TREASURY DEPARTMENT.
Public Health and Marine-Hospital Service of the United States.
WALTER WYMAN, Surgeon-General.

HYGIENIC LABORATORY.—BULLETIN No. 20.
M. J. ROSENAU, Director.
APRIL, 1905.

A ZOOLOGICAL INVESTIGATION

INTO

THE CAUSE, TRANSMISSION, AND SOURCE

OF

ROCKY MOUNTAIN "SPOTTED FEVER."

BY

CH. WARDELL STILES.

WASHINGTON:
GOVERNMENT PRINTING OFFICE.
1905.

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No. 15.—Inefficiency of ferrous sulphate as an antiseptic and germicide. By Allan J. McLaughlin.

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A ZOOLOGICAL INVESTIGATION INTO THE CAUSE, TRANSMISSION, AND SOURCE OF ROCKY MOUNTAIN "SPOTTED FEVER."

By Ch. Wardell Stiles, Ph. D.

Chief of Division of Zoology, Hygienic Laboratory, United States Public Health and Marine-Hospital Service.

Summary.

This paper contains the results of a zoological investigation into the cause, transmission, and origin of the so-called "spotted fever" of the Rocky Mountains and consists chiefly of negative findings.

The disease was first described by Wood (1896), then by Maxey (1899) for Idaho, and by McCullough (1902), Gwinn (1902), Wilson and Chowning (1902, 1903, 1904), Cobb (1902), Anderson (1903), and Gates (1903) for Montana.

It has been known since 1872 in the Bitter Root Valley, but its early history elsewhere seems not to be established. It is known under the various names of "spotted fever," "tick fever," "black fever," "blue disease," "black measles." and "piroplasmosis hominis."

Several suggestions have been made as to the cause of this disease, but the only definite proposition is that advanced by Wilson and Chowning (1902, 1903, 1904), and supported in part, at least, by Wesbrook (*a*), Cobb (*a*), and Anderson (*a*, *b*). According to this theory: (*a*) "Spotted fever" is caused (p. 17) by a protozoon to which the name *Piroplasma hominis* has been given and which infects the red blood corpuscles; (*b*) this parasite is transmitted (p. 20) by ticks (*Dermacentor andersoni*); (*c*) the burrowing squirrel (*Citellus columbianus*) harbors a *Piroplasma* which can not be distinguished from *P. hominis*, and this rodent (p. 24) may perhaps be the original host for the disease.

(*a*) My efforts to find a *Piroplasma* in the fresh blood of 9 cases of the disease in man, and in the stained blood of these and several additional cases, have been negative (p. 19), despite an actual microscopic study of more than 200 hours; Ashburn has had similar negative results; Chowning was unable to demonstrate the parasite to us in the fresh and the stained blood of a typical case; and several investigators who have since examined slides, both from Idaho and Montana cases, have not been able to find the *Piroplasma*. Accordingly, the work of 1904 has failed to confirm the conclusions of 1902 and 1903, and indications are not lacking that at least some of the stages of the supposed *Piroplasma hominis* consist in reality of vacuoles, blood platelets, blood dust, artifacts, and tertian malaria parasites. Wilson and Chowning report that they were able to transmit the disease to rabbits, but I was unable to do this (p. 19).

(*b*) I have likewise been unable (p. 20) to confirm the hypothesis that this disease is transmitted by ticks, for while there is a certain amount of circumstantial evidence against this arachnoid, one of the fundamental premises of the tick theory (namely, the piroplasmatic nature of the disease) is called into question; and, further, cases are known in which a history of tick bite has not been established (p. 22).

(*c*) I am unable to see any valid arguments (p. 25) in support of the view that the burrowing squirrel (*Citellus columbianus*) forms the original host for the disease, while I find several arguments against this hypothesis.

Having failed to confirm the piroplasmatic nature of Rocky Mountain "spotted fever" by direct evidence, I attempt to do so by indirect evidence, namely, by comparing the symptoms reported for this disease with those reported for known piroplasmatic maladies; but in this also my work is negative, for while a thin, watery, anemic condition of the blood, a thick condition of the bile, marked emaciation, prominence of hemoglobinuria, practical absence of skin lesions, and the occurrence of the cases in groups (corresponding to the extreme fertility of ticks) are the striking features described for piroplasmosis in animals, "spotted fever" patients present a thickened blood, fluid bile, no marked emaciation (at least so far as reported), little or no hemoglobinuria (Wilson and Chowning), prominent skin lesions, and the occurrence of isolated cases, widely separated. Accordingly, if this disease is a piroplasmosis, *Piroplasma* has an effect in man markedly different from the effect it has in cattle, sheep, dogs, and horses.

Rocky Mountain "spotted fever" is reported for Idaho, Montana, Nevada, Oregon, Wyoming, ?Washington State, and possibly Utah and Alaska (p. 25). About 200 cases are said to have occurred in the Bitter Root Valley since 1872 (p. 29); of these, 139 have been collated, showing a lethality of 70.5 per cent. It is rather striking that the cases seem to be more or less confined to valleys (p. 30), and infection seems to take place chiefly in the foothills. In the Bitter Root Valley the cases occur chiefly on the west side of the river, and are confined to the months of (January) March to September, inclusive; April, May, and June are the worst months. It is rather striking that more cases seem to develop under moist conditions (p. 33), as on the west side of the river, and during or following a rise in the streams caused by rain or melting snow, than under dry conditions; but the significance of this popular view is not altogether clear. Both sexes and all ages (p. 37) are subject to the disease, but it is more common in males from 21 to 40, and in females from 11 to 40 years of age, than at other times of life; the lethality varies, being in the Bitter Root Valley 45.4 per cent for females from 11 to 20 years of age up to 100 per cent for all patients over 60 years old. So far as one can judge, occupation (p. 38) seems to play a rôle, for a very large percentage of the patients are on farms or are connected with the lumbering industry.

In Idaho, a mild type (p. 39) of the disease exists, with a lethality of about 1 to 3 per cent. In Montana, physicians speak of a mild type (without spots), medium cases, and severe, very fatal cases; some men also speak of cases of "localized" spotted fever (p. 39).

About 90 per cent of the cases give a history of exposure to wet or cold (p. 39). The period of incubation (p. 40) is variously given as 2 to 21 days. The attack may be preceded by a few days of malaise, or the onset (p. 41) may be marked by sudden chill, followed by fever, with or without nausea; the disease usually lasts about 10 to 21 days (p. 42); usually only one case occurs in a family (p. 43), but instances are known in which two members of a family are attacked the same day, or within a few days of each other; it is almost universally admitted (p. 44) that the disease is not contagious, but cases are known where contagion does not seem to be entirely excluded.

The patients may assume a position of general flexion in bed (p. 45), and may have a peculiar urinous odor (p. 45) about them.

The most characteristic and constant symptom is the eruption (p. 45), which usually appears first on the wrists, ankles, and back, about second to fifth, chiefly third day, and spreads rapidly over the rest of the body, lasting about 8 to 21 days, or even several months, and after the fever subsides, becoming visible upon exposure to cold or after a warm bath or active exercise; these spots are petechial and not raised; at first they are rose colored, and disappear momentarily upon pressure, but later they become permanent and assume a dark blue or purplish color; they may coalesce and give a mottled or marbled appearance to the skin; they may or may not be tender to the touch. Desquamation (p. 49) begins about the third week. Jaundice (p. 50) is more or less marked, first noticed in the conjunctivæ. There may be cyanosis (p. 50), or—especially on the scrotum, fingers, or toes—gangrene (p. 50). Hyperesthesia (p. 51) is common, and may be intense. The subcutaneous fat (p. 51) remains.

The face (p. 51) frequently shows a congested, bloated, stupid expression. There may be ringing in the ears (p. 51). The eyes (p. 52) are more or less injected. Photophobia (p. 52) is common, and may be very marked. Nosebleed (p. 52) is more or less frequent. Sore throat (p. 53) is more or less common; breath may be offensive. The tongue at first shows a heavy white or yellowish coat, with red tip and edge, and becomes brownish, dry, and cracked as the fever progresses; the teeth may be covered with sordes.

There may be gurgling and tenderness (p. 55) in the right iliac fossa; tympanites may develop. The joints may become swollen.

Loss of appetite (p. 55) is an early symptom, or in some cases the appetite remains good.

Irritability of and pains in the stomach (p. 56) are reported. Nausea (p. 56) is more or less common, and vomiting may be present. Constipation (p. 58) is very common. The liver (p. 58) may be enlarged to some extent. The gall (p. 60) is fluid. Pancreas may be normal in size or enlarged.

Heart sounds (p. 62) are reported as normal. Pulse (p. 63) is usually full and strong at the onset, but gradually becoming more and more rapid, losing in strength and volume; in ordinary cases it may be 80 to 130, and has been reported as high as 150, or even 186. The blood (p. 63) becomes dark and thick; it shows some decrease in the red cells (to 4,100,000 or to 3,558,000) and may show some increase (12,000 to 15,600) in the white cells, the most interesting feature, according to Anderson, being an increase in the large mononuclears; hemoglobin may fall as low as 50 per cent.

The spleen (p. 67) is uniformly enlarged and tender.

The initial chill (p. 68) may be absent, slight, or severe; and chills or chilly sensations may continue more or less throughout the attack.

The fever develops rapidly, and may register 102° to 104° or 105° F. when the patient takes to bed. It gradually reaches its maximum in 2 to 7 days, when it ordinarily registers 103° to 106°. For temperature charts, see Wilson and Chowning, or Anderson, or page 111.

An irritative cough (p. 71) may exist from the first. Respiration (p. 72) is increased, usually to 26 to 40—in some cases 50 to 60. Edema of lungs develops in a number of cases.

There may be great tenderness or soreness of the muscles.

Some cases are exceedingly nervous, symptoms (p. 74) being so prominent as to remind one of cerebrospinal meningitis. Malaise, restlessness, insomnia, hyperesthesia, jactitation, dizziness, headache, pains, "bone ache," tenderness of neck and lumbar region, photophobia, divergent and convergent squint, delirium, coma, convulsions, and in a few cases opisthotonos have been reported. Autopsy fails to reveal any lesions which would justify the diagnosis of cerebrospinal meningitis. Kernig's sign is absent.

The kidneys are often disturbed; they may be enlarged. Bladder (p. 83) normal or nearly so. The urine is reported as reduced in amount, slightly above normal in color to highly colored; specific gravity 1,018 to 1,030; reaction acid, so far as reported; albumen present or absent; sugar and bile not reported; granular, blood, hyaline, and epithelial casts are reported; hematuria and hemoglobinuria absent or slight.

Menstruation is delayed by attack, and abortion is reported for pregnant women.

Several authors refer to relapses, following muscular exertion or exposure to cold.

Hypostatic pneumonia, rheumatism, and gangrene are among the most frequent complications (p. 87) mentioned; pneumonia predominates in frequency.

Convalescence (p. 87) may be rapid or very slow, lasting ten to twelve weeks or even longer.

Prognosis (p. 88) seems to be favorable (lethality about 1 to 3 per cent) in some places (as in Idaho); but it is very unfavorable in others (as in Bitter Root Valley), where cases in which the eruption is marked show a lethality of about 70 per cent. See also under "sex and age," page 37, and "number of cases," page 29.

Death (p. 89) occurs from third to twenty-ninth day, usually from sixth to twelfth day.

Local physicians agree that diagnosis (p. 89) is not difficult, and even the laity recognize the disease on sight; its peculiar geographic and seasonal distribution, endemic character, severe aching pains in the muscles, joints, bones, neck, and head, appearance of nonelevated spots on the second to seventh day, at first rose-colored and on wrists, ankles, and back, disappearing momentarily on pressure, rapidly spreading to entire body, and becoming darker, and then not disappearing on pressure, the frequency of constipation, the coated tongue, accelerated pulse, temperature, icterus, and expression denoting profound intoxication of entire system, lead the local physicians to the diagnosis of "spotted fever."

It is generally admitted that the disease resembles typhus (p. 90) more than it does any other malady; some cases resemble cerebrospinal meningitis (p. 91); compare also typhoid, dengue, peliosis rheumatica, etc., (p. 92).

Satisfactory specific treatment (p. 92) is unknown.

Clinical histories (p. 100) of cases, and a bibliography (p. 116) of the subject are added.

I have no new theories to present regarding the cause, transmission, and origin of this disease.

During the investigations I incidentally found several new species of parasites which I hope to describe soon, and three of which I propose to dedicate to Doctors Anderson, Ashburn, and Buckley.

Date of manuscript, January 14, 1905.

Introduction.

Pursuant to orders from the Surgeon-General, dated May 2, 1904, I visited the Bitter Root Valley to study the so-called "spotted fever" ("tick fever," "piroplasmosis hominis") from a zoological point of view, and remained there from May 7 to July 6, 1904.

The special object of my detail was to trace the life cycle of the parasite (*Piroplasma hominis*) which had been described as the cause of the disease, to study the tick which was supposed to transmit it, and to trace the disease in the burrowing squirrels, in which it was thought to originate. The points at issue, it will be seen, bore more directly upon discovering some method of prevention than upon a study of the symptomatology.

As the seasonal duration of the outbreak is short, and as the parasite

was classified in one of the most difficult groups, from a standpoint of interpretation, I had upon starting little hope that much could be accomplished in one season; in fact, I viewed the trip as the beginning of an investigation which would probably occupy the spring months for a number of years to come.

From the literature which had appeared upon this malady, especially from the last article by Wilson and Chowning (1904a), I had become somewhat prejudiced in favor of the protozoan theory relative to the origin of this disease, and also in favor of the theory that it was transmitted to man by ticks belonging to the genus *Dermacentor;* regarding the hypothesis of its origin in the burrowing squirrel, however, I must confess that a priori reasons made me very skeptical.

During my stay in the Bitter Root Valley I was able to see 10 patients who I was assured by local physicians presented typical cases of the disease. In 9 of these cases I was able to search in the fresh blood for the parasite. Part of my time was occupied in studying the ticks, the burrowing squirrels, the topography of the region, and other points to be considered in connection with the malady.

Acknowledgments.

I am under numerous obligations to various persons, especially the local physicians in the valley, for many courtesies extended to me during my investigations. Among these I would mention the following in particular:

To Dr. John Jay Buckley, who for years past has spent much time and money in studying "spotted fever," I am under special obligations for the courtesies of his private laboratory and library, as well as for giving me so much time in showing me through the valley, taking me to see patients, and for other courtesies too numerous to mention.

To Dr. Percy M. Ashburn, captain and assistant surgeon, U. S. Army, stationed at Fort Missoula, I am under many obligations for use of his laboratory and his instruments, for accompanying me on various trips through the valley, and for his association in studying the cases. Doctor Ashburn had commenced to study "spotted fever," under instructions from Surgeon-General Robert M. O'Reilly, U. S. Army, before I reached Missoula. After my arrival we worked together, yet independently, constantly checking and criticising each other's work. Our final conclusions were practically identical in all important respects.

To Maj. Z. W. Torrey, in command of Fort Missoula, I am indebted for many official courtesies, including the use of post transportation facilities upon several hard trips through the mountains.

To Dr. Edward W. Spottswood, chief surgeon of the Northern Pacific Railroad Hospital at Missoula, I am indebted for the use of his laboratory and laboratory supplies, as well as for valuable advice.

To President Oscar J. Craig and Prof. M. J. Elrod, of the State University, I am indebted for the use of the university laboratories and supplies. Professor Elrod also kindly took photographs of patients at my request and accompanied me on collecting trips.

To Dr. William Park Mills, Dr. Charles H. Pixley, Dr. Russell Gwinn, Dr. Samuel W. Minshall, Dr. George Hampton Putney, Dr. William B. Parsons, Dr. John T. Brown, Dr. Thomas A. Fitzgerald, Dr. George T. McCullough, Dr. Watkins, Dr.

William A. Glasgow, and Dr. Joseph A. Tremblay (all of Missoula), to Dr. Brice and Dr. E. A. Brooke (of Stevensville), to Dr. Thomas H. Hanbidge (of Victor), and to Dr. J. W. Howard and Dr. George McGrath (of Hamilton) I am indebted for the privilege of seeing actual and suspected cases of "spotted fever," for information, advice, and various other courtesies.

To Sister Superior Gasper, Sister Gabriel, Sister Ignatius, Sister Wilfred, and the other sisters of St. Patrick's Hospital I am indebted for the many courtesies they extended to me at the hospital.

To Doctors Louis B. Wilson and William B. Chowning, of Minneapolis, I am indebted for one of their original slides and for various points of information. I also had the pleasure of personal association with Doctor Chowning for two weeks in Missoula, where we examined a case together.

To Dr. L. A. Gates, of Bridger, Mont., I am indebted for an interesting account of 4 cases of "spotted fever" which he treated, and a table of all 17 cases which have come to his notice.

Last, but not least, I am indebted to Dr. Thomas D. Tuttle, secretary Montana State board of health, for his cordial cooperation and advice.

The fact that I did not accomplish more than I have to report lies in the difficulties of the subject and the shortness of the season, and not in any lack of facilities which it was in the power of the local authorities and profession to offer me.

Historical Review of Literature.

For a proper understanding of the subject of this report it will be well to give extensive historical reviews of various phases of the matter at hand, more especially as no one has as yet collected all of the observations reported.

The first printed account of this "spotted fever" which I have been able to trace is a summary given by Lieut. Col. W. M. Wood (1896, pp. 60–65), Deputy Surgeon-General (retired), U. S. Army, at Boise Barracks, Idaho. He himself had seen no cases, but he collected statements regarding the disease in Idaho from Drs. L. C. Bowers, George Collister, J. K. Dubois, R. M. Fairchild, D. W. Figgins, W. D. Springer, C. L. Sweet, and H. Zipf. The discussions are mainly of symptomatology and will be considered in detail in that part of the present report.

Maxey (1899, pp. 433–438) presented before the Oregon State Medical Society an excellent paper on the disease as found in Idaho. He discussed its seasonal and topographic distribution, symptoms, specific and differential diagnosis, prognosis, and treatment. He is of the opinion that "spotted fever" of Idaho is "an independent, specific disease, and related in no way to any disease described in our text-books on practice."

Commenting editorially upon Maxey's paper, the Medical Sentinel (1899, pp. 456–458), of Portland, Oreg., expressed the view, based solely upon the symptomatology, that "this spotted fever bears a closer resemblance to the papular form of erythema exudativum multiforme than to any other known morbid entity."

McCullough (1902, July, pp. 225–228) presented a paper on "spotted fever" before the Montana State Medical Society at Anaconda, Mont., May 21, 1902. He discussed the distribution of the malady, seasonal occurrence, symptoms, complications, prognosis, treatment, and its name.

In the same number of the same medical journal in which McCullough's paper was printed, there appears a summary of results obtained by Wilson and Chowning (1902c, pp. 238–239), who claim to have found a parasite in the red-blood corpuscles which resembles the parasite of malaria. They think that evidence points to some kind of a tick as its transmitter.

Gwinn (1902) presented a paper on "spotted fever" before the Montana State Medical Society, at Anaconda, May, 1902. He discussed the various possible modes of infection, gave very brief observations on two autopsies, an excellent account of symptoms, diagnosis, prognosis, and treatment. This paper was published in the Missoulian, a daily paper issued in Missoula, but I have been unable to find it in any medical journal.

Wilson and Chowning (1902a, 1902b) investigated this disease for the Montana State board of health. Doctor Wilson reached Missoula on May 16 and Doctor Chowning on May 26. On July 1 they dated a preliminary report which was printed on July 19. Wilson and Chowning reached conclusions which, if correct, are of far-reaching importance. They give a summary of the results obtained upon 6 autopsies. The most important points in the paper are those dealing with the etiology, transmission, and origin of the disease. Briefly stated, these points are as follows: No bacteria of etiologic significance were obtained in any case, but in stained coverslip preparations of (1902) cases 2 to 6 they found certain ovoidal bodies within the red-blood cells; the nature of these bodies was not clear until they examined the fresh blood of case 7: "Intracellular parasites, showing ameboid movements were found. In several of these observations extracellular forms were also found. * * * Probably not more than 1 red cell in 500 in the circulating blood is ordinarily infected." They describe 3 phases of the parasite, which they claim to have transmitted experimentally to rabbits.

The final point they make is that the common gray spermophile (*Citellus columbianus*) possibly forms the regular host for the parasite they found in man.

The views regarding the tick transmission and the origin of the disease in the spermophile are advanced as "hypotheses."

Cobb (1902, pp. 1868–1870) visited the Bitter Root Valley in June, 1902, where he found Wilson and Chowning already at work. His report contains a brief account of the Wilson and Chowning observations. Cobb was in the valley too short a time to thoroughly confirm or refute the Wilson and Chowning hypotheses, but from what he saw it is clear that he was favorably impressed by their views.

On December 31, 1902, Wilson and Chowning (1903a) finished their more complete report to the Montana State board of health.

The paper contains a discussion of the history of the disease, a list of 114 cases they compiled (chiefly from correspondence with local physicians), location, season, table of distribution of cases by months, previous condition of patient, sex and age of patient, types of the disease, clinical histories and autopsy notes of the cases they observed in 1902, a case of "local infection," an excellent general summary of symptoms, morbid anatomy and histology, etiology, "hæmatozoa of spotted fever," inoculation experiments, mode of infection, the gopher as possibly the normal host of the hematozoon, and suggestions relative to future investigations. It is important to note here that at the conclusion of their article they say:

"While the tick-gopher hypothesis is a very alluring one, it must not be forgotten that as yet but few positive facts have been obtained for its establishment. Aside from the collection of clinical evidence attempts must be made to transfer hæmatozoa from infected gophers by direct blood inoculations and through tick bites to various uninfected animals, as the rabbit. A comparison of the results of such inoculations, if successful, with similar inoculations from human cases (see No. 3) should give valuable data as to the identity or dissimilarity of the protozoa from gophers and those from man. In view of the high mortality of 'spotted fever,' it will probably be impossible to get any man to submit to a direct blood inoculation, or tick bite, from patients as has been done recently with yellow fever. But it seems probable that all that is necessary may be done by careful inoculation along the lines here indicated."

During the season of 1903, at the request of the Montana State board of health,

Doctor Anderson investigated the disease on behalf of the Public Health and Marine-Hospital Service, and Doctors Wilson and Chowning continued their studies on behalf of the Montana State board of health.

Anderson (1903c) introduced into medical literature the name "tick fever" for this disease, accepting the tick as the "very probable and almost proved" method of transmission. He reprinted the Wilson and Chowning map of distribution in Montana and added a general map of the States in which the disease is reported. He discussed the geographic distribution, climate, season, occupation of patients, their age and sex, the parasite, and reprinted the list of cases published by Wilson and Chowning, bringing it down to 1903; he discussed method of infection and the symptomatology, gave clinical histories of some of the 1903 cases, with clinical charts and blood counts, autopsy notes, morbid anatomy, prognosis, diagnosis, and treatment. He did not mention the spermophile theory advanced by Wilson and Chowning (1902, 1903), but accepted certain phases of the parasite of man described by Wilson and Chowning as "very probably the cause of spotted (tick) fever."

Gates (1903) gave clinical reports of two cases.

In their third paper, Wilson and Chowning (1904a) cover much the same ground discussed in their second publication (1903a), adding some new observations and omitting the detailed clinical reports. They apparently definitely accept the parasite, which they now name *Piroplasma hominis*, as the cause of the disease, but they still speak of the idea of tick transmission as an "hypothesis."

Since the appearance of this paper, a number of text-books and medical journals have referred to their work and have accepted it, at least to some extent. Manson (1903, pp. 174–176), however, points out that the experimental proof of the Wilson and Chowning hypotheses is lacking, and Nuttall (1904, p. 221) remarks that in "spotted fever" the symptoms differ markedly from those observed in the bovine, ovine, equine, and canine [piroplasmatic] maladies, but he admits (p. 252) that the 1904 paper by Wilson and Chowning is much more convincing than their former articles, so far as the parasite is concerned. Manson (1903) and Nuttall (1904) correct the name of the parasite to *Piroplasma hominis*.

In August, Stiles (1904, pp. 1649–1650; 1904, pp. 362–363) published a preliminary report upon investigations conducted during the spring of 1904.

October 31, 1904, Ashburn delivered an address upon "spotted fever" before the Cincinnati Academy of Medicine. This has not yet been published, but it gave rise to a rather vigorous editorial by Heidingsfeld (1904, pp. 492–493) who attacked the Wilson and Chowning hypotheses.

Craig (1904, pp. 1016–1017) had no opportunity to examine slides of "spotted fever" blood, but he came to the conclusion that the objects described as *Piroplasma hominis* "were not due to the presence of a parasite, but to certain changes, especially in the hemoglobin of the red cells, produced by the disease." He claims to have observed appearances, coinciding in every particular with the description given by Wilson and Chowning, and Anderson of *P. hominis*, in a large number of diseases, especially in fevers, such as typhoid, malaria, smallpox, measles, grippe, and frequently in pneumonia and tuberculosis.

In the Middleton-Goldsmith lecture (Nov. 30, 1904), before the New York Pathological Society, Stiles (1905, pp. 9–21) discussed his results more in detail than given in his preliminary report.

Definition.

Idaho.—Dubois (1896, p. 64) characterizes "spotted fever" as an "acute, febrile, eruptive disease, noncontagious but epidemic, found chiefly in March and April."

Fairchild (1896) speaks of it as a "fever of typhoid type, self-limited, and characterized by a red eruption over whole body."

Springer (1896, p. 61) refers to it as a "continued fever, with typhoid condition and a red eruption general over entire body."

Sweet (1896, p. 61) defines it as a "continued fever, with mild exacerbations; temperature usually not excessive. Cases sometimes pass into an adynamic or typhoid condition."

Maxey (1899, pp. 432–433) defines spotted fever as "an acute, endemic, noncontagious, but probably infectious, febrile disease, characterized clinically by a continuous moderately high fever, severe arthritic and muscular pains, and a profuse petechial or purpural eruption in the skin, appearing first on the ankles, wrists, and forehead, but rapidly spreading to all parts of the body."

Montana.—Gwinn (1902) defines this disease as "an acute, febrile, noncontagious affection, characterized by an eruption of macules which are at first pink, afterwards gradually assuming a purplish or dark blue color."

Historical Review of the Disease.

Idaho.—"Spotted fever" has been known in Idaho for about thirty years (Wilson and Chowning, 1902a, p. 132; Anderson, 1903c, p. 8).

Montana.—According to McCullough (1902, p. 225), "spotted fever" has prevailed in various degrees of intensity in Bitter Root Valley from the earliest history of its settlement by white inhabitants; he states that prior to that time there is convincing evidence that the Indians were also subject to this ailment. Gwinn (1902) states that according to his personal knowledge it has been prevalent in the Bitter Root Valley since 1886, and he is credibly informed that it has existed there since the region was first settled by white men; he adds that the number of cases has of course increased with the number of inhabitants.

Wilson and Chowning (1902a, p. 132; 1903a, p. 28; 1904a, p. 33) report that so far as can be determined the first case of "spotted fever," "black fever," or "blue disease" in the Bitter Root Valley occurred in 1873 (this case—J. W.—occurred in May, near Woodside). At this time there were but few white men in the valley. No authentic information of its occurrence among the Indians, who until 1890 inhabited the valley, has been obtained, though many old residents, including Indians, white trappers, traders, and Catholic priests, were consulted (1903a, p. 28; 1904a, p. 33). It has been recognized as a clinical entity by the local physicians for fifteen or twenty years (1902a, p. 131; 1903a, p. 28; 1904a, p. 31). Anderson (1903a, p. 506; 1903c, p. 8) states that it has been known in the Bitter Root Valley for about twenty years.

In addition to the case in 1873 reported from Woodside by Wilson and Chowning (1903a, p. 28), I might add that Doctor Parsons has kindly called my attention to a second case which occurred at another place in the valley in the same year.

Doctor Buckley, who has spent considerable time and money in tracing back the history of the disease, assured me that he had been unable to obtain any convincing evidence that the Indians had suffered from "spotted fever."

That this disease has existed for some years in other localities than Idaho and the Bitter Root Valley does not seem to be entirely excluded, for the medical history of many parts of the Northwest is as yet unwritten, and there will naturally always remain some cases of disease among the early settlers which will probably be unrecorded and unexplained. To assume that "spotted fever" has made a sudden

spring within the last few years from the Bitter Root Valley to other parts of Montana (as Bridger and Livingston) and to Wyoming is probably not fully warranted in our present knowledge of this malady, for whether these localities actually represent newly infected foci or only newly recognized foci can be definitely determined only by a more complete medical history of these places than is published at present.

Names of the Disease.

Idaho.—In Idaho the disease under discussion is generally spoken of as "spotted fever," because of its eruption, and Maxey (1899, p. 434) states that he knows of no other or better name for it; he adds that, although confusing to physicians unacquainted with the affection, this name admirably describes the disease from a lay standpoint, and very probably will be retained in use by the local profession until future research enables us to classify it where it belongs. Dubois (1896, p. 64) says that *Exanthesis rosalia anthrodynia* more nearly expresses the disease than does "spotted fever." The Medical Sentinel (1899, p. 458) remarks editorially that "the opinion of the writer, based solely upon the symptomatology, is that this spotted fever bears a closer resemblance to the papular form of erythema exudativum multiforme than to any other known morbid entity."

Nevada.—In Nevada this disease is known as "spotted fever."

Montana.—According to McCullough (1902, p. 225) it has been called "black fever" and "blue disease," probably on account of the dusky or bluish appearance a short time before death of those afflicted. He considers the name "spotted fever" a good one, because to the laity it abodes grave responsibility: * * * "many of the cases terminating fatally with the best skill and untiring energy an intelligent physician can give them, we feel the burden is somewhat lightened, and the responsibility is shared by the family and friends if they are prepared for the inevitable." * * * Crain (see Wilson and Chowning, 1902a, p. 33) states that in 1891 the disease was called "black measles" by the valley physicians. Wilson and Chowning (1902a) refer to the malady as the "so-called 'spotted fever' of the Rocky Mountains," but remark (1903a, p. 27) that the name "spotted fever" as applied to it is an unfortunate one, since it has been applied to several other diseases.

Anderson (1903a) proposes the name "tick fever," on account of the relation of the tick to the spread of the disease, and because of the fact that there are already two diseases of man sometimes called "spotted fever." Wilson and Chowning (1904a, p. 31) state that the name "tick fever" [also frequently "wood-tick fever"] was proposed by the local newspapers when the hypothesis of transmission by ticks was advanced by them in 1902; that it seems fairly distinctive, but is open to the objection that it has been previously used as a synonym of "Texas fever" in cattle,[a] and that it does not accurately indicate either symptoms or etiology; since "spotted fever" appears to be the first described infection of man attributed to a "Pyroplasma" [namely Piroplasma], they classify the malady as "*Pyroplasmosis hominis*" [namely *Piroplasmosis hominis*]. Nuttall (1904, p. 221), in referring to the investigations by Wilson and Chowning, uses the term "human piroplasmosis."[b]

Historical Review of Investigations.

For a number of years this disease has been subjected to study by the local physicians in the regions in which it occurs. Maxey, Gwinn, McCullough, and Gates have published their ideas and it is to be

[a] Not to be confused with the African so-called "tick fever" of man, supposed to be transmitted by *Ornithodoros savignyi*.

[b] Not to be confused with "Dum Dum fever."

greatly regretted that other local observers, as Buckley, Mills, Spottswood, Brice, Howard, etc., have not published their many valuable observations.

Dr. Emil Starz, of Helena, State bacteriologist and chemist, Doctor Traphagen, of Bozeman, Doctor Strain, of Great Falls, and Doctor Longeway, then secretary of the Montana State board of health, visited the Bitter Root Valley in the spring of 1902. "After going over the ground, examining the character of the soil, water supply, etc., it was determined that none of these conditions was probably responsible for the disease, and that the investigations should be carried on by pathological and bacteriological examinations of existing cases." In accordance with this conclusion, the State board of health invited Drs. Louis B. Wilson and Wm. M. Chowning, of the Minnesota State University, to study the malady.

Wilson was in the Bitter Root Valley from May 16 to July 14, 1902, and April 22 to May 20, 1903. Chowning was in the same district from May 26 to July 14, 1902, and from May 19 to June 19, 1903. He also visited the valley again, on an unofficial trip in June, 1904, spending about two weeks in Missoula.

Dr. J. O. Cobb, of the U. S. Public Health and Marine-Hospital Service, and Prof. F. F. Wesbrook, of the Minnesota State University, spent the last week of June, 1902, in the Bitter Root Valley, studying the disease.

Dr. J. F. Anderson, of the United States Public Health and Marine-Hospital Service, was in the same locality from May 1 to 30, 1903, when he studied the same cases observed during the same time by Wilson & Chowning.

Dr. Percy M. Ashburn, captain and assistant surgeon, U. S. Army, stationed at Fort Missoula, had an opportunity to observe some features of the disease in 1903, but it was not until the season of 1904 that he undertook a more thorough investigation of the malady.

I arrived at Missoula May 7, 1904, and remained in the Bitter Root Valley until July 6, 1904, studying the cases which occurred during that time.

Specific Cause of Spotted Fever.

Various different theories have been advanced regarding the cause of "spotted fever." Among these the following may be mentioned:

Idaho.—The vera causa is probably of a teluric character (Bowers, 1896, p. 63). Dubois (1896, p. 64) frankly admitted that as yet no cause can be assigned. Figgins (1896, p. 64) states that he never saw any cases except among people who had used water from creeks and surface wells. He has seen it in families who used water from springs and where the entire family had the disease. Springer (1896, p. 62) gives the cause as probably "water; sepsis." Sweet (1896) says that while there are indications which seem to point out this particular affection as a water-borne disease, there are other circumstances which militate against this theory and are in favor of its being akin to malaria, in that it is frequently seen in persons who have been living in the vicinity of newly broken ground, post holes, plowed ground, and in those who have drunk from seepage water from worked soil, etc. Zipf (1896, p. 65) attributes it to malaria. Maxey (1899, p. 434) concludes that the disease "is, in all probability, caused by some peculiar organism, possibly a miasm, though no specific cause has yet been discovered."

Montana.—Gwinn (1902) says that all kinds of theories have been advanced, none of which have been based upon pathological investigations; and none of the theories

are to his mind convincing. McCullough (1902, p. 225) refers to the locality as having a "bearing upon this deplorable affliction with suitable environments, most cases giving a history of exposure to wet and cold, or violent physical exercise and overexertion."

Wilson and Chowning (1902a, 1903a, 1904a) were the first authors to describe a specific organism as cause of the disease. They call special attention to the fact that their bacteriologic cultures failed to show any bacteria of etiologic significance, and they describe bodies which they interpret as protozoa and which they later name "*Pyroplasma hominis*" [namely *Piroplasma hominis*]. The secretary of the Montana State board of health (see report, issued 1903, p. 25) says that the cause has been "attributed to various sources, particularly the water, mineral, and general conditions of the soil, and by some thought to be contained in the snow of the Bitter Root Mountains, as well as various and varied local conditions."

The only definite proposition as to a specific cause is that advanced by Wilson and Chowning (1902a, c, 1903a, 1904a), namely, that "spotted fever" is due to a protozoon parasite which lives in the red blood corpuscles; this protozoon they classify (1904a) as a member of the genus *Piroplasma*.

The genus *Piroplasma*, which is very possibly identical with *Babesia*, seems very clearly to be a protozoon, but its more exact systematic position is at present somewhat uncertain. It is one of the most difficult of all the protozoa to interpret, and little is known of its life cycle. Its synonymy is as follows:

Genus PIROPLASMA Patton, 1895.

1888. ? *Hæmatococcus* Babès [not Agardh, 1828].

1893. *Pyrosoma* Smith and Kilborne [not *Pyrosoma* Péron, 1804], type species, *P. bigeminum* of Texas fever.

1893. ? *Babesia* Starcovici, 1893, July 1, type *Babesia boris* (of bovine hemoglobinuria), whose identity with *P. bigeminum* is not fully established.

1895. *Apiosoma* Wandolleck [not *Apiosoma* Blanchard, 1885], equals *Pyrosoma* Smith and Kilborne, 1893, renamed, hence type species *P. bigeminum*.

1895. *Piroplasma* Patton, equals *Pyrosoma* Smith and Kilborne, 1893, renamed, hence type species *P. bigeminum*.

1904. *Pyroplasma* Wilson and Chowning, for *Piroplasma*.

In view of the comparatively slight knowledge of the present day relative to the structure and life cycle of the members of this genus, hence the uncertainty as to whether *Babesia boris* is really congeneric with *Piroplasma bigeminum*, I retain the name *Piroplasma* in this discussion. Should it be demonstrated that *P. bigeminum* and *B. boris* are congeneric it will of course be necessary to reject the name *Piroplasma* in favor of *Babesia*.

In the genus *Piroplasma* several species are at present known as cause of serious diseases. Thus:

Piroplasma bigeminum (Smith and Kilborne, 1893) is the cause of Texas fever of cattle.

Babesia boris, which may be generically identical with, but specifically distinct from, *P. bigeminum*, is accepted as the cause of hemoglobinuria in cattle.

Babesia oris or *Piroplasma oris* is viewed as the cause of carceag, also known as ovine piroplasmosis.

Piroplasma equi is viewed as the cause of equine piroplasmosis; and

Piroplasma canis is accepted as the cause of canine piroplasmosis.

All of these parasites are supposed (see p. 20) to be transmitted by ticks (*Ixodoidea*) and the symptomatology of the diseases is more or less uniform (see p. 39).

Piroplasma donovani Laveran has been described as a parasite of man, causing non-malarial splenomegaly (Dum Dum fever; Kala-azar) in warm climates; but very serious doubts arise as to whether this organism is a *Piroplasma*, and a new genus (*Leishmania*) has been proposed for it by Ross.

The structures which Wilson and Chowning have found in "spotted fever" have been named—

PIROPLASMA HOMINIS (Wilson and Chowning, 1903) Manson, 1903.

1903. *Pyroplasma hominis* Wilson and Chowning in Anderson, 1903a, p. 506.—Wilson and Chowning, 1904a, p. 48.

1903. *Piroplasma hominis* (Wilson and Chowning, 1903) Manson, 1903, p. 33.—Nuttall, 1904, p. 252.

1904. *Babesia hominis* (Wilson and Chowning, 1903) Chauvelot, 1904, p. 93, in part.

Wilson and Chowning (1902a, pp. 134–135) found structures in the blood of spotted-fever patients which they interpreted as protozoa, but they preferred "to make a fuller study of the life history of the organism before attempting to classify it more definitely than as a hematozoon;" they call attention to its similarity to *Piroplasma bigeminum* and also to the parasite of malaria. For a complete description of this parasite the reader is referred to the articles by Wilson and Chowning (1902a, 1903a, 1904a), Cobb (1902), and Anderson (1903a, 1903c).

Neither Ashburn nor I, in a total of 400 hours of microscopic study of the fresh and the stained blood of typical cases, and in an examination of a typical slide kindly sent to me by Wilson and Chowning, was able to find any *Piroplasma;* and Chowning was unable to demonstrate the organism to us in the fresh and the stained blood of a typical case.

Inoculations.—Wilson and Chowning inoculated rabbits with blood of spotted fever patients, both in 1902 and 1903, and they found the parasites in the blood of the experiment animals. In 1904 I inoculated rabbits with the blood of 3 fatal cases, as follows:

Two rabbits inoculated with blood within thirty minutes after death of patient.

One rabbit inoculated with blood taken from arm during life.

Two rabbits inoculated with blood within two hours after death.

The blood of these rabbits was repeatedly examined, but with negative results.

Since the observations of Wilson (in 1902, 1903), Chowning (in 1902 and 1903, but not in 1904), Cobb (in 1902), Wesbrook (in 1902), and Anderson (1903) all gave positive results, while observations (in 1904)

by Ashburn and myself gave negative results relative to the presence of a *Piroplasma* in the blood of spotted fever patients, I do not feel justified in going to the extreme of stating that no *Piroplasma* is present; all that I can assert is that I was unable to confirm the earlier results.

METHOD OF INFECTION.

Idaho.—Fairchild (1896) states that families using water from the same well are not liable to be affected similarly. Maxey (1899, p. 434) refers to the fact, in connection with cases, that the sole water supply came from melting snow; in other words, the patients drank snow water and became sick, "therefore there must be, in my opinion, some specific cause for this disease, either in the soil over which the water runs or in the snow itself."

Montana.—Wilson and Chowning (1902a, p. 134; 1903a, p. 68; 1904a, p. 44) state that there are no symptoms or lesions which point to the digestive, respiratory, or genito-urinary tracts as the avenue of infection.

Gwinn (1902), in discussing the method of infection, mentions the possibility of its entrance into the body by means of the respiratory tract, the stomach, and the skin, but he reaches no definite conclusions.

Wilson and Chowning (1902a, p. 37) report Hanbidge's interesting note that case 81 (of 1901) drank no water during the season.

The most important theory which comes up for our present consideration is the

TICK THEORY.

Wilson and Chowning (1902a, p. 136) say:

> Since there is no suspicion of "spotted fever" ever having been transferred directly from man to man, and since there is no symptomatic or post-mortem evidence of entrance of the disease, either by way of the digestive tract, respiratory, or genito-urinary system, the writers were led to examine the skin for evidence of direct inoculation by the bite of some temporarily parasitic animal. As has been noted above, in each case under observation during the investigation evidence of tick bites was present. But it is true that in the locality in which the cases occur many persons in the spring of the year are bitten by ticks and yet show no symptoms of "spotted fever." However, the following facts would seem to suggest the hypothesis that the disease is conveyed to man by means of this arachnid.

An important point upon which I desire to place considerable stress is that the tick theory is a secondary hypothesis based upon the idea that "spotted fever" is caused by a protozoon. If the *Piroplasma* theory is correct, the tick theory immediately receives a very strong argument in its favor, for other species of *Piroplasma* are known to have ticks as their intermediate host.

Accordingly, when Wilson and Chowning, in 1902, found what they believed to be a parasite similar to the parasite of malaria and also similar to *Piroplasma bigeminum*, the most natural conclusion for them to draw (reasoning on analogy) was that this organism was transmitted either by a mosquito or by a tick. They found arguments against the view that a mosquito formed the intermediate host and arguments in favor of the tick, hence they adopted the tick theory as a "working hypothesis."

From their point of view, especially on account of their microscopic

interpretations, this "working hypothesis" was most natural, and thoroughly justified, and they show (see above, p. 13) that they thoroughly understood that certain experimental studies were called for in connection with this theory.

Arriving in Missoula this season (1904), it required but a few days to see the zoological points involved and to understand that an experiment with ticks and rabbits was the most important work to be considered, especially as the strictly medical side of the disease was less important to me than the zoological features, leading to its prevention. As stated above (p. 19), I injected blood from three patients into rabbits, but failed to convince myself of the presence of any *Piroplasma* in the inoculated rabbits. Not being able, so far as I could see, to transmit the disease to rabbits, my tick experiment on these rodents had no further purpose.

Quite a number of points have been advanced in support of the tick theory. I have considered these arguments in detail, from the zoological point of view, and have prepared a lengthy discussion covering them. There are, however, so many points of difference of findings and opinion between the supporters of the tick theory and myself that three of my friends, to whom I submitted this manuscript for criticism, have felt that the discussion might be open to a possible interpretation which was most foreign to my mind. On this account I have omitted from the manuscript all points except the following:

8. All of the patients, 23, coming under observation during this investigation had been bitten by ticks. In 14 cases a history was given of severe tick bites two to eight days before the onset of the disease. In a number of other cases an apparently clear history of severe tick bites immediately preceding the onset of "spotted fever" was vouched for by the recovered patients or their friends.—Wilson and Chowning, 1904a, page 52.

In connection with this point it will be necessary to refer to some of the cases.

It must be admitted as very striking that so many cases of "spotted fever" have been preceded by a tick bite, still in a region where it is almost exceptional to go into the woods or fields without being bitten by ticks, it is not excluded that this is a mere coincidence. On account of this latter possibility, the greatest reserve should be exercised in order not to draw a "post hoc, propter hoc" conclusion.

As the valley was thoroughly worked up this year (1904) upon the subject of ticks and as people kept a close lookout for anything resembling a tick or a tick bite, negative evidence obtained from the cases of 1904 is of more value than that obtained prior to 1903. Let us therefore turn to a consideration of the cases which occurred in 1904:

Case 1.—Mr. R., patient of Doctor Buckley; fatal case. History of 2 tick bites in right inguinal region, each surrounded by an undurated inflammatory zone; patient was apparently infected in Grant Creek, namely, outside of the regular district.

Case 2.—E. C., patient of Doctor Buckley; fatal case. History of 3 tick bites April 24, back of the ear; bites caused swelling next day, fever at that time; eruption appeared April 27.

Case 3.—Mrs. R. A., patient of Doctor Fitzgerald; fatal case. Mrs. A. positively denied any history of tick bite, but thought she had been bitten by a chicken louse about April 27. On account of the tick theory she was on her guard for tick bites. Every inch of her body, including hair, pubis, and armpits, was carefully examined by the nurses, but no sign of ticks or tick bites was discovered.

Case 4.—Mrs. M. S., patient of Doctor Merrick; recovered case. Mrs. S. noticed a tick upon her, but claims that it did not bite her; no sign of tick bite was discovered, but there were parts of her body (perineum) which were not examined.

Case 5.—Miss McM., patient of Doctor Mills; fatal case. So far as could be determined, tick bites seem to be excluded in this case. Patient was aware of the tick theory and had been sharply on the lookout for these parasites; she had an unusually delicate and sensitive skin; about three weeks prior to the attack, she found a free tick between her shoulders, but she most positively denied that any ticks had bitten her. Every inch of body was carefully examined by two nurses, but no ticks were found; nor was anything found which could be interpreted as tick bite.

Case 6.—Mr. F. W., patient of Doctor Pixley; fatal case. History of tick bites about May 5; had chill May 13.

Case 7.—G. M., patient of Doctor Howard; fatal case. History of tick bites.

Case 8.—R. K., patient of Doctor Minshall; fatal case. History of tick bites in four places May 14 or 15; tick bites cauterized May 18; chill May 16 or 17.

Case 9.—Mr. G. C. F., patient of Doctor Gwinn; fatal case. History of many tick bites which were cauterized with carbolic acid.

Case 10.—J. B., patient of Doctor Mills; recovered case. Tick bite was denied in this case. An examination of the body showed a slight wound on one ankle, which might have been due to a mosquito, a thorn, a tick, or something else.

Case 11.—Mrs. E., patient of Doctor Mills; fatal case. Tick bites were positively denied. Careful examination of the body showed numerous abrasions, said to be due to mosquito bites which the patient had scratched, but no positive evidence of tick bite was found. This patient and her husband were aware of the tick theory and were prepared to treat the bites

Thus, of the 11 cases which occurred in the Bitter Root Valley 6 patients gave a positive history of tick bites. In none of these cases was the tick determined zoologically, but all specimens were supposed to be *Dermacentor andersoni*. In one of the cases the tick bite was promptly treated with carbolic acid.

In the 5 remaining cases tick bites were denied by the patients and their families. In 2 of these cases, however, it was admitted that ticks had been found crawling on the body. In 1 case the entire body was not carefully examined, but tick bite was denied. In 1 case numerous bites, claimed to be due to mosquitoes, were found. In 1 case a slight abrasion was found which could not be definitely explained. In 1 case it was claimed that the patient had been bitten by chicken lice.

In addition to these Bitter Root Valley cases, I am able to refer to 2 cases reported to me by Doctor Alton:

Case 12.—Patient noticed 4 tick bites in vicinity of left elbow about May 3; is positive regarding the presence of the ticks and also positive that he was not bitten by anything else.

Case 13.—Tick bite twelve days before illness.

Through the kindness of Doctor Gates I am able to give data regarding 17 cases which he observed:

There is no record of any tick bite in 5 of his cases, which occurred 1 each in 1894, 1898, 1900, 1902, and 1904. Of these only the case of 1904 can be given much if any weight.

In 10 of his cases, namely, 1 each in 1900, 1901, and 1902, 4 in 1903, and 3 in 1904, there was history of tick bite.

Two of his cases (15 and 16) of 1904 are especially interesting in this connection:

The patients were husband and wife. On May 29, the husband (case 15), of Bridger, visited the Clark Fork Canyon, some 60 miles south of Bridger, remaining in that vicinity about four days, during which time he was bitten by ticks in five or six places. About seven days from the time he was first bitten, namely, June 5, he arrived home in Bridger, and felt the first symptoms of the disease. Upon reaching home there were two ticks attached to his body, and these, of their own accord or from friction from the clothes, became detached from the body on June 6, and then bit the wife (case 16). The bites occurred during the night, the ticks being removed and killed by the patient the following morning. Following the removal of the ticks, she applied carbolic acid to the bites. On the evening of June 11 she felt chilly, feverish, and a general malaise, and a fatal attack of spotted fever followed.

In this connection it may be pointed out that Doctor Bradbury mentions another instance where husband and wife "had spotted fever at the same time," and he informs me that the wife was taken sick in the morning, the husband in the afternoon of the same day. Neither one was bitten by ticks, so far as could be established.

Wilson and Chowning (1903a, p. 34) record that cases 45 and 46 were two children, aged 3 and 5 years, apparently brother and sister. They are reported as both sick in June, 1899.

Anderson (1903c, p. 16) reports two cases (113 and 114) where the patients were husband and wife. Doctor Heine has written me that "the wife did occupy the same bed as her husband during the period mentioned," namely, during the prodromal symptoms in her husband's case.

In view of these 8 cases (or 6 cases, omitting the two children), it seems to me that the possibility is by no means excluded that, despite the general experience regarding the noncontagiousness of the disease, such close intimacy as sleeping in the same bed might perhaps result in a transmission of the disease to a healthy individual. At least, it must be admitted that such an explanation would account for case 16 of Gates as satisfactorily as do the tick bites; and it must further be admitted that if the tick bites actually did give the infection the proof is still wanting that such method of transmission was not accidental.

Dr. W. L. Samuels (see below, p. 28) has reported to me the case of a girl in 1904 in which no history of tick bite was obtained upon questioning the patient.

In connection with the subject of ticks I am fortunately in a position

to give some data from Idaho also. Doctor Maxey has written me under date of October 21, 1904, as follows:

> In view of the above findings [a] and the further fact that both Doctor McCalla and myself had failed to obtain a history of recent tick bite in a considerable percentage of our cases, we were led to question the theories of Wilson and Chowning even before receiving your letter.

In considering the possibility of infection by ticks it may be remarked that the tick is a very fertile animal, laying hundreds of eggs, and on this account it would be expected on a priori grounds that if "spotted fever" were a tick-borne piroplasmatic disease we ought to find quite a number of cases developing in the locality where one case developed rather than one or two cases each in several widely separated localities (see p. 43). It was the limited number of cases in comparison with the great fertility of the tick which first raised my suspicions against the tick hypothesis.

Tick bites are exceedingly common in the Bitter Root Valley; in fact, *Dermacentor andersoni* is so common that it seems rather strange that all of the patients did not show some history of being bitten by them.

That tick bites are not always of no significance is abundantly demonstrated. Doctor Buckley, for instance, had a patient who was bitten on the arm by a tick; the arm became quite swollen and the man was confined to bed for some days. Doctor Parsons had a patient who showed an extensive lymphangitis following a tick bite. Upon several occasions I have seen lesions from one-fourth of an inch to 3 inches in diameter at the point of the tick bite.

The Burrowing Squirrel (*Citellus columbianus*) as possible source of Spotted Fever.

So far as I am aware, Wilson and Chowning (1902a, p. 136) were the first to suggest that the burrowing squirrel or spermophile represents the original host of this disease. Their grounds for this suggestion were as follows:

> The extreme isolation of cases of "spotted fever," their occasional development in localities removed many miles from the site of any previous case, and the long period existing between the death or convalescence of the last case of any one year before the development of the first case in the following year, would point to the possibility of the red-blood cells of some one of the lower warm-blooded animals being the normal host of the parasitic protozoon in that stage of its cycle not passed within the body of some arachnoid. Of the animals within the infected region, the common gray gopher would probably best fulfill the conditions of such a parasitism.
>
> The writers are at present attempting to obtain data which shall confirm or demolish the above hypotheses.

[a] Namely, the inability of Maxey, Charles E. Simon, and Cole to find any *Piroplasma* in blood smears from Idaho cases.

In their later papers, Wilson and Chowning (1903a, pp. 85–90; 1904a, pp. 53–56) go more into detail regarding their reasons for considering the spermophile hypothesis. Anderson (1903a, c) does not refer to the spermophile theory, this omission being due, as he has informed me, to his absolute rejection of the idea.

Differing as I do with Wilson and Chowning in my results relative to *Piroplasma hominis* and the tick hypothesis, I felt it incumbent upon me to discuss in detail all of the points which have been advanced in support of the spermophile theory, but for the reasons referred to on page 21, with regard to my discussion of the tick theory, I have omitted all the discussion of the spermophile theory also.

Comparison of "Spotted Fever" with Piroplasmatic Diseases of Animals.

GEOGRAPHIC DISTRIBUTION.

"Spotted fever" is reported for Idaho, Montana, Nevada, Oregon, Wyoming, (? Washington State), and possibly Utah and Alaska.

Anderson (1903c, p. 8) states that it does not prevail south of 40° or north of 47°, north latitude; it occurs at an average elevation of about 3,000 to 4,000 feet above sea level.

Idaho.—According to Bowers (1896, p. 63), the disease is endemic in southern and central Idaho over an area of about 4,500 square miles about Boise City. Dubois (1896, p. 64) gives it as occurring within a radius of 50 miles from Boise. Figgins (1896, p. 64) states that it is found only in valleys of the mountain districts, while Zipf (1896, p. 65) says that it occurs more or less every year in the valleys, very seldom in the mountains. Springer (1896, p. 61) confines it to the Snake River Valley and its tributaries, and Fairchild (1896) confines it principally to the same region. Sweet (1896, p. 61) says that it is found in the entire Snake River basin and its tributaries, much of it being seen along the routes of the Oregon Short Line Railroad. Collister, (1896, p. 62) reports it as extending from Pocatello to Huntington, along the Snake River plains. According to Wilson and Chowning (1904a, p. 34), Maxey's paper describes cases in Idaho mostly along the southern foothills of the Boise mountain. The cases seem to be limited largely to the north side of the Snake River Valley from Seven Devils to Haley, and to occur from the latter part of March to the middle of July. This information may possibly have been obtained directly from Maxey, as I do not find these statements in Maxey's paper.

The statements in reference to Idaho by Wilson and Chowning are based upon Maxey's paper and upon the Wood symposium, while those by Anderson are based upon Maxey.

Montana.—McCullough (1902, pp. 225–228) states that careful inquiry shows the disease to be more widely spread than is generally supposed, and that it has prevailed at Camas Prairie, up the Blackfoot, at Phillipsburg, Clinton, Rock Creek, Rattle Snake Valley, and far up the Lo Lo, all regions separate and apart from the supposed infected area in the Bitter Root Valley. He calls special attention to this distribution in order to correct the erroneous idea that the disease is a "bugbear to this particular locality" (Bitter Root Valley). Gwinn (1902) refers to the disease as occurring in the Bitter Root Valley, Rock Creek, and Phillipsburg.

Wilson and Chowning (1902a, p. 132) state that the cases in Montana are confined to the eastern foothills of the Bitter Root Mountains, namely, on the western side of

the Bitter Root Valley in an area from 4 to 10 miles wide and 90 miles long. No case has ever been known to originate in Montana outside of this territory, except 7 cases in an area about half a mile wide and 2 miles long in the narrow canyon of Rock Creek, about 20 miles east of the Bitter Root Valley. Later (1903a, p. 42) they modify this statement to read that "few, if any, cases have ever been known to originate in Montana outside of this territory, etc." Still later (1904a, pp. 33–34) they say that the cases in Montana are confined to the eastern foothills of the Bitter Root Mountains, but they refer also to 8 Rock Creek cases and to 2 cases in a valley near Bridger, Mont., about 250 miles east and 75 miles south of Bitter Root Valley. These latter cases are evidently those reported by Gates (1903, pp. 48–51) at Thermopolis (Wyo.) and near Bridger (Mont.).

Anderson (1903a, p. 506) mentions the disease in Montana, particularly for the Bitter Root Valley (from Lo Lo to Como) and Rock Creek, but later (1903c, p. 4) refers also to Gates's cases near Bridger.

During the season of 1904 I observed (through courtesy of the attending physicians) 10 of the 11 cases in the Bitter Root Valley, distributed as follows:

Case 1.—Supposed to have become infected at or near a sawmill on Grant Creek, 6 miles north of Missoula; this was reported as being the first case in that locality. Patient was taken to Missoula.

Case 2. Patient infected 3 miles west of Victor, on west side of Bitter Root River.

Case 3.—Near Carlton, on west side of Bitter Root River. Patient was taken to Missoula.

Case 4. In Pattee Canyon, on east side of Bitter Root River, 3 miles east of Fort Missoula. There is no evidence that the patient had visited the west side.

Case 5.—Near Woodman, up the Lo Lo Creek, west side of Bitter Root River. Patient was taken to Missoula.

Case 6.—Supposed to have become infected at Harvey Creek, about 20 miles east of Florence.

Case 7. About 8 miles southwest of Hamilton; on west side of Bitter Root River.

Case 8.—Taken sick in Missoula; had not recently been up on the west side of the Bitter Root River, but had visited Bonner, east of Missoula, just prior to illness.

Case 9.—Near Florence, west side of Bitter Root River. Patient taken to Missoula.

Case 10.—Left Iowa six weeks previously; had been in Bozeman five weeks; arrived in Missoula June 2, changing cars en route to Hamilton; began to feel indisposed about the time he changed cars; taken worse in Hamilton (east side of Bitter Root River); later taken to Missoula.

Case 11.—Had been in the United States four weeks; came to Missoula about June 1; remained here five days, then moved to a ranch near Woodman, up the Lo Lo Creek, west side of Bitter Root River. Patient taken to Missoula.

From this will be seen that of the 11 cases in question 6 cases, Nos. 2, 3, 5, 7, 9, 11, appear to have become infected on the west side of the Bitter Root River, while 5 cases show no history of having visited that locality (except case 10, see below, p. 27) immediately prior to infection. One case (No. 4) appears to have become infected on the east side of the Bitter Root River. One case (No. 1) seems to have been infected up Grant Creek. One case (No. 6) appears to have been infected on the west side of Harvey Creek some distance east of the Bitter Root River. One case (No. 8) seems to have become infected either at Bonner or at Missoula; and one case (No. 10) seems to have become infected somewhere between Bozeman and Hamilton, going

via Missoula. From Fort Missoula to a point between Woodside and Hamilton the train runs close to the river on the west side of the stream.

While the predominance of cases on the west side of the Bitter Root River, as reported by Wilson and Chowning and by Anderson, was noticed (6:5) in the season of 1904, the cases were not so confined to that locality as might be expected in view of former statistics. In regard to case No. 6 (Harvey Creek) it is important to note that Wilson and Chowning (1903a, map) report cases Nos. 13, 14, 15 (28 and 30 marked as doubtful), 29, 74, and 107 from the vicinity in which case 1904:6 is supposed to have become infected.

Gates (1905, p. 114) reports 13 cases in or near Bridger from 1900 to 1904, inclusive, 4 of these occurring in 1904.

Alton (1905, p. 110) reports 2 cases in Livingston for 1904; one of these came from Lewiston, the other from Gardiner, Park County.

According to a newspaper clipping dated May 23, 1904, there was a case of spotted fever in Billings, Mont., attended by Doctor Clark.

I have also heard of one case at Dillon, but do not recall the details.

Wyoming.—Gates (1903, p. 48) reports 1 case from Thermopolis; see also Anderson (1903c, p. 4).

According to Wilson and Chowning (1904a, p. 34), Dr. J. J. Bradbury, of Cody, wrote to them that cases occurred in 1903 near Cody and Meeteetse. Anderson (1903c, p. 8) also refers to these cases.

Gates (1905, p. 114) reports, in all, 4 cases for Wyoming, namely, Meyersville, 1 case, 1894; Thermopolis, 2 cases, 1898, and Shoshone River, 1 case, 1901.

Bradbury has written to me (October 31, 1904) that he had had 2 cases at Cody, but none in 1904.

Nevada.—Maj. W. P. Kendall, surgeon, U. S. Army, reported in a letter to Wilson and Chowning (1904a, p. 34) that he saw cases in 1887 in the Quinn River Valley. Kendall's cases are also referred to by Anderson (1903c, pp. 7–8).

Major Kendall has written to me under date of October 23, 1904, that during his tour of duty at Fort McDermitt, Nev., 1885–1889, he saw 10 or 12 cases of the locally so-called "spotted fever;" his medical friends at Winnemucca, Nev., told him that there was a great deal of this disease at a small hamlet some 25 miles distant up the valley, but by them it was not considered to be a dangerous malady, as "they never died." One of the Winnemucca physicians stated that he had seen some 50 cases.

Dr. P. I. Mangan, of Winnemucca, has written to me under date of November 5, 1904, that he has repeatedly heard of a disease termed "spotted fever" that has appeared at Paradise Valley, some 45 miles distant from Winnemucca, and also at Fort McDermitt, but no case was reported in 1904; he is informed that the disease has appeared at isolated places subsequent to sheep shearing, and many persons sup-

pose it to be due to contamination from the sheep; in many cases the patients are very sick, the attack lasting several months; some fatal cases have occurred.

Dr. W. L. Samuels, of Winnemucca, has written to me under date of November 14, 1904, that "spotted fever" makes its appearance from time to time in Paradise and in a locality near the sink of Quinn River. Both of these localities are sheep countries, and the presence of bands of sheep is supposed to pollute the water courses; this is popular belief throughout this section. He says:

I had no cases in 1904, but 2 well-marked cases last year (1903). The first was an old man, a sheep herder, from the Quinn River country. My second case was a schoolgirl, age 18, who had attended a school picnic in Cross Canyon, about 7 miles from town. I questioned her closely as to the possibility of a tick bite, but she said that she did not remember having any ticks on her for months past. However, a band of sheep had been driven through the upper end of the canyon the same week she attended the picnic.

Both of my cases showed marked tendency to collapse, requiring heart stimulants all the time. Outside of that the treatment consisted of maintaining the body temperature and a careful attention to the diet.

I have been informed by the people who have had the disease, and who have seen numerous cases, that if the patient gets chilled the results are fatal, and my experience with the girl mentioned above makes me think there is something in that point. She got the covers off one night, and I spent several hours with her before her condition became satisfactory again.

I think there has been a marked diminution in the number of cases noted in this locality in the last five or six years.

Oregon.—According to Anderson (1903c, p. 8), the mild form of the disease has been reported in eastern Oregon, but he does not state where and by whom it was observed.

Utah.—Sweet (1896) states that the disease does not to his knowledge occur in Utah, but Smith (1905) (see below, p. 116) reports a possible case.

Alaska.—McCullough (1902, p. 225) says that reports show that the same disease occurs in Alaska, and Gwinn (1902) says that he has repeatedly read newspaper accounts of a disease about Klondike which very much resembled "spotted fever."

COMPARISON WITH THE OTHER DISEASES.

Bovine piroplasmosis occurs in the United States, West Indies, Argentine, Southern Venezuela, Uruguay, and apparently Brazil; in Australia, Africa (Algiers, Egypt, Uganda, Kamerun, Cape Colony, German East Africa, etc.); in southwest Russia, Bulgaria, Hungary, Roumania, Turkey, Italy, France, Germany, Finland, Norway, and perhaps in Great Britain (Nuttall, 1904, p. 220). Texas fever may extend from 37° to 38° north latitude to the Gulf of Mexico (for exact area, see maps published by the United States Bureau of Animal Industry). Hemoglobinuria is found in certain swampy regions on the Danube.

Ovine piroplasmosis is reported from Roumania, Italy, Turkey, France, and a similar affection occurs in St. Thomas, West Indies, and in South Africa. Johnson reports piroplasmatic ictero-hematuria for Deer Lodge, Mont. According to Babes, the cases on the lower Danube occur in marshy regions.

Equine piroplasmosis has been observed in South Africa and Germany, and perhaps in Venezuela.

Canine piroplasmosis is reported for Italy, France, and South Africa. Piana and Galli-Valerio (1895) noticed it in dogs which had hunted in marshy localities. Hutcheon (1893, p. 477) states that in South Africa it is very common in coast towns and districts, but comparatively rare in higher inland districts of Cape Colony; nevertheless, it prevailed about Herschel in 1893.

From these data it will be observed that piroplasmatic diseases have a wide geographic distribution, but are not confined chiefly to foothills of mountain ranges; they may be found in swampy valleys.

The question of distribution probably depends more upon the ability of the transmitting tick to develop than upon any other one factor, and as ticks develop in the region of "spotted fever," a comparison of the geographic distribution of the various diseases gives us but meager details upon which to form any judgment regarding the question now at issue.

NUMBER OF CASES.

Montana.—Gwinn (1902) estimates the number of cases he has seen in fifteen years as about 200. Wilson and Chowning (1902a, p. 132; 1903a, p. 28; 1904a, p. 33) think that probably 200 cases of the severe type have occurred (in the Bitter Root Valley) since the disease first appeared; they collate 114 cases (1903a, pp. 32–41) since 1885 which they collected from correspondence with the physicians of the valley, but they give cases 11, 18, 19, 37, 40, 108, 113, and 114 as doubtful. Anderson (1903c, pp. 12–18) increased this compilation to 121 cases.

Taking the cases given in the tables published by Wilson and Chowning and Anderson for the Bitter Root Valley we find the following distribution by years from 1885 to 1892, inclusive:

Year.	Cases.	Deaths.	Percentage deaths.	Year.	Cases.	Deaths.	Percentage deaths.
1885	1	1	100.0	1896	6	6	100.0
1886	1	1	100.0	1897	6	5	83.3
1887				1898	3	2	66.6
1888	3	1	33.3	1899	23	14	60.8
1889	3	3	100.0	1900	12	9	75.0
1890	1	1	100.0	1901	14	10	71.4
1891	6	4	66.6	1902	21	15	71.4
1892	3	1	33.3	Date?	4	2	50.0
1893	4	2	50.0				
1894				Total	114	80	70.17
1895	3	3	100.0				

As these statistics are based upon notes which the local physicians wrote up largely from memory, it is perhaps an open question whether we should draw the conclusion that the disease has increased in frequency since 1898, as the number of inhabitants in the valley have increased, or whether we should attribute the fewer number of cases reported for earlier years to the fact that the earlier cases had passed out of the memory of the local physicians.

Anderson (1903c, pp. 4, 16–19) reports but 9 cases for 1903, with 3 deaths, but he speaks (1903a, p. 40) of 10 cases under treatment.

Wilson and Chowning (1904a, p. 32) refer to having seen 10 cases personally in 1903, and to having collected data from 2 cases which they did not see. To these 12 cases, 7 deaths, should be added the following:

1903, August–September.—R. B., telegraph operator, was at Woodman, on Lo Lo stream. Was taken sick with a chill Saturday evening (August 29), and brought to Missoula Monday morning (August 30), where he later died. He was aware of the "tick theory" which had been published, but claimed that he was not bitten by ticks. Mr. and Mrs. George Kieth say positively that there was no history of tick bite in this case. He was seen by Doctor McCullough and Dr. Parsons.

1903, August–September.—J. G. W., 24 or 25 years old; so far as he knew, he was last bitten by a tick in July. About the last of August he was taken sick and saw Doctor McGrath, who made a diagnosis of "spotted fever" and sent him to St. Patrick's Hospital in Missoula. He reached Missoula September 1 and was treated by Doctor Mills. The case, which was "typical," ended fatally on September 5.

During the season 1904 there occurred in the Bitter Root Valley 11 cases, with 9 deaths. Thus we may complete the above table (p. 29) as follows:

Date.	Cases.	Deaths.	Lethality.
			Per cent.
1885 to 1902	114	80	70.17
1903	14	9	64.29
1904	11	9	81.82
Total Bitter Root Valley, 1885 to 1904	139	98	70.5
Gates's cases near Bridger, 1894 to 1904	17	3	17.6

COMPARISON.—In *bovine piroplasmosis* large numbers of animals may be affected in the same season. In some years 20 per cent of the sheep in the swampy islands of the lower Danube are destroyed by *carceag*.

The occurrence of a large number of cases of a piroplasmatic infection in a given district is natural, when we consider how very prolific an animal the tick is. A female tick lays hundreds of eggs, and it is the next generation (developing from the eggs of an infected female) which carries the infection. Accordingly, for every infected female which lays eggs, there may be hundreds of infected individuals of the next generation, hence piroplasmatic diseases are apt to attack large numbers of patients at about the same time in the same locality, and if "spotted fever" is a piroplasmosis, transmitted by a tick, we should expect a large number of cases to develop in any locality in which one case develops. This, however, is exactly what we do not find in "spotted fever," and this was the first point to lead me to seriously doubt the tick hypothesis. Wilson and Chowning lay considerable stress upon the point that, according to their studies, "in no instance have two or more persons with the same food or water supply been simultaneously stricken with this disease."

LOCALITY OF INFECTION.

Idaho.—Several Idaho observers speak especially of the fact that the disease is found in the valleys; and Collister (1896, p. 631) says that it is rarely found in high mountains. Maxey (1899, p. 434) states that in his opinion it is contracted while

the patients are residing or sojourning in or near the foothills of the mountains; he has carefully investigated this question of residence in every case coming under his observation and has found that without exception there was a history of a longer or shorter residence, just prior to the sickness, in or near the mountains or along some mountain stream where the sole water supply came from melting snow.

Montana.—Gwinn (1902) says that in the Bitter Root Valley at least 90 per cent of the cases occur on the west side of the river. Wilson and Chowning (1902a, p. 132) state that in Montana the cases are confined to the eastern foothills of the Bitter Root Mountains (see also above, p. 25). They describe this range as very rugged, the top being covered with snow until about July 1, and some peaks capped throughout the year; the range on the east side of the valley is less rugged, though the snow remains almost as long in the spring as on the west side; on the foothills the snow melts from sunny exposures as early as February, the bulk of it disappearing in April and May; the climate of the valley is very mild, as is evidenced by the many orchards of apple, cherry, and plum trees; the altitude of the valley is about 3,500 feet above sea level. In their later papers (1903a, pp. 42, 67; 1904a, pp. 33, 34, 43) they practically repeat these statements, laying stress upon the localization of the Bitter Root Valley infection to the foothills on the west side. They do not discuss the topography of the Rock Creek cases.

Anderson (1903a, p. 506; 1903c, p. 8), in discussing the localization in the Bitter Root Valley, says that the disease is sharply localized on the west bank of the Bitter Root River, no case having been known on the east side of the river who had not a short time previously visited the west side. Certain places, he states, seem to be more heavily infected than others.

From the list of cases for 1904, given on page 26, it will be seen that these statements as to localization are borne out to a certain extent, but that while 6 cases occurred on the west side of the Bitter Root River, 1 case certainly occurred on the east side, and in 4 other cases no evidence was obtained that the patients had visited the west side shortly before their illness (except that one had passed through on a train).

In connection with the larger number of cases on the west side it should be mentioned that there is a marked difference in general conditions between the east and west sides. On the west side there is much more timber and underbrush; the west side is narrower than the east and is watered by more numerous mountain streams, as is shown in the map published by Wilson and Chowning (1903a) and Anderson (1903c). In driving through the valley one is struck by the difference in the general conditions of moisture (the east side being less damp) and by the generally better economic and hygienic conditions than on the west side.

In this connection, however, it is interesting to note that there is a short stretch of land, several miles in length, on the west side, between Carlton and Lo Lo, for which neither Wilson and Chowning (1903a, map) nor Anderson (1903c, map) report any cases of spotted fever for the years 1885 to 1903, inclusive. This particular locality resembles, in condition, the east side much more closely than does any other portion of the west side between Lo Lo and Hamilton. Remarking upon this point to one of the farmers in the locality in question, I was

informed by him that in said locality the people had never experienced "spotted fever" and did not fear it.

Whether or not the absence, or at least the apparent absence, of spotted fever from this restricted area between Carlton and Lo Lo is connected with the more advanced condition, as found also on the east side, is one of the points to which future investigation should be directed.

It will be noticed that while Wilson and Chowning (1902a, 1903a, 1904a) and Anderson (1903a, 1903c) speak of the east side of the Bitter Root Valley as the uninfected locality and the west side as the infected locality, other statements Wilson and Chowning make lay stress upon the fact that infection is more or less limited to the foothills on the west side. Maxey also mentions especially the foothills in Idaho.

To Ashburn and to me these references to the foothills seemed to be of no little importance, and they were rather strongly confirmed by our inquiries, for we found the general opinion to be that it was chiefly in the foothills that infection took place. This indicated that the infectious area, or at least the area of more intense infection, was bounded on the east, not by the Bitter Root River, but by the "bench," or foothills, and as a matter of fact the local physicians and inhabitants seemed quite generally to be under the impression that the narrow lowlands close to the river were comparatively, if not entirely, safe so far as infection was concerned.

Comparison.—As shown on page 29, this tendency to limitation of infection in the foothills of mountains does not seem to agree with piroplasmosis in cattle, sheep, and dogs.

SEASONAL DISTRIBUTION.

Practically all authors lay stress upon the fact that the affection under discussion is preeminently a disease of the spring months, but it might be well to direct attention to the fact that any given date (as April 15) in the localities in question does not necessarily correspond in season to the same date in localities in the Southern or Eastern States.

Idaho.—Bowers (1896, p. 63) says that "spotted fever" occurs only during the spring, from about the 1st of March to the middle of May. According to Collister (1896, pp. 62–63) it appears in March and continues until the latter part of June. After Fairchild (1896) it usually prevails from April 1 to July 1. Maxey (1899, p. 434) reports that it invariably occurs during the spring months. The Medical Sentinel (1899, p. 457) says that "the greater prevalence of this fever in the spring suggests that the infection enters the system more easily at this time, or that the morbific agent is to be found in greater profusion. Reasoning on grounds of analogy it would seem that the former is the more likely explanation. No doubt stockmen, herders, and others living in these high altitudes have their powers of resistance considerably lessened by the rigors of a long winter; exposure to cold and deprivation of certain articles of diet producing a condition approaching scurvy. Naturally, then, we would expect in a weakened system an exhibition of virulence from the first pathogenic

microbes to be liberated and distributed by the melting snows of the spring. There is here a resemblance to a water-borne disease, and until Widal's serum test (see below, p. 66) is tried there will be cause for skepticism and a diversity of opinion as to the true nature of the malady."

Montana.—McCullough (1902) says that "spotted fever" occurs in the spring, more likely in April and May than at other times, yet it has occurred as early as January and as late as July. According to Gwinn (1902) time bears a causal relation, as is shown by the fact that the disease does not occur during the latter half of the year; nearly all cases occur in the months of April, May, and June, May being the worst month. Wilson and Chowning (1902a, p. 132; 1903a, pp. 42, 68; 1904a, pp. 35, 43) state that the disease occurs only in the spring. The earliest recorded case began March 17, and the latest about July 20, though most cases occur between May 15 and June 15. There are no records of any cases occurring between August 1 and March 17, though there are rumors of some cases having occurred as early as February. Anderson (1903c, p. 8) agrees essentially with Wilson and Chowning.

Wilson and Chowning (1903a, p. 43) print a table of the cases, distributed by months; they later insert (1904a, p. 35) the cases of 1903. In the following table I add to their cases the Bitter Root Valley cases I have collected.

Month.	Reported by Wilson and Chowning, 1904a, page 35.	Additional cases.	Total.	Remarks.
January	.....	(?)	(?)	See McCullough, 1902; uncertain case, see Cobb, 1902, page 1868.
February	.....	(?)	(?)	Rumors, see Wilson and Chowning, 1902a, page 132; a few cases when March was mild, see Cobb, 1902, page 1868.
March	6	.....	6	
April	24	2	26	The 2 additional cases (1, 2) in 1904.
May	46	7	53	The 7 additional cases (3, 4, 5, 6, 7, 8, 9) in 1904
June	35	2	37	The 2 additional cases (10, 11) in 1904.
July	5	.....	5	
August	.....	2	2	Cases in 1903, see page 30.
September	.....			
"Spring"	10	.....	10	
Total	126	13	139	

Of the 17 cases reported by Gates for another locality, 4 occurred in April, 6 in May, and 7 in June.

It is a more or less popular belief among some of the inhabitants of the Bitter Root Valley that cases of "spotted fever" are more likely to occur during the time that the streams are rising than while they are falling. This would indicate that it occurred either during or following a rise in temperature, such as would melt the snow on the mountains, or during or following a rainfall.

Through the kindness of Prof. Willis Moore, Chief of the United States Weather Bureau, I have been able to obtain the daily temperatures and rainfall for Missoula for the months of February-September, inclusive, 1899-1904, inclusive, so far as these have been recorded by volunteer observers (Prof. M. J. Elrod), and I have attempted to plot the cases of "spotted fever" which are reported for the Bitter Root Valley for the corresponding days. While this study was interesting, it is not entirely satisfactory for several reasons; in the first place,

the exact date of the onset in a number of cases is not given; further, the temperature and precipitation varies some in different parts of the valley, and I could not obtain these data for the exact locality where the cases occurred. Hence the results obtained are only approximate.

I find that of the cases upon which even an approximate comparison could be made 13 cases developed in the valley within seventy-two hours following an increase in temperature at Missoula, but no precipitation was reported for Missoula; 45 additional cases developed in the valley within seventy-two hours following an increase in temperature at Missoula, and precipitation is reported for Missoula during the same period. Thus a total of 58 cases occurred during or following conditions which would result in a swelling of the streams, hence more moist general conditions. One additional case developed during a decrease of temperature at Missoula, without any contemporaneous precipitation.

Without laying too much stress upon these data, because of their incompleteness, it is rather striking that of the 59 cases in which the given data could be compared, 58 of them, or 98.3 per cent, occurred under conditions which would result in an increase in the amount of running water in the valley, thus apparently bearing out the popular view that "spotted fever" is more likely to develop during a rise rather than a fall of the valley streams.

It is also interesting to note that by far the majority of the cases occur on the west side of the river, which is more moist than the east; that the disease seems to be a disease of valleys, which are naturally more moist than are the plains. Future investigations, therefore, have the interesting problem to solve whether this concurrence of moisture with the infection is of any significance or is a mere coincidence. The relation of the moisture to the tick theory is not apparent to me.

Taking the monthly maximum temperature at Missoula in connection with 88 cases which could be plotted, it was found that the lowest maximum was 48.1 F. (2 cases), the highest, 87.6° F. (2 cases); 3 cases occurred with monthly average maximum from 48.1 to 49.6° F., 20 cases with monthly average maximum from 54.2 to 58.6° F., 34 cases with monthly average maximum from 63 to 69° F., 26 cases with monthly average maximum from 71.7 to 76.6° F., 5 cases with monthly average maximum from 80.5 to 87.6° F.

For the same 88 cases, the lowest monthly average minimum temperature at Missoula was 22.2 (1 case), and the highest average minimum, 51.2 F. (2 cases). Four cases occurred with a monthly average minimum from 22.2 to 28.5 F., 38 occurred with a monthly average minimum from 31 to 38.7 F., 44 occurred with a monthly average minimum from 41 to 48.7 F., and 2 with a monthly average minimum of 51.2 F.

For the same 88 cases the lowest difference between the monthly average maximum and minimum temperature for Missoula was 19.6° F. (2 cases), the highest 35.8° F. (2 cases); for 2 cases there was a monthly average difference of 19.6° F; for 70 cases there was a monthly average difference from 21.7 to 29.8° F., inclusive; for 16 cases there was a monthly average difference from 30.4 to 35.8° F.

Thus a monthly average maximum of 63 to 73° F., inclusive, obtained in 59 out of 88 cases, or 67 per cent of the cases; a monthly average minimum of 37 to 46° F., inclusive, obtained in 58 out of 88 cases, or 65.8 per cent of the cases; of 88 cases 70 cases, or 79.5 per cent, occurred during an average monthly difference of 21.2 to 29.8° F. between maximum and minimum temperature, and 55 of these cases, or 60.2 per cent of the 88 cases, occurred during a monthly average difference of 25.2 to 29.7° F., inclusive, between the monthly maximum and monthly minimum.

It would therefore appear that, so far as can be concluded from the data at our disposal, there seems to be some connection, either direct or indirect, between the temperature of the air and the development of cases, in that up to a certain point an increase in temperature is coincident with the development of cases, while beyond that point a further increase in temperature seems not to favor the appearance of new cases. Of course an increase in temperature increases the amount of water resulting from the melting snow, but there finally comes a time when the supply of snow is greatly decreased, hence a further increase in temperature would not have the same effects upon the amount of water in the valley.

Our data upon these points are not sufficiently exact to permit of positive conclusions, but such as the data are they tend to support rather than to negative the popular idea that the melting snow has some direct or indirect connection with the development of cases, or at least they tend to show that conditions which favor the melting of the snow also favor the appearance of cases of spotted fever.

Taking the amount of precipitation, including rain, hail, sleet, and melted snow (not from the mountains), we find that 14 cases occurred in six months showing a total monthly precipitation of 0.37 to 0.68; 50 cases occurred in eleven months showing a total monthly precipitation from 1.02 to 1.98; 3 cases occurred in one month showing a precipitation of 2.78; 7 in a month showing precipitation of 3.84, and 14 in 2 months showing precipitation of 4.19 and 4.53.

The lowest precipitation in any month during which a case developed was 0.37 (1 case); the highest number of cases in reference to precipitation were as follows: 6 cases, 0.65; 9 cases, 1.2; 9 cases, 1.44; 15 cases, 1.28; 7 cases, 3.84; 6 cases, 4.19; 8 cases, 4.53.

From this it is seen that in general fewer cases have developed during the epidemic months with a precipitation under 1 than with

a greater precipitation, and while the greatest number (15) developed with a precipitation of 1.28, the average stands as follows: 2.33 cases between 0.37 and 0.68; 4.5 cases between 1.02 and 1.98; 3 cases at 2.78; 7 cases at 3.84; 7 cases between 4.19 and 4.53.

While these data are too incomplete to permit of definite conclusions, still, such as they are, they are in harmony with the view that cases are more likely to occur coincident with a rising of the streams than with their fall.

COMPARISON.—Texas fever, in the United States, occurs in summer and fall; hemoglobinuria in the Danube region occurs usually in the late spring, summer, and late fall. Carceag is reported for the Danube region, especially in May and June. Canine piroplasmosis prevails at the Cape chiefly in summer and autumn, and is reported in Europe for April, September, and October.

We should not be led astray by this comparison of months. The life history and seasonal occurrence of ticks in different places might vary according to the species of tick involved and according to the climate.

AGE AND SEX OF PATIENTS.

Idaho.—Fairchild (1896) says that all classes and all ages are affected, the rich, the poor, the weak, the robust, the young, the old, males and females, alike. Bowers (1896) agrees that the disease attacks persons in all conditions of life, but males in larger proportion. According to Dubois (1896, p. 64) "spotted fever" attacks persons of all ages, but adults of both sexes are more subject to infection than are children. According to Maxey (1899, p. 434) it is much more frequent in men on account of their exposed occupations, and this will explain also why children are rarely, if ever, affected; the youngest case he knows of was in a patient 6 years old, the oldest in a patient nearly 70 years of age.

Montana.—Gwinn (1902) reports that in his observations he has noted that age, sex, and amount of vitality play little or no rôle in infection.

The first actual statistics concerning age and sex were collected by Wilson and Chowning (1903a, p. 43), based upon 114 cases; they conclude (1903a, p. 68; 1904a, p. 43) that the disease attacks alike patients of any age or either sex, though those whose occupations or pleasures take them to the foothills of mountains in the springtime are most affected; they report one case (1903a, pp. 38, 59) in a child 2 years old and another (p. 32) in a babe 2 days old; they give a very interesting table of 114 cases, and conclude that the cases are too few to warrant elaborate conclusions, but that it is probable that the large number of cases occurring in males (36) of 20 to 40 years, and in females (25) of somewhat younger age (10 to 40), is due to the increased exposure to infection through occupation or pleasure taking them outdoors in the foothills and mountains in the spring of the year.

Anderson (1903c, p. 9) shows that of 121 cases 76 were males and 45 females, the difference in number in the two sexes being probably due to a greater liability to exposure on the part of men on account of occupation. He gives the ages of 15 to 50 as being most liable to infection, and reports 18 months as the youngest and 74 years as the oldest cases recorded.

The Wilson and Chowning table (1903a, p. 43) of 114 cases was increased by them (1904a, p. 36) to 126 cases. It is here increased to 139 cases, to contain the Bitter Root Valley patients observed in August–September, 1903, and in the 1904 outbreak.

The number of cases for Bridger and vicinity is not sufficiently large to permit of comparison by age, but it may be noted that 12 were in males and 5 in females.

Age, years.	Males.							Females.							Both sexes.		
	Compiled by Wilson and Chowning, 1904a, p. 36.		Additional.		Total.			Compiled by Wilson and Chowning, 1904a, p. 36.		Additional.		Total.			Total.		
						Deaths.							Deaths.			Deaths.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Number.	Per cent.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Number.	Per cent.	Cases.	Number.	Per cent.
Inclusive under 5	8	4	....	....	8	4	50.0	5	5	1	1	6	6	100.0	14	10	71.4
6 to 10	6	5	1	0	7	5	71.4	7	4	....	....	7	4	57.1	14	9	64.2
11 to 20	8	5	2	2	10	7	70.0	11	5	....	....	11	5	45.4	21	12	57.1
21 to 30	17	13	2	2	19	15	78.9	7	3	3	3	10	6	60.0	29	21	72.4
31 to 40	24	19	1	1	25	20	80.0	11	7	....	....	11	7	63.6	36	27	75.0
41 to 50	8	6	....	....	8	6	75.0	2	1	....	....	2	1	50.0	10	7	70.0
51 to 60	3	2	....	....	3	2	66.6	2	1	....	....	2	1	50.0	5	3	60.0
61 to 80	4	4	1	1	5	5	100.0	2	2	....	....	2	2	100.0	7	7	100.0
Not stated	1	1	[a]1	1	2	2	100.0	....	0	[b]1	0	1	0	0	3	2	66.6
Total	79	59	8	7	87	66	75.9	47	28	5	4	52	32	61.5	139	98	70.5

[a] Baskerville, 1903. [b] Merrick's case, 1903.

This table, October 31, includes 11 additional cases for Bitter Root Valley, 1904.

So far as can be judged from these statistics, the lethality of this disease for the Bitter Root Valley has been as follows:

Average of all cases, 70.5 per cent.
Average of all males, 75.9 per cent.
Average of all females, 61.5 per cent.
For females from 11 to 20 years, 45.4 per cent.
For males under 5 years, females 41 to 50 years, and 51 to 60 years, 50 per cent.
For females from 6 to 10 years, and all cases from 11 to 20 years, 57.1 per cent.
For females from 21 to 30 years, and all cases 51 to 60 years, 60 per cent.
For females from 31 to 40 years, 63.6 per cent.
For all cases from 6 to 10 years, 64.2 per cent.
For males from 51 to 60 years, 66.6 per cent.
For males from 11 to 20 years, and all cases from 41 to 50 years, 70 per cent.
For all cases from 21 to 30 years, 72.4 per cent.
For males from 41 to 50 years, and all cases 31 to 40 years, 75 per cent.
For all males, average, 75.7 per cent.
For males from 21 to 30 years, 78.9 per cent.
For males from 31 to 40 years, 80 per cent.
For males from 61 to 80 years, females 61 to 80 years, all cases 61 to 80 years, and females under 5 years, 100 per cent.

Comparison.—In Texas fever, the disease attacks both sexes, but seems to be more seldom in calves than in adults; Starcovici says that cows are rarely attacked with hemoglobinuria, and that the disease is unknown in calves; also that only adult sheep are affected with carceag. According to Robertson (1901, p. 327), canine piroplasmosis attacks both young and old dogs; Nocard and Motas (1902, p. 275) found young dogs to be much more susceptible than adults.

OCCUPATION OF PATIENT.

Idaho.—"Spotted fever" is mostly confined to teamsters, who camp out during the summer months (Figgins, 1896, p. 64). Stockmen in general, but particularly sheep men, prospectors, and miners, are special prey of this disease, as their occupations take them into the mountains. These men are exposed to all kinds of weather day and night, sleep on damp ground, in damp beds, their meals are irregular, their food is coarse and poorly cooked, and proper personal cleanliness seems to be out of the question (Maxey, 1899, p. 434). Spotted fever occurs among stockmen, miners, and other mountaineers (Medical Sentinel, 1899, p. 456).

Montana.—Anderson (1903c, p. 8) states that all occupations that cause the person to be exposed to the bite of ticks, such as stockmen, and especially sheep herders, miners, prospectors, lumbermen, ranchmen, and those whose duties take them into the brush, are subject to the disease.

Wilson and Chowning (1902a, p. 132; 1903a, p. 42; 1904a, pp. 34–35) state that the population of the Bitter Root Valley is made up largely of fairly well-to-do ranchers, the majority of whom have come from Missouri, Georgia, and the Carolinas. They are, as a rule, cleanly and healthy. The lumber industry is an important one, and many cases of spotted fever have arisen about sawmills and on ground recently cleared of timber.

Taking the occupations as given by Wilson and Chowning for cases 1 to 121, and adding others reported by Anderson (1903c) and those collected for the Bitter Root Valley by Ashburn and myself, we have the following data:

Thirty-eight patients were children, or boys and girls, as follows: 4 schoolboys, 4 schoolgirls; 30 children, occupation not given, but probably most of them lived on farms.

Twenty-six patients were housewives or housekeepers, probably the majority on farms.

Twenty patients were connected with farms, as follows: 19 farmers or ranchmen, 1 farmer's daughter.

Sixteen patients were connected with the lumber industry, as follows: 10 lumbermen, 2 lumber jacks, 1 lumber cruiser, 1 sawmill man, 1 business man, who had been at a sawmill.

Eleven patients are listed as laborers, but the kind of labor is not stated.

Eleven patients are not listed relative to their occupation. Two patients are listed as trappers; 2 patients are listed as prospectors; 4 patients are listed (1 each) as stonemason, teamster, miner, and telegraph operator (who was in the valley on other work at the time.)

Two female patients are listed (1 each) as nurse and school-teacher.

In this list it is striking that a very large number of the patients were either on farms or connected with the lumber industry, but it should be remembered that farming and lumbering are the chief occupations in the valley; hence, other things being equal, these occupations would be expected to furnish a large percentage of the patients.

In Gates's statistics the following occupations are given:

Four children (2 boys, 2 girls), 1 housewife, 6 ranchmen, 1 sheep herder, 1 stockman, 1 trapper, 1 freighter, 1 nurse.

TYPES OF CASES.

Idaho.—The type of the disease which appears in Idaho, as described by Doctor Maxey, is apparently very much milder than that of the severe form appearing in Montana, though all cases show the peculiar eruption. (Wilson and Chowning, 1902a, p. 132; 1903a, p. 44; 1904a, p. 37.)

Montana.—Gwinn (1902) divides the cases into mild, medium, and most severe.

Most physicians in the Bitter Root Valley who have had experience with the disease recognize but one type—a severe and usually fatal form—the principal diagnostic feature of which is the "spots." Several physicians recognize a mild type in which there are no "spots." There is much difficulty in the accurate diagnosis of the mild type and, though its existence must be recognized, yet during the investigations herewith reported all of the examinations except 1 were made on cases of the severe type. (Wilson and Chowning, 1902a, p. 132; 1903a, p. 44; 1904a, p. 36.)

Some of the physicians in the Bitter Root Valley speak of cases of "local infection" with "spotted fever." Wilson and Chowning (1903a, p. 61) report one such case, but do not include it in their statistics. I am unable at present to admit these cases. (See Stiles, 1905, pp. 14–15).

COMPARISON:

Smith and Kilborne (1893, p. 15) describe for Texas fever two types, an acute fatal type and a mild, rather prolonged, usually nonfatal type.

For canine piroplasmosis, acute and chronic types are described.

SYMPTOMATOLOGY.

The symptomatology of the disease under consideration has been discussed by Wood (1890), Maxey (1899), McCullough (1902), Gwinn (1902), Wilson and Chowning (1902, 1903, 1904), Anderson (1903), and Gates (1903). Wood and Maxey have described the symptoms as they are observed in Idaho and the other authors as they are observed in Montana. As no one has as yet brought all these observations together it may be well to do so at this time in connection with the symptoms observed in Montana in 1904.

As stated in the introduction (see above, p. 10), my trip did not contemplate a study of symptomatology, but it naturally became necessary for me to familiarize myself more or less with this phase of the subject.

The symptoms are quite well marked and very constant (Maxey, 1899, p. 435).

All symptoms and lesions indicate that the disease is due to a specific infection (Wilson and Chowning, 1903a, p. 68; 1904a, p. 43).

PREVIOUS CONDITION OF PATIENT.

Idaho.—Bowers (1896, p. 63) refers to exposure to cold, drafts, and dampness as auxiliary causes.

Montana.—Gwinn (1902) says that at least 90 per cent of the cases give a history of having been exposed to wet or cold, or both, from one-half to three days prior to the attack; and although the exposure and constipation are so constant a feature as to be apparently operative in the cause, yet little stress can be laid upon them from the fact that they do not have such effect at other times of the year or in other localities. Wilson and Chowning (1902a, p. 132; 1903a, p. 43; 1904a, p. 35) say that

the age and sex and general health of the patient appear to have no part in determining susceptibility to the disease; a large number of cases give a history of recent exposure to wet or cold or of overexertion shortly before the attack, but in several cases all such history has been absolutely excluded; many of the patients have suffered somewhat from indigestion and constipation immediately prior to the attack, in others no such condition existed.

Period of Incubation.

Idaho.—The period of incubation is uncertain, probably 10 to 21 days (Bowers, 1896, p. 63).

Montana.—Gwinn (1902) says that in so far as exposure to wet and cold may be a cause, the onset is on an average of 18 hours after exposure. Anderson (1903a, p. 21) gives the incubation as 3 to 10 days, usually about 7. Wilson and Chowning (1904a, p. 37), arguing on the theory of transmission by ticks, state that though all cases occurring in 1902 and 1903 gave a history of tick bites (see, however, p. 30) shortly before the onset of the symptoms, only the following cases gave a clear history of a definite incubation period:

	Days.
Cases 94, 97, 117, 124, 125	2
Case 96	3
Case 119	5
Case 116	6
Case 112	7
Case 115	8
Cases 120, 121	2 to 5

Several of the cases which occurred in 1904 give points of comparison upon the period of incubation.

Arguing on the tick hypothesis, it may be noted that:

1904, case 2 was bitten by ticks April 24, and fever was noticed the following day.

1904, case 8 was bitten by ticks May 15, and was taken sick the following day.

1904, Gates's case 15 started for Clarke Fork Canyon May 29, remaining in that vicinity about 4 days, during which time he was bitten by ticks in five or six places; he showed first symptoms 7 days after first tick bite.

1904, Gates's case 16 showed symptoms 5 days after tick bite.

Not arguing on the tick hypothesis, it may be noted that—

1904, case 3 moved in January to house in which she was taken sick on May 3, making a maximum of about 4 months in the infected locality before she became ill.

1904, case 10 left Iowa 6 weeks prior to onset, 5 of these 6 weeks being spent in Bozeman, Mont.; thus this patient was in Montana a maximum of 6 weeks prior to illness.

1904, case 11 had been in the United States 4 weeks, in the Bitter Root Valley 19 days, and at the house where she was taken ill 14 days prior to onset of the disease.

1904, Gates's case 15 was taken ill about 7 days after he visited the locality in which infection is supposed to have taken place; and

1904, Gates's case 16 was taken ill 6 days after sleeping with a case (15) in its initial stage.

These data would indicate that either upon basis of the tick hypothesis or upon basis of infection by some other unknown method, the disease may develop within 6 days after exposure; the maximum

and minimum time between exposure and onset can not be well established from the data now at our disposal.

Arguing upon the tick hypothesis it would seem that the disease might develop within a day after exposure.

Comparison.—Smith and Kilborne (1893, p. 15) point out that the term "period of incubation" is used in two different senses in connection with Texas fever. In experimental cases of Texas fever it is 6 to 10 days after the cattle are inoculated with Texas fever blood, the time depending on the number of parasites originally introduced, the predisposition and age of the animals, and the season of the year. Starcovici gives about 14 days as incubation period for hemoglobinuria, and "about 8 days (?)" for carceag. For canine piroplasmosis the period is given as 10 days from the date of visiting a tick belt (Robertson); Nuttall found it to be 13 to 21 days in dogs bitten experimentally by ticks; upon subcutaneous injection the period varies from 3 to 10 days.

Onset.

Idaho.—During incubation there is a slight headache and a feeling of lassitude and inaptitude for work; during the first week following this period the patient complains of chilly feelings, nausea, loss of appetite, intense headache, pain in the back and legs, a muscular soreness and stiffness of the entire body, and he takes to bed with a temperature of 102° to 105° F., pulse 90 to 120 (Bowers, 1896, p. 63). According to Collister (1896, p. 63) many cases are taken suddenly without previous malaise, some with a severe chill and others with more or less chilly shudderings frequently referred to the spinal regions; still others have little or no chill. Dubois (1896, p. 64) says that there is usually no prodromal stage and a patient is stricken down without warning with severe frontal headache, photophobia, nausea, lassitude, persistent anorexia, and intensely severe pains in joints and muscles. According to Fairchild (1896, p. 62) the attack is sometimes ushered in by a chill, but usually by two or three days of malaise, with severe headache, particularly in the back part; also shooting pains throughout the body and limbs, usually more severe in the bowels and back; the pain is neuralgic in type. Figgins (1896, p. 64) states that the disease begins with a chill, pains in the extremities, muscular soreness, and fever, the temperature ranging from 100° to 105° F., pulse 100 to 120. Springer (1896, p. 61) says there is a feeling of malaise for a few days, followed by a chill; the fever then sets in, ranging from 103° to 105° F. According to Sweet (1896, p. 61) the onset is usually accompanied by severe break-bone pains. According to Zipf (1896, p. 65) the onset is sudden, with high fever, violent headache, coated tongue, backache, and flushed face. Maxey (1899, p. 435) states that the patient first notices a general malaise, loss of appetite, and flashes of heat and cold, but no marked chill; the bones and muscles soon begin to ache, and by the second day the patient feels sick enough to take to his bed; he already feels very weak and depressed, and pains in the back, in the joints and muscles of the extremities, and in the head become quite severe; the bowels are constipated.

Montana.—In a few cases the disease seems to be preceded by a prodromal period of malaise for a few days; the attack comes on by either a well-marked chill or chilliness, simultaneous with fever, general aching and soreness of the entire body, and a flushed dusky red color of the skin. The chilliness, although most severe at the onset, often continues more or less throughout the attack, coming on at intervals—generally mornings—and becoming lighter day after day until within a week or so the chills seem but little more than chilliness from light covering (Gwinn, 1902).

According to McCullough (1902, p. 226), the onset may be marked by a sudden and severe chill and dizziness, with high fever following, associated with intense soreness seemingly of the entire muscles of the body, or it may come on insidiously,

a feeling of malaise for a few days, gradually growing worse and merging into a well-defined "bone ache" and slight chilly sensations, mostly in the morning.

Wilson and Chowning (1902a, p. 132) say that many of the cases are preceded by a short period of malaise; this is followed by a marked chill, which is usually most severe at the beginning of the attack and recurs at irregular intervals, though with decreasing severity; at the onset there is a severe aching in the bones and muscles, with pain in the back and joints; the patient is usually very weak and the headache may be severe; constipation at this period is usually present; there is considerable restlessness; a bronchial cough is frequently present; the urine is small in amount and highly colored; albumen is sometimes present; the skin is dry and the tongue, even at the onset, is thickly coated; the coat at first is white, but it becomes brownish as the fever increases, while the tongue becomes dry and cracked; sordes appear early, and may be quite pronounced; indeed, the whole facies in these respects is like typhoid. Wilson and Chowning (1904a, p. 37) also report that cases Nos. 94, 96, 97, 115, 116, 117, 119, and 120 gave a history of soreness about the tick bite and pains radiating therefrom which continued until the initial chill.

According to Anderson (1903a, p. 50; 1903c, p. 21), the patient may have chilly sensations, malaise, and nausea for a few days, then there is a distinct chill, and the person takes to bed; there is some pain in the back and head, soreness of the muscles and bones, causing a sensation as if the limbs were in a vise; bowels constipated; tongue with heavy white coat, red edge and tip; conjunctivæ congested, becoming yellowish; urine usually small in amount, with albumen and a few casts; slight bronchitis after a few days; nose bleed, sometimes quite severe, is always present. In case 74 (1903a, p. 15) the symptoms were about three days in reaching their height.

Comparison.—In Texas fever the fever usually precedes the outward symptoms by several days; pulse and respiration rise with the temperature; loss of appetite always, and cessation of rumination usually accompany the high fever after the third or fourth day.

Hemoglobinuria begins with exhaustion, loss of appetite, and fever; carceag with chill, exhaustion, and fever. Canine piroplasmosis is ushered in by a fever and loss of appetite, followed by increasing prostration, ending in complete helplessness.

Duration.

Idaho.—The period of incubation is uncertain, probably 10 to 21 days; from the appearance of prodromal symptoms until convalescence, 12 to 20 days; average period of convalescence, 1 month, very infrequently several months; in 1 case, 2 years elapsed before sunlight was borne without intense cephalalgia (Bowers, 1896, p. 63). According to Figgins (1896, p. 64) the duration is 14 to 42 days, the eruption showing for 4 or 5 months after the patient is up and around, especially when subject to heat or physical exercise. Fairchild (1896, p. 62) gives the duration as 14 to 28 days. Springer (1896, p. 61) reports duration as 2 to 3 weeks. Sweet (1896, p. 61) states that the duration is 3 weeks to 3 or 4 months, i. e., although the fever may not last longer than 21 days, the lesions may continue for months. The disease usually terminates in course of 2 or 3 weeks by lysis, usually in recovery (Medical Sentinel, 1899, p. 457).

Montana.—Gwinn (1902) reports that one case persisted 3 months before recovery.

Gates (1905) reports convalescence in 14 of his 17 cases as follows: In a few days, 1 case; 10 days, 1 case; 12 days, 2 cases; 14 days, 1 case; 15 days, 1 case; 18 days, 1 case; 20 days, 1 case; 21 days, 1 case; 22 days, 2 cases; about 25 days, 1 case; 28 days, 1 case; not given, 1 case.

See also Deaths, page 89.

In the 2 Bitter Root Valley recovery cases of 1904 1 (No. 4) was

taken sick about May 8 and was able to sit up May 21, thus giving a duration of 13 days from onset to convalescence; 1 (No. 10) noticed first symptoms June 2 and was discharged from the hospital June 17, giving a duration of 15 days.

Comparison.—Extremely acute cases of canine piroplasmosis may last only 24 hours, but it appears more often to last 3 to 6 days; subacute cases last about 10 days; chronic cases, 21 to 62 days. Starcovici states that in hemoglobinuria the fever lasts about 5 days. In Texas fever the continuous high temperature rarely lasts longer than 8 to 10 days.

Epidemic Character.

Under the heading "Seasonal distribution" (p. 32) it is shown that spotted fever occurs chiefly during the spring months and in certain localities, and on page 44 it is shown that all authors agree that it is not contagious. The question naturally arises as to the distribution of cases by families.

Idaho.—Bowers (1896, p. 63) states that spotted fever occurs sporadically. Figgins (1896, p. 64) says that he has seen it in families who used water from springs and where the entire family had the disease. In the experience of Fairchild (1896) the cases have been sporadic, and it is exceptional to find more than 1 or 2 cases in the same house. Sweet (1896), however, says that frequently several cases occur in a household, again only a single case. This latter fact is often due to the patient's returning home ill after sojourning in another place up to the malaise.

Montana.—It is common for quite a number of the patients to come down with the disease near the same time; then perhaps there will be a respite for a week or ten days, when there will be a number of other almost simultaneous attacks in the neighborhood. If upon further investigation this proves true it may be significant in detecting the cause. (Gwinn, 1902.)

According to Wilson and Chowning (1903a, p. 68; 1904a, p. 43), in no instance have two or more persons with the same food or water supply been simultaneously stricken with the disease.

In no instance which Ashburn and I saw personally was there more than one patient in the same house, but the following cases are interesting, and possibly important, in this connection:

Cases 45 and 46 (Doctor Gwinn's patients in 1899) were two children, 3 and 5 years old, respectively (Wilson and Chowning, 1903a, p. 34). Gwinn writes me that he believes some confusion has arisen in connection with the dates of these two cases.

Cases 113 and 114 (Doctor Heine's patients at Butte in 1893) were husband and wife (Anderson, 1903c, p. 16). They were living together, and one was taken sick ten days later than the other.

In 1903 there occurred 3 cases (father and two sons) of illness in the family of A. M., on Lo Lo Creek. None of these cases is reported either by Wilson and Chowning or by Anderson:

One boy was sick with a disease which his mother supposed was "spotted fever." His father went to town to obtain medicine for the boy, and encountering a storm he became thoroughly "wet through." The father was then taken sick and was seen at the hotel in Missoula by Doctors Gwinn and Wilson. Doctor Gwinn informs me

that the father's case was one of spotted fever. A second son was also taken sick, according to his mother, also with "spotted fever." In case these patients all had "spotted fever," we here have an instance in which 3 cases occurred in one and the same family during the same spring, all the patients living in the same room in a cabin and at least two of the patients being sick at the same time. It must, however, be recalled that although I have the mother's statement to the effect that all three had "spotted fever," and although this diagnosis is accepted as correct by the neighbors, we have a physician's opinion in regard to only one of the cases.

Not far from the farm on which these cases occurred is the house in which (1904) case 5 occurred. Ashburn and I visited this house and obtained the information that at least one other case of "spotted fever," and possibly still a third case, had occurred in this same house, but the 3 cases were not simultaneous, and only 1 of them occurred in 1904.

On page 23 (see above) mention is made of a husband and wife who both had spotted fever at the same time (1900) in Boise. Doctor Bradbury writes me that the wife was taken ill at 10 a. m. and the husband was taken ill at 5 p. m., the same day.

Gates (1905, p. 112) reports two cases (15 and 16) in husband and wife, in 1904; the wife was taken ill about 6 days later than the husband.

Comparison.—Texas fever appears suddenly and, as a rule, at about the same time in all animals of a herd which have been exposed to the same infection together. In hemoglobinuria all of the cases of disease last in a given place scarcely longer than 14 days, then the outbreak disappears.

Is "Spotted Fever" Contagious?

Idaho.—The disease is not contagious (Bowers, 1896, p. 62; Sweet, 1896; Maxey, 1899, p. 433; Medical Sentinel, 1899, p. 457).

Montana.—Authors writing for Montana admit that there is no evidence that the disease is contagious (McCullough, 1902, p. 225; Gwinn, 1902; Wilson and Chowning, 1902a, 1903a, 1904a; Anderson, 1903a, c). Wilson and Chowning (1903a, p. 68; 1904a, p. 43) state that there is not even a suspicion [see, however, above, p. 23] of its ever having been transferred directly from one human being to another, except in one instance, in which an infant (case No. 17), born while the mother was suffering from the disease, developed marked purpura on the second day after birth. This child (case No. 17) was born 4 days before the death of the mother; early on the second morning after birth the physician's (Doctor Coughenour) attention was called to the child's fever and jaundiced appearance; the babe had but few spots and began to recover on the ninth or tenth day of his sickness (Wilson and Chowning, 1903a, p. 33; Anderson, 1903c, p. 13).

Regarding the noncontagious character of "spotted fever," all information which Ashburn and I obtained in the Bitter Root Valley, as well as our own personal experience, are in accord with the generally accepted view. See, however, the discussion on page 23, which seems to open up the question as to whether the malady is not contagious under some circumstances.

Comparison.—None of the known piroplasmatic diseases are directly contagious, but they must first pass through a tick, between two successive patients.

Position in Bed.

Owing to the muscular and articular soreness, the patients lie in a position of general flexion for hours without moving (Bowers, 1896, p. 64).

See also Restlessness, page 75.

Odor.

There is a peculiar urinous odor to many of the patients, even when the greatest care and cleanliness are exercised in nursing them. This odor was especially marked in 1904 in cases 2, 3, 7, and 11.

In case 12, upon removing the clothing, the odor of the body led the physician to ask the patient whether he had ever had measles, to which he replied in the affirmative.

Skin.

general condition.

Idaho.—During the first week the skin is usually dry, later it is somewhat moist, and night sweats are common during the third week (Bowers, 1896, p. 63). Collister states that the skin shows no abnormal appearances. According to Figgins (1896, p. 64) it is swollen and sensitive to the touch. Maxey (1899, p. 435) describes the skin as dry and harsh.

Montana.—Gwinn (1902) speaks of the flushed, dusky-red color of the skin; if it is pressed while in a congested condition, it appears blanched upon removal of the fingers and is much slower in regaining its color than in health; this fact being true, in spite of the strong pulse, leads Gwinn to believe that the blood current is obstructed in the capillaries, and this belief is supported also by the nature of the eruption, the oft-occurring gangrene, and the really thick unoxygenated blood; whether this obstruction be due to changes or to alteration in the blood is hard to determine, but Gwinn inclines to the latter explanation; in addition to the eruption, the skin takes on a congested, jaundiced color; the congested and cyanotic condition of the skin causes a bloated, stupid expression of the face in most cases, which is a very diagnostic symptom. Gwinn also states that at the beginning of the second week there may be noticed a glazed appearance of the skin which, upon close examination, is found to be due to epithelium coating; it begins to scale up and shed off at the beginning of the third week.

Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37) describe the skin as dry; there is a peculiar ashy paleness present, more readily observed in children and women, a few days before death (1902a, p. 132; 1903a, p. 63; 1904a, p. 38); about the second week, ordinarily, the skin presents a *glazed appearance.*

Anderson (1903a, p. 33) speaks of the lividity on dependent portions of skin and thighs (case 120); he also refers (1903c, p. 38) to a marbled appearance of the skin.

Gates (1903, p. 48) reports hot and dry skin in one case; he later (1905, p. 111) reports the skin as very dry in an additional case (1903, case 11).

spots.

location.

Idaho.—The spots first appear about the ankles and wrists, and by the end of the first week they have extended over the entire body. (Bowers, 1896, p. 63.) According to Collister (1896, p. 63) they first appear on the feet and from there spread over the body. Dubois (1896, p. 64) states that they first appear on the palms of the hands and extend from there over the body. Fairchild (1896) reports that they first are seen on the legs and arms, and soon cover the whole body. Fairchild (1896) says

that the eruption, in many cases, invades the eyes, making them very sensitive to light. According to Figgins (1896, p. 64) the spots first affect the face and hands, then the trunk, and, finally, the extremities. Maxey (1899, p. 436) states that the eruption first shows itself on the wrists and ankles, next on the forehead and chest, and in 24 to 36 hours spreads over the entire surface of the body; even the palms of the hands, soles of the feet, and the scalp are spotted.

MONTANA.—The spots are first discernible on the back, requiring about 24 hours to appear over the remainder of the body. (Gwinn, 1892.) McCullough says that they first appear more frequently upon the back, or simultaneously with ankles or wrists, and extend over the entire body.

According to Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 38) the eruption appears first about the wrist and ankles or back. It then extends over the entire body, the abdomen usually being last involved. Sometimes it spreads very rapidly, the entire surface being covered in 12 hours, but more usually one or two days pass before it reaches the maximum. The scalp, palms of the hands, and soles of the feet are frequently covered with the rash. Occasionally, though perhaps rarely, purplish spots are found on the mucous membrane of the inside of the cheeks.

Anderson (1903c, pp. 22, 38, 39) states that the spots appear first on the wrists and ankles, then on the arms, legs, forehead, back, chest, and, last and least, on the abdomen. They are never abundant on the abdomen, but other portions of the body are in some cases literally covered by the eruption. They are most abundant on the wrists, ankles, arms, and back (post-mortem). The abundance of the spots apparently bears no relation to the severity of the attack.

Gates (1903, p. 49) records a case where the spots first appeared on the buttocks, back, and thighs; the face was only slightly affected; and another case in which the spots appeared on the forehead, back of hands, wrists, and ankles. Later he (1905, p. 111) reports for his case 11 that the spots first appeared on the palms of the hands and on the feet, and they invaded the scalp in this case. In case 15 the spots appeared first on feet and hands. In case 16 they appeared first on feet, ankles, hands, and wrists.

TIME OF APPEARANCE.

Idaho.—The eruption appears during the first week about the fourth or fifth day of the attack (Bowers, 1896, p. 65) and matures during the second week. Accordi g to Dubois (1896, p. 64) it appears early, within 48 to 72 hours. Fairchild (1896) reports its appearance about the third or fourth day; Figgins (1896, p. 64) from the third to the fifth day; Springer (1896, p. 61) from the second to the fifth day; Sweet (1896) says the eruption appears within 1 to 3 days; Maxey (1899, p. 436) says the spots appear on the third to the seventh day of the fever.

Montana.—The spots usually appear about 3 days after the beginning of the sickness, but a few cases show no eruption until late, or a few hours before death (Gwinn, 1902). According to McCullough (1902, p. 226) they usually appear upon the third to the sixth day. Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37–38) give more exact statistics on this subject; in their last summary (1904a) they say that the eruption usually begins from the second to the fifth day after the chill; in 126 cases collated, the eruption appeared on the second day in 11 cases, on the third in 65, on the fourth in 22, on the fifth in 7, on the sixth in 3, on the seventh in 2, on the eighth in 2 (both doubtful), on the ninth in 2, and on unknown dates in 12 cases. Anderson (1903a, p. 507; 1903c, p. 22) says that the spots usually appear on the third day.

Gates (1905, p. 114) reports the eruption as appearing on the second or third day in 1 case, third day in 1 case, third or fourth day in 1 case, fourth day in 5 cases, fifth day in 3 cases, sixth day in 1 case; no record in 5 cases.

In the Bitter Root Valley cases of 1904 the spots were first noticed 2 days after the first chill in 1 case, 2 days after initial fever in 1 case, third day after first chill in 1 case, fourth day after chill in 3 cases, fourth day after initial symptoms in 1 case, record incomplete in remaining cases.

DURATION.

Idaho.—Bowers (1896) says that the spots reach the stage of absorption and desquamation in 8 to 21 days. They disappear slowly during the period of absorption, which is very variable—a few weeks to several months. After Collister (1896, p. 63), they can be seen for weeks, sometimes for months, after recovery, especially if the patient becomes a little chilly. Dubois (1896, p. 64) says that they fade slowly and may be discerned weeks after convalescence has set in. Fairchild (1896) reports that the eruption continues throughout the disease or until convalescence is fairly established, when it gradually fades. When exposed to cold the remains of the spots can be seen for months after complete recovery. Springer (1896, p. 61) agrees that the eruption continues throughout the attack. It is faintly visible often for many weeks, and is visible even after convalescence, especially if the surface becomes chilled; there is considerable irregularity regarding its disappearance. After Zipf (1896, p. 65), the spots stay out even after the patient feels well again. Maxey (1899, p. 436) says that as the fever declines the spots fade, but any temporary exacerbation of fever or free perspiration will freshen them temporarily, and a characteristic feature of this disease is that for several weeks after the fever has subsided the spots will show under the skin after a warm bath or active exercise.

Montana.—The spots fade as the fever subsides, but may not entirely disappear for weeks or months after convalescence is established (Wilson and Chowning, 1902a, p. 32; 1903a, p. 63; 1904a, p. 38). Anderson (1903a, p. 507; 1903c, p. 23) says that the spots fade as the fever declines, but show distinctly on slight return of fever or free perspiration. Warm baths produced spots 10 months after recovery.

Gates (1905, p. 115) reports that the spots were visible 4 months after recovery in his case 2 (1898), and in case 15 (1904) many spots were still visible 6 weeks after the temperature reached normal.

CHARACTER.

Idaho.—According to Bowers (1896, p. 63) the spots are due to a sanguineous exudate, probably into or beneath the corium; they have a characteristic red color; on their first appearance they are one-eighth to one-fourth inch in diameter, and disappear momentarily on pressure; in 3 or 4 days they enlarge one-fourth to one-third inch in diameter, become papular, and are modified only slightly on pressure. In fatal cases they turn dark purple before death. Collister (1896, p. 63) describes the eruption as flat, rosy-red papules about one-sixth inch in diameter; when they are pressed upon, especially soon after they have formed, their color disappears, but later they do not disappear on pressure. Dubois (1896, p. 64) refers to the spots as fine, red, round papules of an erythematous nature. "The term exanthesis rosalia arthrodynia more nearly expresses the disease than spotted fever." Fairchild (1896) describes the eruption as hemorrhagic in appearance; the spots vary in size from one-eighth to one-half an inch in diameter; sometimes they coalesce, covering a large surface; they do not disappear on pressure. Figgins (1896, p. 64) refers to the spots as raised, first of light-red color, assuming a dark-purple hue as the disease progresses; eruptive patches one-fourth to 1 inch in diameter, mostly of circular form. According to Springer (1896, p. 62) the eruption does not fade on pressure and in many cases there are large areas of almost black (hemorrhagic) skin, especially in

front of the tibia; the spots vary in size, averaging about one-eighth inch in diameter; they appear over the entire body, are papular, evidently extravasation of blood beneath the skin, fading to a faint blue in 4 to 30 days; they often remain for months as faint blue indurated spots on exposure to cold. According to Zipf (1896, p. 65) the spots are red in the center and blue at the margin. Maxey (1899, p. 436) states that on first appearance the spots are a bright rose color, round, unelevated, and vary in size from that of a pin head to that of a split pea; on pressure they disappear, but return quickly when released from pressure; they may or may not be tender; pathologically the eruption appears to be an extravasation of blood into the deeper layers of the skin.

Montana.—The eruption has the color and at a distance appears very much like measles, but it is unlike the latter in being macular; when first out the macules almost disappear on pressure; they are often situated at a hair follicle, and they vary in size—when the eruption is new, from a pin head up to a split pea; gradually the macules become darker, assuming a purple or dark-blue color, becoming harder and harder to eliminate by pressure; they increase in size so that many of them become confluent in places, producing a mottled appearance; after they become dark they have exactly the appearance of a hemorrhage under the skin; but whether or not they are a true diapedesis Gwinn (1902) is unable to say. McCullough (1902, p. 226) refers to the resemblance to a turkey egg, caused by the spots; the spots also vary and "may be seen in the form of a petechia extending to a decided ecchymosis upon an area of the body the hand would cover;" the macules partially disappear under pressure early in the eruptive stage, but later they become permanent and take on a darker hue, losing the pink tinge that predominates when the eruption first appears.

According to Wilson and Chowning (1902a, p. 132; 1903a, pp. 62–63; 1904a, p. 38) the macules are at first rose colored and consist of circular spots, varying in size from 1 to 5 mm. in diameter; they are not elevated; at first they disappear on pressure, but quickly reappear; they are sometimes tender to the touch; the appearance ordinarily rapidly changes, the macules becoming permanent, assuming a dark-blue or purplish color, and increasing in size until by confluence a mottled or marbled appearance may be given to the skin, especially on the dependent portions; in some cases the marbling covers the entire body; the color now no longer disappears on pressure; in some cases the eruption at no time becomes confluent and only small brownish or purplish petechiæ may be present, giving a speckled appearance which has been likened to that of a turkey's egg.

Anderson (1903a, p. 507; 1903c, p. 22) says that at first the spots are of a bright red color, macular at all times, from a pin point to a split pea in size; at first they disappear readily on pressure and return quickly, but if the case is a severe one they soon become darker, and in some cases are almost purple; from about the sixth to the tenth day of the disease they fail to disappear on pressure and are distinctly petechial in character; in favorable cases, about the fourteenth day they begin to lose their petechial character and disappear slowly on pressure; in some cases the eruption consists of small, brownish spots, giving a turkey egg appearance; Koplik's spots are not present (Anderson, 1903c, p. 36). Gates (1903, p. 49) in describing a case says that the petechiæ increased in size and number very rapidly during the first 2 weeks, forming large, irregularly shaped spots from the size of a little finger nail to spots one-half by one-half inch in diameter; they darkened in color, becoming bluish, with surrounding yellow tinge; they were slow in disappearing, some trace being visible 7 months after recovery.

In 1904 the skin of case 1 was mottled. In case 2 the spots were not sore to the touch. In case 3 they first appeared on the arms. In case 4 the eruption covered the legs to the knees; on the 14th it was reported as elevated and extended over trunk to face and arms. In

case 6 the spots were on the legs and trunk when patient was first seen by physician. In case 7 they were very slow and backward in appearance, in fact, and never became very pronounced. In case 8 no spots were noticed at 10 a. m. May 21 (fourth day), but at 2 p. m. they appeared quite distinct on wrist and chest; at 5 p. m. all spots disappeared; the skin then became decidedly mottled; on May 24 spots appeared in decided manner over entire back, arms, and chest. In case 9 spots were first noticed on the ankles. In case 10 first symptoms were noticed on June 2, first spots on June 6. There was profuse perspiration on May 13 in case 12, a few days after tick bite the "arm had swelled up as large as two arms and was red as a beet," the spots disappeared from the face, forehead, arms, body, and legs in the order named.

PATHOLOGICAL FINDINGS.

Wilson and Chowning (1904a, p. 42), reporting upon the pathological findings, say that the skin over all dependent portions of the body presented a marbled appearance; over nondependent portions it was covered with petechiæ; in all cases small wounds of the skin due to tick bites were present; the capillaries of the skin are distended with blood, which contains an excess of leukocytes; many of the red cells have escaped from the vessels into the surrounding tissues; in some cases blood-pigment granules are present in old extravasations; in a few cases phagocytes, containing infected red cells, are present, but not so many infected cells are found as are present in the spleen, kidney, and liver. Anderson (1903c, p. 38) speaks of the congested capillaries and minute extravasations in the rete extending into the stratum mucosum.

COMPARISON.

The spotted condition of the skin is one of the most striking clinical features of so-called "spotted fever," although it is maintained by some physicians that a mild, nonfatal type of the disease occurs in which these spots are not present.

In the articles referred to as summarizing piroplasmatic diseases no mention seems to be made of exactly similar regular conditions in cattle, sheep, and dogs. Starcovici, however, refers to a "jelly-like, hemorrhagic, subcutaneous edema" in hemoglobinuria of cattle and "a yellow jelly-like edema, here and there under the skin" in carceag of sheep. Smith reports, for Texas fever, that "the skin presents nothing abnormal to the unaided eye. * * * In one case the hair on the abdomen and the inner aspect of the thighs was matted into little tufts by dried blood; the skin showed at such places a bluish elevated spot, and when incised a little blood was found in the subcutis; this may be what has been called blood sweating."

If "spotted fever" is a piroplasmosis, we would therefore seem to have in man a skin lesion which marks this disease as quite different, clinically, from similar diseases in other animals.

DESQUAMATION.

Idaho.—A browny desquamation is usual about the third week; subjects that die at this stage present a resemblance to smallpox, due to the loss of epidermis from the apices of the papules (Bowers, 1896, p. 63). Figgins (1896) gives the third or

fourth week as the time of desquamation. According to Maxey (1899, p. 436) the desquamation during convalescence is somewhat peculiar, and can best be studied on the feet; the spots seem to cause a circumscribed death of the skin. A short time ago he examined Mr. D., one of his spotted-fever cases, who was taken sick 2 months previously; patient was sick 18 or 20 days, and made a rapid and perfect recovery. It was a warm day; he had taken a brisk walk just before coming to the office, and when stripped for examination spots could be dimly seen in the skin on the hands and feet, but none on the body. On the soles of the feet were many round, white areas of dead epidermis, corresponding to the former location of the spots, and when this dead epidermis was removed it gave to the thick skin of the sole a punched-out appearance. Over the body desquamation is hardly noticeable, but it does occur.

Montana.—McCullough (1902, p. 227) reports a peeling of the eruptive spots upon patients who recover. According to Wilson and Chowning (1902a, p. 132; 1903a, p. 63; 1904a, p. 38), desquamation begins about the third week and extends over the whole body, but is slight except over the most affected areas. Anderson (1903c, p. 23) agrees that when convalescence is well advanced desquamation begins and extends over the entire body.

JAUNDICE.

In addition to the eruption the skin takes on a congested, jaundiced appearance, well marked in the conjunctivæ (Gwinn, 1902). According to McCullough (1902, p. 226) jaundice accompanies the majority of cases, depicted over the entire body, and well marked upon the conjunctivæ. According to Wilson and Chowning (1902a, p. 132; 1903a, p. 63; 1904a, p. 38) the skin ordinarily shows some jaundice. Anderson (1903a, p. 507; 1903c, p. 23) states that the skin is always jaundiced to a greater or less degree, first noticed in the conjunctivæ. He reports (p. 32) the skin of case 120 as distinctly yellow; on post-mortem (p. 33) deeply jaundiced.

In 1904 jaundice was noticed to a greater or less extent in practically all the Bitter Root Valley cases. It was reported especially in case 9 for skin and conjunctivæ.

Comparison.—Jaundice of the skin is reported for Texas fever as "of rare occurrence;" in dogs the skin becomes "icteric, may become chrome yellow in color;" icterus was "very slight or not evident" in dogs which Nuttall studied; icterus was noticed "in the mucous membranes of the eye, mouth, and skin in 30 out of 63 cases observed" in France. Icterus is not reported by Starcovici for hemoglobinuria in cattle.

CYANOSIS.

The congested and cyanotic condition of the skin causes a bloated, stupid expression of the face in most cases, which is a very diagnostic symptom (Gwinn, 1902).

Case 11 (1904) showed cyanosis June 25–26 (seventh and eighth days).

GANGRENE.

Idaho.—Sloughing of the scrotum occurred in one case (Bowers, 1896, p. 63). Fairchild (1896) also reports occasional sloughing of limited areas, such as of the scrotum, etc.

Montana.—Gangrene is not an infrequent complication and may affect the fingers, toes, the region about the lobe of the ear, or in fact almost any part of the body; in one case the odor of the exhalations and sputum gave strong evidence of gangrene of the lungs (Gwinn, 1902). According to McCullough (1902, p. 226) gangrene frequently affects the toes, fingers, and dependent portions of the body. Wilson and

Chowning (1902a, p. 132; 1903a, p. 63; 1904a, p. 38) report that in some cases the skin becomes gangrenous over considerable areas, as on the elbows, fingers, toes, lobes of the ears, scrotum, etc. Anderson (1903a, p. 507; 1903c, p. 23) states that in very severe cases there may be gangrene of the fingers and toes, and still more frequently of the skin of the scrotum and penis; in case 120 the epidermis over scrotum sloughed off from an area about 2 to 5 centimeters in diameter (Anderson, 1903c, p. 33).

Gates (1905, p. 115) reports that in his case 6 (1900) the skin of the scrotum and legs became gangrenous before death, and Buckley noticed gangrene in case 1 (1904).

HYPERESTHESIA.

Idaho.—Bowers (1896, p. 64) reports hyperesthesia on the surface of the body.

Montana.—McCullough (1902, p. 227) says there is an exaggerated superficial hyperesthesia of the skin, as well as deep soreness, the patient fearing to be moved or touched.

In the cases observed in 1904, hyperesthesia was especially marked in Minshall's case (No. 8), and in Buckley's case (No. 2).

FAT.

See also Emaciation, page 74.

The panniculus adiposus was about normal in case 120 (Anderson, 1903c, p. 32).

In 1904, case 11, upon autopsy, showed a good amount of subcutaneous fat when incision was made from manubrium to pubis.

HEAD.

FACE.

Idaho.—Early in the disease the face acquires a dusky flush, and is slightly swollen; the expression becomes listless, dull, and heavy (Bowers, 1896, p. 63). Zipf (1896, p. 65) reports flushed face.

Montana.—The congested and cyanotic condition of the skin causes a bloated, stupid expression of the face in most cases, and this is a very diagnostic symptom (Gwinn, 1902). There is a glazed appearance of the face, bordering upon being copper colored, and taking a bluish or dusky hue as the disease advances.

In all severe cases more or less edema of the face and extremities is present. This may be marked, and may appear as early as the third day of the disease (Wilson and Chowning, 1902a, p. 133; 1903a, p. 64; 1904a, p. 40). There was considerable swelling of the legs and face the last day or two in cases 24 and 72 (Wilson and Chowning, 1903a, pp. 32, 37; Anderson, 1903c, pp. 13, 15); in cases 117 and 120 there was also more or less swelling of the face and limbs (Anderson, 1903c, pp. 27, 32).

Gates (1905, p. 111) reports the face of his case 14 as slightly flushed.

In 1904 the face of case 1 was reported as congested; of cases 8 and 10, flushed; of case 5, slightly flushed; of case 3, as placid, blotched with eruption, and then darkly flushed (May 13); of case 6, as florid; case 11, as dusky.

EARS.

Wilson and Chowning (1903a, p. 44) report ringing in the ears for case No. 109; the patient had been partially deaf for 2 or 3 days at the beginning of the sickness; case 89 (p. 49) experienced deafness (but had had quinine); deafness is also recorded for case 107. Anderson (1903c, p. 36) reports ringing in the ears in case 121.

EYES.

See also Jaundice, page 50.

Idaho.—The conjunctivæ are more or less injected (Bowers, 1896, p. 63). The eruption in many cases, invades the eyes causing severe pain, and making them very sensitive to light.

Montana.—Jaundice is well marked upon the conjunctivæ (McCullough, 1902, p. 226; Gwinn, 1902). According to Wilson and Chowning (1902a, p. 132; 1903a, p. 63; 1904a, p. 38) jaundice may be quite marked in the conjunctivæ, the vessels of which are injected from the outset. Anderson (1903a, p. 507; 1903c, p. 21) says that jaundice is first noticed in the conjunctivæ, the vessels of which are congested from the outset.

The pupils react normally to light and accommodation (Wilson and Chowning, 1902a, p. 133; 1904a, p. 38; Anderson, 1903c, p. 23). Gates (1905, p. 111) reports the conjunctivæ of case 11 congested; eyes of his case 14 as dull, conjunctivæ slightly congested; case 15, conjunctivæ congested; case 16, eyes much congested.

In 1904, the conjunctivæ were reported as injected in case 8, but as not injected in case 13; the eyes were reported as injected in cases 2 and 10; in case 2 they were quite bloodshot on May 8; they were normal in case 3 on May 10; in case 1 the sclerotics were much injected.

Case 2 complained of sore eyes early in the attack; there was no tenderness of the eyeballs in case 11, but some hours before death the pupils were widely dilated, despite the fact that the patient was receiving large doses of morphine.

An examination of the eyes of case 9 by Gwinn showed the media a little blurred, so that the granular appearance of the retina could not be seen; the larger retinal blood vessels were quite plain, however; there was no swelling or blurring of the disk; no hemorrhagic petechiæ in the retina.

In case 10 there was a slight convergent strabismus; on June 10, the mother states that last night patient cried out that his eyes were turning out; she examined, and saw that a divergent squint (outward rotation) of the left eye was present; this later disappeared; the eyes were suffused. The pupils in case 13 were reported as dilated and irresponsive to light. In case 8 the eyes reacted to light up to May 28.

PHOTOPHOBIA.

Idaho.—In many cases the patient is very sensitive to light (Fairchild, 1896). Dubois, (1896, p. 64) also reports photophobia. In one case two years elapsed before sunlight was borne without intense cephalalgia (Bowers, 1896, p. 63).

Montana.—Wilson and Chowning (1903a, p. 46) report photophobia for case 107. Gates (1905, p. 113) reports photophobia for his case 16 (1904).

In 1904 photophobia was more or less present in cases 2 to 11, except possibly case 8. It was especially marked in cases 2 and 10.

NOSE.

EPISTAXIS.

See also Hemorrhage, page 63.

Idaho.—Epistaxis occurs in some cases (Figgins, 1896, p. 64).

Montana.—Epistaxis is present (McCullough, 1902, p. 227) and not uncommon

(Gwinn, 1902). Two physicians have noted epistaxis; it was not observed in 1902, but was seen in 3 cases in 1903 (Wilson and Chowning, 1903a, p. 65; 1904a, p. 40). According to Anderson (1903c, pp. 21, 23, 32, 36–37), however, nosebleed is always present, usually from the end of the first week, and is sometimes quite severe; he reports it for cases Nos. 120 and 121.

In 1904, nosebleed was reported as not present in case 9; slight in case 6; frequent, but not excessive, during last five days in case 7.

MOUTH AND THROAT.

See also Cough, page 71.

Idaho.—Sore mouth and congested fauces are met with (Bowers, 1896, p. 64). Many cases suffer from congestion of the throat, which is very sensitive and painful and interferes with swallowing (Fairchild, 1896).

Montana.—The mouth is parched and dry (McCullough, 1902, p. 227); there was a bad taste in the mouth in case 89 (Wilson and Chowning, 1903a, p. 49); also in case 120 (Anderson, 1903c, p. 30). Gates (1903, p. 48) reports sore throat in one case. Wilson and Chowning (1903a, p. 49) report intense thirst in case 89.

In 1904, the lips were dry, blood stained, and crusted in cases 2 and 7; sordes were present on lips and tongue in case 11.

There was no eruption in the mouth or throat in cases 3, 9, and 10.

In case 3 the fauces and pharynx were much injected and showed adherent mucus-pus.

Sore throat was not noticed in cases 7 and 10; in case 2 there was marked irritation in the throat, and it was very sore to the touch; in case 3 the throat was at one time very sore, but it improved later; in case 5 the throat was slightly sore, but showed no marked injection; in case 5 sore throat was an early symptom; in case 8 the throat was slightly sore.

In case 2 there was marked huskiness of voice.

In cases 2 and 8 there was intense thirst.

Comparisons.—The pharynx and larynx are hyperemic in hemoglobinuria, and the mucosa is swollen in carceag.

BREATH.

Gwinn (1902) states that in one case the odor of the breath gave evidence of gangrene of the lungs. Anderson (1903c, p. 29) reports a peculiar sweetish odor to the breath in case 120.

In 1904, case 3 presented an unusually offensive breath, which during the last stages became absolutely nauseating; the breath of this patient during health was said to be very offensive. Cases 9 and 11 presented a more or less urinous odor to the breath.

TONGUE.

Idaho.—Bowers (1896, p. 64) reports the tongue as thick and furred. Collister (1896, p. 63) says that it is usually covered with a whitish or yellowish fur, but in severe typhoidal cases is dry, red, and glazed. Fairchild (1896) describes it as coated by a whitish fur with red edges early in the disease; later, the whitish coat usually disappears and the tongue becomes red and frequently dry and brown or black. Springer (1896, p. 62) says that the tongue has a yellowish-white coat with

red edges; in the later stages it becomes red, or dry and brown. According to Springer (1896) the tongue is always coated, a thin, white fur persisting for a long time. Zipf (1896, p. 65) also reports coated tongue. Maxey (1899, p. 435) reports the tongue as at first covered with a whitish coating, but about the time the fever reaches its maximum the tongue changes to a dry, brownish condition, which continues until the general symptoms begin to ameliorate, when the tongue clears in an irregular manner, beginning at the base.

Montana.—According to Gwinn (1902) the tongue at first has a white coat, but it soon becomes brown and is accompanied by sordes. Wilson and Chowning (1903a, pp. 44, 46, 62; 1904a, p. 37) describe the tongue as dry with heavy white or yellowish coat in the middle, but red at the tip and along the edges; it may be thickly coated even at the onset (1902a, p. 132); while the coat is whitish at first, it becomes brownish as the fever progresses, and the tongue may become dry and cracked (1902a, p. 132). Anderson (1903a, p. 21, 23, 29, 31) agrees that at first the tongue has a heavy, whitish coat with red edge and tip; later it becomes dark brown; in case 117 it was coated throughout the disease; in case 120 it was furred on April 29, and on May 6 it showed a heavy white coat with red tip and margins; in case 121 it had a heavy white coat in the center with red tip and edges. Gates (1903, p. 49) says in regard to one case when first seen that the tongue was coated white on the sides and was rather dry.

In 1904 the tongue was moist in case 1; tremulous and with a heavy white moist coat in case 3; it had a heavy white moist coat in cases 5 and 10; in case 8 there was a heavy white coat, which became dry and brown; in case 7 there was a heavy moist coat which became dry, brown, and somewhat glazed; in case 9 the tongue was heavily coated; in case 11 it was red and moist, with white streaks; in case 13 the tongue was furred.

Gates (1905, p. 111) reports that in his case (11) the tongue quivered very much when extruded, and it was loaded with a heavy dirty-brown coat, the sides of the tongue were so livid as to be almost blue; his case (14) showed a grayish coat on the tongue; in his case (16) the tongue became very dry early in the disease.

TEETH.

Gwinn (1902) and McCullough (1902, p. 227) say that sordes are present. Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37) state that sordes appear early and may be quite pronounced. Anderson (1903c, p. 23) also agrees that the teeth may be covered with sordes.

NECK.

In case 109 there was no stiffness of the muscles of the neck and back, though some pain was present on pressure over the spinal process of the vertebrae, especially in the dorsal lumbar region. (Wilson and Chowning, 1903a, p. 45.)

In case 115 the postcervical glands were enlarged, particularly on the right side; in case 116 the axillary glands were swollen and sore the day after the tick bite; in case 120 the mesenteric and retroperitoneal glands were pale, but not enlarged (autopsy).—Anderson, 1903c, pp. 24, 25, 33.

In 1904, case 9 showed no stiffness of the neck, and the head could be readily bent forward; in case 6 there was general rigidity of the

body on May 20; in case 7 there was cervical tenderness on first day of illness; case 11 had cervical tenderness for several days.

In cases 3 and 10 the cervical glands were somewhat enlarged.

ABDOMEN.

There was considerable gurgling and tenderness in the right iliac fossa in case 120 (Anderson, 1903c, p. 31); tympanites is never excessive (1903c, p. 23). Anderson (1903c, p. 120) reports the mesenteric and retroperitoneal glands as pale and not enlarged. Abdominal tympanites usually appears 1 or 2 hours before death. (Wilson and Chowning, 1904a, p. 40.) Gates (1905, p. 111) reports tympanites as moderate in his nonfatal case 11 on the 10th day.

In 1904, the abdomen was not distended or painful in case 3 (May 10, 1904); it was generally tender, especially on right side in case 8 (May 26, 1904), and was painful and tender in case 10.

Autopsy of case 11 (1904) showed the abdominal organs in normal relation and position.

PERITONEUM.

In case 11 (1904), the peritoneum was normal; there was a considerable amount of straw-colored material present in peritoneal cavity.

COMPARISONS.—In hemoglobinuria the peritoneum near the duodenum is always yellowish and jelly-like, swollen and ecchymotic.

OMENTUM.

In case 97 the omentum was somewhat discolored, showing postmortem degeneration; in case 94 it was slightly hemorrhagic. (Wilson and Chowning, 1903a, pp. 56–58.)

See also page 68.

EXTREMITIES.

Idaho.—Bowers (1896, p. 63) speaks of swollen joints, the joint lesions developing with the spots; the swelling disappears during the period of absorption and convalescence.

In 1904 the knee jerks were normal in case 10; patellar reflex was exaggerated and ankle clonus pronounced in case 13.

In case 2 the finger nails turned purple about half an hour before death.

GENITALIA.

See also page 50.

Bowers (1896, p. 63), reports that the scrotum and testicles are swollen in severe cases. Fairchild (1896) and others say that sloughing occasionally takes place over limited areas, such as of scrotum, etc.

DIGESTIVE SYSTEM.

MOUTH, TONGUE, TEETH.

See pages 53–54.

APPETITE.

Idaho.—The loss of appetite is early and the relish for food is not regained until the patient is quite convalescent (Bowers, 1896, p. 63). Dubois (1896, p. 64) reports persistent anorexia. In many cases the appetite is lost (Fairchild, 1896). There is

first a loss of appetite, but after the first 5 or 6 days in many cases it returns and remains good throughout the attack (Maxey, 1899, p. 435).

Montana.—McCullough (1902, p. 227) reports an apathy for food and nourishment. Wilson and Chowning (1902a, p. 133; 1903a, p. 64; 1904a, p. 40) state that at the onset of the disease the appetite is usually good and food is well retained and assimilated. Anderson (1903c, p. 23) gives the appetite as often good throughout the first week. Gates (1903, p. 48) reports it as completely absent in one case when first seen; in another case food and medicine were taken well until the last 36 hours. He (1905, pp. 111–116) reports loss of appetite for cases 11, 14 (complete), and 15.

In 1904, appetite was fairly good in case 8 on May 22; in case 4 there was complete loss of appetite on May 13; appetite was absent in cases 5 and 7, in the latter case throughout the attack; case 6 had difficulty in swallowing (May 20).

Comparison.—For Texas fever, Smith and Kilborne (1893, p. 20) report that loss of appetite always, and cessation of rumination usually, accompany the high fever after the third or fourth day. Loss of appetite is also reported for hemoglobinuria in cattle. Nuttall (1904, pp. 232–233) records for canine piroplasmosis that loss of appetite is a constant and early symptom, observed in all cases upon which he has experimented. The dogs refuse all food in later stages; they may drink much water but refuse milk (Hutcheon, 1899). This symptom is also noted by Lounsbury and Robertson. In France loss of appetite is noted at onset in acute cases, and the appetite is not regained; anorexia is also observed in chronic cases.

STOMACH.

Idaho.—Springer (1896, p. 62) reports irritability and severe pains in the stomach. In some cases (Fairchild, 1896) it shows marked irritability.

Montana.—Anderson (1903c, p. 38), in his summary of lesions, reports the stomach as normal, but he says (p. 33) that in case 120 it was apparently normal, except hypostatic congestion over dorsal surface of fundus. Wilson and Chowning (1904a, p. 41) say that in some cases the dependent portions of the stomach were hyperemic.

In 1904 "stomach ache" was reported as absent in case 5 but present in case 7. "Cramps in stomach" were reported for case 2.

Upon autopsy, case 11 (1904) showed injection about cardiac end, otherwise it appeared normal.

See also page 57.

NAUSEA AND VOMITING.

Idaho.—Nausea is common, and vomiting an occasional symptom (Bowers, 1896, pp. 63–64). Collister (1896, p. 63) says that nausea and vomiting are present until the fourth or fifth day of the fever. Dubois (1896, p. 64) reports nausea as an early symptom.

Montana.—Vomiting may be present to a greater or less degree (McCullough, 1902, p. 227). About the beginning of the second week nausea and vomiting develop and continue in fatal cases to the end; in some cases nausea is present from the onset (Wilson and Chowning, 1903a, p. 64; 1904a, p. 40). According to Anderson (1903c, p. 23) there may be at first a little nausea, but the appetite is often good throughout the first week; in fatal cases nausea becomes more persistent during the second week and lasts until the end; he reports (1903c, pp. 16, 34) vomiting as an initial symptom in case 97, and nausea present in case 121. Gates (1903, p. 49) reports vomiting once in one case; he (1905, pp. 111) reports vomiting for his case 11, but there was no vomiting in his case 16, who took food well at all times.

In 1904, nausea was reported as present in case 7, absent in cases 5 and 8. Vomiting was noticed twice (May 5) in case 2; case 5 vomited practically everything taken; case 7 vomited throughout the attack, the vomit being greenish-yellow and containing blood (May 20), thought to be from nose; cases 10 and 11 vomited early in the attack.

INTESTINES.

Wilson and Chowning (1904a, p. 42) report the intestines as normal upon post-mortem, except slight hypostatic congestion in two cases, throughout their entire extent.

The stomach and intestine were normal in case 91. In case 93 the stomach was congested (hypostatic?) over dorsal portion; intestine normal. In case 97 intestine was slightly discolored and distended by gas. In case 107 there was nothing abnormal except that the colon was distended by gas. In case 89 there was nothing abnormal except a slight congestion (probably hypostatic) in upper portion of the jejunum. In case 94 nothing abnormal except intestine distended with gas (Wilson and Chowning, 1903a, pp. 48, 51, 54, 56, 58). In case 120 the small intestine was empty and showed no inflamation or congestion except hypostatic (Anderson, 1903c, p. 33).

None of the glands were enlarged in cases 89, 91, 93, 107; Peyer's patches pale and not congested in case 120.

The mesenteric vessels were congested in case 93.

In case 11 (1904) the upper portion of the intestine appeared normal; solitary glands and Peyer's patches appeared somewhat swollen; cecum showed considerable injection, which continued more or less throughout the colon.

Comparisons.—Smith and Kilborne (1893, p. 34) state regarding Texas fever that "The lesions of the intestines are limited to hyperæmia and pigmentation. Beginning with the duodenum, there is found generally an abundance of bile and more or less injection and pigmentation of the villi appearing in the form of closely set points and fine lines. The remainder of the small intestine may show with the stomach more or less marked congestion, or there may be patches marked by the injection of minute vessels. In many of the cases examined the mucosa was pale and concealed by a thin layer of a grayish pasty consistency made up largely of desquamated epithelium. * * * In the large intestine we find more or less hyperæmia and pigmentation in longitudinal lines corresponding to the summits of the folds of the mucous membrane. This condition is more marked in the cæcum and rectum than in the colon and seems to be associated with the constipated condition. Thus the cæcum is in some cases distended with very hard, dry, fecal balls and some may be found in the rectum. In some cases no abnormal condition of the large bowel is discoverable."

For hemoglobinuria of cattle Starcovici says that in the duodenum ecchymoses or small ulcers are constant; the mucosa of the small intestine is always much swollen and covered with a thick, yellowish jelly-like mass; the mucosa of the large intestine constantly shows ecchymoses and swelling. In carceag the rectum contains hard or soft slimy masses of manure mixed with blood; the mucosa regularly has extensive hemorrhages along the folds, and the bases of the folds are covered with a crumbling or pulpy, dirty brown scab.

In canine piroplasmosis there is a catarrhal inflammation of the small intestine, more intense about the duodenum; the lumen contains a viscid mucus often mixed with blood; the large intestines are slightly but not uniformly inflamed, and contain much viscid mucus (Hutcheon). Mucosa is infiltrated and congested on a level with the duodenum in a few cases.

CONSTIPATION; DIARRHEA.

Idaho.—The bowels, as a rule, are constipated and the abdomen retracted, but occasionally marked diarrhea occurs; there is no tympanites or tenderness of the abdomen (Fairchild, 1896). Bowers (1896, p. 64) reports constipation as usual during the entire illness. According to Figgins (1896, p. 64) the bowels are either constipated or quite loose; in some cases typhoid symptoms in malignant form are noticed. Springer (1896, p. 62) says that the bowels, as a rule, are constipated, and Sweet (1896, p. 61) agrees that constipation is usual, although diarrhea sometimes occurs. Maxey (1899, p. 435) states that the bowels remain constipated throughout the entire course of the disease.

Montana.—Most of Gwinn's cases (1896) were suffering more or less from indigestion and constipation at the time of the attack. According to Wilson and Chowning (1903a, p. 64; 1904a, p. 40) constipation is usually present from the beginning. According to Anderson (1903c, pp. 23, 29) constipation is present throughout the course of the attack; tympanites is never excessive; there is occasionally gurgling in the right iliac fossa. In case 119 improvement was interrupted by attack of acute indigestion. Constipation is reported for cases 118 (p. 27) and 121 (p. 36). Bowels were loose from the onset in case 97 (p. 16), and they were regular after initial constipation in case 117 (p. 27).

In one case (Gates, 1903, p. 49) a number of watery evacuations were produced by the action of elaterium. Gates (1905, pp. 111–112) reports constipation present in his cases 14 and 15; in case 14 the bowels became loose later.

In 1904 the bowels were at first normal in case 3, but after use of salt enemata they became loose (May 10); case 5 was at first slightly constipated, but the bowels afterwards became normal (May 18); in case 6 nothing abnormal was noticed; in case 7 they acted regularly and without assistance, there was no diarrhea; in case 10 they were loose; case 13 was constipated.

In two cases I examined microscopically for intestinal parasites, with negative results.

COMPARISONS.—In Texas fever the bowels are as a rule constipated during the high fever, and on post-mortem examination the large bowels (cecum and colon) are found in some cases compactly filled with small, very firm, hard balls of dung. As the fever subsides the feces again become softer and are then found more or less deeply tinged with bile. In hemoglobinuria of cattle there is colic, constipation, with hard feces surrounded by bloody mucus. In carceag there is colic, and hard, bloody stools.

LIVER.

Size.—Gwinn (1902) reports slight enlargement of the liver in one autopsy.

The liver is somewhat, though not markedly, enlarged (Wilson and Chowning, 1902a, p. 133); pain on pressure is absent (1904a, p. 40); pale in color and of normal consistency (1904a, p. 42); the capillaries are distended with blood containing an excess of leukocytes; many red cells contain parasites; the infected cells are frequently contained within phagocytes; there is acute parenchymatous hepatitis, with very marked fatty degeneration; some of the cases show considerable blood pigment.

Anderson (1903c, pp. 23, 31, 33, 36, 38) reports the liver as normal or usually slightly enlarged; pale, fatty in appearance; in parts areas are outlined by bile pigment; in some areas outlined by enlarged bile ducts; sections usually show fatty infiltration, bile capillaries full.

Wilson and Chowning (1903a, pp. 48, 51, 53, 54, 56, 58) report the liver as normal in size for case 94, rather swollen for case 97, appreciably enlarged for case 91, some-

what enlarged for 89, enlarged though not markedly so for case 93, noticeably enlarged for case 107. Anderson (1903c, p. 33) reports the liver as enlarged (weight 92.5 ounces) for case 120.

In 1904 the liver was not enlarged (on May 10) in case 3; in case 11, on post-mortem, it was apparently somewhat enlarged.

Comparison.—In Texas fever and hemoglobinuria the liver is reported as enlarged. In canine piroplasmosis the liver may be normal in size in acute cases or in some cases it may be enormously enlarged.

Color.—The liver was apparently normal in color in cases 94, 97; it was paler than normal in cases 89, 91, 107, and quite pale in case 93 (Wilson and Chowning, 1903a, pp. 48, 51, 53, 54, 56, 58); it was pale also in case 120 (Anderson, 1903c, p. 33).

In 1904, the liver was paler than normal in case 11, with yellowish tinge, apparently due to fat.

Comparisons.—In Texas fever the color of the surface is usually paler than in normal livers, and in most cases of a peculiar mottled appearance. The mottling is due to minute irregular grayish-yellow patches, usually 1 mm. or less in diameter. Starcovici reports the liver as pale and marbled in hemoglobinuria and pale in carceag. Nuttall reports, in reference to canine piroplasmosis, that the liver in an acute case was yellowish; it is usually congested, at times inflamed, and of mahogany or saffron color.

Section.—In case 97 the liver cut easily and was quite light in color on section; in case 107 it was of normal consistency and showed no congestion; in case 89 it showed no adhesions, and in cases 89, 91 it was of normal consistency and was not congested; it was normal in consistency in case 93 (Wilson and Chowning, 1903a, pp. 48, 51, 53, 54, 56, 58). In case 120 it was fatty in appearance, and in some areas outlined by engorged bile ducts (Anderson, 1903c, p. 33).

In 1904, section of the liver of case 11 was decidedly pale. What little blood flowed was also very pale; tissue was firm but apparently not fibroid.

Comparison.—In Texas fever, when incised, the parenchyma of the liver was remarkably bloodless in most cases, and a lac-colored thick blood poured from the cut ends of the larger hepatic veins; the color of the cut surface was either a uniformly brownish-yellow or else mottled as on the surface; the mottling, on closer scrutiny with the naked eye or hand lens, was found to be due to a paler yellowish discoloration of the zone bordering the intralobular veins; this zone of discoloration was the wider the more prolonged the disease, and in a few cases involved the entire lobule; parallel to this degenerative process the consistency of the organ became less resistant, more doughy, and brittle. In thin razor sections of fresh tissue the most striking phenomenon was the filling up of the ultimate bile canaliculi so that the hepatic cells were inclosed in polygons of yellow lines forming a beautiful network; when the liver is teased and crushed, the contents of these bile canaliculi may be found floating free in the form of rods, sometimes with Y-shaped ends; this stasis or filling up of the ultimate bile capillaries was present in nearly all animals examined; it was most pronounced in those whose death followed quickly after a high fever; in one case killed in the early days of the fever the liver was the seat of marked congestion, the bile stasis not having taken place yet. The extent of this stasis varies considerably. It may be seen in small isolated areas or else it may involve a large continuous territory. Owing to absence of connective tissue between the lobules it is quite impossible in fresh sections to make out accurately its distribution. It seems to be most frequently met with in the innermost or hepatic zone of the lobule, but it may also be found involving the entire lobule.

Small bile ducts between the lobules are often found injected, and rarely lines of yellow injection may be visible to the unaided eye.

"Associated with the occlusion of the biliary canaliculi and ducts is a more or less extensive fatty degeneration of the hepatic cells. This is most advanced in prolonged cases of disease. In several which came under our observation the fatty changes were so extensive that cells free from large quantities of fat could not be seen. Among other abnormal appearances may be mentioned the presence of irregular yellow clumps of pigment in the hepatic cells and of stellate masses or blood-red needle-like crystals of very minute size. In one case large branched thrombi were found in some of the hepatic veins. * * * The injection of the bile canaliculi is seen only in Müller's fluid preparations or in alcoholic material cut directly without imbedding. The extent and location of the injection are variable. It may appear over an entire lobule or only a small portion of it. The fatty degeneration so regularly seen in fresh material shows itself in sections of hardened material in a peculiar vacuolated appearance of the cell protoplasm, the fat having been dissolved out. The vacuolation may be more pronounced near the center of the lobule, where the individual vacuoles may be as large as red corpuscles. Of these there may be several in a single cell, very little of the protoplasm remaining. The cell protoplasm of the peripheral zone of the lobule is uniformly vacuolated, the vacuoles being very small.

"Another change that is of considerable importance in estimating the pathological effect of the disease is a tendency toward necrosis of the inner zone of the lobule. This process, which shows itself to the naked eye as a faint paler mottling of the liver tissue limited to the inner zone of the acini, seems to begin around the central vein and extend toward the periphery. It is characterized by a degeneration and loss of the nuclei of the parenchyma cells." Smith and Kilborne, 1893, pp. 28–30.

Starcovici reports for hemoglobinuria that the center of the lobule is necrotic with gall stasis. In carceag the liver is friable; the finer changes of the liver consist in a collection of leukocytes, and the larger vessels show here and there gall stasis, and there is parenchymatic degeneration and fatty degeneration of liver cells, especially in the center of the lobes.

Nuttall reports for canine piroplasmosis that his cases showed but slight gross changes.

GALL BLADDER.

In case 107 the gall bladder and its contents appeared normal; in case 93 it was distended with fluid bile; in case 94 it contained 1.5 ounces of fluid of a dark yellow color, and the gall ducts were patulous (Wilson and Chowning, 1903a, pp. 48, 54, 58).

In case 11 (1904) the gall bladder was distended with fluid bile, the ducts were patulous; there were no gallstones.

COMPARISON.—In Texas fever the bile is found in the gall bladder in considerable quantity (one-half pint to a quart) after death. As might be anticipated from the description of the changes in the liver, this fluid is greatly altered. The usual limpid greenish fluid is replaced by an almost semisolid mass. As it flows from the incised bladder it has been aptly compared to chewed grass. The presence of mucus makes it cohesive enough to be drawn out into long flat bands as it flows. When it is allowed to stand quietly in a cylindrical vessel a layer of flakes settles down, which occupies not infrequently one-half of the entire column. The supernatant fluid is much darker than normal bile. The suspended matter appears to be made up chiefly of small yellowish flocculi or flakes. A deep-yellow tinge is imparted to all vessels and to the hands coming in contact with it. When examined under the microscope the suspended particles are resolved into amorphous yellowish masses mingled with bright golden points barely visible at 500 diameters. The common bile duct has

always been found pervious, and in many cases an abundance of bile is found in the small intestine (Smith and Kilborne, 1893, p. 31).

For bovine hemoglobinuria, Starcovici states that there is thick, dark bile in the gall bladder.

In canine piroplasmosis the bile is usually thick, sirupy, grumous or dark green, and distends the gall bladder.

Thus, if "spotted fever" is a piroplasmosis, the disease differs in its effects upon the gall of man from the effects shown in bovine and canine piroplasmosis.

PANCREAS.

Anderson (1903c, pp. 33, 38) states that the pancreas is about twice its normal weight; in case 120 it was normal in appearance, except enlargement (5 ounces).

In case 11 (1904) the pancreas was apparently normal.

CIRCULATORY SYSTEM.

HEART.

Upon autopsy, the epicardium usually contained a few petechial hemorrhages near the base of the left ventricle; pericardium was normal; the myocardium was softened; the right ventricle was filled with dark fluid blood, the left was almost empty or contained only a small clot; the capillaries of the heart are distended; there is not much extravasation of red cells, but considerable round-cell infiltration; all the cases show considerable parenchymatous degeneration; those cases in which round-cell infiltration is marked also show swelling of the muscle-fiber nuclei with fragmentation (Wilson and Chowning, 1904a, pp. 41, 42).

Pericardium.—The pericardium was normal in cases 107, 89, 91, 93, 97, and 94 (Wilson and Chowning, 1903a, pp. 48, 51, 52, 54, 56, 58); also in case 120 (Anderson, 1903c, p. 33).

The pericardial cavity contained an excess of fluid in cases 107 and 91 (Wilson and Chowning, 1903a, pp. 48, 51); fluid was not increased in cases 93 and 97 (1903a, pp. 53, 56), and about 2 ounces of fluid was present in case 120 (Anderson, 1903c, p. 33).

In case 11 (1904) there was an apparent excess of clear, straw-colored fluid.

Epicardium contained no hemorrhagic areas in case 107; in case 93 it showed on ventral surface of left ventricle several small hemorrhagic areas (Wilson and Chowning, 1903a, pp. 48, 54).

There were no ecchymotic areas over the surface of the heart in case 89; in cases 97 and 94 there were hemorrhagic areas over both ventricles; in case 91 there was one small ecchymotic spot in the right ventricle (Wilson and Chowning, 1903a, pp. 51, 53, 56, 58). In case 120, there were a few small hemorrhages over the left ventricle near the interventricular groove under the pericardium; small chicken-fat clots were found in the auricles (Anderson, 1903c, p. 33).

The heart was normal in size in cases 107, 97, and 94 (Wilson and Chowning, 1903a, pp. 48, 56, 58); it was somewhat dilated in case 89 (1903a, p. 51).

In case 11 (1904) the heart was distended with blood; in general, it seemed normal.

The muscle was normal or perhaps a trifle softened in case 107, apparently somewhat softened in cases 91, 93, and soft in case 97 (Wilson and Chowning, 1903a, pp. 48, 53, 54); much softened and pale in case 89, soft in case 97 (1903a, pp. 51, 56). In case 120 the myocardium of the right heart was somewhat pale and flabby (Anderson, 1903a, p. 33).

In case 11 (1904) the muscle was well nourished.

The right side contained dark-red blood in case 107, dark fluid blood in case 89; blood in cases 91 and 93 apparently darker than normal (Wilson and Chowning, 1903a, pp. 48, 51, 53, 54). In case 120 the right heart was half filled with blood (Anderson, 1903a, p. 33).

In case 11 (1904) the right side contained small clots, white and red.

The left heart was contracted and empty in cases 107 and 89; it was empty in case 91; there was small clot in left heart of case 93 (Wilson and Chowning, 1903a, pp. 48, 51, 53, 54).

In case 11 (1904) the left ventricle was partially contracted and contained chicken-fat and red clots.

In case 97 the valves were normal (Wilson and Chowning, 1903a, p. 56).

In case 11 (1904) the valves of both sides were apparently normal.

The endocardium was normal in case 94 (Wilson and Chowning, 1903a, p. 58) and 120 (Anderson, 1903c, p. 33).

Gates (1905, pp. 111–113) reports that heart sounds were normal in case 14; the heart action was weak in case 16 from first day and became very weak and irregular, with low arterial pressure.

In 1904 the heart sounds were clear and normal in cases 3, 5, 7, and 11.

Comparison.—In Texas fever at autopsy the right ventricle is always distended with blood, fluid or clotted, according to the time elapsing between death and the examination. The left ventricle is usually firmly contracted and may contain a small quantity of fluid or clotted blood. The clots are quite firm and very rarely mixed with firmer, pale-yellowish clots. A very constant lesion is the extravasation of blood beneath the epicardium and endocardium. This is mainly restricted to the left ventricle, although petechiæ are not infrequently met with on the right ventricle. On the external surface of the heart the petechiæ are usually grouped along the interventricular groove and near the base, although cases occur in which the whole ventricular surface is sprinkled over with them. The inner surface of the left ventricle shows larger patches of extravasation, usually on or at the base of the papillary muscles. On the large vessels at the base of the heart within the pericardial sac there are frequently very delicate shreds of tissue or patches in a hyperæmic condition. The heart muscle, on closer inspection, is observed to have its minute vessels markedly injected, and in fresh sections the capillary network is found densely packed with red corpuscles. In cases which have succumbed after the subsidence of the fever the heart muscle is quite pale. Cloudy and fatty changes of the fibers are in some cases quite marked, in others absent or restricted to a small number of fibers (Smith and Kilborne, 1893, p. 26).

In carcag the pericardium and pleura usually show abundant ecchymoses.

In canine piroplasmosis the pericardium contains a variable amount of serous fluid; ecchymoses are around the heart, largely in left ventricle (Hutcheon); in France it is reported that the pericardium contains yellow or bloody fluid; not infrequently one observes numerous petechiæ about the apex or beneath the endocardium of the left heart; the heart may be pale (acute case).

AORTA.

In case 11 (1904) the arch of the aorta seemed unusually small.

PULSE.

Idaho.—The pulse is more or less accelerated, often greatest at the beginning of convalescence; in the onset it is sluggish and lacks force; in fatal cases it is not usually greatly quickened; it may be slower than in health (Bowers, 1896, p. 63). Collister (1896, p. 63) says that it does not run very high, not often above 110 in adults. Fairchild (1896) reports it as usually slow and full, from 85 to 110. Springer (1896, p. 62) gives it as 100 to 130 in ordinary cases. Maxey (1899, p. 435) reports it from 80 to 120 per minute, at first full and bounding, later becoming soft, but not irregular.

Montana.—At first the pulse is full and strong, it gradually gains in rapidity and loses in strength and volume (Gwinn, 1902). According to McCullough (1902, p. 226) the pulse varies from 80 to 120 in typical cases, and lacks volume and regularity as the disease advances. Wilson and Chowning (1902a, p. 133; 1903a, p. 63; 1904a, p. 38) say that at the onset the pulse is usually full and strong, but gradually becomes more and more rapid while it loses in volume and strength, very much as in diphtheria; in fatal cases in adults it may reach 150 per minute some days before death; the rapidity of the pulse is sometimes out of all proportion to the temperature, as may be also the respiration.

Anderson (1903a, p. 507; 1903c, p. 22) reports that the pulse appears out of all proportion to the temperature, usually running from 110 to 140. A pulse of 120 is not unusual with a temperature of 102°; it is rather thready, though sometimes full and strong, occasionally dicrotic in the first week (1903c, p. 22). In case 120 (1903a, p. 29) the circulation was feeble on compressed areas and extremities.

Gates (1903c, pp. 48, 49) reports 186 as the highest pulse rate observed; one case at the end of the first week was almost pulseless; he reports (1905, p. 112) slow pulse and repeated chills as special features of his case 14.

Comparison.—In Texas fever the pulse and respiration rise with the fever. * * * As the fever subsides and recovery begins the great weakness of the animal still keeps the pulse very high for a time, especially when the animal is moved about or excited in any way. The respirations, on the other hand, are apt to fall below the normal in this same period. When death approaches the heart beats increase in number as they grow feebler, and the respirations fall with the body temperature below the normal. (Smith and Kilborne, 1893, p. 18.)

In canine piroplasmosis the pulse is weak and rapid; in acute cases it beats 120 to 160 a minute, is thready, and often intermittent.

HEMORRHAGE.

See also Epistaxis, page 52.

There is a marked tendency to hemorrhage—nose, throat, lungs, and bowels, each have been known to be affected, and the blood loses power of coagulation. (McCullough, 1902, p. 226.)

BLOOD.

Referring to their table, Wilson and Chowning (1904a, pp. 39, 40) conclude that the blood shows a marked reduction of red blood cells and hemoglobin, with a slight increase of leukocytes at times; the reduction of red cells is particularly marked just before death in fatal cases, and in recovering cases just before convalescence. Preparations taken from organs at autopsy, as well as those from the living patient, show a marked poikilocytosis and anemia. (Wilson and Chowning, 1903a, p. 67.)

None of the cases 1 to 11 (1904) were in a condition which would strike the observer as being anemic. A blood count might have shown some tendency to anemic condition, but from the general appearance of the patients such condition was not evident.

COMPARISONS.—In Texas fever, hemoglobinuria, carceag, and canine piroplasmosis, anemia is reported as present and in many cases as pronounced or intense. If "spotted fever" is a piroplasmosis, the action of the parasites in respect to anemia is far below the action of other members of the genus *Piroplasma*.

CONSISTENCY AND COLOR.

Gwinn (1902) took blood in five or six cases from the arm, and in all of them it was found to be dark and thick, with the power of coagulation partly or entirely lost; it regained a bright scarlet color upon being shaken up with the air. These facts, taken together with the frequent eruption, the frequent complication of gangrene, and the fact that the whole system seems to be affected, naturally would lead one to suspect the blood to be the part mostly affected. He also speaks of "the really thick unoxygenated blood."

Wilson and Chowning (1902a, p. 133; 1903a, p. 64; 1904a, p. 38) state that when removed for examination the blood appears somewhat darker than normal, as well as somewhat less fluid; on exposure to air the color brightens perceptibly.

In the Bitter Root Valley cases of 1904 the dark, thickened condition of the blood was a very prominent symptom. Not infrequently the blood was so thick and flowed so slowly as to be of some inconvenience in making blood smears. In case 3, I cut into a blood vessel within fifteen minutes after death, and the blood was so thick that I had to add salt solution in order to draw the blood into a syringe. In case 11, the blood was so thick about 12 hours before death that the operation of bleeding the patient was performed with difficulty.

COMPARISON.—For Texas fever, Smith and Kilborne (1893, p. 21) describe the blood as follows: "Another character of this disease, the most constant and valuable of all and of which the hæmoglobinuria or "red water" is but a part, is the thinness of the blood. * * * Soon after the high temperature sets in the blood begins to grow thin, and after some days of fever it has become very pale and watery. * * * The difference between the drop of rich red blood issuing from a slight cut of the skin in healthy cattle and the thin, pale drop oozing from such a cut in Texas fever is very marked. This difference is due to the loss of red corpuscles which give the blood its characteristic color. Associated with this there may be in some cases a marked bloodlessness of the skin in the later stages. A number of small incisions are often required to obtain a few drops of blood. In some cases, shortly before death, the blood slowly trickles from a slight incision for some time before it is checked by the natural process of coagulation.

"When freshly drawn blood is allowed to stand the serum forced out of the clot has in the acute stage a very dark-red color, indicating the presence of much coloring matter in solution. As regards the coagulability, which some observers have regarded as feeble, we have no facts pointing in one direction. In a few cases the coagulation appeared retarded; in others it appeared to be normal in rapidity and effectiveness. As will seen further on, the condition of the blood must vary considerably from time to time. At one time it may contain the débris of destroyed corpuscles equal in number to one-tenth, or even one-fifth, of all circulating in the body. That under such circumstances its coagulability may be affected is evident. Frequently, however, the blood comes under observation when the destruction of red corpuscles has ceased, and the products have either been excreted or metamorphosed. In this way conflicting observations may perhaps be harmonized. In general, we may say that the coagulability of the blood is not much altered."

Starcovici reports the blood in hemoglobinuria as pale and lac-colored.

Nuttall (1904, pp. 235, 236, 237) reports the blood in canine piroplasmosis as profoundly altered, pale and watery, and with coagulation retarded; the serum is tinged with hemoglobin.

The difference in the blood in "spotted fever" on the one hand, and in piroplasmatic diseases on the other hand, is thus seen to be quite marked. In a piroplasmosis we naturally expect to find marked changes in the blood; but from our present knowledge it would appear that in Texas fever, hemoglobinuria, and canine piroplasmosis these changes are in one direction (the blood becoming thin, watery, and pale), while in "spotted fever" they are in the opposite direction (the blood becoming thick, molasses like, and dark). If now "spotted fever" be a true piroplasmosis, it would seem that a genus (*Piroplasma*) of protozoa has very different effects upon the blood of man from those noticed in cattle and dogs.

Viewed from the consistency of the blood, the condition noted in "spotted fever" can not at present be said to support the theory that this disease is a true piroplasmosis.

RED CELL COUNT.

Wilson and Chowning (1902a, p. 133; 1903a, p. 64) appear to be the first to give the red cell count in this disease; they found it to be 4,100,000, 4,200,000, 4,300,000, 4,400,000, and 4,500,000, respectively, in 5 cases examined; they call attention to the fact that these counts were made in an altitude of about 3,500 feet, where the normal count is above rather than under 5,500,000; they give a table of counts (1904a, p. 39) for cases 89, 94, 107, 115–120, 122–124. Anderson gives blood counts for several cases and concludes (1903a, p. 507; 1903c, p. 22) that there is a progressive decrease in red cells, but as soon as the temperature becomes normal an increase begins.

COMPARISON.—In Texas fever there is a tremendous decrease in the red cell count. Thus, Smith and Kilborne (1893, pp. 38 to 41) report a decrease in acute cases from 6,290,000, July 31, to 2,025,000, August 28; 7,171,000, August 13, to 1,675,000, August 29; 5,000,000, August 13, to 2,645,000, August 25.

In the mild nonfatal type the decrease is slower.

For canine piroplasmosis Nuttall (1904, pp. 238–239) reports the following cases: Typical acute case, 5,240,000 first day to 2,200,000 late the fifth day; typical chronic case, 5,840,000 first day to 1,200,000 late the fifteenth day.

LEUKOCYTES.

Wilson and Chowning (1902a, p. 133; 1903a, p. 64) report a slight increase of leukocytes—from 12,000 to 13,000 (or 14,000, see 1903a, p. 50)—in 4 cases examined; further counts are given in 1904a, p. 39.

Anderson (1903a, p. 507; 1903c, p. 22) says that the white cells are increased in number, varying from 8,000 to 12,000, the most interesting feature being an increase in the large mononuclears, which in an average of 2 cases gave 11.4 per cent.

Polymorphonuclear leukocytes	77.7
Large mononuclear leukocytes	11.4
Small lymphocytes	10.0
Eosinophiles	.9
	100.0

In case 11 (1904) the leukocytes ran up to 15,600 (Ashburn). It is a point of some interest that the increase in large mononuclears, as reported by Anderson, is in harmony with protozoan infection in so far that in malaria and kala-azar there is also an increase in large mononuclears, but in canine piroplasmosis it is the polynuclears which are increased.

COMPARISONS.—For Texas fever Smith and Kilborne (1893, p. 50) say: "Any unusual increase in numbers was not noted in stained preparations of any case which came under observation. In some cases an abnormal crowding together of leukocytes was observed in dried preparations, which crowding must be regarded as having existed within the blood vessels, for there was no time for any massing together after the blood had left the vessels."

In canine piroplasmosis there may be considerable leukocytosis, the number of leukocytes being increased 2, 3, or 4 times the normal, so that instead of having 7,000 to 8,000 (normal), as many as 40,000 may be counted; the multiplication almost entirely affects the polynuclear elements, this being especially marked in slow-running cases (Nuttall, 1904, p. 238).

HEMOGLOBIN

In 5 cases examined during various stages of the disease, the hemoglobin was 50 to 60 per cent; one child of 12 years, examined 2 months after convalescence, showed Hb. 80 per cent (Wilson and Chowning, 1902a, p. 133; 1903a, p. 64; 1904a, p. 40). Anderson (1903a, p. 507; 1903c, p. 22) reports a steady but never very rapid decrease in the percentage of hemoglobin, one case going as low as 50 per cent.

COMPARISON.—For canine piroplasmosis (Nuttall, 1904, p. 238) there is a great fall in the percentage of hemoglobin, namely, to 13, 12, 6.4, or even to 3.5 per cent.

WIDAL TEST.

The Widal reaction with *Bacillus typhosus* is not present. (Wilson and Chowning, 1902a, p. 133; 1903a, p. 64; 1904a, p. 40; Anderson, 1903a, p. 507; 1903c, pp. 22, 33, 37.)

PARASITES.

See also page 19.

Freshly drawn blood from patients during their illness when examined with a one-twelfth oil immersion objective shows parasites sparingly in the red blood cells (Wilson and Chowning, 1902a, p. 133; 1903a, p. 64; 1904a, p. 40). Blood was examined from 3 recovered cases, 1 of 2 months, 1 of 1 year, and 1 of 2 years without finding the hematozoon (1903a, p. 64; 1904a, p. 40). Case 115, examined 14 days after patient had been discharged by physician, still showed parasites in the blood (1904a, p. 40).

Anderson (1903a, 1903c) also reports the parasites in the blood.

Ashburn and I have failed to confirm these observations; see pages 19.

COMPARISON.—It will be recalled that Wilson and Chowning state that in "spotted fever" probably not over 0.2 per cent of the red cells in the circulating blood are infected, but the parasites are more common in the spleen and in certain other portions of the body.

For Texas fever, Smith and Kilborne (1893, pp. 61–65) state that the numbers of infected corpuscles circulating in the blood during the high fever is usually quite small; 0.1 to 1 per cent would be a fair estimate in most cases. Toward the fatal termination there may be from 5 to 10 per cent of the corpuscles with the pyriform

parasites present. Larger numbers of parasites are found within corpuscles in the capillary blood of congested areas, as is seen by the following table of a case in which 2 to 3 per cent of the circulating corpuscles were infected before the cow was killed:

"In blood from skeletal muscles, very few infected corpuscles.
"In blood from the right heart, very few infected corpuscles.
"In blood from marrow of sixth rib, very few infected corpuscles.
"In blood from the left heart, 2 to 3 per cent infected corpuscles.
"In blood from lung tissue, 2 to 3 per cent infected corpuscles.
"In liver tissue, 10 to 20 per cent infected corpuscles.
"In kidney tissue, 10 to 20 per cent infected corpuscles.
"In hyperæmic fringes of omentum, 50 per cent infected corpuscles.
"In heart muscle, 50 per cent and many free parasites."—Smith and Kilborne, 1893, page 62.

In some cases the liver blood was infected to 40 or 50 per cent, and kidney blood to 80 or 90 per cent.

Starcovici reports for hemoglobinuria that 90 per cent of the corpuscles in the kidneys may show infection, but fewer infected corpuscles are found in the circulating blood. In carceag the blood in the spleen and in the hemorrhagic edema may be infected to 5 or 10 per cent. In the larger vessels scarcely 1 per cent.

For canine piroplasmosis, Nuttall (1904, p. 228) reports that the parasites occur in the blood throughout the body, being most numerous in the internal organs.

SPLEEN.

The spleen is uniformly enlarged and tender on palpation (Wilson and Chowning, 1902a, p. 133; 1903a, p. 64; 1904a, p. 40). It was 3 to 3½ times its normal weight; the capsule was distended and thinned; on section the tissue was found dark red and so soft as to be in most cases confluent; the outlines of the Malpighian bodies were obliterated; the omentum covering the spleen was usually congested (Wilson and Chowning, 1904a, p. 41). The spleen shows an engorgement with red blood cells and leukocytes; the outline of the Malpighian bodies are lost; there is a marked infiltration of leukocytes, mostly of the polynuclear type, in the region of the Malpighian bodies; there is much blood pigment, both free and within phagocytes; many piroplasmata are present, both free and within red cells, many of which have been taken up by phagocytes.

According to Anderson (1903c, p. 23), the spleen is enlarged early and may extend 1 or 2 inches below the costal margin; on post-mortem (1903c, p. 38) it is usually purple in color, soft, diffluent, and from 3 to 4 times its normal weight; the vessels are engorged with blood; many mononuclears are present containing from 1 to 4 red blood cells; there is no free pigment. In case 35 (1903c, p. 13) the spleen was much increased in size, and this was the only abnormal appearance at post-mortem; in case 121 (1903c, pp. 35–36) it was enlarged and easily palpable 1 inch below costal margin.

Comparison.—In Texas fever the grayish Malpighian bodies and the whitish trabeculæ have all disappeared from view within the distended pulp; a microscopic examination shows that the enlargement and peculiar color of the spleen tissue is due to an engorgement with red blood corpuscles. With this engorgement there may be associated a variable number of large cells containing coarse granules and from 2 to 12 red corpuscles, or else the remains of these corpuscles in the form of irregular clumps of yellowish pigment. The pigment is also free in masses of variable size. Examination of fresh pulp from spleens of healthy cattle shows that the presence of large quantities of free pigment of the form described is not uncommon. (Smith and Kilborne, 1893, page 28.)

In carceag the follicles are usually indistinct and the spleen is hyperemic.

In hemoglobinuria of cattle the follicles are seldom distinct.

Size.—The size and weight of the spleen on post-mortem have been reported as follows:

7 ounces after 4 hours in paper, case No. 107, Wilson and Chowning, 1903a, page 48.

9 ounces, case No. 94, Wilson and Chowning, 1903a, page 58.

17 ounces, after 12 hours in paper, case No. 3, Wilson and Chowning, 1903a, page 54.

22 ounces, after 8 hours in paper, case No. 91, Wilson and Chowning, 1903a, page 53.

25 ounces, after 12 hours in paper, case No. 107, Wilson and Chowning, 1903a, page 48.

3 times normal weight, case No. 97, Wilson and Chowning, 1903a, page 56.

20 ounces, 1 hour after removal, case No. 120, Anderson, 1903c, page 33.

In cases of 1904, it was enlarged in Nos. 3, 7, 10, and 11; dullness was increased in case 5, but not obtainable in case 8.

Comparison.—The spleen is enlarged in Texas fever (very much enlarged, hence the name splenic fever), in hemoglobinuria in cattle, and in carceag in sheep, and is often 3 to 4 times natural size in canine piroplasmosis.

Color.—The spleen is reported as dark in cases 91, 94, 107, 120; very dark in cases 93, 97.

In 1904 the spleen of case No. 11 on post-mortem was of a slaty purple in color.

Comparison.—In Texas fever the spleen is reported as dark brownish-red, dark in hemoglobinuria of cattle; in canine piroplasmosis it is reported as "pale, bloodless, like other organs; scarcely stains paper when smeared thereon" (Robertson); Nuttall observed little change; in France it is dark.

Capsule.—The capsule of the spleen is reported as stretched and thin in cases 89, 91, 93, 107; it stripped easily in cases 94, 97.

Comparison.—In Texas fever the ordinarily rather thick whitish capsule is very much distended and attenuated, so that the dark pulp shows through it very distinctly.

Pulp.—The spleen pulp is reported as soft and diffluent in cases 89, 91, 93, 107, 120; almost fluid and deep red in case 97; decidedly diffluent, of deep yellowish-red color, in case 94.

In case 11 of 1904, the spleen was soft and easily torn.

Comparison.—In Texas fever the pulp may be firm or it may be partly diffluent, welling out as a semifluid mass from the incised retracting capsule. It is reported as soft in carceag and canine piroplasmosis (France).

Adhesions. In case 93 (of 1902), the spleen was adherent to the gut. In case 11 of 1904, it was bound down by posterior adhesions, and adhesions to stomach.

In case 107 the portion of the omentum covering the spleen was darkened and apparently disintegrating; in case 89 it was dark, congested, and soft; in case 91 it was pale and apparently normal; in case 93 it did not differ from the omentum elsewhere (Wilson and Chowning, 1903a, pp. 48, 51, 54).

TEMPERATURE.

CHILL.

Idaho.—During the first week following the malaise the patient complains of chilly feelings (Bowers, 1896, p. 63). Some cases begin with a severe chill, and others with more or less chilly shudderings frequently referred to the spinal region; still

others with little or no chill (Collister, 1896, p. 63). The disease is sometimes ushered in by a chill (Fairchild, 1896, p. 62). Figgins (1896, p. 64) states that the attack begins with a chill. According to Springer (1896, p. 61), the chill follows the malaise. Maxey (1899, p. 435) states that the patient feels flashes of heat and cold, but no marked chill.

Montana.—The attack comes on by either a well-marked chill or by chilliness, simultaneous with fever; the chilliness, although most severe at the onset often continues more or less throughout the attack, coming on at intervals, generally mornings, and becoming lighter day after day until within a week or so it seems but little more than chilliness from light covering (Gwinn, 1902). According to McCullough (1902, p. 226), the onset may be marked by a sudden and severe chill or by slight chilly sensations, mostly in the morning. Wilson and Chowning (1902a, p. 132; 1903a, p. 61; 1904a, p. 37) report that the malaise is followed by a well-marked chill, which is usually most severe at the beginning and recurs at irregular intervals, though with decreasing severity. Anderson (1903a, p. 507; 1903c, p. 21) states that for a few days the patient may have chilly sensations, and finally there is a well-marked chill; he reports cases as follows:

Case 97 began with chills and vomiting, and with a rapid rise in temperature (pp. 16–17).

Case 115 was bitten by ticks April 1; complained of being chilly on April 7 or 8 (p. 24).

Case 116 was bitten by tick April 13; severe chill on April 19 (p. 26),

Case 117 was bitten by tick April 16; marked chill on April 20 (p. 27).

Case 118 was bitten by tick; chill April 20 (p. 27).

Case 120 found tick bites April 28; had chill same day (p. 29).

Gates (1903, p. 48) reports for one patient a hard chill during the last of the 1st week; he (1905, pp. 111–112) also reports chill for cases 11 and 14, in the latter case 10 days after the bite; later the chill recurred; pronounced chill for case 16.

During the 1904 season, chills occurred in cases 2, 3, 5, 6, 8, and 9.

Comparison.—Chills are reported for carceag.

FEVER.

Idaho.—During the first week following the incubation, the patient takes to bed with a temperature of 102° to 105° F., pulse 90 to 120; in favorable cases and in those of moderate severity there is a gradual decline in fever during the second week; the temperature varies in different cases; there is a daily rise during the first 4 or 5 days; the evening temperature is about 1° to 1½° higher than the morning remission; a temperature of 102½° to 104° is not uncommon by the fourth or fifth day; having reached its acme, the fever persists for several days; at the end of the second and during the third week the fever falls by lysis to an evening record of 98.4° (Bowers, 1896, p. 63). According to Collister (1896, p. 63) the febrile stage gradually follows the chill; it continues 2 or 3 weeks; it is not common to find a temperature over 103°, except in occasional cases. Fairchild (1896) states that in some cases febrile action runs high; it usually ranges from 101° to 104½° or 105°, and is continuous, showing but slight remissions. Figgins (1896, p. 64) reports the fever as remittent; the temperature ranges from 100° to 105°; pulse, 100 to 120. Springer (1896, p. 61, 62) says that after the chill the fever sets in, ranging from 103° to 105°; the fever ranges high and continues from 10 to 14 days; then it intermits for the following week or two. Zipf (1896, p. 65) says that the usually sudden onset is accompanied by high fever; the fever is continuous, lasting one to two weeks and is out of proportion to the danger of the disease; it also leaves the patient weak for weeks. Maxey (1899, p. 435) describes the fever as of the continuous type, beginning on the first day and rising gradually until it reaches 102° to 103° on the third or fourth day, when the eruption

usually appears; it is highest on the fourth to seventh day, corresponding to the period of most profuse eruption; there is a difference of 1° to 1.5° F. between the minimum morning and maximum evening temperature, which difference is maintained until about the tenth to fifteenth day, when the temperature line becomes erratic, for at this time it is apt to take sudden jumps up and then as suddenly down, while at the same time the average temperature is gradually going down, until by the end of the third week the patient is entirely free from fever. The Medical Sentinel (1899, p. 457), speaking editorially, refers to the fever as remittent in type.

Montana.—The fever comes on with or rapidly follows the initiative chill, so that upon the first visit the temperature is usually 102° to 104° F.; it becomes gradually higher day after day until it reaches its maximum in 2 to 7 days, when it ordinarily registers 103° to 106°; there seems to be a slight evening rise above that of mornings; in probably all cases except the mildest, one may be misled in the latter days of the attack in thinking the fever abated upon feeling the skin, or by the thermometer registered in the axilla, while the rectal temperature shows to the contrary; this difference in temperature is apparently caused by the slow, feeble, obstructed circulation, the exterior and the extremities becoming cool from a lack of blood supply; in cases where recovery takes place, the fever begins to abate about the fourteenth day and gradually recedes until it disappears, on an average, on the twenty-first day; 2 cases had subnormal temperature mornings and about 1° of fever at 6 p. m. almost during the entire attack. (Gwinn, 1902.)

McCullough (1902, p. 226) says that the temperature and pulse assume the form of most continued fevers, both gradually increasing, until the acme of the disease is reached in nonfatal cases about the end of the second week; very high temperature is not usual, ranging from 102° to 106°.

According to Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37) after the initial chill, fever rapidly develops, and may reach 103° to 104° F. on the second day; it gradually increases and reaches its maximum in from 5 to 7 days, when it may register 105° to 107° F. (rectal temperature); the difference between rectal and axillary temperature is sometimes as much as 2° F.; usually a slight evening increase and morning decrease are noted; the temperature occasionally becomes normal or subnormal 18 to 24 hours before death; when recovery occurs, it is by lysis, much as in typhoid; the diminution of the fever begins about the end of the second week and reaches normal about 2 weeks later.

Anderson (1903a, p. 507; 1903c, p. 21) states that before the distinct chill there is little or no fever in the morning, with a slight rise in the afternoon; after the chill there is an abrupt rise, and from then on the fever gradually rises in the evening, with a slight morning remission. The maximum is usually reached on the eighth to the twelfth day; then, in a favorable case it gradually falls, becoming normal about the fourteenth to the eighteenth day, usually going to subnormal for a few days; in fatal cases the fever remains high, from 104° to 105° or 106° F., and the morning remissions are very slight or not present.

Gates (1903, pp. 48, 49) reports the temperature for two cases; in one case the patient was given small doses of aconite and spirit of nitrous ether and small doses of alcohol until the fever was reduced and bowels moved freely; the patient was sponged with cool and cold water, as needed for high temperature; the fever ran an irregular course, with great variation, reaching at times a temperature 104.5° F., and again sinking to 97° F. This low temperature was observed during the last of the first week of the disease, at which time the patient was in a state of collapse, being almost pulseless and having a hard chill at the time.

Comparisons.—In Texas fever, if the temperature of exposed animals be taken once daily—say, in the morning—it will be found that at the onset of the disease it will rise within 24 hours from the normal to 104° F. or even higher. In the following 24 hours it may rise to 105° or 107° F. The continued daily record will then

show a high temperature until the disease terminates fatally or in recovery. In the former case it may fall from 2° to 4° below the normal just before death. When recovery ensues, it falls as quickly to or even below the normal as it rose in the beginning of the attack. If the temperature be taken twice daily—in the morning and the evening—a new set of phenomena appear. The temperature at the outset rises during the day, is highest in the evening, and may be low again in the morning. This oscillation, partly a normal occurrence, may be noticed for 3 or 4 days in some cases, the morning temperature gradually rising until it is as high as the evening temperature. The high temperature then remains continuous until the end of the fever. (Smith and Kilborne, 1893, p. 16.)

For canine piroplasmosis, Nuttall (1904, pp. 232–233) reports:

"South Africa: Fever recorded in all cases, and may be present when the dog appears well, thus constituting usually the first symptom. Fever starts at 104.2° to 105.4°, and oscillates or rises to 105° or 106.6°, even 107° F. In chronic cases (Chart V) there may be great oscillations in temperature, which may fall below normal (97° to 98° F., about 36° C.) and again rise. Toward death the rectal temperature gradually falls far below normal; in three of my dogs 98.2°, 97.2°, 90° F. (32.2° C.) were recorded, respectively, when last taken.

"France: In acute cases fever at onset may exceed 40° C. (104° F.), is maintained usually 2 to 3 days, then the temperature falls below normal, even down to 33° C. (91.4° F.). Rarely temperature is seen to oscillate, then gradually fall. In young dogs, which die very quickly, initial fever may be absent, parasites appear in the blood, and temperature sinks until death.

"In chronic cases fever usually absent; slight when present; rarely exceeds 40° C. (104° F.). May be overlooked; lasts 36 to 48 hours, then falls. In one case a 'quartan fever,' with remissions, as in the human malaria, observed."

RESPIRATORY SYSTEM.

BRONCHITIS; COUGH.

Idaho.—There is considerable bronchial irritation, cough lasting during convalescence or as long as there is any appearance of the eruption (Figgins, 1896, p. 64). Fairchild (1896) agrees that a slight cough accompanies the disease. According to Maxey (1899, p. 435) there is occasionally some bronchial cough, which may or may not be accompanied by some pain in the lungs.

Montana.—An irritative cough generally exists from the first, but not to an extent to be especially noticeable (Gwinn, 1902). Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37) state that a bronchial cough is frequently present at the onset. Anderson (1903a, p. 507; 1903c, p. 21) refers to a slight bronchitis after a few days; always present in the second week (1903c, p. 23). Gates (1905, pp. 111–113) reports cough for his cases, Nos. 11 (tenth day, especially well-marked symptom), 14, 15 (most troublesome), 16 (some, but not so much as usual).

In the 1904 cases no cough was recorded for cases 7, 9, and 13; in case 3 there was no cough on May 10, but the patient coughed freely on May 14; there was some cough in case 10.

Bronchial glands. In case 11 (1904) the bronchial glands were found enlarged at autopsy.

THYMUS.

In case 11 (1504) remnants of the thymus were seen upon removal of the sternum.

Respiration is notably deeper, fuller, and more labored from the first than in health, as well as increased in frequency; the respiratory acts are labored and increased in frequency in proportion to the amount of cyanosis rather than the amount of fever. (Gwinn, 1902.)

According to Wilson and Chowning (1902a, p. 133; 1903a, pp. 64–65; 1904a, p. 40) the respiratory rate sometimes reaches 60 per minute in the adult, though ordinarily it does not run above 36 per minute; like the pulse rate, it is frequently out of all proportion to the temperature; it is regular, but usually shallow; it may be labored and accompanied by rattling, due to accumulation of mucus in the upper air passages, during the last day or two of life; Cheyne-Stokes respiration has not been observed.

Anderson (1903a, p. 507; 1903c, p. 23) states that the respiratory rate is always increased, usually varying from 26 to 40 per minute, in some cases reaching 50 to 60; it is regular, but often shallow. He reports hurried respiration for case 37 (Howard's case, p. 12), between 40 and 50 for case 56 (Howard's case, p. 14), 24 for case 90 (Brice's case, p. 16) the second day after onset, 32, two days after onset, increasing gradually to 45, for case 97 (Burton's case), taking his data from Wilson and Chowning, 1903a. In case 117 (p. 27) the respiration was at first normal, became more rapid and labored until a few hours before death, then gradually weaker: 26 and 28 for case 118 (p. 28).

Gates (1903, p. 50) reports in one case that the respiration varied from 30 to 40 throughout the course of the disease and continued until after all signs of heart action had ceased. He (1905, p. 113) reports Cheyne-Stokes respiration during last of second week in his case 16.

In the cases of 1904 respiration became poor (7) in case 2, the patient at times struggling for breath on May 8; Buckley observed Cheyne-Stokes respiration in this case. In case 3 it was slightly harsh and prolonged anteriorly; it became slightly stertorous on May 12; on May 13 the lung sounds were very harsh all over the front and back, with large coarse rales; the lungs were rapidly filling with fluid; for 5 or 10 minutes respiration would be quiet and regular, then very rapid (60) for a time; on May 14 the filling of the lungs progressed rapidly, edema well marked; on May 15 there was much noise in breathing. In case 5 the respiration suddenly ceased on May 21; artificial respiration was resorted to and in 5 minutes the patient breathed, was roused, and quite rational; failure of respiration continued to occur at intervals, patient apparently forgetting to breathe; hypodermics of morphine acted as a respiratory stimulant (Mills). In case 7 respiration became harsh, especially on right side. In case 11 respiration was depressed, falling on June 24 to 8 (patient receiving large doses of morphine).

Edema of lungs developed in case 2.

COMPARISONS.—For Texas fever, see above, page 63.

In canine piroplasmosis breathing is accelerated, subsequently labored, irregular, and finally very shallow; in acute cases respirations are 36 to 48 per minute (accelerated), labored, gasping, and at times, especially in young dogs, accompanied by whining sounds; examination of thorax negative.

PLEURAL CAVITIES.

No adhesions were reported in cases 107, 89, 91, and 120. In case 93 the pleura was intensely adherent over entire surface of both lungs; the adhesions were very thick and fibrous; there was a history of pleurisy several years before. (Wilson and Chowning, 1903a, pp. 47, 51, 52, 54; Anderson, 1903c, p. 33.)

In 1904 the pleural cavities of case 11 were normal except a few adhesions between left lung and pericardium.

COMPARISONS.—In canine piroplasmosis, the peritoneal and thoracic cavities may contain fluid (Hutcheon); sometimes there is brownish serous exudate in thorax; in Lounsbury's chronic case there were pericarditis and pyothorax.

LUNGS.

See also Gangrene; page 50.

Wilson and Chowning (1904a, pp. 41, 42) report hypostatic congestion; all the lungs show considerable congestion and swelling of the capillaries; many red blood cells containing parasites are present; in most cases many phagocytes are found which have taken up infected red cells and pigment granules; in one case there was considerable broncho-pneumonia; pleura was normal.

Anderson (1903c, pp. 33, 38) reports that the plurae are normal and do not contain excess of fluid; lungs show hypostatic congestion, occasionally pneumonia; in case 120 there were no consolidated areas, except a few points resembling emboli.

See also Complications, page 87.

Upon autopsy, the lung tissue in case 93 was fully crepitant; there was slight hypostatic congestion on both sides; in cases 89 and 91 both lungs were normal, except hypostasis on both sides; in case 107, the lungs were apparently normal, no pneumonia (Wilson and Chowning, 1903a, pp. 47, 51, 52, 54). In case 120, the lungs were normally inflated, no consolidated areas, except a few points resembling emboli (Anderson, 1903c, p. 33).

During the season of 1904, case 2 died of edema of the lungs; in case 3 also edema of the lungs developed; in case 8, slight edema of the lungs developed; there was edema of the right lung May 29, and on May 30 both lungs were edematous. In case 11, on autopsy, the right lung was lead color on upper surface, very dark posteriorly; several dark spots one-eighth inch in diameter anteriorly, but apparently old; entire lung edematous; posterior portion extremely congested, in a condition of hypostatic pneumonia, and sinks in water; left lung shows same appearance as right, except pneumonic area is less marked and less extensive.

COMPARISON.—In Texas fever the lungs are, as a rule, healthy; there is, in many cases, pulmonary edema, with or without emphysema, noticeable after death; in a few instances foci of dark red hepatization were observed in one of the principal lobes, which involved one or several lobules. (Smith and Kilborne, 1893, p. 26.)

In canine piroplasmosis the lungs are rarely affected (Hutcheon); Nuttall noted edema and pinkish frothy fluid in the bronchi and trachea; in France apoplectiform foci have been found; in young dogs dying quickly, usually there are acute edema and reddish foamy secretion in bronchi and trachea.

Hypostatic lobar pneumonia herds have been recorded in carceag.

MUSCULAR SYSTEM.

See also Pains, page 76.

Gwinn (1902) reports rigidity of muscles of neck and back in one case. Several authors refer to the soreness and stiffness of the muscles.

Anderson (1903c, p. 23) says that the soreness of the muscles and bones causes the patient to change position often; muscular soreness is often very severe even in mild cases and lasts until recovery.

Wilson and Chowning (1903a, p. 47) report some tenderness on pressure along the spine (case 107), especially in the dorsal region, though this may have been due in part to the general soreness of the muscles.

In case 11 (1904) the muscles were well developed and of good color.

COMPARISON.—In Texas fever the lean meat may be of a brownish mahogany color and possess a peculiar sickening odor, or it may be normal in color or perhaps a trifle paler. In carceag the muscles are reported as pale and flabby.

EMACIATION.

See also Fat, page 52.

Emaciation has not been reported in connection with "spotted fever," but it seems to be quite a prominent symptom in some piroplasmatic diseases.

Thus, for Texas fever, Smith and Kilborne report that there may be extreme emaciation during the period following the fever. For canine piroplasmosis, Nuttall (1904, pp. 231, 326) reports that there is a loss of weight which is greatly increased during the final stages, and appears to be more marked in long-continued cases; there is great emaciation.

NERVOUS SYSTEM.

See also Pains, page 76; Photophobia, page 52; Extremities, page 55.

Idaho.—All cases are very nervous, sleepless, and throughout the disease suffer intensely; there is a hyperemic condition of the nervous system, as is shown by the general neuralgic pains; in my opinion it is a hybrid between typhoid fever and cerebrospinal fever, the disease having many symptoms common to both (Fairchild, 1896). Springer (1896, p. 62) says that the patients are usually very nervous and irritable. Sweet (1896) reports that the nervous system is sometimes involved; in such cases there is marked hyperpyrexia.

Montana.— McCullough (1902, p. 227) reports indifference to surroundings. According to Wilson and Chowning (1902a, pp. 132–133; 1903a, p. 63; 1904a, pp. 38), aside from the headache at the beginning, many patients show no nervous symptoms until just prior to death; a low muttering delirium, as in typhoid, is present in some severe cases, the patient being but partly rational; in the severe stages, picking at mouth, ears, and bed clothing is present; except at the onset the disease is remarkable for its freedom from pain.

Gates (1903, p. 50) reports for one case that there seemed to be a profound impression on the nervous system from the very first symptoms of the disease; muttering delirium and a semicomatose condition, from which the patient could be aroused only with much effort, were early and prominent symptoms. Later he (1905, p. 113) states in regard to his case 16 (1904) that the intensity with which the disease attacked the nervous system was marked from the onset; a low muttering delirium came on during the first week; during the second week the patient was in a heavy stupor from which she could be aroused with difficulty, but when aroused she would answer questions correctly and then, perhaps, talk at random; the condition of the mind approached normal during the third week.

During the season of 1904 nervous symptoms were prominent.

In case 2 hyperesthesia (see above, p. 51) was extreme, the weight of a palpating hand or even of the bed clothing caused extreme pain. For case 4 severe intracranial and supraorbital neuralgia, May 12, was reported; condition improved under codeine; in general the nervousness of the patient was marked in this case, as also in case 5. In case 6, who was of a neurotic temperament, the nervous symptoms were prominent, very marked, and constant; twitching of muscles, etc. On May 19, when touched anywhere on the body the patient was thrown into a state of tonus. In case 7 nervous disturbance was important and marked throughout illness. The patient thrashed around in bed, rolling head and throwing the arms around. He could not be made to lie on the left side for a minute at a time, but would immediately throw himself upon his right side or his back. In case 8 nervousness increased May 24; dullness increased with slight delirium; May 25 hyperesthesia was very marked; there was high nervous tension, the muscles of the back and limbs became very rigid; all nervous symptoms increased May 26, 27, and 28; a drink of water would produce spasm of pharynx and diaphragm; reflex excitability was so intense for 24 hours before death that a slight touch used in putting a spoon to the mouth, or sponging, etc., would cause spasm and rigidity of entire body. In case 11 the severity of the nervous symptoms pointed strongly to cerebrospinal meningitis. In case 13 there was picking at the bed clothing, muttering, and restless rolling from side to side.

MALAISE.

Idaho.—During incubation there is a feeling of lassitude and inaptitude for work (Bowers, 1896, p. 63). Many cases are taken suddenly without previous malaise (Collister, 1896, p. 63). Lassitude is mentioned by Dubois (1896, p. 64). Fairchild (1896, p. 62) says there are usually 2 or 3 days of malaise. Springer (1896, p. 61) states that there is a feeling of malaise for a few days preceding the chill, and Maxey (1899, p. 435) reports that the patient first notices a general malaise.

Montana.—In a few cases the disease seems to be preceded by a prodromal period of malaise for a few days (Gwinn, 1902). According to McCullough (1902, p. 226) the attack may come on insidiously with a feeling of malaise for a few days, gradually growing worse and merging into a well-defined "bone ache."

Wilson and Chowning (1902a, p. 132; 1903a, p. 61) and Anderson (1903a, p. 507; 1903c, p. 21) agree that many cases are preceded by a short period of malaise.

In 1904 case 3 complained of slight malaise on May 10; on May 12 this case showed stupor.

RESTLESSNESS AND INSOMNIA.

Idaho.—Sleeplessness is common during the first week (Bowers, 1896, p. 64). On account of the fever and the soreness and the pains in the extremities and back the patient rolls and tosses in a restless effort to find a comfortable position (Maxey, 1899, p. 435).

Montana.—There is considerable restlessness (Wilson and Chowning, 1902a, p. 132; 1903a, p. 62; 1904a, p. 37). Anderson (1903c, p. 23) says that the soreness of the

muscles and bones causes the patient to change position often; he reports (pp. 14, 15) marked jactitation for cases 56 and 57, and says (p. 28) that restlessness in case 118 was allayed by darkening the room.

Gates (1905, p. 111) reports insomnia as especially well marked in his case No. 11, persistent in case 14, not troublesome in case 15.

In 1904 all cases were reported as very restless, especially cases 2, 6, 7, and 11. Case 2 was markedly drowsy on May 4.

DIZZINESS.

McCullough (1902, p. 226) reports dizziness among the initial symptoms. This was not complained of in cases 1 to 11 (1904).

HEADACHE.

Idaho.—Cephalalgia is the most common and persistent symptom in the development of this disease; the pain is intense and persists without intermission; it is referred to the frontal region or to the occiput, or to the entire head (Bowers, 1896, p. 64). Collister (1896, p. 63) states that there is generally a severe headache. According to Dubois (1896, p. 64) the patient may be stricken down without warning with a severe frontal headache. Severe headache, particularly in the back part of the head, is mentioned by Fairchild (1896, p. 62) as an early symptom. Sweet (1896) also mentions headache. Violent headache is recorded by Zipf (1896, p. 65) as an initial symptom. On the second day the pain in the head becomes quite severe (Maxey, 1899, p. 435).

Montana.—There is a general aching and soreness of the whole body (Gwinn, 1902). According to Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37) the headache may be severe at the onset. Anderson (1903a, p. 507; 1903c, p. 21) reports some pain in the head; he gives (1903c, pp. 24, 27) headache as following the tick bites in case 115 and as present in case 118. Gates (1905, pp. 111–113) reports for his cases 11 and 16 severe headache; for case 11, frontal headache following the chill.

Headache was present, to a greater or less degree, in cases 1 to 11 during 1904. In case 1 it was reported as frontal; in case 5 it began at the time of the chill; in case 8 it was very severe; in case 11 it was both frontal and occipital.

PAIN, OTHER THAN HEADACHE.

Idaho.—During the first week following the incubation period there is pain in the back and legs and a muscular soreness over the entire body; it is severe in the lumbar region or along the entire spine and in the lower extremities (Bower, 1896, pp. 63, 64). Collister (1896, p. 63) states that there is pain of a rheumatic character in the back and limbs, mostly referred to the joints. Dubois (1896, p. 64) mentions intensely severe pains in joints and muscles as an early symptom; this pain "is boring, breaking, and stabbing, and appears to penetrate into the very substance of the bones." Fairchild (1896, p. 62) reports shooting pains throughout the body and limbs, usually more severe in the bowels and back; it is neuralgic in type and is controlled only by morphine; the pain may appear early, and continues throughout the attack. Figgins (1896, p. 64) records pains in the extremities, and muscular soreness. According to Springer (1896, p. 62) the patient usually complains of severe pains throughout the body, especially in the back and stomach, and in many cases in the larger joints; these pains are not continuous, but are of a shooting character and cause the patient to cry out on any movement. Sweet (1896, p. 61) says that the onset is usually accompanied by severe breakbone pains; the intense

boneache is suggestive of dengue. Zipf (1896, p. 65) reports backache. According to Maxey (1899, p. 435) the bones and muscles soon begin to ache, and on the second day the patient feels sick enough to take to his bed; he already feels very weak and depressed, and the pains in the back and in the joints and muscles of the extremities are becoming quite severe; accompanying the bronchial cough there may or may not be some pain in the lungs.

Montana.—At the onset there may be intense soreness seemingly of the entire muscles of the body (McCullough, 1902, p. 226). According to Gwinn (1902) there may be general aching and soreness of the whole body. Patients while conscious usually complain most bitterly of general aching and soreness, and prefer to be moved as little as possible, especially after the disease has progressed a few days. Sometimes the pain is referred to as being more in the head, in other cases the back, while in others it does not seem to predominate at any single point. Wilson and Chowning (1902a, p. 132; 1903a, p. 62; 1904a, p. 37) state that the onset is accompanied by a severe aching in the bones and muscles, with pain in the back and joints. The patient is usually very weak (1903a, p. 45). In case 109 there was no stiffness of the neck or back, though some pain was present on pressure over the spinal processes of the vertebræ, especially those in the dorso-lumbar region. They also report (1904a, p. 37) that cases Nos. 94, 96, 97, 115, 116, 117, 119, and 120 give a history of soreness about the tick bite, and pains radiating therefrom which continued until the initial chill, but (1902a, p. 133; 1903a, p. 63; 1904a, p. 38) except at the onset the disease is remarkable for its freedom from pain. Anderson (1903a, p. 507; 1903c, p. 21) reports for the onset some pain in the back and head and soreness of the muscles and bones. He states (1903c, pp. 21, 23) that the pain in the head and back is usually severe during the first week, while the soreness of the limbs causes a sensation as if the limbs were in a vise, and causes the patient to change position frequently. The soreness of the muscles is often very severe in mild cases and lasts until recovery. Anderson (1903c, p. 16) records pains in head, back, and upper and lower limbs and soreness of muscles in all parts of the body for case 90; (p. 26) case 116 was bitten by tick under the arm on April 13, and the chill on April 19 was followed by aching pains; the wound and axillary glands were sore and swollen on April 13, soreness was less marked after a few days, but still present; it became much worse on April 19, with shooting pains radiating from the axilla, through the shoulder, down the arm and side of the body; aching pains in the back followed the severe chill and extended over the whole body; (p. 27) case 118 complained of aching pains in the back and limbs; (p. 29) case 119 experienced soreness from the wound made by the tick bite; pain and swelling was present, extending down the side of the head behind the ear to right side of neck; (p. 29) case 120 had pain in back and limbs on day preceding removal of ticks, the pains continued the next day; (p. 36) case 121 had pain in bones and joints, and (pp. 34, 37) backache on May 10, severe pain in back and limbs on May 14, backache and soreness of muscles of legs and arms on May 17, no pain in head or back on May 23. Anderson (1903c, p. 40) advises Dover's powders or morphine sulphate to relieve the pain. Gates (1905, pp. 111–113) states that in his case 11 the headache was followed by severe aching of all of the skeletal muscles, especially severe in the calves of the legs; case 14 complained of pains in the tibiæ, intense backache, pains in ankles and knee joints, and great muscular soreness including muscles of eye; case 15 complained of general muscular soreness, severe headache and backache, and great exhaustion; case 16 complained of aching in limbs and back.

In 1904, case 1 complained of "general body aching;" case 2 had some pain in back and head, and complained of general soreness after appearance of the eruption; case 3 complained of backache; case 4 complained of suprascapular and lumbar pains, and later of pains in

the head; case 6 complained of headache, backache, and legache, and general aching and pain; case 7 complained only of headache, pain in the abdomen and back of the neck; later (June 8) pain in the neck disappeared, but on June 9, and on June 15, patient complained of pain in the knees; case 11 complained especially of pains in the neck and lumbar region; case 12 complained of great soreness all over the body, the feet were exquisitely tender, and continued so until he left the hospital.

MIND.

Idaho.—The mental processes become dulled and the patient is listless and apathetic (Bowers, 1896, p. 64).

Montana.—In all attacks attaining a marked degree of severity the patient's mind is affected, first noticeable from incoherent and rambling speech when the temperature happens to be high, and if the case be dangerous in severity the disturbed thought sooner or later merges into delirium, usually followed by coma and death (Gwinn, 1902).

According to Anderson (1903a, p. 507; 1903c, p. 23) the mind is usually clear, even in severe cases, until within a few hours of the end (see cases 89, 116–121).

Case 7 (1904) was reported as answering questions intelligently up to within a few hours of death. Illusions were reported for case 4 on May 13; on 15th, illusions, weariness, and sleeplessness; illusions disappeared on May 19; case 3 showed stupor on May 12.

DELIRIUM.

Idaho.—Delirium occurs in severe cases; it is of a typhoid character, and due to fever or to toxemia (Bowers, 1896, p. 64).

Montana.—See also Mind, page 78.

According to McCullough (1902, p. 226) delirium usually manifests a very active part in the symptoms, and may be low and muttering, or only a mental hebitude, the patient being partially rational. Anderson (1903c, p. 12) reports marked delirium for case 36 (Buckley's case, 1897); case 74 (Putney's case, 1900) showed delirium about the fourth day (p. 14); case 102 (McGrath's case, 1902) was delirious much of the time after the fourth day (p. 11). Gates (1903, pp. 48, 50) records slight delirium in one case; in another case a muttering delirium and a semicomatose condition were among the early and prominent symptoms. Later (1905, pp. 115) he reports that the delirium in his case No. 7 (1901) lasted for two weeks.

In 1904 case 1 presented delirium, which soon passed to coma; delirium was present in cases 2, 3, and 6; in case 9 the mind wandered and passed to delirium; in case 11 the delirium was at times violent; case 12 showed delirium at no time, but case 13 was delirious.

COMA.

Coma and death usually follow the delirium (Gwinn, 1902). According to Anderson (1903a, p. 507; 1903c, p. 23), quoted from Wilson and Chowning (1903a), case 37 (Howard's case, 1898) was entirely comatosed for 3 days; case 8 (Gwinn's case, 1899) showed a semicomatose condition as an early sympton; case 118 (1903c, p. 28) passed into a state of semiconsciousness, gradually increasing to total unconsciousness, which gradually passed away, having lasted 72 hours. Gates (1903,

p. 50) reports a patient in a semicomatose condition, from which he could be aroused only with much effort (early symptom). Case 6 (1900) was in a very deep stupor for 10 days prior to death (Gates, 1905, p. 115).

In 1904, coma in case 1 followed the delirium and lasted till death; coma was also present in case 2 for some hours before death; case 3 was marked by the sudden onset and prolonged duration of coma; in case 6 coma preceded death a few hours; in case 8 coma was present to some extent May 26, and almost complete coma was reported May 27.

CONVULSIONS.

Case 95, a child of 2 years, had convulsions (Wilson and Chowning, 1903a, p. 60).

In 1904, case 7 showed convulsions about 8 hours before death, and there was almost a state of convulsion in case 6.

OPISTHOTONOS.

Idaho.—"In one case I found marked opisthotonos during the fourth week of the disease, which proved fatal. In this case there were marked cerebral conditions." (Fairchild, 1896.)

Montana.—Gwinn (1902) in one case observed rigidity of the muscles of the neck and back, very much resembling that of cerebrospinal meningitis. Wilson and Chowning (1902a, p. 133; 1903a, pp. 50, 57; 1904a, p. 38) and Anderson (1903c, p. 23) state that there is no opisthotonos.

In 1904, cases 2 and 3 presented toward death a tendency to opisthotonos, but the patients had received strychnine. Two unpublished cases of 1902 were described to me (not by a physician) as presenting marked opisthotonos. In Howard's case 7 there was slight opisthotonos reported during the convulsions.

BRAIN AND SPINAL CORD.

In one autopsy, Gwinn (1902) has been told, there was found a serous fluid in the fourth ventricle of the brain.

There is a slight congestion of the capillaries of the meninges; a few vessels of the cortex contain infected red blood cells; there is some distention of the pericellular spaces in the cortex; little or no chromatolysis is shown by Nissl's stain (Wilson and Chowning, 1904a, p. 43). The meninges of the brain and spinal cord showed a slight congestion, apparently hypostatic; there was no basilar (or other) meningitis (Wilson and Chowning, 1904a, p. 42). The vessels are somewhat congested in the spinal cord, as in the brain, and contain a small number of infected corpuscles; in one case the anterior horn cells show considerable chromatolysis with Nissl's stain; no fiber degeneration is shown by Weigert's stain (Wilson and Chowning, 1904a, p. 43).

Upon autopsy the brain substance was normal in color and consistency in cases 91 and 107; examination of the central nervous system in cases 89, 93, 94, and 97 was not permitted (Wilson and Chowning, 1903a, pp. 48, 52, 53, 55, 56, 58). Anderson (1903c, p. 33) makes no mention of the central nervous system of case 120.

In 1904, case 11, the veins on the surface of the brain were distended with blood; no pus nor lymph was found at base of skull; ventricles

appeared normal, except for distention of veins; a small amount of bloody serum was found in one lateral ventricle, the blood probably coming from a cut vein; on section the cerebrum appeared about normal; basal ganglia on section appeared normal; section of pons and cerebellum showed nothing abnormal.

The meninges over the occipital lobe of case 91 were congested (hypostatic), but otherwise normal; in case 107 the meninges were normal, except a slight congestion (hypostatic) in the vessels of the pia over the occipital lobe; there was no evidence of meningitis (Wilson and Chowning, 1903a, pp. 52, 48).

In case 11 (1904) the dura showed outer surface injected, but otherwise normal; removal of dura showed some adhesions at the vertex between the membranes and the brain substance, the area of adhesions being small.

In case 91 there was no inflammation or marked congestion in either of the meninges of medulla and cord; in case 107 there was no inflammation of the upper 6 inches of medulla and cord (Wilson and Chowning, 1903a, pp. 48–49, 55).

In case 11 (1904) section of the medulla showed nothing abnormal; the spinal cord showed considerable injection of the vessels, probably hypostatic; no lymph exudation or other evidence of inflammation was present; dura was normal; cross sections of cord at 1-inch intervals showed nothing abnormal; 15 cc. of clear spinal fluid was aspirated through the lumbar region before the canal was cut open.

Doctor Alton says that case 13 may possibly have been a case of meningeal irritation following measles, or may have been due to tick bite and infection from that source (no autopsy).

Local physicians have repeatedly spoken of the resemblance of "spotted fever" to cerebrospinal meningitis, and, in fact, the resemblance in some cases is very striking. Although the central nervous system has been examined upon autopsy in only a few cases, still it would seem rather remarkable that none of these autopsies showed the pathological lesions in case this disease were actually meningitis. At present, therefore, we are not justified in concluding that the "spotted fever" under discussion is cerebrospinal meningitis

Comparison.—In Texas fever no nervous lesions were found which can be regarded in any sense as peculiar to or characteristic of the disease. It may be said, in general, that the brain shared the general tendency toward the injection of the capillary system. The vessels of the pia and the plexuses were engorged, and over the frontal lobes and near the great transverse fissure it was more or less pigmented—a condition also met with in other diseases. The gray matter of the cerebrum and especially of the cerebellum appeared of a more pinkish color. The white substance was normal in color, the ventricles free from fluid (Smith and Kilborne, 1893, pp. 25–26).

In canine piroplasmosis a slight congestion of the meninges is found in some cases.

KERNIG'S SIGN.

Kernig's sign is absent (Wilson and Chowning, 1902a, p. 133; 1903a, pp. 49, 50, 57; 1904a, p. 38).

URINARY SYSTEM.

KIDNEYS.

Idaho.—Collister (1896, p. 63) states that the kidneys are often disturbed.

Montana.—Gates (1905, pp. 115) mentions severe parenchymatous inflammation for his case 10 (1903), and says that the kidneys suffered especially in case 12 (1903). Gwinn (1902) reports slight enlargement of the kidneys in one case on post-mortem. Referring to pathology Wilson and Chowning (1903a, p. 67; 1904a, p. 42) state that in all cases examined one or both kidneys showed small subcapsular hemorrhages on the ventral surface; the cortex on section was congested. Upon microscopic examination numerous phagocytes were found, each containing from 1 to 8 faintly outlined red blood cells, in nearly every one of which is a parasite; the kidney shows acute parenchymatous nephritis. Later (1904a, pp. 42, 43) they add that the capsule strips readily and there is some extravasation of red-blood cells, particularly in the cortex. Anderson (1903c, p. 38) summarizing autopsies on 7 cases (including the 6 autopsies reported by Wilson and Chowning, 1903a) states that the kidneys are enlarged; capsule usually not adherent; small subcapsular hemorrhages on ventral surface; on section, congested and swollen cortex; pyramids well outlined and deep red in color; small hemorrhages in pelvis; microscopically there are minute extravasations of blood in cortex and under the capsule; veins filled with blood; nuclei of convoluted tubules stain poorly; cells granular and in some places detached; newly formed casts in tubules; bladder normal and usually with small amount of dark urine.

In case 3 there were small subcapsular hemorrhages in both kidneys. In case 89 a small ecchymotic area was present immediately underneath the capsule on the anterior surface of the right kidney; the left kidney showed no ecchymotic areas, but otherwise was like the right kidney. In case 91 several minute hemorrhagic spots were present in the capsule over the ventral surface of each kidney. In case 94 the left kidney was normal in size and there was a hemorrhagic area near upper pole and on anterior surface; the right kidney was normal, except on section (see below). In case 97 the left kidney was about normal in size; the right kidney was small; fetal lobation was present. In case 107, 2 or 3 ecchymotic hemorrhages were present in the ventral portion of the capsule over the left kidney.—Wilson and Chowning, 1903c, pp. 48, 51, 53, 55, 56, 58. Anderson reports (1903c, p. 33) in reference to case 120 that the kidneys were enlarged; the left kidney weighed 10 ounces; there were minute subcapsular hemorrhages, especially over the greater curvature.

In case 11 (1904) the right kidney measured 12 by 7 cm.; the left kidney measured 13 by 7 by 6 cm., areas of distinct pallor were scattered over the surface.

In cases 89 and 94 the capsule stripped easily. In case 97 the capsule was adherent and slightly hemorrhagic in the left kidney, but the capsule stripped easily from the right kidney.—Wilson and Chowning, 1903a, pp. 51, 56, 58. Anderson (1903c, p. 33) reports the capsule as adherent on the left kidney in case 120.

In case 11 (1904) the capsules of both kidneys were adherent, carrying substance with them in removal.

In case 3 the cortex of both kidneys, on section, was slightly congested. In case 89 the section of the cortex appeared slightly congested. In case 91 the section of both kidneys was congested, but otherwise normal. In case 94 the cortex of the left kidney was normal in thickness, with hemorrhagic areas over the cut surface; the pelvis was normal; in the right kidney similar hemorrhagic areas were seen, but

to a less extent. In case 97 section of the left kidney showed slightly hemorrhagic areas the cortex was normal in thickness; on section the cortex of the right kidney was thin and dark, and hemorrhagic areas were seen over the whole surface, extending down to the pelvis. In case 107 the cortex of both kidneys, on section, was found congested.—Wilson and Chowning, 1903a, pp. 48, 51, 53, 55, 56, 58. Anderson (1903c, p. 33) reports for case 120 that on section the cortex was congested, the pyramids well outlined, and small hemorrhages about 1 mm. in diameter were present in the pelvis.

In case 11 (1904) the section of the cortex of the left kidney showed little if any alteration; the cortex of the right kidney appeared somewhat pale and thickened to Ashburn and myself, but to Mills it appeared about normal.

Comparison. In Texas fever, Smith and Kilborne (1893, pp. 31–32) report that "in a considerable number of cases a sero-sanguinolent condition of the connective tissue and fat about the kidneys is observed. In a few cases the ventral surface of the organ appeared like two large blood blotches. The portion of the abdominal wall upon which the dorsal surface of the kidneys rest is free from these effusions.

"The kidneys themselves, like the other organs affected by this disease, vary more or less in color, according to the severity and stage of the disease. In those cases which succumb early in the fever and in which the bladder is filled with port wine colored urine, the kidneys are enlarged and of a uniform dark brownish-red color throughout. The usual markings are pretty well effaced. When fresh sections are examined from different regions, the vascular system is found quite uniformly engorged and distended with red corpuscles. The section is likewise sprinkled over with very minute pigment particles. Sometimes irregular masses of red corpuscles, run together as it were, are met with in the vessels of the pyramids. Lesions of the secreting structures are not discoverable. Hemorrhages are uncommon. In those cases which succumb after the hæmoglobinuria and the fever have passed away, the kidneys are paler than usual and the texture is quite flabby. Sections of the fresh tissues show in the cortex a considerable amount of pigment. In some cases the convoluted tubules are the elected seat of pigment deposit, and the epithelium of these tubes may be so filled with yellowish-red pigment that they are easily traceable in their windings by their decided color. Fatty changes are occasionally met with in the epithelium, and the straight tubules of the pyramids may be filled with fat globules. Degenerative or necrotic changes of the epithelium were not noticed in sections of hardened tissue from a few cases stained in various ways. In those cases in which the capillaries were filled with red corpuscles, the latter were usually all infected with Texas-fever parasites. The pelvis and its ramifications were usually found beset with blood extravasations." For hæmoglobinuria of cattle, Starcovici states that the kidney capsule is hemorrhagic; a large hemorrhagic spot is present over each kidney, extending to the peritoneum. The kidneys are large, stiff, brittle, and dark red. The lining membrane of the pelvis is swollen and ecchymotic. The kidney changes consist in an overfilling of the vessels, and especially of the glomeruli, with blood, and peculiar desquamative and fibrinous yellow masses in the lumena of the uriniferous tubules, the epithelia of which appear compressed and show parenchymatous alteration.

For canine piroplasmosis, Nuttall (1904, pp. 236–237) records that according to Hutcheon the kidneys are more or less congested, at times edematous, with dark-brown cortex; according to Robertson, they are pale and friable; in France they are usually greatly congested, and the capsule strips easily, revealing numerous petechiæ; on section, the cortex seems to be congested, and shows petechiæ; blood from kidney is very rich in parasites; in an acute case, yellowish-red fluid exuded on section.

SUPRARENALS.

In case 11 (1904) the gross appearance of the left suprarenal was apparently normal; the right suprarenal appeared congested, otherwise normal.

BLADDER.

Wilson and Chowning (1904a, p. 42) report that upon autopsy the bladder was normal, and contained a small amount of urine which was darker than normal. Anderson (1903c, pp. 33, 38) reports the wall of the bladder apparently normal, the contents about 4 ounces of urine (case 120).

In case 3 the ventral wall of the bladder was congested; 1 ounce of urine of normal color was present. In case 89 the bladder and urine (about 3 ounces present) were normal. In case 91, 2 ounces of urine of normal color were present. In case 107 about 1 to 2 ounces [or ½ ounce?] of apparently normal urine was present.—Wilson and Chowning, 1903a, pp. 48, 52, 55.

Anderson (1903c, p. 33) reports for case 120, that the bladder was apparently normal and contained about 4 ounces of urine.

COMPARISONS.—Smith and Kilborne (1903) report for Texas fever that the bladder may show a few ecchymoses on its inner surface, and most cases contain 1 to 4 quarts of urine holding more or less hemoglobin in solution.

In hemoglobinuria of cattle the bladder is filled with dark red to black urine, which contains much hemoglobin, but usually no red-blood corpuscles.

In carceag, the bladder contains pale to red-brown urine with hemoglobin.

For canine piroplasmosis, Nuttall (1904, pp. 236–237) says that the bladder may appear normal (Hutcheon, Robertson), and may contain urine which is generally dark brown like "pontac." In France prune-juice-like urine is found in an acute case.

URINE.

Quantity.—Idaho.—In the Idaho cases the urine is reported as "scanty" by Bowers (1896, p. 64), Fairchild (1896), Springer (1896, p. 62), and Maxey (1899, p. 435).

Montana.—For Montana patients the urine is reported as reduced to one-half its normal amount (Wilson and Chowning, 1902a, p. 133; 1903a, p. 64; 1904a, p. 40; Anderson, 1903c, pp. 21, 23). Gates (1903, p. 49) gives it as 32 ounces for 24 hours following his first visit to 1 case; it then gradually diminished until 2 days before death, when there was complete anuria. Later (1905, pp. 111–113), Gates reports the urine as lessened in amount in case 11; much lessened at times in case 15; very scant at end of 1st week in case 16, but increased to normal amount by end of 2d week.

Wilson and Chowning (1903a, p. 58) give the urine in the bladder as normal in case 94.

In 1904 in case 3 urine was passed normally on May 10, and abundantly and involuntarily on May 15; in case 5 it was passed normally; in case 7 it was regular; case 11 passed 6 ounces in 24 hours on June 25 and 8 ounces in 24 hours, June 26; in cases 12 and 13 it was scanty. The quantity of urine was not measured regularly.

Color.—For *Idaho* patients the urine is reported as of high color (Bowers, 1896, p. 64; Fairchild, 1896; Maxey, 1899, p. 435).

For *Montana* patients it is reported as "slightly above normal in color or as highly colored" (Wilson and Chowning, 1902a, pp. 132, 133; 1903a, p. 64; 1904a, pp. 37, 40); Anderson (1903c, p. 17) reports it highly colored in case No. 97; Gates (1903, pp. 48, 49) reports it as highly colored in one case and dark in another case. Later, Gates (1905, pp. 111–113) reports the urine as almost brown in case 11; highly colored in case 14; and dark in case 15.

In 1904, the urine was reddish yellow and turbid in cases 5 and 11, with a heavy yellowish precipitate in case 11; it was of high color in case 13.

Specific gravity.—For *Idaho* cases it is reported as of high specific gravity by Bowers (1896, p. 64).

In 1904, the specific gravity was 1018 for case 11, 1022 for case 9, 1028 for case 5, and 1030 for case 13.

Reaction.—Maxey (1899, p. 435) gives it as acid in Idaho.

In 1904 the reaction was acid in cases 5, 9, and 11.

Albumen.—In *Idaho* cases no albumen was found by Fairchild (1896) and Maxey (1899, p. 435).

In *Montana* Wilson and Chowning (1903a, p. 64; 1904a, p. 40) found a small amount of albumen in each of 5 cases examined. Anderson (1903c, p. 23) states that a small amount of albumen was found in all cases examined, but his protocol of case 121 (pp. 34–37) denies albumen for this case; he gives albumen (pp. 31, 32) for case 120. Gates (1903, pp. 48, 49) gives slight amount of albumen for one case when first seen, and also for another case during the second week, while it was absent when first seen. Later (1905, pp. 111–113) Gates reports some albumen for his case 11; none for case 14; a trace in case 15; some albumen was found at times in case 16.

In 1904 albumen was found in case 5 (May 18), a trace was present in case 9, and a small amount in case 11. No albumen was found in cases 12 and 13. In none of these cases was a daily test made.

Sugar.—No sugar was found in 1904 in cases 9, 11, 12, and 13.

Urea.—Urea was 3.5 per cent in case 11 (1904).

Bile.—No bile (Gmelin) was present in case 11 (1904).

Casts.—Both granular and blood casts were found in each of 5 cases examined by Wilson and Chowning (1903a, p. 64), and later (1904a, p. 40) in 4 additional cases. Anderson (1903c, pp. 21, 23) states that granular, hyaline, and epithelial casts may be found; (p. 36) he found no casts in case 121; no casts (p. 31) were found in case 120 on May 8, but granular and epithelial casts were present on May 11. Gates (1903, p. 49) reports some hyaline and granular casts for one case when first seen (May 11), and an enormous number of granular, blood, and epithelial casts for the same case in the last sample [date?] taken. Later (1905, p. 112) he reports numerous blood and epithelial casts for case 15 (1904), and says that the urine of case 16 contained many blood and epithelial casts, but later, by the end of the second week, it was free from casts.

In 1904 granular casts were abundant in case 9. Case 11 contained abundant vaginal and small round epithelial cells, and numerous blood, epithelial, and granular casts, also much granular débris.

Hematuria.—Anderson (1903c, pp. 31, 32) found no red blood cells in case 120, and Gates (1903, p. 49) reports red and white blood cells in the last sample taken in one case. See also blood casts, under "Casts," above, p. 84.

In 1904 case 9 showed no blood. Case 11 showed free red and white blood cells (Ashburn).

Hemoglobinuria.—Hemoglobinuria is reported as absent or very slight by Wilson and Chowning (1903a, p. 64; 1904a, p. 40).

Deposits.—Maxey (1899, p. 435) says, in reference to *Idaho* cases, that the "urine

is loaded with amorphous urates," and Anderson (1903c, p. 31) reports heavy deposit of phosphates in case 120, in *Montana*.

Post-mortem.—Anderson (1903c, p. 38) states that usually a small quantity of highly colored urine is found in the bladder on post-mortem examination. (See under Bladder, p. 83.)

Comparison.—For Texas fever, Smith and Kilborne describe the urine as follows:

"Next to the high temperature the condition of the urine demands our attention. The one sign regarded as peculiar and pathognomonic in this disease is the discharge of urine having the color of blood. This color is not due to a discharge of blood from the kidneys and subsequent breaking up of the red corpuscles, but to a filtration of the coloring matter of broken-down red corpuscles (hæmoglobin) already in solution in the circulation into the urine in the excretory structures of the kidneys. This fact was first pointed out in 1868 by R. Cresson Stiles. In using the term hæmoglobinuria this is all that is meant in this report. * * * Hæmoglobinuria may be said to be present in most acute fatal cases of Texas fever. Out of 46 fatal cases in which urine was in the bladder after death, hæmoglobin was present in 33 cases. A careful examination of the notes will show that in 13 negative cases the animals were killed in the earliest stages of the fever, or else they died or were killed after the number of blood corpuscles had been greatly reduced and the acute stage of the disease was over. In the former cases the hæmoglobin had not yet been set free from the corpuscles; in the latter cases it had probably been eliminated one or more days before death. How frequently 'red water' is passed before death we can not state with any degree of certainty, since its discharge may wholly escape observation. We have a record of hæmoglobinuria in but four cases: In No. 43 on the third day before it was killed (probably 12 to 24 hours before death), and in No. 198, 24 hours before death. In some of these cases it so happened that the urine was passed while the animal was undergoing examination. It is interesting to note in connection with the statements made that in No. 44 no 'red water' was found in the bladder after death, although it had been passed 4 days previously. Whether hæmoglobinuria is always present in acute cases of Texas fever, it is impossible to state definitely. As it seems to depend upon the rapidity with which the red blood corpuscles are infected and destroyed, a slower destruction may allow other organs to take charge of the débris and thus forestall the discharge of hæmoglobin in the urine. In the notes will be found the record of hæmoglobinuria in but one acute case which recovered (No. 49), while in a number of cases in which the urine was collected, sometimes in the height of the fever, sometimes after it had departed, no hæmoglobinuria was detected. In this solitary case the high temperature first appeared August 18. On August 23, the temperature being still above 105°, the urine was free from hæmoglobin, but contained a small quantity (0.05 per cent) of albumen. On August 27 the temperature had become normal, but a second paroxysm followed soon after, and on September 4 and 5 the urine was of a port-wine color. Urine collected September 6 was again of normal color. The urine during the fever, when free from hæmoglobin, contains in many instances a small quantity of albumen. The specific gravity may at first be high (1,030 to 1,040), and it may be strongly alkaline and effervesce with acids as in health, but, as the disease progresses, and when the animal eats but little, its specific gravity will fall to 1,010 to 1,020; it fails to effervesce with acids and is faintly alkaline or even slightly acid. When the fever has subsided the urine has been observed to be in a few cases very watery, i. e., of very low specific gravity and feeble in color. Within one or two weeks, however, the normal condition is restored.

"The urine which contains the coloring matter of the blood varies, as might be expected, very much in depth of color, according to the concentration of the hæmoglobin. It may have a very light claret color, or it may be so deeply tinted as to appear opaque and blackish. In a test tube when viewed by transmitted light it may barely permit the light to pass unless diluted with water. Such urine is, as a rule, entirely

free from suspended matter and blood corpuscles. The latter may sometimes be found in small numbers when the urine is permitted to stand, and they may be derived from small hemorrhages in the pelvis of the kidney, quite regularly observed at autopsies. The coloring matter, as has been stated above, is derived from corpuscles broken up within the circulation, and not outside in the bladder. When such urine is treated with a little acetic acid a brownish flocculent precipitate, probably of the derivatives of hæmoglobin, appears. When boiled, a brownish flaky precipitate forms, which rises to the surface as a scum. As might be expected such urine always reacts in the presence of the usual tests for albumen. * * * Suffice it to say that in very opaque urines the precipitate is quite abundant and corresponds when Esbach's test is applied, to from 1 to 3 per cent of albumen.

"The * * * hæmoglobinuria is * * * occasionally observed during life, and probably with the aid of a catheter may be seen much more frequently. * * * Very little need be said of the other characters of 'red water.' When found in the bladder after or collected shortly before death its specific gravity is usually low (1,010 to 1,020) and it is feebly alkaline or acid. There is no effervescence with acids. After standing, a few granular casts and rarely urates are found in the very slight sediment. The greater the number of days before death that it is collected the more nearly it approaches normal urine as regards specific gravity and alkalinity."

Starcovici reports bloody urine as regular in the severer cases, but not observed in the lighter forms of bovine hemoglobinuria. In carceag bloody urine is less frequent.

For canine piroplasmosis, Nuttall (1904, pp. 232–233) reports as follows:

South Africa: "All cases I have observed have been acute, and hæmoglobinuria was present, also albuminuria. The urine was claret or brownish-red in color, or resembling coffee grounds. Lounsbury and Robertson consider this brown coloring an unfavorable symptom, indicating a fatal termination. Hæmoglobinuria was noted by Hutcheon (1899). It may be absent in fatal cases, as in redwater (Robertson, p. 329). In one urine I examined I found the reaction acid, albumen, hæmoglobin, bile salts, and pigments, a considerable deposit consisting of spermatozoa (chiefly), granular casts, epithelium, leucocytes, granular detritus, crystals of salts, and a few erythrocytes. There were no spermatozoa or bile salts and pigment present on the day preceding death (dog 1). No hæmoglobinuria was observed in the chronic case recorded in Chart V."

France: "In acute cases urine albuminous at onset before parasites can be found in the blood. Albuminuria persists until death, increasing with number of parasites present. Hæmoglobinuria: Urine pink, dark red, blackish, like prune juice or coffee grounds, according to its degree. No erythrocytes in urine. Oxyhæmoglobin may amount to 2.5 per cent. Hæmoglobinuria appears soon after parasites are seen in the blood, and in very acute cases persists until death, and is found in bladder at autopsy. Hæmoglobinuria inconstant, noted in 3 out of 6 cases by Nocard and Almy; this may be due to its being at times very transitory. Nocard and Motas observed more or less lasting and severe hæmoglobinuria in 43 out of 63 dogs. Bile pigment present in cases showing icterus and hæmoglobinuria. Reaction acid only found neutral twice, alkaline once. Polyuria rare.

"In chronic cases urine usually slightly albuminous at start, condition lasting 15–20 days. Hæmoglobinuria very rare; lasts 1 to 2 days. Urine may be icteric. Reaction acid only once found neutral; this attributable to other causes (sugar found)."

It will thus be seen that while Wilson and Chowning report hæmoglobinuria as absent or very slight in "spotted fever," this is a prominent symptom in piroplasmatic diseases. If, therefore, "spotted fever" is a piroplasmosis, it differs in this very characteristic symptom quite markedly from other maladies caused by parasites of the same genus.

GENITAL SYSTEM.

See *Genitalia*, page 55.

Several women have been taken sick while pregnant. Case 121 was two months pregnant (Anderson, 1903c, p. 34), but apparently did not abort.

Wilson and Chowning (1904a, p. 42) report the uterus as apparently normal in the 3 females examined.

In case 11 (1904) patient aborted; the uterus, upon autopsy, measured 11 by 13 cm., was soft, but normal in appearance for a recently delivered uterus; on section it was normal; the vagina showed slight bloody discharge; the ovaries were normal; right ovary contained corpus luteum.

In case 3 menstruation occurred for one hour on May 14, and then stopped; it began again during the night of May 14–15, and flowed freely until death. Her former menstruation was on April 11, so that the disease appears to have delayed her menses. Case 5 menstruated just prior to attack.

RELAPSES.

Case 53 (Gwinn's patient 1899) relapsed after abortive treatment (Wilson and Chowning, 1903a, p. 35; Anderson, 1903c, p. 15). According to Gwinn (1902) relapse is favored by getting up from bed too soon, or by muscular exertion, or exposure to cold.

We saw no relapses in 1904.

COMPARISON.—In carceag there is usually 1 attack; in some cases there is 1 or 2 days of remission, then a second attack.

COMPLICATIONS.

Idaho.—Some cases develop rheumatic trouble, particularly of the larger joints.

Montana.—Hypostatic pneumonia, rheumatism, gangrene, and hemorrhagic diathesis seem the most usually to complicate the disease (McCullough, 1902, p. 226); pneumonia predominates in frequency as a complication, and such involvement of the lungs along with the predominating illness generally terminates the case.

Hypostatic pneumonia is a frequent complication for a day or so before death; one case had to all appearances genuine lobar pneumonia; one case gave well-marked symptoms of acute inflammatory rheumatism as complication; one case was complicated with abscess and gangrene (Gwinn, 1902).

The symptoms noted are sometimes complicated by gangrene, hypostatic pneumonia, articular rheumatism, etc.; hypostatic pneumonia sometimes develops; lobar pneumonia occasionally occurs as a complication, and usually hastens the end (Wilson and Chowning, 1904a, p. 40).

According to Anderson (1903c, p. 23) lobar pneumonia is a frequent complication in fatal cases; cases 44, 74, and 75 were complicated with pneumonia (1903c, p. 15).

CONVALESCENCE.

Idaho.—Convalescence is established during the third week, and is usually prolonged (Bowers, 1896). It is remarkably slow; and may be prolonged for months (Dubois, 1896, p. 64). It usually begins by or follows a stage of profuse sweating (Fairchild, 1896), and during convalescence the cough remains (Figgins, 1896, p. 64). Sweet (1896, p. 61), on the other hand, states that convalescence is usually rapid.

Montana.—Convalescence in case 78 began at the end of 23 days, and about the

twelfth day in case 103. It was very slow in cases 113 and 114, 10 or 12 weeks passing before the patients were able to work; health afterwards was not so good (Anderson, 1903c, pp. 14, 15, 16, 17). See also Duration, page 42.

Gates reports that his case No. 15 suffered from severe intercostal neuralgia during convalescence.

Comparisons.—In carceag convalescence lasts about 14 days.

PROGNOSIS.

Idaho.—Delirium or involvement of the nervous system is a bad prognostic sign; the amount of fever is deceptive, as fatal cases may have a temperature not exceeding 103° (Bowers, 1896, p. 64). Prognosis is, as a rule, quite favorable, if the patient is transferred to the lower valleys, where he can have home comforts and proper care; the disease appears to be more malignant in some localities than it is in others; the recovery is, in the majority of cases, complete (Maxey, 1899, p. 438).

Montana.—If the patient be promptly and thoroughly treated as here set forth (see below, p. 92) within 12 to 24 hours after the onset, nearly all attacks can be abated; but when the case is seen later, and the disease be not broken up, about 60 per cent or more prove fatal (Gwinn, 1902). In milder cases, where symptoms are not so marked, in which the jaundice, delirium, high temperature, eruption, and systemic infection are slight or entirely absent, the prognosis is usually favorable; it is very grave in typical severe cases, but there is no doubt that many mild and moderately severe cases recover; where the systemic infection is pronounced, jaundice very plainly discernible over entire body, delirium of low muttering type, hemorrhagic diathesis portrayed in the dusky appearance of the eruption, these cases invariably die; typical cases run a well-defined course, and patients surviving the fourteenth to sixteenth day are likely to recover (McCul'ough, 1902, pp. 25, 27).

In Montana, cases of the mild type of the disease, which show no spots, are as yet too indefinitely differentiated to permit of their inclusion with those of the severe type which invariably develop the eruption; that such cases exist there can be no doubt; they are never fatal; on the other hand, the cases which are marked by the eruption have a mortality of 70 to 80 per cent (Wilson and Chowning, 1902a, p. 133; 1903a, p. 65; 1904a, p. 40). It is unsafe to prognosticate a favorable termination in a case of mild initial symptoms, since many such cases rapidly become fatal (Wilson and Chowning, 1903a, p. 66). Prognosis of cases in Idaho, Nevada, and Wyoming is much more favorable (Wilson and Chowning, 1904a, p. 41).

The abundance of the eruption apparently bears no relation to the severity of the disease (Anderson, 1903c, p. 39).

Gates (1905, p. 115) states that in his experience the cases in children have been mild.

LETHALITY.

See also page 37.

Idaho.—The lethality is about 2.5 per cent; in fleshy subjects the disease is a serious affection, but particularly in the aged it is fatal; from the fifth to the eighth decade the lethality progressively increases from 5 to 50 per cent (Bowers, 1896, p. 64). Collister (1896, p. 63) reports that the death rate is not very high; in children it will not exceed 1 per cent, and in old age it varies from 4 to 5 per cent. According to Dubois (1896, p. 64), the lethality persists during the entire attack from 14 to 28 days; it is not high, but weak subjects, and even strong ones, succumb from intercurrent affections of the bowels, kidneys, or heart; it may be called a nonfatal disease. In Fairchild's experience (1896) the death rate is low, perhaps 2 or 3 per cent. Figgins (1896, p. 64) has seen but 1 fatal case in about 60 cases he treated, extending over a period of 14 years; death in this case, he believes, was superinduced by years of dissipation and by age. According to Springer (1896, p. 62) the death

rate is probably about 1 or 2 per cent, but higher in old people; death usually occurs from exhaustion. Sweet (1896) says that the death rate is slight, and Zipf (1896, p. 65) states that the disease is very seldom fatal; he has heard of only 1 fatal case this year (1896), and this patient had always been of weak constitution.

Montana.—Spotted fever is very fatal, but perhaps not more so than Asiatic cholera or yellow fever; it is more fatal in adult males than in women and children. Hanbidge reports 12 fatal cases in 16; Gwinn, 30 fatal in 40 severe cases; St. Patrick Hospital, 12 fatal in 15; McCullough, about 75 per cent fatal (McCullough, 1902, pp. 225, 227). Anderson (1903c, p. 38) gives the case mortality as about 70 per cent. The mortality varies within narrow limits from year to year; some years as many as 90 per cent of those attacked dying. Wilson and Chowning (1903a, p. 65; 1904a, p. 41) give tables showing the case mortality for various ages of males and females (see above, under "Sex and age," p. 37). Cases which are marked by the eruption have a mortality of 70 to 80 per cent, but cases without the eruption are never fatal (1902a, p. 133; 1903a, p. 65; 1904a, p. 42).

Comparison.—In Texas fever the lethality varies greatly. The time of the outbreak will largely decide whether practically all of the animals attacked die or all survive; a midsummer outbreak, when acute in its nature, is the most fatal. From this there may be all gradations toward the mild, nonfatal form of late summer. (Smith and Kilborne, 1893, p. 23). Starcovici gives the lethality as about 50 per cent for hemoglobinuria and 50 to 60 per cent for carceag.

In Texas fever death usually occurs from the fourth to the fourteenth day; in hemoglobinuria, in various stages of the disease; in carceag, usually from the second to the fifth day.

DEATH.

Idaho.—Death in adults and the aged results from toxemia and exhaustion (Bowers, 1896, p. 63). Death usually results from exhaustion (Springer, 1896, p. 62). It is usually due to lowered vitality from other causes, such as bad air and surroundings (Sweet, 1896).

Montana.—Wilson and Chowning (1904a, p. 41) have tabulated 88 cases with reference to date of death and have shown that in 69 of these death occurred from the sixth to eleventh days, inclusive. Adding to these statistics the cases recorded by Gates and those I have collected we find that death occurred on the—

	Cases.		Cases.
Third day in	1	Twelfth day in	6
Fourth day in	1	Thirteenth day in	4
Fifth day in	3	Fourteenth day in	2
Sixth day in	13	Fifteenth day in	2
Seventh day in	13	Eighteenth day in	1
Eighth day in	14	Twenty-second day in	1
Ninth day in	11	Twenty-seventh day in	1
Tenth day in	13	Twenty-ninth day in	1
Eleventh day in	9		

DIAGNOSIS.

SPECIFIC DIAGNOSIS.

Idaho.—After once seeing and recognizing spotted fever the diagnosis is easy; there is no occasion for making a mistake; even the laity recognize it on sight; its peculiar habitat and endemic character, the severe aching pains in the muscles, joints, bones, and head, the absence of gastro-intestinal symptoms, the temperature range, the

invariable appearance on the third to the seventh day of a profuse eruption of rose-colored, unelevated spots, first noticeable on the wrists and ankles, and rapidly spreading over the entire body, the frequency of constipation, and the marked debility noticeable during convalescence all go to make up a clinical picture characteristic only of spotted fever; in 3 or 4 cases in which I have used Ehrlich's diazo test the result has been negative (Maxey, 1899, pp. 436, 437). Epistaxis (see, however, p. 52), diarrhea, iliac tenderness, and gurgling are said to be seldom, if ever, present (Medical Sentinel, p. 457).

Montana.—Generally bad feeling, coated tongue, constipation, accelerated pulse and temperature, the expression denoting profound intoxication of the entire system with some grave illness, the unusual, intense soreness all over the body, affecting both bones and muscles, perhaps more marked along the spine and back of the neck and head, the icterus appearing from the fifth to tenth day of illness, and the characteristic eruption following, leave little room for doubt regarding the type of illness with which we have to contend (McCullough, 1902, p. 226).

According to Anderson (1903a, p. 508, 1903c, p. 39), cases occurring in infected localities and presenting a history of tick bites, chill, pain in head and back, muscular soreness, constipation, macular eruption, first on the wrists and ankles, appearing on the third day of illness, becoming petechial in character, do not present difficulty in diagnosing spotted (tick) fever; blood examination should be made in all suspicious cases.

While the different cases of spotted fever vary to no inconsiderable degree, this variation in symptomatology is perhaps not greater than it is in many other diseases. As for the blood examination to find the parasite, as a test in diagnosis, I must take the position that this is not at present upon a firm foundation. Ashburn and I are as expert with the microscope as the average physician, yet we were not able to find the parasite in the cases we examined, although we spent a total of 400 hours of actual microscopic work, equivalent to 80 days' work of 5 hours each.

DIFFERENTIAL DIAGNOSIS.

Idaho.—This disease differs principally in the occurrence of the symptoms from our occasional mountain fever, which seems to be similar to the mountain fever of the eastern Rocky Mountain region, a typhomalaria, or at least a modified typhoid (Sweet, 1896). "As I have known physicians to call it 'dengue fever,' cerebrospinal meningitis, typhoid, rheumatic purpura, typhus, and measles, I may be pardoned for taking up the differential diagnosis and calling your attention to the salient points of difference in support of the theory that this spotted fever is an independent, specific disease, and related in no way to any disease described in our text-books on practice."—Maxey, 1899, p. 436.

TYPHUS.

Idaho.—Maxey (1899, p. 438) states that he has known one or two physicians who invariably diagnosed spotted fever as "typhus fever," but he calls attention to the fact that typhus is an epidemic, contagious, malignant disease, more prevalent in the winter season and in thickly populated or crowded districts, and attacks men, women, and children alike; the onset is abrupt, with chill, followed by a violent fever and pain in the head; the eruption, red and measly, appears on the fifth to seventh day; there is also a peculiar mottling of the skin all over the body except the face.

Montana.—"Spotted fever more nearly resembles typhus fever than any other continued fever with which I am familiar, with the exception that it is not conta-

gious" (see above, p. 44).—McCullough, 1902, p. 225. Gwinn (1902) says: "I have been unable to find any marked difference by which to distinguish it [spotted fever] from this disease [typhus], save that typhus is by most authors given as a contagious disease, while the affection under discussion certainly is not contagious, and does not occur in crowded, filthy places, like jails, etc., as does typhus. Had this difference not existed I should never have claimed that this disease was one not described by medical literature. That one is contagious while the other is not is such a radical difference that one can not be justified in calling them the same. Either we have a disease to contend with heretofore undescribed by authors or most of our authors are mistaken as to the contagiousness of typhus." Anderson (1903c, p. 40) says: "Spotted (tick) fever, I think more closely resembles typhus fever than other disease, and cases of typhus fever occurring in a locality in which spotted fever prevails would, without a blood examination and close bedside observation, cause much trouble in diagnosis. In typhus we have a longer period of incubation, absence of a history of tick bites, the eruption which first appears on the abdomen and chest, its intensely contagious character, especially prevalent in the winter months, not limited to a short time in the spring, and marked nervous symptoms. As before mentioned, two cases of spotted fever have never been known to occur in the same family the same season (see, however, above, p. 43), thus conclusively showing the noncontagious character of the disease."

TYPHOID.

Idaho.—Many patients after a few days pass into a typhoid condition (Springer, 1896, p. 62). Epistaxis, diarrhea, and other abdominal symptoms, the scattering, petechial eruptions not appearing until the end of the first week, the absence of severe pains in the muscles of the limbs and back, the nerve symptoms, the characteristic tongue, and the greater prevalence in the autumn, render the differentiation quite plain; in the 3 or 4 cases of spotted fever in which Ehrlich's diazo test has been used the result has been negative (Maxey, 1899, p. 437).

Montana.—At the onset, the whole facies in spotted fever is in certain respects typhoid (Wilson and Chowning, 1902a, p. 132; 1903a, p. 62; 1904a, p. 37). Anderson (1903c, p. 39) says that typhoid resembles spotted fever, but the rose spots appearing first on the abdomen—papular in character—diarrhea, Widal reaction, and presence of typhoid bacilli in cultures from the blood of typhoid fever, and the presence of parasites in the red cells of spotted fever (see, however, above, p. 90), suffice to separate distinctly the two diseases.

MENINGITIS.

Idaho.—Before death, children have symptoms of meningitis (Bowers, 1896, p. 63). Maxey (1899, p. 437), in speaking of meningitis, refers to its occurrence in winter, its malignancy, its predilection for children, sudden onset with chill or convulsion, the severe headache or vomiting, painful rigid or contracted condition of the muscles of the back of the neck, with opisthotonos, the irregular fever, the scattering petechial or purpural eruption not present in all cases, the hyperesthesia, disorders of special senses, stupor or coma.

Montana.—Gwinn (1902) mentions in one case rigidity of the muscles of the neck and back, very much resembling that of cerebrospinal meningitis; when spotted fever is seen after the eruption begins to darken it may be distinguished from cerebrospinal meningitis by the absence of the typical vomiting, the rigidly contracted muscles of the neck and back, the herpes, and the irregular, low-averaging temperature of the latter disease. McCullough (1902, p. 225) considers spotted fever as distinct from meningitis. Anderson (1903c, p. 39) says in reference to meningitis that "the stiffness of the muscles of the neck, photophobia, sensitiveness to sudden noises, headache, and rigidity of the muscles of the back and neck, with the not altogether constant irregularly situated rash, should not cause much trouble."

It seemed to me that cases occur which are not so easily differentiated symptomatically from some cases diagnosed as cerebrospinal meningitis, as the above remarks would lead one to believe. However, "spotted fever" does not show post-mortem lesions which would justify us in classifying it as meningitis. (See also page 79.)

DENGUE.

Idaho.—Dengue or breakbone fever is an epidemic, contagious disease, found only in subtropical climates. Its onset is abrupt with chill and intense pains in head and back, followed by high fever for from 1 to 5 days, when there is an intermission of all symptoms for a day or two, followed by a second paroxysm of fever and pain. There is a scarlatinal rash during the first paroxysm, and a characteristic erythematous rash or rubeolus eruption accompanies the second paroxysm. Nausea and vomiting are common, and the average duration is only 8 days (Maxey, 1899.)

Montana.—Dengue is a disease of tropical or subtropical countries, whereas spotted fever occurs at an elevation of from 3,000 to 4,000 feet above sea level. The swollen joints, pleomorphic eruption over the joints, never petechial, apyretic period, and short course of the disease would differentiate it from spotted fever (Anderson, 1903c, p. 39).

PURPURA HÆMORRHAGICA.

Purpura haemorrhagica occasionally accompanies severe cases, the hemorrhagic spots becoming as large as the thumb nail (McCullough, 1902, p. 226). Purpura in this region [Bitter Root Valley] is manifested by the eruption being generally confined to the lower extremities, a less systemic disturbance than in spotted fever (Gwinn, 1902).

PELIOSIS RHEUMATICA.

In peliosis rheumatica, the sore throat, multiple arthritis with purpura and urticaria, and comparative rarity of the disease, offer a sufficiently distinct clinical picture (Anderson, 1903c, p. 39).

"BILIOUS FEVER."

"If seen at first, and it be a mild attack, it [spotted fever] very much resembles bilious fever or biliousness with constipation. However, if the patient has been chilly and the skin appears congested, the eyes congested as well as jaundiced, and it be at a locality and time when this disease may be suspected, it should not be overlooked in the diagnosis. If it be a severe attack, the danger of mistaking the two diseases is very much lessened by the pronounced chill, high fever, and inordinate aching, they being more severe than in biliousness" (Gwinn, 1902).

MEASLES.

Idaho.—If a case of spotted fever is not seen until the spots are well out, it might be mistaken for a case of measles; but when we take into consideration the epidemic and contagious nature of measles, the characteristic catarrhal symptoms referable to the respiratory tract, the elevated, crescentic, crimson eruption, and the presence of Koplik's spots, we should not hesitate long (Maxey, 1899, p. 438).

Montana.—When the eruption is new it may be mistaken for that of measles, but close examination shows the eruption to be purely macules, and not in the least elevated (Gwinn, 1902).

TREATMENT.

GENERAL PRINCIPLES.

Idaho.—"No abortive or reliable curative treatment has as yet been discovered. The disease is self-limited and a large portion of cases recover without any internal medication. Treatment of individual cases is governed by the rational and symp-

tomatic conditions present. On the theory that the infection enters through the alimentary canal, I employ intestinal antiseptics and evacuants and a supportive treatment."—Bowers, 1896, p. 64. "No medication will relieve the pain and fever; but quinine, dissolved in aromatic sulphuric acid, in comparatively large doses, gives the best results to those who can withstand the treatment."—Dubois, 1896, p. 64. "It is a self-limited disease, and drugs have little or no effect upon the attack. I treat on expectant plan principally. When pain is severe I control it with morphia, and to lessen the hyperæmia of the cord, etc., I usually give a mixture of bromide and ergot. When temperature goes above 103° F., I bathe with tepid water, and, if indicated, give small doses of acetanilid or phenacetin. Quinine has given no results in my hands. I keep patients in a recumbent position constantly, overcome constipation with salines, and confine them to a milk diet."—Fairchild, 1896. "I never employ any other than such as is used in ordinary malarial fevers, and that of symptomatic nature."—Figgins, 1896, p. 64. "Expectant. Morphine for pain; salines for constipation; sponge baths and antipyretics for high temperature. Diet, milk."—Springer, 1896, p. 62. "Milk diet; a cholagogue followed by frequent alcohol hot baths, with usually very little positive medication. As routine, I usually relieve the dengue ache with salol, quinia salts, and some coal-tar products in very small doses until free diaphoresis is obtained. I give little but a placebo in mild cases."—Sweet, 1896. "Treatment is entirely symptomatic. The hygienic and sanitary surroundings should be the best possible to obtain. Frequent baths and changes of bedding add materially to the patient's comfort. For the fever I usually use cold sponging, with occasional doses of acetanilid or phenacetin and codeine, or Dover's powders may be required to relieve the pain and restlessness. During convalescence stimulants, iron and bitter tonics are in order."—Maxey, 1899, p. 438.

Montana.—"I have tried many remedies, but found most of them to do but little or no good, and often harm if they should be pushed in amount or number. The treatment which has served me best is what might be termed eliminative and supportive treatment. The old rule of keeping the head cool and feet warm should be closely observed. The patient should be frequently turned in bed after the disease is well established, in order to prevent hypostatic pneumonia, and to cool the underside of the body, which may be superheated while the upper side is cool."—Gwinn, 1902. "The best results are obtained by systematic and eliminative treatment" (McCullough, 1902, p. 227). "Until the past season [1903] the treatment of this disease has been purely symptomatic, but after the discovery of the parasite, Doctor Wilson and the writer suggested the use of quinine in large doses, preferably hypodermatically" (see below, p. 94).—Anderson, 1903c, p. 40. "Many drugs have been used in the treatment of 'spotted fever,' but while some of them are important as stimulants, sedatives, etc., none of them—except perhaps quinine—seem to have any specific action on the disease through destruction of the parasites."—Wilson and Chowning, 1904a, p. 57.

SURROUNDINGS.

"The room should be kept dark and as free from noise as possible."—Anderson, 1903c, p. 41. "Darkening of the room and hot sponge baths add much to the comfort of the patient."—Wilson and Chowning, 1904a, p. 57.

DIET.

Idaho.—"The diet and bowels should be properly regulated, particularly after the eruption is well out, for I have found in the majority of my cases that at this time the appetite is apt to return to the patient, and the physician's judgment will be taxed to decide just what and how much food may be allowed."—Maxey, 1899, p. 438.

Montana.—"Milk, buttermilk, broths, soft eggs, and soft toast may all be allowed. The whisky may be administered in an eggnog."—Anderson, 1903c, p. 41.

TICK BITE.

"As soon as a person is bitten by a tick, the insect should be removed and the place cauterized with 95 per cent carbolic acid. There is sometimes difficulty in removing the tick, but by applying ammonia, kerosene, or carbolized vaseline it can usually be detached without trouble."—Anderson, 1903a, p. 41.

"As soon as a person is bitten by a tick, the arachnid should be removed and the wound cauterized with a 95 per cent carbolic acid."—Wilson and Chowning, 1904a, page 56.

In 3 cases during 1904 in which this precaution was adopted it did not seem to inhibit the development of the disease, for all 3 patients died. Further, while the precaution may be good, so far as the tick bite is concerned, I do not see what good effect may be expected from it in aborting a piroplasmosis.

PURGATION.

"To act on the bowels, if the patient be an adult and robust, I begin by giving 20 grains of the mild chloride of mercury well rubbed together with the same amount of sodium bi. carb., to be followed in 12 hours by an ounce dose of magnesium sul. This will keep the bowels active for 1 to 3 days, after which I give the same remedies in sufficient quantity to make the bowels act 2 or 3 times every 24 hours."—Gwinn, 1902.

"For cases encountered within the first 3 or 4 days I begin with: R. Hydrarg. chlor. mite et sodii bicarb., āā. gr. i. Tablets no. xii. Sig.: One tablet every half hour until all are taken.

"This rarely fails to produce free purgation and unloads the intestinal canal, thereby relieving any tendency to hepatic engorgement. Should catharis be insufficient, a clyster containing a teaspoonful of turpentine should be given. Then with a view of eliminating the noxious accumulations in the system, and in some degree modifying the jaundice, give: R: Sodium phosphatis ʒii. Sig.: Teaspoonful given in hot water every 3 hours

"By administering this remedy at above-named intervals the bowels are kept free, diaphoresis increased, and muscular soreness diminished."—McCullough, 1902, pp. 227–228.

In case 118, bowels were kept open with calomel (Anderson, 1903c, p. 28).

QUININE.

"In 5 cases in which it [quinine] was used systematically and in large doses the results were most happy, all recovering. Five cases which did not have the treatment died. Of course, 10 cases is too small a number on which to base very positive conclusions, but I hope that the use of quinine will be followed in the future treatment of the disease. Quinine bimuriate, 1 gram, should be given hypodermically every 6 hours. If there is great objection to the use of the needle, the sulphate, 1 gram every 4 hours, may be given by mouth; but the irritable condition of the stomach at times may prevent. The use of quinine should be begun as soon as the diagnosis is made, and persisted with in decreasing doses as convalescence begins. Some of the valley physicians seemed to fear that quinine depressed the heart and caused nervous symptoms; but I am of the opinion that the great good the drug does more than counterbalances these effects. I strongly advise the early and continuous use of large doses of quinine." Anderson, 1903c, p. 40. "The good results that have followed the administration of large doses of quinine—the 5 cases in which it was used having recovered—give much hope that this disease, which is so much dreaded, may in future be robbed of many of its terrors."—Anderson, 1903c, p. 7.

Quinine has been used by the physicians of the Bitter Root Valley in small doses by mouth for a number of years in the treatment of this disease. During the spring of 1902 the writers urged, on purely theoretical grounds, the use of large doses of the drug intravenously, hypodermically, or per rectum. Cases 94 and 95 were given quinine per mouth and rectum to the point of cinchonism, with some apparent beneficial results. Both cases died, though No. 95 was convalescent from spotted fever and died of a complicating pneumonia. During 1903 cases Nos. 115, 118, 119, 122, 124, 125, and 126 were treated with doses ranging from 5 grains by mouth (case No. 119) to 60 grains subcutaneously (case No. 118). All of these cases except No. 125 recovered, though case No. 119 died later of a complication (acute gastritis). Case 125 was an old debilitated man, and had the most abundant infection of all the cases examined by the writers. The remaining 5 cases occurring in 1903 and untreated with quinine were all fatal. These cases are too few on which to base conclusions, but are sufficiently suggestive to warrant a further trial of the treatment. In this connection it is worthy of note that Theiler (1903) has recently made the observation that in South African equine malaria, a disease caused by *Piroplasma equi*, the pyroplasmata rapidly disappear from the blood of infected horses on the administration of quinine and ammonium chloride."—Wilson and Chowning, 1904a, p. 57.

In connection with the cases which occurred in 1903, it may be noted that Gates's case (No. 10, 1903) was not reported as having been treated with quinine, but recovered. As this case was known to Anderson and to Wilson and Chowning, it must be concluded that they did not include it in the statements that the 5 cases which did not have quinine treatment died.

So far as I was able to learn, the quinine treatment in large or comparatively large doses is not new in the Bitter Root Valley. Doctor Mills, for instance, gave quinine in large doses per mouth some years ago, but his results were so unsatisfactory that he discarded its further use. Doctor Howard has been giving quinine for 6 years in large doses twice a day, and claimed to have had very good success with it up to 1904. He states that under quinine treatment 6 out of 7 cases recovered. Doctor McGrath has been using quinine for about 5 years; of 6 quinine cases 5 recovered. One physician has lost 2 or 3 cases under quinine treatment—3 grains every 2 or 3 hours. Doctor Brook informed me that one of his quinine cases died after 12 days of treatment; he also lost another quinine case. Doctor Gwinn used quinine from 1887 to 1890; some patients recovered, others died. He then rejected it. He tried it again about 1897, giving about 1 dram per day, but as all of these cases died he again rejected it. He used it in 2 cases in 1903, and one case recovered; the other died under the same treatment.

Quite a number of local physicians with whom I spoke maintained that except in imported malaria cases quinine could not be given in that locality so freely as in lower eastern and southern localities, without more serious effect, but this view was not shared by two other physicians.

Early in 1904 Doctor Mills stoutly combated the quinine treatment

on the ground that his experience with it had been so unsatisfactory. Ashburn also had grave doubts regarding its value and in case 3 he opposed it rather strongly.

In the 2 Bitter Root Valley cases of 1904 which recovered, no quinine was used, and in the 5 cases in which it was used the patients not only showed no improvement, but died; in cases 12 and 13 also it was not used and the patients recovered.

COMPARISONS.—In connection with the statement by Wilson and Chowning quoted above, that Theiler observed that *Piroplasma* in the blood of horses in South Africa rapidly disappear on the administration of quinine and ammonium sulphide, it might be added that Nuttall (1904, pp. 248–249), in discussing canine piroplasmosis, says:

"Apart from the specific treatment recorded above, there is very little to note regarding treatment. In South Africa, Hutcheon (1893, p. 477, and 1899, p. 400) recommended the use of repeated doses of ammonium chloride and belladonna, a form of treatment tried by Borthwick at Port Elizabeth with 'excellent results.' Subsequently Hutcheon obtained encouraging results from the use of quinine, benzoate of soda, and carbolic acid. Robertson (1901, p. 332) states that he has tried quinine, calomel, ammonium chloride, extract of belladonna, carbolic acid, and finally benzoate of soda without satisfactory results. Carbolic acid appeared, in fact, to hasten death. He obtained the best results from a 'calomel pill to start with, then a calomel and quinine pill four times a day.' Without stating the dose, he says that very large amounts of calomel are needed. Hutcheon does not appear to approve of the calomel treatment. In other words, the evidence as to treatment in South Africa appears to be somewhat contradictory.

"In Europe, Piana and Galli-Valerio (1895) attributed the recovery of the one dog they saw suffering from Piroplasmosis to the use of quinine. Almy (10, x, 1901, p. 379) treated his dogs with quinine bromhydrate, but observed no effect therefrom, the remedy being as ineffective as quinine has been shown to be in the treatment of Tristeza (*Piroplasmosis boris*).

"Evidently there is no known remedy for canine Piroplasmosis, and it is open to question whether or no the dogs which have been successfully treated would not have recovered anyhow."

CALCIUM SULPHIDE AND CREOSOTE.

"Some physicians speak well of calcium sulphide, and others of creosote."—Anderson, 1903c, p. 40.

PAIN.

"For the severe pain in the head and back during the first week, Dover's powders or morphine may be used."—Anderson, 1903c, p. 40.

Morphine is used in this disease by a number of the local physicians, and Mills called my attention to the large doses which the patients could take. Case 11 (1904) in one day received 9 grains within 8 hours, and at another time 4.5 grains in 2 hours without noticeable effect.

SKIN.

"To produce an active skin, lower fever, and reduce pain, phenacetin acts well, and may be given as seems to be needed in 10 to 15 grain doses for a few days succeeding the attack, without lowering the patient's vitality to a noticeable degree."—Gwinn, 1902.

BATHS.

"After from 2 to 4 days, when the chilliness has ceased and when the patient will tolerate cool baths, I substitute them for the phenacetin. It often occurs in the later stages of this malady that the surface of the body will be cold and the axillary temperature even below normal, resultant from the obstructed or weakened circulation, while the thermometer will show a high fever if registered in the rectum. In this event a blanket may be added to the patient's covering to warm the periphery and extremities of the body."—Gwinn, 1902.

Anderson (1903c, pp. 37, 40) reports that warm sponge baths or packs relieved the congestion of the skin, reduced the fever, and allayed restlessness; the spots became brighter after the bath.

ENEMATA.

"An enema of cool normal salt solution lowers central temperature, and supplies the kidneys with fluid with which to eliminate poison from the system."—Gwinn, 1902.

FEVER.

"For the fever warm spongs baths or packs are useful and refreshing to the patient. After a bath the spots lose their dark color and become much brighter."—Anderson, 1903c, pp. 40–41.

DIURESIS.

"To produce an active diuresis I know of nothing better than to encourage the patient in drinking an abundance of fluid; potass. acetate and liq. amm. acetas act well during the first stages, while digitalis is well given during the latter stages when also needed to support the heart. As a drink during the first stages I recommend water, lemonade, and buttermilk. I have seen excellent results from beer as a heart stimulant and diuretic after the patient becomes weak. All drinks should be cold and taken frequently in small amounts."—Gwinn, 1902. "The patient should be encouraged to drink large quantities of water to flush out the kidneys." Anderson, 1903c, p. 40.

HEART.

"For a fagged heart and respiratory effort I know of nothing better than strychnia, digitalis, and alcoholics."—Gwinn, 1902. "Heart action should be watched, and any tendency to weakness of that organ should be stimulated by one-thirtieth grain strychnia given hypodermatically as directed."—McCullough, 1902, p. 228. "The heart should be supported by strychnine, whisky, or other appropriate cardiac stimulants."—Anderson, 1903c, p. 40.

DELIRIUM.

"For the active delirium and sleeplessness I have found no more effectual remedy than: R. Chloral hydratis et Kali bromidi āā. ʒiii; tinct. hyocyam., ʒii; flu. ext. glycyrrhiza. ʒss; aq. menth. pip. q. s., ʒiii. M. Sig. 2 teaspoonfuls in 2 tablespoonfuls of water when very restless, and repeat in 6 hours if needed.

"The high fever and moderate delirium call for the ice bag applied to the head, cold sponge bath if not too much body tenderness, and occasional 10-grain doses of antipyrine."—McCullough, 1902, p. 228.

SALINE SOLUTION.

"Rectal and subcutaneous injections of normal salt solutions were given. The combined use of the above and hot packs, together with hot elder water and liquor ammonii acetatis internally, produced only slight diaphoresis, and that mostly about the head."—Gates, 1903, p. 49.

Normal salt solutions, in rectal, vesical, and subcutaneous injections, were repeatedly used in 1904 with good temporary effect.

OXYGEN.

Oxygen was repeatedly resorted to in emergencies in 1904 with temporary good effect.

BLEEDING.

Case 11 (1904) was bled twice with very marked temporary relief. On June 27, p. m., about 12 ounces of blood were taken, after which the patient had a good night's rest; the next morning the spots were much lighter in color. On June 28, p. m., about 1 ounce of blood was taken, followed by noticeable temporary improvement; the next morning the spots were lighter, but the patient died later in the day.

SUPPORTIVE TREATMENT.

"As supportive treatment as well as germicidal, and having a particular action on the blood, I have found useful: R. hydrarg. bichlor., gr. i; liq. pot. arsenitis, ʒ ii; tinct. ferri chlor., ʒ ss.; acid phos. dil., ʒ i; syr. limonis q. s., ʒ vi. M. sig. Teaspoonful in wineglass of water four times a day.

"In addition to the above medication, the special symptoms require careful treatment best adapted to such complications individually."—McCullough, 1902, p. 228.

PREVENTION.

"In the way of prophylaxis, I have advised an occasional dose of calomel, the drinking of boiled water, and thorough protection against cold and wet."—Gwinn, 1902.

"If, as seems very probable and almost proved, the tick is the means by which the disease is spread, the question of the prevention of the disease resolves itself into the destruction of the tick. This is an almost impossible task over such a large area, especially of such varied topography. When conditions will permit, burning the undergrowth and stubble will be an effective method for the destruction of ticks. This may be done either in the early fall or, preferably, in the early spring, when the ticks are just beginning to move about."—Anderson, 1903c, p. 41.

"In view of the almost certain rôle of the tick in the conveyance of pyroplasmosis to man, measures should be taken to reduce the numbers and limit the spread of this arachnid. The burning of underbrush, sawdust, etc., wherever practicable, is recommended. Persons going into the brush in the infected area should use all possible precautions to prevent ticks from biting them. As soon as a person is bitten by a tick, the arachnid should be removed and the wound cauterized by 95 per cent carbolic acid."—Wilson and Chowning, 1904a, p. 56.

Definite statements regarding prevention can not be made until the cause of the disease and its method of transmission are definitely known. All that can be said at present seems to be that people in the valley should take the best possible care of themselves during the spring months, and in case one member of a family is taken sick no other person should occupy the bed with the patient.

SEQUELÆ.

Idaho.—Dubois (1896) states that no constitutional symptoms are left. The only sequela which Maxey (1899, p. 438) has noticed is a little stiffening of the knee joints, lasting some weeks.

Gates (1905, p. 115) reports endocarditis as sequela in his case 8.

One of the cases of 1903 had not fully regained the use of his legs in the summer of 1904.

Autopsies.

The following autopsies have been made:

Idaho.—Apparently none.

Montana.—1897: Case 35, by Buckley, reported briefly in Wilson and Chowning, 1903a, page 33, and Anderson, 1903c, page 13. 1902: Case 89, by Wilson, Longeway, Brethauer, and Buckley, reported in Wilson and Chowning, 1903a, page 50; case 91, by Wilson, Brice, Longeway, and Buckley, reported in Wilson and Chowning, 1903a, page 52; case 93, by Wilson and Spottswood, reported in Wilson and Chowning, 1903a, page 54; case 94, by Wilson, Chowning, and Buckley, reported in Wilson and Chowning, 1903a, page 56; case 97, by Chowning, reported in Wilson and Chowning, 1903a, page 55; case 107, by Wilson and Longeway, reported in Wilson and Chowning, 1903a, page 47. 1903: Case 120, by Anderson, Wilson, Gwinn, Mills, Olson, Pixley, and Spottswood, reported in Anderson, 1903c, page 32. 1904: Case 11, by Ashburn, Mills, and Stiles, reported in Stiles, 1905, p. 109–110.

(?): Case —, by Hanbidge and Gwinn, reported briefly in Gwinn, 1902.

(?): Case —, briefly referred to by Gwinn, 1902.

Pathology.

Idaho.—"The pathological conditions, as at present understood, may be described with a large-sized interrogation point."—Maxey, 1899, pp. 434–435. Fairchild (1896) has seen no autopsies. The lesions, so far as Figgins (1896, p. 64) knows, are confined to the skin.

Montana.—Gwinn (1902) reports slight enlargement of the liver and kidneys, together with slight degeneration, in one case on autopsy.

The gross lesions are very uniform: intense rigor mortis appears early (Wilson and Chowning, 1904a, p. 41).

The changes are those which can be ascribed to interference with capillary circulation; the extravasation into and pigmentation of the skin account for the persistence of the spots for long periods after the recovery of the patients; there is acute parenchymatous degeneration of the heart muscles, spleen, liver, and kidney; the central nervous system is but little affected (Wilson and Chowning, 1904a, p. 43).

Anderson (1903c, p. 38) summarizes the post-mortem findings in 7 cases (6 of 1902, 1 of 1903) in the Bitter Root Valley.

Rigor mortis was intense in cases 89, 91, and 93 (Wilson and Chowning, 1903a, pp. 50, 52, 54), but not in case 120 (Anderson, 1903c, p. 32).

For further details see special organs.

Clinical histories.

During my work I did not take clinical notes, as I had so many other things to occupy my time. Clinical notes were, however, taken by the attending physicians and to some extent also by Doctor Ashburn. As both Wilson and Chowning (1902a, 1903a, 1904a) and Anderson (1903a, c) had studied the clinical side, and as I was looking especially for the life history of the parasite as a basis for prevention, I naturally neglected to go into an especially detailed clinical study, blood

counts, etc., such as I should have done had time permitted. Furthermore, some of the hospital charts became lost, so that part of the notes had to be written from memory or from brief memoranda.

BITTER ROOT VALLEY CASES, 1904.

1904. Case 1.—Attending physician, Doctor Buckley (notes written from memory later by Doctor Buckley: No microscopic examination of blood).

Male, age 66 years. Seen by Doctor Buckley, April 29, 1904. When first seen he had two wood-tick bites in the right inguinal region, each surrounded by an indurated inflammatory zone and showing an area of blackened skin about $\frac{1}{16}$ inch in diameter at the center. Patient had frontal headache and general body aching. Temperature 102° F. Thought he had caught cold.

April 30, temperature 103°. Sclerotics much injected and face congested. Tongue moist. Pulse not high.

May 1, restless. No sleep night before. Bowels open. Temperature 103.5° F., a. m.; 104°, p. m. Mottling of skin noticed. Ecchymotic spots the size of a dollar seen on the abdomen. Scrotum black. That evening patient was feeling better and Doctor Buckley was telephoned he need not see him. May 2, worse. Spots dark purple. Temperature, a. m., 104°; p. m., 105°. Began to have delirium, which soon passed to coma, which lasted until death, on May 3, 1904.

1904. Case 2.—Attending physician, Dr. J. J. Buckley (notes written later by Doctor Ashburn from hospital chart and from statement by Doctor Buckley: Patient seen by Doctors Buckley, Ashburn, and Stiles; microscopic examination of dried blood from lips by Doctor Stiles, unsatisfactory).

Female, age 7 years 4 months. Born in Ravalli County, 15 miles south of present home.

Family history, negative.

Personal history (previous to present illness): Scarlatina in fall of 1903, mild. No other sickness.

Present illness: April 24, 1903, bitten back of ear by 3 ticks. Bites caused swelling next day. No discoloration; swelling was over upper part of sterno-mastoid, below bites. Had fever from that time on.

April 27, eruption showed on arms, shoulders, hips, knees, ankles, palms, and soles. Mother thought she had measles. She had also complained of sore eyes, some pain in back and head. Continued that way with increasing eruption and some darkening of it (browning) until May 2, when seen by Doctor McGrath and pronounced spotted fever. Brought to Missoula May 3. Complained of intense general soreness after appearance of eruption.

May 4: More irritation in throat. Very sore to the touch. Very drowsy. Cramps in stomach. Restless in afternoon.

May 5: Headache. Vomited twice. Sore throat. Very restless all night. Rash darker in color but not sore to the touch; patient very thirsty.

May 6: After sponging rested all forenoon, retained milk, some water, and all medicine. Throat much better. Not so thirsty as yesterday. Rested all afternoon. No vomiting. At 5 p. m., cramps in stomach.

May 7: Respiration a little more irregular; retains everything; apparently weaker, but pulse good; p. m., breathing very poor, but change for the better late in the

evening; breathing easy after a hot bath; slept calmly about two hours; became very restless again about 10.30 p. m.; pulse very poor at 12.

May 8: Choking spells about 12.30 a. m.; breathing very heavy; finally becomes unconscious; eyes bloodshot, pupils very much dilated; spots out in profusion; pulse good, but great struggle at times for breath.

May 9: Patient very restless during the night until about 3 a. m., then sank into a comatose condition; much weaker; spots very purple; p. m., sinking spell; improved after subcutaneous saline injection; lungs filling up; mouth full of bloody matter.

May 10: Finger nails turned purple, beginning at the tips about 1 a. m.; died with very little struggle at 1.25 o'clock a. m.

1904. Case 3.—Attending physician, Doctor Fitzgerald (notes prepared partially from hospital records, partially at bedside by Doctor Ashburn; patient seen and microscopic examination of fresh and stained blood by Doctors Fitzgerald, Buckley, Ashburn, and Stiles).

Female, age 28 years. Born in New Brunswick.

Family history: Father died of piles (?), one brother of consumption. Mother and four brothers and sisters living and well. History otherwise negative.

Personal history (previous to present illness): Had 3 children. Measles in childhood, bowel trouble last summer. No other sickness.

History previous to admission: Moved to present home in January. Had great fear of ticks and has watched for them and has not been bitten by them. Has had bites by chicken lice—last, probably about April 27. May 3 had chill. Backache and fever followed, and on May 7 eruption appeared on arms. Since that time, too, throat has been very sore.

Condition on admission: Date, May 10. First seen by Doctor Fitzgerald on May 7, 1904.

Symptoms: Sore throat, fever, slight malaise, but claims to feel well. Body presents on all parts a discrete maculo-papular eruption, very measly in appearance, but no crescentic arrangement. No purpura or petechiæ. Fauces and pharynx much injected and show adherent mucus-pus. Cervical glands enlarged. No eruption in mouth or throat. The tongue tremulous and has heavy, white, moist coat. Face placid. Blotched with eruptions. Eyes normal; mind clear. Heart sounds clear and regular. Lungs—Respiration slightly harsh and prolonged anteriorly; more so behind. Blowing over right upper lobe posteriorly. No cough. Spleen enlarged. Liver not enlarged. Abdomen not distended, tender, or painful. Menstruated April 11th. Bowels have been regular; now loose from salt enemata. Urine passed normally.

Diagnosis: Spotted fever.

Treatment: Seventh to 10th, calomel first day and quinine sulphate, grains 15, every 4 hours. Next day salol, grains 5, and quinine, grains 10, every 4 hours. Strychnine, grain, $\frac{1}{30}$, every 6 hours. Saline enema, 1 pint every 3 hours. Probably none of this was carried out as prescribed.

May 11: Arrived at Sisters Hospital this a. m. Nervous on admission. Looks and says she feels better. Throat not so sore. Spots unchanged, possibly a trifle darker. Not petechial. Skin presents faintest yellow tinge. No jaundice of sclerotics or tongue. Bowels moved after salt solution. Was very drowsy all afternoon; restless between 4 and 5 o'clock. Retained saline enemas. Patient apparently thirsty. Took nourishment; resting easy at 7.30 p. m.

May 12: At about 8 o'clock became drowsy; by 8.30 very stupid. Stupor continued all night with rapid respiration. Coma from 12 p. m. to 3 or 4 a. m. Bladder full at 3 a. m., when it was emptied for vesical irrigation. Now, 9.25 a. m., in deep sleep or coma. Pulse 80 and of good quality. Respiration 39 and slightly stertorous.

Blotches have disappeared from face and ordinary spots abundant there. Spots generally darkening. Now plum color, but not distinctly petechial. Lungs—Sounds very harsh all over front and back, with large coarse râles; 6 p. m., comatose all day; respiration rapid. Face dusky.

May 13: Quinine discontinued last night. Patient then comatose, breathing very rapidly and promised to die in a few hours, at most, as lungs were rapidly filling with fluid. This morning face darkly flushed, coma deep, great perspiration. Respiration will be for a time quiet and regular, for 5 or 10 minutes, and then very rapid (60) for a time. Spots no darker.

May 15: Filling of lungs progressed yesterday; edema well marked. Spots increased in size and darkened. Menstruation began for 1 hour yesterday and stopped. Began again last night and flowed freely until death. Urine and feces passed involuntarily and abundantly. Coughed freely yesterday. Finger nails this a. m. dark, face purple, much noise from breathing. Extremely offensive odor to breath. Dependent side of face turned black immediately after death, at 8 o'clock a. m. to-day.

1904. Case 4.—Attending physician, Doctor Merrick (notes prepared by Doctor Ashburn, from memoranda by Doctor Merrick; patient seen and microscopic examination of blood made by Doctors Merrick, Ashburn and Stiles).

Diagnosis: Spotted fever.

May 12. Symptoms: Severe intra-cranial and supra-orbital neuralgia, supra-scapular and lumbar pains. Tongue slightly furred and fissured. Anorexia. These symptoms dated from May 8, 1904. An eruption covering the legs to the knees.

May 13: The neuralgia considerably improved under codeine. The eruption increasing. Complains of illusions. Anorexia.

May 14: Eruption elevated and extended over trunk to face and arms. General condition prostrated. Pulse full. Blood examination for parasites negative.

May 15: Eruption dusky in morning, becomes brighter through the day, completely covers body and palms of hands. Complains of pains in head. Sleeplessness and illusions and weariness. Many red blood corpuscles are deformed. Blood examined by Doctor Ashburn.

May 16: Eruption possibly cleared. Patient feeling better; bowels moved freely. Temperature, 100; respiration, 20; pulse, 80. Anorexia and illusions persist, with prostration. Doctor Ashburn and Doctor Stiles made blood examination. No organisms were found.

May 17: Headache continues; fever 101.4. Illusions and prostration. No eruption on back, fading on arms and face. General condition of anxiety.

May 18: Headache relieved; condition improved. Eruption fading. Temperature, 100; pulse, 90. Sat in chair for one hour. Complains of weakness.

May 19: Temperature normal; pulse and respiration normal. Illusions disappear. Spots fading, only perceptible on limbs. Pains gone; tongue still coated; appetite returning.

May 21: Patient sitting up; weak and extremely nervous; spots about entirely faded away. Appetite improved and patient sleeps well. No anatomical lesions apparent.

N. B.—Doctor Merrick's note about the eruption covering the whole body evidently refers to its general distribution, as it was nowhere abundant, and was the lightest eruption occurring in any case this year.

1904. Case 5.—Attending physician, Doctor Mills (notes prepared by Doctor Ashburn; patient seen and microscopic examination of blood made by Doctors Mills, Ashburn, and Stiles).

Female; school-teacher; age 24 years; single.

Family history: Parents and brothers and sisters are living and in good health. No hereditary disease in family.

Personal history (previous to present illness): Measles in childhood. No other sickness. Been living at Woodman, in this county, since last September, teaching school. Kept house. No chickens. Just finished menstruating.

History previous to admission: Positive she has had no tick bites or other bites of any kind. Chill night of the 12th. Headache and fever continued then to present. Came here next day. Eruption appeared yesterday afternoon.

May 16. Symptoms: Quite calm facies; tongue has heavy, white, moist coat. Throat slightly sore, but shows no marked injection. No appetite. Bowels at present slightly constipated. Face slightly flushed. Spots on face, arms, hands, and body, etc. Heart sounds clear and regular. Breathing normal and sounds clear, front and back. Splenic dullness increased. Hepatic apparently diminished. Passing urine normally. Vomits practically everything taken by mouth. No nausea or pain in stomach. Headache present.

May 18: Condition good. Heart rapid and slightly weak and patient vomits a great deal, but otherwise is doing well. Bowels O. K. Patient slept well last night. Spots not brighter and seem to be fading. Mind clear. Headache improved.

May 23: Informed to-day by Doctor Mills that patient continued to do well until afternoon of May 21, when her respiration ceased. As heart was acting well, artificial respiration was resorted to. In five minutes patient breathed, roused, and was quite rational. Failure of respiration continued to occur at intervals, patient apparently forgetting to breathe. She died about 7 a. m. May 22. Hypodermics of morphine apparently acted as respiratory stimulant (Dr. Mills). Urine examined by Ashburn, passed May 18, contained albumen; reddish, yellow, turbid, acid, 1.028; no microscopic examination.

Later notes: Temperature on fourth day was 103° F. On day of death was 104° F. Treatment consisted principally of morphine in considerable doses. Diet was light, but almost everything was vomited unchanged soon after taking. This was also true of beer, which the patient had requested.

1904. Case 6.—Attending physician, Dr. Charles Pixley (patient seen and microscopic examination of blood made by Doctors Pixley, Mills, Ashburn, and Stiles).

Report of a Case of so-called "Spotted Fever" in 1904. By Charles Pixley, M. D.

Male patient, F. W., aged 21 years, of rather neurotic temperament. At one time he had used tobacco to excess. Two weeks prior to my first visit, which was on May 16, 1904, he had worked at Harvey Creek, a few miles east of Rock Creek, and parallel; also opposite to Quigley.

Patient was bitten by ticks at Harvey Creek; also at home at Missoula after his return. He had brought bedding home with him.

He had been complaining for about a week before I saw him. Two days prior to my visit he had fallen against the door at home, and he then went to bed. When first seen by me he had a temperature of 103° F., with headache, backache, and legache; also felt restless. Small pink macular spots were present on legs, arms, and trunk; face of a florid complexion, but without visible spots.

May 16, p. m.: Temperature, 103; respiration rapid; pulse good, but rapid; nervous symptoms marked; restless; talk a little incoherent.

May 17, a. m.: Temperature, 102; nervous symptoms more marked; p. m., temperature rising until 9 p. m., 103.6; rash still more marked.

May 18, a. m. Temperature, 101.8; p. m., 103.8; restless, twitching of muscles, labored breathing, rash becoming purple and more plentiful.

May 19: Temperature about the same as on previous day; pulse weaker; extremely nervous, restless; respiration rapid and labored; delirious all the time; thrown into a state of tonus when touched lightly anywhere on the body.

May 20: All symptoms more marked; temperature about the same; rash profuse; delirium the most marked symptom; general rigidity of all parts of the body; twitching of face muscles; unable to swallow.

May 21: Died at 2.30 a. m., preceded by coma; temperature at 1 o'clock was 104.

Treatment was at first purging with calomel; continued use of calomel in ¼-grain doses every 4 hours, also inunction of blue ointment; quinine per mouth in 5-grain doses; whisky, strychnine, and morphine throughout the last 3 days.

The following additional notes on this case were made by Doctor Ashburn:

Personal history (previous to present illness): Working in lumber camp on Harvey Creek, where no spotted fever had previously been.

Present illness: Bitten by tick at Harvey Creek about May 5 (date not exact). Bitten again after coming home. May 13, taken sick with chill, fever, general pain, and aching. 14th, fell in door, sick. Seen by Doctor Pixley May 16. Temperature 103.5° F. Slightly delirious. Nervous twitchings and trembling. Spots beginning to appear on flanks and legs, unusually small. Temperature ranged higher in afternoon (to about 104°). Kept growing worse, twitching, tossing all the time. Spots increased gradually and darkened until at death patient had blue areas the size of the hand. Became more sensitive and irritable until at last the slightest touch would cause violent jump, almost a convulsion. Breathing irregular when awake, regular when sleeping. Had sore throat early and very slight nose bleed. Bowels O. K. Raving almost to time of death. Comatose for a few hours. Urine not examined. Physical examination negative except the skin. Rash when seen by me very fine.

Treatment: Calomel internally, mercurial inunctions. Quinine, 5 grains every 4 hours. Morphine as necessary. Formalin about 5 minims at end. Died Saturday night, May 21.

1904. Case 7.—Attending physician, Dr. J. W. Howard (patient seen also and microscopic examination made by Doctors McGrath, Ashburn, and Stiles).

Report of a case of so-called "Spotted Fever" at Hamilton, 1904, by Dr. J. W. Howard.

G. M., male, aged 10 years, was brought to Hamilton from his home, 8 miles southwest of here, on the west side of the Bitter Root River. He came under my observation May 14, 1904.

The eruption at the time was very faintly marked, though he had been sick for 3 or 4 days; temperature, 103° F.; pulse, 110; very restless; appetite absent throughout entire sickness. Died May 22, 8 a. m.

The nose bled, not excessively, but frequently, for the last 5 days before death. The eruption was very backward and slow in coming out, and in fact never became pronounced. He had a convulsion 8 or 10 hours before death; do not know whether he had others later; during the convulsion he had slight opisthotonos. Disturbance of nervous system was marked throughout his illness by thrashing around in bed, rolling of the head, and throwing the arms; he could not be made to lie upon his left side for a minute at a time, but would immediately throw himself upon his right side or his back. Answered questions intelligently up to within a few hours of death.

Never complained of pain at any time, except occasionally when asked if his head ached, he would answer "Yes." Was seen by Doctors Ashburn and Stiles on May 20; they took blood samples for examination; examination negative; temperature ranged throughout from 97.5° to 105° F.; pulse from 85° to 125° per minute; bowels and urine both acted regularly and without assistance. The basis of treatment was quinine sulphate in large doses; other medication as indicated from time to time.

In addition to the above account I may give the following notes made by Doctor Ashburn:

Family history: Father killed. Mother alive and well. One sister died of whooping cough. One sister recovered from spotted fever under quinine 3 years ago.

Personal history (previous to present illness): Had chills and fever 6 years ago. Measles and whooping cough. No other sickness.

History previous to admission: May 11, complained at noon of being sick. Had nausea and headache. Slept that afternoon and was feverish and had headache and stomachache. On washing neck on first day complained of tenderness. May 13, spots showed on hips.

May 14, seen by Doctor Howard. Then stupid, restless. Temperature 103.5° F.; pulse, 115. Tongue had heavy moist coat. No pain except in bowels, from cathartic. No diarrhea, cough, or sore throat. After medicine, bowels loose. Has eaten practically nothing yet.

Physical signs: Temperature from 103° in evening to as low as 99° in morning. Vomiting more or less all through attack. Vomit greenish yellow.

Present condition, May 20: Expression dull. Very restless and tossing. Nauseated much of time. Nosebleed at times. Lips dry, blood stained, and crusted. Tongue dry, brown, and somewhat glazed. Vomit contains blood, thought to be from nose.

Eruption general, fine purplish. Less marked on face. Neck shows discoloration, yellowish. Heart sounds clear; respiration harsh, especially on right side. Spleen enlarged. Convulsions at 8 p. m., May 21. Died at 2 a. m., May 22. The daily dosage of quinine to May 20, was: 45, 45, 36, 48, 32, and 35 grains.

1904. Case 8.—Attending physician, Doctor Minshall; patient seen by Doctors Minshall, Gwinn, Ashburn, and Stiles; microscopic examination made by Ashburn and Stiles.

[Dr. Minshall's notes.]

Bitten by tick in 4 places Sunday night, May 15, 1904. Slight chill in afternoon of May 17. Was sick the following night and day. Was seen by Doctor Gwinn the evening of the 18th, who cauterized and dressed the tick bites. Temperature at that time said to have been 102. Severe headache, but no pain in back or limbs. I took charge of patient on May 19, at 5 o'clock p. m. Headache, but no pain elsewhere; coated tongue, intense thirst, but no nausea.

May 20: Condition much the same as yesterday, bowels moving freely.

May 21: No headache, no pain, no spots at 10 a. m., but skin presents a mottled appearance. At 2 p. m. spots appear quite distinctly on wrists and chest. By 5 p. m. all spots have disappeared. Skin of a decidedly mottled appearance.

Sunday, 22d: No change from yesterday. Appetite fairly good, mind clear, no pain in any part of the body.

Monday, 23d: No change since Saturday, except patient sleeps more, mind less active, some muttering during sleep.

Tuesday, 24th: Spots make appearance in decided manner over entire back, upper limbs, and chest. Nervous symptoms increase, more dullness, with slight delirium; in fact, sleeps most of time.

Wednesday, 25th: Eruption all over the body. Hyperesthesia of skin very marked. High nervous tension, muscles of back and limbs very rigid.

Thursday, 26th: Condition much the same as yesterday, with more or less coma.

Friday, 27th: Almost complete coma; all nervous symptoms increased.

Saturday, 28th: All conditions of two previous days more pronounced. A drink of water produces spasm of pharynx and diaphragm.

Sunday, 29th: Previous conditions more marked; slight edema of right lung noticed at 8 o'clock p. m.

Monday, 30th: Both lungs quite edematous. Remained in this condition during the day, dying at 9.15 o'clock p. m.

No photophobia at any time, pupils reacting to light up to and including 28th instant.

[Doctor Ashburn's notes.]

Tick bites, May 14; chill, 16th; fever, slight sore throat, and eruption on 23d.

May 26: Eruption profuse and measly everywhere but face. Temperature 103. Delirious. Tongue heavy, white coat, becoming dry and brown. Abdomen generally tender, especially right side. Hyperesthesia marked. Face flushed, conjunctivæ injected.

May 29: Comatose, spots purple, face still free. Respiration 50, pulse 138, temperature 103. Splenic dullness not obtainable.

May 30: Seen at 8.30 p. m. with Doctor Minshall and Doctor Stiles. Patient comatose. Lungs filled with fluid, so that loud rattling was heard throughout the room. Bubbling and crackling râles everywhere. Pulse weak, respiration 50. Patient evidently about to die, and decided to use bleeding followed by intravenous injection of 1:1000 formaline to normal salt solution. Patient died while this was being done. Informed by Doctor Minshall, nurse, and mother that reflex excitability had been so intense for 24 hours that slight touch used in putting spoon to lips, sponging, etc., would cause spasm and rigidity of whole body.

1904. Case 9.—Report on case "spotted fever" in 1904, by R. Gwinn, M. D.

G. C. T., of Florence, Mont., farmer, aged 32 years. Has been tick bitten every day or so for the whole spring; he has disinfected the bites with carbolic acid. On the 26th instant, was taken with chills, fever, and headache. Seen by Dr. J. F. Brice, of Stevensville, Mont., on 27th instant, who administered "calomel and fever medicine." Came to St. Patrick's Hospital at 4 p. m. to-day, when I find the patient complaining of headache and general soreness. Examination reveals many tick bites, together with an eruption all over like that of the "spotted fever." Pulse 92, temperature 103.2 under axilla. Pupils large. Respiration slightly accelerated. Tongue heavily coated. No apparent eruption in mouth or throat.

Diagnosis: The so-called "spotted fever."

May 29: Seems about the same. Pulse 100, temperature 103.2.

May 30: Patient much the same, except eruption darker and the skin and conjunctivæ more "jaundiced." Pulse 104, temperature 103.2. Doctors Mills and Stiles see the case with me, when a search of 1 hour by each of us for the parasite described by Doctors Wilson and Chowning is made with a negative result.

May 31, a. m.: Pulse 104, temperature in axilla 103.4; rested during latter part of night; injection of warm soapsuds causes good action of the bowels. Examination of eyes shows media a little blurred, so that the granular appearance of the retina can not be seen. The larger retinal blood vessels are quite plain, however; no swelling or blurring of disk; no hemmorhagic petechiæ in retina.

There is no stiffness of neck, and the head can readily be bent forward. No herpes. There is no pain complained of at all to-day; not even headache, and there was scarcely any yesterday.

May 31, p. m.: Pulse 122, axillary temperature 102.5, rectal temperature 104.8, respiration 28. Urine S. G. 1,022, acid, no sugar, trace of albumen, granular casts abundant, no blood. Patient's mind has been wandering for 2 days. Worse now.

June 1, a. m.: Pulse 120, rectal temperature 103.8 at 9 a. m. At 2 p. m., pulse 130, weak and irregular. At 8 p. m., pulse 120, rectal temperature 103.8; patient is delirious.

June 2, 9 a. m.: Pulse 130, weak and irregular; patient grew weaker and died at 8 p. m. from heart failure.

(The treatment in this case was symptomatic.)

The following additional notes were made by Doctor Ashburn:

Personal history (previous to present illness): Mumps, measles, scarlatina. No typhoid. Five years ago had spotted fever, which was aborted by Doctor Brice. Felt worse then at beginning than now. Did not have eruption.

History previous to admission: May 29: Been working in hills west of Florence all spring. Had very many tick bites which he cauterized with carbolic. May 25, had chill, followed by fever and mild pains. Took calomel. Headache more or less since, but not constant.

Condition on admission: May 28: Measly eruption, not thick, appeared about ankles. It now shows on ankles, face, and arms; possibly elsewhere, nowhere marked. Tongue white, moist, coated. Eyes suffused and light hurts them. Urinous (?) odor to breath. Bowels O. K. No cough, nose bleed, or especial discomfort. Urine passed about as usual. Spleen not demonstrably enlarged, not tender. Liver, heart, and lungs O. K. Abdomen not tender, shows eruption.

June 1: No photophobia, no pain. Apparent jaundice. Mind not so clear as at last note. Doctors Chowning, Stiles, and myself found nothing in blood.

1904. Case 10.—Attending physician, Doctor Mills (notes prepared by Doctor Ashburn; patient seen and microscopic examination of blood made by Doctors Mills, Ashburn, and Stiles).

J. B. Male, age 6 years. Had measles. Family history good.

Personal history (previous to present illness): Left Iowa 6 weeks ago. Been in Bozeman 5 weeks. June 2, arrived in Missoula and went on to Hamilton. Felt badly that night and ate no dinner. Ate none next day and vomited. Pain in abdomen, occiput, and back of neck, lasting to present, but now less marked. Fever. Seen by Doctor McGrath June 5, and eruption appeared June 6.

Symptoms: Bright and intelligent. Face flushed. Eyes injected, slightest convergent strabismus. Tongue white and moist. Cervical glands somewhat enlarged. Throat and mouth show no eruption or soreness. Some cough present. Spleen enlarged. Abdomen painful and tender. Measly eruption on hands, arms, feet, legs, and buttocks, palms, and soles. Pain and tenderness in abdomen, occiput, and neck. Bowels loose.

Blood examination: Fresh specimen negative. Large diplococcus in stain, looking like contamination.

June 8: Taking calomel and paregoric; room darkened. Has slept much of the time. Free from pain in the neck. Still has it in abdomen. Harsh breathing over right lower lobe.

June 9: Not seen by me. Doctor Mills reports spinal tenderness again. Pain still in abdomen. Spots not darkened. Condition much the same.

June 10: Appearance excellent. Very slight convergent squint still present. Spots fading on legs and body, not on soles. Pain still present in neck and abdomen. Tongue lightly coated. Knee jerks normal. Mother states that last night he cried that his eye was turning out. She looked, and divergent squint outward rotation of

left eye) was present. She calmed him to sleep and the outward rotation was not present this morning.

June 12: Still complaining somewhat of pain in neck and belly. Spots fading. Doing well.

June 15: Pain in knees; temperature normal. Patient sitting up in bed and eating well. Spots all disappeared, but a few discernible on palms.

June 17: Patient left hospital practically well.

1904. Case 11.—Attending physician, Doctor Mills (notes prepared by Doctor Ashburn from hospital chart and bedside; patient seen and microscopic examination made by Doctors Mills, Ashburn, and Stiles).

Monday, June 20, 1904: Mrs. E., aged 23 years, married, one 9-months-old child living. Now pregnant in the sixth month. Patient born in the Tyrol, Austria, and arrived in this country 4 weeks ago. Came to Missoula 19 days ago. Remained in town 5 days and removed to a ranch purchased on the Lo Lo.

Saturday evening, June 18: Patient felt entirely well. That day she had merely tasted and then refused to eat veal because of bad odor. She had gone barefooted during the day and felt chilly, but it was a very cold day. That night she had occipital headache and could not sleep.

Sunday, the 19th: She vomited. All day she ate nothing and felt feverish. At 9.30 p. m. she started for Missoula, reaching here at 1 a. m. Slept at a hotel and came to hospital this morning. On her way to town she vomited bitter fluid. At present she has frontal headache, some tenderness of the back of the neck. Many abrasions on the feet and legs, all said to be due to scratching mosquito bites. Tick bites denied. Eyes slightly injected, otherwise normal. No tenderness of eyeballs, scalp, or general surface. Tongue moist and shows a moderate white coating. No eruption. Spleen not demonstrably enlarged and not tender. Bowels regular. Temperature, 101° F.; pulse, 105. Patient has had a slight cough for 2 weeks. Examination of lungs negative. Heart sounds normal.

June 22: Condition not so good. Pulse more rapid. Scattered measly eruption on body and limbs. Examination of the blood shows no protozoa. Leukocyte count 15,600.

June 23: Spleen not demonstrably enlarged. Fetal movements felt. Examination of lungs shows decidedly harsh respiratory sounds over the left apex anteriorly and posteriorly and over entire right lung posteriorly. Tongue red and moist, with white streaks. Eruption abundant on body, limbs, and face; measly, not petechial. Urinous (?) odor of breath.

June 24: Had respiratory depression last night, respiration falling to 8. Fetal movements felt at noon today. Spinal puncture made at 4.30 p. m., negative. Patient's general condition not so good as yesterday. Face somewhat dusky, spots darker, but not hemorrhagic. Sordes on lips and tongue. Patient very nervous. Urine, reddish-yellow, turbid; heavy yellowish precipitate; acid, specific gravity 1.018; albumen present in small amount; no sugar; no bile (Gmelin); urea, 3.5 per cent; abundant vaginal epithelium and small round epithelium; numerous blood, epithelial, and granular casts; free red and white blood cells; much granular débris.

June 25: Condition worse. Delirium constant and marked. Only 6 ounces of urine past 24 hours. Drawn by catheter. No fetal movements felt. Pulse very rapid and weak. Cyanosis present.

June 26: Miscarriage at 2 a. m. Very rapid and not followed by much bleeding. Child lived an hour and showed no spots. Patient very restless and constantly delirious; at times violent. Alcohol seems to aggravate this. Face cyanosed, spots purple and somewhat increased in size. Urine, 8 ounces in past 24 hours.

June 27: Patient was bled last evening to the amount of 12 ounces, with marked

temporary improvement: good night's rest followed. This morning restless and very delirious. Resists all attempts at passive motion. Urine passed involuntarily. Pupils widely dilated notwithstanding the administration of large amounts of morphine. Yesterday had 9 grains between 2 and 10 p. m. Face and spots not so dusky as yesterday. Lungs examined anteriorly and found as at last note. Harsh sounds at right apex.

June 28: Bled 1 ounce last night. Very restless and delirious this morning. Morphine, grains 4.5 from 7 to 10 a. m., with no apparent effect. Lungs edematous. Patient died at 11.30 o'clock a. m.

Autopsy, 3 hours after death: Body of well-nourished woman, showing no marks of violence except some small scabbed abrasions on the leg, phlebotomy incision on arm, and needle puncture below left breast and on arms. Purple discoloration on dependent portions of body. All measly eruption has disappeared from the upper portion of body. Posterior of body blue, shows many spots, very dark blue or purple, from 1 to 3 or 4 mm. in diameter. Over the left buttock and coccyx are a few purple areas (1 by 4 to 6 cm.) that look like extravasations. Slight bloody discharge from the vagina. Calvarium being removed shows outer surface of dura injected, but otherwise normal. Removal of dura shows some adhesions at the vertex between the membranes and the brain substance. Area of adhesions small. Veins on surface of brain are distended with blood. No pus or lymph seen. Base of skull and dura normal. Ventricles opened and appear normal, except for some distension of the veins. Small amount of bloody serum, blood of which very probably came from cut veins, in one lateral ventricle. Cerebrum on section appears about normal. Basal ganglia on section appear normal. Section of medulla, pons, and cerebellum shows nothing abnormal. Incision from manubrium to pubis shows good amount of subcutaneous fat. Muscles well developed and of good color. Peritoneum normal. Removal of sternum shows remnants of thymus gland. Pleural cavities normal, except a few adhesions between the left lung and the pericardium; latter contains an apparant excess of clear straw-colored serum. Heart distended with blood. Left ventricle partially contracted. Removal of right lung shows it lead color on upper surface, very dark posteriorly. Several dark spots one-eighth inch in diameter, apparently old, anteriorly. Entire lung edematous. Posterior portion extremely congested; in a condition of hypostatic pneumonia, and sinks in water. Bronchial glands enlarged and black. Removal of left lung shows same appearance as right except the pneumonic area is less marked and less extensive. Heart on removal seems normal, muscle well nourished; left side contains chicken fat and red clots. Valves normal. Right side contains a few small clots, white and red. Valves apparently normal. First part of aorta normal. Arch of aorta seems unusually small, just permitting little finger to enter. Examination of abdomen shows peritoneum normal, considerable amount of straw-colored serum present. Organs in normal relation and position. Spleen enlarged and bound down by posterior adhesions and adhesions to stomach. Soft, easily torn; slaty-purple in color. Liver apparently somewhat enlarged; paler than usual, with yellowish tinge, which is probably fat. Section of liver is decidedly pale; what little blood flows is also very pale; tissue is firm, though not apparently fibroid. Gall bladder distended with fluid bile, duct patulous. No gallstones. Gross appearance of left suprarenal shows it apparently normal. Size of left kidney 13 by 7 by 6 cm. Section shows the cortex little if at all altered. Capsule is adherent, carrying substance in removal. Areas of distinct pallor scattered over the surface. Right suprarenal appears congested, otherwise normal. Right kidney 12 by 7 cm. Cortex pale and thickened. (Doctor Mills thinks it normal.) Capsule adherent in same way. Pancreas apparently normal. Stomach shows injection about cardiac end, otherwise appears normal. Intestines removed, opened, and washed. Upper portion appears normal. Solitary glands and Peyer's patches appear somewhat swollen. Cecum shows considerable injection, which continues more or less throughout the colon. The uterus is enlarged

to the size of 2 fists and measures 11 cm. broad by 13 cm. long; soft, normal in appearance for a recently delivered uterus. The left ovary normal, as is the right, which contains corpus luteum. On section womb appears normal. Aspiration of spinal canal through the lumbar region withdrew 15 c. c. clear spinal fluid. Dura apparently normal. Spinal cord removed. On removing dura from cord showed considerable injection of vessels, which was probably hypostatic. No lymph exudation or other evidence of inflammation. Cross section of the cord at 1-inch intervals shows nothing abnormal. No scales present and organs could not be weighed.

The following notes have been sent to me by Dr. R. D. Alton, of Livingston, Mont.:

1904: (?) Case 12.—R. S., age 71. United States, came to St. Lukes Hospital, having been sent to me with the diagnosis "spotted fever."

Upon admission, May 14, 1904, he stated that he noticed 4 ticks in the vicinity of the left elbow, about 10 days ago.

Upon closely questioning him, stated he saw the ticks 11 days ago, or on the 3d of May. He further said he saw the rash on the body a few days after observing the ticks; that the arm "swelled up as large as two arms and was as red as a beet."

From the arm the rash spread all over the body. He is positive regarding the presence of the ticks. He is equally positive that he was not bitten by anything else. Upon removing his clothing the odor of the body led me to ask him if he had ever had measles, to which he replied, yes.

Temperature on admission was 102° F. This gradually declined to normal.

At no time was he delirious. He complained of great soreness all over the body; his feet were exquisitely tender and continued so until he left the hospital, May 27, 1904.

The urine on admission was scanty and highly colored, no sugar or albumen found. His diet while in the hospital consisted of fruits, vegetables, buttermilk, lemonade, etc.

He was given a combination of equal parts of Pulv. Glycyrrhizæ Comp. and Potassium Bitart., in water, to regulate the bowels, with Basham's Mixture after meals. In addition, he was given sour Rhine wine during the day.

He made an uneventful recovery, the rash disappearing from the face, forehead, trunk, arms, body, and lower extremities in the order named.

On the 25th of May his daughter called on him; from her I learned that measles were present in the vicinity from which he came—Lewiston, Meagher County, Mont.

The history, together with your negative findings, lead me to believe it was practically a case of measles, occurring in an old, poorly nourished, scorbutic patient.

1904: (?) Case 13.—On Sunday, June 12, 1904, I saw Charles E., age 26, United States, at Gardiner, Park County, Mont.

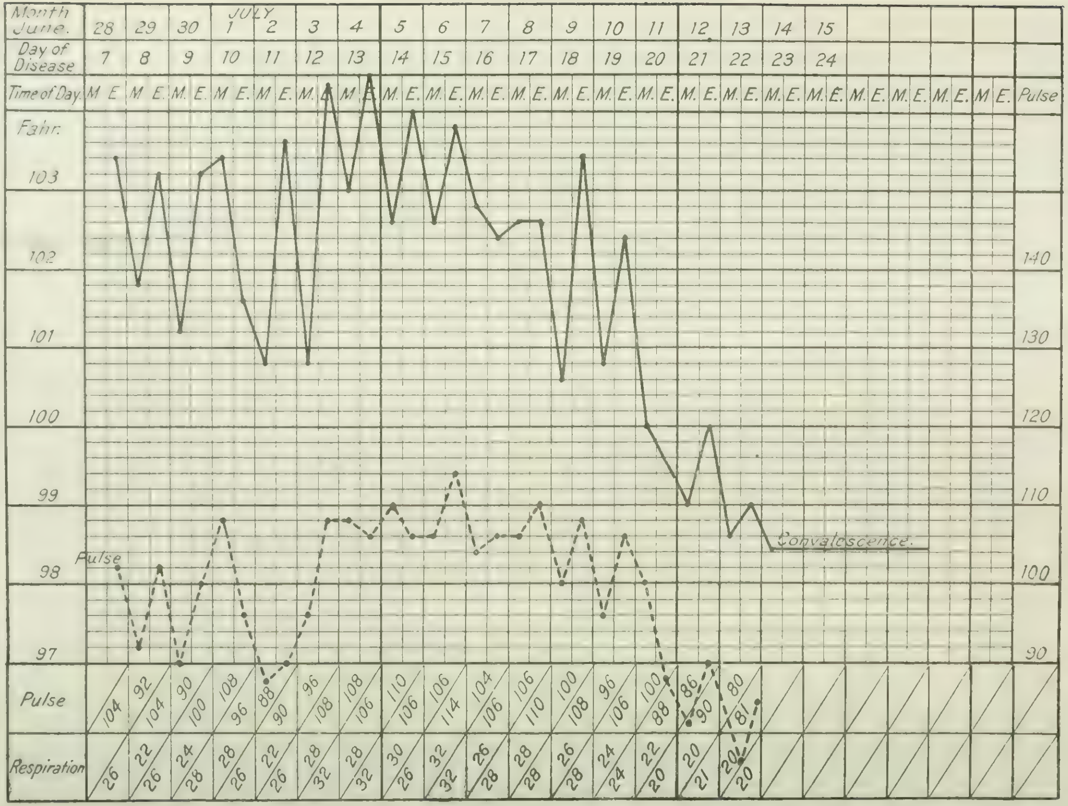
Upon examination found a cicatrix on the inner aspect of the left leg, middle third. Was told he had been bitten by a tick, 12 days previously.

At the time of my visit he was delirious, picking at the bed clothing, muttering, and rolling restlessly from side to side. Pupils dilated and irresponsive to light, patellar tendon relex exaggerated, ankle clonus pronounced. Tongue furred, bowels constipated, urine scanty and high colored. No albumen or sugar, Sp. gr. 1030. Temperature in axilla at 5.41 p. m., 104° F.

Ordered Sod. Bromide, grains xv, every 3 hours to produce rest, ice bags to head, and a hot, mustard, footbath.

While preparing to catheterize, he passed a fair quantity of urine, involuntarily. Further ordered he be given a glass of water every hour, with a glass of equal parts milk and water, every 4 hours. I inclose specimens of blood taken June 13, 1904.

On the 13th was informed by telephone that he had rested well, nervous symptoms



Month June.
28 29 30 JULY 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
Day of Disease
7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
Time of Day.
Pulse
Fahr.
103
102
101
100
99
98
97
140
130
120
110
100
90
Pulse
Convalescence.
Pulse
Respiration

abating, kidneys acting freely, bowels moved from an enema given on the evening of the 12th June.

On the 14th improvement continued, temperature at 5.30 p. m., 102° F.

This may be a case of meningeal irritation following measles, or may be due to the tick bite and infection from that source.

I might mention that there was no cough or conjunctival congestion.

A Clinical Report of Four Cases of Piroplasmosis Hominis, with Table of Seventeen Cases seen by the Author, by L. A. Gates, M. D., Bridger, Mont.

Case 11.—C. B.; age, 26; male; occupation, ranchman; residence, Sage Creek, 20 miles southeast of Bridger, Mont. I saw this patient first July 1, 1903, being the tenth day of the disease. Patient gives a history of having been bitten by a tick June 21 on the left leg, 7 inches below the knee. The small wound caused by the bite became very sore, and there gradually developed around it an irregular, oblong dark bluish-red spot. The patient also states that he was bitten by a tick on the 22d and 23d, but only the first bite became sore.

On the 24th he had a very severe chill and says he has had considerable fever since that time; says he has been unable to sleep at all or eat anything for the past three days. Following the chill, a very severe headache came on, followed by severe aching of all of the skeletal muscles. He said "They ache like the toothache," being especially severe in the calves of the legs. On the 27th he vomited; on the 28th he noticed a few red spots on the palms of his hands, which he said seemed to be just under the skin and looked like the eruption of the measles. There were also, at this time, a few spots beginning to appear on the feet.

On examination this tenth day of the disease, we find the patient's countenance bears a look of nervous anxiety. The eyes are bright, with considerable congestion of the conjunctiva, the tongue quivers very much when protruded and is loaded with a heavy, dirty, brown coat; sides of the tongue are so livid as to be almost blue. A petechial eruption is seen in the skin and involves every portion from the soles of the feet to and including the scalp. They vary in color from bright red to bluish red. The greater number are seen on the extremities, where in places, two or more, by coalescence, form irregularly shaped spots. They vary in size from a pin head to the size of a dime, the larger ones being very irregular in outline. The skin is very dry.

There is some cough and on auscultation numerous dry rales are heard over the posterior portion of both lungs. The abdomen is moderately tympanitic. The urine, which is almost brown in color and lessened in amount, contains some albumen. The pulse is 104, weak and irregular, temperature, 103° F. The accompanying chart shows the temperature, pulse, and respiration from the seventh to the twenty-third day of the disease, at which time the patient was convalescent. On the eleventh day the patient was unable to urinate and the catheter was passed—urine very dark. The general course of the disease was severe in this case from the onset, though no complications occurred. Especially well marked was the cough and insomnia. The temperature touched normal on the twenty-third day, from which time the patient made rapid progress to complete recovery.

Case 14.—C. F. P.; male; age, 41 years; occupation, ranchman; residence, 4 miles southwest of Bridger, Mont. This patient was bitten on the leg by a tick April 14. On the 18th he felt a general soreness all over the body, which has gradually increased up to the first time I saw him, April 23. On the 21st he had a chill, followed by loss of appetite, headache, backache, and fever.

Examination: The face was slightly flushed, eyes dull, conjunctiva slightly injected, tongue has a grayish coat, bowels are constipated. There is an annoying, dry cough. No abnormal sounds on auscultation of the lungs; heart sounds normal; pulse rate, 70, strong and regular. The urine is high colored, but contains no albumen. Small

petechiæ are present over the entire body except the face. There is great muscular soreness, including the muscles of the eye. Has slept but little the last 2 or 3 nights. Temperature, 102⅘° F.

April 24: Patient feels better, eruption is not so well marked. He had a chill this a. m.; temperature, 102° F.; pulse, 64.

April 25: Patient complains bitterly of backache and pain in ankles and knee joints; temperature, 103° F.; pulse, 64.

April 26: Patient has had two chills this a. m.; anorexia complete; says he feels very tired; temperature, 102⅘° F.; pulse, 72.

April 27: Bowels moved twice during last 24 hours without laxative or enema. The cough is not so troublesome; the eyes are very sensitive to light; the spots are very conspicuous; temperature, 101⅘° F.; pulse, 62, full and regular.

April 28: Complains of frontal headache and severe, heavy, aching pains in the tibiæ; the spots are very red; the insomnia persists; temperature, 103° F.; pulse, 70.

April 29: Patient has had a slight chill this a. m.; the pain has left the legs; the eruption shows very bright and red; bowels loose; urine much reduced in quantity, no albumen; temperature, 102.7° F., pulse, 66.

May 1: Patient says he feels very much better; free from all muscular soreness and pain; slept fairly well last night; has some desire for food; the spots appear very dark red; temperature, 101.5° F.; pulse, 70. From this date the patient gradually recovered strength and was able to resume his work about the ranch June 1. This case was below average in severity for cases occurring in this valley. Of special interest in connection with this case are the slow pulse and repeated chills.

The following facts, I believe, are unique as regards the theory of the tick-bite origin of the disease, being, so far as I am aware, the only recorded instance in which a tick, after having bitten a patient suffering from "spotted fever" has then become detached from the patient, bitten a second person, and this second individual thereby contracting the disease. This is what actually occurred. The second party developed the disease in its most severe type, ending in death. (See p. 113.)

On May 29, Mr. C. A. H., of Bridger, Mont., visited the Clarke Fork Canyon, some 60 miles south of this place. He remained in the vicinity of the canyon about 4 days and while there was bitten in 5 or 6 places by ticks. About 7 days from the time he was first bitten by the ticks, viz, June 5, he arrived at his home in Bridger, at which time he felt the first symptoms of the onset of the disease. When he arrived home there were two ticks attached to his body which of their own accord, or from friction of the clothes, became detached from the body on June 6 and then bit his wife, who, on June 12—that is, after a period of incubation of 6 days—developed the first symptoms which marked the first symptoms of the disease. The petechiæ occurred on the 3d day. The following is a rather meager clinical history of the disease as it occurred in husband and wife.

Case 15.—C. A. H., male, age 46; occupation, stockman; residence, Bridger, Mont.

June 10: The patient is a large man, weighing about 198 pounds; has indulged very freely in alcoholics the past 3 years; has never been sick excepting 2 years ago, when he had smallpox. On the legs and abdomen are the marks of several tick bites, one of them on the ankle being very tender and around it a dark, bluish spot. There are now a number of small petechial spots on feet and hands, this being the fourth or fifth day since the onset. The conjunctivæ are congested, temperature 103⅘° F., pulse 97. He complains of general muscular soreness, severe backache, headache, and of being very tired and exhausted. No appetite whatever. Bowels are constipated, urine dark and shows a trace of albumen. During the course of the disease, which lasted 20 days in this case, the dry cough was most troublesome. Nervous symptoms were not marked. The urine was much lessened at times; besides a slight amount of albumen, it contained numerous blood and epithelial

casts. The spots appeared over the entire surface, being large and irregular on the legs, very bright red at the height of the disease, fading to a brownish pink color as the fever subsided. Many spots were yet visible 6 weeks after the temperature reached normal. Highest temperature observed was 104.1° F. The insomnia in this case was not troublesome. A severe intercostal neuralgia occurred during convalescence.

Case 16.—Mrs. G. H. (wife of C. A. H., case 15), age, 43; housewife; residence, Bridger, Mont. This patient is a very fleshy woman, with pendulous abdomen. She received bites from two ticks, which had previously bitten case 15, during the onset of the disease in that case. This occurred June 6. One tick bit this patient on the abdomen in the hypogastric region; the other on the leg. The bites occurred during the night; the ticks were removed and killed by the patient the following morning. Following the removal of the ticks she applied carbolic acid to the bites. On the evening of the 11th she felt chilly, feverish, and general malaise. The morning of the 12th she arose as usual, but had a pronounced rigor and such severe headache and aching in limbs and back as to compel her to return to bed. The temperature that evening rose to 104° F. During the first 12 days of the disease it ranged between 102° and 104.4° F., for 5 days keeping close to the 104° F. mark. After the twelfth day of the disease the temperature gradually fell and the eighteenth day it registered 97° F., when an unfavorable prognosis was given, though in every other way the patient seemed on the road to recovery. The following four days the temperature remained about normal.

The respirations, early in the course of the disease, reached 40 per minute, at which rate they remained until the last week of the disease. Cheyne-Stokes respiration was observed during the last of the second week, some cough, but not so much as is usually observed. The heart action was weak from the first, and during the second and third weeks became very weak and irregular, with very low arterial pressure. The rate of pulsation varied from 120 to 140 per minute. The tongue, mouth, and pharynx became very dry early in the disease. No vomiting occurred, and food was well taken at all times. The urine became very scant at the end of the first week and contained many fatty, blood, and epithelial casts and, at times, some albumen. By the end of the second week the urine had increased to normal amount and was free from albumen and casts. The intensity with which the disease attacked the nervous system was marked from the onset. A low, muttering delirium came on during first week. During the second week patient was in heavy stupor from which she could be aroused with difficulty, but when aroused would answer questions correctly and then perhaps talk at random. The condition of the mind approached the normal during the third week. The eyes were very sensitive to light and very much congested. The petechiæ commenced to appear on the third day, first on feet, ankles, hands, and wrists. It rapidly spread all over the body and was very thickly distributed on the back. During the third week the patient each day expressed herself as feeling better than on the day previous. The temperature, after having reached 97° F., became normal. The respiration was easier and all symptoms seemed to indicate a beginning convalescence. On the twenty-second day of the disease the patient said to the nurse, "I feel a pain around my heart." The nurse turned her on her side, in which position she seemed to rest a few minutes and then breathed her last.

The points of interest in this case were mode of infection (see also, p. 23); high temperature at onset, which persisted; rapid breathing; great congestion of the kidneys; extreme weakness of heart action; the decided effect on the nervous system; subnormal temperature, and sudden death without warning, when apparently recovering from the disease.

No. case.	Year.	Date onset.	Patient's initials.	Residence.	Sex.	Age.	Occupation.	Eruption appeared.
						Yrs.		
1	1894	June ...	C. G	Myersville, Wyo.....	Male ...	5	.....	Second or third day.
2	1898	May 24..	L. M.....	Thermopolis, Wyo....	....do...	23	Ranch hand.	Fourth day.
3	1898	June ...	D. W.....	.....do.....	....do...	17	Freighter....	No record ..
4	1900	April ...	.....	7 miles SE. Bridger, Mont.	....do...	29	Sheep herder	.....do.....
5	1900	April 17.	E. G	1 mile SE. Bridger, Mont.	Female.	6	.....	Fourth day.
6	1900	April 15.	J. A.....	.....do.....	Male ...	39	Ranch hand.	Third or fourth day.
7	1901	May	.....	Shoshone River, Wyo.	....do...	52	Ranchman ..	No record ..
8	1902	May 15..	W. S.....	South of Bridger, Mont.	....do...	31	Trapper	Fifth day...
9	1902	May	A. W	Near Bridger, Mont ..	Female.	8	.....	No record ..
10	1903	May 9...	M. H	7 miles SW. Bridger, Mont.	....do...	67	Nurse	Sixth day...
11	1903	June 21.	C. B	Sage Creek, 25 miles SE. Bridger, Mont.	Male ...	26	Ranchman ..	Fifth day...
12	1903	June 9..	H. L.....	5 miles SE. Bridger, Mont.	Female.	47	Housewife...	Fourth day.
13	1903	June 5..	W. C.....	10 miles SW. Bridger, Mont.	Male ...	6	.....	No record ..
14	1904	April 18.	C. F. P....	4 miles SW. Bridger, Mont.	....do...	41	Ranchman ..	Fourth day.
15	1904	June 5..	C. A. H...	Bridger, Mont.....	....do...	46	Stockman ...	Fifth day...
16	1904	June 12.	Mrs. G. H.	.....do.....	Female.	43	Housewife...	Third day ..
17	1904	May 9...	J. N	2 miles N. Bridger, Mont.	Male ...	45	Ranchman ..	Fourth day.

Date of tick bite and location.	Death or convalescence.	Remarks.
No history of bite, but was much exposed.	Convalescence after 10 days..	This was a very mild case, as is always the case in children, so far as my observations go.
.....do	Convalescence after 21 days..	A very severe, though uncomplicated case. The petechiæ coalescing to form great irregular spots, which could still be seen 4 months after recovery.
.....do	Convalescence after 3¼ weeks.	This man did not call a physician, and took no medicine. The spots were very large and bright.
Was bitten, date not known..	Was in bed but a few days ...	This man came to the office with eruption well developed. Was not seen again by me.
No history of bite, but was out among the sagebrush every day, and so very much exposed to ticks.	Convalescence after — days..	Mild case, few spots, not petechial in character.
.....	Died the twenty-ninth day of the disease.	This is the most severe case I have seen. Spots were large and on the legs became black and gangrenous before death. The skin on the scrotum was also gangrenous. Very deep stupor for 10 days before death.
Bitten on leg	Slow recovery after 4 weeks..	Spots could be seen 4 months after recovery. Delirious 2 weeks.
Bitten several places	Recovery after 18 days	Severe case. Endocarditis as sequela.
No record; exposed	Did not remain ill in bed for 2 weeks.	Very mild. Slight fever, general malaise, eruption rose color, not petechial.
Bitten on leg and thigh May 4 and 8.	Died on eighth day of the disease.	Severe parenchymatous inflammation of kidneys.
Bitten on leg June 15, 16, and 17.	Recovery after 22 days	Type of disease severe. No after effects.
Bitten several places during first week of June.	Slow recovery after 20 days ..	Kidneys suffered especially.
Bitten on back of head; no date.	Convalescent twelfth day	This little patient was seen by me but once, and was in bed about 8 or 10 days.
Bitten on leg April 11	Recovery after 12 days	Disease was of medium severity. Pulse never above 70. A greater part of the time it was 64.
Several bites from May 30 to June 5 on legs and abdomen.	Convalescent twenty-second day.	Case from which ticks became detached, and then bit wife of patient, thereby infecting her.
Bitten on leg and abdomen by ticks from case 15.	Died on twenty-second day of disease.	This case was bitten by ticks from case 15, which had drawn blood from case 15 during onset of disease.
No record	Convalescent fifteenth day...	This patient is much given to drink, and would not be likely to know whether or not a tick had bitten him. This case was certainly as spotted as could be.

A Possible Case of "Spotted Fever" in Utah, by R. J. Smith, M. D.

[Personal letter, dated May 22, 1904, to Dr. Thomas D. Tuttle, secretary Montana State board of health.]

* * * * * * *

I have a case that looks to me very much like wood-tick disease, "spotted fever."

The patient, female, age 24, was visiting in Idaho, where within 2 miles of the place she was visiting, there were 2 cases of "spotted fever," but I have been unable to obtain information as to onset, symptoms, etc. One young man recovered, left in very weak condition.

Ten days ago this patient of mine was bitten in four places by 2 ticks, the ticks penetrating the right leg below the knee.

The parts became very painful and swollen and in three or four days the patient suddenly became very ill, severe nausea, intense headache, backache, sore muscles, pains in limbs; could not sit up without dizziness and nausea; did not vomit.

Her mother brought her home on Friday; I was called Saturday, 21st, and found temperature 103.5°, pulse 92, eyes very bright, some roseolous spots on wrists, arms, back, abdomen, ankles; patient very nervous, stiff neck, no spinal tenderness, no hyperesthesia; numb all over, some twitching of muscles of forearm and legs. Temperature at 1.30 p. m., 103.5°; 4 p. m., 103°; 10 p. m., 103°; this (Sunday) a. m., 104°.

Temperature same all evening and night. Bathing had no effect. When face was first touched with cold water during the bathing chill came on; quite severe. No elevation of temperature afterwards.

This morning spots all over body, on palms and soles; not papular except here and there; look like the roseola of typhoid fever; no pain, no nervousness, slight hacking cough, nausea completely relieved, no tenderness over spine, an occasional pain in ankles, muscles without stiffness, neck freely movable, eyes very bright, urine free; temperature 104, pulse 98; bowels moved freely; slept well, no opiates used. Gave cicutine hydrobromate, $\frac{1}{30}$ grain, every hour until pain and stiffness relieved last evening—4 doses removed pain, etc.

My diagnosis is held in abeyance.

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