has been found to be very serviceable. It is not poison-

ous, but quite corrosive, and care should be taken to protect the eyes and hands from accidental splashing:

	Gallon.
Crude carbolic acid	½
Crude sulphuric acid	½

These two substances should be mixed in tubs or 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 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.

Formalin. Formalin is being highly recommended as a disinfectant when used in a 5 per cent. solution. The floors and walls should be thoroughly wet with it.

Ordinary slaked lime. Although it does not possess the disinfecting power of the substances given above, slaked lime is nevertheless very useful. It is well adapted for disinfecting the surface of yards and pens. It is very good to apply to the ceilings and walls of stables.

There are a number of other substances that may be used, such as a solution of blue vitriol or creolin.

In disinfecting stables and pens all litter which has accumulated should be removed before applying the disinfectant. As the litter itself is infected it should be disinfected as well as the stable floor and walls. The most efficient method for disinfecting the litter is fire. The practice of washing the floors and ceiling with water before applying the disinfectant has in most instances the disadvantage that the water carries the microörganisms to be destroyed into cracks and possibly through the floor, where they will not be affected by the later application of the germicide. It is deemed safer to simply use dry cleaners, avoiding dust as much as possible, and to burn the sweepings or to *thoroughly* wet them with a strong disinfectant. The disinfectant is then applied in sufficient quantity to thoroughly saturate the surfaces, including the adhering particles of dirt. The solutions available for stable disinfection are cheap enough to admit of this precaution.

In the application of disinfectants it is well to use a broom and thoroughly scrub the floor and lower part of the walls. This is neces-

sary in order to get the disinfectant through the dirt and into the crevices of the floor. The disinfectants can be applied to the ceilings and upper parts of the side walls with a spray pump.

It is often desirable to disinfect yards where infected animals have been kept. It is well in such cases to carefully scrape together and burn the litter, after which the surface of the soil must be disinfected. For this the milk of lime or a very liberal coating of slaked lime has been recommended. The burning of the surface such as can be done by covering it with a layer of old straw and burning is a more certain destroyer, especially if animal parasites are to be eliminated.

The practical use of disinfectants is a matter requiring much attention if good results are to be attained. It is not wise to trust the disinfection of pens and stables to their owners, unless they are men well versed in the knowledge of disinfection. The failure to properly disinfect stalls and stables is frequently the secret of the failure to stop the ravages of infection.

In the employment of commercial disinfectants, it is necessary also to know the destructive value of the solutions for the organism to be destroyed. There are many so-called disinfectants that, in the strength of the solutions recommended, are little if any better than a poor *antiseptic*. In the destruction of pathogenic microörganisms it is important to keep in mind that the disinfectant must be able to kill the organisms under the conditions in which they are existing.

REFERENCES

1. BRACKEN. Disinfection and disinfectants. 1901.

2. DORSET. Some common disinfectants. *Farmers' Bulletin 345. U. S. Dept. of Agric.*, 1908.

3. POPE. Practical methods of disinfecting stables. *Farmers' Bulletin, No. 480, U. S. Dept. Agric.*, 1912.

4. RIDEAL. Disinfectants and disinfection. London, 1895.

5. ROSENAU. Disinfection and disinfectants. 1902.

6. YOUNG. Notes on disinfectants and disinfection. *Reprinted from the 10th Report of the State Board of Health of Maine*, 1898. (Contains full bibliography on disinfectants and results of experiments.)

APPENDIX
I

STATE SANITARY REQUIREMENTS GOVERNING ADMISSION OF LIVE STOCK

Veterinarians are often asked the health requirements of animals for shipment from one state to another. To afford such assistance as possible on that subject the following pages containing a summary of the regulations of each state are appended. These abstracts of the law were made and published by the Bureau of Animal Industry and issued December 11, 1915.

Where more detailed information is desired, correspondence with the official of the state in question is necessary. This should be done in practically every instance as the laws are being changed from year to year. It is believed, however, that this summary of the state laws will afford an efficient guide to those interested in the subject.

ALABAMA

Horses, mules, and asses.—Health certificate and, if exposed to glanders, mallein test chart must accompany same. Designate each animal as mare, gelding, stallion, jack, jennet, horse mule, or mare mule.

Cattle.—Health certificate, including tuberculin test, for breeding and dairy cattle over 6 months of age and feeding and grazing cattle over 2 years of age. Calves from tuberculous mothers are not admitted. Cattle for feeding under 2 years of age require affidavit of owner that he will keep them separate from other cattle during feeding period.

No ticky cattle, horses, or mules shall be brought into Alabama. Cattle from the area quarantined for splenetic fever shall be accompanied by certificate of inspection or dipping.

Dogs.—Health certificate, stating no exposure to disease.

Hogs.—Health certificate, stating no exposure to cholera or other contagious disease.

Sheep.—Health certificate.

Who may inspect.—Any legally qualified veterinarian who is indorsed by his State veterinarian or by the United States Bureau of Animal Industry.

Official.—State veterinarian, Auburn, Ala., to whom copy of all certificates must be sent.

ARIZONA

Horses, mules, and asses.—Health certificate, preferably including mallein test.

Cattle.—Health certificate. Tuberculin test for dairy or breeding cattle.

Hogs.—Health certificate and isolation at destination two weeks or until released by State veterinarian.

Sheep.—Health certificate for all. Certificate of dipping under official supervision when from any territory classed by the Government as infected.

Who may inspect.—Horses, cattle, and hogs: Any State, Federal, or county veterinarian, or other veterinarian when his certificate is approved by the State veterinarian or State sanitary board at point of origin. Sheep: Federal veterinarian.

Official.—State veterinarian, Phoenix, Ariz., to whom duplicate certificate should be sent in advance.

ARKANSAS

Horses, mules, and asses.—Health certificate, stating particularly that stock is free from ticks.

Cattle.—Health certificate for dairy or breeding cattle, including tuberculin test by official veterinarians.

Hogs.—Must be free from and not exposed to contagious or infectious disease. Swine for exhibition at fairs must be immunized by the Dorset-McBride-Niles serum method and be accompanied by certificate showing same.

Sheep.—Must be free from and not exposed to contagious or infectious disease.

Who may inspect.—Veterinary inspectors of the Bureau of Animal Industry or official veterinarians of the State of origin.

Official.—State veterinarian, Old State House, Little Rock, Ark.

CALIFORNIA

Horses, mules, and asses.—Health certificate. In lieu of health certificate horses, mules, and asses may be brought into California when accompanied by signed statement of State veterinarian or other livestock sanitary authority stating each animal in shipment is free from and has not recently been exposed to any communicable disease.

Cattle.—Dairy cattle and breeding bulls over 6 months of age, health certificate, including tuberculin test. In lieu of health certificate and tuberculin test record dairy cattle and breeding bulls may be brought into California when accompanied by signed statement of State veterinarian or other live-stock sanitary authority stating animals originated in herds free from tuberculosis and other communicable diseases.

Sheep.—In accordance with Federal regulations

Hogs.—Health certificate.

Exemptions.—Animals accompanying emigrant outfits are exempt from all inspection requirements. Animals for theatrical and exhibition purposes are exempt from all inspection requirements provided they do not remain in California.

Note.—Transportation companies should ascertain if California has any special regulations in effect covering the State in which the shipments originate before accepting animals for shipment.

Who may inspect.—Any qualified veterinarian who is a graduate of a duly recognized and accredited veterinary college.

Official.—State veterinarian, Sacramento, Cal.

COLORADO

Horses, mules, and asses.—Health certificate.

Cattle.—Health certificate and tuberculin-test chart for bulls for breeding purposes and female cattle over 6 months old intended for dairy purposes.

Hogs.—Hogs for breeding purposes must be accompanied by affidavit from owner or seller showing them to be free from hog cholera or exposure thereto and a copy of same be sent to the State veterinarian of Colorado. Cars carrying hogs destined to Colorado for purposes other than immediate slaughter must, before loading, be properly disinfected as required by the United States Bureau of Animal Industry.

Sheep.—In compliance with regulations issued by the United States Department of Agriculture.

Who may inspect.—Official veterinarians, State or Federal, or a licensed veterinarian whose certificate is approved by the State veterinarian or like officer.

Official.—State veterinarian, Denver, Colo.

CONNECTICUT

Horses, mules, and asses.—Permit and ophthalmic mallein test.

Cattle.—For neat cattle over 6 months of age, permit from commissioner on domestic animals, health certificate, including tuberculin-test chart, properly filled out and certified to by a qualified veterinarian in any other State who is approved by the authority having jurisdiction of diseases of domestic animals in that State. This certificate must contain a description of each animal, including age, breed, sex, and color, or numbered ear tags, so that animals may be easily identified. When certificate as above described is not provided neat cattle may be taken into the State under a permit from the commissioner on domestic animals and held in quarantine at the place designated until examined and released by the commissioner or his agent.

Hogs.—None.
Sheep.—None.
Who may inspect.—Commissioner or his agent.
Official.—Commissioner on domestic animals, State Capitol, Hartford, Conn.

DELAWARE

Horses, mules, and asses.—None.
Cattle.—Cattle for dairy or breeding purposes admitted to the State on permit from the live-stock sanitary board or must be accompanied by certificate, including tuberculin-test chart showing animals to be free from tuberculosis.
Hogs.—None.
Sheep.—None.
Who may inspect.—Federal or State inspector or veterinarian whose certificate must be approved by State live-stock sanitary board.
Official.—Secretary, State live-stock sanitary board, Wilmington, Del.

DISTRICT OF COLUMBIA

Horses, mules, and asses.—None.
Cattle.—Permit from Chief of Bureau of Animal Industry or health officer of District of Columbia and, except for cattle for immediate slaughter, certificate of tuberculin test by a veterinary inspector of the Bureau of Animal Industry or an official veterinarian of the health department of the District of Columbia or of the State from which the animal is brought. Said certificate must show the place and the date of test and be issued within 30 days of date of entry; also temperature chart, description of the animal or animals, age, markings, and tag numbers if tagged.

Cattle for immediate slaughter may enter the District of Columbia without the tuberculin test, but must be accompanied by a permit as indicated above and tagged by an official of the Bureau of Animal Industry or of the District of Columbia before entry, except that cattle under 6 months old, castrated cattle, and cattle shipped in cars consigned to an establishment having United States meat inspection may enter the District of Columbia for immediate slaughter without permit or tagging.
Hogs.—None.
Sheep.—None.
Officials.—Chief, Bureau of Animal Industry, Washington, D. C.; health officer, Washington, D. C.

FLORIDA

Horses, mules, and asses.—Ophthalmo-mallein test for glanders, recorded on the "Uniform interstate live-stock health certificate."
Cattle.—Tuberculin test, recorded on same form as above.
Hogs.—Protective dose anti-hog-cholera serum within 30 days prior to shipment; or "double treatment" at least 30 days prior to shipment.
Sheep.—In accordance with Federal regulations.
Who may inspect.—Federal and State veterinarians and their deputies.
Transportation vehicles.—Cars, boats, and other vehicles used in transportation of live stock into Florida shall be disinfected in compliance with the regulations of the United States Bureau of Animal Industry governing interstate shipments of live stock.
Officials.—The veterinarian of the Florida State board of health, Jacksonville, Fla.

GEORGIA

Horses, mules, and asses.—None.
Cattle.—All cows, heifers, or bulls shipped or driven into the State must be accompanied by a health certificate, including tuberculin-test record, and on order of proper State official are subject to retest in from 30 to 60 days after arrival in the State. Any cattle not accompanied by a certificate as above described must be held at the State line until inspected and certified to by the State veterinarian of Georgia or his duly accredited deputy, the expense of such inspection to be paid by the owner of said cattle.

All cattle destined to Georgia must be free of ticks, and the owner or shipper must make the following affidavit:

..................Ga., ...191 .

I, ..., under oath, declare that I have carefully inspected and disinfected the following-described cattle, complying with the provisions of the law regulating the suppression and control of infectious and contagious diseases of live stock in the State of Georgia, and the supplemental rules issued for this purpose by the Department of Agriculture, and offer them for shipment from .... in .... County, to .... in .... County, of Georgia, via .... Describe the cattle here...........

....
These cattle are free of ticks. Should they upon inspection while in transit be found infested with ticks, I agree to pay all cost incidental to feeding and disinfection while these cattle are held in quarantine, this cost to be a bona fide lien upon these cattle which shall be paid before the cattle are delivered at destination.

<div align="center">(Owner)</div>

(Signed) ....

<div align="right">(Agent)</div>

Hogs.—If shipped to recognized slaughtering centers for immediate slaughter, none.

Breeding hogs must be inoculated by the serum-alone method not more than 14 days prior to shipment, and the hog and crate must be disinfected in a 2 per cent cresylic-acid compound solution prior to shipment. Shipments must be accompanied by the following affidavit, copy of which shall be sent to the State veterinarian direct:

<div align="center">BREEDERS' SWINE HEALTH CERTIFICATE</div>

I, ..., under oath declare that the following-described swine:

Description of hogs.	Inoculated with anti-hog-cholera serum.		
	Date.	Cubic centimeters.	Serum maker's name.
....	....	....	....
....	....	....	....
....	....	....	....
....	....	....	....

offered by me for shipment from......................to......................
by express are not infected with and have not been exposed to cholera or any other contagious disease. As a special safeguard against exposure during transportation they have been inoculated with the doses of anti-hog-cholera serum hereinbefore stated.

Both hog and crate have been thoroughly disinfected in a 2 per cent solution of cresol compound, U. S. P.

....
<div align="center">(Signature of shipper.)</div>

Sworn to and subscribed before me this .... day of ...., 191...

....
<div align="center">(State title of officer taking oath.)</div>

Sheep.—None

Who may inspect.—Federal veterinarians, State veterinarians, or properly qualified deputies.

Official.—State veterinarian, Atlanta, Ga.

IDAHO

Horses, mules, and asses.—Health certificate, including mallein-test chart.

Cattle.—Health certificate, including tuberculin-test chart.

Hogs.—Health certificate showing hogs to have been immunized by the Dorset-McBride-Niles serum method within 15 days from date of shipment into State. Said certificate must also state that cholera has not existed on the premises from which the hogs were shipped for a period of at least 6 months prior to date of shipment.

Sheep.—Bucks must be dipped under State supervision upon arrival. Sheep can not come farther than 2 miles within the State line until inspected by live-stock inspector in this State.

Who may inspect.—Federal, State, and properly qualified assistant State veterinarians.

Official.—State veterinarian, Boise, Idaho.

ILLINOIS

Horses, mules, and asses.—Certificate of health, showing the animals to be free from contagious or communicable diseases.

Cattle.—All importations of bulls cows, or heifers exceeding the age of 9 months must be covered by a certificate of health, including the tuberculin test administered within 30 days prior to date of shipment or by a permit for their consignment in quarantine for feeding purposes only. Bulls, cows, or heifers less than 9 months of age and steers or spayed heifers must be covered by an affidavit certifying to their classification.

Hogs.—Certificate of health showing the animals to be free from cholera or exposure thereto by being removed from cholera-infected premises.

Note.—Regulations which apply to cattle and swine shall not be interpreted as covering shipments consigned to public stock yards at Chicago, Peoria, or East St. Louis, Ill.

Who may inspect.—State veterinarian and his assistants; inspectors of the United States Bureau of Animal Industry.

Officials.—State veterinarian, Springfield, Ill.; secretary State board of live-stock commissioners, Springfield, Ill.

INDIANA

Horses, mules, and asses.—Health certificate, including certificate of soundness for stallions and jacks, together with an affidavit that they are free from any contagious, infectious, or communicable disease.

Cattle.—Health certificate, including tuberculin test for dairy or breeding cattle indorsed by State or Federal authorities; calves under 6 months of age, health certificate showing that they are from tuberculin-tested and free-from-tuberculosis mothers. Affidavit for cattle intended for feeding purposes.

Sheep.—Health certificate showing that they have been dipped if passing through public stock yards.

Hogs.—For breeding purposes must have health certificate issued in triplicate showing them to be free from disease and that they have not been exposed to disease. For feeding or stocker purposes a permit must be obtained to bring them in, subject to vaccination and quarantine for 30 days on premises of the owner at destination.

Who may inspect.—Veterinary inspectors of the United States Bureau of Animal Industry or authorized State or deputy State veterinarians.

Official.—State veterinarian, Indianapolis, Ind.

IOWA

Horses, mules, and asses.—Health certificate, including mallein test.

Cattle.—For dairy and breeding purposes, health certificate, including tuberculin test.

Cattle other than dairy and breeding cattle, except steers and cattle for immediate slaughter, shall be accompanied by a certificate of health and an affidavit certifying that the title of such cattle will not be transferred and that they will not be used for

other purposes than feeding or slaughter without first notifying the State veterinarian and having them subjected to the tuberculin test.

Hogs.—Except for immediate slaughter, must be accompanied by a certificate of health certifying that they have been immunized with Dorset-McBride-Niles anti-hog cholera serum not more than 30 days prior to date of importation when the serum alone is used and not less than 30 days prior to date of importation when the simultaneous method is used.

Sheep.—Health certificate.

Who may inspect.—Federal, State, or assistant State veterinarians or any graduate veterinarian whose certificate is endorsed by the parties having charge of live-stock sanitation in the State where shipment originates.

Official.—State veterinarian, Des Moines, Iowa.

KANSAS

Horses, mules, and asses.—Health certificate.

Cattle.—Tuberculin-test certificate for dairy and breeding cattle. All other classes of cattle admitted by complying with Bureau of Animal Industry's requirements to move interstate.

Hogs.—Special permits required on hogs entering the State or moving from point to point within the State for any purpose other than immediate slaughter.

Sheep.—No restrictions.

Who may inspect.—Inspectors of the United States Bureau of Animal Industry, veterinarians and inspectors having a commission from the State live-stock sanitary commissioner.

Official.—State live-stock sanitary commissioner, Topeka, Kans.

KENTUCKY

Horses, mules, and asses.—Health certificate. If originating in an area quarantined on account of Texas fever, they shall be disinfected and loaded in a cleaned and disinfected car.

Cattle.—Health certificate, including the tuberculin test for breeding and dairy cattle over 6 months of age.

Health certificate only for stockers and feeders.

Health certificate and tuberculin test not required when cattle are consigned to the Bourbon Stock Yards, Louisville, Ky.

All cattle shall be loaded in cleaned and disinfected cars or crates.

Hogs.—Health certificate stating that hogs have been immunized against cholera by the serum-virus method at least 21 days before shipment or the serum-alone method within 5 days of shipment and sprayed or washed in a disinfectant solution before loaded.

Hogs from public stockyards accepted for immediate slaughter only. All hogs shall be loaded in cleaned and disinfected cars or crates.

Sheep.—Health certificate for pure-bred sheep. Dipping certificate also for stock sheep, except when consigned to points where vats are available for dipping immediately after arrival.

All sheep must be loaded in cleaned and disinfected cars. or crates.

Who may inspect.—State or Federal inspectors or veterinarians whose certificates are indorsed by live-stock sanitary officials of the State in which shipment originates.

Official.—State veterinarian, Frankfort, Ky.

LOUISIANA

Horses, mules, and asses.—Health certificate showing freedom from all contagious, infectious, and communicable diseases.

Cattle.—Dairy and breeding cattle shall be free from tuberculosis and must be tested with tuberculin before entering the State. Railroad agents or owner of cattle must mail certificate to secretary and executive officer of State live-stock sanitary board immediately following arrival of cattle at destination. No tuberculin test is required for cattle under 6 months old. Calves from tuberculous cows shall be rejected.

Hogs.—All swine shipped into the State of Louisiana must be accompanied by a certificate of health showing their freedom from contagious, infectious, or communicable diseases or exposure thereto, certifying that no infectious swine disease has existed in the locality from which shipment originates within a period of 6 months; otherwise certificates must show that such swine have been immunized by the Dorset-McBride-Niles serum-alone method not more than 20 days prior to date of shipment. Railroad stockyards are considered infectious, and no hogs yarded or loaded through them will be accepted in the State of Louisiana for any purpose other than immediate slaughter (48 hours).

Hog-cholera virus or virulent blood should not be shipped by serum manufacturers into the State except by written permission issued by the secretary and executive officer.

Sheep.—Health certificate from qualified veterinarian 24 hours before shipping showing freedom from infectious, contagious, or communicable disease.

Who may inspect.—Federal veterinarians, State veterinarians, deputy State veterinarians, assistant State veterinarians, and other veterinarians provided they are graduates of veterinary schools or colleges recognized by the United States Department of Agriculture and their competency and reliability certified to by authorities in charge of live-stock sanitary control work in the State where shipment originates.

All health certificates and test charts must be made in triplicate on official uniform blanks. The original must be attached to waybill of shipment. Duplicate must be sent to secretary and executive officer in ample time to reach him before arrival of stock. Triplicate should be sent to the proper State official in the State where inspection is made and where shipment originates. Tuberculin-test charts must show that at least 3 temperatures were taken before injection 2 or 3 hours apart and 5 temperatures after injection 2 hours apart, beginning 10 hours after injection of tuberculin.

Official.—Secretary and executive officer of State live-stock sanitary board, Baton Rouge, La.

MAINE

Horses, mules, and asses.—Any person or persons bringing horses into the State of Maine must have a permit and shall notify the live-stock sanitary commissioner within 48 hours after their arrival, who shall at once cause the same to be examined either by a physical examination or to be tested with mallein, or the blood test used, at the expense of the owner, and if an animal is found to be glandered no compensation shall be allowed. No permit or examination will be required for horses used in circuses or to perform on the stage.

Cattle.—That no neat stock (calves, cows, steers, oxen, or bulls) or stags of any age shall be allowed to enter the State from any other State or county, either for dairy purposes or for slaughter (except cattle in transit under the control of the Federal Government), without a permit duly authorized by the live-stock sanitary commissioner, said permit to accompany the shipment. Such animals shall be tested with tuberculin within 30 days of arrival, regardless of any other test made, and shall be held in quarantine upon the premises of the owner until released by the live-stock sanitary commissioner.

Hogs.—Swine imported into Maine shall be kept in quarantine for 90 days on the premises of the owner, who shall notify the live-stock sanitary commissioner upon the arrival; said quarantine may be sooner removed by said commissioner.

Sheep.—None.

Transportation companies (express, railroad, or steamship) shall notify the live-stock sanitary commissioner of the arrival of live stock at their destination.

Who may inspect.—Qualified veterinarians authorized by the live-stock sanitary commissioner.

Official.—Live-stock sanitary commissioner, Augusta, Me.

MARYLAND

Horses, mules, and asses.—None.

Cattle.—Health certificate for feeding cattle and tuberculin test for dairy and breeding cattle accompanied by test chart.

Hogs.—Health certificate.

Sheep.—None.

Who may inspect.—State veterinarian, deputies, and inspectors of the United States Bureau of Animal Industry.

Official.—Chief veterinary inspector, 120 North High Street, Baltimore, Md.

MASSACHUSETTS

Horses, mules, and asses.—Permit required on shipments from New York, Rhode Island, and Connecticut only. Examination on arrival.

Cattle.—Neat cattle for dairy or breeding purposes unless consigned to quarantine station in Brighton stock yards, must be accompanied by a permit of Massachusetts department of animal industry. Tuberculin test required if over 6 months of age, made either by a veterinarian approved by proper official of State where shipment originates or by agent of Massachusetts department on arrival at destination. Quarantined until released by department. Permit not required if for immediate slaughter consigned to abattoir under Federal inspection.

Hogs.—None.

Sheep.—None.

Who may inspect.—Qualified veterinarians whose record of inspection and test is approved by the officials in charge of live-stock interests in the State where the shipment originates. The tests of United States inspectors of the Department of Agriculture are accepted at all times.

Official.—Commissioner of animal industry, room 138, State House, Boston, Mass.

MICHIGAN

Horses, mules, and asses.—Health certificate, including mallein test.

Cattle.—Health certificate for dairy and breeding cattle, including tuberculin test.

Hogs.—None.

Sheep.—None.

Who may inspect.—Veterinarians graduated from an accredited veterinary college and authorized by State officials.

Officials.—State veterinarian, Lansing, Mich.; president live-stock sanitary commission, Lansing, Mich.

MINNESOTA

Horses, mules, and asses.—All branded horses, mules, or asses imported into Minnesota must be accompanied by a health certificate, including mallein test, certifying that animals have been examined and mallein tested within 30 days prior to date of shipment and found free from glanders.

Cattle.—Cattle for breeding or dairy purposes must be tuberculin tested.

Cattle of New York State must show certificate of health and tuberculin test issued and made by an inspector of the United States Bureau of Animal Industry or the chief veterinarian of the New York department of health.

Hogs.—Health certificate.

Sheep.—Health certificate.

Who may inspect.—State veterinarians or assistants, Federal veterinarians, and veterinarians acting under authority of State live-stock sanitary board.

Official.—Secretary and executive officer, live-stock sanitary board, Old Capitol, St. Paul, Minn.

MISSISSIPPI

Horses, mules, and asses.—Health certificate.

Cattle.—Health certificate. Tuberculin test for dairy and breeding cattle.

Hogs.—Health cerrificate.

Sheep.—Health certificate.

Who may inspect.—State veterinarian, assistant State veterinarians, inspectors of the Bureau of Animal Industry.

Official.—State veterinarian, Agricultural College, Miss.

MISSOURI

Horses, mules, and asses.—None specifically required. The statute of the States forbid the importation of animals affected with glanders, farcy, or nasal gleet.

Cattle.—Health certificate for dairy and breeding cattle, including tuberculin test. If any animal in a lot inspected is found tuberculous, the words "exposed to tuberculosis on day of inspection" shall be written on the certificate of health of such animals as pass. Cattle for pasturing, feeding, or immediate slaughter admitted on permit from the State Veterinarian without tuberculin test. Regulations do not apply to cattle shipped to the public stockyards at Kansas City, St. Joseph, and St. Louis, or for exhibition at any fair or live-stock show.

Hogs.—None, except to Pettis County. Hogs to Pettis County must be immunized by a graduate veterinarian.

Sheep.—None specifically required. The statutes of the State forbid the importation of sheep affected with any contagious disease.

Who may inspect.—Official veterinarian, State or Federal, or graduate veterinarian, whose certificate shall be approved in writing by State veterinarian or like officer.

Official.—State veterinarian, Columbia, Mo.

MONTANA

Horses, mules, and asses.—Health certificate, including mallein test. Horses, mules, and asses may be shipped without inspection to quarantine yards at Miles City, Billings, or Dillon, providing the waybills bear the notation "Consigned to quarantine yards at ————. Montana." Animals so shipped will be tested and inspected at owner's expense by a representative of the Montana live-stock sanitary board before being released from quarantine yards. Wild, unbroken, range (but not pasture) horses, mules, or asses may be shipped into Montana on a clinical health certificate, providing each individual animal is given a clinical chute inspection.

Animals for temporary racing, exhibition, or speed purposes may be shipped in on a clinical health certificate.

Stallions or jacks.—In addition to mallein test a certificate of soundness, original of which must accompany shipment and copy mailed to the stallion registration board at Bozeman, Mont., at least 10 days before the importation of stallion or jack into the State. No stallion or jack which is neither pure bred nor grade shall be imported into the State of Montana for breeding purposes. A "grade" is defined as an animal whose sire or dam, but not both, is a registered pure-bred animal.

Cattle.—Cattle for dairying, strictly pasture breeding, all pure breeds and all bulls over 6 months of age must be accompanied by a tuberculin-test chart. Cattle for feeding purposes or for slaughter where no inspection is maintained must be accompanied by a clinical health certificate. Cattle for slaughter where an inspection is maintained do not require a health certificate, but the waybill must be marked "For immediate slaughter," giving the name of the abattoir.

All cattle of any class originating in the State of Wisconsin or New York destined to the State of Montana must be accompanied by a certificate of health issued by a veterinary inspector of the United States Bureau of Animal Industry, the inspection to be made in accordance with the regulations of the live-stock sanitary board and the laws of Montana. Health certificates and test charts are good for 30 days. The original certificate must accompany the shipment to its destination and a duplicate immediately forwarded by the veterinarian making the inspection or test to the State veterinary surgeon, Helena, Mont.

Hogs.—Hogs for feeding or breeding purposes must be accompanied by a clinical health certificate stating the animals do not come from a public stockyard or a district in which hog cholera has existed during the past 6 months, and providing the animals have not been double vaccinated, or at least 90 days have elapsed since they were double vaccinated. Hogs may be shipped from a district where hog cholera has existed during the past six months provided they are shipped not earlier than 30 days and not later than 60 days after receiving the single vaccination and, provided further, that they have been kept since vaccination on premises on which hog cholera has not existed during the past 6 months.

Hogs for slaughter.—Same as for breeding and feeding.

Hogs for exhibition.—All swine to be exhibited in Montana at State or county fairs must be accompanied by a certificate showing they have been immunized by the single vaccination not less than 15 days and not more than 60 days prior to their shipment.

All hogs must be loaded through clean and disinfected pens and chutes into disinfected cars, and must not be unloaded en route in any public stockyard unless the stockyard has been specially disinfected for that purpose.

Sheep.—Health certificate and shipped in disinfected cars. Sheep for grazing or feeding purposes must be inspected upon their arrival at railroad destination in Montana by a Montana inspector, at owner's expense, and quarantined for 90 days on land owned, leased, or controlled by owner of the sheep. Bucks and ewes for dissemination to other bands for breeding purposes shall be dipped twice, with an interval of 10 days, under the supervision of a Montana inspector at the owner's expense and quarantined for at least 90 days on land owned, leased, or controlled by the owner. In all sheep shipments 5 days' notice must be given the State veterinary surgeon's office at Helena before the arrival of the sheep in Montana.

Disinfection of cars.—Disinfection of cars does not apply to box cars which have not been previously used for stock shipments.

Dogs.—Until further notice no dogs or any animals of the canine species will be permitted to enter the State of Montana from the States of Oregon and Minnesota and the counties of Asotin, Garfield, Columbia, Walla Walla, and Benton, in the State of Washington, and the counties of Nez Perce, Idaho, Washington, Canyon, Owyhee, and Latah, in the State of Idaho, unless accompanied by a permit issued by the State veterinary surgeon at Helena, Mont., and an official health certificate from a recognized veterinarian at point of origin stating animals are free from rabies and have not been exposed to rabies during the past 6 months.

Who may inspect.—Federal, State, graduate deputy State veterinarians, or graduate veterinarians whose certificates are indorsed by their State veterinarian or live-stock sanitary board.

Official.—State veterinary surgeon, Helena, Mont.

NEBRASKA

Horses, mules, and asses.—Health certificate.

Cattle.—For dairy or breeding purposes over 6 months old, health certificate including tuberculin test. For feeding, grazing, or range purposes, permit from the deputy State veterinarian of Nebraska without tuberculin test. If not accompanied by a health certificate, cattle will be inspected at destination at owner's expense. For exhibition purposes, permit from deputy State veterinarian without tuberculin test, provided accompanied by proper health certificate. Exhibition cattle remaining in the State three months or more shall be subject to tuberculin test at owner's expense. Cattle for immediate slaughter admitted without inspection. Cattle originating in the States of Illinois and New York shall not be transported, trailed, or driven into the State of Nebraska unless accompanied by certificate of health and tuberculin test issued by an inspector of the United States Bureau of Animal Industry.

Hogs.—Certificate of health showing freedom from all contagious and infectious diseases and that no contagious disease has existed in the locality in which the shipment originated for a period of six months previous to the time of shipment. Railroad cars used for such shipments must be thoroughly disinfected with a 5 per cent solution of carbolic acid before hogs are loaded. Such hogs shall not be unloaded while in transit into any public stockyard. If feeding and watering are necessary, it must be done in the car. No hogs intended for shipment into the State of Nebraska shall be loaded from or unloaded into any public stockyards or ordinary chutes, but must be loaded from wagons and unloaded in the same manner. Hogs shipped to public stockyards for immediate slaughter where Government inspection is maintained need no inspection.

Sheep.—Health certificate stating that they are free from all contagious and infectious disesases. When such shipments originate in a territory where lip-and-leg ulceration or scabies exists, the certificate must show freedom from these diseases.

All shipments of any live stock coming into Nebraska without a proper health certificate as above indicated shall be reported to the deputy State veterinarian by

railroad agent at destination.   Such live stock will be allowed to be taken to the final destination, but will be quarantined on the premises of the owner for inspection and test by an authorized agent of the State at owner's risk.   All animals found to be diseased will be disposed of as directed by the deputy State veterinarian.

Who may inspect.—Federal or State veterinarians or graduate veterinarians authorized by the deputy State veterinarian.

Official.—Deputy State veterinarian, Lincoln, Nebr.

NEVADA

Horses, mules, and asses.—Health certificate, including mallein-test certificate. Physical examination of stallions and jacks for dourine.   Certificate and records of mallein test mailed to State quarantine board, veterinary division, University of Nevada, Reno, Nev., on day of shipment.

Cattle.—Health certificate, including tuberculin test certificate for dairy and breeding cattle.   Exception made in case of range cattle transferred from the ranges of other States to the ranges of Nevada.   In lieu of certificate of inspection owner must mail a statement giving the origin and destination of shipment and the number of bulls, cows, steers, and calves included in same.

Hogs.—None.

Sheep.—Before entrance into State for grazing must notify board (State sheep commission) or any inspector in writing.   Notice not required for sheep in transit unless they remain in State or are unloaded to feed and rest for a longer period than 48 hours.

Who may inspect.—State veterinarians, veterinarians of agricultural colleges and experiment stations, Federal veterinary quarantine officers, or graduate veterinarians certified to by State veterinarians or live-stock sanitary officials.

Officials.—State quarantine board, veterinary division, University of Nevada, Reno, Nev.; executive officer, State sheep commissioners, Lovelace, Nev., or secretary State sheep commission, Reno, Nev.

NEW HAMPSHIRE

Horses, mules, and asses.—None.

Cattle.—Health certificate, including the tuberculin test for all cattle except calves under 6 months old.   Permits allowing shipment will be issued upon receipt of test charts approved by proper live-stock sanitary officials of the State in which the shipment originates.

Hogs.—None.

Sheep.—None.

Who may inspect.—Veterinarians approved by proper live-stock sanitary officials of State of origin.

Official.—Commissioner of agriculture (division of animal industry), Concord, N. H.

NEW JERSEY

Horses, mules, and asses.—None.

Cattle.—Health certificate for dairy and breeding cattle, including tuberculin test.

Hogs.—None.

Sheep.—None.

Who may inspect.—Official veterinarians of the State or competent veterinarians whose health certificate is approved in writing by State officials.

Official.—Secretary State board of health, Trenton, N. J.; secretary commission on tuberculosis in animals, Trenton, N. J.

NEW MEXICO

Horses, mules, and asses.—Health certificate.

Cattle.—Health certificate, including tuberculin test, for dairy cattle or cattle intended for the breeding of dairy cattle.

Hogs.—Subject to hog laws of 1915.   Details obtained from the cattle sanitary board, Albuquerque, N. Mex.

Sheep.—Health certificate. Bucks must be dipped at unloading point.

Who may inspect.—Official veterinarians, State or Federal, for cattle. Sheep must be inspected by a Federal veterinarian before shipment and by State inspector at destination.

Officials.—Secretary cattle sanitary board, Albuquerque, N. Mex.; secretary sheep sanitary board, Albuquerque, N. Mex.

NEW YORK

The movement into the State of New York of domestic animals suffering from any contagious or infectious disease is prohibited, and persons bringing such animals into the State are held responsible.

Horses, mules, and asses.—Must be free from contagious or infectious disease. Such animals from States bordering on New York are required to be shipped subject to health certificate either before or after entrance into the State. Ophthalmic test is accepted as an official test.

Cattle.—Neat cattle for dairy or breeding purposes must be accompanied by certificate of health showing satisfactory tuberculin-test record, such test to be made by a veterinarian approved by proper official of his State, or if not so accompanied must be held in quarantine at destination within State until duly examined by a representative of the State department of agriculture and released

Hogs.—Must be free from contagious or infectious disease.

Sheep.—Must be free from contagious or infectious disease.

Who may inspect.—Federal inspectors, inspectors indorsed by the proper official of the State from which the shipment comes, and the commissioner of agriculture, or duly authorized representatives.

Official.—Chief veterinarian, Albany, N. Y.

NORTH CAROLINA

Horses and asses.—Health certificate when for breeding purposes.

Cattle.—Health certificate, including certificates of tuberculin test when for breeding or dairy purposes.

Hogs.—Health certificate for breeding purposes.

Sheep.—Health certificate for breeding purposes.

Who may inspect—State veterinarians or any veterinarian whose certificate he will indorse; also United States inspectors.

Officials.—State veterinarian, Raleigh, N. C.

NORTH DAKOTA

Horses, mules, and asses.—Health certificate, including mallein test made within 30 days prior to entry into State. Certificate for stallions should, in addition, show the animals to be free from infectious, contagious, or transmissible disease or unsoundness.

Cattle.—Health certificate for all cattle. Cattle over 6 months of age that can be used for breeding or dairy purposes must be accompanied by tuberculin-test chart health certificate.

Cattle that originate or are shipped from the States of New York and Wisconsin and South St. Paul, Minn., must be accompanied by certificate issued by a veterinary inspector of the United States Bureau of Animal Industry. Test and inspection must be made within 30 days of shipment.

Swine.—Health certificate stating that no infectious swine disease exists or has existed in locality from which shipment originated within 6 months prior to date of shipment, unless the swine are certified by a duly accredited Federal or State veterinarian as having been immunized by the Dorset-McBride-Niles hog-cholera immune serum. Swine brought into the State for exhibition purposes at State and county fairs must be accompanied by a certificate stating that such swine have been immunized by the Dorset-McBride-Niles hog-cholera serum.

Sheep.—Health certificate showing them to be free from scabies, lip-and-leg ulceration, or exposure thereto within 30 days prior to date of shipment.

All live stock of any class originating in the State of South Dakota destined to the State of North Dakota must be accompanied by a certificate of health issued by a veterinary inspector of the United States Bureau of Animal Industry or by a veterinarian registered by the United States Bureau of Animal Industry to test horses going to Canada.

Who may inspect.—Federal, State, or deputy State veterinarian or graduate veterinarian whose inspections are indorsed by officials in charge of live-stock sanitary work in the State where inspection is made.

Duplicates of all certificates must be forwarded to the live-stock sanitary board, Bismarck, N. Dak. Certificates issued by veterinarians failing to do this will be refused recognition.

All tests and inspections must be made within 30 days of shipment of stock. ·

Officials.—State veterinarian, Bismarck, N. Dak.; bacteriologist, Agricultural College, North Dakota; State live-stock sanitary board, Bismarck, N. Dak.

OHIO

Horses, mules, and asses.—None.

Cattle.—Health certificate, including tuberculin test for dairy and breeding cattle 6 months of age and over. Tuberculin test must be made within 6 weeks prior to the importation of cattle into this State.

Hogs.—None.

Sheep.—None.

Who may inspect.—Inspectors of the United States Bureau of Animal Industry, veterinarians in the employ of the State Board of agriculture, and veterinarians whose competency, trustworthiness, and reliability are vouched for by the authority in charge of the control of animal diseases in the State from which the animals are shipped.

Official.—State veterinarian, Columbus, Ohio.

OKLAHOMA

Horses, mules, and asses.—Health certificate showing mallein test and stating particularly that stock is free from ticks.

Cattle.—Health certificate, including tuberculin test for dairy or breeding cattle.

Hogs.—For purposes other than immediate slaughter, certificate showing that they have not been exposed to hog cholera for at least 6 months previous to time of shipment and that cars containing them were cleaned and disinfected; that they were not loaded or unloaded en route into public stockyards or stock pens.

Sheep.—None, other than compliance with Federal regulations when shipped from areas under quarantine for scabies.

Who may inspect.—State veterinarians or graduate veterinarians from a school recognized by the United States Bureau of Animal Industry.

Official.—President, Oklahoma State board of agriculture, Oklahoma City, Okla.

OREGON

Horses, mules, and asses.—Health certificate, including mallein, complement-fixation, or other officially accepted test. Imported stallions and mares coming direct from European ports need not be mallein tested.

Cattle.—Health certificate, except for immediate slaughter, including tuberculin test for all dairy and breeding cattle. All cattle excepting settlers' and homesteaders' effects, imported into the State of Oregon from that territory east of the Mississippi River and north of the Tennessee-North Carolina north-boundary line must first receive a written permit from the State veterinarian to be moved into the State of Oregon before such movement can be made. All cattle originating in the States of New York, Wisconsin, or South Dakota must be tuberculin tested by a Federal veterinarian unless otherwise ordered. All cattle from Illinois to be tuberculin tested by Federal veterinarian or veterinarian approved in writing by State veterinarian of Illinois.

Hogs.—Health certificate, except for immediate slaughter, stating that no infectious disease exists or has existed in the locality from which said shipment originated within a period of 6 months prior to shipment. In instances where a veterinarian is so far remote as to prevent examination an affidavit from the owner certifying the animals to be free from exposure to cholera for past 6 months will be accepted in lieu of health certificate. Certificate showing animals to have been immunized by the Dorset-McBride-Niles hog-cholera immune serum is desired where this treatment has been given, stating whether single or double treatment has been given, amount of serum injected, time of injection, and brand of serum used; also, animals must be dipped in a 2 per cent standard disinfecting solution prior to shipment if double treatment has been given; also, animals must be held 30 days after immunization if double treatment has been administered. Disinfected cars, crates, and yards to be used in moving all hogs into or within the State, except those for immediate slaughter.

Sheep.—Health certificate from States in quarantine. Animals must be free from disease. Notice must be given State veterinarian or nearest deputy, stating, by telegraph, telephone, registered letter, or in person, time and place when and where sheep crossed State line, locality from which they came, name and residence of owner or owners and person in control of same, and numbers, brands, and character of the animal. Sheep from quarantined States must be dipped once. Range bucks must be dipped twice after arrival.

Duplicate certificate of inspection to be forwarded this office by veterinarian making inspection, and railroad agent at port of entry into Oregon to forward shipping; also inspection data.

Who may inspect.—Official veterinarians, State or Federal, graduate veterinarians when approved in writing by State veterinarian or like officer for animals, excepting sheep. Sheep to be inspected by official veterinarians, if possible, State or Federal.

Officials.—State veterinarian and secretary, State live-stock sanitary board, Salem, Oreg.

PENNSYLVANIA

Horses, mules, and asses.—Must be free from transmissible diseases.

Cattle.—Apparently healthy calves under 6 months of age and those older for immediate slaughter can be admitted without a health certificate or tuberculin test. Southern cattle for immediate slaughter and those for temporary exhibition purposes can be admitted only on a special permit. All others are to be accompanied by health certificate and a satisfactory tuberculin test.

Hogs.—Must be free from transmissible diseases. Hogs for purposes other than immediate slaughter, if hauled, must be transported in cleaned and disinfected cars or other conveyances. Such swine must not be handled through public stockyards or pens.

Sheep.—Must be free from transmissible diseases.

Who may inspect.—State veterinarian, officially certified inspectors in the State from which cattle originate, agents of the Pennsylvania State live-stock sanitary board, and inspectors of the United States Bureau of Animal Industry.

Official.—State veterinarian and secretary State live-stock sanitary board, Harrisburg, Pa.

RHODE ISLAND

Horses, mules, and asses.—Ophthalmic mallein test, either before or after arrival.

Cattle.—Physical examination; if cattle suspicious, tuberculin test ordered by cattle commissioner.

Hogs.—None.

Sheep.—None.

Who may inspect.—Cattle commissioners of Rhode Island.

Official.—State veterinarian, Providence, R. I.

SOUTH CAROLINA

Horses, mules, and asses.—Health certificate. Mallein test of any exposed animals.

Cattle.—Health certificate, except when intended for immediate slaughter. Tuberculin test for dairy and breeding cattle over 6 months old.

Hogs.—Health certificate, except when intended for immediate slaughter.
Sheep.—Health certificate, except when intended for immediate slaughter.
Who may inspect.—Official veterinarians, State or Federal.
Official.—State veterinarian, Clemson College, S. C.

SOUTH DAKOTA

Horses, mules, and asses.—Health certificate, including mallein test, ophthalmic test being accepted.

Cattle.—Steers, health certificate; bulls and female cattle, health certificate and tuberculin test, except female cattle shipped direct from Mexico, the States of Texas, Arizona, New Mexico, Nevada, Colorado, Utah, Idaho, Wyoming, and Montana, which will be received on health certificate and affidavit of consignee that same will not be used, sold, or offered for sale for dairy or domestic purposes.

Shipments of female cattle from the above points when not made direct must be tuberculin tested, the intradermal test being accepted.

All shipments originating in the State of New York, whether made direct or indirect, must be inspected and tested by an inspector of the United States Bureau of Animal Industry.

Hogs.—For immediate slaughter, health certificate; for breeding purposes, health certificate, and must be shipped in crates or cleaned and disinfected cars, and not loaded or unloaded through any public stockyards.

Sheep.—Health certificate. All bucks and pure-bred sheep for breeding purposes will be held in quarantine at State line or rail or boat destination for 60 days and dipped twice under State supervision. Live-stock sanitary board must be notified of probable time of arrival, that quarantine and dipping may be arranged for.

Who may inspect.—State veterinarian or one of his deputies of the State where shipment originates or an inspector of the United States Bureau of Animal Industry.

Official.—State live-stock sanitary board, Pierre, S. Dak.

TENNESSEE

Horses, mules, and asses.—Health certificate. Horses, mules, and asses originating in a quarantined area quarantined on account of the existence of southern, splenetic, or Texas fever outside of the State of Tennessee shall not at any time be transported, driven, or allowed to drift therefrom into any portion of this State unless they are dipped in a standard arsenical solution either at point of origin, in transit, or on arrival at destination.

Cattle.—For breeding and dairy purposes, health certificate, including tuberculin test of all cattle over 6 months old.

Hogs.—Health certificate. Hogs from public stockyards accepted for immediate slaughter only.

Sheep.—Health certificate.

Who may inspect.—State and Federal inspectors or other qualified veterinarians who are approved by the live-stock sanitary control official of the State in which the shipment originates.

Official.—State veterinarian, State Capitol, Nashville, Tenn.

TEXAS

Horses, mules, and asses.—Health certificate, including mallein test.

Cattle.—Dairy and breeding cattle over 6 months old and cattle for exhibition purposes at any fairs within the State must be accompanied by a certificate of inspection showing them to have been tuberculin tested within 60 days prior to time of entering the State.

Hogs.—Hogs for breeding and stocking purposes or hogs intended for exhibition at any fair within the State must be accompanied by a certificate of inspection showing them to have been immunized by the Dorset-McBride-Niles serum method and to have been dipped or otherwise disinfected in 3 per cent solution of cresol compound, U. S. P.

Sheep.—Health certificate, except when intended for immediate slaughter.

Who may inspect.—Federal, State or other veterinarians whose certificates are indorsed by officials in charge of the live-stock sanitary control work in the State where inspections are made.

Officials.—Chairman live-stock sanitary commission, Fort Worth, Tex.; State veterinarian, Fort Worth, Tex.

UTAH

Horses, mules, and asses.—No horses, mules, or asses shall be admitted into the State unless accompanied by health certificate, including mallein-test chart; and no stallions or jacks shall be admitted unless accompanied by certificate showing that they are not afflicted with dourine, and mares must be certified to as being free from contagious abortion, the tests to have been made not more than 20 days next prior to date of shipment from State of origin.

Cattle.—For dairy or breeding purposes health certificate showing that they are not affected with contagious abortion, and that they have been examined and subjected to the tuberculin test within 40 days prior to shipment and are free from tuberculosis or other contagious disease. In tuberculin and mallein tests at least 3 temperatures must be taken before the injection of tuberculin or mallein, and these not more than 3 hours apart, and 4 temperatures taken after injection not more than 2 hours apart, and beginning not earlier than 10 hours after injection.

Swine.—All swine shipped into the State must be accompanied by health certificate stating that they are free from any infectious or communicable diseases and that no such disease has existed on the premises from which the swine were shipped for a period of at least 6 months prior to shipment. Further, said certificate must show that the swine have been immunized by the Dorset-McBride-Niles hog-cholera serum within 10 days of the date of shipment.

Who may inspect.—Veterinary inspectors of the United States Bureau of Animal Industry, State or deputy State veterinarians of the State in which the shipment originated.

Official.—State inspector, Salt Lake City, Utah.

Sheep.—When any owner or person in charge of sheep desires to bring such sheep into the State from an adjoining State they shall notify the State board of sheep commissioners in writing of such intention at least 10 days before entering the State, indicating the time and place where such sheep shall enter. Provided, however, that no notice will be required when sheep are in transit through the State on railroad cars.

Officials.—President and secretary State board of sheep commissioners, Salt Lake City, Utah.

VERMONT

Horses, mules, and asses.—Must be accompanied by one of the three documents enumerated below:

(a) Permit from Vermont live-stock commissioner as for cattle.

(b) Certificate of inspection and mallein test by a veterinarian whose competency and reliability are certified to by the authorities charged with the control of live-stock sanitary work in the State in which inspection has been made.

(c) Certificate of inspection and mallein test signed by an inspector in the employ of the United States Bureau of Animal Industry.

Cattle.—Must be accompanied by a permit from Vermont live-stock commissioner specifying the number of head and the State or country from which shipment is made and destination in Vermont.

Hogs.—None.

Sheep.—None.

Who may inspect.—Live-stock commissioner and his veterinarians. Tests made in another State for shipment into Vermont are accepted when approved by the proper official of that State.

Official.—Live-stock commissioner, White River Junction, Vt.

VIRGINIA

Horses, mules, and asses.—None.

Cattle.—Health certificate for dairy and breeding cattle, including tuberculin test made within the preceding four months.

Hogs.—Brought into Virginia for purposes other than immediate slaughter to be accompanied by certificate of health by qualified veterinarian properly indorsed by officials of State of origin showing animals to be free from cholera or exposure thereto for period of 6 weeks prior to shipment. Said certificate of health must be presented to State veterinarian of Virginia and approved by him before the animals shall be received into State.

Sheep.—None.

Who may inspect.—Inspectors of the United States Bureau of Animal Industry, State veterinarians, and qualified veterinarians whose certificates are approved in writing by the State veterinarian or live-stock sanitary official of the State in which animals originate.

Official.—State veterinarian, Richmond, Va.

WASHINGTON

Horses, mules, and asses.—Physical inspection.

Cattle.—Tuberculin test for dairy and breeding cattle and special permit from the commissioner of agriculture.

Hogs.—Physical inspection and immunization.

Sheep.—Physical inspection.

Who may inspect.—State veterinarian, assistant State veterinarians, and inspectors of the United States Bureau of Animal Industry.

Official.—Commissioner of agriculture, Olympia, Wash.

WEST VIRGINIA

Horses, mules, and asses.—Certificate of good health from approved veterinarian.

Cattle.—Tuberculin test for dairy and breeding cattle over 6 months old; certificate of good health from approved veterinarian for feeding and grazing cattle.

Hogs.—Certificate of good health from approved veterinarian.

Sheep.—Certificate of good health from approved veterinarian.

Who may inspect.—State veterinarians or their assistants and inspectors of the United States Bureau of Animal Industry.

Official.—Commissioner of agriculture, Charleston, W. Va.

WISCONSIN

Horses, mules, and asses.—Health certificate for native horses. If from localities where glanders is prevalent, all shall be mallein tested. Range horses, branded western, mallein tested.

Cattle.—For dairy, breeding, or when mingled with or intended to be mingled with breeding or dairy cattle after being shipped into the State of Wisconsin, tuberculin test prior to shipment if 6 months old or over. Shipments of calves less than 6 months old shall have statement filed with the bill of lading that the calves are from tuberculin tested dams and fed on milk from clean herds. The term "feeders," "stockers," or "stock cattle" shall be construed as applying to cattle to be shipped into Wisconsin intended or used for immediate feeding purposes, to be held on certain designated premises and not mingled with dairy or breeding cattle or cattle intended for dairy or breeding purposes. The owner or shipper or his agent who shall be in charge of such cattle shall file a certified statement with the State veterinarian that the cattle contained in such shipment will not be mingled with dairy or breeding cattle, and that he will in no manner dispose of same to anyone within the State of Wisconsin unless for immediate slaughter within 10 days; that such cattle will remain in his possession until so slaughtered or reshipped out of the State. Such cattle shall not at any time be tuberculin tested after being shipped into the State of Wisconsin unless application has been first filed with the State veterinarian, who will designate a qualified veterinary surgeon to make such test, which shall be at the expense of the owner.

Swine.—Swine shall have certificate of health certifying to one of the following:

1. None shall have been treated with the "double method" within 30 days of shipment. If so immunized, state date of treatment.

2. If from district within 5 miles of hog-cholera outbreak, must either be immune or have had a treatment of "serum alone" not less than 10 days nor more than 30 days before shipment.

From noninfected districts shipment may be made by owner filing a certificate with the carrier, and a copy must also be sent to the State veterinarian at Madison certifying that such shipment originates from hog-cholera free district.

Who may inspect.—Federal, State, assistant State, or veterinarians whose integrity and competency are vouched for by the official in charge in the State of origin.

Official.—State veterinarian, Madison, Wis.

WYOMING

Horses, mules, and asses.—Health certificate.

Cattle.—Neat cattle, health certificate. All dairy cattle, bulls, and female cattle, registered or pure bred, over 6 months old, health certificate, including tuberculin test. All cattle originating in the States of New York and Wisconsin must be accompanied by Federal health certificate and test chart. Cattle originating in an area under Federal quarantine for any disease must be accompanied by a health certificate issued by an inspector of the United States Bureau of Animal Industry.

Hogs.—For purposes other than immediate slaughter, health certificate showing them free from all contagious, infectious, and communicable diseases and certifying that no infectious swine disease exists or has existed in the locality from which said shipment originated within the period of 6 months; otherwise certificate must show that they have been immunized by the Dorset-McBride-Niles hog-cholera serum not more than 30 days prior to date of shipment.

Live stock of any class originating in the State of Illinois will not be permitted to enter Wyoming.

Who may inspect.—Veterinary inspectors of the United States Bureau of Animal Industry, State veterinarians, or authorized deputies or assistants, or a graduate veterinarian whose reliability and competency are certified to by the proper State authorities in which the animals originate.

Official.—State veterinarian, Cheyenne, Wyo.

Sheep.—Send 10 days' notice to secretary State board of sheep commissioners, Cheyenne, Wyo., inclosing 3 cents for each sheep and 25 cents for each buck. All sheep to be dipped twice at destination within 15 days after arrival in a dip prescribed or recognized by the State board of sheep commissioners for scabies.

Who may inspect.—Federal or State inspectors.

Official.—Secretary-treasurer State board of sheep commmmissioners, Cheyenne, Wyo.

APPENDIX
II

REGULATIONS GOVERNING THE MEAT INSPECTION OF THE UNITED STATES DEPARTMENT OF AGRICULTURE

The question often arises whether or not the flesh of an animal which is suffering from certain morbid conditions is fit for human food. Questions also arise relative to the organization of the Federal Meat Inspection Service and definitions of the terms used in connection with meat inspection. In order to assist those desiring information on these points and also on the present regulations concerning the disposition of carcasses of animals that are found to be suffering from certain diseases, the 17 regulations of the Bureau of Animal Industry dealing with these questions are appended. The regulations concerning the reinspection and preparation of meat and meat products, market inspection and other matters pertaining to the shipment of meat and meat products required by the Federal Government are omitted. Those who are especially interested in this subject should secure from the Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., Order 211 on Regulations Governing the Meat Inspection of the United States Department of Agriculture.

It is the practice generally for boards of health in cities having local meat inspection to adopt the federal regulations. It is also well for practitioners to follow them in inspecting isolated cases for their clients.

Regulation I. Definitions.

SECTION 1. For the purposes of these regulations the following words, phrases, names, and terms shall be construed, respectively, to mean—

Paragraph 1. **The meat inspection act, or act of June 30, 1906**, or act of Congress of June 30, 1906: "An Act Making appropriations for the Department of Agriculture for the fiscal year ending June thirtieth, nineteen hundred and seven," approved June 30, 1906 (34 United States Statutes at Large, pages 674 to 679), as reenacted by "An Act Making appropriations for the Department of Agriculture for the fiscal year ending June thirtieth, nineteen hundred and eight," approved March 4, 1907 (34 United States Statutes at Large, pages 1260 to 1265).

Paragraph 2. **The imported meat act:** The "free list" clause and paragraph 545 of an act entitled "An Act To reduce tariff duties and to provide revenue for the Government, and for other purposes," approved October 3, 1913 (38 United States Statutes at Large, pages 114, 152, 159).

Paragraph 3. **The food and drugs act:** "An Act For preventing the manufacture, sale, or transportation of adulterated or misbranded or poisonous or deleterious foods, drugs, medicines, amd liquors, and for regulating traffic therein, and for other purposes," approved June 30, 1906 (34 United States Statutes at Large, pages 768 to 772), as amended by "An Act To amend section eight of the food and drugs act approved June thirtieth, nineteen hundred and six," approved August 23, 1912 (37 United States Statutes at Large, pages 416 and 417), and by "An Act To amend section eight of an act entitlted 'An Act For preventing the manufacture, sale or transportation of adulterated or misbranded or poisonous or deleterious foods, drugs, medicines, and liquors, and for regulating traffic therein, and for other purposes,' approved June thirtieth, nineteen hundred and six," approved March 3, 1913 (37 United States Statutes at Large, page 732).

Paragraph 4. **The department:** The United States Department of Agriculture.

541

Paragraph 5. **Bureau:** The Bureau of Animal Industry of the United States Department of Agriculture.

Paragraph 6. **Inspector:** An inspector of the Bureau of Animal Industry.

Paragraph 7. **Bureau employees:** Inspectors and all other individuals employed in the Bureau of Animal Industry who are authorized by the chief of bureau to do any work or perform any duty in connection with meat inspection.

Paragraph 8. **Official establishment:** Any slaughtering, meat canning, curing, smoking, salting, packing, rendering, or other similar establishment at which inspection is maintained under these regulations.

Paragraph 9. **Official station:** One or more official establishments included under a single supervision.

Paragraph 10. **"Inspected and passed," or "U. S. inspected and passed," or "U. S. inspected and passed under the act of Congress of June 30, 1906," or "U. S. inspected and passed by Department of Agriculture," or any authorized abbreviations thereof:** That the carcasses, parts of carcasses, meat, meat products, or meat food products so marked have been inspected and passed under these regulations, and that at the time they were inspected, passed, and so marked, they were found to be sound, healthful, wholesome, and fit for human food.

Paragraph 11. **"Passed for sterilization":** That the carcasses, parts of carcasses, meat, meat products, or meat food products so marked have been inspected and passed on condition that they be rendered into lard or tallow as prescribed by regulation 15 or otherwise sterilized by methods approved by the chief of bureau.

Paragraph 12. **"U. S. inspected and condemned," or any authorized abbreviations thereof:** That the carcasses, parts of carcasses, meat, meat products, or meat food products so marked are unsound, unhealthful, unwholesome, or otherwise unfit for human food.

Paragraph 13. **"U. S. retained":** That the article so marked is held for further examination by an inspector to determine its disposal.

Paragraph 14. **"U. S. suspect," or any authorized abbreviation thereof:** That the animal so marked is suspected of being affected with a disease or condition which may require its condemnation, in whole or in part, when slaughtered, and is subject to further examination by an inspector to determine its disposal.

Paragraph 15. **"U. S. condemned."** That the animal so marked has been inspected and found to be immature, or in a dying condition, or to have died otherwise than by slaughter, or to be affected with any other condition or with any disease that will require condemnation of its carcass.

Paragraph 16. **"U. S. refused entry":** That the article so marked, offered for importation, contains a preservative not permitted by these regulations, but contains no substance in conflict with the laws of the foreign country from which exported, and has not been found to be otherwise unsound, unhealthful, unwholesome, or unfit for human food.

Paragraph 17. **Inspection legend:** A mark, or a statement, authorized by these regulations, on an article or on the container of an article, indicating that the article has been inspected and passed for food by an inspector.

Paragraph 18. **Carcass:** All parts, including viscera, of a slaughtered animal that are capable of being used for human food.

Paragraph 19. **Primal parts:** The usual sections, cuts, or parts of the dressed carcass commonly known in the trade, such as sides, quarters, shoulders, hams, backs, bellies, beef tongues, and beef livers, before they have been cut, shredded, or otherwise subdivided preliminary to use in the manufacture of meat food products.

Paragraph 20. **Meat product:** Any edible part of the carcass of any cattle, sheep, swine, or goat, which is not manufactured, cured, smoked, processed, or otherwise treated.

Paragraph 21. **Meat food product:** Any article of food or any article which enters into the composition of food for human consumption, which is derived or prepared, in whole or in part, from any portion of the carcass of any cattle, sheep, swine, or goat, if such portion is all or a considerable and definite portion of the article, except such articles as organo-therapeutic substances, meat juice, meat extract, and the like, which are only for medicinal purposes and are advertised only to the medical profession.

Paragraph 22. **Meat and products:** Carcasses, parts of carcasses, meat, products, food products, meat products, and meat food products of, or derived from, cattle, sheep, swine, and goats, which are capable of being used as food by man.

Paragraph 23. **Meat or product:** Any part or all of meat and products.

Paragraph 24. **Immediate container, or true container:** The unit can, pot, tin, canvas, or other receptacle or covering in which any meat or product is customarily delivered to consumers.

Paragraph 25. **Shipping container, or outside container:** The box, bag, barrel, crate, or other receptacle or covering inclosing any meat or product packed in two or more immediate or true containers.

Paragraph 26. **Person:** Natural persons, individuals, firms, partnerships, corporations, companies, societies, and associations, and every agent, officer, or employee of any thereof. This term shall import both the plural and the singular as the case may be.

Paragraph 27. **Subsidiary:** Any individual, firm, partnership, corporation, company, or association, in whose name any business is done, controlled, or owned, in whole or in part, directly or indirectly, by another.

SECTION 2. Wherever in these regulations the phrase "inspected and passed under the provisions of (or according to) the act of Congress of June 30, 1906" is authorized or required to be used, the phrase "U. S. inspected and passed by Department of Agriculture" may be substituted therefor.

SECTION 3. On and after three years from the date of the order adopting these regulations the phrase "inspected and passed under the provisions of (or according to) the act of Congress of June 30, 1906" shall not be used as an inspection legend, unless hereafter expressly authorized by the Secretary of Agriculture upon its being shown to his satisfaction that continuance of the use thereof for a longer period is equitable and is rendered necessary in order to utilize stocks of labels on hand or ordered at the time this regulation takes effect.

Regulation 2. Scope of Inspection.

SECTION 1. Every establishment in which cattle, sheep, swine, or goats are slaughtered for transportation or sale as articles of interstate or foreign commerce, or in which carcasses, parts of carcasses, meat, meat products, or meat food products of, or derived from, cattle, sheep, swine, or goats are, wholly or in part, canned, cured, smoked, salted, packed, rendered, or otherwise prepared for transportation or sale as articles of interstate or foreign commerce which are capable of being used as food for man, shall have inspection under these regulations, except as expressly exempted by regulation 4.

SECTION 2. All cattle, sheep, swine, and goats and all meat and products entering an establishment at which inspection is required by these regulations, and all meat and products prepared, in whole or in part, therein, shall be inspected, handled, prepared, and marked as required by these regulations.

Regulation 3. Organization of Force.

SECTION 1. Meat inspection is conducted, under the direction of the Secretary of Agriculture, by the Bureau of Animal Industry. All permanent employees engaged in the work of meat inspection are appointed upon certification of the United States Civil Service Commission that they have passed the examination prescribed by that commission. These employees are classified as shown in the following sections of this regulation. Promotions are made on the basis of efficiency, deportment, and length of service.

SECTION 2. **Inspectors in charge.** These are inspectors assigned to supervise and perform official work at each official station. Such employees report directly to the chief of bureau and are chosen by reason of their fitness for responsibility as determined by their records in the service. At stations where slaughtering is conducted, only veterinary inspectors are placed in charge.

SECTION 3. **Veterinary inspectors.** All applicants examined for these positions must be graduates of veterinary colleges, accredited by the United States Civil Service Commission, having a course of not less than three years leading to a degree. Veteri-

nary inspectors perform all final post-mortem examinations and enforce the sanitary requirements in their respective departments, under the direction of the inspector in charge.

SECTION 4. **Traveling veterinary inspectors.** These employees inspect official stations and the conduct of operations and ascertain whether the regulations and instructions governing meat inspection are properly observed. They also confer with and instruct bureau employees with a view to uniformity and efficiency of the service, and report thereon, with recommendations, to the chief of bureau.

SECTION 5. **Laboratory Inspectors.** These employees possess technical education and training in the microscopical and chemical examination of meat and products, and their inspections are conducted in laboratories located at various slaughtering centers. Pathological laboratories are also maintained, to which diseased specimens may be sent, when necessary, for diagnosis.

SECTION 6. **Lay inspectors.** These employees are laymen who assist veterinary inspectors in ante-mortem and post-mortem inspections, supervise the curing, canning, packing, and other preparation, handling, and marking of meat and products, examine such articles to detect unsound or unfit conditions, assist in the enforcement of sanitary requirements, and perform various other duties.

Regulation 4. Applications for Inspection or Exemption; Retail Butchers, Retail Dealers, and Farmers; Declarations for Inedible Products Establishments.

SECTION 1. *Paragraph 1.* The proprietor or operator of each establishment of the kind specified in section 1 of regulation 2 shall make application to the Secretary of Agriculture for inspection or for exemption from inspection, or shall file with the Secretary of Agriculture the declaration prescribed by paragraph 1 of section 6 of this regulation. Every application and declaration under this regulation shall be made on a form furnished by the Bureau of Animal Industry, Washington, D. C. In cases where inspection or exemption is already in effect, new applications for inspection or exemption shall not be required. In cases of change of ownership or change of location, a new application shall be made.

Paragraph 2. Triplicate copies of plans, properly drawn to scale, and of specifications, including plumbing and drainage, of plants shall accompany, and the prints or diagrams required by section 2 of regulation 13 should accompany, applications for inspection.

Paragraph 3 Each application shall specify the names and addresses of all the applicant's subsidiaries doing any of the business described in section 1 of regulation 2 and the location of each establishment of such subsidiaries. Each subsidiary making an application shall specify the name and address of the person, firm, corporation, or association of which it is a subsidiary.

Paragraph 4. Notice in writing shall be given to each applicant granted inspection, specifying the establishment to which the same applies.

Paragraph 5. Inspection or exemption may be refused, or if granted may be revoked, for any false statement in the application therefor.

SECTION 2. Retail butchers and retail dealers in meat and meat food products, supplying their customers, upon making application, pursuant to section 1 of this regulation, may be exempted from inspection. To each one so exempted a numbered certificate of exemption shall be furnished for use with transportation agencies to procure the movement of his products in interstate or foreign commerce. No certificate shall be issued unless all the premises on which the products are prepared and handled are maintained in a sanitary condition. Failure by certificate holders to maintain sanitary conditions or to conform to such of these regulations as apply to them shall be cause for withdrawal of exemption and the cancellation of certificates. Such exempted establishments shall conform to the same regulations as govern official establishments in regard to labelling and the use of dyes, chemicals, and preservatives.

SECTION 3. No holder of a certificate of exemption shall use the same for any purpose except for making shipments in supplying his own customers.

SECTION 4. The carcasses and products of animals slaughtered by any farmer on the farm, provided thay can be identified as such and are sound, healthful, wholesome, and fit for human food, and otherwise meet the requirements of these regulations,

may be transported in interstate or foreign commerce under the provision of section 8 of regulation 25. In order to procure the transportation of such products, a farmer need not apply for exemption from inspection.

SECTION 5. Inspectors shall make inspections to ascertain whether any of these regulations applying to retail butchers, retail dealers, farmers, or other persons has been violated.

SECTION 6. *Paragraph 1.* The proprietor or operator of each grease rendering or grease refining establishment, and of every other establishment which has not been granted inspection or exemption, which prepares or ships any article or articles derived wholly or in part from cattle, sheep, swine, or goats, for interstate or foreign commerce, or in the District of Columbia, a Territory, or other place under the jurisdiction of the United States, which article or articles it is claimed are not capable of being used as food by man, shall file with the Secretary of Agriculture a declaration that none of such articles are for human consumption, nor will be sold or shipped from such establishment otherwise than in compliance with these regulations, and that no article for human consumption derived wholly or in part from cattle, sheep, swine, or goats will be prepared at or in such establishment, or will be sold or shipped therefrom. Such establishments may be inspected at any time to ascertain whether any article derived wholly or in part from cattle, sheep, swine, or goats is prepared therein for human consumption, or whether any declaration filed or offered for filing is false in any particular.

Paragraph 2. The proprietor or operator of each establishment which has been granted inspection or exemption and which prepares therein any article or articles derived wholly or in part from cattle, sheep, swine, or goats for interstate or foreign commerce, or in the District of Columbia, a Territory, or other place under the jurisdiction of the United States, which article or articles it is claimed are not capable of being used as food by man, shall file with the Secretary of Agriculture a declaration that none of such articles are for human consumption nor will be sold or shipped from such establishment otherwise than in compliance with these regulations.

Paragraph 3. The Secretary of Agriculture may refuse to file, or if previously filed may withdraw from his files and cancel, any declaration which is false in any particular or the terms of which are violated in any respect.

Regulation 5.   Official Numbers and Inauguration and Withdrawal of Inspection.

SECTION 1. *Paragraph 1.* To each establishment granted inspection an official number shall be assigned. Such number shall be used to identify all inspected and passed meat and products prepared in the establishment.

Paragraph 2. Two or more official establishments under the same ownership or control may be granted the same official number, provided a serial letter is added in each case to identify each establishment and the products thereof.

Paragraph 3. No meat or product shall be handled or prepared in an official establishment for a subsidiary of the proprietor or operator, nor shall any article handled or prepared therein be sold or transported in interstate or foreign commerce by or in the name of a subsidiary of the proprietor or operator, unless such subsidiary is named in an application of the establishment for inspection, and is granted inspection in such establishment, under these regulations.

SECTION 2. Each official establishment shall be separate and distinct from any unofficial establishment in which any meat or product is handled.

SECTION 3. Inspection shall not be begun if an establishment is not in a sanitary condition nor unless the establishment provides and agrees to maintain adequate facilities for conducting such inspection.

SECTION 4. When an application for inspection is granted, the inspector in charge shall, at or prior to the inauguration of inspection, inform the proprietor or operator of the establishment of the requirements of these regulations. If the establishment, at the time inspection is inaugurated, contains any meat or product which has not theretofore been inspected, passed, and marked in compliance with these regulations, the identity of the same shall be maintained and it shall not be transported or offered

for transportation in interstate or foreign commerce, or otherwise dealt with, as inspected and passed under these regulations. The establishment shall adopt and enforce all necessary measures, and shall comply with all such directions as the inspector in charge may prescribe, for carrying out the purposes of this section.

SECTION 5. Inspection may be withdrawn from any official establishment which violates or fails to comply with any provision of the meat inspection act of these regulations.

SECTION 6. Inspector and other bureau employees shall report to the inspector in charge all violations and failures under section 5 of the regulation of which they have knowledge, and the inspector in charge shall report the same to the chief of bureau.

Regulation 6. Assignment of Bureau Employees.

SECTION 1. The chief of bureau shall designate an inspector in charge of the inspection at each official station, and assign to said inspector such assistants as may be necessary.

SECTION 2. For the purpose of any examination or inspection, bureau employees shall have access at all times, by day or night, whether the establishment be operated or not, to every part of any official establishment to which they are assigned.

SECTION 3. Each bureau employee will be furnished with a numbered official badge, which he shall not allow to leave his possession. This badge shall be sufficient identification to entitle him to admittance at all regular entrances and to all parts of the establishment and premises to which he is assigned, and to any place, at any time, for the purpose of making an inspection pursuant to section 3 of regulation 23.

SECTION 4. No bureau employee shall be detailed for duty at an establishment where any member of his family is employed by the establishment. Bureau employees are forbidden to solicit, for anyone, employment at any official establishment, or by any officer, manager, or employee thereof.

Regulation 7. Facilities for Inspection.

SECTION 1. Office room, including light and heat, shall be provided by official establishments, rent free for the exclusive use, for official purposes, of the inspector and other bureau employees assigned thereto. The room or rooms set apart for this purpose shall meet with the approval of the inspector in charge and shall be conveniently located, properly ventilated, and provided with lockers suitable for the protection and storage of bureau supplies and with facilities suitable for the dressing of bureau employees.

SECTION 2. Each official establishment shall inform the inspector in charge, or his assistant, when work in each department has been concluded for the day, and of the day and hour when work will be resumed therein. Whenever any meat or product is to be overhauled or otherwise handled in an official establishment during unusual hours, the establishment shall, a reasonable time in advance, notify the inspector in charge, or his assistant, of the day and hour when such work will be commenced, and such articles shall not be so handled except after such notice has been given. No department of an official establishment shall be operated except under the supervision of a bureau employee. All slaughtering of animals and preparation of meat and products shall be done within reasonable hours, and with reasonable speed, the facilities of the establishment being considered. No shipment of any meat or product shall be made from an official establishment until after due notice has been given to the inspector in charge or his assistant.

SECTION 3. When one inspector is detailed to conduct the work at two or more official establishments where few animals are slaughtered or where but a small quantity of any meat or product is prepared, the inspector in charge may designate the hours during which such establishment may be operated.

SECTION 4. No work shall be performed at official establishments during any day on which such work is prohibited by the law of the State or Territory or District of Columbia in which the establishment is located. However, the department requires that it be judicially determined that such work is so prohibited.

SECTION 5. When required by the chief of bureau or the inspector in charge, the following facilities and conditions, and such others as may be essential to efficient conduct of inspection, shall be provided by each official establishment:

(a) Satisfactory pens, equipment, and assistants for conducting ante-mortem inspection and for separating, marking, and holding apart from passed animals those marked "U. S. suspect" and those marked "U. S. condemned."

(b) Sufficient natural light, and abundant artificial light at times of the day when natural light may not be adequate, at places for inspection. Such places shall be kept sufficiently free of steam and vapors for inspection to be properly made.

(c) Racks, receptacles, or other suitable devices for retaining such parts as the head, tongue, tail, thymus gland, and viscera, and all parts and blood to be used in the preparation of meat food products or medical products, until after the post-mortem examination is completed, in order that they may be identified in case of condemnation of the carcass; equipment, trucks, and receptacles for the handling of viscera of slaughtered animals so as to prevent contact with the floor; trucks, racks, marked receptacles, tables, or other necessary equipment for the separate and sanitary handling of carcasses or parts passed for sterilization.

(d) Tables, benches, and other equipment on which inspection is performed, of such design, material, and construction as to enable bureau employees to conduct their inspection in a ready, efficient, and cleanly manner.

(e) Sanitary water-tight metal trucks or receptacles for holding and handling diseased carcasses and parts; such trucks or receptacles to be marked in a conspicuous manner with the phrase "U. S. condemned," in letters not less than 2 inches high, and, when required by the inspector in charge, to be equipped with facilities for locking or sealing.

(f) Adequate arrangements, including disinfectants, for cleansing and disinfecting hands, for sterilizing all implements used in dressing diseased carcasses and for disinfecting hides, floors, and such other articles and places as may be contaminated by diseased carcasses or otherwise.

(g) In establishments in which slaughtering is done, rooms, compartments, or specially prepared open places, to be known as "final inspection places," at which the final inspection of retained carcasses shall be conducted. Final inspection places shall be sufficient in size and their rail arrangement and other equipment shall be adequate to prevent carcasses and parts passed for food or sterilization from being contaminated by contact with condemned carcasses or parts. They shall be equipped with hot water, stationary washstands, and sanitary tables and other apparatus essential to a ready, efficient, and sanitary conduct of the inspection. The floors shall be of sanitary construction and shall have proper sewer connections, and when the final inspection place is part of a larger floor it shall be separated by a curb and railing.

(h) In each establishment at which any condemned article is held until a day subsequent to its condemnation, a suitably located room or compartment in which the same shall be placed. This room or compartment shall be secure, rat proof, and susceptible of being kept clean, including a sanitary disposal of the floor liquids. It shall be equipped for secure locking, and shall be held under a lock furnished by the department, the key of which shall not leave the custody of a bureau employee. The door or doors of such room or compartment shall be conspicuously marked with the phrase "U. S. condemned," in letters not less than 2 inches high.

(i) Rooms, compartments, and receptacles in such number and in such locations as the needs of the inspection in the establishment may require, in which carcasses and products may be held for further inspection. These shall be equipped for secure locking and shall be held under locks furnished by the department, the keys of which shall not leave the custody of bureau employees. Every such room, compartment, or receptacle shall be conspicuously marked with the phrase "U. S. retained," in letters not less than 2 inches high.

(j) Adequate facilities, including denaturing materials, for the proper disposal of condemned articles in accordance with these regulations. Tanks which, under these regulations, must be sealed shall be properly equipped for sealing as may be specified by the chief of bureau.

(*k*) Docks and receiving rooms, to be designated by the establishment, with the approval of the inspector in charge, for the receipt and inspection of all meat and products as provided in section 4 of regulation 18.

(*l*) Suitable lockers in which brands bearing the inspection legend shall be kept when not in use. All such lockers shall be equipped for locking with locks to be supplied by the department, the keys of which shall not leave the custody of bureau employees.

SECTION 6. Inspectors shall furnish their own implements, such as knives, steels, and triers, for conducting inspection, and shall cleanse their hands and implements as prescribed by paragraph 3 of section 7 of regulation 8.

Regulation 8.   Sanitation.

SECTION 1. Prior to the inauguration of inspection, an examination of the establishment and premises shall be made by a bureau employee and the requirements for sanitation and the necessary facilities for inspection specified.

SECTION 2. Triplicate copies of plans, properly drawn to scale, and of specifications, including plumbing and drainage for remodeling plants of official establishments and for new structures, shall be submitted to the chief of bureau in advance of construction.

SECTION 3. *Paragraph 1.* Official establishments, establishments at which market inspection is conducted, and premises on or in which any meat or product is prepared or handled by or for persons to whom certificates of exemption have been issued, shall be maintained in sanitary condition, and to this end the requirements of paragraphs 2 to 8 inclusive, of this section shall be complied with.

Paragraph 2. There shall be abundant light, both natural and artificial, and sufficient ventilation for all rooms and compartments, to insure sanitary condition.

Paragraph 3. There shall be an efficient drainage and plumbing system for the establishment and premises, and all drains and gutters shall be properly installed with approved traps and vents.

Paragraph 4. The water supply shall be ample, clean, and potable, with adequate facilities for its distribution in the plant. Every establishment shall make known, and whenever required shall afford opportunity for inspection of, the source of its water supply and the location and character of its reservoir and storage tanks.

Paragraph 5. The floors, walls, ceilings, partitions, posts, doors, and other parts of all structures shall be of such materials, construction, and finish as will make them susceptible of being readily and thoroughly cleaned. The floors shall be kept watertight. The rooms and compartments used for edible products shall be separate and distinct from those used for inedible products.

Paragraph 6. The rooms and compartments in which any meat or product is prepared or handled shall be free from odors from dressing and toilet rooms, catch basins, hide cellars. casing rooms, inedible tank and fertilizer rooms, and stables.

Paragraph 7. Every practicable precaution shall be taken to keep establishments free from flies, rats, mice, and other vermin. The use of rat poisons is prohibited in rooms or compartments where any unpacked meat or product is stored or handled; but their use is not forbidden in hide cellars, inedible compartments, outbuildings, or similar places, or in storerooms containing canned or tierced products. So-called rat viruses shall not be used in any part of an establishment or the premises thereof.

Paragraph 8. Dogs shall not be admitted into official establishments, except upon permission of the inspector in charge, for the purpose of destroying rats. Dogs which are admitted shall be kept free from tape-worm infestation. Such examinations shall be made to determine freedom from infestation as the chief of bureau may prescribe. Contamination by the excreta of these animals shall not be permitted, nor shall the dogs be allowed to eat the raw viscera of cattle, sheep, swine, or goats.

SECTION 4. Adequate sanitary facilities and accommodations shall be furnished by every official establishment. Of these the following are specifically required:

(*a*) Dressing rooms, toilet rooms, and urinals, sufficient in number, ample in size, conveniently located, properly ventilated, and meeting all requirements as to sanitary construction and equipment. These shall be separate from the rooms and compart-

ments in which meat and products are prepared, stored, or handled. Where both sexes are employed, separate facilities shall be provided.

(*b*) Modern lavatory accommodations, including running hot and cold water, soap, towels, etc. These shall be placed in or near toilet and urinal rooms and also at such other places in the establishment as may be essential to assure cleanliness of all persons handling any meat or product.

(*c*) Properly located facilities for disinfecting and cleansing utensils and hands of all persons handling any meat or product.

(*d*) Cuspidors of such shape as not readily to be upset and of such materials as to be readily disinfected. They shall be sufficient in number and asccessibly placed in all rooms and places designated by the inspector in charge, and all persons who expectorate shall be required to use them.

SECTION 5. Equipment and utensils used for preparing, processing, and otherwise handling any meat or product shall be of such materials and construction as will make them susceptible of being readily and thoroughly cleaned and such as will insure strict cleanliness in the preparation and handling of all meat and products. Trucks and receptacles used for inedible products shall bear some conspicuous and distinctive mark and shall not be used for handling edible products.

SECTION 6. Rooms, compartments, places, equipment, and utensils used for preparing, storing, or otherwise handling any meat or product, and all other parts of the establishment, shall be kept clean and sanitary.

SECTION 7. *Paragraph 1.* Operations and procedures involving the preparation, storing, or handling of any meat or products shall be strictly in accord with cleanly and sanitary methods.

Paragraph 2. Rooms and compartments in which inspections are made and those in which animals are slaughtered or any meat or product is processed or prepared shall be kept sufficiently free of steam and vapors to enable bureau employees to make inspections and to insure cleanly operations. The walls and ceilings of rooms and compartments under refrigeration shall be kept reasonably free from moisture.

Paragraph 3. Butchers and others who dress or handle diseased carcasses or parts shall, before handling or dressing other carcasses or parts, cleanse their hands of grease, immerse them in a prescribed disinfectant, and rinse them in clean water. Implements used in dressing diseased carcasses shall be thoroughly cleansed in boiling water or in a prescribed disinfectant, followed by rinsing in clean water. The employees of the establishment who handle any meat or products shall keep their hands clean, and in all cases after visiting the toilet rooms or urinals shall wash their hands before handling any meat or product or implements used in the preparation of the same.

Paragraph 4. Aprons, frocks, and other outer clothing worn by persons who handle any meat or product shall be of material that is readily cleansed, and only clean garments shall be worn. Knife scabbards shall be kept clean.

Paragraph 5. Such practices as spitting on whetstones, placing skewers or knives in the mouth, inflating lungs or casings, or testing with air from the mouth such receptacles as tierces, kegs, casks, and the like, containing or intended as containers of any meat or product, are prohibited. Only mechanical means may be used for testing.

SECTION 8. The wagons and cars in which any meat or product is transported shall be kept in a clean and sanitary condition. Wagons used in transferring loose meat and products between official establishments shall be closed or so covered that the contents shall be kept clean.

SECTION 9. *Paragraph 1.* Second-hand tubs, barrels, and boxes intended for use as containers of any meat or product shall be inspected when received at the establishment and before they are cleaned. Those showing evidence of misuse rendering them unfit to serve as containers for food products shall be rejected. The use of those showing no evidence of previous misuse may be allowed after they have been thoroughly and properly cleaned. Steaming, after thorough scrubbing and rinsing, is essential to cleaning tubs and barrels.

Paragraph 2. Interiors of tank cars about to be used for the transportation of any meat food product shall be carefully inspected for cleanliness even though the last previous content was edible. Lye and soda solutions used in cleaning must be thoroughly removed by rinsing with clean water. In their examinations bureau employees shall enter the tank with a light and examine all parts of the interior.

SECTION 10. The outer premises of every official establishment, embracing docks and areas where cars and wagons are loaded, and the driveways, approaches, yards, pens, and alleys, shall be properly drained and kept in clean and orderly condition. All catch basins on the premises shall be of such construction and location and be given such attention as will insure their being kept in acceptable condition as regards odors and cleanliness. The accumulation on the premises of establishments of any material in which flies may breed, such as hog hair, bones, paunch contents, or manure, is forbidden. No nuisance shall be allowed in any establishment or on its premises.

SECTION 11. No establishment shall employ in any department where any meat or product is handled or prepared, any person affected with tuberculosis or other communicable disease.

SECTION 12. When necessary, bureau employees shall attach a "U. S. rejected" tag to any equipment or utensil which is insanitary, or the use of which would be in violation of these regulations. No equipment or utensil so tagged shall again be used until made sanitary. Such tag so placed shall not be removed by anyone other than a bureau employee.

Regulation 9. Ante-Mortem Inspection.

SECTION 1. *Paragraph 1*. An ante-mortem examination and inspection shall be made of all cattle, sheep, swine, and goats about to be slaughtered in an official establishment before their slaughter shall be allowed.

Paragraph 2. Such ante-mortem inspection shall be made in pens on the premises of the establishment in which the animals are about to be slaughtered, except as provided in paragraph 3 of this section.

Paragraph 3. At each official station where there are public stockyards, upon approval of the chief of bureaus, ante-mortem inspection may be conducted at the scales or in the pens of the yards. Inspection under this paragraph shall be performed only on animals presented for inspection by an official establishment. Except as provided in section 7 of this regulation, every animal marked as a suspect on such inspection shall be slaughtered at an official establishment of the official station at which the inspection was made. If any such animal be not so slaughtered or disposed of in compliance with section 7 of this regulation, then thereafter no ante-mortem inspection shall be done under this paragraph for the official establishment which presented the animal for inspection, and ante-mortem inspection for that establishment shall be performed only in pens on its premises in accordance with paragraph 2 of this section. Upon the chief of bureau being satisfied at any time that inspection at scales or in pens of public stockyards is being used for speculative or other unfair or unjust purposes by an official establishment or by anyone in whose behalf it presents animals for inspection under this paragraph, then he shall require ante-mortem inspection for that establishment thereafter to be made only in accordance with paragraph 2 of this section. The chief of bureau may at any time withdraw ante-mortem inspection, in whole or in part, from any public stockyards.

Paragraph 4. If an animal marked as a suspect on inspection at public stockyards be not slaughtered by the establishment by which it was presented for inspection, then such animal shall be removed from the place of inspection only under the supervision of a bureau employee, and until slaughtered in compliance with paragraph 3 of this section or disposed of pursuant to section 7 of this regulation, shall remain under the supervision of a bureau employee. Every animal marked as a suspect on inspection in the pens of an official establishment shall be slaughtered on the premises of that establishment unless disposed of pursuant to section 7 of this regulation.

Paragraph 5. The withdrawal of ante-mortem inspection from public stockyards, in whole or in part, shall not be a substitute for, but shall be in addition to, any penalty for violating these regulations elsewhere prescribed by these regulations or prescribed by the meat inspection act.

SECTION 2. *Paragraph 1*. All animals plainly showing on ante-mortem inspection any disease or condition that under these regulations would cause condemnation of their carcasses on post-mortem inspection shall be marked "U. S. condemned" and disposed of in accordance with section 8 of this regulation.

Paragraph 2. All hogs plainly showing on ante-mortem inspection that they are affected with either hog cholera or swine plague shall be marked "U. S. condemned" and disposed of in accordance with section 8 of this regulation.

Paragraph 3. If a hog has a temperature of 106° F. or higher, and is of a lot in which there are symptoms of either hog cholera or swine plague, in case of doubt as to the cause of the high temperature, after being marked for identification, it may be held for a reasonable time, under the supervision of an inspector, for further observation and taking of temperature. Any hog so held shall be reinspected on the day it is slaughtered. If upon such reinspection, or when not held for further observation and taking of temperature, then on the original inspection, the hog has a temperature of 106° F. or higher, it shall be condemned and disposed of in accordance with section 8 of this regulation.

Paragraph 4. All animals showing on ante-mortem inspection symptoms of rabies, tetanus, milk fever, or railroad sickness shall be marked "U. S. condemned" and disposed of in accordance with section 8 of this regulation.

Paragraph 5. Immature animals offered for ante-mortem inspection at any of the places specified in this regulation, and animals found dead or in a dying condition on premises of an official establishment, shall be marked "U. S. condemned" and disposed of in accordance with section 8 of this regulation.

Paragraph 6. All animals which, on ante-mortem inspection, do not plainly show, but are suspected of being affected with, any disease or condition that, under these regulations, may cause condemnation, in whole or in part, on post-mortem inspection, shall be so marked as to retain their identity as suspects until final post-mortem inspection, when the carcasses shall be marked and disposed of as provided elsewhere in these regulations, or until disposed of in accordance with section 7 of this regulation.

Paragraph 7. All seriously crippled animals and animals commonly termed "downers", if not marked "U. S. condemned" under paragraph 1, 2, 3, or 4, shall be marked and treated as suspects in accordance with paragraph 6, of this section.

Paragraph 8. Animals which are known to have reacted to the tuberculin test and which are to be slaughtered at an official establishment shall be marked and treated as suspects in accordance with paragraph 6 of this section.

SECTION 3. All animals required by these regulations to be treated as suspects, or to be marked as suspects, or to be marked so as to retain their identity as suspects, shall be marked by or under the supervision of a bureau employee "U. S. suspect," or with such other distinctive mark or marks to indicate that they are suspects as the chief of bureau may adopt. No such mark shall be removed except by a bureau employee.

SECTION 4. *Paragraph 1.* All hogs, even though not themselves marked as suspects, which are of lots one or more of which have been condemned or marked as suspects under section 2 of this regulation for either hog cholera or swine plague, shall, so far as possible, be slaughtered separately and apart from all other animals passed on ante-mortem inspection.

Paragraph 2. All animals required to be marked as suspects shall be set apart and, except as hereinafter provided, shall be slaughtered separately from other animals at an official establishment. In order to avoid unnecessary suffering, crippled animals and animals commonly termed "downers" should be slaughtered without delay.

SECTION 5. In all cases of emergency slaughter, except as provided in section 23 of regulation 11, the animals shall be inspected immediately before slaughter, whether theretofore inspected or not. When the necessity for emergency slaughter exists the establishment shall notify the inspector in charge of his assistant so that such inspection may be made.

SECTION 6. *Paragraph 1.* When any condition is suspected in which the question of temperature is important, such as hog cholera, swine plague, Texas fever, anthrax, blackleg, pneumonia, or septicemia, and in the case of animals commonly termed "downers," the exact temperature shall be taken and recorded.

Paragraph 2. If any animal has a temperature indicating a diseased condition, in case of doubt as to the cause of the high temperature, after being marked for identification, it may be held for a reasonable time, under the supervision of an inspector, for further observation and taking of temperature, before its final disposal is determined.

Section 7. *Paragraph 1.* The slaughter of an animal which has been marked as a suspect on account of pregnancy or on account of having recently given birth to young, and which has not been exposed to any infectious or contagious disease, is not required. Such animal, together with its young, may be released for breeding or dairy purposes, and when released shall be promptly removed from the stockyards or premises of the establishment where inspected. At the time the animal is released, and immediately before removal, the suspect mark if detachable shall be detached by a bureau employee, who shall report his action to the inspector in charge.

Paragraph 2. Vaccine animals with unhealed lesions of vaccinia, accompanied by fever, which have not been exposed to any other infectious or contagious disease, are not required to be slaughtered and may be disposed of in accordance with paragraph 1 of this section.

Section 8. Animals marked "U. S. condemned" shall be killed by the establishment, if not already dead, and shall not be taken into an establishment to be slaughtered or dressed, nor shall they be conveyed into any department of the establishment used for edible products, but they shall be disposed of and tanked in the manner provided for condemned carcasses in regulation 14. The "U. S. condemned" tag shall not be removed from, but shall remain on, the animal when it goes into the tank. The number of such tag shall be reported to the inspector in charge by the bureau employee who affixed it and also by the bureau employee who supervises the tanking of the animal.

Regulation 10.   Post-Mortem Inspection.

Section 1. A careful post-mortem examination and inspection shall be made of the carcasses and parts thereof of all cattle, sheep, swine, and goats slaughtered at official establishments. Such inspection and examination shall be made at the time of slaughter, except in cases of emergencies provided for in section 23 of regulation 11.

Section 2. The head, tongue, tail, thymus gland, and all viscera, and all parts and blood to be used in the preparation of meat food products or medical products, shall be held in such manner as to preserve their identity until after post-mortem examination has been completed, in order that they may be identified in case of condemnation of the carcass.

Section 3. *Paragraph 1.* Each carcass, including all parts and detached organs thereof, in which any lesion of disease or other condition is found that might render the meat or any organ unfit for food purposes, and which for that reason would require a subsequent inspection, shall be retained by the bureau employee at the time of inspection and taken to the place designated for final inspection. The identity of every such retained carcass, part, and detached organ thereof shall be maintained until the final inspection has been completed. Retained carcasses shall not be either washed or trimmed unless authorized by the inspector.

Paragraph 2. Such devices and methods as may be approved by the chief of bureau may be used for the temporary identification of retained carcasses, parts, or organs. In all cases the identification shall be further established by affixing "U. S. retained" tags as soon as practicable and before final inspection. These tags shall not be removed except by a bureau employee.

Section 4. Each carcass or part which is found on final inspection to be unsound, unhealthful, unwholesome, or otherwise unfit for human food shall be conspicuously marked on the surface tissues thereof by a bureau employee at the time of inspection "U. S. inspected and condemned." Condemned detached organs and parts of such character that they can not be so marked shall be immediately placed in trucks or receptacles which shall be kept plainly marked "U. S. inspected and condemned" in letters not less than 2 inches high. All condemned carcasses, parts, and organs shall remain in the custody of a bureau employee and shall be tanked as required in these regulations at or before the close of the day on which they are condemned, or be locked in the "U. S. condemned" room or compartment. Condemned articles shall not be allowed to accumulate unnecessarily in the condemned room or compartment.

Section 5. *Paragraph 1.* Carcasses and parts passed for sterilization shall be conspicuously marked on the surface tissues thereof by a bureau employee at the time of inspection "Passed for sterilization." All such carcasses and parts shall be steril-

ized in accordance with regulation 15 and until so sterilized shall remain in the custody of a bureau employee.

Paragraph 2. In all cases where carcasses showing localized lesions of disease are passed for food or for sterilization the diseased parts shall be removed before the "U. S. retained" tag is taken from the carcass, and such parts shall be condemned.

SECTION 6. Carcasses and parts found to be sound, healthful, wholesome, and fit for human food shall be passed and marked as provided in these regulations.

SECTION 7. Hog carcasses found before evisceration to be affected with an infectious or contagious disease, including tuberculosis, shall not be eviscerated at the regular killing bed or bench, but shall be retained and separated from other carcasses and taken to the final inspection room or place and there opened and examined. This requirement, however, may be waived for those slaughter floors where the number of animals slaughtered per hour is small and on which the inspection facilities are such as permit a ready, efficient, and sanitary performance of the final inspection without such separation.

SECTION 8. *Paragraph 1.* When a carcass is to be dressed with the skin or hide left on, the skin or hide shall be thoroughly washed and cleaned before evisceration.

Paragraph 2. All hair, scurf, and dirt shall be removed from hog carcasses and the carcasses thoroughly washed and cleaned, before any incision is made for inspection or evisceration.

Paragraph 3. Skins and hides from animals condemned for tuberculosis or any disease communicable to man or other animal may be removed from the establishment, except as provided in section 2 of regulation 11, for tanning or other industrial use; but they shall be removed for these uses only after disinfection as follows: Each skin or hide shall be immersed for not less than five minutes in a 5 per cent solution of liquor cresolis compositus, or a 5 per cent solution of carbolic acid. The process of skinning and dipping shall be conducted entirely in the retaining room, or other specially prepared place approved by the inspector in charge, and under the supervision of a bureau employee.

SECTION 9. The sternum of each carcass shall be split and spread apart at the time of slaughter so as to expose the lungs, heart, liver, and thoracic cavity, in order to allow proper inspection and drainage.

SECTION 10. Carcasses or parts of carcasses shall not be inflated with air. Transferring the caul or other fat from fat to lean carcasses is prohibited.

SECTION 11. When only a portion of a carcass is to be condemned on account of slight bruises, either the bruised portion shall be removed immediately and tanked or the carcass shall be immediately placed in a retaining room and kept until chilled and the bruised portion then removed and tanked.

Regulation 11. Disposal of Diseased Carcasses and Parts.

SECTION 1. The carcasses or parts of carcasses of all animals slaughtered at an official establishment and found at the time of slaughter or at any subsequent inspection to be affected with any of the diseases or conditions named in other sections of this regulation shall be disposed of according to the section of this regulation pertaining to the disease or condition. Owing to the fact that it is impracticable to formulate rules covering every case and to designate at just what stage a process becomes loathsome or a disease noxious, the decision as to the disposal of all carcasses, parts, or organs not specifically covered by these regulations shall be left to the inspector in charge.

SECTION 2. All parts, including hides, hoofs, horns, viscera, intestinal contents, fat, and blood, of animals the carcasses of which show lesions of anthrax, regardless of the extent of the disease, shall be condemned and immediately incinerated or otherwise completely destroyed. The killing bed upon which the animal was slaughtered shall be disinfected with a 1 to 1,000 solution of bichlorid of mercury, and all knives, saws, cleavers, and other instruments which have come in contact with the carcass shall be treated as provided in paragraph 3 of section 7 of regulation 8 before being used upon another carcass.

SECTION 3. *Paragraph 1.* The following principles are declared for guidance in passing on carcasses affected with tuberculosis:

Principle A. No meat should be used for food if it contains tubercle bacilli, or if there is a reasonable possibility that it may contain tubercle bacilli, or if it is impregnated with toxic substance of tuberculosis or associated septic infections.

Principle B. Meat should not be destroyed if the lesions are localized and not numerous, if there is no evidence of distribution of tubercle bacilli through the blood or by other means to the muscles or to parts that may be eaten with the muscles, and if the animal is well nourished and in good condition, since in this case there is no proof, or even reason to suspect, that the flesh is unwholesome.

Principle C. Evidences of generalized tuberculosis are to be sought in such distribution and number of tuberculosis lesions as can be explained only upon the supposition of the entrance of tubercule bacilli in considerable number into the systemic circulation. Significant of such generalization is the presence of numerous uniformly distributed tubercles throughout both lungs, also tubercles in the spleen, kidneys, bones, joints, and sexual glands, and in the lymph glands connected with these organs and parts, or in the splenic, renal, prescapular, popliteal, and inguinal glands, when several of these organs and parts are coincidentally affected.

Principle D. Localized tuberculosis is tuberculosis limited to a single or several parts of organs of the body without evidence of recent invasion of numerous bacilli into the systemic circulation.

Paragraph 2. The meat of animals affected with tuberculosis shall be disposed of as follows:

Rule A. The entire carcass shall be condemned if any of the following conditions occur:

(*a*) When it was observed before the animal was killed that it was suffering with fever.

(*b*) When there is a tuberculous or other cachexia, as shown by anemia and emaciation.

(*c*) When the lesions of tuberculosis are generalized, as shown by their presence not only at the usual seats of primary infection but also in parts of the carcass or in the organs that may be reached by the bacilli of tuberculosis only when they are carried in the systemic circulation. Tuberculous lesions in any two of the following mentioned organs are to be accepted as evidence of generalization when they occur in addition to local tuberculous lesions in the digestive or respiratory tracts, including the lymph glands connected therewith: Spleen, kidneys, uterus, udder, ovary, testicle, adrenal gland, and brain or spinal cord or their membranes. Numerous tubercles uniformly distributed throughout both lungs also afford evidence of generalization.

(*d*) When the lesions of tuberculosis are found in the muscles or intermuscular tissue or bones or joints, or in the body lymph glands as a result of draining the muscles, bones, or joints.

(*e*) When the lesions are extensive in one or both body cavities.

(*f*) When the lesions are multiple, acute, and actively progressive. (Evidence of active progress consists in signs of acute inflammation about the lesions, or liquefaction necrosis, or the presence of young tubercles.)

Rule B. An organ or a part of a carcass shall be condemned under any of the following conditions:

(*a*) When it contains lesions of tuberculosis.

(*b*) When the lesion is localized but immediately adjacent to the flesh, as in the case of tuberculosis of the parietal pleura or peritoneum. In this case not only the membrane or part affected but also the adjacent thoracic or abdominal wall is to be condemned.

(*c*) When it has been contaminated by tuberculous material through contact with the floor or a soiled knife or otherwise.

(*d*) Heads showing lesions of tuberculosis shall be condemned, except that when the heads of hogs from carcasses passed for food of for sterilization and the lesions are slight, are calcified or encapsulated, and are confined to lymph glands in which not more than two glands are involved, the head may be passed for sterilization after the diseased tissues have been removed and condemned.

(*e*) An organ shall be condemned when the corresponding lymph gland is tuberculous.

Rule C. Carcasses showing lesions of tuberculosis should be passed for food when the lesions are slight, localized, and calcified or encapsulated, or are limited to a single or several parts or organs of the body (except as noted in Rule A), and there is no evidence of recent invasion of tubercle bacilli into the systemic circulation. Under this rule carcasses showing such lesions as the following may be passed, after the parts containing the lesions are removed and condemned in accordance with Rule B:

(*a*) In the cervical lymph glands and two groups of visceral lymph glands in a single body cavity, such as the cervical, bronchial, and mediastinal glands, or the cervical, hepatic, and mesenteric glands.

(*b*) In the cervical lymph glands and one group of visceral lymph glands and one organ in a single body cavity, such as the cervical and bronchial glands and the lungs or the cervical and hepatic glands and the liver.

(*c*) In two groups of visceral lymph glands and one organ in a single body cavity, such as the bronchial and mediastinal glands and the lungs, or the hepatic and mesenteric glands and the liver.

(*d*) In two groups of visceral lymph glands in the thoracic cavity and one group in the abdominal cavity, or in one group of visceral lymph glands in the thoracic cavity and two groups in the abdominal cavity, such as the bronchial, mediastinal, and hepatic glands, or the bronchial, hepatic, and mesenteric glands.

(*e*) In the cervical lymph glands and one group of visceral lymph glands in each body cavity, such as the cervical, bronchial, and hepatic glands.

(*f*) In the cervical lymph glands and one group of visceral lymph glands in each body cavity, together with the liver when the latter contains but few localized foci. In this class of carcasses, which will be chiefly those of hogs, the lesions of the liver are considered to be primary, as the disease is practically always of alimentary origin.

Rule D. Carcasses which reveal lesions more severe or more numerous than those described for carcasses to be passed (Rule C), but not so severe nor so numerous as the lesions described for carcasses to be condemned (Rule A), may be rendered into lard or tallow or otherwise sterilized in accordance with regulation 15, if the distribution of the lesions is such that all parts containing tuberculous lesions can be removed.

SECTION 4. *Paragraph 1.* The carcasses of all hogs marked as suspects on antemortem inspection shall be given careful post-mortem inspection, and if it appears that they are affected with either acute hog cholera or swine plague, they shall be condemned.

Paragraph 2. Carcasses of hogs which show acute and characteristic lesions of either hog cholera or swine plague in any organ or tissue, other than the kidneys or lymph glands, shall be condemned. Inasmuch as lesions resembling lesions of hog cholera or swine plague occur in the kidneys and lymph glands of hogs not affected with either hog cholera or swine plague, carcasses of hogs in the kidneys or lymph glands of which appear any lesions resembling lesions of either hog cholera or swine plague shall be carefully further inspected for corroborative lesions. On such further inspection—

(*a*) If the carcass shows such lesions in the kidneys or in the lymph glands or in both, accompanied by characteristic lesions in some other organ or tissue, then all lesions shall be regarded as those of hog cholera or swine plague, and the carcass shall be condemned.

(*b*) If the carcass shows in any organ or tissue, other than the kidneys or lymph glands, lesions of either hog cholera or swine plague, which are slight and limited in extent, it shall be passed for sterilization in accordance with regulation 15.

(*c*) If the carcass shows no indication of either hog cholera or swine plague in any organ or tissue other than the kidneys or lymph glands, it shall be passed for food, unless some other provision of these regulations requires a different disposal.

SECTION 5. *Paragraph 1.* Carcasses of animals showing generalized actinomycosis shall be condemned.

Paragraph 2. Carcasses of animals in a well-nourished condition showing uncomplicated localized actinomycotic lesions may be passed after the infected organs or parts have been removed and condemned, except as provided in paragraph 3 of this section.

Paragraph 3. Heads affected with actinomycosis (lumpy jaw), including the tongue, shall be condemned, except that when the disease of the jaw is slight, strictly localized, and without suppuration, fistulous tracts, or lymph-gland involvement, the tongue if free from disease, may be passed.

SECTION 6. Carcasses of animals affected with, or showing lesions of, any of the following named diseases or conditions shall be condemned:

(*a*) Blackleg.
(*b*) Hemorrhagic septicemia.
(*c*) Pyemia.
(*d*) Septicemia.
(*e*) Texas fever.
(*f*) Malignant epizootic catarrh.
(*g*) Unhealed vaccine lesions.
(*h*) Parasitic ictero-hematuria in sheep.

SECTION 7. Any individual organ or part of a carcass affected with carcinoma or sarcoma shall be condemned. In case the carcinoma or sarcoma involves any internal organ to a marked extent, or affects the muscles, skeleton, or body lymph glands, even primarily, the carcass shall be condemned. In case of metastasis to any other organ or part of a carcass, or if metastasis has not occurred but there are present secondary changes in the muscles (serous infiltration, flabbiness, or the like), the carcass shall be condemned.

SECTION 8. Carcasses of animals showing any disease such as generalized melanosis, pseudo-leukemia, and the like, which affects the system of the animal, shall be condemned.

SECTION 9. All slight, well limited abrasions on the tongue and inner surface of the lips and mouth, when without lymph-gland involvement, shall be carefully excised, leaving only sound, normal tissue, which may be passed. Any organ or part of a carcass which is badly bruised or which is affected by a tumor, an abscess, or a suppurating sore, shall be condemned; and when the lesions are of such character or extent as to affect the whole carcass, the whole carcass shall be condemned. Parts of carcasses which are contaminated by pus shall be condemned.

SECTION 10. All carcasses of animals so infected that consumption of the meat or meat food products thereof may give rise to meat poisoning shall be condemned. This includes all carcasses showing signs of either—

(*a*) Acute inflammation of the lungs, pleura, pericardium, peritoneum, or meninges.
(*b*) Septicemia or pyemia, whether puerperal, traumatic, or without any evident cause.
(*c*) Gangrenous or severe hemorrhagic enteritis or gastritis.
(*d*) Acute diffuse metritis or mammitis.
(*e*) Polyarthritis.
(*f*) Phlebitis of the umbilical veins.
(*g*) Traumatic pericarditis.
(*h*) Any acute inflammation, abscess, or suppurating sore, if associated with acute nephritis, fatty and degenerated liver, swollen soft spleen, marked pulmonary hyperemia, general swelling of lymph glands, or diffuse redness of the skin, either singly or in combination.

Immediately after the slaughter of any animal so diseased, the premises and implements used shall be thoroughly disinfected as prescribed elsewhere in these regulations. The part of any carcass coming into contact with the carcass or any part of the carcass of any animal covered by this section, other than those affected with the diseases mentioned in (*a*) above, or with the place where such diseased animal was slaughtered, or with the implements used in the slaughter thereof, before thorough disinfection of such place and implements has been accomplished, or with any other contaminated object, shall be condemned. In case the contaminated part is not removed from the carcass within two hours after such contact the whole carcass shall be condemned.

SECTION 11. From the standpoint of meat inspection, necrobacillosis (lip-and-leg ulceration) may be regarded as a local affection at the beginning, and carcasses in which the lesions are so localized may be passed for food if in a good state of nutrition, after removing and condemning those portions affected with necrotic lesions. On the

other hand, when emaciation, cloudy swelling of the glandular organs, or enlargement and discoloration of the lymph glands are associated with the affection, it is evident that the disease has progressed beyond the condition of localization to a state of toxemia, and the entire carcass should therefore be condemned as both innutritious and noxious. Septicemia or pyemia may intervene as a complication of the local necrosis, and when present the carcass shall be condemned in accordance with section 6 (*c*, *d*) of this regulation.

SECTION 12. When extensive lesions of caseous lymphadenitis, with or without pleuritic adhesions, are found in the lungs, or if several of the visceral organs contain caseous nodules and the carcass is emaciated, the carcass shall be condemned. When the lesions of caseous lymphadenitis are limited to the superficial glands or to a few nodules in an organ, involving also the adjacent lymph glands, and the carcass is well nourished, the meat may be passed after the affected parts are removed and condemned.

SECTION 13. Carcasses showing any degree of icterus with a parenchymatous degeneration of organs, the result of infection or intoxication, and those which show an intense yellow or greenish yellow discoloration without evidence of infection or intoxication, shall be condemned. Carcasses affected with icterus, the result of conditions other than those before stated in this section, but which lose such discoloration on chilling, shall be passed for food, while those which do not so lose such discoloration may be passed for sterilization. No carcass affected with icterus may be passed for food or for sterilization unless the final inspection thereof is completed under natural light.

SECTION 14. Carcasses which give off the odor of urine or a sexual odor shall be condemned. When the final inspection of such carcasses is deferred until they have been chilled, the disposal shall be determined by the heating test.

SECTION 15. *Paragraph 1.* Carcasses of animals affected with mange or scab in advanced stages, or showing emaciation or extension of the inflammation to the flesh, shall be condemned. When the disease is slight, the carcass may be passed.

Paragraph 2. Carcasses of hogs affected with urticaria (diamond skin disease), *Tinea tonsurans, Demodex folliculorum,* or erythema may be passed after detaching the affected skin, if the carcass is otherwise fit for food.

SECTION 16. *Paragraph 1.* Carcasses of cattle (including the viscera) infested with tapeworm cysts known as *Cysticercus bovis* shall be condemned if the infestation is excessive or if the meat is watery or discolored. Carcasses shall be considered excessively infested if incisions in various parts of the musculature expose on most of the cut surfaces two or more cysts within an area the size of the palm of the hand.

Paragraph 2. Carcasses of cattle showing a slight infestation, that is, not to exceed ten cysts, as determined by a careful examination of the heart, muscles of mastication, tongue, diaphragm and its pillars, and of portions of the carcass rendered visible by the process of dressing, may be passed for food after removal and condemnation of the cysts, with the surrounding tissues, provided the carcasses and parts, appropriately identified by retained tags, are held in cold storage or pickle for not less than twenty-one days under conditions which will insure proper preservation; and provided, further, that if the temperature at which such carcasses and parts are held in cold storage does not exceed 15° F., the period of retention may be reduced to six days. Carcasses which show no cysts except in the heart may be passed for food after retention in cold storage or pickle as above provided, irrespective of the number of cysts in the heart.

Paragraph 3. Carcasses of cattle showing a moderate infestation, that is, a greater number of cysts than mentioned in paragraph 2, but which are not so extensively infested as indicated in paragraph 1, of this section, may be passed for sterilization. In case such carcasses are not sterilized as required by regulation 15, they shall be condemned.

Paragraph 4. Fats of carcasses passed for food or for sterilization under the provisions of paragraphs 2 and 3 may be passed for food provided they are melted at a temperature of not less than 140° F. The edible viscera, except the lungs and heart, of carcasses passed for food or for sterilization under the provisions of paragraphs 2 and 3 may be passed for food without refrigeration or other process of sterilization provided they are found to be free from infestation upon final inspection. The in-

testines, weasands, and bladders from beef carcasses affected with *Cysticercus bovis* which have been passed for food or for sterilization may be used for casings after they have been subjected to the usual methods of preparation and may be passed for such purpose upon completion of the final inspection.

Paragraph 5. The inspection for *Cysticercus bovis* may be omitted in the case of calves under six weeks old. The routine inspection of calves over six weeks old for *Cysticercus bovis* may be limited to a careful examination of the surface of the heart and such surfaces of the body musculature as are rendered visible by the process of dressing

SECTION 17. Carcasses of hogs affected with tapeworm cysts (*Cysticercus cellulosæ*) may be passed for sterilization, but if the infestation is excessive the carcass shall be condemned.

SECTION 18. *Paragraph 1.* In the disposal of carcasses, edible organs, and parts of carcasses showing evidence of infestation with parasites not transmissible to man, the following general rules shall govern: If the lesions are localized in such a manner and are of such a character that the parasites and the lesions caused by them may be radically removed, the nonaffected portion of the carcass, organ, or part of the carcass may be passed for food after the removal and condemnation of the affected portions. If an organ or a part of a carcass shows numerous lesions caused by parasites, or if the character of the infestation is such that complete extirpation of the parasites and lesions is difficult and uncertainly accomplished, or if the parasitic infestation or invasion renders the organ or part in any way unfit for food, the affected organ or part shall be condemned. If parasites are found to be distributed in a carcass in such a manner or to be of such a character that their removal and the removal of the lesions caused by them are impracticable, no part of the carcass shall be passed for food. If the infestation is excessive the carcass shall be condemned. If the infestation is moderate the carcass may be passed for sterilization, but in case such carcass is not sterilized as required by regulation 15 it shall be condemned.

Paragraph 2. In the case of sheep carcasses, affected with tape-worm cysts located in the muscles (*Cysticercus ovis*, so-called sheep measles, not transmissible to man) the carcass may be passed after the removal and condemnation of the affected portions; provided, however, that if upon the final inspection of sheep carcasses retained on account of measles the total number of cysts found embedded in muscle or in immediate relation with muscular tissue, including the heart, exceeds five, this shall be taken to indicate that the cysts are so generally distributed and so numerous that their removal would be impracticable, and the entire carcass shall be condemned or passed for sterilization, according to the degree of infestation. If not to exceed five cysts are found upon final inspection, the carcass may be passed after the removal and condemnation of the affected portions.

Paragraph 3. Carcasses of animals found infested with gid bladder worms (*Cœnurus cerebralis, Multiceps multiceps*) may be passed after condemnation of the affected organ (brain or spinal cord).

Paragraph 4. Organs or parts of carcasses infested with hydatid cysts (*Echinococcus*) shall be condemned

Paragraph 5. Livers infested with flukes shall be condemned.

SECTION 19. Carcasses of animals too emaciated or anemic to produce wholesome meat. and carcasses which show a slimy degeneration of the fat or a serous infiltration of the muscles, shall be condemned.

SECTION 20. Carcasses of animals in advanced stages of pregnancy (showing signs of parturition), also carcasses of animals which have within ten days given birth to young and in which there is no evidence of septic infection, may be passed for sterilization; otherwise, they shall be condemned.

SECTION 21. *Paragraph 1.* Carcasses of calves, pigs, kids, and lambs too immature to produce wholesome meat shall be condemned. Such carcasses shall be considered too immature to produce wholesome meat if (*a*) the meat has the appearance of being water-soaked, is loose, flabby, tears easily, and can be perforated with the fingers; or (*b*) its color is grayish red; or (*c*) good muscular development as a whole is lacking, especially noticeable on the upper shank of the leg, where small amounts of serous infiltrates or small edematous patches are sometimes present between the muscles; or

(*d*) the tissue which later develops as the fat capsule of the kidneys is edematous, dirty yellow or grayish red, tough, and intermixed with islands of fat.

Paragraph 2. All unborn and stillborn animals shall be condemned.

Paragraph 3. Meat and organs such as lungs and livers which have been condemned on account of parasitic infestation or invasion, and the flesh of immature and unborn animals and of animals which have been condemned on account of emaciation and recent parturition, may be utilized at official establishments in the manufacture of poultry feed, provided that such organs or tissues are sterilized by thorough cooking, steam rendering, or desiccation under high temperature. If so utilized, such organs and tissues shall be handled and prepared in rooms or places separate and apart from those in which edible products are handled, prepared, or stored.

SECTION 22, Hogs which have entered the scalding vat alive or which have been suffocated in any way shall be condemned.

SECTION 23. When it is necessary for humane reasons to slaughter an injured animal at night or on Sunday or a holiday when the inspector can not be obtained, the carcass and all parts shall be kept for inspection, with the head and all viscera except the stomach, bladder, and intestines held by the natural attachments. If all parts are not so kept for inspection, the carcass shall be condemned. If on inspection of a carcass slaughtered in the absence of an inspector any lesion or condition is found indicating that the animal was sick or diseased, the carcass shall be condemned.

Regulation 12. Carcasses of Animals Slaughtered Without Ante-Mortem Inspection.

SECTION 1. No carcass of an animal slaughtered in the United States which has not had ante-mortem inspection by a bureau employee shall be brought into an official establishment except that carcasses of cattle, sheep, swine, and goats, slaughtered by a farmer on the farm, to which the head and all viscera other than the stomach, bladder, and intestines, are held by the natural attachments, may be received for inspection at official establishments where there is a veterinary inspector, upon the conditions prescribed in this section. After receipt in an official establishment, every such carcass shall be inspected, and if found to be free from disease and otherwise sound, healthful, wholesome, and fit for human food, it shall be marked with the inspection legend. If found to be diseased, unsound, unhealthful, unwholesome, or otherwise unfit for human food, it shall be marked "U. S. inspected and condemned" and destroyed for food purposes as provided in regulation 14.

Regulation 13.   Tank Rooms and Tanks.

SECTION 1. All tanks and equipment used for rendering or preparing inedible products shall be in rooms or compartments separate from those used for rendering or preparing edible products. There shall be no connection, by means of pipes or otherwise, between tanks, rooms, or compartments containing inedible products and those containing edible products.

SECTION 2. Every official establishment shall file with the department blue prints or other accurate diagrams showing all underground pipe lines and other equipment used to convey edible products and those used to convey inedible products, with a description giving the exact location, terminals, and dimensions of such pipes and other equipment and of all gates, valves, or other controlling apparatus, and designating the lines used for conveying edible products and those used for conveying inedible products, and shall also file a copy thereof with the inspector in charge. Like prints or diagrams of alterations in existing tank rooms or tanks and of new tank rooms or tanks of official establishments shall be furnished to the department and approved by the chief of bureau before the same are constructed. If no such underground pipe line or equipment is used for any of the purposes mentioned in this section, a written statement certifying to that fact and duly signed by the proprietor or operator of the establishment shall be filed with the department.

SECTION 3. *Paragraph 1.* In conveying to the inedible-product tank carcasses of animals which have been condemned on ante-mortem inspection, they shall not be taken through rooms or compartments in which any meat or product is prepared, handled, or stored.

Paragraph 2. Under no circumstances shall the carcass of any animal which has died otherwise than by slaughter be brought into any room or compartment in which any meat or product is prepared, handled, or stored.

Paragraph 3. No dead animal shall, under any circumstances, be brought from outside the premises of an official establishment into any room or compartment thereof where any meat or product is prepared; nor, unless permission therefor in advance shall be obtained from the Secretary of Agriculture, shall any dead animal be brought into rooms or compartments where inedible products are prepared. "Dead animal," within the meaning of this paragraph, shall be construed to include any animal which died without having been inspected under these regulations.

Paragraph 4. Inedible fats from outside the premises of an official establishment shall not be received except into the tank room provided for inedible products, and then only when their receipt into the tank room produces no insanitary condition on the premises. When so received, they shall not enter any room or compartment used for edible products.

Regulation 14.   Tanking and Denaturing Condemned Carcasses and Products.

SECTION 1. *Paragraph 1.* Condemned meat and products at official establishments having facilities for tanking shall, except as hereinafter provided, be disposed of by tanking as follows: The lower opening of the tank shall first be securely sealed by a bureau employee; then the condemned meat and products and a sufficient quantity of coloring matter or otehr substance to be designated by the department shall be placed in the tank in his presence, after which the upper opening shall also be securely sealed by such employee, who shall then see that a sufficient force of steam (not less than 40 pounds) is turned into the tank and maintained a sufficient time effectually to destroy the contents for food purposes.

Paragraph 2. The seals of tanks shall be broken only by a bureau employee, after the product has been rendered as provided in paragraph 1 of this section. The drawing off of the contents of such tanks shall be supervised by a bureau employee. Samples shall be taken by bureau employees as often as required to determine whether the fat or grease is effectively denatured.

Paragraph 3. Rendered fats and greases condemned on reinspection shall be destroyed for food purposes by denaturing with coloring matter or other designated substance.

SECTION 2. Any meat or product condemned at an official establishment which has no facilities for tanking shall, under the supervision of a bureau employee, be denatured with crude carbolic acid or other prescribed agent, or destroyed by incineration. When such meat or product is not incinerated, all containers thereof shall be opened, and all meat shall be freely slashed with a knife, before the denaturing agent is applied.

Regulation 15.   Rendering Carcasses and Parts into Lard and Tallow, and Other Sterilization.

SECTION 1. Carcasses and parts passed for sterilization may be rendered into lard or tallow provided that such rendering is done in the following manner: The lower opening of the tank shall first be securely sealed by a bureau employee, then the carcasses or parts shall be placed in the tank in his presence, after which the upper opening shall be securely sealed by such employee, who shall then see that a sufficient force of steam is turned into the tank. Such carcasses and parts shall be cooked at a temperature not lower than 220° F. for a time sufficient to render them effectually into lard or tallow.

SECTION 2. Establishments not equipped with steaming tanks for rendering carcasses and parts into lard or tallow as provided in section 1 of this regulation may render such carcasses or parts in open kettles under the direct supervision of a bureau employee. Such rendering shall be done at a temperature and for a time sufficient to render the carcasses and parts effectually into lard or tallow, and shall be done only during regular hours of work.

SECTION 3. *Paragraph 1.* Carcasses and parts passed for sterilization and which are not rendered into lard or tallow may be utilized for food purposes provided they are first sterilized by methods, and handled and marked in a manner, approved by the chief of bureau.

Paragraph 2. Any carcasses or parts prepared in compliance with paragraph 1 of this section may be canned if the container be plainly and conspicuously marked so as to show that the product is second grade, class, or quality and has been sterilized.

Regulation 16. Marking, Branding, and Identifying Meat and Products

SECTION 1. *Paragraph 1.* The chief of bureau may approve and authorize the use of abbreviations of marks of inspection under these regulations. Such abbreviations shall have the same force and effect as the respective marks for which they are so authorized to be used

Paragraph 2. Except for the purpose of submitting a sample or samples of the same to the chief of bureau for approval, no person shall make or prepare, or cause to be made or prepared, the inspection legend, or any abbreviation, copy, or representation thereof, without the written authority therefor of the chief of bureau given in advance.

Paragraph 3. No person shall affix or place, or cause to be affixed or placed, the inspection legend, or any abbreviation, copy, or representation thereof, to or on any meat or product, except under the supervision of a bureau employee.

Paragraph 4. No person shall fill or cause to be filled, in whole or in part, with any meat or product, any container bearing, or, within the United States, any container intended to bear, the inspection legend, or any abbreviation, copy, or representation thereof, except under the supervision of a bureau employee.

Paragraph 5. No person shall affix or place, or cause to be affixed or placed, the inspection legend, or any abbreviation, copy, or representation thereof, to or on any container of any meat or product, except under the supervision of a bureau employee.

Paragraph 6. No person shall make, prepare, affix, or use, or cause to be made, prepared, affixed, or used, the inspection legend, or any abbreviation, copy, or representation thereof, except in compliance with these regulations.

Paragraph 7. Advertisements, photographs, and other representations of any meat or product prepared exclusively in official establishments, which contain copies or reproductions of the inspection legend and which are not false or misleading, may be permitted when approved in advance by the chief of bureau.

Paragraph 8. All marks of inspection shall be carefully applied and securely affixed.

Paragraph 9. No person shall remove or cause to be removed from an official establishment any article which these regulations require to be marked in any way unless the same is clearly and legibly marked in compliance with these regulations.

SECTION 2. *Paragraph 1.* Each carcass which has been inspected and passed in an official establishment shall be marked at the time of inspection with the inspection legend and with the number of the establishment. Each primal part shall be likewise marked before it leaves the establishment in which it is first inspected and passed, except as provided in paragraphs 2 and 6 of this section and section 6 of regulation 25.

Paragraph 2. Primal parts which have been inspected and passed but do not bear the inspection legend may be transported from one official establishment to another official establishment, for further processing, in a car, wagon, or other closed container, if the car, wagon, or container be sealed with a departmental seal bearing the inspection legend in compliance with these regulations.

Paragraph 3. All primal parts which have been inspected and passed shall, after processing, bear, plainly and legibly, the inspection legend and the number of the official establishment at which the processing was completed.

Paragraph 4. Inspected and passed sausages and other meat food products in animal casings, of the ordinary "ring" variety or larger, shall bear on the casings the inspection legend and the number of the establishment. Inspected and passed smoked sausages and other meat food products in animal casings, of the smaller varieties, such as Frankfort and Vienna styles, shall bear on the casings one or more marks to each chain or two or more marks to each bunch. When additional markings are required by these regulations, they shall be likewise applied.

Paragraph 5. Meat food products in animal casings, other than sausage, shall be branded with the name of the product, the statement "composed of," and the names of the ingredients arranged in the order of their percentages.

Paragraph 6. Any meat or product of such character or so small that it can not be marked with a brand, and which has been inspected and passed, but does not bear the inspection legend, may be transported in a closed container bearing the inspection legend and other marks required by these regulations. When such container has been opened, the contents thereof may not be further transported in interstate or foreign commerce unless reinspected and packed in a container or containers bearing the inspection legend and other marks required by these regulations. The chief of bureau may authorize meat and products of such character or so small that they can not be marked with a brand, which have been inspected and passed, but do not bear the inspection legend, to be removed from an official establishment in open containers when such articles have been sold by the establishment exclusively for consumption in the city or town at or in which the establishment is located. The chief of bureau may withdraw such privilege of removal in open containers if the same be in any way abused, or if the establishment make any sale of any meat or product which is unsound, unhealthful, unwholesome, or otherwise unfit for human food.

Section 3. *Paragraph 1.* When cereal not in excess of 2 per cent is added to sausage, the product shall be marked with the phrase "cereal added." When water in excess of 3 per cent and cereal are added to certain kinds of sausage, as provided in paragraphs 4 and 5 of section 6 of regulation 18, the product shall be marked "sausage, water, and cereal." When water, but no cereal, is added to certain kinds of sausage, as provided in paragraph 5 of section 6 of regulation 18, the addition of water need not be stated.

Paragraph 2. When coloring matter is used in the preparation of casings, as provided in paragraph 3 of section 6 of regulation 18, the product shall be marked with the phrase "artificially colored."

Section 4. Official establishments shall furnish such ink brands, burning brands, and like devices for marking meat and products as the chief of bureau may require. In advance of manufacture, complete and accurate descriptions and designs of the same shall be submitted to and approved by the chief of bureau. Every such brand and device which bears the inspection legend shall, immediately upon being manufactured, be delivered into the custody of the inspector in charge of the establishment, and shall be used only under the supervision of a bureau employee. When not in use for marking inspected and passed meat and products, all such brands and devices bearing the inspection legend shall be kept locked in properly equipped lockers or compartments, the keys of which shall not leave the possession of a bureau employee.

Section 5. All brands and devices furnished by the bureau for marking articles with the inspection legend, including self-locking seals and presses for lead and wire seals, shall be used only under the supervision of a bureau employee, and when not in use for marking, shall be kept locked in properly equipped lockers or compartments, the keys of which shall not leave the possession of a bureau employee.

Section 6. No brand or device shall be false or misleading. The letters and figures thereon shall be of such style and type as will make a clear impression. The inspection legend and establishment number on brands shall be separate and apart from trade names, marks, or other devices.

Section 7. *Paragraph 1.* Except as provided in paragraphs 2 and 3 of this section and in section 6 of regulation 25, when any inspected and passed meat or product for domestic commerce is moved from an official establishment, or from a place having market inspection under these regulations, the shipping container shall bear a domestic meat label which has been submitted to and received the approval of the department and conforms to the following specifications: The label shall be printed with black ink on white paper of good quality, and shall be not less than $2\frac{3}{4}$ by 4 inches in size. The phrase "domestic meat label" shall be printed inside the border across one end of the label. The word "establishment" and the official number shall constitute the top line of the label and shall be printed in type of such size and style as to make them the most conspicuous part of the label. The following statement shall be printed in uniform style: "The meat or meat food product contained herein has been inspected

and passed under the provisions of the act of Congress of June 30, 1906."[1] The name and address of the establishment, or the name only, may also be printed on the label, at the bottom thereof, in type of such size and style as to be less conspicuous than the establishment number. No word or statement,[1] except as permitted by this paragraph and no picture or other advertising matter, device, or design, shall appear upon the domestic meat label, which in form and substance shall be as follows:

```
┌─────────────────────────────────────────────┐
│  ┌───┐  ┌─────────────────────────────────┐  │
│  │   │  │   ESTABLISHMENT  999            │  │
│  │ L │  │                                 │  │
│  │ A │  │   ──────────────────            │  │
│  │ B │  │                                 │  │
│  │ E │  │   THE MEAT OR MEAT FOOD PRODUCT │  │
│  │ L │  │   CONTAINED  HEREIN  HAS  BEEN  IN- │
│  │   │  │   SPECTED  AND  PASSED  UNDER  THE │
│  │ T │  │   PROVISIONS  OF  THE  ACT  OF  CON- │
│  │ A │  │   GRESS  OF  JUNE  30,  1906.   │  │
│  │ E │  │                                 │  │
│  │ M │  │   ──────────────────            │  │
│  │   │  │                                 │  │
│  │ C │  │   SMITH, JONES & RANKIN,        │  │
│  │ I │  │   NEW YORK, N. Y.               │  │
│  │ T │  │                                 │  │
│  │ S │  └─────────────────────────────────┘  │
│  │ E │                                        │
│  │ M │                                        │
│  │ O │                                        │
│  │ D │                                        │
│  └───┘                                        │
└─────────────────────────────────────────────┘
```
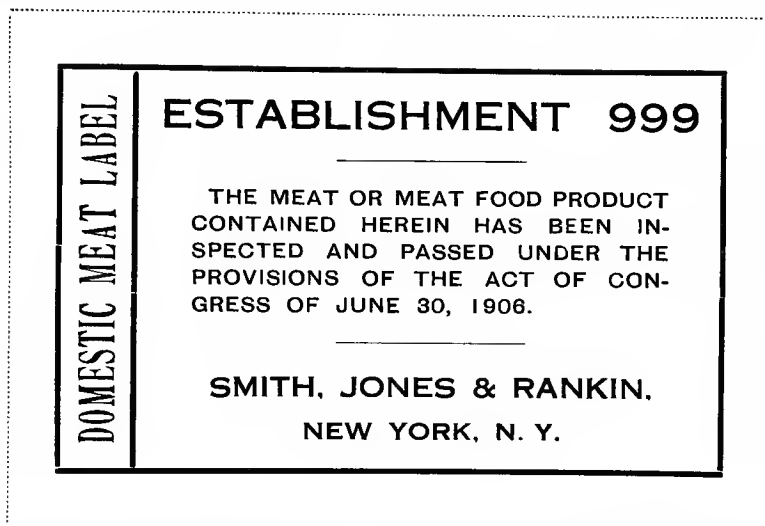
Paragraph 2. When any meat or product prepared in an official establishment for domeistc commerce has been inspected and passed and is inclosed in a cloth wrapping as a shipping container, such wrapping shall bear the inspection legend and establishment number applied by an ink brand, except in those cases in which the inspection legend and establishment number on the articles themselves are clearly legible through the wrapping.

Paragraph 3. The use of domestic meat labels is not required on containers bearing trade labels which have been approved by the department and on which the inspection legend and establishment number appear in plain view after the package is prepared for shipment

Paragraph 4. Domestic meat labels shall be affixed to packages in the manner prescribed in paragraph 2 of section 1 of regulation 24 for affixing meat inspection stamps to export packages.

SECTION 8. The shipping or outside containers of meat and products for export shall be marked in compliance with sections 1 and 6 of regulation 24.

SECTION 9. Both ends of each container, such as tierces and tank cars, of inedible grease, inedible tallow, or other inedible rendered fat, shall be painted white and conspicuously stenciled or burned with the name of the product and the word "inedible" in letters not less than two inches high, or, in the case of tank cars, not less than four inches high.

SECTION 10. *Paragraph 1.* Tank cars carrying inspected and passed product between official establishments shall be equipped for sealing and be securely sealed with seals bearing the inspection legend furnished by the department and affixed by bureau employees.

---

[1]For further information regarding the wording of the inspection legend, see sections 2 and 3 of regulation 1.

Paragraph 2. Each tank car carrying inspected and passed product from an official establishment to any destination other than an official establishment shall have securely affixed thereto a label showing the true name of the product, the inspection legend, the number of the official establishment, and the date of loading.   Before the car is removed from the place where it is unloaded, the carrier shall remove or obliterate such label.

Paragraph 3. When inspected and passed products for export are transferred from tank cars to other containers on boats, such transfer shall be under bureau supervision, and the containers on the boats shall be likewise labeled.

Regulation 17.   Labeling

SECTION 1. *Paragraph 1.* When any inspected and passed meat or product is placed or packed in any can, pot, tin, canvas, or other receptacle or covering in an official establishment, there shall be attached to such container or covering a trade label as hereinafter described.

Paragraph 2. No container or covering which bears or is to bear a trade label shall be filled, in whole or in part, except with articles which have been inspected and passed in compliance with these regulations and which are sound, healthful, wholesome, fit for human food, and strictly in accordance with the statements on the label.   No such container or covering shall be filled, in whole or in part, and no trade label shall be affixed, except under the supervision of a bureau employee.

SECTION 2. *Paragraph 1.* Trade labels shall bear the true name of the meat or product contained in the package, and, except as provided in paragraphs 2 and 5 of this section, shall bear, in prominent letters and figures of uniform size, the phrase "U. S. inspected and passed under the act of Congress of June 30, 1906,"[1] and the number of the official establishment at which the meat or product was prepared, or, if processed, the number of the establishment at which last processed.   Such labels may also bear any other statement, not false or misleading, which has been approved by the department.

Paragraph 2. Trade labels within the meaning of these regulations shall include printed, lithographed, or embossed labels, stickers, seals, wrappers, and receptacles. Metal containers on which the inspection legend is embossed may, with the approval of the department, bear the inspection legend in abbreviated form.

Paragraph 3. Stencils, box dies, inserts, tags, so-called "liners" and "circles" and like devices shall not bear the inspection legend or any abbreviation or representation thereof, nor shall any of them be used in an official establishment unless previously approved by the department.

Paragraph 4. All sealed tin containers of inspected and passed meat and products filled in an official establishment shall have embossed thereon the number of that establishment.   When so marked, the establishment number may be omitted from the trade label or wrapper.   Trade labels shall not be affixed to containers so as to obscure the embossed establishment number.

Paragraph 5. When any meat or product is placed in cartons, or in wrappers of paper or cloth, or in such other containers as the department may approve, the inspection legend and the establishment number may be embodied in a sticker or seal prominently displayed with the trade label, but not necessarily a part thereof.   Such stickers or seals shall not be used without the approval of the department, and shall be securely affixed to the containers under the supervision of a bureau employee after an approved trade label has been affixed.

Paragraph 6. No detachable device bearing the inspection legend or any abbreviation or representation thereof shall be affixed to any meat or product or the container thereof.

SECTION 3. *Paragraph 1.* No trade label shall be used until it has been approved in its final form by the department.   Duplicates of new trade labels in the form of sketches, proofs, or photographic copies shall be submitted through the inspector in charge to the department for approval.   After trade labels from approved sketches

---

[1]For further information regarding the wording of the inspection legend, see sections 2 and 3 of regulation 1.

or proofs are printed, lithographed, or embossed, they shall be submitted in quadruplicate through the inspector in charge for final approval and filing.

Paragraph 2. All trade labels, whether in the form of sketches, proofs, or finished labels, which are submitted to the department for final approval, shall, when the chief of bureau shall so require, be accompanied by a statement showing the kinds and percentages of the ingredients of the product on any container of which it is desired to use the label.   Approximate percentages may be given in cases where the percentages of ingredients may vary from time to time, if the limits of variations are stated.

SECTION 4. Trade labels shall be used only on products for which they are approved. They shall not be applied to any meat or product the container of which bears any statement that is false or misleading.

SECTION 5. Trade labels to be affixed to packages of any meat or product for foreign commerce may be printed in a foreign language.   The inspection legend and the official establishment number shall in all cases appear thereon in English; but, in addition, may appear, literally translated, in foreign languages.

SECTION 6. The name of anyone to whom inspection is granted may appear, without qualification, upon the label or the container of an article prepared for him by the official establishment at which he was granted inspection.   When an article is prepared by an official establishment for a person other than one of those to whom inspection has been granted at that establishment, and the name of such person is to appear upon the label or container thereof, a statement shall be made on the label to the effect that the article was prepared for such person, or the term "distributer" or "distributers" or "distributed by," or other equivalent term, shall be used thereon in connection with the name of such person, or the name of such person shall be used thereon followed by the word "brand" or "style" in the same size and style of lettering as the name of such person.   Whenever the name of such person appears on the label, it shall be prominently placed and lettered and shall not be used so as to be either false or misleading.

SECTION 7. *Paragraph 1.* No meat or product, and no container thereof, shall be labeled with any false or deceptive name; but established trade names which are usual to such articles and are not false or deceptive and which have been approved by the Secretary of Agriculture may be used.

Paragraph 2. No statement, word, picture, design, or device which conveys any false impression or gives any false indication of origin or quality shall appear on any label.   For example:

(*a*) The picture of any swine shall be allowed only on labels used in connection with pork products.

(*b*) Such terms as "special," "fancy," "selected," "best," "finest," "absolutely pure," "100 per cent pure," and the like, without qualification, shall be allowed on labels only in connection with products the quality of which justifies the use of such terms.

(*c*) Names of counties, States, and Territories, and such other geographical names as the department may approve, may be used on labels only when followed by the word "style," "type," "cut," or "brand," in the same size and style of lettering as the geographical name, unless the products for which the labels are intended are prepared in the localities named; provided, that when a geographical name by reason of long usage is recognized as a generic term, indicating a certain style, type, or brand, such a name may be used without the words "style," "type," or "brand," when accompanied by a statement showing the State or Territory in which the product is prepared, if prepared in a State or Territory, and showing the locality in which the product is prepared, if not prepared in a State or Territory.   For example, sausage of the kind commonly known as Vienna sausage may be labeled either "Vienna style sausage" or "Vienna sausage, made in Illinois."   In the latter case the words showing the place of manufacture need not be in the same size and style of lettering as the name of the product, but shall be plain and conspicuous.

(*d*) Names indicative or imitative of distinctive types or breeds of live stock shall not be used on labels unless the products for which such labels are intended are actually derived from carcasses of animals of the type or breed specified.

(*e*) The word "ham," without any prefix indicating the species of animal from which derived, shall be used on labels only in connection with pork hams.

(*f*) The word "fresh" shall not be used on labels in connection with any meat or product the ingredients of which, in whole, or in part, have undergone any process of curing.

(*g*) Such terms as "meat extract" or "extract of beef," without qualification, shall not be permitted on labels in connection with products prepared from organs or parts of the carcass other than fresh flesh. Extracts prepared entirely from parts of the carcass other than fresh flesh shall not be labeled "meat extract," but may be properly labeled with the true names of the parts from which prepared, as, for example, "liver extract." The terms "beef extract" and "extract of beef" without qualification shall be applied only to extracts of fresh beef. Extract of cured beef or of other cured meat shall be designated respectively as "extract of cured beef," "extract of cured meat" or "cured-meat extract." In the latter case the words "cured" and "meat" shall appear on one line in the same size and style of lettering and shall be connected by a hyphen. When beef extract or meat extract is mixed with extract from cured meat or extract derived from the other parts of the carcass, such mixture shall be designated as "compound meat extract," and, in addition, there shall appear on the label a statement showing the ingredients, other than fresh flesh, which have been used in preparing the extract. In the case of fluid extract the word "fluid" shall also appear on the label, as, for example, "fluid extract of beef." The word "fluid" merely indicates a lower percentage of solid matter.

(*h*) Such terms as "country," "farm," and the like, shall not be used on labels in connection with meat and products unless such meat and products are actually prepared in the country or on the farm. However, if the articles are prepared in the same way as in the country or on the farm, these terms, if qualified by the word "style" in the same size and style of lettering, may be used. Sausage containing cereal shall not be labeled "country style," and lard not rendered in an open kettle shall not be designated as "country style."

(*i*) The word "leaf" shall not be used in connection with lard prepared from fat other than leaf fat.

SECTION 8. A meat food product when composed of more than one ingredient shall not bear a label with a name stating or indicating that the product is a substance which is not the principal ingredient contained therein, even though such name be an established trade name. The term "principal ingredient," as used in this section, shall be construed to mean that such ingredient is equal to or exceeds in amount the other ingredients combined, exclusive of cereal and water. If the ingredients are stated on the label, they shall appear in the order of their percentages. For example, sausage containing pork and beef shall not be labeled "pork sausage," but shall be labeled "pork and beef sausage." However, if the pork ingredient equals or exceeds 50 per cent of the meat content, the sausage may be labeled "pork sausage, beef added." A product consisting of veal, pork, and beef shall not be labeled "veal loaf," but may be designated as "veal, pork, and beef loaf." However, if the veal ingredient is not less than 50 per cent of the meat content of the product, the product may be labeled "veal loaf, pork and beef added," the words "pork" and "beef" to appear in the order of their percentages, as above indicated.

SECTION 9. *Paragraph 1.* When a meat food product contains an added substance or substances, the label shall show the added substance or substances except as provided in the succeeding paragraphs of this section.

Paragraph 2. When cereal is added to sausage within the limit prescribed by paragraph 4 of section 6 of regulation 18, there shall appear on the label in a prominent manner, contiguous to the name of the product, the statement "cereal added." When water in excess of 3 per cent and cereal are added to certain kinds of sausage as permitted by paragraph 5 of section 6 of regulation 18, the same shall be labeled "sausage, water, and cereal;" but when no cereal is added, the addition of water need not be stated.

Paragraph 3. When cereal is added to any meat food product other than sausage in quantities not exceeding 5 per cent, the statement "cereal added" shall appear on the label in a conspicuous manner contiguous to the name of the product, and if any such product contains cereal in quantities exceeding 5 per cent, then "cereal" shall appear as a part of the name of the product in uniform size and style of letters, for example, "potted meat and cereal:" *Provided, however,* That, products such as meat

loaves, pâtés, soups, tripe with onion sauce, Irish stew, stewed kidneys, hash, chile con carne, tamales, boiled dinners, chop suey, scrapple, and the like, may contain cereal and similar substances without the presence of such substances being indicated on the labels.

Paragraph 4. When edible parts of the head or viscera, or other similar edible parts, are added to any meat or product bearing a specific name, such as "meat," "beef," "pork," "veal," and the like, there shall appear on the label, in a prominent manner and contiguous to the name of the product, the statement "meat products added," provided such parts are not in excess of 20 per cent. If this percentage is exceeded, the words "and meat products" must appear as a part of the name of the product and in the same size and style of lettering. The percentage of such parts added to any meat or product shall be based on the weight of the meat ingredient of the product exclusive of added substances. When a potted, deviled, or similar article of food is prepared exclusively from the above mentioned parts, the product shall be labeled "potted meat products," "deviled meat products," and the like.

Paragraph 5. Lard may have added thereto not to exceed 10 per cent of lard stearin without the presence of added stearin being shown on the label. When more than 10 per cent of lard stearin is added to lard, there shall appear on the label, contiguous to and in the same size and style of lettering as the name of the product, the statement "lard stearin added."

Paragraph 6. When not over 20 per cent of oleo stearin, beef fat, or mutton fat are added to lard, there shall appear on the label, contiguous to and in the same size and style of lettering as the name of the product, the statement "oleo stearin added," "beef fat added," or "mutton fat added," respectively, as the case may be.

Paragraph 7. Mixtures, of which the lard ingredient equals or exceeds in amount the other ingredients combined, may be labeled "lard compound," provided all the ingredients in the mixture are stated on the label in a prominent manner in the order of their percentages and preceded by the statement "composed of," or "made from," or an equivalent statement.

Paragraph 8. Labels for mixtures, other than oleomargarin, consisting of fat derived from carcasses of cattle, sheep, swine, or goats and any vegetable oil, shall bear the names of the ingredients in a prominent manner, in the order of their percentages, preceded by the statement "composed of," or "made from," or an equivalent statement. Tierces and barrels containing "compound," or "lard substitutes," or "lard compound," shall, immediately after filling, be legibly marked on one end, and on the side near the end, with the true name of the product. Tin pails, drums, tubs, and similar containers of such products shall bear the true name of the product also on the side at the time of filling.

Paragraph 9. Any meat or product containing any benzoate of soda shall be plainly labeled so as to show the presence and the percentage amount of such benzoate of soda.

Paragraph 10. When permitted coloring matter is used in the preparation of lard or other prepared animal fats under the provisions of paragraph 3 of section 6 of regulation 18, there shall appear on the label, in a prominent manner and contiguous to the name of the product, the statement "artificially colored."

SECTION 10. *Paragraph 1.* When the weight of any meat or product, prepared at an official establishment, or imported, prior to September 3, 1914, appears upon a label or container, it shall be the correct weight, and the words "net," "gross," "not less than," or a similar statement shall appear in direct connection therewith.

Paragraph 2. All meat and products in package form, prepared at official establishments, or imported, on or after September 3 1914, shall have the quantity of the contents thereof plainly and conspicuously marked on the outside of the package in terms of weight, measure, or numerical count: *Provided,* That such reasonable variations and tolerances and also exemptions as to small packages shall be permitted as shall be established by rules and regulations made pursuant to the food and drugs act.

SECTION 11. *Paragraph 1.* No marks of Federal inspection which have been previously used shall be again used for the identification of any meat or product except as provided in paragraph 2 of this section.

Paragraph 2. All stencils, marks, labels, or other devices, whether relating to any meat or product or otherwise, on previously used containers, shall be removed or

obliterated before such containers are used for any meat or product, unless such stencils, marks, labels, or devices correctly indicate the article to be packed therein and such containers are refilled under the supervision of bureau employees.

SECTION 12. *Paragraph 1.* All labeling of meat and products required to be inspected by bureau employees shall be in compliance with these regulations.

Paragraph 2. No person shall apply or affix, or cause to be applied or affixed, any label to any article prepared or received in an official establishment or to any container thereof except in compliance with these regulations.

Paragraph 3. No person shall, in an official establishment, fill, or cause to be filled, in whole or in part, any container with any article required by these regulations to bear a label, except in compliance with these regulations.

Paragraph 4. No person shall remove or cause to be removed from an official establishment any meat or product bearing a label unless such label be in compliance with these regulations.

LIST OF AUTHORS

Valenti, 500
Valentin, 358
Vallée, 161, 228, 249, 253, 302, 310, 454, 456, 457, 458, 459, 460, 483
Vallet, 358, 362
Van de Velde, 32
Van Ecke, 60
Van Es, 146, 191, 208, 454, 456, 460
Van Gehuchten, 397, 398, 403
Van Recklinghausen, 79
Varnell, 285, 288
Vaughan, 507, 514
Vaughn, 8, 508, 515
Venuta, 467
Viborg, 37, 242, 386
Vicchi, 37
Villejean, 161
Villemin, 147
Vincenheller, 342
Virchow, 110, 147
Visgocqi, 37
Voges, 356, 359, 379, 380, 381, 383, 384, 397
Waldeyer, 299
Waldinger, 386
Walker, 411
Wall, 208
Walley, 408, 411, 414, 420, 441
Walraff, 242
Wandelleck, 328
Ward, 23, 27, 29, 40, 41, 69, 70, 71, 72, 74, 82, 182, 184, 189, 190, 405, 410, 411, 484, 485, 494, 495, 498
Warming, 302
Washburn, 42, 43, 44, 45, 46, 233, 472
Wasilewsky, 359, 363
Wasserman, 239, 241, 401, 508, 509, 515
Watson, 363, 372
Way, 144, 146, 403
Webb, 346
Weber, 141, 145, 190, 353, 369
Wedemann, 199, 200, 208

Weichel, 23
Weichselbaum, 111
Weidlich, 432
Weis, 288, 300
Welch, 29, 33, 58, 418, 421, 432
Wenyon, 302
Wernich, 507
Westbrook, 390
Westphal, 238
Wetzl, 346
Wharton, 406
Wherry, 146
White, 411
Wilhelms, 419, 420
Williams, 21, 23, 29, 146, 198, 200, 202, 203, 205, 208, 349, 362, 366, 370, 372, 387, 390, 398, 403, 453, 461, 463, 466, 508, 515
Williamson, 191
Wills, 189, 441
Wilson, 60, 61, 62, 66, 68, 208
Wilson-Barker, 372
Wiltshire, 346
Winslow, 31, 33
Wolbach, 432
Wolff-Eisner, 170
Wolffhügel, 500
Wohner, 253
Wood, 405, 410
Woodhead, 198, 208, 411
Woolley, 68, 325
Wright, 146, 254, 256, 270, 346
Wurtz, 190
Xylander, 431
Youatt, 474, 482
Young, 522
Zell, 401, 403
Zeller, 208
Zinsser, 515
Zschokke, 26, 40, 41, 180, 228, 436, 437, 439, 454, 500
Zurn, 110, 281, 325
Zwick, 199, 200, 208

INDEX