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THE DISEASES OF NEWBORN LAMBS WITH SPECIAL EMPHASIS ON BACTERIOPATHOLOGY

Ching-tuan Cheng, Thesis (Ph.D.)

Fifty-five newborn lambs affected with a variety of diseases were included in this study and the problem was attacked hematologically, bacteriologically and pathologically.

The hematological investigation consisted of studying the blood from both normal sheep and pathological lambs. The blood values obtained from normal sheep served as a control. The results were briefly summarized in three tables and discussed in detail in connection with the individual cases. The average blood values of normal lambs were - red blood cells 10,444,286 per cmm, hemoglobin 10.9 gm. per 100 cc, white blood cells 7,979 per cmm with differential count of 28% neutrophils, 70% lymphocytes, 1% monocytes and 1% eosinophils, while those of normal ewes were - red blood cells 8,607,000 per cmm, hemoglobin 10.08 gm per 100 cc, white blood cells 8,263 with differential count of 30% neutrophils, 65.25% lymphocytes, 1% monocytes and 4% eosinophils.

In the bacteriological studies, 41 cultures of aerobes and 7 anerobes were isolated from various cases. By means of morphological, biochemical, and pathogenicity tests, these cultures were identified and grouped under 9 genera. They were - Staphylococcus, Diplococcus, Neisseria, Escherichia, Bacterium, Pasteurella, Corynebacterium, Actinomyces and Clostridium. Two cultures were undetermined. Twenty (48.78%) of the 41 original aerobic cultures were recovered from inoculated mice, 13 (31.7%) of which were definitely virulent. Rabbits were found to be quite resistant to the cultures and were not suitable for pathogenicity tests.

As a result of pathological and bacteriological studies, 15 different diseases were diagnosed, namely, white muscle disease, staphylococcic

infection, navel infection, docking infection, castration wound, bronchopneumonia, pasteurellosis, enterotoxemia, meningitis, peritonitis, enteritis, gastric ulceration and perforation, dietary deficiency, congenital anomalies, and an undetermined group. Most of these diseases were studied more or less in detail and a number of conditions such as congenital interstitial bronchopneumonia, patent ductus arteriosus, patent foramen ovale, gastric ulcers and perforation, and encephalomalacia were reported for the first time.

The histopathology of white muscle disease in lambs was studied in detail. The finding of muscle giant cells (skeletal and cardiac) in the cases of white muscle disease and their significance in myo-regeneration are considered new to medical science and of vital importance in explaining the pathological phenomena involved. The specific function of the muscle giant cells and the modes of their formation were fully discussed. Their function is to remove the dead muscle fibers, and they were formed either by fusion or by nuclear proliferation without cytoplasmic division.

The cardiac muscle was observed to possess the capacity for regeneration, and this is also considered new to medical science.

The nature and origin of the so-called "anitschkow myocyte" and "Aschoff cell" were fully discussed and evidence was given bearing on the position of these cells in the myocardium. In short, "Anitschkow myocyte" was found to simulate very much the regenerating cardiac muscle cells and the "Aschoff cell" the cardiac muscle giant cells.

The fancy and peculiar terms such as "repair clubs of muscle", "multinucleated syncytial masses", "large multinucleated plasmatic masses", "syncytial sprouts with multiple nuclei", "cytoplasmic syncytium with a large number of nuclei", and "multinucleated cells in muscle" were explained and designated as synonyms of either skeletal or cardiac muscle giant cells.

The nature and the origin of the so-called "muskelzellenschlauche" of Waldeyer was discussed in detail. This term designates a group of

regenerating skeletal muscle cells together with one or more muscle giant cells.

The nature and origin of the so-called "Aschoff body" was discussed. This body is probably a mass of regenerating cardiac muscle cells together with one or more cardiac muscle giant cells.

A brief and concise statement was given concerning the pathological processes involved in muscular disturbances. The processes involved are designated as "myo-inflammation and scar-tissue repair" for tissue reaction to s severe injury and "myo-degeneration and myo-regeneration" for tissue reaction toward a mild injury.

A new technic of opening the cranial cavity for brain examination was described. This was done by making a frontal section through an imaginary line, passing through nasal cavities, floor of orbits and about half an inch above the ears until it reached the occipital region of the skull. Then, by gentle tipping and with the help of the blunt tip of a pair of scissors, a greater part of the brain (the entire cerebrum) was slipped out of the upper half of the skull and exposed, leaving the cerebellum and medulla oblongata in the lower half.

The nature of the various diseases of newborn lambs was stressed, and suggestions were made for the control of some of the more important conditions.

THE DISEASES OF NEW-BORN LAMBS WITH SPECIAL EMPHASIS ON BACTERIO-PATHOLOGY

by

Ching-tuan Cheng

A THESIS

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The diseases of new-born lambs with special emphasis on bacterio-pathology

Ching-tuan Cheng

I. Introduction

Formerly, in this country as elsewhere, most lambs were born in May on grass (so-called "grass-lambing") and practically few, if any, diseases of the new-born were encountered. Under the present condition of marketing lambs early in the fall, "shed-lambing" during the early spring has been practiced. Often as a result of inclement weather, unsuitable conditions and unsanitary surroundings in the sheds together with overcrowding, the diseases of new-born lambs have increased and extensive losses each year are reported by farmers, extension men, and veterinarians. Under this type of management, a number of interesting diseases in new-born lambs are encountered and unfortunately not much detailed work has been done in this particular field. In order to get a better understanding of these diseases so as to form a sound basis for diagnosis, treatment, and control, the present investigation has been undertaken since early spring of 1944 and the results are herewith presented.

II. Historical review

For a historical review of the diseases of new-born lambs, it seemed advisable to divide the literature into separate categories so that a clearer and better idea concerning individual diseases might be obtained. The following categories such as: diseases of new-born lambs in general, arthritis, enteritis, enterotoxemia, pneumonia, white-muscle disease, and miscellaneous diseases were followed. Under each category, a chronological order of the literature as it appeared during the past fifty years will be observed.

1. Diseases of new-born lambs in general

In 1902, Craig and Bitting[21] were probably the first in this country to pay some attention to the diseases of sheep. They discussed such diseases as white scours of lambs, peritonitis, encephalitis, cerebrospinal meningitis, and arthritis.

Gallagher's[30] first work on "Diseases of Sheep" appeared in 1921 and it was revised later by Schoening in 1933. In this bulletin, a number of diseases related to young lambs were discussed among which were: necrobacillosis of navels, white scours, joint ill, peritonitis, and meningitis. They believed that Actinomyces necrophorus was the etiological agent of necrobacillosis, bacteria or parasites the causes of meningitis, and Bacterium coli communis the cause of white scours. They also mentioned that meningitis was more often seen in lambs than in older sheep.

Jungherr and Welch[46], in 1927, reported on lamb diseases from Montana. Of the lamb diseases brought to their attention that year, the most important by far was dysentery. The other three diseases--umbilical infection, coccidiosis, and the bradsot-like disease--were also included but were considered to be of lesser importance. They felt that the coccidia must be regarded as possibly pathogenic factors in "stiff lambs" and the white muscle lesions of a degenerative nature were probably secondary in nature. Vibrion septique was thought to be the etiology of the bradsot-like disease.

In 1929, Baker's book on "Sheep Diseases" (2nd ed.)[4] was published. In the section dealing with the diseases of young lambs, he mentioned the following conditions: congenital defects (imperforate anus, hernia, skin drying, umbilical hemorrhage, suffocation), pinning (retention of the meconium), navel-ill, white scours, goiter, constipation, etc. A brief account of each diseased condition as to its cause, symptoms, and treatment was given.

Marsh (1929)[59] mentioned the condition of "stiff lambs" as one of the several obscure diseases. According to him, at least three separate diseases were ordinarily regarded under the general name "stiff lambs", and they were umbilical infection, stiff lambs, and white-muscle disease.

Two years later (1931), Miessner & Koser[65] reported on lamb diseases in Germany: "About half of all examined lambs showed a specific infection and, of these, infectious

pneumonia (B. bipolare ovisepticaum) was the most prevalent (43%). Other organisms isolated from cases of pneumonia were B. pyogenes, Streptococci, and the bacillus of Dammann-Freese. B. Coli was rather common (23%) while anaerobes were found to a lesser extent. Some of the non-bacterial diseases were ascribed to dietetic errors." In the same year (1931), Mills[66] gave a brief account of the following lamb diseases, describing the nature and advice as to the treatment: white scours, B. necrophorus infection, and spotted liver disease.

In 1932, Marsh's article[60] on "Prevention of Diseases in Young Lambs" was published. The author dealt with this subject under three categories, namely, diseases due to management of the ewe during pregnancy, infectious diseases (necro-bacillosis, arthritis, dysentery, pneumonia), and miscellaneous diseases. Under the last category, he specifically discussed stiff-lambs or white-muscle disease. The white appearance of the affected groups of muscle fibers was due to a deposit of tricalcium phosphate, and the diseased condition was brought about by a disturbance of normal calcium and phosphorus balance.

Turner[69] in 1932, reported a rather lengthy article on "Field and Experimental Studies of Sheep Diseases". He considered joint-ill (pyemic arthritis) of lambs as a result of navel infection, goiter as iodine deficiency, and scours as bacterial infection.

Howarth (1934)[42] published a brief account on the following diseases of lambs: navel ill, dysentery, pneumonia,

hemorrhagic septicemia (Pasteurella ovisepticus), polyarthritis, and white-muscle disease. Only gross lesions were described for the white muscle disease.

In 1936, Oppermann[80] described two main diseased conditions found in the lambing pen, namely, the paralytic disease and hemorrhagic septicemia of lambs. The former was considered to be caused by navel infection and nutritional disturbance, while the latter was caused by Pasteurella bacteria with dusty hay and litter as the predisposing factors.

The Montana Station workers (1937)[70] published a summary of practical information dealing with shed sanitation, care of pregnant ewes, necrobacillosis of the liver, joint disease, paralysis, dirty eating, wool-eating, lamb dysentery, pneumonia, sore eyes, stiff lambs, and blackleg.

Two years later (1939), the Oregon Station[78] published a preliminary report dealing with the cause of four range diseases of sheep, such as lunger disease, stiff-lambs, lamb dysentery, and pregnancy disease, but no definite conclusions were made.

In 1942, Dykstra et al[23] stated that "control of sheep diseases is essentially a problem of prevention rather than of treatment". In dealing with diseases of young lambs, a special discussion was given to each of the following, namely, sanitation at lambing, scours, blackleg, cleanliness in docking and castrating, malignant edema, polyarthritis, stiff lamb disease, navel ill, etc. However, the authors had the idea that "polyarthritis and stiff lamb disease were

sometime classed as the same disease, because they cause inflammation of the joints"

In the same year (1942), an article entitled "Preventing Lamb Losses" was prepared by Thorp[98]. In this article, the importance and means of preventing losses of lambs from diseases were considered. The author expressed his idea by saying: "It has been estimated that marketing only 4/5 of a lamb crop means no profit for the farmer. At least 3/4 of this loss occurs during the first portion of the lamb's life and is largely preventable." Nine different diseased conditions such as navel infection, scours or dysentery, pneumonia, "stiff lambs", etc. , were mentioned and methods of control were discussed.

Last year (1944), the National Veterinary Association of Great Britain and Ireland[35] made a report in which some diseases of new-born lambs were included. Among those mentioned were: lamb dysentery (Cl. welchii, Type B), pulpy kidney disease (Cl. welchii, Type D), pyemia (Staphylococcus aureus), joint-ill (Streptococcus of Lancefield's group C.), Erysipelothrix infection, and Fusiformis necrophorus infection. A brief account of the nature of the disease, its diagnosis, symptoms, pathology, treatment, and control was given for most of these conditions.

2. Arthritis (polyarthritis, joint-ill, lameness, stiff lambs, navel-ill, erysipelothrix infection)

A number of different terms have been used to designate inflammation of joints resulting from infection such as: arthritis, polyarthritis, joint-ill, navel-ill, lameness,

"stiff-lambs", and erysipelothrix infection. Some of these terms are very misleading and cause a lot of confusion. For the sake of uniformity, the term "arthritis" is re-emphasized and used here.

From the literature, it is apparent that two forms of arthritis have been encountered in lambs, namely, the acute and chronic forms. The acute form is non-specific and is associated with pyogenic organisms while the chronic one is specific and associated with Erysipelothrix rhusiopathiae.

In 1925, Cornell and Glover[20] reported cases of arthritis due either to streptococci or swine erysipelas. It was stated that more outbreaks were found to be caused by streptococci. On the other hand, swine erysipelas was responsible for only one outbreak during the period of their investigation.

Seddon (1929)[88] made a general review on the subject of "lameness in lambs", and he pointed out that several quite distinct affections of lambs, different in cause, in symptoms, and in age of animals affected, were included under the term "lameness".

Two years later (1931), the Montana Station[68] investigated the same problem in lambs from two weeks to several months of age. The cause of this disease was shown to be an infection of a specific bacillus which gained entrance to the body through the navel cord at birth, or through the docking wound. Loss from this cause could be prevented by disinfecting the navel at birth with tincture of iodine and by proper docking technic using a hot iron. This same

information was later published from the same Station in the same year[69], while the etiological agent, (swine erysipelas) was identified and reported six years later (1937)[71].

The Department of Agriculture of New South Wales (1931)[72] found two forms of arthritis in lambs and young sheep, namely, a non-suppurative specific infection (E. rhusiopathiae) and a non-specific type associated with pyogenic organisms (B. pyogenes).

In 1933, Howarth[43] described cases of polyarthritis which appeared in lambs after docking and castrating. A strain of E. rhusiopathiae was found to be the causal agent.

In the same year (1933), Seddon[89] worked with a specific arthritis in sheep which was defined as a synovitis and osteo-arthritis due to a specific bacillus (resembling B. pyogenes), and manifested by lameness. It affected sheep from four weeks to seventeen months of age. Attempts to treat the condition failed.

Blakemore[10] described a disease (joint-ill) of lambs in 1939. This disease was characterized by lameness, rapid emaciation, suppuration of joints, synovial bursal and tendon sheath, but other pyemic changes were very rarely observed. A non-hemolytic streptococcus, antigenically related to Lancefield's Group C was isolated from the affected lambs and the disease was experimentally reproduced. The affected lambs were treated with several daily doses of 1.5 grams of sulfanilamide and the results were very satisfactory.

3. Enteritis

Only two references have been noticed in connection with this disease, and both happened to be published in the same year (1933). However, the entity of these cases is doubtful.

Caré[17] reported that lambs 14-30 days old suffered from a disease in which the affected lambs showed stiffness with muscle inflammation and degeneration. These lambs, when fed on milk, recovered completely in 2-3 days. He attributed the cause of this condition to the ingestion of coarse food unsuitable for a young lamb and enteritis resulted. Judging from this report, Caré might have been dealing with a case of white-muscle disease.

Heller[39] found cases of infectious enteritis of young lambs in California. He mentioned that "it appears to be of the enterotoxemia type of infection, as it can regularly be transmitted by oral administration of filtered intestinal contents". However, the author considered that "it is highly improbable that an anaerobe causes the disease", because he was unable to isolate from such cases any bacteria other than colon bacilli which would produce a poison capable of producing infectious enteritis.

4. Enterotoxemia (Pulpy kidney disease, bradsot-like disease, etc.)

This disease has been investigated by quite a number of research workers and references were recently collected by the writer. In this article, however, only some of the more important works are presented.

In 1932, Bennetts[6] gave a rather complete account of investigations carried out upon the etiology of infectious enterotoxemia in Australia, and it was found that the causal organism was a new species of anaerobe closely related to Cl. welchii, the lamb dysentery bacillus, and Cl. paludis. Intestinal stasis was thought to be the predisposing factor which enabled the organism to proliferate in the intestine and produce an exotoxin. Active immunization was reported to give promising results.

Four years later (1936), Boughton and Hardy[11] reported outbreaks of the disease in Texas. The causal organism was identified as Cl. welchii Type D. This was the first time that the Cl. welchii Type D enterotoxemia was reported in this country.

Newsom and Thorp[74], in 1938, were able to demonstrate that 45% intestinal filtrates from 256 lambs dead of overeating were shown to be toxic for laboratory animals, and 12 out of 20 of the filtrates proved fatal to sheep when injected in amounts varying from 5 to 50 cc. Heating the filtrates to 60° C. for 30 minutes rendered them a toxic. The toxin was neutralized by antisera made from Cl. welchii Types B and D, but not by Types A and C. They concluded that "while overeating seems to differ from enterotoxemia on the basis of symptoms and lesions, the intestinal contents of lambs dead of the two diseases contain toxic substances that seem to be identical".

In the same year (1938), Roberts[84] described a simple

method by which sheep could be made to succumb to enterotoxemia by ingestion of the causal organism. He employed both in vitro and in vivo experiments by using a definite amount of Cl. welchii Type D and a varying amount of milk, and was able to produce the disease at will. In conclusion, he pointed out that the large amount of casein present after ingestion of large quantities of milk would reduce the acid of the abomasum for a sufficient length of time to allow Cl. welchii Type D to propagate and produce enough toxin to cause the disease. For the same reason, a lamb that ingested a small amount of milk would have less casein so that the free acid of the abomasum would inhibit the bacterial growth and consequently no disease would occur.

The next year (1934), Shaw, Muth, and Seghetti[90] reported on the occurrence of "pulpy kidney" disease in Oregon and Cl. perfringens Type D was found to be the cause. Methods of prevention by the use of antitoxins were tried and proved successful.

Boughton & Hardy[12], in 1941, gave a general account of the disease as it occurred in lambs and kids of Texas and they stressed as control measures either passive immunization of susceptible animals or removal to rough, hilly and sparse grass pasture together with penning the animals overnight in a dry lot.

In the same year (1941), Oregon Station workers[79] were able to isolate eleven different strains of the organism responsible for "pulpy kidney" disease. All the strains were found to be potent toxin producers.

Last year (1944), Britton and Cameron[14] made a report of ten outbreaks of so-called enterotoxemia in California, and they felt that "the theory that it is due to absorption of the toxin of Cl. welchii Type D is inadequate". They apparently believed that the chief etiological factor in these outbreaks was the quantity and quality of the ration and the mechanism in fatal cases appeared similar to that operating in "inhibitory ileus" of human beings. They carried on some experiments since last year in order to substantiate this theory. The results were published this year[15] and in summary, they claimed that so-called entero-toxemia was a form of acute indigestion resulting from primary intestinal atony.

5. Pneumonia (bronchopneumonia, laikipia lung disease, pleuropneumonia.)

In 1923, Spray[95] made a bacteriological study of pneumonia in sheep. Two types of pneumonia were studied, namely, a rather edematous type frequently observed in spring lambs and a purulent chronic type found in older sheep. He isolated Pasteurella ovisepticum, a gram-positive diplococcus, and a gram-negative diplococcus. The Pasteurellae were found highly pathogenic for mice, guinea pigs, and rabbits.

Mettam[63], in 1930, described a disease known as "laikipia lung disease" in sheep and lambs. In fact, this condition is a pleuro-pneumonia. This disease ran an acute course in lambs and yearlings but was generally chronic in adults.

Certain complications such as acute arthritis, necrotis lesionsin the mouth, and acute ophthalmia were also found. The causal organism was a pleomorphic gram-negative coccobacillus which was pathogenic for the guinea pigs, rabbit, goat, sheep, and cattle, but not for pigeons.

Two years later (1932), Delpy[22] reported cases of pneumoenteritis in sheep from Persia. According to the post-mortem lesions three clinical types were described, namely, a pneumonic type, a type characterized by intestinal congestion, and a third type which presented lesions similar to hemorrhagic septicemia. Two organisms, a Pasteurella and an undetermined gram-negative rod, were isolated from such cases. An immune serum was prepared from sheep and a formalized bacterin was prepared and each proved satisfactory.

In 1939, Koshelev[49] made investigations on the etiology of pneumonia in sheep and lambs in the Crimea, Russia. The clinical, post-mortem findings, and etiology of the disease were discussed. Various bacteria such as Bacterium coli, Salmonella paratyphi, Pseudomonas pyogenes, and various cocci were isolated from the carcasses of 77 lambs and 5 sheep, but they were all considered secondary invaders. Experimental transmission in laboratory animals and lambs failed. Specific treatment with various sera was of little value but the improvement of hygienic condition was considered to be effective.

Besides these special references, pneumonia of lambs was also reported or considered by Miessner and Koser[65],

Marsh[60], Howarth[42], Veterinary Research Laboratory, Montana[70], and Thorp[98]. These references were previously mentioned in this article.

6. White Muscle disease ("stiff-lamb" disease)

White muscle disease or so-called "stiff-lamb" disease was first reported and described by Metzger and Hagan[64] in 1927 from New York. It affected lambs from two to eight weeks of age and was manifested chiefly by a disturbance of locomotion that usually became worse until either the animals died or were destroyed by the flock owner. The condition was due to muscular changes, the cause of which was not discovered, although apparently it was not the result of an infection. The lesions of the skeletal muscle and heart were briefly described both macroscopically as whitish patches and microscopically as degenerative and not inflammatory in nature. There was practically no cell infiltration by polymorphonuclear or other wandering cells. However, an occasional phagocytic cell was observed.

In the same year (1927), this disease was reported from Montana by Jungherr and Welch[46] but it was regarded as secondary to coccidiosis.

Two years later (1929), Welch, Marsh, and Tunnicliff[103] reported this disease again in Montana. The disease occurred largely in lambs confined to small pens and shed during the first two or three weeks of their life, followed by an unusual amount of exercise on pasture.

The following year (1930), it was reported from Oregon[77].

A chemical analysis of the white muscle from lambs was made and about 0.9 per cent of calcium was found as compared to 0.12 per cent calcium in the normal muscle of the animals. In the same year, workers at New York (Cornell) Station were able to produce the disease experimentally by feeding the pregnant ewes with high-protein grain ration plus alfalfa hay combined with limited exercise.

In 1931, Heath[38] reported on the occurrence of "stiff disease" of lambs in Alberta, Canada, and isolated an acid-fast bacillus. It would seem that Heath's work was not done on a true case of white-muscle disease. The Montana Agricultural Experimental Station (1931)[68] carried on an experiment with lambs and proved that lack of exercise and sunlight would not produce stiff lambs as was suspected two years before. Willman et al (1931)[104] confirmed the earlier work in producing the disease experimentally by feeding pregnant ewes high protein grain mixtures together with alfalfa or clover hay combined with a lack of exercise. In the same report, the authors mentioned that this disease had been reported from Pennsylvania, Maryland, Ohio, Michigan, Wisconsin, Montana, Oregon, and Nevada.

Marsh[60], in 1932, mentioned that the white appearance of muscle fibers in "stiff lambs" disease was due to a deposit of tricalcium phosphate and claimed that there was a disturbance of normal calcium and phosphorus balance in the affected lambs.

In 1934, Slagsvold and Lund-Larsen[93] described a peculiar

disease of young lambs and calves in Norway. The chief lesions consisted of muscular degenerative changes, having some similarity to hyaline degeneration but differing from it by a more pronounced tendency to deposition of calcium. No specific organism or condition was demonstrated as the cause. Adequate rations of minerals and vitamins given to pregnant ewes was considered as an effective preventive measure. Apparently, the authors were dealing with white muscle disease.

In the same year (1934), Willman et al[105] published the results of four years' work and gave a rather extensive account of the cause and histopathology of "stiff-lamb" disease. They were able to produce this condition by feeding the pregnant ewes a ration (the "stiff-lamb" ration) consisting of second-cutting alfalfa hay and a mixture of 3 parts of oats, 3 parts of barley, and 4 parts of cull beans, and proved that neither exercise for the ewes during the winter nor heavy feeding of concentrates or creep-feeding of the lambs was the chief causal factor. They agreed with Metzger and Hagan's[64] work in most respects but claimed that clumps of mononuclear phagocytes and neutrophils were found in the lesions.

The next year (1935), Lee and Scrivner[50] made a study of cases of "stiff-lambs" in Wyoming. They found that the gross lesions were somewhat variable and consisted of arthritis, white skeletal muscle lesions, grayish white areas and streaks in the heart and in the kidneys. Microscopic examination of the white skeletal muscle lesions showed degeneration,

cellular and calcium salt infiltrations. The heart and kidney lesions were found to be areas of coagulation necrosis and purulent foci. Chemical examination showed an increase in calcium and phosphorus. Besides, the authors were able to isolate three different kinds of bacteria from heart blood, namely, bi-polar rods, cocci, and non-spore-forming rods, and typical lesions were reproduced experimentally in lambs and rabbits by means of inoculation with these bacteria. Apparently, Lee and Scrivner were dealing with cases of white-muscle disease complicated by bacterial infection.

Willman et al (1935)[106] sought to determine if the disease was caused by a lack of vitamin C (ascorbic acid). They could not prevent the disease by the addition of ascorbic acid to the new-born lambs. These lambs were from ewes fed with "stiff lamb" ration as long as five weeks after birth. The results indicated that the disease was not due to a vitamin C deficiency.

In 1936, Willman et al[107] tried to test whether the hay was the cause of the trouble. The so-called "stiff-lamb ration" was fed to the ewes out mixed clover and timothy hay was used in place of alfalfa hay. Ten of the 19 lambs in this lot became stiff, which indicated that the concentrate mixture and not the hay was the primary cause of the disease.

In 1937, three articles dealing with various phases of white-muscle disease appeared. Anderson's[3] article stated that "the calcium of the serum of lambs affected with stiff-lamb

disease was lower and the phosphorus higher than that of normal lambs. Many of the diseased lambs showed higher glucose and non-protein nitrogen values, and lower chloride values than normal lambs." The second article was from the Montana Veterinary Research Laboratory[70] which summarized the extent of the disease as follows: "On some farms the losses due to white-muslce disease reached 30-40% and on others, 5% or less. A third of them eventually recovered." The third article on "stiff-lambs" was published by the Wyoming Department of Veterinary Science[114] and a paragraph of it read as follows: "A farm flock of about 400 ewes produced about 10 per cent stiff lambs one year without navel infection when fed on rather poor winter roughage supplemented by native hay during stormy weather. For two years following, when the ration included alfalfa hay, grain, and a mixture of minerals, only 1 per cent of lambs developed symptoms of stiffness". These results seemed to be quite contradictory to the results obtained by Willman and his associates.

Briggs et al (1938)[9] made an attempt to study the cause and cure of "stiff joints". They bought 12 ewes that had typical stiff lambs at side, and these animals were then placed on small wheat pastures. The mothers were fed nothing in addition to the abundant wheat pasture but a simple mineral mixture. A heavy milk flow resulted from this feed. The affected lambs nursed well and made noticeable recoveries.

In the same year (1938), Schofield[86] made a study of the

histo-pathology of "stiff lamb" disease. He found degeneration and fracture of the muscle fibers with calcareous infiltration of the fragments. Cellular infiltration was not extensive but both polymorphonuclear and round cells were present. In the final stages there was cellularity of the affected muscular tissues due to proliferation of fibroblasts and multiplication of the nuclei of the sarcolemma. There was moderate cellular infiltration and numerous portions of damaged muscle remained in the cellular mass. Besides, the liver was found to have small clusters of 10-20 polymorphonuclear cells and a definite hyperplasia of the secretory epithelium was observed in the enlarged thyroid.

Willman et al[108], in 1938, undertook experiments to detect the effect of feeding daily doses of cod liver oil to the lactating ewes and by supplementing the "stiff-lamb" ration with di-basic sodium phosphate. Since this work was not completed, no definite conclusion was drawn.

In 1939, Schofield and Bain[87] reported that "stiff lamb" disease was an aphosphorosis because they found that a small group of affected lambs recovered after receiving doses of dilute phosphoric acid in milk. Sholl[92] in the same year reported the occurrence of stiff-lamb disease in Michigan where losses from the affection were very heavy (30% loss on some farms). Blood chemistry determinations were made, and low creatinine and high non-protein nitrogen were found. Vawter and Records (1939)[100] observed "white muscle" disease of lambs from ewes receiving alfalfa hay alone but not in

ewes receiving alfalfa plus rolled oats. The authors had the feeling that the disease might be due to a phosphorus deficiency, because the alfalfa hay in Reno, Nevada, was somewhat low in phosphorus whereas rolled oats was a good source of phosphorus. Besides, they felt that the term "stiff lambs" was confusing and should be separated from the term "white muscle" disease.

Willman et al (1939)[109] continued the work with the "stiff lamb" ration supplemented with phosphorus, but no significant results were found after using this element.

In 1940, Willman et al[110] summarized the results of the three previous years work. The disease was of nutritional origin and was not present at birth. The feeding of maize silage, bone meal or sodium phosphate to the ewes failed to prevent the condition, as did drenching the young lambs with the equivalent of 60-180 mgm. ascorbic acid per week. The incidence of the disease appeared to be markedly reduced in a controlled experiment in which unextracted wheat germ meal was added to the basal maternal diet of oats, barley, cull beans and lucerne hay.

In the same year (1940), Mills[67] considered that the term "stiff lambs" referred merely to a single symptom and not to any special entity and he felt that it was advisable to discuss the conditions under a heading which related to etiology rather than to symptoms. He recognized four causes of lameness in lambs, to which the term "stiff lambs" was commonly applied, and they were: (1) Erythropelothrix

rhusiopathiae infection, (2) tetanus, (3) "feed allergy" in old lambs, and (4) "feed allergy" in unweaned lambs. The allergenic substance also appeared to be present in the ewe's milk. Apparently, Mills considered "white muscle" disease as a symptom of "feed allergy" although he did not mention it.

Willman et al[111] in 1941 continued experiments on the "stiff lamb" ration with and without the addition of unextracted wheat-germ meal. With the ewes fed the "stiff lamb" ration, eleven of twenty-seven lambs became stiff. No stiff lambs developed in the wheat-germ supplemented lot. In the same year (1941) the Oregon Station[79] reported the occurrence of "stiff lambs" and stated that no organism was found in connection with the disease.

In 1942, Melass[62] carried out further investigation on the cause of stiff lamb disease and mentioned that several possible factors were responsible for the condition. Of these factors, high protein content of the ration (cystine deficiency), presence of toxic substance in the ration, and the question of mineral inbalance were all possible but needed further study.

Sályi (1942)[85] described a disease in young lambs which showed cardiac and skeletal muscle dystrophy. The termination of the disease was generally fatal; bronchopneumonic, arthritic, and enteric complications often supervened before death. The gross lesions were typical grey or greyish-yellow areas in the heart and skeletal musculature. Bacteriological investigations proved negative, and nutritional deficiencies

were suspected. Probably Salyi was dealing with typical cases of white muscle disease complicated by some other infections.

Willman et al (1944)[112] conducted an experiment with three lots each of 24 ewes and their lambs to determine whether or not "stiff lamb" disease could be prevented by feeding supplements of vitamin E to a ration (cull beans and alfalfa hay) that produced the disease. As a result, 36 of 38 lambs became stiff when raised by ewes fed this ration, and one when raised either by ewes fed in addition two doses weekly of 9 cc. per ewe of an 8.25 per cent solution of mixed tocopherols in cottonseed oil twenty-four days before lambing or by treating the lambs with 2 cc. of an olive oil solution of 140 mgm. of alpha-tocopherol acetate. Treatment of the affected lambs with 100 mgm. of water soluble disodium salt of d. l-alpha tocopherol phosphoric acid ester was tried and proved quite effective in uncomplicated cases.

Besides those special works just cited, white muscle disease was also briefly mentioned by Marsh,[59,60] Howarth[42], Montana Veterinary Research Laboratory[70], Oregon Station[78], Dykstra et al[23], Thorp[98], and Care[17] under the heading "diseases of new-born lambs in general".

7. Miscellaneous Diseases

(1) Bacterium coli infection. This diseased condition was reported by Volkova[102] in 1938. Eighteen new-born lambs

died following an illness lasting from 20 minutes to 30 hours after the appearance of initial symptoms of trembling, stumbling, and high temperatures but no diarrhea. In each case, the bacteriological examination showed cultures of Bacterium coli virulent both for laboratory animals and for a lamb when given intravenously.

(2) Dysentery. Lamb dysentery was reported by Welch et al from Montana[103], 1929. It was stated that dysentery occurring in Montana differed in so many respects from the English lamb dysentery that the results obtained in Great Britain were of little or no value in controlling the disease in Montana. The authors claimed that a combination of cold, wet weather and the presence of strains of intestinal bacteria was responsible for such diseased condition rather than a specific pathogenic micro-organism.

(3) Navel infection. In 1934, Bullard[16] published a case report on navel infection of a lamb. The causal agent was Staphylococcus aureus and the disease was septicemic and associated with metastatic abscess formation.

(4) Necrobacillosis. As noted above, necrobacillosis of the navel was mentioned by Gallagher[30] and that of the liver by the Montana Veterinary Research Laboratory[70] and Mills[66]. In 1944, Marsh[61] reported on cases of necrobacillosis affecting the rumen. Marsh believed that lamb losses from necrobacillosis might occur as a result of primary infection of rumen from which secondary invasion of the liver and sometimes the diaphragm and lungs resulted.

In all cases, according to Marsh, Fusiformis necrophorus was isolated.

(5) Paraplegia. Snieckiene[94], in 1937, reported cases of enzootic paraplegia of lambs from Lithuania. The illness was described as progressive paraplegia and lasted from about four days to three weeks. The post mortem findings included pale, flecked skeletal and heart muscles, renal congestion, degeneration of liver and catarrh of the respiratory and digestive tracts. The cause was unknown.

(6) Pasteurellosis. Pasteurellosis was found to occur as an epizootic among Algerian sheep in 1909 by Caze[18]. Micro-organisms were found in great numbers in the blood, spleen, liver, lungs, and supra-renals.

(7) Septicemic infection by swine erysipelas. In 1919, Christiansen[19] made a post-mortem report on a lamb 38 hours old which revealed hemorrhagic enteritis, great enlargement of mesenteric glands, degenerative changes in other organs, and small hemorrhages under the endocardium and epicardium. There had been a high mortality of lambs in this flock. Swine erysipelas bacilli were isolated in pure culture.

III. Experimental studies

1. Materials and methods

(1) Sources of materials. The new-born lambs used in this study came from two sources, namely, from the flock at Michigan State College and from farms located in the State. During an entire lambing season extending from March to June

of the year 1944, more than a hundred lambs were brought to the experimental barn or laboratory of this Department for treatment or autopsy. This is a report of 55 cases which were autopsied and studied with the idea of trying to get a better understanding of the diseases of lambs. Of the 55 cases, 37 came from the college flock while 18 came from farms in the State. Besides the 55 pathological cases, 16 healthy or normal animals from the college sheep flock were used in various control studies, making a total of 71.

(2) Methods of study.

A. Methods for studying blood and blood chemistry.

(a) Blood studies on normal animals. Since a study of this kind is rather new and data from other workers meager, it was necessary to determine the blood picture and blood chemistry of normal lambs for control purposes and normal ewes as a check-group before attempting to study pathological cases.

A 2-inch 16-gauge needle or 3-inch 15-gauge needle (the so-called "California bleeding needle") was used to draw 15 cc. of blood from the jugular vein (from each of 8 normal lambs and 8 normal ewes) into a test-tube containing dry potassium oxalate (seven drops of a 30 per cent solution delivered from a 1 cc. pipette after having been evaporated to dryness in a hot oven). As soon as enough blood was obtained and the needle was loosened from the vein, at least three blood smears were immediately made by tipping the needle and allowing a small drop of blood to collect on

the end of the clean slides after which each drop was smoothly pushed forward by the end of another clean slide so as to get a uniform smear. The smears were instantly dried by waving them in the air and they were then saved for differential counts. At the same time, the blood-sample in the test-tube was constantly but gently rotated in order to prevent the blood from clotting. Each of these blood samples was to be used for the cellular count, hemoglobin determination, and chemical analysis. The time for bleeding such normal sheep was purposely arranged to cover a considerable length of time. The normal lambs were bled twice, once on May 4, 1944, and again on June 1, 1944, each bleeding being carried out on four animals. The ewes were bled at five different times, one each on April 4, April 6, April 7, April 20, and four on June 1, 1944.

The counting of blood was made as soon as the blood samples reached the laboratory. The ordinary method of blood counting was employed so that the detailed procedure will not be mentioned here. Leake and Guy's platelet dilution fluid was used for red blood cell counts. The diluting fluid for white blood cell counts consisted of 8 cc. distilled water in a small vial to which was added 4 drops of Gram's crystal violet. Both diluting fluids were found to be quite satisfactory. For mixing blood cells, the blood pipette rotor (Bryan-Gerry)* was used and it seemed to be very well adapted to the work. In order to cleanse the diluting pipettes, a set of home-made suction bottles with the

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* Sold by A. S. Aloe Co., St. Louis, Missouri.

aid of a rubber pipette adaptor was found to be quite efficient.

For hemoglobin determination, Sheard-Sanford's method was strictly followed[40]. The necessary solutions were accordingly prepared, and the reading was made by means of a Cenco-Sheard-Sanford photolometer. The value of hemoglobin in terms of gram per 100 cc. of blood was determined according to the calibration chart provided by the Central Scientific Co., Chicago, Ill.

For chemical analysis of the blood, the methods of W. S. Hoffman[40] were followed with the exception of blood chloride determination. Five different blood components were determined by these methods, namely, glucose, uric acid, creatinine, non-protein nitrogen (N.P.N.), and urea-nitrogen. Whitehorn's method[36] was used for the determination of blood chloride.

The blood smears were stained with Wright's stain, dried, and counted under the microscope by the so-called "battlement" or "palisade" method.[52]

The results of the preliminary study of blood are summarized and arranged in Tables (1) and (2).

Table (1) The blood picture of normal lambs and ewes.

Series No.	Identification No.	Breed	Age (Days)	Date Bled	R. B. C. per c.mm.	Hb. Gm.%
1	14a-223	Shropshire	46	5/4/44	10,340,000	11.0
2	22-179	"	39	"	11,950,000	8.6
3	27-469	"	35	"	10,980,000	12.5
4	32-979	Oxford	53	"	9,070,000	7.8
5	943	Black Top Delaine	78	6/1/44	10,370,000	12.1
6	944	"	69	"	10,670,000	12.5
7	945	"	64	"	9,730,000	11.8
8	950	Rambouillet	72	"	10,550,000	11.1
Average of normal standard			For 7 lambs		10,444,286	10.90
			For 8 lambs		10,457,800	10.92
1	7356E	White-face Grade	4 yr.	4/4/44	6,830,000	10.10
2	7358E	Hampshire	5 yr.	4/6/44	6,590,000	9.20
3	7362E	Shropshire	4 yr.	4/7/44	8,880,000	12.70
4	7393E	"	2 yr.	4/20/44	10,900,000	10.40
5	12	"	5 yr.	6/1/44	9,380,000	11.40
6	13	"	5 yr.	"	7,490,000	8.45
7	19	"	3 yr.	"	7,390,000	9.20
8	25	Rambouillet	4 yr.	"	10,900,000	9.20
Average or normal standard					8,607,000	10.08

Note:
R. B. C.....Red blood cell count
Hb.....Hemoglobin value
W. B. C.....White blood cell count
N.....Neutrophils
L.....Lymphocyte
M.....Monocyte
** Lamb 950 probably had some infection, and it might be omitted, although the averaged values of 8 animals were calculated in the table for reference.

nd ewes.

	R. B. C. per c.mm.	Hb. Gm.%	W. B. C. per c.mm.	Differential leucocyte count, %				
				N	L	M	E	B
	10,340,000	11.0	10,650	19	80	1		
	11,950,000	8.6	7,050	34	66			
	10,980,000	12.5	5,400	20	77	1	2	
	9,070,000	7.8	5,400	39	58	1	2	
	10,370,000	12.1	9,250	39	60		1	
	10,670,000	12.5	8,950	22	77		1	
	9,730,000	11.8	9,150	21	75	1	3	
	10,550,000	11.1	19,000	58**	35		7	
	10,444,286	10.90	7,979	28	70	0.57=1	1.3=1	
	10,457,800	10.92	9,356	31	66	0.5=1	2	
	6,830,000	10.10	5,850	20	76	2	2	
	6,590,000	9.20	10,450	36	63			1
	8,880,000	12.70	5,700	13	79		8	
4	10,900,000	10.40	15,400	40	56		4	
	9,380,000	11.40	5,300	38	56		6	
	7,490,000	8.45	7,150	38	62			
	7,890,000	9.20	6,450	28	65	1	6	
	10,900,000	9.20	9,800	28	65		7	
	8,607,000	10.08	8,283	30	65.25	0.37=1	4	0.12=0

E.....Eosinophils
B.....Basophils

t might be
animals were

Table (2). Chemical analysis of blood of normal lambs and ewes.

Series No.	Identification No.	Date Bled	Glucose Mgm.%	Uric Acid Mgm.%	Creatinine Mgm.%	Chloride Mgm.%	N.P.N. Mgm.%	Urea-N Mgm.%
Lamb 1	14a-223	5/4/44	61.00	1.55	0.80	390	38.00	17.8
2	22-179	"	71.11	1.55	0.78	460	27.6	12.2
3	27-469	"	65.00	1.50	0.80	410	32.8	12.2
4	32-979	"	80.00	1.55	1.40	440	35.6	13.0
5	943	6/1/44	57.50	1.90	1.20	460	27.0	15.8
6	944	"	67.00	2.35	1.10	470	36.0	13.5
7	945	"	62.00	2.00	1.20	470	36.0	14.9
8	950*	"	55.50	1.30	1.00	420	26.0	13.0
Averaged value or Normal Standard			64.89	1.71	1.04	440	32.37	14.17
Ewe 1	7356(E)	4/4/44	38.00	0.22	0.40	350	30.0	16.8
2	7358(E)	4/6/44	85.00	1.10	0.80	420	30.0	7.5
3	7362(E)	4/7/44	30.71	2.22	0.40	365	20.0	14.9
4	7393(E)	4/20/44	51.82	1.65	0.80	460	24.5	11.2
5	12	6/1/44	43.30	2.10	1.20	380	41.0	18.2
6	13	"	37.00	1.90	1.10	440	41.0	21.8
7	19	"	50.00	1.48	1.00	450	36.0	14.9
8	25	"	52.00	1.95	1.20	460	28.0	15.8
Averaged value or Normal Standard			48.48	1.58	0.84	415.62	31.31	15.14

Note:*
The chemical analysis of blood of lamb No. 950 did not show any marked difference from the other normal animals so that it was included in the calculation of averaged value for the normal standard. Probably, this may be one reason to explain why this lamb appeared normal at the time of bleeding although its blood picture showed some kind of infection as mentioned above (Table 1).

Table (1) shows the average blood cell value of 8 normal lambs. The average erythrocyte count was 10,444,286 per c.mm., leukocyte count 7,979 per c.mm., and hemoglobin 10.9 grams per 100 cc. of blood. The average differential count showed 28% neutrophils, 70% lymphocytes, 1% monocyte, and 1% eosinophil. If these values are compated with those of normal ewes, it is interesting to note that while the red blood cell count of normal ewes (8,607,000 per c.mm.) is decidely lower and eosinophils of the same animals (4%) decidedly higher, the remaining figures were nearly the same.

From Table (2) and referring to the average values, one generalization can be drawn, the blood glucose in normal lambs is much higher than that found in normal ewes (64.89 mg. per 100 cc. blood in lambs against 48.48 mg. per 100 cc. blood in ewes), while the other elements in the blood approximate each other.

In view of the fact that the results of this preliminary study on blood were rather constant in almost every respect, it seemed justifiable to use these values as a normal standard and as a control for the pathological cases.

(b) Blood studies on pathological cases. The blood from pathological cases was studied by using the same methods employed for the normal animals. For the entire lambing season of last year, a total of 12 pathological cases was available for blood study. The results are herewith briefly summarized in Table (3). The detailed account of the findings will be presented in connection with each individual case.

Table (3). A summary of blood studies on pathological lamb.

Case No.	R. B. C.* per c.mm.	Hb. gm.%	W. B. C. per c.mm.	Differential leucocyte count								Chemica
					Neutrophils							
				L	s	b	j	m	M	B	E	G
7338	10,400,000	11.4	12,150	12	6	79	3					41.66
7343	5,810,000	6.3	14,900	14	3	46	7	29	1			15.00
7344	8,990,000	11.4	800	74	8	8	4	6				28.00
7393	6,740,000	6.8	23,900									75.55
7394	8,710,000	12.0	14,600	18	73	7			2			74.44
7356	12,250,000	9.8	11,600	7	88	4			1			196.00
7358	9,370,000	6.3	13,250	2	35	47	9	7				115.00
7362	8,670,000	9.8	9,950	35	56	6	3					97.50
7400	10,070,000	10.3	6,150	18	46	4	29	3				77.77
7406	10,390,000	10.3	1,700	6	5	43	24	22				200.00
7415	8,970,000	7.8	14,150	2	78	18			2			170.00
7474	9,070,000	10.8	2,200	37	40	21				1	1	88.75

*Explanation of the abbreviations:

R. B. C.....Red blood cell count
Hb.....Hemoglobin
W. W. C.....White blood cell count
L.....Lymphocytes
M.....Monocytes
E.....Eosinophils
B.....Basophils

s.....segmenters
b.....bands
j.....juveniles
m.....myelocytes

G
U
C
C
N
U

count		Chemical analysis of blood, Mgm./100 cc.						Erythrocytes	Diagnosis
B	E	G	U	CE	Cl	N.P.N.	UN.		
		41.66	2.00	2.10	400	106.00	55.30	Normocytic	Staphylococcic infection
		15.00	0.12	0.20	480	25.00	20.00	Some poikilocytic, hypochromic.	Navel infection
		28.00	0.70	0.60	400	35.80	30.00	Normocytic; some nucleated	Navel infection, etc.
		75.55	3.12	0.80	520	67.00	39.00	"	"
		74.44	1.10	0.80	460	16.00	11.20	Normocytic; few poikilocytes	
		196.00	1.78	1.65	560	162.00	108.00	"	White muscle disease
		115.00	4.16	3.76	550	118.50	98.00	Microcytic hypochromia; poikilocytic	White muscle disease, etc.
		97.50	1.10	1.00	430	34.00	11.20	Normocytic	Docking infection
		77.77	0.50	0.70	380	15.00	9.90	Some crenated & poikilocytic	Acute Meningitis
		200.00	1.44	1.65	420	21.00	20.70	Marked poikilocytosis	Acute peritonitis
		170.00	2.60	1.30	460	41.00	28.30	Microcytic hypochromia; some nucleated	Bronchopneumonia & encephalitis
1	1	88.75	2.60	1.15	350	30.00	18.20	Normocytic	Bronchopneumonia and constipation

G.....Glucose
U.....Uric acid
Ce.....Creatinine
Cl.....Chloride
N.P.N.....Non-protein nitrogen
UN.....Urea-nitrogen

B. Bacteriological methods.

(a) Technic of culturing organs and isolating bacteria.

The lamb was killed (or brought in as a dead carcass), skinned and opened according to the usual autopsy procedure which will be described later. A careful examination of the carcass was then made, and the liver, spleen and heart-blood were cultured for bacteria and smears from these materials made for preliminary bacterial examination as a routine. If gross lesions were present, the material from the lesions was also cultured and smears made.

In order to make such cultures and smears, a simple but effective technic was devised which is stated as follows: An old autopsy-knife was heated and used to sear the surface of the left diaphragmatic lobe of the liver. A sterile inoculating needle was used to bore deeply into the liver at the spot which had been seared, after which it was twisted around in order to get the liver material as well as blood. The needle was withdrawn and immediately several streaks were made on one-third of a blood-agar plate. The smear from the liver was made on at least two clean slides, one to be stained with Wright's stain and the other with Gram's stain.

After the surface of a portion of the spleen had been seared, a pair of sterile scissors and forceps were used to remove a small piece of tissue and a direct smear was made on another one-third of the same blood-agar plate used for the liver. Smears for preliminary bacteriological examination were also prepared.

The heart-blood was cultured using the following technic: The ventral surface of the heart was seared, and a sterile pipette with a pointed end was pushed through the wall of the right auricle in order to get a small amount of blood and a smear was made on the remaining one-third of the same blood-agar plate (liver and spleen). At the same time, a drop of blood was used to prepare smears on slides and these were stained for the preliminary bacteriological examination.

The gross lesions were prepared for culturing by searing the surface. After that, either a sterile needle was used for getting the fluid exudate from a pyogenic process or a scissors and forceps were used to remove a small piece of the affected tissue if the lesion was firm. Smears from such materials were made on blood-agar plates as well as on clean glass slides. For culturing anaerobic micro-organisms, the material from various lesions was transferred into deep brain broth which had been boiled for fifteen minutes and cooled without agitation.

After all the cultures were made, the blood-agar plates were incubated at 37°C. for 24-48 hours and observations made daily for visible bacterial growth after which they were left at room temperature for several days.

In order to isolate bacteria free from contamination, the following procedure was followed. Whenever visible bacterial growth was observed on the blood-agar plates, smears from the colonies were made and stained with Gram's

stain in order to determine whether or not the culture was pure. If the culture was pure and not contaminated, a single colony was transferred onto a blood-agar slant. If the culture was a mixed, a small portion was diluted with a few cubic centimeters of sterile nutrient broth and plated until a pure culture was obtained. If the culture was contaminated and worthless, the original blood-agar plates were discarded. The transfers were incubated at 37°C. for 24 hours, and the examination for pure culture was performed again. If the cultures were found to be pure, they were kept in a refrigerator as stock cultures. In maintaining such stock cultures, a transfer was made and rechecked once for every three weeks or sometimes two weeks for those which showed sparse growth.

By using the methods and technics described, 41 aerobic cultures and 7 anaerobic cultures were isolated from pathological lambs throughout the period of this study.

(b) Bacteriological tests.

a. Preparation of media. Most of the media were prepared according to the Manual of Methods for Pure Culture Study of Bacteria[2]. Modifications of media preparation will be described. With the exception of litmus milk, dextrin, and maltose, all media were sterilized in the autoclave at 15 pounds pressure (121°C.) for 20 minutes, and the pH adjusted to approximately 7.3-7.5 (fermentable media with pH 7.0-7.1 as an exception) by means of Beckman pH-meter. For sterility tests of media, at least one "pilot" was selected from each lot and incubated at 37°C. for 24 hours.

Basal fermentation medium.

Formula

Bacto-tryptose.....2.0 per cent
Beef extract.....0.7 " "
NaCl.....0.5 " "
Distilled water .....q.s.(amount for completion)

Procedure. The basal medium was used to prepare all the different media for fermentation tests. Five liters of this medium were prepared at one time by dissolving the necessary ingredients in water and autoclaving as usual and kept as stock solution for later use.

Blood-agar plates.

Formula

Bacto-tryptose agar.... .....q.s.
Distilled water.....q.s.
Defibrinated sheep blood.. .....5.0 per cent

Procedure. Sufficient amount of Bacto-tryptose agar was suspended in cold distilled water and heated until the agar was dissolved. It was then autoclaved and cooled to about 45°C. The blood was added and mixed gently by a whorling action, and then poured aseptically into Petri dishes.

Blood-agar slants. The Bacto-tryptose agar was dissolved, tubed, autoclaved, and cooled as for the plates. The sheep blood was then proportionally added to each tube by aseptic methods and mixed so as to get a uniform slant. After the slants were cooled and the sterility test proved satisfactory, 0.5 cc. of nutrient broth was added to the base of each slant.

Deep brain medium (for anaerobic cultures)

Formula

Sheep brain.....1 part
Distilled water.....1 part
Peptone (Difco).....2 per cent
Dextrose.....0.1 per cent
NaCl.....0.5 " "

Procedure. The brain was finely ground in a mortar with an equal amount of distilled water and the other ingredients added. The mixture was then heated until the peptone was completely dissolved and the pH was adjusted to approximately 7.6-7.8. The medium was tubed and sterilized as usual.

Fermentable media. The basal medium was used to prepare 18 different kinds of fermentable media. They were as follows: arabinose, dextrin, dulcitol, fructose, galactose, glucose, glycerol, inositol, inulin, lactose, maltose, mannitol, raffinose, salicin, sorbitol, sucrose, trehalose, and xylose. Of these 18 media, 2 of them (dextrin and maltose) were prepared separately by cold filtration, while all the others were made by adding the fermentable substances in amounts of 0.1%-0.5% (0.1% for the rare substances) to the basal medium, tubing, and autoclaving as usual with brom cresol purple as the indicator.

Since dextrin and maltose are complex compounds which may be broken down to simpler forms by high temperature in the autoclave, these two substances were specially prepared by cold filtration. The needed amount of fermentable substance (dextrin or maltose) was calculated, dissolved in

a certain amount of distilled water and filtered through a sterile Berkefeld filter. The sterility test was performed, and at the same time the basal fermentation medium with brom-cresol purple indicator was tubed and autoclaved. If the sterility test of the filtrate proved satisfactory, it was added aseptically to the sterile tubed basal medium to make a 0.5 per cent final concentration after which another sterility test was made.

The fermentable substances were used in the following concentrations:

Arabinose	.1 per cent	Lactose	.5 per cent
Dextrin	.5 " "	Maltose	.5 " "
Dulcitol	.1 " "	Mannitol	.1 " "
Fructose	.1 " "	Raffinose	.1 " "
Galactose	.1 " "	Salicin	.1 " "
Glucose	.5 " "	Sorbitol	.1 " "
Glycerol	.1 " "	Sucrose	.5 " "
Inositol	.1 " "	Trehalose	.1 " "
Inulin	.1 " "	Xylose	.1 " "

Litmus milk. Litmus milk was prepared by adding litmus powder to freshly-separated skim milk until a suitable color was obtained. The milk was then tubed and sterilized by placing the medium in flowing steam for 30 minutes daily for four days. During warm weather, it was necessary to place the medium in the refrigerator between the first and second heating.

Gelatin medium.

Formula (for 400 cc.)

Gelatin	2.0 g.
Tryptose (Bacto)	4.0 g.
Na_2HPO_4	0.75 g.
Glucose	0.40 g.
Sodium thioglycollate	0.40 g.
Beef extract	0.80 g.
Distilled water	400.00 cc.

Procedure. The ingredients were dissolved, tubed, and autoclaved as usual.

Medium for H_2S production.

Formula (for 400 cc.)

Liver infusion (Huddleson, 1934)	200 cc.
Distilled water	200 cc.
Witte's peptone	4 g.
NaCl	2 g.

Procedure. The liver infusion was prepared first by heating for 1.5 hours in flowing steam 1 lb. of fresh and grease free beef liver in 500 cc. of tap water and filtering through a wire screen. 200 cc. of the infusion were mixed with other necessary ingredients and the mixture was dissolved, tubed, and sterilized.

Medium for indole formation.

Formula (for 250 cc.)

Bacto-tryptone	5.0 g.
Na_2HPO_4	0.5 g.
Glucose	0.25 g.
Agar	0.25 g.
Beef extract	1.75 g.
Distilled water	250.00 cc.

Procedure. Same as for preparing gelatin medium.

Medium for nitrate reduction.

Formula (for 500 cc.)

Bacto-tryptose	10.0 g.
Na_2HPO_4	1.0 g.
Glucose	0.5 g.
Agar	0.5 g.
KNO_3	0.5 g.
Beef extract	3.5 g.
Distilled water	500.0 cc.

Procedure. Same as for preparing gelatin medium.

Nutrient broth. The following formula was used, and the procedure was the same as that used for preparing basal fermentation medium.

Beef extract	0.5 per cent
NaCl	0.5 " "
Peptone (Difco)	1.0 " "
Distilled water	q.s.

Potato medium. Giltner's method[32] was used for preparing this medium. Several good-sized sound potatoes were carefully cleaned, and by means of a cork borer they were cut into cylinders, about 5 cm. long and 1.2 cm. in diameter, followed by diagonal cutting each with an ordinary knife. These slants were then soaked in a dilute aqueous solution (1:500) of Na_2CO_3 for 30 minutes and washed in several changes of distilled water. A glass rod (2 cm. x 0.3 cm.) was placed at the bottom of each test-tube, and the treated potato slant was placed into the test-tube followed by the addition of about 1 cc. of distilled water. These tubes were immediately plugged with cotton and sterilized in the autoclave.

Starch-agar plates.

Formula (for 400 cc.)

Beef extract	2.8 g.
Peptone	2.0 g.
Agar	6.0 g.
Starch (soluble)	0.8 g.
Distilled water	400.0 cc.

Procedure. The ingredients were dissolved, autoclaved as usual, and the plates poured.

b. Biochemical tests. For all biochemical tests, the methods as recommended in the Manual of Methods for Pure Culture Study of Bacteriae were generally used. The inoculations were made directly from a 24 hour nutrient broth culture in each case and an uninoculated tube or plate of the particular medium involved was used as a control which was subjected to the same technic and to the same conditions as the inoculated ones. These inoculated tubes or plates were incubated at 37°C. for varying lengths of time, and the tests were always performed in duplicate and sometimes in triplicate. The tests are outlined as follows:

Motility test. Nutrient broth was inoculated and incubated for 12 hours. At the end of the incubation period the motility of the micro-organisms was determined by hanging-drop method.

Observation for turbidity, sedimentation, and pellicle formation. These observations were checked in nutrient broth culture after incubation for 24, 48, and 72 hours. The daily readings were recorded but the one following the 48 hour incubation period will be the only one presented.

Hemolytic test. Production of hemolysin was determined by streaking the cultures on blood-agar plates and reading after incubation for 24, 48, and 72 hours.

Starch hydrolysis. The ability of the micro-organisms to hydrolyze starch was detected by growing the cultures on starch-agar plates for one week and then covering the surface with a 50% alcoholic solution saturated with iodine.

Growth and pigment-formation on potato medium. The ability to grow and to form pigment on potato medium was checked by inoculating potato slants and recording the results at intervals similar to those for fermentation tests during the two week incubation period.

Liquefaction of gelatin. The inoculated tubes were incubated at 37°C. for two weeks, and during this period they were examined for liquefaction at intervals of 48 to 72 hours. In making the readings, the tubes were placed in the refrigerator for about one hour until the gelatin medium in the negative control tubes solidified.

H_2S determination. H_2S production was detected by observing the blackening of lead-acetate paper strips suspended over the medium during an incubation period of two weeks with the readings being made at intervals of 48 to 72 hours.

Indole test. Indole production was determined by the Thorp and Tanner[97] modification of the Gore[1] Technic in which solution No. 1 and 2 (Manual of Methods for Pure Culture Study of Bacteria, 1936, up to date revision)[2] were applied to narrow strips of sterile filter paper instead of to cotton plugs. These strips were carefully labeled with pencil and suspended over the appropriate inoculated medium which had been incubated for four days. The tubes were then placed in a boiling water-bath for 15 minutes and the results read after removing the tubes from the bath.

Nitrate reduction. After incubation for four days, the reduction of nitrate was determined by using solutions of

sulphanilic acid and a-naphthylamine.[2]

Litmus milk. The reaction of micro-organisms in litmus milk was checked by incubating the milk cultures for two weeks and reading at same intervals of time as for the fermentation tests.

Fermentation test. The ability of the micro-organisms to react upon various fermentable media (including 12 carbohydrates, 5 alcohols and 1 glucoside) was determined by inoculating these media and incubating for two weeks. Readings of the reactions were made at 24 hour intervals for the first three days and at 48 hour intervals for the remaining time with doubtful cases being checked with the Beckman pH-meter.

The results of biochemical tests. The results of the biochemical tests will be briefly given in Table (4).

Table (4)

The biochemical characteristics of bacterial cultures isolated from pathological lesions.

Strain No.	Culture No.	Source	Gram stain & form	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Identification
(3)	7334	Heart blood	Gram-negative rod	−	−	+	+		−	−	−	−	+	−	−	−	+	−	+	+	+	−	−	−	−	+	+	−	−	+	+	−	−	Pasteurella
(4)	7335	"	"	−	−	+	+	−	+	−	−	−	+	[illegible]	−	−	+	−	+	+	+	−	−	−	−	+	+	−	−	+	+	−	+	"
(13)	7371a	Pericardial sac	"	−	−	+	+	−	−	+	+		+	−	−	−	+	−	+	+	+	−	−	−	−	+	+	−	−	+	+	−	+	"
(22)	7372b	Lung	"	−	−	+	+	−	−	+	+	−	+	−	−	−	−	−	+	+	+	−	−	−	−	−	+	−	−	+	+	−	+	"
(24)	7372b	"	"	−	−	+	+	−	−	+	+	−	+	−	−	−	−	−	+	+	+	−	−	−	−	−	+	−	−	+	+	−	+	"
(32)	7471a	Bronchus	"	−	−	+	+	+	+	−	+	−	−	−	−	−	−	−	+	+	+	−	−	−	−	−	+	−	−	+	+	−	+	"
(5)	7338	Heart blood	"	+	−	+	+	−	+	+	+	r	−	−	−	(+)	(+)	(+)	(+)	(+)	(+)	(+)	−	−	(+)	(+)	(+)	−	(+)	(+)	(+)	(+)	(+)	Escherichia
(27)	7708c	carpal arthrosis	"	+	−	+	+	−	−	+	+	"	−	−	−	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	−	(+)	(+)	(+)	−	+	+	+	+	+	"
(28)	7703c	Lung	"	+	−	+	+	−	−	+	+	"	−	−	−	(+)	(+)	(+)	(+)	(+)	(+)	(+)	−	−	(+)	(+)	(+)	−	(+)	(+)	(+)	(+)	(+)	"
(2)	7319	Lung	"	−	−	+	+	−	−	+	−	−	+				+		+	+	+	+	+	−	+	+	+	−	−	+	+	−	+	[illegible]
(8)	7340	"	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	+	+	+	+	−	+	+	+	−	−	+	+	−	+	"
(9)	7350	"	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	+	+	+	−	−	+	+	+	−	−	+	+	−	+	"
(10)	7351	"	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	+	+	−	+	−	+	+	+	−	−	+	+	−	−	"
(11)	7353	Heart blood	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	+	+	+	+	−	+	+	+	−	−	+	+	−	+	"
(30)	7394b	"	"	−	−	+	+	−	−	−	−	−	+	−	−	−	+	−	+	+	+	+	−	−	+	+	+	−	−	+	+	−	−	"
(32)	7401b	"	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	+	+	+	−	−	+	+	+	−	−	+	+	−	+	"
(36)	7410	Lung	"	−	−	+	+	−	−	−	−	−	+	−	−	−	+	−	+	+	+	+	+	−	+	+	+	−	−	+	+	−	+	"
(37)	7418	"	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	+	+	+	−	−	+	+	+	−	−	+	+	−	+	"
(38)	7443	"	"	−	−	+	+	−	−	+	−	−	+	−	−	−	+	−	+	−	+	+	−	−	+	+	+	−	−	+	+	−	+	"
(42)	7474	"	"	−	−	+	+	−	−	+	−		−	−		−	+	−	+	+	+	+	+	−	+	+	+	−	−	+	+	−	+	"
(34)	7401b	Bronchus	Gram-negative diplococcus	−	−	+	+	−	−	+	−	−	−	−	−	−	B		−	b	−	−	b	−	−	−	b	b	−		−	b	b	Neisseria
(15)	7362a	Liver	Gram-negative coccobacillus	−	−	+	+	−	+	+	−	r	−	−	−	+	+		+	+	+	−	−	+	+	+		+	+	−	+	+	+	Undetermined
(7)	7338	Pericardial sac	Gram-positive coccus	−	−	+	+	+	+	+	−ccr.		+	B	−	−	+Rt	−	+Rt	−	+Rt	+	−	−	+Rt	+	+Rt	−	−	−	+Rt	−	−	Staphylococcus
(23)	7371b	Lung	"	−	−	+	+	+	+	+	−	"	+	B	−	−	+	−	"	−	"	+	−	−	"	+	+	−	−	−	"	−	+	"
(29)	7394a	carpal joint	"	−	−	+	+	+	+	+	−	"	+	B	−	−	+Rt	−	−	−	"	+	−	−	+	+	+	−	−	+	−	−		"
(13)	7398	Right stifle	"	−	−	+	+	−	+	+	−	"	+	M	+	−	+	−	"	+Rt	"	+Rt	−	+	+	+	+	+	+	+	+	+	−	"
(73)	7373a	Lung	"	−	−	+	+	−	+	+	−	"	+	−	+	−	+	−	"	+Rt	+	+	−	+Rb	+	+	+	+	+	+	+	−	+	"
(41)	7460c	"	"	−	−	+	+	−	+	+	−	"	+	−	+	−	+	−	"	+Rt	+	+	−	+Rb	+	+	+	−	+	+Rt	+	−	−	"
(6)	7337	Liver	"	−	−	+	+	−	−	+	−	r	+	Y	−	−	+Rt	−	"	−	+Rt	+	−	−	+Rt	+Rt	+	−	−	−	+	−	−	"
(31)	7430	Heart blood	"	−	−	+	+	−	−	+	−	r	+	Y	−	−	+	−	+	−	+	+	−	−	+	+Rt	+	−	−	−	+	−	+	"
(12)	7358a	Right stifle	"	−	−	+	+	+	−	+	−	[illegible]	+	−	+	+Sc	+Rt	−	+Rt	−	+Rt	+	−	−	−	+Rt	−	−	−	−	+Rt	+	−	Undetermined
(14)	7358c	Pericardial sac	Gram-positive diplococcus	−	−	+	+	+	+	+	− ccr		−	−	−	−	+	−	+	+Rt	+	−	−	+Rt	+Rt	+	+Rt	+	+	+Rt	+Rt	+	−	Diplococcus
(21)	7372a	Lung	"	−	−	+	+	+	+	+	− ccr		−	−	+	−	+	−	+	+	+	−	−	+Rt	+	+	+	+	+	+	+	+	−	"
(25)	7373c	"	"	−	−	+	+	−	+	+	−	r	−	−	+	−	+	−	+	+	+	−	−	+	+	+	+	−	+	+	+	+	−	"
(39)	7463a	"	"	−	−	+	+	−	+	+	−	ccr	−	−	+	−	+	−	+	+	+	−	−	+	+	+	+	+	+	+	+	−	−	"
(40)	7469b	"	"	−	−	+	+	−	+	+	−	"	−	−	+		+	−	+	+	+	−	−	+	+	+	+	−	+	+Rt	+	−	−	"
(19)	7365	Spinal cord	Gram-positive diphtheroid	−	−	+	+	+	+	+	−	ccr	−	−	+	−	+	−	+	−	+	−	−	−	+	+	−	−	−	−	−	−	+	Corynebacterium
(26)	7393a	Round ligament of liver	"	−	−	+	+	+	+	+	−	"	−	−	+	−	+	−	−	−	+	−	−	−	+	+	−	−	−	−	−	−	−	"
(35)	7402	Peritoneal fluid	"	−	−	+	+	+	+	+	−	"	−	−	+	−	+	−	−	−	+	+	−	−	−	−	−	−	−	−	−	−	−	"
(17)	7362c	Spinal cord	"	−	−	+	+	+	+	+	−	cc	+	−	+	−	+	−	+	−	+	−	−	−	−	+	−	−	−	−	−	−	+	"
(16)	7362b	"	"	−	−	+	+	−	−	+	−	−	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	"

Explanation of the figures used in Table (4)

(1) Gelatin liquefaction, indole production, motility test, pellicle formation, and starch hydrolysis:

+ .....positive test
- .. ..negative test

(2) H_2S production, hemolysin production:

+ .....marked
± .....slight
∓ .. ..very slight
- ....negative

(3) Sedimentation, turbidity:

+ .....marked
± .....moderate
∓ .....slight
∓ .....very slight

(4) Potato medium:

B ....growth and brown pigment
M ..... growth and creamery color
Y .....growth and yellowish pigment
- ....no growth

(5) Litmus milk:

a .....acid
b .....basic
c .....coagulation
r ..... reduction
d .....digestion
g .....gas formation

(6) Fermentation tests:

+ .....complete change to yellow color (marked acid)
± .. ..partial change to yellow color (moderate acid)
∓ ....slightly acid
(+) .. ..marked acid with gas production
(±) .....moderate acid with gas production
(∓) .....slightly acid with gas production
Rb .... returned to basic reaction
B .....basic reaction

From Table (4), it will be noted that 41 cultures isolated from various pathological cases were subject to biochemical tests, and, as a result, 7 genera of bacteria were identified and 2 cultures were undetermined. The identified genera (7)

included: Staphylococcus, Di-plococcus, Neisseria, Escherichia, Bacterium, Pasteurella, and Corynebacterium, while the undetermined ones were a gram-positive coccus and a gram-negative coccoid bacillus. In respect to their frequency, Bacterium was isolated from 11 cases (26.8%), Staphylococcus from 8 cases (19.2%), Pasteurella from 6 cases (14.7%), Diplococcus and Corynebacterium from 5 cases each (12.2%), Escherichia from 3 cases (7.3%), Neisseria, gram positive coccus, and gram-negative coccoid bacillus from 1 case each (2.4%).

Besides those cultures just mentioned, 5 cultures of Actinomyces necrophorus and 2 cultures of Clostridium welchii were isolated from various cases and cultivated in deep-brain broth.

c. Pathogenicity tests.

Preparation of inoculum. The micro-organisms were inoculated directly from the stock cultures into nutrient broth and incubated at 37°C. for 24 hours. The broth cultures were transferred to 15 c. c. centrifuge tubes and centrifuged (Sorvall centrifuge) at 4140 r.p.m. for 20 minutes until the supernatant fluid was clear and could be discarded into a beaker by inverting the tubes without disturbing the bacteria collected at the bottom. The bacteria were washed twice with sterile salt solution (0.85% NaCl) and re-suspended to give a turbidity the same as the standard tube-antigen for the Brucella agglutination test.

Inoculation of experimental animals. Both rabbits and mice were used for this test. They were housed in clean wire-made cages and fed with ordinary ration with an ample supply of water. During a preliminary period of at least one week before the test, the animals were checked for health and all the unhealthy ones were discarded.

The mouse was held by the tail and head with the aid of an assistant. The right side of the lower abdomen was swabbed with 70% alcohol and 0.5 c.c. of the suspended inoculum was injected intraperitoneally with a sterile 1.0 cc. hypodermic syringe having a 26 gauge needle. One mouse out of every five was injected with 0.5 c. c. of sterile 0.85% NaCl solution to serve as a control. Two mice were kept in one cage with one of them having the tip of

one ear clipped for identification. The cage was carefully labeled and all the mice were observed three times daily for signs of illness or death.

The rabbit to be injected was confined in a wooden box with the head free. One of the ears was moistened with 70 per cent alcohol and 2 c.c. of the suspended bacteria were injected intravenously into the marginal ear vein. Two rabbits were put together in one cage with the hair on the back of one of them clipped to serve for identification. The cage was labeled as usual and the observations made under the same condition as for the mice.

Technic of culturing tissues of experimental animals. If the experimental rabbits survived the pathogenicity tests, they were killed five days after inoculation by the intravenous injection of air (30 c.c. syringe) through the marginal ear vein. Each rabbit was then autopsied and the tissues cultured using the same technic as employed for the lambs. However, the rabbits were not skinned.

If the experimental mice showed no symptoms and survived the pathogenicity tests, they were killed five days after inoculation by grasping the tail of each mouse with a pair of long forceps and hitting the head on a cement floor. As soon as they were killed, each was stretched on a piece of wooden board with the aid of four thumb tacks. The mouse was then skinned, opened, and cultures made from the liver using the same method employed for culturing the tissues of pathological lambs. However, in culturing the heart-blood

and spleen of mice, the following procedures were found to be more applicable. The ventral surface of the heart was seared as usual. A sterile pair of forceps was used to grasp the sides of the heart and by squeezing the forceps a drop of blood would usually ooze from a hole made in the middle of the seared spot with the aid of a sterile needle having a small and pointed loop at the tip. As soon as the blood was oozing out, the needle was introduced into the heart in order to get a good sized drop of blood with which to smear over 1/3 of a blood-agar plate.

The spleen was cultured by searing an area near either end (preferably the ventral end). A sterile needle (the same needle used for culturing heart-blood) was introduced through the seared area into the spleen along its long axis. Since the subterminal part of the spleen was seared and consequently weakened, it was easy to introduce the sterile needle through the weakened spot into the spleen pulp in order to secure sufficient material for plating. This procedure was facilitated by grasping the extreme end of the spleen with a pair of forceps.

Identification of bacteria isolated from experiment animals.

The bacteria isolated from rabbits and mice were stained and the biochemical tests were made with the following media: Glucose, lactose, sucrose, glycerol, NO_3 broth, indole medium, starch-agar plate, litmus milk, and gelatin. The reactions of the micro-organisms in these media and the

characteristics upon staining were compared to those of the original cultures in order to check their identity.

Results of pathogenicity test. The results of the test are arranged in Table (5).

Table (5). Results of Pathogenicity Tests.

Culture No.	Results of Test Mouse	Rabbit	Bacteria Recovered from	Remarks
7308	-	-		*
7334	x		H.L.S.	Mouse died in 15 hr.
7335	x	x	H.S.	" " " 21 hr., rabbit in 24
7336	x	-	L.S.	" very sick, killed in 40 hr.
7337	-	-	S.(contaminated)*	
7338	x		H.L.S.	Mouse died in 30 hr.
		x	H.	Rabbit died in 28 hr.
7344	x	-	H.S.	Mouse died in 38 hr.
7350	x	-	H.L.S.	Mouse died in 60 hr.
7351	x	-	H.L.S.	Mouse died in 24 hr.
7353	x	-	H.	*
7358a	x	-	L.	*
7358b	-	-		*
7358c	-	-		*
7362a	-	-		*
7362b	-			*
7362c	-	-		*
7365	x		H.L.S.	*
7371a	x		H.L.S.	Died in 38 hr.
7371b	x	x	H.	*
			S	*
7372a	-			*
7372b	x		H.L.S.	*
7373a	-			*
7373b	x		S.	*
7373c	-			*
7393a	x		L.S.	*
7393b	x	-	H.L.S.	Mouse died in 14 hr.
7393c	x	-	H.L.S.	Mouse died in 15 hr.
7394a	x	-	H.L.S.	Mouse died in 18 hrs.
7394b	-			*
7400	-			*
7401a	x		H.L.S.	Died in 22 hrs.
7401b	-			*
7401c	-			*
7406	x	-	H.L.P.S.	Mouse died in 4 days.
7410	-		L.S.(contaminated)	*
7415	-			*
7443	-			*
7460a	-	-		*
7460b	-	-		*
7460c	-	-		*
7474	-		L.S. (contaminated)	*
Total: 41	20x	3x	25(including contaminated ones)	13(mice became very sick or died within 4 days after inocultaion.

Note: Explanation of table.
x.....positive test
-.....negative test
*.....Killed in 5 days.
H.....Heart blood
L. .....Liver
S. .....Spleen
P. ..Peritoneal fluid

The results of the experiment in Table (5) showed that 20 (48.78%) out of the 41 original cultures were recovered from the experimental mice, of which 13 (31.7%) were definitely virulent. These 13 virulent cultures either caused the death of mice or made them seriously ill within four days after inoculation, while the remaining 7 cultures of the 20 positive tests did not induce any noticeable symptoms up to five days after inoculation at which time the mice were killed for examination. As for the rabbits, only 3 positive tests were obtained from 22 inoculated animals which indicates that rabbits were quite resistant to the cultures isolated from lambs and were not suitable for such work. Incidentally, rabbits were first used as the test animal in this experiment. Later on, when they were found to be rather indifferent to the cultures, mice were used instead.

C. Pathological Methods.

(a) Technic used for autopsy.

The lambs were either bled to death or brought dead to the laboratory for autopsy. In either case, they were immediately skinned and carefully examined for gross lesions in every part of the body, including the brain, cranial nerves, and sometimes the spinal cord. In order to expose the brain, a frontal cut was made through the nasal cavities extending along an imaginary line just below the orbits and above the base of the ears until it reached the occipital region. Then, by a gentle tipping and with the help of the blunt tip of a pair of scissors, a greater part of the brain (the entire cerebrum) was slipped out of the upper half of the skull and exposed, leaving the cerebellum and medulla oblongata in the lower half. (Fig. 1, 2)

(b) Technics for preparing pathological tissues.

The tissues were fixed in Zenker's fluid and sometime in 10% formol saline. They were then washed, dehydrated, embedded and sectioned by ordinary histological technics, and finally stained either with hematoxylin-eosin, Gram-Weigert, Mallory's anilin-blue, or by all three methods.[56]

2. Presentation of cases

Case 7356 (Grade, male)

This was a typical case of white-muscle disease or so-called "stiff-lamb" disease.

History. This lamb was owned by Mr. Savage of Albion, Michigan, and was delivered to the pathology barn on 4/3/44. The lamb was showing evidence of white-muscle disease and was unable to rise. The animal made no progress following the time of delivery and on the morning of April 4th would not nurse. It was then sacrificed and studied. The lamb was 5 weeks old.

Etiology. Undetermined. No bacteria were isolated from this case.

Blood studies.

(a) Blood picture

Erythrocytes	12,250,000 per c.mm.
Leucocytes	11,600 " "
Hemoglobin	9.8 g./100 cc.
Differential count (%)	
Lymphocytes	7
Neutrophils	93
segmenters	(88)
bands	(4)
Monocytes	1
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm/100 cc.)

Glucose	196.00
Uric acid	1.78
Creatinine	1.65
Chloride	560.00
N.P.N.	162.00
Urea-N	108.00

From the results of the blood studies, it is interesting to note that the chemical analysis of blood as well as the blood picture of this case resembled quite closely that of Case No. 7358. All blood elements which had been studied were decidedly increased, and yet on the other hand the number of lymphocytes was very much decreased. The neutrophilia was also very pronounced, but it showed mostly mature forms. In general, the peculiar feature of increased value in blood elements seemed to be quite unique for white-muscle disease. The erythrocytes were more or less normal, although a few poikilocytes were observed.

Gross lesions.

Skeletal muscles. The skeletal muscularis on both sides of the body were involved, and the lesions appeared as superficial whitish patches or streaks.

Liver. The liver showed some fatty degeneration.

Other organs. Apparently normal.

Microscopic lesions.

Liver. The liver was very much congested and showed cloudy swelling and fatty degeneration. Most blood vessels as well as the sinusoids were distended with blood. The fatty degeneration was rather extensive and diffuse, beginning at the central part of each lobule and extending gradually in a diminishing gradation toward the periphery. The affected cells became granular and vacuolated due to the presence of fat globules in the cytoplasm, and the nuclei were either pushed to one side or were pyknotic.

Kidneys. The kidneys were rather congested and showed evidence of cloudy swelling, fatty degeneration and hemorrhage. The convoluted tubules were generally somewhat dilated, and the lumina together with the subcapsular spaces were filled with varying amounts of debris and hyaline casts. The epithelium of some convoluted tubules were affected by marked fatty degeneration, the condition being especially pronounced in the terminal portion of the proximal convoluted tubules. Occasionally, some interlobular petachiae were observed and some disintegrated erythrocytes were present in the collecting tubules.

Skeletal muscle. In this case, quite a number of skeletal muscles were more or less involved. Seven blocks of muscle were taken from various areas over the body for microscopic study. The general characteristics of the lesion were more or less the same, namely, myodegeneration followed by either myo-regeneration or fibrosis. (See also Case No. 7358). The degenerative changes of the muscle were characterized by a diffuse and fragmentary necrosis with some fibroblastic activity. The affected muscle fibers generally became swollen and wavy, followed by contraction or shrinkage and consequently broke into small pieces. As soon as the muscle fibers broke into pieces, some of the more severely affected ones underwent hyaline or Zenker's degeneration[13]. (Fig. 3, 4) These dying muscle-fibers lost their characteristic striations and

nuclei and became more or less coagulated. They were stained either pinkish or dark purple with hematoxylin-eosin and some of them appeared to contain a certain kind of deposit. (Fig. 4, 5, 6). In some instances, however, the destructive process was not so severe and only the sarcoplasm was hyalinated whereas the sarcolemma with the nuclei was not affected.

Following the degenerative changes, a regenerative process was going on. In this regenerative process, the most striking feature was proliferation of the fibroblastic cells and the nuclei of unaffected muscle fibers or sarcolemmae. Occasionally, a few polymorphonuclear leucocytes and plasma cells were observed. The fibroblastic proliferation resulting in fibrosis was to strengthen the weakened areas. The newly-formed nuclei from the normal muscle fibers or surviving sarcolemmae migrated toward the injured ends and each carried a small amount of sarcoplasm and transformed either gradually into muscle giant cells or directly into regenerating muscle fibers. The nuclear proliferation and cell division were so extensive that in certain spots a great mass of such newly-formed cells were present, (Fig. 4, 9, 11, 12), which have been referred to as "Muskelzellenschlauche" of Waldeyer[27]. The formation of such structures has been the subject of much discussion since early history of medical science.[13, 26, 27, 28, 37] In fact, some of these newly formed cells were regenerating muscle fibers while others

were muscle giant-cells. The function of the muscle giant cells was to remove the dead muscle tissue and that of the regenerating muscle-fibers was to fill the gaps left by the dead tissues. In quite a number of instances, the writer was able to demonstrate a muscle giant cell engulfing a piece of dead muscle of considerable size (Fig. 7, 7a, 8, 9), while in another he frequently encountered several such giant-cells each containing one or several food vacuoles filled with small pieces of tissue debris (Fig. 12, 10). Therefore, in such cases, the function of muscle giant-cells was beautifully illustrated. These giant-cells were so active that they had no definite shape. They somewhat resembled amebae and acted almost like them. The cells usually thrust out parts of their bodies in order to engulf the dead muscle tissue (Fig. 7, 7a, 8, 9). As soon as the dead tissue was surrounded, digestion began. Gradually the pieces of dead muscle fiber diminished in size until they were completely removed, leaving food vacuoles behind (Fig. 11). After the dead tissue was completely digested and absorbed, the food vacuoles disappeared and the giant-cells were then ready to repeat the amazing process (Fig. 12).

These muscle giant-cells were formed either by fusion of the newly-formed regenerating cells (Fig. 13, 14) or by mitosis without cytoplasmic division (Fig. 15). The formation of giant-cells has been also a problem of great

controversy in the medical field and quite a number of theories[37] have been formulated. Some workers thought that they might be formed by cell fusion, others maintained that they were derived by mitosis without cell division, and still others insisted that they came into existence by both fusion and mitosis. Since there was no actual concrete evidence to support the various theories, much confusion existed. From now on, it is hoped that the definite evidence of the formation of muscle-giant cells in the case of white-muscle disease of the new-born lambs may serve as a sound basis for further investigations as to the formation of other kinds of giant cells and the confusing condition, which is still existing in this particular field, may be unraveled.

The size of the muscle giant-cells was variable; one of the largest measuring about 0.05 mm. X 0.07 mm. The nuclei were either spherical or oval in shape, but all were provided with a heavy distinct nuclear membrane, fine or nearly clear nucleoplasm, and from one to three well defined nucleoli. The number of nuclei varied and from two to twenty-five were observed (Fig. 7, 7a, 9, 12). Their arrangement in the cell was irregular and indefinite.

In a section stained with aniline-blue and eosin, it was observed that the necrotic muscle-fibers were stained orange to purplish-blue or blue, the normal muscle-fibers red, the fibroblastic tissue blue with small spindle-shaped

yellowish nuclei, and the regenerating muscle-fibers and muscle giant-cells grey with clear colorless nuclei containing yellowish nucleoli (Fig. 6, 7, 7a, 8). The regenerating muscle-fibers generally possessed faint to distinct fibrils and some of them also contained cross striations (Fig. 6, 8, 11), while the muscle giant-cells were more or less homogeneous in nature. By means of aniline-blue and eosin stain, the morphology of nuclei and the staining affinity of both giant-cells and the regenerating muscle-fibers were exactly the same, indicating clearly that they were all derived from the same parent tissue, i.e., muscle, and were not of extra-muscular origin.

Generally speaking, if myodegeneration were not too severe and myo-regeneration followed a complete or total restoration of the affected muscle is possible. In this case, the myo-regeneration is carried out by the process of nuclear proliferation from unaffected muscle-fibers or sarcolemmae so as to form muscle giant-cells for the removal of devitalized tissue and regenerating muscle-fibers for the filling in the gaps left by the dead tissue. The fibroblastic activity is reduced to a minimum. In order to substantiate these facts, a lamb recovered from "white muscle" disease was slaughtered early last fall (1944) and no lesions were found whatsoever. The lamb chops were boiled or fried and tasted just as good and tender as those from normal lambs. Forbus[26, 27] in his study on degeneration and regeneration of the rectus abdominus in human

pneumonia also found the "total restoration of the affected muscle". If the total restoration of the affected muscle is effected, a question naturally arises: "How about the fate of muscle giant-cells after they have performed their duty in removing the dead tissue?" Regarding this, the writer has not been able to get any evidence for making a definite statement, but he is inclined to feel that after the muscle giant-cells have completed their task, they may disintegrate and be removed or they may be gradually transformed into regenerating muscle-fibers. Probably, the latter process is the actual case.

On the other hand, if myo-degeneration were very severe and destructive and thus less chance for myo-regeneration were provided, the fibroblastic activity would be more pronounced and the gaps left by the dead tissue would be replaced by fibrous or sometimes by adipose tissues and the coagulated dead muscle tissue would remain for some time. Quite a number of such instances were observed, and such muscle tissue would be atrophied and the area was considered to be more serious than ordinary.

Discussion and summary.

1. This was a typical case of "white-muscle" disease in which the most striking features were hepatic disorder and myo-degeneration followed by myo-regeneration.
2. A pronounced leucocytosis (neutrophilia) was observed, the cause of which was not definitely known.

3. The dehydrated condition of the animal, the marked hepatic disorders, and the extensive destruction of skeletal muscles would naturally induce the presence of high value of all six blood constituents studied. This particular increased value in blood constituents seemed to be rather unique in the case of "white-muscle" disease and it was suggested that it might serve as a criterion for diagnosis.
4. The existence of muscle giant-cells was well illustrated in this case. This finding is considered to be new to this particular disease and even new to medical science. For a more detailed discussion of this matter, reference is made to the general discussion of this article.
5. It is suggested that "white-muscle" disease is a better and more illustrative term for this diseased condition than the so-called "stiff-lamb" disease, because the latter may be confused with quite a number of conditions such as arthritis, laminitis, paralysis, docking infection, etc., in which cases animals would also show a stiff gait.

Case 7358 (Hampshire, male)

This was a case of white muscle disease complicated by abscess-formation and arthritis, the latter probably the result of a previous bacteremia.

History. The animal showed unilateral paralysis of the right and rear legs on 3/31/44. It was unable to stand and nurse, but by holding the animal up it would suck vigorously. Six days later when the animal was about 15 days old, it was bled and killed.

Etiology. Three different micro-organisms were isolated, namely, one culture of Staphylococcus and an undetermined gram-positive coccus from the right stifle joint and one Diplococcus from the pericardial sac (Table 4: culture No. 7358 a,b,c.).

Blood studies.

(a) Blood picture

Erythrocytes	9,370,000	per c.mm.
Leucocytes	13,250	" "
Hemoglobin	6.3	g./100 cc.
Differential count (%)		
Lymphocytes	2	
Neutrophils	98	
segmenters	(35)	
myelocytes	(7)	
juveniles	(9)	
bands	(47)	
Monocytes	0	
Eosinophils	0	
Basophils	0	

(b) Blood chemistry (mgm./100 cc.)

Glucose	115.00
Uric acid	4.16
Creatinine	3.76
Chloride	550.00
N.P.N.	118.50
Urea-N	98.00

The blood picture of this lamb showed a low hemoglobin and a leucocytosis. A state of marked neutrophilia existed together with an excessive number of immature forms and a greatly reduced number of lymphocytes. The erythrocytes exhibited characteristics of microcytic anemia with marked poikilocytosis.

Similarly, the blood chemistry of this case was abnormal. All six components included in this study were greatly increased, presenting a rather unique and interesting feature for a case of white muscle disease. (See case #7356) The glucose was about twice as high as normal, uric acid 2.5 times, creatinine 3 times, N.P.N. 3.5 times, urea-N 7 times, and the chloride somewhat higher.

Gross lesions.

Liver. Possibly showed some fatty changes.

Spleen. Enlarged.

Kidneys. Both showed a yellowish zone in the extra-medullary area.

Joints. The carpal joint of the right front leg and the stifle joint of the right rear leg showed accumulations of greenish colored semi-caseated exudate.

Skeletal muscle. Showed some pyogenic foci and some whitish streaks which were suspected of being white-muscle disease.

Lymph nodes. Prescapular, popliteal, and deep-inguinal lymph nodes were greatly enlarged and edematous.

Other organs. Apparently normal.

Microscopic lesions.

Liver. The liver was rather congested and showed evidence of moderate cloudy swelling.

Kidneys. The kidneys were congested and some hyaline casts were found at various levels from the glomeruli down the convoluted tubules into the collecting tubules. The casts in the larger collecting tubules were more abundant and pronounced so that they might have appeared as a yellowish zone during macroscopic examination. Here and there, some perivascular lymphocytic infiltrations were found throughout the cortex, and a marked cloudy swelling was observed in the epithelium of the convoluted tubules.

Joint capsule of carpus. The joint capsule of the right carpus was inflamed and showed lesions of recent abscess formation with marked polymorphonuclear leucocytic infiltration.

Skeletal muscle. The skeletal muscle presented a very interesting but difficult to explain series of lesions. Some sections of the muscle appeared normal, while others showed two different lesions. These lesions were (1) recently formed abscesses and (2) myodegeneration together with myoregeneration. To explain this condition, it should be remembered that on autopsy of this lamb, both pyogenic foci and white-muscle disease were revealed. Consequently, two different kinds of lesions would be expected.

The abscesses (Fig. 12a) were localized in the muscle and each was characterized by varying amounts of muscular

necrosis, marked polymorphonuclear leucocytic infiltration and absence of limiting walls. The average size of these abscesses was about 0.6 mm. in diameter. The muscle fibers adjacent to each abscess were generally broken up into small pieces and separated from the remaining normal fibers. They had lost their striations, stained acidophilic, and the nuclei were undergoing pyknosis or karyolysis.

The myodegeneration was (Fig. 7, 12) quite different from the abscessation just described and a detailed account is given for case #7356 in which similar lesions were found.

Other tissues. Other tissues such as brain, heart, spleen, intestine, lymph nodes, etc., were also included in this study, but they were all found to be normal.

Discussion and summary.

1. This lamb suffered from white muscle disease and complicated by abscess-formation and arthritis, the latter two conditions undoubtedly the result of a previous bacteremia.
2. The animal was somewhat anemic, probably the result of being unable to stand and nurse for several days before it was killed. It was due to this anemic condition that the blood chloride was considerably increased.
3. The results obtained from blood studies revealed a marked state of neutrophilia and high values in various blood components. These increased values in blood components resembled quite closely those found in case #7356 (a case of "white-muscle" disease), indicating

that these findings might be of significance in diagnosing such a disease, although this case (case 7358) was complicated by bacteremia with abscess-formation, and arthritis which would also induce the increase in polymorphonuclear leucocytes and uric acid.

Case 7337 (Rambouillet, male)

This was another case of staphylococcic infection but it was not so severe as that of case 7338.

History. This lamb was from a parasite-free experimental group. It had been getting weak for a few days and died during the night of 3/26/44. The lamb was about 10 days old.

Etiology. A culture of Staphylococcus was isolated from the liver. (Table 4: culture No. 7337)

Gross lesions. The spleen showed some petechial hemorrhages, and the mesenteric blood-vessels seemed to be somewhat congested. The left upper eyelid showed marked conjunctivitis, and the nasal turbinates were quite congested. No other visible lesions were observed.

Microscopic lesions.

Liver. Congestion, cloudy swelling, and tiny necrotic foci with cell infiltration were found in the liver. The congestion was generalized and very marked. The sinusoids were very much dilated and engorged with blood which pushed the adjacent hepatic-cell chords far apart. In some portions of the liver, several such dilated sinusoids were united, forming a small, rounded area filled with blood. These areas were located in the intermediate zone of the lobule. The hepatic cells were undergoing various degrees of cloudy swelling and necrosis. The necrotic foci (somewhat similar to those found in case 7338) varied in size from a

tiny area about 0.05 mm. in diameter to about 0.25 mm. in diameter and they were usually located in the parenchyma of the lobules. Each of the tiny necrotic foci was characterized by the death of one or several hepatic cells among which was a group of activated Kupffer cells. (Fig. 16) Occasionally, bacterial emboli were found in the midst of such necrotic spots. In the larger necrotic foci, more liver cells were involved and actual phagocytosis of the dead cells by Kupffer-cells was observed together with some polymorphonuclear leucocytic infiltration. The cytoplasm of some of these dead cells was acidophilic and stained light red in color with hematoxylin and eosin, while that of others stained a deep purple color.

Spleen. The spleen was greatly damaged. In the central portion of the spleen, quite extensive hemorrhages were observed in which most of the lymphoid tissue had been replaced by red blood cells and only few normal Malpighian corpuscles were observed.

Lungs. The lungs were rather congested, otherwise they were not affected.

Kidneys. The kidneys showed evidences of marked congestion, involving mostly the interstitial tissues. Some hemorrhage was present in the interstitial tissue. The epithelial lining of most portions of the convoluted tubules had undergone varying degrees of cloudy swelling so that all these cells did not appear normal.

Other tissues. No abnormalities were found in other tissues.

Case 7338 (Shropshire, female)

This was a case of staphylococcic infection, resulting in the formation of small necrotic foci, abscesses, and hemorrhages in many tissues of the body.

History. This lamb was born on 3/24/44 and was brought to the pathology barn three days later. The lamb was quite stiff but suckled as usual and appeared to be bright. On the morning of 3/28/44, the lamb refused to nurse and at 11:30 it was killed.

Etiology. A hemolytic Staphylococcus was isolated from the nodule-like abscesses of the skeletal muscle and its physiological characteristics are referred to in Table 4 (culture No. 7338).

Blood studies.

(a) Blood picture

Erythrocytes	10,400,000 per c.mm.
Leucocytes	12,150 " "
Hemoglobin	11.4 g./100 cc.
Differential count (%)	
Lymphocytes	12
Neutrophils	88
segmenters	(6)
bands	(79)
juveniles	(3)
Monocytes	0
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm./100 cc.)

Glucose	41.66
Uric acid	2.00
Creatinine	2.10
Chloride	400.00
N.P.N.	106.00
Urea-N.	55.30

As compared with the normal standard (Table 1), the blood picture of this case showed moderate leucocytosis with an abnormal increase in the number of immature cells of the neutrophilic series and considerable decrease in lymphocytic cells. (Fig. 17) The values of erythrocytes and hemoglobin were apparently normal. Regarding blood chemistry, the glucose and chloride were somewhat lower than normal, while the uric acid, creatinine, N.P.N., and urea-N were higher than normal.

Gross lesions.

Skeletal muscle. Showed some small nodules that varied in size from rice grains down to circumscribed areas 1 to 2 mm. in diameter; such nodules were also found in the diaphragm. (Fig. 18)

Lungs. Several small abscesses on diaphragmatic lobes which measured from 2 to 12 mm. in diameter. (Fig. 18)

Heart. Pericardium adherent to heart with fibrinous exudate; myocardium studded with numerous abscesses varying from 2 to 10 mm. in diameter; one abscess almost filling the lower part of the left ventricle. (Fig. 18)

Kidneys. Showed many subcapsular abscesses varying from 1 to 3 mm. in diameter. On cross section, right kidney showed three abscesses at the junction of the cortex and medulla. (Fig. 18)

Other organs. Not visibly affected.

Microscopic lesions.

Liver. The lesions in the liver consisted of moderate congestion, cloudy swelling, moderate to marked focal polymorphonuclear leucocytic infiltration, tiny necrotic foci and hemorrhage. The necrotic foci seemed to be of two different kinds, i.e., an older lesion and a recent one (Fig. 19, 20), possibly due to the showering of staphylococci, whereas the moderate congestion of the sinusoids and the cloudy swelling of hepatic cells were more or less the same throughout the organ. Occasionally, small areas of hemorrhage were observed. (Fig. 20)

Intestine. The intestine showed lesions of enteritis, involving the mucosa and submucosa. The superficial one-fifth of the mucosa was necrotic and part of it was desquamated, while the remaining portion was not much damaged but infiltrated with an excessive number of polymorphonuclear leucocytes. The submucosa was rather congested and evidence of lymphadenitis of the Peyer's patches were noticed. The muscular coat and serosa appeared to be normal.

Lungs. The lungs were rather congested, as most of the arteries as well as the capillaries were filled with blood. In some of the blood vessels, emboli were observed. There were some small areas of hemorrhage in the peri-bronchial region, resulting probably from the rupture of the congested capillaries in the alveolar walls.

Kidneys. The kidneys showed cloudy swelling, congestion, some glomerular hemorrhage, focal necrosis, and abscess formation. The cloudy swelling was rather generalized. The capillaries in the interstitial tissue were very much congested and those in the glomerular tufts were also somewhat distended. In some cases, glomerular hemorrhage occurred. Red blood corpuscles were observed in some of the capsular spaces as well as in some of the convoluted tubules.

The necrotic areas were localized, and they beautifully illustrated the staphylococcic infection. One of the necrotic foci was observed to originate from an infected glomerulus. In this glomerulus, the micro-organisms (Staphylococci) were found in five masses lodging within the glomerular tuft as bacterial emboli. Due to the presence of Staphylococci and their toxin in the particular spot, a marked cell infiltration was present. These mobilized cells consisted mostly of polymorphonuclear leucocytes, some reticulo-endothelial cells, and a few fibroblasts. The bacterial toxin seemed to be so strong that it not only killed all the cells in the glomerulus but also damaged the surrounding convoluted tubules and interstitial tissue, involving an area about 0.55 mm. by 0.45 mm. in diameter. The affected cells showed acidophilic staining with pyknotic nuclei or karyolysis. (Fig. 21) Another necrotic focus was found in the interstitial tissue

of the cortex but the glomeruli were not involved and no bacterial forms could be observed. (Fig. 22)

According to their location, two kinds of abscesses were found in the kidneys, namely, the subcapsular abscesses and cortico-medullary abscesses. The subcapsular abscesses (Fig. 23) were medium in size and irregular in shape, involving both cortex and the capsule. They originated in the outer part of the cortex and as they grew larger, they often became associated with the capsule. These abscesses could be recognized by the marked cell infiltration (mostly poly-morphonuclear leucocytes) and liquefaction of necrotic cells with their outer surface being limited by the thickened and partly disintegrated capsule of the kidney. The tissues surrounding these abscesses were also damaged as evidenced by the deformed structure, acidophilic cytoplasm and pyknotic nuclei. The cortico-medullary abscesses (Fig. 24) were large and each of them could be distinguished from the others by the irregular shape, marked cell infiltration, and purulent exudate which was composed of polymorphonuclear leucocytes, fibrin and clumps of staphylococci. The tissue adjacent to these abscesses was also affected as indicated by marked polymorphonuclear infiltration and proliferation of fibroblasts. Clumps of Staphylococci were found in most of the lesions and they were beautifully demonstrated by means of Gram-Weigert's stain. (Fig. 25)

Heart. In the heart, the staphylococcic infection resulted in the formation of focal necrotic areas, marked cell infiltration, fibroblastic proliferation, abscesses, and some hemorrhage under the endocardium. The necrotic areas and abscesses were mostly found in the myocardium but a few were also observed in the epicardium. These lesions could be divided into two kinds, old and recent, which can probably be accounted for by the showering of staphylococci as noticed in the liver. The lesions were more wide spread and involved larger and more irregular areas than those found in the cortex of kidneys. The older lesions were characterized by fibroblastic proliferation or fibrosis whereas the recent lesions (Fig. 26) were distinguished by more polymorphonuclear infiltration. In either case, no definite limiting walls could be observed. The tissues adjacent to these lesions showed loss of striations with the sarcoplasm staining acidophilic and the nuclei were undergoing pyknosis, karyorrhexis, or karyolysis.

In some parts of the epicardium, the adipose tissue was greatly thickened and some abscesses were present. Due to the rupture of certain epicardial abscesses, the pericardium became involved and consequently adhered to the epicardium at various points.

In one area on the endocardium, there was hemorrhage, and the blood was escaping into the heart chamber.

Skeletal and diaphragmatic muscles. In the skeletal muscles and diaphragm, varying degrees of necrosis and two kinds of abscesses were observed. The muscular necrosis seemed to be initiated by a preliminary shrinking of the muscle fibers due to staphylococcal toxin followed in sequence by breaking up of the fibers, hyalination of the sarcoplasm with loss of striations, and finally nuclear pyknosis and karyolysis. The damaged cells had an acidophilic affinity so that the sarcoplasm was stained either bright pink or pale yellow in color. The diaphragmatic muscle seemed to be more sensitive to the staphylococcal toxin than the skeletal one, because the toxic effect on the diaphragmatic muscle tended to be diffuse as well as focal.

Both old and recently formed abscesses were present in the skeletal muscles and diaphragm, and the respective characteristics resembled quite closely those found in the heart. (Fig. 27)

Other tissues. Other tissues showed nothing abnormal.

Discussion and summary.

1. This was a case of staphylococcal infection together with liberation of exotoxin and the subsequent formation of both recently formed and older lesions in various tissues of the body, especially in the liver, heart, skeletal muscles and diaphragm.

2. Due to the presence of bacterial infection, a state of leukocytosis (neutrophilia) was found.
3. The high values of N.P.N., urea-N, creatinine, and uric acid in the blood apparently resulted from the destruction of various body tissues by necrotic process, abscess formation and renal suppression by acute nephritis, while the high value of blood glucose might also be due to the nephritis alone. The blood cloride was somewhat lower than the normal, and that was accounted for by the existence of enteritis.

Case 7343 (Hampshire, male)

This was a case of navel infection due to Actinomyces necrophorus which was complicated by bronchopneumonia and a mild catarrhal enteritis.

History. This lamb was born at 9:00 P.M. 3/14/44 and three days later an umbilical infection was noticed. It was brought to the pathology barn 3/17/44 and immediately treated with pneumonia serum (15 cc. intraperitoneally) and sulfathiazole(1 cc. infected subcutaneously in vicinity of navel). On the same day, a milk feeding program (containing sulfathiazole in excess) at three hour intervals was started. However, on 3/19/44, the lamb began to nurse an Oxford ewe that was in the same pen, so that the afternoon feedings were cut off if the lamb refused to drink. On 3/21/44, the condition of the lamb seemed to be improving and the navel lesion was almost healed. On 3/26/44, the lamb was somewhat off feed and the serum therapy was repeated. The following two days the lamb declined in vigor and was killed on 3/29/44. At that time, the navel was quite well healed except for a small scab.

Etiology. A pleomorphic beaded Gram-negative filamentous micro-organism, morphologically indistinguishable from Actinomyces necrophorus, was isolated from the lungs and cultured in deep brain broth. The same organism was also found in the smear made from liver abscesses.

Blood studies.

(a) Blood picture

Erythrocytes	5,810,000 per c.mm.
Leucocytes	14,900 " "
Hemoglobin	6.3 g./100 cc.
Differential count (%)	
Lymphocytes	14
Neutrophils	85
segmenters	(3)
bands	(46)
juveniles	(7)
myelocytes	(29)
Monocytes	1
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm./100 cc.)

Glucose	15.00
Uric acid	0.12
Creatinine	0.20
Chloride	480.00
N.P.N.	25.00
Urea-N	20.50

The results of blood studies in this case revealed that the number of erythrocytes was reduced to about 1/2 of the normal standard (Table 1), and hemoglobin by 1/3, while the leucocytes increased nearly twice with many immature neutrophilic cells and a reduced number of lymphocytes. Most of the erythrocytes were hypochromic and poikilocytic and some of them were nucleated. As to blood chemistry, the glucose was about 1/4 normal (Table 2), uric acid 1/13 normal, and creatinine 1/5 normal, whereas the chloride and urea-N were a little higher.

Gross lesions.

Lungs. The lungs showed lesions of bronchopneumonia and were

studded with large abscesses which varied in size from 2 to 12 mm. in diameter. These abscesses were somewhat circumscribed and adherent to the parietal pleura. (Fig. 28)

Ribs. The ribs on the right side had been broken at one time and bony calluses were present. There were also circumscribed abscesses on the parietal pleura close to the bony calluses. (Fig. 28)

Liver. Liver was studded with large prominent circumscribed abscesses which contained a greenish-yellow pus. (Fig. 28)

Spleen. Spleen was enlarged but showed no visible abnormalities.

Other organs. Other organs such as heart, kidneys, etc., were apparently normal.

Microscopic lesions.

Liver. The liver was severely damaged, resulting in the formation of very interesting abscesses and other tissue reactions closely associated with them. The abscesses might have been started as a group of small abscesses and coalesced to form "multiple abscesses" in one area or as a single solitary abscess in another. As the solitary abscess grew larger, they assumed a more or less spherical shape, while the multiple ones were irregular due to the crowding effect or the pressure exerted by individual abscesses of the group. In one instance, it was observed that the multiple abscesses joined together and coalesced

to form a large single one. These abscesses (Fig. 29) were variable in size, ranging from about 2 mm. to 8 mm. in diameter. Each of these abscesses was surrounded by a rather thick fibrous wall (about 0.3 mm.), a layer of fibroblastic cells infiltrated with some lymphocytes, numerous macrophages, a few inactive or dying polymorphonuclear leucocytes, a layer of deeply-stained cell debris, and finally the cheesy central core. The cheesy central core of the abscesses stained acidophilic and was devoid of any recognizable structure. These are the chief characteristics of a necrophorus abscess. Due to the presence of such abscesses, the surrounding liver tissue was affected. The sinusoids were much congested and the endothelial cells rather active, while the hepatic cells themselves were undergoing various phases of cloudy swelling and fatty degeneration together with either normal or pyknotic nuclei.

Lungs. The lungs were affected by multiple and at the same time spreading abscesses, presenting a more malignant process than that found in the liver. In fact, a case of suppurative bronchopneumonia actually existed. Moreover, it was complicated by an acute bronchopneumonia. The abscesses were not well limited and this could be interpreted to that the lesions were of more recent existence than those found in the liver. It is well to point out that the disease was initiated by navel infection which

progressed inward through the umbilical vein to the liver, resulting in the formation of abscesses in that organ first. From the liver, the organisms were carried to heart and finally to lungs through the pulmonary circulation. The abscesses were similar to those found in the liver, but the boundaries were more irregular. (Fig. 30) Due to the rupture of some of the abscesses located near the surface of the lungs and subsequent fibroblastic activity, various parts of the lungs became adherent to the pleura. The adhesion was so extensive that some parts of the thoracic muscle were also involved and damaged as shown by their acidophilic and hyalinated sarcoplasm and pyknotic nuclei.

Spleen. The spleen appeared to be in a state of hyperactivity. The sinuses of the red pulp were engorged with lymphocytes so that the Malphigian corpuscles appeared as if they were abnormal.

Intestine. The intestine showed evidences of a mild catarrhal enteritis, involving only the superficial part of the mucosa where some epithelial cells were desquamated and some polymorphonuclear leucocytic infiltration was noticed. Occasionally, a group of necrotic cells in the crypts near the muscularis mucosae was observed. Probably, this is one reason why the lamb refused to drink for the last few days during its life.

Kidneys. The kidneys showed a slight degree of cloudy swelling, otherwise they were normal.

Other tissues. Other tissues such as heart, skeletal muscles, etc., were also included in the study of this case, but no abnormalities were found.

Discussion and summary.

1. This lamb was not vigorous for the last few days of its life and apparently did not get enough food due to the presence of enteritis, bronchopneumonia, and hepatitis. As a result, the animal was rather anemic and the values of blood glucose and uric acid were thus also greatly reduced. The blood value of N.P.N., urea-N, and creatinine were also quite low, and that was possibly due to acute hepatic insufficiency resulting from the extensive destruction of liver tissue by abscess formation.
2. The leukocytosis (neutrophilia) was the usual consequence of a bacterial infection.
3. Both solitary and multiple abscesses were found in the liver as well as in the lungs and the characteristic features of such typical Actinomyces necrophorus abscesses were noticed.

Case 7344 (Oxford, male)

This was a case of navel infection complicated by bronchopneumonia and encephalomalacia.

History. This lamb was born on 3/19/44 and was noticed to be dull and showed marked in-coordination when about 11 days old. Both ewe and lamb were brought to the pathology barn, and the lamb was given 25 cc. pneumonia serum intraperitoneally. The next morning, the lamb did not nurse its mother and showed increasing in-coordination and dullness. It was killed the same morning at 10:30.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs and its physiological characteristics are listed in Table (4): culture No. 7344.

Blood studies.

(a) Blood picture

Erythrocytes	8,990,000 per c.mm.
Leukocytes	800 " "
Hemoglobin	11.4 g./100 cc.
Differential count (%)	
Lymphocytes	74
Neutrophils	28
segmenters	(8)
myelocytes	(6)
juveniles	(4)
bands	(8)
Monocytes	0
Eosinophils	0
Basophils	0

(b) Blood chemistry (mgm./100 cc.)

Glucose	28.00
Uric acid	0.70
Creatinine	0.60
Chloride	400.00
N.P.N.	35.80
Urea-N	30.00

The blood picture of this lamb is interesting. Both erythrocytes and leukocytes were decreased in number as compared with the normal, and a state of leucopenia apparently existed. The number of leucocytes was reduced to about 1/10 of the normal value and an excess of immature neutrophiles were found. The erythrocytes looked rather normal morphologically, although a few nucleated cells were present.

In chemical analysis of blood, the glucose was found to be about 1/2 normal, and uric acid and creatinine were also somewhat lower.

Gross lesions.

Umbilicus. Not completely healed and a large cicatricial area about 1 cm. by 2 cm. in size was observed.

Lungs. Bronchopneumonia involving apical and anterior cardiac lobes of the right lung. Only slight pneumonia on left side.

Ribs. Two ribs broken on right side of the body.

Brain. Congested. Cloudy fluid present beneath the meninges in the sulci. When cut into halves, a marked softening and typical lesions of encephalomalacia were noted in the anterior end of the cerebrum.

Other organs. Other organs appeared normal.

Microscopic lesions.

Liver. The liver was only slightly damaged, and the lesions consisted of moderate congestion, slight cloudy swelling and diffuse but mild fatty degeneration.

Adrenal glands. The adrenal glands were rather congested, especially in the zona fasciculata. Due to congestion, the glands were somewhat enlarged.

Lungs: The lungs showed a recent and severe broncho-pneumonia with small suppurative foci, involving the right apical and cardiac lobes. There was an intense inflammation of the bronchial wall with its lumen filled with exudate and sometimes desquamated cells. These affected bronchi were each surrounded by a ring of alveoli filled with an inflammatory exudate consisting either of polymorphonuclear leukocytes with small amount of fibrin, a little more fibrin and less polymorphonuclear leukocytes, or edematous fluid only. A short distance from the consolidated areas, the alveoli contained some macrophages and a few dying polymorphonuclear leukocytes. These consolidated areas were interrupted by areas of congestion, collapse and in the cardiac lobe especially, compensatory emphysema. In the right apical lobe, the outer layer of pleural connective tissue was injured and slightly thickened, and a section of it had ruptured so that the inflammatory exudate from the pneumonia underneath came out and collected on the surface of this part of the lung.

Brain. The lesions in the brain seemed to start from the cerebral meninges and then extended gradually to the cerebral cortex. The cerebral medulla was not involved. The cerebral meninges were very much congested and in the affected areas, most of the cellular structure of the pia mater was partly dissolved and detached from the underlying

cerebral cortex. A typical purulent exudate consisting of both macrophages and polymorphonuclear leukocytes was found and an active phagocytosis was present. This was evidence that the acute purulent meningitis had been going on for some time (Fig. 31). In the cerebral cortex a lesion of more recent development was noticed, since the predominating features were necrosis of cortical tissue, marked polymorphonuclear leukocyte infiltration, and, in some parts, death of brain tissue. No macrophages were found in the lesion of this area, so that the death of brain tissue must be attributed to the proteolytic action of polymorphonuclear leukocytes. In some parts of the more severely affected brain, the lesion extended quite deeply into the cerebral cortex, involving the neuroglia layer, the layer of small pyramidal cells, the layer of large pyramidal cells and a small outer portion of the layer of polymorphous nerve cells. The affected nerve cells became granular in appearance and acidophilic in reaction with their nuclei undergoing pyknosis and karyolysis, whereas the affected neuroglia became loosened and broken and thus lost their fibrous characteristics (Fig. 31).

Other tissues. Other tissues such as heart, spleen, intestine, skeletal muscles, etc., were not affected and appeared normal.

Discussion and summary.

1. This was a case of navel infection complicated by bronchopneumonia and encephalomalacia.

2. A state of marked leukopenia existed.
3. Since the adrenal glands were rather congested, the low value of blood glucose might be due to hypo-adrenalism.
4. The low values of blood creatinine and uric acid possibly resulted from the acute hepatic insufficiency, although the liver damage was not severe when subjected to microscopic study.

Case 7372 (Shropshire, male)

This was a case of navel infection leading to a typical acute suppurative bronchopneumonia as a result of mixed infection with three species of bacteria as mentioned below under etiology.

History. This lamb came from Cesor Farms, New Hudson, Michigan, and was one of a pair of twins born on 2/29/44. This animal was never active from the time it was born and had been discharging watery serous exudate from the nose for about three weeks. The lamb died on April 11th.

Etiology. One culture of Pasteurella and another of Diplococcus (Table 4: culture No. 7372 a, b.) were isolated from the lungs. In addition, a micro-organism indistinguishable from Actinomyces necrophorus was also isolated from the lungs.

Gross lesions. Typical bronchopneumonia with numerous small abscesses spread throughout the lungs. There was a large abscess (about 1 cm. x 2 cm.) of caseated pus on the anterior end of the urinary bladder, indicating that the infection had gone through the urachus to the bladder. Other organs seemed to be normal.

Microscopic lesions. Only lung tissue was saved for microscopic study and an acute suppurative bronchopneumonia with typical lesions somewhat like cases 7401 and 7410 was observed. The pneumonia was still in the acute stage with polymorphonuclear leucocytic infiltration as the predominating

feature, whereas the abscesses seemed to be in the beginning stage although the initial infection was undoubtedly there shortly after birth. (Fig. 32)

Case 7393 (Shropshire, female)

This was a case of navel infection resulting in abscess formation in the lungs, pneumonia, hemorrhage in liver, glomerulo-nephritis and arthritis.

History. The lamb was noticed lame for several days before being brought to the pathology barn on 4/17/44. There was a marked enlargement of the right carpal joint. The lamb was also lame in the right rear leg. The animal was killed three days later when it was 26 days old.

Etiology. Two cultures of Escherichia were isolated, one from the lung and the other from the right carpal abscess, and one species of Corynebacterium was cultured from the round ligament of liver of this case. (Table 4: Culture No. 7393, a, b, c.)

Blood Studies.

(a) Blood picture.

Erythrocytes	6,740,000	per c.mm.
Leucocytes	23,900	" "
Hemoglobin	6.8	g./100 cc.

(b) Blood chemistry (Mgm./100 cc.)

Glucose	75.55
Uric acid	3.12
Creatinine	0.80
Chloride	520.00
N.P.N.	67.00
Urea-N.	39.00

In comparison with the normal standard (Table 1), the values obtained for erythrocytes and hemoglobin were decidedly

lower and that for leucocytes was decidedly higher. The differential count was not included in this study. The values of all blood components studied were higher than normal (Table 2) except that of the creatinine which was in the lower normal limit.

Gross lesions.

Subcutaneous tissue. There was a subcutaneous abscess present at the tailhead and one in the region of the ilium.

Navel. The navel was infected. The round ligament of the liver was approximately 1 cm. in thickness and contained a core of greenish, cheesy pus.

Lungs. There were localized abscesses in the right apical lobe and the left cardiac lobe of the lungs.

Joints. The right carpal joint was enlarged and distended with greenish pus, and the right hip joint was greatly distended with the same kind of exudate.

Other organs. Apparently normal.

Microscopic lesions.

Round ligament of liver. The bacterial infection of the umbilicum and its extension along the round ligament of liver resulted in necrosis, suppuration, erosion, and fibrosis. The inner portion of the ligament was first attacked and the lesion gradually spread from there outward with a zone of coagulated dead cells, a zone of macrophages mixed with dying polymorphonuclear leucocytes, a zone of dense fibroblastic cells, macrophages and polymorphonuclear leucocytes, a zone of looser fibroblastic cells, macrophages,

plasma cells and lymphocytes, and finally a zone of fibrosis.

Liver. The liver was somewhat congested and showed cloudy swelling and hemorrhage. The sinusoids were moderately dilated and distended with an excessive number of erythrocytes and neutrophils. The cloudy swelling was more or less generalized, although it was mild in nature. Surrounding the areas of hemorrhage, a polymorphonuclear leucocytic infiltration was present. The hemorrhages were irregular in shape and variable in size, ranging from a minute unlimited area to those of considerable size which were surrounded by a thin fibrous wall.

Lungs. The lungs showed lesions of suppurative bronchopneumonia. The areas adjacent to the suppurative foci were infiltrated with polymorphonuclear leucocytes while those away from the suppurative foci showed atelectasis of the alveoli and bronchopneumonia with macrophage infiltration.

Intestine. The blood vessels of the intestine were rather congested but no other lesions were apparent.

Kidneys. Most glomeruli were swollen and stuffed with an excessive number of proliferative cells so that a case of proliferative glomerulo-nephritis existed. The lumina of convoluted tubules were greatly dilated and filled with a considerable amount of edematous fluid mixed with some albuminous casts. The epithelium of the convoluted tubules showed various degrees of cloudy swelling and some of the cells were desquamated.

Heart. Just beneath the endocardium, some fatty degeneration of the myocardium was noted.

Joint capsule. The joint capsule of the right carpus showed typical lesionsof an acute suppurative arthritis.

Other tissues. Apparently normal.

Summary and discussion.

1. The animal suffered from navel infection resulting in abscess-formation and bronchopneumonia of the lungs, hemorrhages in the liver, glomerulo-nephritis and arthritis.
2. This lamb was anemic as revealed by the low value for red blood cells and hemoglobin. Since this lamb was attacked by acute arthritis and was lame, apparently it did not get enough food in order to maintain its blood level.
3. The leucocytosis could be the result of various conditions such as abscess-formation, nephritis, acute arthritis, pneumonia and even hemorrhages in the liver. Probably, the combined effect of all these conditions was the real or actual cause of such a marked leucocytosis in this case.
4. The high values of all blood components (except creatinine) were directly related to the renal suppression due to nephritis, although acute arthritis would also call forth an increase in blood uric acid and anemia might sometimes induce a high level of blood chloride.

Case 7394 (Grade, Male)

This was probably another case of novel infection with Staphylococcus sp. and a gram-negative rod resulting in the formation of a variety of lesions such as necrosis in liver, abscesses in heart, skin and suocutaneous tissue, and arthritis in the carpal joint.

History. This lamb was brought in by Mr. Wakefield, County Agent, on 4/11/44. It was the smaller of a twin and the ewe had died. The lamb had an enlarged left carpal joint and was quite lame. The animal became weaker, could not get up and was killed at 3:00 P.M., 4/21/44 at the age of 20 days.

Etiology. A hemolytic Staphylococcus was isolated from left carpal joint and a gram-negative rod (Bacterium sp.) from heart-blood (Table 4: culture 7394 a, b.)

Blood studies.

(a) Blood picture

Erythrocytes	8,710,000 per c.mm.
Leucocytes	14,600 " "
Hemoglobin	12 g./100 cc.
Differential count (%)	
Lymphocytes	18
Neutrophils	80
segmenters	(73)
bands	(7)
Monocytes	2
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm./100 cc.)

Glucose	74.44
Uric acid	1.10
Creatinine	0.80
Chloride	480.00
N.P.N.	18.00
Urea-N	11.20

In comparing the results of blood studies in this case with the normal standard (Tables 1 & 2), the number of erythrocytes was somewhat lower, hemoglobin somewhat higher, and leukocytes decidedly higher with neutrophils (80%) predominating over lymphocytes (18%). The structure of erythrocytes was rather normal, although a few poikilocytes were occasionally observed. In respect to blood chemistry, the values of N.P.N. and Urea-N were rather low while the values of other elements seemed to fall within the normal range.

Gross lesions.

Suocutaneous tissue. There was an abscess about 1 1/2 cm. in diameter in the subcutaneous tissues near the thoraco-abdominal wall.

Joint. The left carpal joint was swollen and distended and the joint capsule was thickened and the cavity filled with cloudy flocculent exudate. A smear made from the exudate showed active phagocytosis. (Fig. 33).

Heart. Numerous abscesses were present in the heart. There was a marked pericarditis and myocarditis, the percardium being tightly adhered to the myocardium. (Fig. 34)

Other organs. Apparently normal.

Microscopic lesions.

Liver. The liver showed evidences of congestion, cloudy swelling, some diffuse fatty degeneration, some hydropic degeneration, and necrotic foci with marked polymorphonuclear

leucocytic infiltration. In general, the various lesions of liver in this case resembled quite closely those found in Case #7337 with the exception of diffuse fatty and hydropic degenerations which were not found in that case.

Heart. There were numerous abscesses in the heart and they were found mostly in myocardium and a few in the epicardium. Due to the fact that some of the epicardial abscesses ruptured and the inflammatory exudate went into the pericardial sac, the pericardium became involved. As a result, a case of suppurative myocarditis, epicarditis and pericarditis actually existed. The abscesses were irregular in shape, variable in size (0.15 mm. X 0.12 mm. to 5 mm. X 4 mm.), and were characterized by marked cell infiltration (both polymorphonuclear leucocytes and macrophages) surrounded by an extensive fibrosis (Fig. 35). The endocardium, however, was not affected.

Joint capsule. The joint capsule of left carpus showed a typical lesion of an acute suppurative arthritis somewhat like that found in Case #7393.

Skin abscess. The skin lesion was partially opened but possessed all the general characteristics of a typical abscess. Except at the point where its opening was located, the abscess was bounded on its outer surface by a thin layer of epidermis together with a small portion of outer corium and on its inner surface by a thick layer of fibroblastic tissue, which extended down the muscle tissue

underneath. On this account, most parts of the inner corium and stratum subcutaneum had been destroyed and replaced by fibrosis.

Other tissues. Those tissues such as intestines, kidneys, etc., were also studied, but they showed no abnormalities.

Summary and discussion.

1. This lamb was small and weak due to the presence of necrosis and hemorrhages in liver, abscesses in heart, skin, subcutaneous tissues, and arthritis in carpal joint.
2. Both bacterial infection and abscess-formation resulted in a marked neutrophilia.
3. Since the liver was damaged, it apparently could not perform its functions properly in maintaining the normal level of blood urea and non-protein nitrogen. These values were rather low.

Case No. 7362 (Shropshire, male)

This was a case of docking infection which resulted in abscess-formation of the spinal cord, mild associated conditions were an enteritis, and some disturbances in the liver and kidneys.

History. This lamb showed bilateral paralysis of the rear limbs on 3/31/44 and was brought to the pathology barn with its mother. The lamb was kept under observation for an entire week and showed no improvement. The animal, however, remained very bright, nursed regularly, and it seemed doubtful whether this was "stiff-lamb disease". On 4/7/44, at the age of 9 days, the lamb was bled and killed.

Etiology. One species of undetermined gram-negative coccoid bacillus was isolated from the liver and two species of Corynebacterium were isolated from the spinal cord. (Table 4: Culture No. 7362, a, b, c.)

Blood Studies.

(a) Blood picture

Erythrocytes	8,870,000 per c.mm.
Leucocytes	9,950 " "
Hemoglobin	9.8 g./100 cc.

Differential count (%)

Lymphocytes	35
Neutrophils	65
segmenters	(56)
bands	(6)
juveniles	(3)
Monocytes	0
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm/100 cc.)

Glucose	97.50
Uric acid	1.10
Creatinine	1.00
Chloride	430.00
N.P.N	34.00
Urea-N	11.20

Except for some leucocytosis with increased neutrophils, reduced lymphocytes, and slight increase in glucose, the various values of the blood elements of this lamb were not much different than those of normal lambs. The morphology of erythrocytes was normal.

Gross lesions.

Skeletal muscles. There was some atrophy of the muscles of the rear limbs.

Tail-head. In the region of the tailhead at the docking site, a greenish colored pus was present. This suppurative lesion extended into and involved the spinal cord as far as the center of the lumbar area. (Fig. 36)

Other organs. Apparently normal.

Microscopic lesions.

Liver. The liver showed congestion, cloudy swelling, diffuse fatty degeneration, and occasional tiny necrotic foci with a polymorphonuclear leucocytic infiltration. The lesions were somewhat similar to those found in the liver of Case #7356.

Intestine. The intestine showed lesions of a mild enteritis, involving particularly the mid-gut. There was a superficial necrosis involving both the epithelium and lamina propria with a moderate macrophage reaction in the subnecrotic zone.

Kidneys. The kidneys were suspected of having a congenital cystic condition. Here and there, the proximal convoluted tubules were dilated to the cystic stage with urinary excretion and sometimes contained albuminous casts. Some fibroblastic proliferation was also observed in these affected areas. As the animal was young, this condition was believed to have developed in intra-uterine life. The epithelium of the convoluted tubules showed cloudy swelling, and occasionally small foci of lymphocytic infiltration were noted.

Brain. Brain was slightly congested, otherwise it seemed normal.

Spinal cord. The spinal cord was severely injured by the docking infection which resulted in abscess-formation and fibrosis. The tissue reaction seemed to start in the grey substance of the cord along the central canal and the lesion gradually spread to the surrounding areas. The epithelial lining of the central canal was first attacked, and as these cells were involved, a polymorphonuclear leucocytic infiltration took place. As the process developed, more of the surrounding tissues became involved and more polymorphonuclear leucocytes migrated to the area. In some

sections of the affected cord, both perivascular polymorphonuclear leucocytic infiltration and infiltration along the commissures were readily observed. When these affected tissues became necrotic and partly phagocytized by the polymorphonuclear leucocytes or partially dissolved by their proteolytic enzyme, a semi-liquid abscess was formed. In general, a recently formed abscess (Fig. 37) had the following characteristics: a liquified center with some semi-solid exudate, a zone of dark-stained material consisting mostly of dying polymorphonuclear leucocytes and some fibrin, and a zone of affected nervous tissue infiltrated with active polymorphonuclear leucocytes. The affected nerve cells and neuroglia took eosin stain and the nuclei had either disappeared or undergone pyknosis.

In the more advanced or older lesion, the spinal abscess had slightly different manifestations. Besides all the characteristics mentioned previously for the recently formed abscess, two other elements were observed, namely, a marked infiltration of macrophages in the 3rd layer and a pronounced fibrosis in the 4th layer. As a result, there were five different zones in the older abscess instead of three zones as were found in the more recent one. The endothelium of capillaries located on the walls of the older abscesses was proliferating and showed evidence of hyperplasia, suggesting that their activity was still

proceeding. The etiological agent, the diphtheroids, was observed in the inner second layer of the abscesses.

Other tissues. Other tissues were more or less normal.

Summary and discussion:

1. This was a case of docking infection resulting in abscess-formation of the spinal cord, mild enteritis, and some disturbances in the liver and kidneys.
2. The animal showed bilaterial paralysis in the rear limbs due to involvement of the lumbo-sacral region of the spinal cord by abscess-formation and naturally the nerves supplying the rear limbs had ceased to function.
3. A bacterial infection of the tail-head caused the mild neutrophilia.
4. Blood glucose was slightly increased. The reason for that was probably the result of impaired liver function.
5. The lamb remained bright throughout life, which indicated that the mild enteritis and disturbances in the liver and kidneys were recent developments and general systemic involvement was not apparent.

Case 7365 (Hampshire, male)

This was a case of docking infection and somewhat resembled Case #7362. However, this case was more acute and serious, since it had a combined infection of Actinomyces necrophorus and Corynebacterium sp., resulting in the formation of abscesses around the central canal of the spinal cord, hemorrhages in the arachnoid membrane of the meninges, and an acute valvular endocarditis.

History. This lamb was noticed to have some paralysis of the rear limbs on the morning of 4/8/44. It nursed well when held up to the mother. The lamb was found dead at 8:00 A.M. two days later when it was about one month old.

Etiology. Corynebacterium sp. and Actinomyces necrophorus were isolated from the spinal cord. Actinomyces necrophorus was cultured in deep brain broth, while the general characteristics of the Corynebacterium sp. are referred to in Table 4: Culture No. 7365.

Gross lesions.

Urinary bladder. The bladder was distended to the extent that urine was present in the pelvis of the kidneys producing a hydronephrosis. However, the urethra was open.

Tailhead. The docking wound was not healed. There was considerable inflammation present about this area. Upon splitting the vertebral column, exudate was found in the vicinity of the cauda equina and the inflammatory process

extended up the central canal to a point located at the posterior part of the lumbar region. In the lumbar region, there was an extensive hemorrhage around the spinal cord approximately 2.5 cm. in length.

Bicuspid valve. The bicuspid valve showed some areas of inflammation.

Other organs. No visible lesions were observed.

Microscopic lesions.

Spinal cord. Hemorrhage was rather extensive in the arachnoid membrane. It was observed that the blood flooded the subdural space for a considerable area, covering the whole dorsal half of the spinal cord. The abscess (Fig. 38) around the central portion of the cord was recently formed and it resembled rather closely that found in Case #7362. Besides this abscess, there were four greatly inflamed or abscess-like spots located in the perivascular areas, two on each of the mixed lateral tracts of Cajal. In fact, one of these four areas was already transformed into a small abscess. In these lesions, the capillaries were considerably dilated. The endothelium was swollen, loosened, and polymorphonuclear leucocytes had migrated through the capillary walls. The endothelial layer was surrounded by a clear zone filled with edematous fluid and cells and limited by a delicate layer of fibrous tissue which was in turn surrounded by a clear space of about the same size as the previous mentioned area. Finally, a dense layer of fibrin was noticed at the outermost limit of the

lesion. The etiological agents, Corynebacterium sp. and Actinomyces necrophorus, were observed in the exudate of the central abscess, and it indicated clearly that this case was one of mixed infection (Fig. 39).

Bicuspid valve. The lesion in the bicuspid valve of the heart consisted of hemorrhage, necrosis, and a marked polymorphonuclear leucocytic infiltration mixed with some fibrin. A short distance from the affected area, the fibroblastic cells were actively proliferating, although a definite wall segregating the inflamed are had not formed. Actually, the lesion in the bicuspid valve constituted what is known as acute valvular endo-carditis.

Case 7370 (Grade, male)

This lamb suffered from infection of the castration wound.

History. This animal was castrated about three weeks before death which occurred on 4/10/44. The lamb was noticed to be stiff in the left hind leg on the previous day. Upon talking with the owner, it was felt that this might have been a case of "stiff-lamb disease".

Gross pathology. The scrotum had been partially removed, and in healing, the wool and skin had turned in. The wound had failed to heal and the inflammatory process extended from the scrotal region along the left abdominal wall and as far forward as the end of the penial sheath. No other visible lesions were observed and apparently this was not a case of white-muscle disease, since no muscular lesions were observed.

Case 7282 (Hampshire, male)

This was a case of bronchopneumonia.

History. This animal was born on 2/28/44. It was noticed to be droopy, ceased to suckle, and breathed with difficulty as though it had pneumonia. It was then treated with pneumonia serum and mineral oil. The lamb died when it was three days of age.

Gross pathology

Lungs. Frothy mucous exudate was present in the trachea and bronchi. Some congestion of the mucous membrane of the lower trachea and bronchi was noticed. Moreover, an acute fulminating and rapidly-spreading bronchopneumonia was found in the apical, cardiac and anterior one-fourth of the diaphragmatic lobes of both lungs.

Liver. There were small white foci about 1 mm. in diameter throughout the liver. The liver was friable and slightly increased in size.

Other organs. Apparently normal.

Bacterial culture. Negative.

Case 7308 (Southdown, male)

This was a case of passive hyperemia resulting from an open ductus arteriosus and complicated with bronchopneumonia.

History. This lamb was born on 2/29/44. On 3/5/44, the lamb was noticed breathing heavily and was given 20 cc. of pneumonia serum subcutaneously. Two days later, the same dose of serum was again given. After each injection, the lamb appeared brighter and more active. However, it was found dead on 3/9/44.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs. See Table 4: culture No. 7308 for its general characteristics.

Gross lesions.

Lungs. There was a marked congestion of the lungs with some recent pneumonia on the right side involving the apical, cardiac and small portion of the diaphragmatic lobes.

Liver. The liver was very much congested. This organ was enlarged by at least one-third.

Heart. The ductus arteriosus was open which allowed the free passage of blood into the aorta from the pulmonary artery.

Other organs. Apparently normal.

Microscopic lesions.

Liver. The liver showed acute venous congestion with marked diffuse fatty degeneration and necrosis. Fat globules were present

not only in cells located at the periphery of the hepatic lobule but also in the intermediate and central parts of the lobule. Almost every cell in the lobule was involved. Some cells contained a number of various sized fat globules in which case the nucleus was still centrally located, while others contained just one large fat globule and the nucleus was pushed to one side of the cell and flattened so that the cell resemoled those of adipose tissue. In some lobules, the necrosis had extended almost around the central vein. (Fig. 40) The hyperemia was generalized, but in some spots it was so extensive that the sinusoids had ruptured and consequently hemorrhage occurred.

Intestine. Due to acute passive hyperemia, the intestine was also affected. The intestinal capillaries were very much congested, and there was some mucoid degeneration of the epithelium.

Lungs. The lungs were congested and showed lesions of a typical bronchopneumonia of recent development. The polymorphonuclear cell infiltration was the predominating feature throughout the pneumonic areas.

Other tissues. Other tissues were more or less normal.

Case 7329 (Black Top Delaine, female)

This was a case of interstitial bronchopneumonia with marked passive congestion of liver and kidneys.

History. This lamb was born on 3/21/44 and was found dead at 7:30 A.M. of the same day.

Etiology. No bacteria were isolated from this case.

Gross lesions.

Lungs. No evidence that the lamb had ever breathed.

Liver. Slightly mottled.

Other organs. Apparently normal.

Microscopic lesions.

Lungs. The lesions in the lungs were interesting. It was a case of interstitial bronchopneumonia having developed during fetal life. In addition to all those general characteristics found in a typical bronchopneumonia, there was a marked lymphocytic infiltration. These lymphoid cells were found mostly in the alveolar walls, but some of them were also found in the peribronchial and perivascular regions. Here and there, groups of these cells were in aggregate forming tubercle-like structures. (Fig. 41, 42) The alveolar walls were thickened while the alveoli were either infiltrated with macrophages and a few polymorphonuclear leucocytes or distended with edematous fluid. The bronchioles contained inflammatory exudate, consisting of some mucous material mixed with

polymorphonuclear leucocytes. In certain areas of the affected lungs, there was evidence of fibroblastic proliferation in the alveolar walls as well as in the peribronchial and perivascular region, while in others--- especially in those near the bronchial walls quite a number of plasma cells were observed. Occasionally, giant cells were found in certain alveoli. In general, the alveoli were more or less in an actelectatic condition, but this condition was interrupted here and there by areas of compensatory emphysema which indicated that the lamb had breathed for some time after birth.

Liver. The liver was exceedingly congested and showed marked fatty degeneration. The sinusoids were greatly enlarged and filled with an excessive number of blood cells. The fatty degeneration was pronounced and diffuse, involving practically every hepatic cell in the liver. Most hepatic cells contained one to several large fat globules so that the nuclei either were pushed to the side of the cell or were deprived of nutrition and showed karyolysis.

Kidneys. The kidneys showed marked congestion and hemorrhage, involving both parenchyma and interstitial tissues. Throughout the cortex and medulla, an excessive number of erythrocytes were observed within the inter-lobular capillaries and capillaries in the medullary area. The glomeruli were also rather congested and a case of glomerular hemorrhage actually existed so that blood cells

were found inside quite a number of the convoluted tubules as well as in the collecting tubules.

Other organs. Other organs were not affected and seemed normal.

Case 7350 (Shropshire, female)

This was a case of bronchopneumonia with probably some other complication.

History. The lamb was owned by A. E. Holtforth, Fenton, Michigan. It was a large well-nourished lamb about three weeks of age. The animal died suddenly the previous evening (3/25/44) and the carcass was beginning to decompose and was badly bloated when brought to the laboratory for autopsy.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs. (Table 4: culture No. 7350)

Gross pathology.

Stomach. On the lesser curvature of the stomach, there was a large area of hemorrhage and erosion of the mucous membrane. Surrounding this area, the folds of the stomach were thickened to about 1.0 cm. and were very hard.

Lungs. Pneumonia was present in the right apical and cardiac lobes. The pneumonia did not appear to be of sufficient extent to cause death.

Heart. There was an extension of the inflammatory process to the pericardium with some hemorrhage on the pericardium and excess of pericardial fluid.

Other organs. Apparently normal.

Case 7351 (Shropshire, male)

This was another case of bronchopneumonia with probably some other complication.

History. The lamb was small and weak and had not been doing well since birth. The animal came from the same farm as Case #7350. It died on 3/25/44 when it was about one week old.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs, and its general characteristics are mentioned in Table 4: culture No. 7351.

Gross pathology. There was a very slight amount of patchy pneumonia in the right apical lobe. Besides this, no other lesions were observed.

Case 7353 (Southdown, female)

This lamb suffered from an acute hemorrhagic bronchopneumonia.

History. This animal had not been able to stand on its front legs and died in the morning of 4/1/44 when it was about two days old.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from liver, heart blood, and lungs. The general characteristics of this micro-organism are given in Table 4: culture No. 7353.

Gross lesions.

Trachea. Contained frothy foam.

Lungs. Showed reddened areas which were solidified, suggesting either hemorrhage or beginning bronchopneumonia.

Mesenteric lymph nodes. Congested and somewhat enlarged.

Other organs. No visible lesions observed.

Microscopic lesions. The lungs showed a very acute hemorrhagic type of bronchopneumonia. It was the most severe case of bronchopneumonia observed during the entire lambing season last year. The lungs were exceedingly congested and hemorrhagic. In fact, hemorrhage was the most predominating feature of the lesion, and almost everywhere in the lung an excessive number of erythrocytes were observed. The bronchioles were very badly inflamed and the lumina as well as the surrounding alveoli were filled with either inflammatory cells, fiorin, or erythrocytes. (Fig. 43) The consolidated areas were interrupted by areas of congestion,

collapse, and emphysema, following the same pattern as a typical case of bronchopneumonia. The serosa was also very much congested and swollen.

Cases 7371 & 7373 (Shropshire female & male)

These two cases showed bronchopneumonia complicated with Actinomyces necrophorus infection of the lungs.

History. These two lambs came from the Cesor Farm, New Hudson, Michigan. One of them was born on 2/28/44, while the other on 3/8/44. Both were given serum treatment for pneumonia, but they died on the same day (4/11/44) in spite of the treatment.

Etiology. Two species of bacteria were isolated from the lungs and pericardial sac of case 7371 (Table 4: culture No. 7371 a, b) and three from the lungs of case 7373 (Table 4: culture No. 7373 a, b, c). A micro-organism morphologically similar to Actinomyces necrophorus was isolated from the lungs of each case by deep brain broth culture.

Gross pathology.

Lungs. The lungs were greenish brown in color and the exudate in the lower trachea was also greenish brown and had a putrid smell. Numerous greenish colored abscesses were present throughout the lungs and there were adhesions of the lungs to the ribs. In case 7371, there was a fibrinous pericarditis in addition to the lung lesions.

Case 7401 (Shropshire, female)

This was a case of acute suppurative bronchopneumonia complicated by a mild enteritis.

History. This lamb was 24 days old and was found dead at 9:00 A.M. 4/24/44. The lamb had been nursing a goat and appeared normal the day before its death.

Etiology. Three different kinds of micro-organisms were isolated from this lamb, namely, one from heart blood (Bacterium sp.) and two from the lungs (Pasteurella sp. and Neisseria sp.). Their general characteristics are referred to in Table 4: cultures No. 7401 a, b, c.

Gross lesions. Upon removing the skin, the carcass was found to be rather emaciated and the lamb had a severe diarrhea.

Intestine. There was a slight enteritis present in the ileum. No formed feces were found in the rectum.

Lungs. Bronchopneumonia was present in the apical and cardiac lobes of the right lung. There was only slight involvement of the left lung. The pneumonia would not be considered severe enough to be the entire cause of death.

Microscopic lesions. Only lung tissue was preserved for study. Both the pneumonia and the suppurative foci were in beginning active stages, and much lung tissues had been damaged especially by the suppurative processes. In one instance, a relatively large area of about 4 mm. by 5 mm. was occupied and destroyed by a spreading abscess.

Case 7410 (Hampshire, male)

This was a case of acute suppurative bronchopneumonia complicated with enteritis.

History. The lamb showed mild scouring on 4/27/44 and was found dead the next morning. It was about 25 days old.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lung. (Table 4: culture No. 7410)

Gross lesions.

Lungs. Bronchopneumonia was present on both apical, cardiac and anterior third of the right diaphragmatic lobes. The pneumonia was progressive as there was a marked zone of congestion between the pneumonic area and the normal lung.

Intestine. There was slight congestion of the Peyer's patches and the mucosa of the small intestine. No formed feces were found in the colon or rectum.

Other organs. Apparently normal.

Microscopic lesions. In studying the sections taken from right anterior cardiac lobe and right diaphragmatic lobe of the lungs, a case of typical acute suppurative bronchopneumonia was noted. The bronchioles were severely damaged and their lumina were filled with inflammatory exudate consisting mostly of polymorphonuclear leucocytes and desquamated epithelial cells. The alveoli surrounding each inflamed bronchiole contained mostly polymorphonuclear leucocytes, indicating that the case was still in acute stage. Here and there, abscesses of recent development

were found. Farther out from the affected areas, a few macrophages mixed with some polymorphonuclear leucocytes were found in the alveoli, yet the latter seemed to be predominating. The pneumonic areas were spreading quite extensively and were only occasionally interrupted by areas of marked congestion, collapse, and emphysema.

Case 7415 (Hampshire, female)

This was a case of bronchopneumonia complicated with encephalitis (probably listerellosis) and enteritis.

History. This lamb was born on 3/7/44 and its twin sister died about three days later (Case No. 7310). On 5/3/44, the animal was down on its side with the head thrown back and the feet were moving as though the animal were running. The animal was then killed.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs. (Table 4: culture No. 7415).

Blood studies.

(a) Blood picture

Erythrocytes	8,970,000 per c.mm.
Leucocytes	14,150 " "
Hemoglobin	7.8 g./100 cc.

Differential count (%)

Lymphocytes	2
Neutrophils	96
segmenters	(78)
bands	(18)
Monocytes	2
Eosinphils	0
Basophils	0

(b) Blood chemistry (Mgm./100 cc.)

Glucose	170.00
Uric acid	2.60
Creatinine	1.30
Chloride	460.00
N.P.N.	41.00
Urea-N.	28.30

The blood picture of this case showed a marked leucocytosis neutrophilia with neutrophilic cells (96%) greatly predominating over lymphocytes (2%). Both hemoglobin value

and the number of erythrocytes were somewhat lower than the normal values (Table 1), and there was structural evidence of microcytic hypochromia of the erythrocytes with an occasional occurrence of nucleated young cells.

From the results of chemical analysis of the blood, the glucose was found to be about 2.6 times higher than the normal standard whereas uric acid, N.P.N., and urea-N were all somewhat above normal.

Gross lesions.

Lungs. The right apical and cardiac lobes showed "cold pneumonia" and there seemed to be no recent extension of the process. In the left diaphragmatic lobe, there were some spots which appeared to be hemorrhages.

Liver. It showed what appeared to be some cloudy swelling.

Brain. It seemed edematous although it was quite firm otherwise.

Other organs. Apparently normal.

Microscopic lesions.

Lungs. In a study of those tissue sections taken from the right apical and right cardiac lobes of the lungs, a case of typical bronchopneumonia of several weeks duration was revealed. The characteristic features of the lesion were metaplasia of the small bronchioles and thickening of the alveolar walls as a result of fibroblastic activity. In general, the pneumonia was in a stage of resolution with a predominance of macrophages carrying on active phagocytosis, although groups of polymorphonuclear leucocytes were

observed here and there in the alveoli and bronchioles.

In a section taken from the left diaphragmatic lobe, however, a different pneumonic lesion was found. There were some hemorrhages in this portion of the lungs indicating that a state of hemorrhagic bronchopneumonia actually existed. This condition appeared to be the result of a recent extension of the old pneumonic lesion.

Thalmus. In the thalmus, quite a number of acute inflammatory foci were found. These foci were scattered throughout the cortex, especially in the perivascular areas, and each was characterized by a marked focal infiltration of both mononuclear and polymorphonuclear leucocytes, constituting a lesion commonly known as "perivascular cuffing". (Fig. 44) This lesion resembled that observed in listerellosis and other infections. The nervous tissue, however, was not greatly damaged so that undoubtedly this was a case of beginning encephalitis. The brain was not cultured.

Liver. The liver was only moderately congested, but it showed a marked cloudy swelling and some diffuse fatty degeneration.

Intestine. In the small intestine, while both duodenum and the last portion of ileum near the caecum were normal, a greater part of the jejunum and ileum were severely affected with acute enteritis. A greater part of the mucosal epithelium was necrotic and partly desquamated, filling the intestinal lumen with inflammatory exudate which consisted of dead tissues and some leucocytes.

Summary and discussion.

1. Since this lamb exhibited bronchopneumonia, encephalitis, and enteritis, the chance for its survival was meager.
2. The animal was slightly anemic and some of the vital organs (lungs, brain, liver, and intestine) were diseased. Apparently, the animal was under-nourished. Due to the presence of anemia, the blood chloride was a little higher than normal.
3. The blood glucose, uric acid, creatinine, N.P.N., and Urea-N were all higher than normal, which might result from the hepatic disorder and possibly the dehydrated condition.

Case 7443 (Shropshire, male)

This was another case of bronchopneumonia complicated with Actinomyces necrophorus infection in the lungs.

History. This lamb was about 64 days old. It was noticed sick on 5/11/44 and was brought to the pathology barn. There was marked abdominal breathing and the lamb was quite inactive. The lamb was treated with pneumonia serum for two days but no improvement was noted. The lamb died on 5/13/44.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs (Table 4: culture No. 7443) and a micro-organism indistinguishable from Actinomyces necrophorus was also isolated from the lung abscesses.

Gross pathology. There was a marked bronchopneumonia involving the apical, cardiac and about half of the diaphragmatic lobes. The right apical lobe contained many greenish abscesses.

Case 7474 (Oxford, male)

This animal suffered from bronchopneumonia complicated by cecal and colonic constipation.

History. This animal showed severe abdominal breathing and was brought to the pathology barn on 4/28/44 and was treated with pneumonia serum and sulfathiazole. The animal showed improvement and began to eat and nurse well until 5/21. On that day and for the four days following, deep abdominal breathing was noted and the abdomen began to be distended. The lamb was killed on 5/25/44 when it was about 7 weeks old.

Etiology. A gram-negative rod (Bacterium sp.) was isolated from the lungs. (Table 4: culture No. 7474)

Blood studies.

(a) Blood picture

Erythrocytes	9,070,000 per c.mm.
Leucocytes	2,200 " "
Hemoglobin	10.8 g./100 cc.

Differential count (%)

Lymphocytes	37
Neutrophils	61
segmenters	(40)
bands	(21)
Monocytes	0
Eosinophils	1
Basophils	1

(b) Blood chemistry (Mgm./100 cc.)

Glucose	88.75
Uric acid	2.60
Creatinine	1.15
Chloride	350.00
N.P.N.	30.00
Urea-N	18.20

The blood studies of this case revealed that except for a moderate leukopenia in which the percentage of lymphocytes (37%) was outnumbered by that of neutrophils (61%), the other constituents of the blood did not seem to be affected and did not deviate conceivably from the normal standard (Tables 1 & 2). Moreover, the erythrocytes were morphologically normal and contained a suitable amount of hemoglobin.

Gross lesions.

Lungs. A "cold pneumonia" was found in the apical, cardiac and a small section of the diaphragmatic lobes of the right and left sides of the lungs. There was no indication of acute progression. Resolution was taking place.

Intestines. The cecum and large colon were distended with solid, dehydrated feces, and the mucosa was slightly edematous. Due to constipation, fluid material had backed up so that a considerable amount of it was present in the fourth stomach.

Other organs. The other organs were apparently normal.

Microscopic lesions.

Lungs. A typical bronchopneumonia was present. Most of the pneumonic areas were dominated by macrophages and a few polymorphonuclear leucocytes were present, indicating that resolution was taking place. However, in some smaller areas where the polymorphonuclear leucocytes were still quite numerous, there was some hemorrhage present. These areas of hemorrhage might account for some of the later symptoms of labored breathing and inactivity but

which was undoubtedly caused for the most part by the cecal and colonic constipation.

Kidneys. The kidneys were congested, and some dark brown pigment was found at the base of the stratified epithelium of papillae just adjacent to the pelvis.

Other tissues. The other tissues were more or less normal.

Summary and discussion.

1. This was a case of bronchopneumonia complicated by cecal and colonic constipation. The dyspnea and severe abdominal breathing indicated such a condition.
2. The blood studies revealed some abnormalities of the blood and there was a moderate leukopenia with some immature forms of neutrophilic leucocytes present.
3. The low value of blood chloride was directly related to the pneumonic condition.
4. The lamb might have been saved if an enema had been given in order to relieve the cecal and colonic constipation and the serum and sulfathiazole treatment renewed so as to clear up the pneumonic condition.

Case 7334 (Hampshire, male)

The lamb suffered from bacteremia due to Pasteurella infection.

History. This case and the next one (Case 7335) had the same history. The lambs were born as twins at 11:00 P.M., 3/20/44. Both of these lambs were very strong and apparently normal. The ewe had plenty of milk and the lambs suckled very well even up to 3:00 P.M., 3/21/44. At 7:00 P.M. of the same day, these two lambs were found dead in sleeping position.

Etiology. A culture of Pasteurella (Table 4: culture No. 7334) was isolated from liver, heart-blood, and spleen.

Gross lesions.

Skin. Subcutaneous hemorrhages were found over the back region. These hemorrhages varied from 2 to 10 mm. in diameter.

Muscles and fasciae. Large blotchy hemorrhages were found in the semitendinosus muscle, and also in the intercostal muscles on the left side of the body. Some hemorrhages were also noticed in the fasciae over the intercostal muscles on the right side and in the fascia anterior to the elbow joints.

Liver. A few subcapsular hemorrhages were observed in the liver and the organ was covered by some whitish fibrin.

Small intestine. The small intestines were quite edematous and the Peyer's patches were markedly congested and edematous. The edema was so severe that the Peyer's patches resembled blebs.

Stomach and spleen. Petechial and ecchymotic hemorrhages were found in the stomach wall and under the serosa at the lesser curvature of stomach. Hemorrhages were present in the subcapsular area of the spleen.

Omentum and peri-orbital fat. Petechial hemorrhages were noted in these tissues.

Other features. There was an excess of pleural, pericardial, and peritoneal fluids. The ductus arteriosus was almost closed. No hemorrhages were observed in the brain, although the blood vessels were rather congested. The heart, lungs, and kidneys were apparently normal.

Microscopic lesions.

Liver. The liver showed marked congestion with cell infiltration and hemorrhages. The sinusoids were very much distended and filled with blood cells. The polymorphonuclear leucocytic infiltration was rather diffuse, but they were more concentrated around the portal trinities. The hemorrhages seemed to originate from the portal vessels and spread from there to the surrounding tissues and gradually increased in size.

Spleen. The lesion of the spleen was an interesting one. There was a marked serosal congestion and hemorrhages without observable involvement of the Malpighian corpuscles or splenic pulp. In the serosa, the capillaries in some parts were congested while in most of the other parts they were enormously enlarged, being at least thirty times as large as the normal ones. (Fig. 45) Hemorrhage took

place in the areas where the engorged capillaries were located. In the inner portion of the spleen, both spleen pulp and Malpighian corpuscles seemed to show but little if any changes.

Skeletal muscles. The skeletal muscles showed marked congestion in some parts, cell infiltration in others, and in still others necrosis and hemorrhage. In the areas of hemorrhage, the blood was observed either in between the muscle-fibers or in open spaces formed by the separated necrotic muscle tissue. The necrotic muscle fibers had lost the striations and nuclei and took a lighter stain, while others had broken into small irregular pieces. In some of those showing a more advanced stage, the pieces of dead muscle-fibers were shrunken and crowded together, leaving irregular spaces which were replaced by connective tissue and adipose cells. These shrunk and crowded masses of dead muscle had changed their characteristics and were stained blue instead of purplish pink with Mallory's anilin blue.

Kidneys. The kidneys were rather congested, and some hyaline casts were observed from glomeruli down to the collecting tubules, indicating increased permeability of the glomerular endothelium.

Case 7335 (Hampshire, female)

This animal also suffered from acute bacteremia with Pasteurella infection and the condition was somewhat similar to case 7334.

History. Same as Case 7334.

Etiology. A gram-negative rod (Pasteurella sp.) was isolated from liver, heart-blood, and spleen. For general characteristics of the etiological agent, see Table 4:: culture No. 7335.

Gross lesions. The gross lesions were almost the same as for those recorded for case 7334 except that blotchy hemorrhages were found in the muscles ventral to the pubis.

Microscopic lesions. Only a few pieces of tissues were preserved and studied, but most of the lesions found in the various tissues resembled quite closely those observed in case 7334.

Skeletal muscle. Hemorrhage was present in the subcutaneous connective tissue and between the muscle fibers. The lesions were similar to those found in case 7334.

Skin over loin. The skin over loin region was very much congested and some hemorrhages were present in the subcutaneous tissue and in the muscle just beneath.

Liver. The liver was severely congested and such lesions as cloudy swelling, fatty degeneration, necrotic foci, and hemorrhages were present. The sinusoids were greatly engorged. The hemorrhages were found in the parenchyma

of the liver as well as in the subcapsular region. The cloudy swelling was diffuse but it was especially pronounced in those cells not being affected with fatty changes. Fat globules were seen in most of the liver cells, although they were more conspicuous around the central vein of each lobule than around the periphery. Here and there throughout the liver, tiny necrotic foci were observed and each of them consisted of a small group of dead hepatic cells which had lost their nuclei and normal architecture, and took a little deeper hematoxylin stain than the normal cells. The cells in close contact with such foci were also affected as their cytoplasm showed an affinity for eosin stain and the nuclei showed pyknosis.

Case 7399 (Shropshire, male)

This was a case of enterotoxemia.

History. This lamb was 34 days old and was found dead on the morning of 4/24/44. No previous symptoms were noted.

Etiology. Clostridium welchii, Type D.

Gross lesions.

Thoracic cavity. Excess of fluid in the pleural cavity; edema of the lungs; excessive pericardial fluid with fibrinous coagulum in percardial sac; petechial hemorrhages on the thymus.

Abdominal cavity. Blotchy hemorrhages (Fig. 46) on the jejunum, ileum and colon; congestion of Peyer's patches; cecum and colon filled with grayish liquid ingesta. The fourth stomach was distended with ground grain and coagulated milk. The kidneys were soft and markedly congested in certain areas. There was excess of peritoneal fluid.

Microscopic lesions. Since this animal was found dead for some time before autopsy, the only tissue taken for microscopic study was small intestine. From the sections where the blotchy hemorrhages were present, it was found that most of the hemorrhages were confined to the submucosal area. The hemorrhage was rather extensive and it seemed to be initiated by the weakened congested blood vessels in that particular region. Moreover, the solitary lymph nodules in submucosa were greatly enlarged and were infiltrated by

quite a number of macrophages and some polymorphonuclear leucocytes which furnished another indication of involvement of the Peyer's patches. (Fig. 47)

Case 7426 (Oxford Grade, male)

This was a case of entero-toxemia.

History. This lamb came from the farm owned by Mark Williamson, Lapeer, Michigan. Eight or ten of the largest and fattest lambs had died and this was one of them. One bunch of 35-40 ewes and their lambs in which most of the death losses had occurred was fed about 3/4 bu. of grain per day, but they apparently did not get enough exercise. This lamb died at the age of about five weeks without noticeable symptoms.

Gross pathology.

Carcass in general. The carcass was well-nourished.

Body fluids. There was an excess of pericardial, pleural, and peritoneal fluids.

Stomach. The stomach and rumen contained dark colored ingesta. Some milk was found in the fourth stomach, the mucous membrane of which was badly congested.

Small intestine. The Peyer's patches of the small intestine stood out distinctly and were surrounded by a zone of congestion.

Lungs. The lungs were rather edematous.

Other organs. Other organs were normal.

Culture. Negative.

Cases 7434-'35-'36 (Black Top Delaine: male, female, male)

These three cases were considered to be enter-toxemia and they resembled rather closely case 7426.

History. These three lambs were owned by Gerald Fleming of Munith, Michigan, and were about three weeks old when brought to the laboratory for autopsy. These lambs were the largest of the group of the same age and died suddenly on 5/7/44. Shortly before death, the animals went down on their sides with the head thrown back and kicked violently and died within half an hour. No previous symptoms were noticed.

Gross pathology. The carcasses were not examined until for at least 30-45 hours after death so there was evidence of post-mortem change. In general, the gross pathology of these lambs resembled quite closely that found in case 7426 except that there were some extensive hemorrhages on the endocardium and on the mucosal folds of the fourth stomach.

Case 7400 (Rambouillet, male)

This was a case of acute meningitis involving mostly the cerebral meninges.

History. This lamb was brought to the pathology barn on 4/8/44 together with its twin and mother. The lamb showed bilaterial stiffness in the rear legs and was somewhat emaciated. The animal did not gain and remained thin throughout the observation period of about two weeks. It never nursed vigorously. On 4/24/44, the lamb was down and kicking its legs as though it were running. It was killed when about 43 days old.

Etiology. A Staphylococcus was isolated from the brain lesion. (Table 4: culture 7400).

Blood studies.

(a) Blood picture

Erythrocytes	10,070,000 per c.mm.
Leucocytes	6,150 " "
Hemoglobin	10.3 g./100 cc.

Differential count (%)

Lymphocytes	18
Neutrophils	82
segmenters	(46)
bands	(4)
juveniles	(29)
myelocytes	(3)
Monocytes	0
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm./100 cc.)

Glucose	77.77
Uric acid	0.50
Creatinine	0.70
Chloride	380.00
N.P.N	15.00
Urea-N	9.90

The above data showed that there was a marked neutrophilia existing in this case, although the values obtained from the blood counts appeared to be normal. Some of the erythrocytes were crenated and poikilocytic, while others were more or less normal. In this particular instance, the significance of the differential count for a diagnosis was brought out.

In the blood analysis, except for the glucose value which was just a little higher than normal (Table 2), all other constituents were decidedly lower, especially the creatinine and uric acid.

Gross lesions.

Brain. The cerebrum showed some whitish areas beneath the meninges in the various sulci.

Liver. Liver showed some fatty degeneration.

Other tissues. Apparently normal.

Microscopic lesions.

Cerebrum. The cerebrum was very much congested and especially so was the cerebral meninges. The meninges were greatly swollen and infiltrated with excessive number of polymorphonuclear leucocytes and macrophages. In a number of areas the affected meninges had become detached from the cerebrum and an active suppurative process was going on. The exudate of this suppuration consisted mainly of infiltrating cells mingled with the detached meninges. The ground tissue of the cerebrum, however, was not much affected. (Fig. 48)

Liver. The liver showed some fatty degeneration of the diffuse type.

Spinal cord. The spinal cord and other tissues were more or less normal.

Other tissues. Other tissues were more or less normal.

Summary and discussion.

1. This lamb suffered from acute cerebral meningitis with some hepatic disturbance.
2. The animal was never active and never nursed vigorously during its life, which could be accounted for by the presence of cerebral meningitis.
3. In the blood studies, a state of neutrophilia was found. Such a neutrophilia was possibly related to the meningitis, whereas the high value in blood glucose but low value in blood creatinine, uric acid, N.P.N., and Urea-N, might all be explained on the basis of hepatic disturbances and inactivity.

Case 7406 (Rambouillet, male)

This was a case of acute peritonitis following the previously existing bronchopneumonia.

History. This lamb was about 45 days old. The animal had been sick about three weeks previously and was given pneumonia serum. For three days before it was killed, this lamb had been showing marked abdominal breathing. It was killed on 4/25/44.

Etiology. A species of Corynebacterium was isolated from the peritoneal fluid and its general characteristics are referred to Table 4: Culture 7406.

Blood studies.

(a) Blood picture

Erythrocytes	10,390,000	per c.mm.
Leucocytes	1,700	" "
Hemoglobin	10.3	g./100 cc.

Differential count (%)

Lymphocytes	6
Neutrophils	94
segmenters	(5)
bands	(43)
juveniles	(24)
myelocytes	(22)
Monocytes	0
Eosinophils	0
Basophils	0

(b) Blood chemistry (Mgm./100 cc.)

Glucose	200.00
Uric acid	1.44
Creatinine	1.65
Chloride	420.00
N.P.N.	21.00
Urea-N	20.70

The results of blood studies revealed that the lamb had a marked leukopenia with young neutrophilic cells (89%) overwhelming the lymphocytes (6%). The structure of erythrocytes showed marked poikilocytosis. The value of glucose was about three times higher than that of the normal (Table 2) and the creatinine was also a little high. The high value of urea-N might be due to technical error.

Gross lesions.

Lungs. Pneumonia was present in the apical, cardiac and anterior fringe of the diaphragmatic lobes. This pneumonia was of the so-called "cold" type and there was no acute progressive line of demarkation between the pneumonic area and normal tissue. The bronchi leading to the pneumonic lobes were being cleared of exudate.

Abdominal cavity. A marked peritonitis was present throughout the abdominal cavity.

Intestines. The intestines were adhered to each other with fibrin. The visceral peritoneal walls and adjacent structures were quite edematous and swollen.

Kidneys. The kidney capsule was quite edematous and black pigment was present in the pelvis of each kidney.

Other organs. No visible lesions were found in the other organs.

Microscopic lesions.

Lungs. The lungs showed lesions of a typical bronchopneumonia of several weeks duration. The bronchioles were only

slightly damaged and some of them showed evidence of metaplasia. In the alveolar walls, there was some fibroblastic proliferation going on. In general, the pneumonia of this lamb was in a beginning stage of resolution and was not very extensive, because it was confined to certain small spots.

Intestines. A marked and extensive peritonitis existed on the intestines with much involvement of the serosa and less so for the muscular coat. The serosa was greatly congested, thickened, and became adhered to the mesentery. (Fig. 49) These affected areas were infiltrated by polymorphonuclear leucocytes. The muscular coat of the intestine was also thickened and somewhat swollen. The submucosa and mucosa, however, were not much affected, although some serous exudate was seen in the intestinal lumen just next to the mucosa.

Liver. The liver was rather congested, and a rather marked cloudy swelling and some fatty degeneration were present. Along the surface of liver, an active inflammatory process and necrosis were in progress, the liver cells were destroyed and were being replaced by cell infiltration and fibrosis. Some of the necrotic processes extended deeply into the parenchyma of the liver and varied in size from pin-point areas to those about 0.3-0.5 mm. in diameter. (Fig. 50)

Kidneys. The capsule of kidneys was severely inflamed and very much thickened. The inflammatory exudate was more or

less organized and mixed with the adipose tissue and fibrin so that the capsule became tightly adhered to the kidney on one hand and the peritoneal membrane on the other. The thickened capsule together with its organized inflammatory exudate measured about 4 - 5 mm. in some parts. The parenchyma of the kidneys, however, seemed to show little or no involvement, although there was some cloudy swelling in the epithelial cells of the convoluted tubules.

Other organs. Other organs were normal.

Summary and discussion.

1. This animal suffered from an acute peritonitis with complication of hepatic disorders and resoluting pneumonia.
2. The presence of bronchopneumonia and acute peritonitis were responsible for the leukopenic neutrophilia. Again, it was due to these conditions that the animal showed dyspnea at first and marked abdominal breathing later on while it was still alive.
3. The high value in blood glucose and creatinine was directly related to hepatic disturbances as the liver was rather severely inflamed and necrotic, especially along its entire surface.
4. The Corynebacterium sp. isolated from the peritoneal fluid was pathogenic for a mouse and caused the same diseased condition (acute peritonitis) when the organism was injected intr-peritoneally.

Case 7336 (Shropshire, female)

This lamb suffered from acute hemorrhagic enteritis together with some passive congestion.

History. This lamb was born at 8:00 A.M. 3/24/44 and died at 8:00 A.M. the next day. Shortly before death, the animal was kicking violently and trembling.

Etiology. A species of Escherichia was isolated from the liver, heart blood, and spleen. The general characteristics of this micro-organism are referred to in Table 4: culture No. 7336.

Gross lesions. A complete autopsy was performed, but no lesions were found, except that the intestines were very much congested and an acute enteritis was suspected.

Microscopic lesions.

Liver. The liver was rather congested and showed some small hemorrhages. The hepatic cells showed evidence of cloudy swelling and some of them exhibited a slight degree of fatty degeneration.

Spleen. There were hemorrhages in the spleen pulp and a moderate congestion of the serosal capillaries. In the areas of hemorrhage, the lymphoid tissue was largely replaced by red blood corpuscles and these areas appeared deep red in color with hematoxylin-eosin stain in contrast to the light yellow color of the normal portion of the spleen. In these affected areas, the Malpighian corpuscles were also involved. There were only a few

lymphoid cells left in each of the corpuscles and the central arterioles were greatly enlarged and distended with blood.

Small intestine. Typical lesions of hemorrhagic enteritis were found in the small intestine. The mucosa was severely damaged and a greater part of the epithelium was necrotic and desquamated. The capillaries were involved and hemorrhage resulted. In the lumen of intestine, an inflammatory exudate consisting of desquamated epithelial cells, blood cells of various types, and a small amount of fibrin was present. The submucosa was also affected, and it showed marked leucocytic infiltration and congestion. The muscular coats and the serosa, however, were not affected, although some congestion was noticed in both. (Fig. 51)

Kidneys. The kidneys showed evidence of some passive hyperemia, otherwise they were more or less in a normal condition.

Other organs. Those other tissues included in this study were all normal.

Case 7364 (Hampshire, male)

This lamb suffered from acute enteritis.

History. The lamb was born on 3/16/44 and was found dead a few hours later. No visible symptoms were observed.

Gross pathology. There was a marked enteritis extending approximately two feet from the pylorus of the stomach to the rectum. The mucosa of the entire gut was congested and the Peyer's patches stood out distinctly. The contents of the intestinal tract were liquid.

There was approximately 100 cc. of the peritoneal fluid present. This fluid was slightly blood tinged.

No other visible lesions were observed besides those mentioned above.

Bacterial culturing. A hemolytic gram-positive anaerobic rod was isolated from the intestinal contents.

Case 7433 (Southdown, female)

This was a case of gastric ulcers and perforations.

History. The lamb was about two months old. It died during the night of 5/8/44 without showing any previous symptoms.

Etiology. Undetermined.

Gross lesions. Upon skinning the lamb, a well nourished carcass was found. A complete autopsy was done, including brain examination. The only lesions observed were perforation of the stomach and two deep ulcers at the pylorus.

Microscopic lesions. The stomach mucosa was found very much congested. The mucosal epithelium showed a slight degree of necrosis on the surface. This slight necrosis may have been due to post-mortem changes. In the ulcerated areas, however, the entire epithelium together with the gastric glands was severely damaged and had undergone varying degrees of necrosis. As the necrotic foci extended deeper and deeper, the muscularis mucosae and submucosa were also involved. At the same time, marked polymorphonuclear leucocytic infiltration occurred. As the lesion extended to the submucosa, it spread sidewise more rapidly than toward the serosa so that a greater part of submucosa was affected. The blood vessels in this area were damaged, resulting in hemorrhage, the connective tissue was called into action, and an active fibrosis was present. As a result, the necrosis (as in the case of gastric ulcer in man) stopped at the submucosa, but the

fibrosis extended outward deep into the muscular layers where most of the muscular bundles were pushed far apart and a state of fibrous hyperplasia existed. (Fig. 52)

Case 7440 (Black Top Delaine, female)

This was a case of starvation and dietary deficiency.

History. This lamb was noticed to be inactive and the abdomen was quite distended. It looked like a "skim-milk fed" calf. The animal died in the morning of 5/10/44 when 50 days of age.

Gross pathology.

Stomach. The rumen was filled with fibrous roughage but the fourth stomach was empty except for a small hair-ball.

Kidneys. The left kidney was slightly larger than the right one and the pelvis of this kidney was slightly distended.

Bones. The skeletal bones of this animal were smaller than those of normal well-fed lambs of the same age. The ribs were easily broken.

Culture. Negative.

Case 7537 (Rambouillet, male)

This was also a case of nutritional deficiency, somewhat similar to case 7440.

History. This animal was noticed "going light" and showed some paralysis of the limbs for about one week before death, which occurred about three months of age.

Gross pathology. The lamb showed very little fat on the carcass. The skeletal muscles were small and not well developed. The bones were small, easily bent, and cut easily with a scissors. No other visible lesions were observed, and the bacterial culture was negative.

Case 7326 (Rambouillet, male)

This was a case of failure to develop anal and urethral openings.

History. The lamb was thought to be bloated on 3/17/44, but upon examination it was found to have no anal opening or urethral opening. An operation was performed which provided for an artificial anal opening but the urethra could not be found. The lamb died during the night of 3/18/44 and was autopsied the next morning.

Gross pathology

Intestinal tract. The intestinal tract posterior to the stomach was greatly distended with gelatinous fecal material. Although much of the material had passed out, a considerable amount was still retained. A marked atony and dilatation of the colon and rectum was noticed.

Bladder & genital organs. The urinary bladder and genital organs were normal, but the urethra was blind due to lack of development of the penis.

Other organs. No other gross lesions observed.

Case 7352 (Rambouillet, female)

This lamb suffered from a patent foramen ovale.

History. The animal was one of a set of twins born on 3/28/44 and was found dead a few hours later without showing any noticeable symptoms.

Gross lesions.

Abdominal cavity. Upon opening the abdominal cavity, the blood vessels were found to be markedly distended which indicated passive congestion.

Liver. The liver was increased in size and showed congestion.

Small intestine. The Peyer's patches were quite conspicuous and surrounded by engorged blood vessels.

Heart. The foramen ovale was open, and an ecchymotic hemorrhage was located in the region of one of the semilunar valves.

Other organs. Apparently normal.

Culture. Negative.

Case 7392 (Oxford, male)

This was a case of ruptured liver.

History. The lamb was born dead on 4/20/44.

Gross pathology.

Lungs. The lungs contained no air, indicating that the animal was born and had never breathed.

Liver. The right side of the liver was ruptured, probably due to abnormal passage during the act of birth.

Other organs. Apparently normal.

Culture. Negative.

In the preceding pages, a total of 42 cases of diseases of the new-born lambs has been presented. Besides these 42 cases, there were 13 more cases whose diagnoses could not be made due either to lack of symptoms or to insufficient pathological findings. No bacteria were isolated from the internal organs. To furnish some information as a basis for further investigations, the 13 undetermined cases are presented herewith in Table (6).

Table (6)

Cases of diseases of the new-born lambs with undetermined diagnosis.

Case No.	History of the case	Gross pathology
7303	Southdown, female. Born on 3/7/44 and lived only for about one hour. It was bloated when born and remained so during life.	Fourth stomach and accessary stomach were distended with gas. There seemed to be no stricture either in the inlet or outlet of the stomach, and no motile forms or bacteria were observed upon microscopic examination of the stomach contents. All other organs were apparently normal. Culture--Negative.
7304	Southdown, female. The lamb had the same history as case 7303 but less bloat in the stomach.	Nothing abnormal.
7309	Oxford, male. This lamb was about two days old. It showed no symptoms and was the most vigorous of the triplet lambs. It died on 3/9/44.	No lesions. Culture--Negative.

Table (6) (Cont'd)

Case No.	History of the case	Gross pathology
7310	Hampshire, female. This animal was one of a set of twins which was born on 3/7/44. The lamb appeared weak and died at the age of three days.	Except some particles of fibrin on the surface of liver, no other lesions were found.
7311	Hampshire, male, about 3 days old. The lamb was noticed breathing heavily and pneumonia was suspected. It was then treated with pneumonia serum and saline and glucose (5%). It died on 3/10/44.	Extensive subcutaneous hemorrhages were found in the vicinity of both hind legs, and marked hemorrhage was found in the gastrocnemius muscles. The ductus arteriosus showed a small opening. Other organs were apparently normal. Culture--Negative.
7321	Southdown, male, about 24 hours old. This lamb was born bloated on 3/12/44 and lived only for a few hours. It had a history similar to cases 7303-'04	Subcutaneous hemorrhages were present along the left side of the back line from the shoulder to the tail region. No food was present in the stomach. The stomach was distended with gas to the point where it reached the pubis. No other visible lesions were found.
7322	Oxford, female. This lamb was weak and had been given 90 cc. of saline and glucose (5%) previous to death. It was found dead on 3/14/44 when six days old.	No visible lesions were observed. Culture--Negative.

Table (6) (Cont'd)

Case No.	History of the case	Gross pathology
7325	Southdown, male. This lamb was born at 5:00 A.M., 3/15/44. It was stiff at birth and made no effort to move. 80 cc. of saline glucose (5%) were given subcutaneously and 4 oz. of colostral milk from the ewe orally. The lamb died when it was about two days old.	No visible lesions were found except a slight edema of the stomach wall. Culture--Negative.
7354	Rambouillet, female. This lamb was born on 3/14/44 and was graded as 1-A at birth. Without any noticeable symptoms, the animal died at the age of about 20 days.	No gross lesions were observed. Culture--Negative.
7367	Shropshire, male. The lamb was born as one of a pair of twins on 4/9/44 and was found dead shortly after birth.	Spleen. The spleen showed many petechial hemorrhages under the capsule. Heart. One petechial hemorrhage was found under the epicardium. The ductus arteriosus was open as would be expected. Lungs. Only partly expanded, indicating that the lamb lived for a very short time. Stomach. A few petechial hemorrhages were present on the folds of stomach. Other organs. Apparently normal. Culture--Negative.
7388	Shropshire, female. This lamb was about five days old and died on 4/18/44 without any noticeable symptoms.	No other visible lesions were observed except a few subcutaneous hemorrhages in the vicinity of the tailhead. Culture--Negative.

Table (6) (Cont'd)

Case No.	History of the case	Gross pathology
7283	Hampshire, male, about 3 days old. The lamb became droopy on the afternoon of 3/1/44 and died the next morning.	No gross lesions were found. Culture--Negative.
7284	Shropshire, female, about 24 hours old. This lamb was one of a set of triplets. It was a small lamb and died on 3/2/44 without any noticeable symptoms.	No lesions of the internal organs were observed. Culture--Negative.

IV. Discussion of Results

Fifty-five cases of new-born lambs affected with a variety of diseases were included in this study and the problem was attacked hematologically, bacteriologically, and pathologically.

The hematological investigation consisted of studying the blood from both normal sheep and pathological lambs. The blood values obtained from normal sheep served as a control. The results are briefly summarized in Tables (1, 2, 3) and discussed in detail in connection with the individual cases. For the sake of comparison, the results recorded by a number of other workers such as Allcroft[1], Fraser[29], Holman[45], Josland[45], Norris and Chamberlin[75], and Shearer and Stewart[91] were consulted. In general, the results of blood studies on normal sheep compare fairly well with those of other workers except some minor differences. Norris and Chamberlin found the total average of white blood corpuscles for lambs to be 5,500 per c.mm. as compared to 7,979 per c.mm. for our normal lambs. The neutrophils were 52 per cent as compared to our finding of 30 per cent in sheep. The blood glucose values for sheep were high (75 mgm.% as compared to our 48.48 mgm.%). In Fraser's work, the neutrophils of lambs were high (54% as compared to our record of 28%). This may be due to a lack of uniformity among thevarious workers, in the technics used, and to different environmental conditions. Regarding the study of blood in diseased lambs, no previous references were available for making a comparison. Work of this kind is comparatively new to the field of veterinary medicine.

In the bacteriological studies, 41 cultures of aerobes and 7 anaerobes were isolated from various cases. By means of morphological, biochemical, and pathogenicity tests, these cultures were identified and grouped under 9 genera. They were: Staphylococcus, Diplococcus, Neisseria, Escherichia, Bacterium, Pasteurella, Corynebacterium, Actinomyces and Clostridium. Two cultures were undetermined. Bacterium, Staphylococcus, and Pasteurella were most frequently encountered, while Neisseria and the undetermined cultures were encountered the least number of times. (Table 4). Twenty (48.78%) of the 41 original aerobic cultures were recovered from inoculated mice, 13 (31.7%) of which were definitely virulent. Rabbits were found to be quite resistant to the cultures isolated from lambs and were not suitable for pathogenicity tests.

The pathological and bacteriological studies revealed 15 different types of diseases among the 55 cases. They were: White-muscle disease, staphylococcic infection, navel infection, docking infection, castration wound, bronchopneumonia, pasteurellosis, enterotoxemia, meningitis, peritonitis, enteritis, gastric ulcers and perforation, dietary deficiency, congenital anomalies, and an undetermined group. Most of these diseases were studied more or less in detail, and the following diseases or conditions such as congenital interstitial bronchopneumonia, patent ductus arteriosus, patent foramen ovale, gastric ulcers and perforation, and

encephalomalacia are reported for the first time. The finding of muscle giant cells in the cases of white-muscle disease and their significance in myo-regeneration are considered new to medical science and of vital importance in explaining the pathological phenomenon involved. In the following paragraphs, a brief discussion of some of the more important cases is given.

1. Congenital interstitial bronchopneumonia (Case 7329)

This case was found in a lamb born on 3/21/44 and found dead at 7:00 A.M. the same day. Apparently, the pneumonic condition must have developed during intra-uterine life. The histopathology resembled quite closely that found in human cases reported by MacCallum[52] in 1919 and Sprunt[96] in 1938. It was also somewhat similar to the so-called "progressive pneumonia" in sheep as described by Marsh[57,58] in 1923 and reported by the Montana Station[71] in 1937. This finding would indicate that more intensive studies should be made on the etiology and epidemiology of lamb pneumonia.

2. Patent ductus arteriosus (Case 7308)

This was found in a lamb born on 2/29/44 and died on 3/9/44. The patency of ductus arteriosus was complicated by bronchopneumonia and a species of Bacterium was isolated from the lungs. Ordinarily, as reported by Barclay, Barcroft, and Barron[5], the closure of ductus arteriosus and foramen ovale in lambs occurs in a few minutes after birth. Since this particular duct was not closed, the lamb

suffered rather severely from passive hyperemia and pneumonia. In human cases of patent ductus arteriosus, Wilson and Lubschez[113] (1942) considered it as insignificant in children (mortality rate 0.38%), while Keys and Shapiro[48] (1943) regarded it as a predisposing factor to various diseases such as subacute bacterial endocarditis, congestive failure, cardiac hypertrophy, and pulmonary dilation.

3. Encephalitis or probably listerellosis (Case 7415)

This was a case of bronchopneumonia complicated by encephalitis and enteritis, but the condition of encephalitis is of special interest to the writer. From microscopic examination, the lesions in the thalmus (so-called "pericascular cuffings") were somewhat similar to those found in cases of ovine listerellosis reported by Gill[31], Jungherr[47], Biester and Schwarte[8], Graham et al[34], Pallaske[81], and Pomeroy et al[83]. The brain was not cultured in this particular case, therefore the etiological agent was not determined.

4. Bronchopneumonia (15 cases)

Bronchopneumonia is common in new-born lambs. During the lambing season last year (1944), about 15 cases of bronchopneumonia (either with or without complication) were encountered, and most of them were of the acute type. Quite a variety of micro-organisms were isolated from the affected lungs, such as: Actinomyces necrophorus, Bacterium, Diplococcus, Escherichia, Neisseria, Pasteurella, and Staphylococcus. These findings agree quite well with those of Spray[95], Mettam[63], Delpy[22], and Koshelev[49], although

Actinomyces necrophorus is reported here for the first time as one of the causal agents in lamb pneumonia. Taking the results of the present and previous investigations, it seems that the causal agents of lamb pneumonia are variable and not specific.

5. Navel, docking, and castration infections.

These three different types of infection are discussed together, because they are more or less artificially produced by unsanitary technics, improper disinfection of the navel and insanitary surroundings. In the lambing season last year (1944), 5 cases of navel infection, 2 cases of docking infection, and 1 case of castration infection were encountered. The causal organisms (Actinomyces necrophorus, Bacterium, Corynebacterium, Coccoid bacillus and Staphylococcus), like in pneumonia, were also variable and not specific. These diseases were mentioned and discussed by many workers in the field of veterinary research long ago, but they still remain a great problem to the sheep industry. In order to prevent such losses, it seems necessary to pay more attention to aseptic surgical operations, disinfection and environment.

6. Peritonitis (7406)

Cases of peritonitis in lambs were briefly discussed by Craig and Bitting[21] in 1902 and by Gallagher[30] in 1921. No causal agents were mentioned. In the present investigation, the case of acute peritonitis (case 7406) needs special attention. The causal agent was a species of pleo-morphic

gram-positive diphteroid (Corynebacterium) and the typical lesions were beautifully presented.

7. Pasteurellosis (Cases 7334 & 7335)

Six cultures of Pasteurella were isolated from various organs of pathological lambs, but only two cases (7334 & 7335) were considered to be the typical pasteurellosis. These two cases were found in a pair of twin lambs born on 3/20/44 and which died the next day. The disease was characterized as an acute bacteremia and blotchy hemorrhages were found in subcutaneous tissue and muscles in both cases. Since Pasteurellosis was reported by Caze[18], occurring as an epizootic among Algerian sheep, this gives the writer a clear-cut impression that Pasteurellosis in new-born lambs needs special attention.

8. Staphylococcic infection.

Two cases (7337-38) staphylococcic infection were found during the course of this study. The disease was characterized by acute septicemia associated with metastatic necrotic foci and abscess-formation which agrees quite closely with the condition reported by Bullard[16] (1934).

9. Enterotoxemia (Cases 7399, 7426, 7434, 7435, 7436)

Five cases of enterotoxemia were encountered during the course of this study, which indicates that young lambs have the same condition as seen in older sheep and feed lot lambs, although the feed and environment are different.

10. Undetermined cases.

A group of 13 undetermined cases was found during the

course of this study. The cause of death in these cases was not determined and no gross lesions were observed. This group presents a challenge to experimental ingenuity.

11. White-muscle disease (Cases 7356-58)

Two cases (7356-58) of white-muscle disease were encountered during the course of this study. One (Case 7358) of these two cases was complicated by bacteremia leading to abscess-formation and arthritis, while the other (Case 7356) was not. In the complicated case, three different kinds of bacteria (a Staphylococcus, an undetermined gram-positive coccus and a Diplococcus) were isolated, whereas in the uncomplicated or typical case no bacterial forms were found. Apparently, a typical (or uncomplicated) case of white-muscle disease is not caused by any bacterial infection, and that agrees very well with the work of the New York (Cornell) Station[64, 73, 105] and the Oregon Station[79]. On the other hand, Jungherr and Welch[46] found coccidia in cases of "stiff-lamb" disease, Heath[38] found an acid-fast bacillus in a similar diseased condition, while Lee & Scrivner[50] isolated 3 kinds of bacteria (bipolar rods, cocci, and non-spore forming rods) from lambs with this malady. The results of the present investigation as illustrated by Case 7358 clearly indicate that this latter group of workers were actually dealing with white-muscle disease complicated by some other infection. The blood studies on white-muscle disease revealed high values in all six blood constitutions

studied which agreed rather closely, except for minor differences, with the findings of Anderson[3] and Sholl[92].

In those two cases (7356-58) studied and presented above, no cardiac involvements were found. Just recently, however, the writer had an opportunity to study a case of white-muscle disease in a young lamb (Case 6598) from our department collection. The pathological processes in the heart followed rather closely those found in the skeletal muscle but more mitotic figures were noted in the regenerating muscle cells (Fig. 53) and smaller muscle giant cells were seen (Fig. 54). The cell with the mitotic figure or with the so-called "peculiar nuclear structure" found in the heart was first described by Anitschkow in 1913 and was called the "Anitschkow myocyte"[19a]. Anitschkow believed that these cells originated in the muscle fibers of the heart but this observation was disregarded by Ehrlich and Lapan[19a] in 1939 and Clawson and Downey[19a] in 1941. Ehrlich and Lapan were convinced that this cell arose from the interstitial tissues of the heart and called it a "myocardial reticulocyte" while Clawson and Downey thought it should be called a "cardiac histiocyte" since the term "reticulocyte" is applied to a red blood cell. Since then, it has been generally agreed that it develops from cardiac interstitial tissue and not from the myocardial muscular fibers. Boyd[13] described this cell as "a cardiac histiocyte which in inflammation shows increased cytoplasm, a highly characteristic serrated bar of chromatin in the center of

the nucleus, and fibrils radiating from the bar to the periphery." Apparently, Boyd did not notice that the "peculiar nuclear structure" was nothing more than a beautiful mitotic figure (probably in the metaphase stage) in an actively-proliferating regenerating muscle cell in the heart. In fact, cells of such kind, in the heart, as pointed out by Ehrlich and Lapan[19a], were not only frequently observed in rheumatic fever but also in a number of pathologic states, such as sepsis, meningococcic bacteremia, scarlet fever, pericarditis nodosa, and subacute bacterial endocarditis. Undoubtedly, certain regenerating cardiac muscle cells with mitotic figures may be expected under those conditions.

Another interesting but important point should be mentioned here in regard to regeneration of heart muscle. Boyd[13] stated that "plain muscle and heart muscle fibers do not regenerate; union of the divided parts is by scar tissue". From the results of present investigation it seems that the heart muscle has the ability to regenerate at least under certain conditions (white-muscle disease in lamb case 6598). Moreover, since the so-called "Anitschkow myocytes were observed in a number of pathologic conditions of the heart by Ehrlich and Lapan[19a] and in syphilitic myocarditis (gumma of heart) as reported by many other workers[13,28,76], further evidence was furnished to substantiate the fact that cardiac muscle might also regenerate under some of these conditions.

The histo-pathology of white-muscle disease in lambs was briefly worked out first by Metzger and Hagan[64] in 1927. They mentioned four main points, namely, (1) whitish streaks or patches as the gross lesion in the muscle; (2) it is degenerative and not inflammatory; (3) practically no cell infiltration by polymorphonuclear or other wandering cells; (4) occasionally a phagocytic cell was found. The results of the present investigation agree in certain respects with their findings but not in all. Apparently, Metzger and Hagan did not notice muscle giant cells nor the proliferative activities of the regenerating muscle cells which are so vital and significant to the pathological processes of this particular disease. Following Metzger and Hagan, quite a number of research workers, Jungherr and Welch[46], members of the Veterinary Research Laboratory of Oregon Station[77], Heath[38], Marsh[60], Slagsvold and Lund-Larson[93], Lee and Scrivner[50], Scholfield[86], Salyi[85], and Willman et al[105-111], were actively engaged in studying the pathological aspect of the disease. Up to this moment, little advance has been made toward the solution of the problem, and confusion still exists. It seems rather strange that not a single worker has described the microscopic lesions in the heart.

To state briefly, the histo-pathology of white-muscle disease involves myodegeneration followed by myo-regeneration. In the degenerative changes, the important feature is hyalination of the affected muscle fibers together with some

fibroblastic activity, while in the regenerative process, the nuclear proliferation from the unaffected muscle fibers or from the survived sarcolemma lead to the formation of muscle giant cells and regenerating muscle fibers. The function of the muscle giant cells is to remove the dead tissues while that of regenerating muscle fibers is to fill the gaps left by damaged tissue. If the damage was so severe that the affected muscular nuclei were dead, fibroblastic proliferation and subsequent fibrosis would take place and the lesion would be filled with fibrous and adipose tissue. In the heart, the lesions are somewhat similar to those found in the skeletal muscle but more mitotic figures in the regenerating muscle fibers and smaller giant cells were observed.

Regarding the subject of "myodegeneration and myoregeneration" in general, it was noticed that many workers in the field of pathology have been actively engaged in such a study. As early as 1862, Virchow[101] was able to find the so-called "repair clubs of striped muscle" in regenerating muscle. He mentioned that these repair clubs closely resembled myeloplaxes. In 1899, Lejars[51] and his friend Pillet described tuberculosis of the muscle with formation of multinucleated giant-cells from the striated fibers. Since their description was brief and poorly illustrated (hand drawing), their work has long been disregarded. Moreover, Lejars and his friend did not appreciate the significance of the giant cell in tuberculosis muscle, because they did not know the exact function of the cell and process of its

formation. In 1901, Marchand[37] mentioned such "repair clubs of striped muscles" and included them among the source of foreign body giant cells.

Mallory[55], 1914, explained the various appearances of injured striped muscle and stated that "when the whole muscle fiber is killed, it is not regenerated. When only a part of one of the fibers is destroyed, active regeneration of the part which has not undergone necrosis takes place from the part which remains uninjured. The intact nuclei undergo rapid direct division and each forms several dozen separate nuclei.....The new nuclei migrate to the periphery and the injured end of the muscle-fiber.....The process of regeneration of muscle-fibers is complicated and made confusing by the presence of numerous leucocytes attracted by the injurious agent and by the injured tissue. Sometimes the necrotic muscle is removed through the action of polymorphonuclear leukocytes, which usually invade the fibers; .....but at other times only endothelial leukocytes are present. They attack necrotic muscle substance from the periphery and gradually dissolve it, working slowly from without inward. Mitoses in these endothelial leucocytes are not infrequent. Occasionally, multiple mitoses occur from which multinucleated cells result. More often some of the endothelial leucocytes fuse to form foreign body giant-cells. Sometimes the two processes of regeneration and of removal of the necrotic muscle-tissue of the same muscle-fiber overlap and render the picture difficult to interpret".

According to Mallory, myo-regeneration was apparently possible and multinucleated giant cells did exist. These two points agree very well with our findings. However, his observation on the removal of dead muscle tissue by polymorphonuclear leucocytes or endothelial leucocytes, and the formation of multinucleated giant-cells from endothelial leucocytes was quite contrary to our findings and was very misleading.

In 1926, Forbus[26] investigated the degeneration and regeneration of the rectus abdominis in pneumonia. He pointed out that following degeneration of the muscle (i.e. hyaline degeneration), regenerative changes occurred which led to the total restoration of the affected muscle. This regenerative process was described in detail, the essential of which was as follows: "The hyaline contractile substance is removed through the activity of mononuclear wandering phagocytic cells which are of extramuscular origin.---The muscle nuclei of the old fiber proliferate and produce new muscle cells, their cytoplasm being derived from the sarcoplasm of the old partially preserved fiber. From those muscle cells, the new fiber develops, either from growth of the individual cell or from fusion of several cells". The findings in white-muscle disease agree in most respects with the work of Forbus except for the origin of the phagocytic cells found in the process of myo-regeneration and the fusion of several cells to form new fiber. According to our findings, the phagocytic cells (the muscle giant cells) are of muscular origin and Forbus' fused-cells are in reality

multinucleated muscle giant cells.

In the same year (1926), Forbus[27] conducted an experimental study on myodegeneration and myoregeneration using vital stains. As a result, he confirmed his previous work and stated that "the phagocytic cells found within the persistent sarcolemma, which together with the muscle cells derived from the muscle nuclei form the "Muskelzellenschlauche" of Waldeyer, are wandering cells of extra-muscular origin and have no connection with the muscle cells", although he mentioned that "muscle cells as well as phagocytic cells of the "Muskelzellenschlauche" are capable of being stained by intravenous injection of vital dyes (carmine or trypan blue) during their development, and hence such vital staining alone is insufficient for differentiating between cells of muscle origin and cells of extramuscular origin". Unfortunately, Forbus did not use Mallory's anilin blue stain in studying the preserved tissues, otherwise he would have realized that the muscle giant cells and the regenerating muscle cells would have exhibited the same staining affinity and some nuclei of these two newly-formed cells would appear exactly the same. This would have indicated clearly the muscular but not extra-muscular origin of the muscle giant cells.

Haythorn[37], in 1929, published an extensive review on the subject of "multinucleated giant cells". He mentioned the term "Muscle Giant Cells" but hesitated to believe that they existed. He was of the opinion that "while muscle repair clubs may resemble foreign body giant cells, it is

doubtful if they can take part in their formation in any capacity other that that of a foreign body which attracts them". In fact, he agreed with Mallory and Forbus and considered the so-called "foreign giant cells" in myo-regeneration as of extra-muscular origin.

In 1931, Goettsch and Pappenheimer[33] reported their results on the study of nutritional muscular dystrophy in the guinea pig and raboit. They were able to find the same processes of myodegeneration followed by myo-regeneration but agreed entirely with Forbus in respect to the origin of the so-called invading "phagocytic histiocytes". They also found large multinucleated plasmatic masses, lying against the necrotic remains of the muscle substance. In fact, according to our interpretation, the large multinucleated plasmatic masses are nothing more than muscle giant cells and the so-called "phagocytic histiocytes" are most likely the developing muscle giant cells.

The following year (1932 a, b), Fishback and Fishback[24,25] made an experimental study on muscle degeneration by using different types of trauma. They found that a moderate diffuse infiltration of polymorphonuclear leucocytes and lymphocytes was induced within 24 hours after injury but such infiltration began to fade at 72 hours. At the end of five days there were no inflammatory cells left. Seventy-two hours after injury, phagocytic cells containing debris appeared, and at the end of five days, marked early muscle cell proliferation occurred around the margin of patches of the fiber

degeneration. There were numerous irregular single cells with fibrillar extensions, and syncytial sprouts with multiple nuclei. On the 18th and 21st days a few areas of almost normal sized new fibers were found. The so-called "syncytial sprouts" with multiple nuclei, according to our explanation, are probably the muscle giant cells.

Two years later (1934), Pappenheimer and Goettsch worked on the problem, "Nutritional myopathy in ducklings". The myopathy was induced by a special diet consisting of skimmed milk powder, casein, corn-starch, lard, cod liver oil, yeast, salts, and paper pulp. The pathological changes were found in the skeletal muscles as hyaline or waxy degeneration. In those cases which survived longer, there was an extreme cellular response in which myoblasts, histiocytes, and leucocytes of different types took place. Occasionally, basophilic regenerating myocytes were present.

Madsen, et al[54], in 1935, published work on "Nutritional myodegeneration of herbivora fed normal and synthetic rations". A myo-degeneration was found in the lesions and an active myophagocytosis was observed to occur shortly after injury. They did not mentioned, however, the type of cells responsible for myophagocytosis.

In 1943, Boyd[13] discussed giant cells and stated "when the individual macrophages are unable to deal with particles to be removed, they fuse together to form multinucleated giant cells.---Such a foreign body giant cell is a cytoplasm; a syncytium containing a large number of nuclei. In this

form, it seems to have greater phagocytic power than when the cells act singly". The writer agrees with Boyd's interpretation regarding the function of giant cells and partly to the formation of giant cells, but not with his idea as to the origin of muscle giant cells. Boyd also mentioned that "some giant cells are formed by amitotic division of the nucleus" which is apparently not found in the muscle giant cells. Again in his book, Boyd stated that "Healing in the muscle depends on the kinds of muscle. Plain muscle and heart muscle fibers do not regenerate; union of the divided parts is by scar tissue. Striated muscle has greater reparative power. The extent of regeneration depends on the type of injury. In an incised wound with no loss of substance, the nuclei of the sarcolemma proliferate and form multinucleated syncytial masses while the sarcous substance puts out bands which bridge the gap and eventually become striated. On the other hand, when the muscle fibers have been destroyed, the debris is removed by mononuclear phagocytes, which may form giant cells, and the gap is closed by fibrous tissues". The writer agrees in certain respects with Boyd's statement as to the processes involved in myo-regeneration but does not agree with his idea of myophagocytosis by mononuclear phagocytes nor to the origin of this particular group of giant cells from mononuclear phagocytes. In fact, Boyd's "multinucleated syncytial mass" should be regarded as one of the muscular giant cells.

In the same year (1943), Forbus[28], in dealing with Aschoff cells, stated: "The nature and the origin of the curious single and multinucleated giant cells which are so characteristic of the typical Aschoff body has been a subject of long discussion. These cells have been described very carefully by a number of workers. The earliest was Aschoff and he thought it most likely that they arise from what he called the adventitial cells of the blood vessels. Later, Geipel expressed the opinion that they are probably derived from ordinary connective tissue cells. Since the early descriptions, opinion as to the nature of these cells have varied, as indicated by the many names given to them, for example, fibroblasts, connective tissue cells, endothelial cells, epithelioid cells, muscle cells, and so forth. The recent studies by McEven, in which modern, supravital, cytological techniques were used, seem to show that the Aschoff cells most likely arise from undifferentiated mesenchymal elements of loose connective tissue. In line with these observations, it would seem most likely that the development of the peculiar Aschoff cells constitutes simply a part, and yet a rather peculiar part, of the healing reaction of the connective tissue." Apparently, no one has been able to ascertain the exact nature and origin of the so-called "Aschoff cells". Referring to the study on white muscle disease of new-born lambs, it was observed that the muscle giant cells in the heart were smaller than those found in the skeletal muscle but possessed all the

characteristics of the so-called "Aschoff cells". This leads the writer to believe that the so-called "Aschoff cells" are probably nothing more than cardiac muscle giant cells and the "Aschoff bodies" in myocardium are only masses of tissue consisting of regenerating cardiac muscle cells and cardiac muscle giant cells together with a few fibroblasts, occasional plasma cells, lymphocytes, and polymorphonuclear leucocytes. Further investigation is needed in order to clear up this situation.

In the same year (1943), Ogilvie[76] mentioned gumma of heart (syphilitic myocarditis) and stressed the existence of giant cells. He stated: "Here and there, a giant cell is present. But, giant cells are, as a rule, less numerous in a gumma than they are in a tuberculoma. Further, while having the same irregular shape and granular, acidophilic cytoplasm, they are generally smaller and contain fewer nuclei". Under such conditions, the cardiac muscle giant cells might also exist. Further investigation might reveal this fact.

In the discussion of foreign-body giant cells, Ogilvie stated: "They are known as foreign-body giant cells, because they are frequently found in relation to foreign bodies such as cat-gut or vegetable-matter embedded in the tissues. They also occur in the neighborhood of pathological deposits which are difficult to absorb such as urates, inspissated secretions, fat and keratin, and other features in tissues chronically inflamed by bacterial infection.....Some of them contain cell-debris or fat and it is likely that their

main capacity is, indeed, that of phagocytosis, but apart from this their function is obscure. Nor is their origin clear. Probably most are derivatives of histiocytes, others of fibroblasts. And it would appear that the commonest mode of formation is repeated amitotic division of the nucleus without division of the cytoplasm, though sometimes fusion of cells is possibly the method". Apparently, Ogilvie had little concrete evidence to support his statement regarding the origin, function and mode of formation of the so-called "foreign-body giant cells". Perhaps the evidence obtained from our finding the muscle giant cells in white-muscle disease of young lambs may serve as a basis for further investigation of other kinds of giant cells.

In a brief discussion of the subject on "myodegeneration myo-regeneration" as presented in the foregoing paragraphs, it seems quite possible that the pathological phenomena observed in the cases of white muscle disease of new-born lambs may serve as a guiding principle in the study of a number of other diseased conditions involving muscular disturbances, such as tuberculosis, pneumonia, typhoid fever, rheumatic fever, syphilitic myocarditis, scarlet fever, and etc. In these muscular disturbances involving myodegeneration and myo-regeneration, the tissue reaction to injury would follow in one way or another the same pathological pattern as described in cases of white-muscle disease. The fancy term "Muskelzellenschlauche" of Waldeyer is in reality a mass of regenerating skeletal muscle cells and skeletal

muscle giant cellstogether with a few fibroblasts. The so-called "Aschoff body" in the myocardium is most likely a group of regenerating cardiac muscle cells, cardiac muscle giant-cells and occasionally a few lymphocytes, plasma cells, and polymorphonuclear leucocytes. The "Anitschkow myocyte" is considered to be a regenerating cardiac muscle cell with an active mitotic figure, while the "Aschoff cell" is probably nothing more than a cardiac muscle giant cell. Furthermore, the terms such as "repair clubs of muscle", "multinucleated syncytial masses", "multinucleated plasmatic masses", "syncytial sprouts with multiple nuclei", "Cytoplasmic syncytium with a large number of nuclei", and "multinucleated cells in muscle" are actually designations for muscle giant cells.

In addition, the writer feels that in any muscular disburbances, the fundamental pathological processes involved can be briefly stated as follows:

(1). If a strong etiological agent is met and the injury is severe, the tissue reaction would follow an inflammatory process. The affected muscle fibers would undergo various phases of necrosis and an active infiltration of polymorphonuclear leucocytes and other wandering cells (macrophages and later on lymphocytes or plasma cells) would be induced and ausecessation may result. If the animal survives or lives longer, repair could take place and the lesions would be filled with scar tissue. No myo-regeneration is possible under such conditions. In short, the whole process may be

designated as "myo-inflammation and scar-tissue repair". In a very rare instance, this process was observed in white-muscle disease.

(2) If the etiological agent was mild or not so strong and the damage to the muscular tissue was not severe, the typical "myodegeneration and myoregeneration" as illustrated in the case of white-muscle disease would take place.

V. Summary and conclusion

1. Fifty-five cases of new-born lambs affected with a variety of diseases were included in this investigation and the problem was studied by means of hematological, bacteriological, and pathological methods.
2. From the hematological aspect, both cellular determinations and chemical analyses of the blood were studied in detail and discussed fully in connection with the various individual cases. A similar blood study on normal sheep and lambs (controls) was also included.
3. The bacteriological studies included the isolation of 41 cultures of aerobes and 7 anaerobes from various cases. Nine genera of bacteria were identified, namely, Staphylococcus, Diplococcus, Neisseria, Escherichia, Bacterium, Pasteurella, Corynebacterium, Actinomyces, and Clostridium. Two cultures were undetermined.
4. As a result of pathological and bacteriological studies, 15 different types of diseases were diagnosed, including: white-muscle disease, staphylococcic infection, navel infection, docking infection, castration wound, broncho-pneumonia, pasteurellosis, enterotoxemia, meningitis, peritonitis, enteritis, gastric ulceration and perforation, dietary deficiency, congenital anomalies, and an undetermined group. Most of these diseases were studied more or less in detail and a number of conditions were reported for the first time.

5. The finding of muscle giant cells (skeletal and cardiac) in the cases of white-muscle disease and their significance in myo-regeneration are considered new to medical science and of vital importance in explaining the pathological phenomena involved.

6. The specific function of muscle giant cells and the modes of their formation were fully discussed.

7. A brief and concise statement was given concerning the pathological processes involved in muscular disturbances. The processes involved are designated as "myo-inflammation and scar-tissue repair" for tissue reaction to a severe injury and "myo-degeneration and myo-regeneration" for tissue reaction toward a mild injury.

8. The histopathology of white-muscle disease in lambs was studied in detail, and the pathological phenomena observed were suggested as a guide for the study of a number of human diseases with muscular involvement such as, tuberculosis, pneumonia, typhoid fever, rheumatic fever, syphilitic myocarditis, scarlet fever, and etc.

9. The cardiac muscle was observed to possess the capacity for regeneration, and this is also considered new to medical science.

10. The nature and origin of the so-called "Anitschkow myocyte" and "Aschoff cell" were fully discussed and evidence was given bearing on the position of these cells in the myocardium.

11. The fancy and peculiar terms such as "repair clubs of muscle", "multinucleated syncytial masses", "large multinucleated plasmatic masses", "syncytial sprouts with multiple nuclei", "cytoplasmic syncytium with a large number of nuclei", and "multinucleated cells in muscle" were explained.

12. The nature and the origin of the so-called "Muskelzellenschlauche" of Waldeyer was discussed in detail.

13. The nature and origin of the so-called "Aschoff body" was discussed and suggestions concerning further investigations were mentioned.

14. A new technic of opening the cranial cavity for brain examination was described.

15. The nature of the various diseases of new-born lambs were stressed, and suggestions were made for the control of some of the more important conditions.

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Fig. 1. Case 8069.

Lateral view of the skull to show the technic of opening cranial cavity for brain examination.

Fig. 2. Case 8041.

Ventral view of the frontal section of cranial cavity showing greater part of the brain and optic nerves.

- Plate 1 -

Fig. 3. Case 7356.

Longitudinal section of skeletal muscle showing myodegeneration. (H & E), X 160.

Fig. 4. Case 7356.

Longitudinal section of skeletal muscle showing fragmentary necrosis of muscle fibers and nuclear proliferation of surviving fibers. (H & E), X 780.

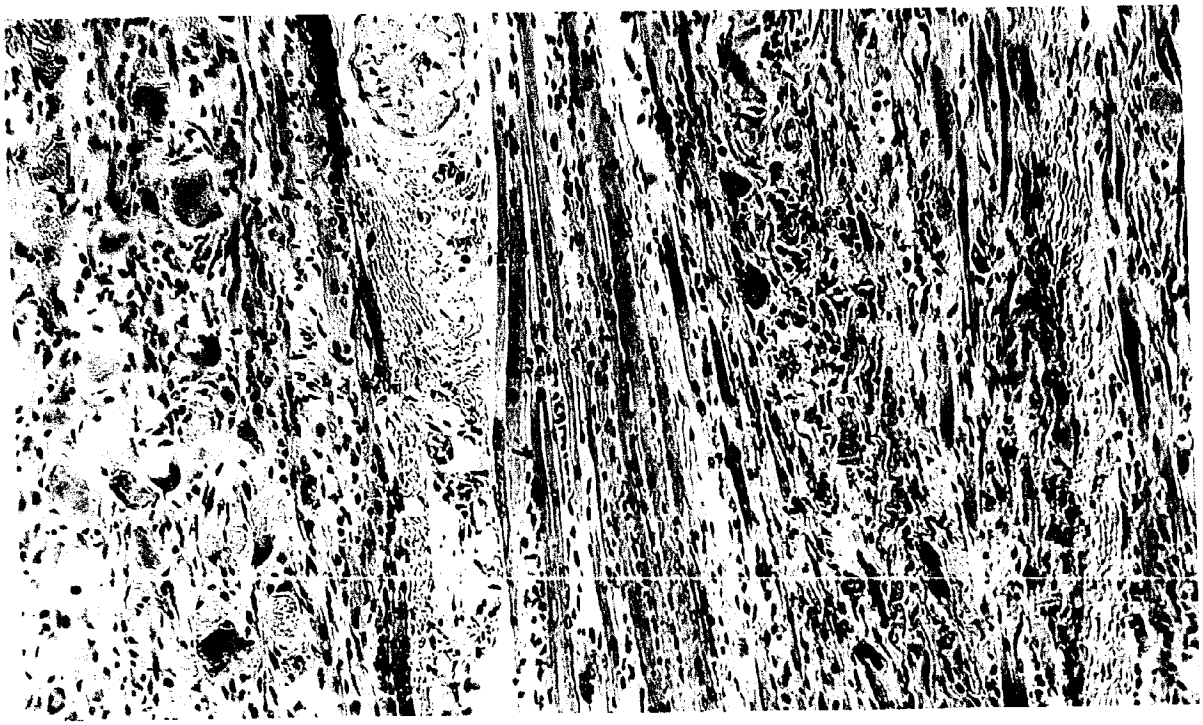


Fig. 5. Case 7356.

Cross section of skeletal muscle showing degeneration of the fibers. (H &E), X 116.

Fig. 6. Case 7356.

Skeletal muscle showing giant cell and some of the severely-damaged fibers. (Aniline blue), X 780.

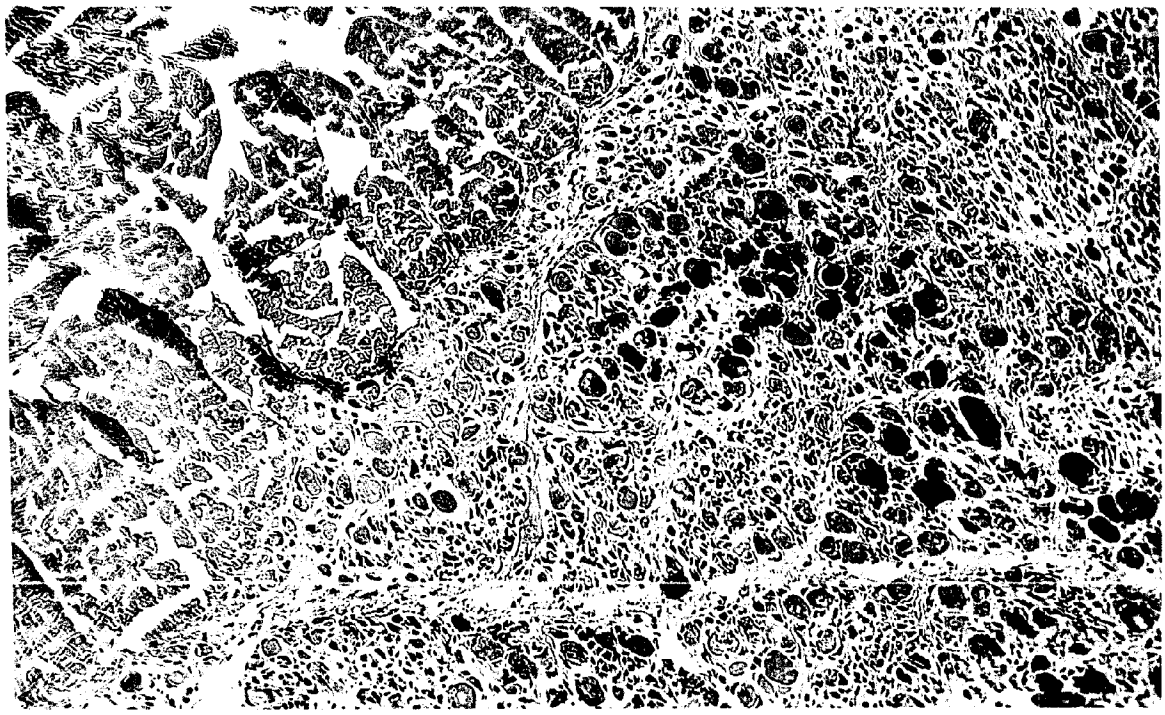


Fig. 7. Case 7358.

Muscle showing giant cells and necrotic muscle-fibers. (Aniline blue), X 780.

Fig. 7a. Case 7358.

Same but different field. (Aniline blue), X 780.

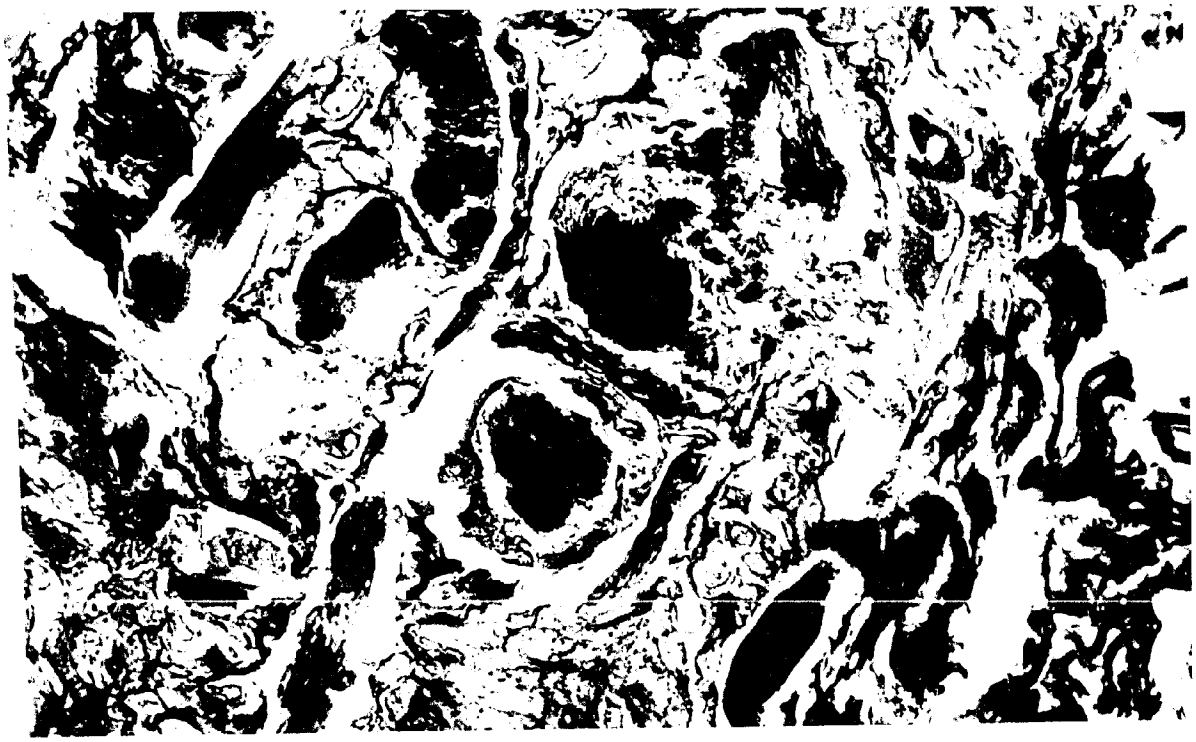


Fig. 8. Case 7356.

Skeletal muscle showing giant cell engulfing necretic muscle. A regenerating muscle fiber at the side. Note the similarity between the two in morphology of nuclei and affinity toward the same stain. (Aniline blue), X 780

Fig. 9. Case 7356.

Muscle showing giant cell engulfing necrotic muscle. A number of regenerating muscle fibers also in the same field. (H & E), X 780.

Fig. 10. Case 7356.

Muscle showing giant cell containing food vacuoles. (H & E). X 750.

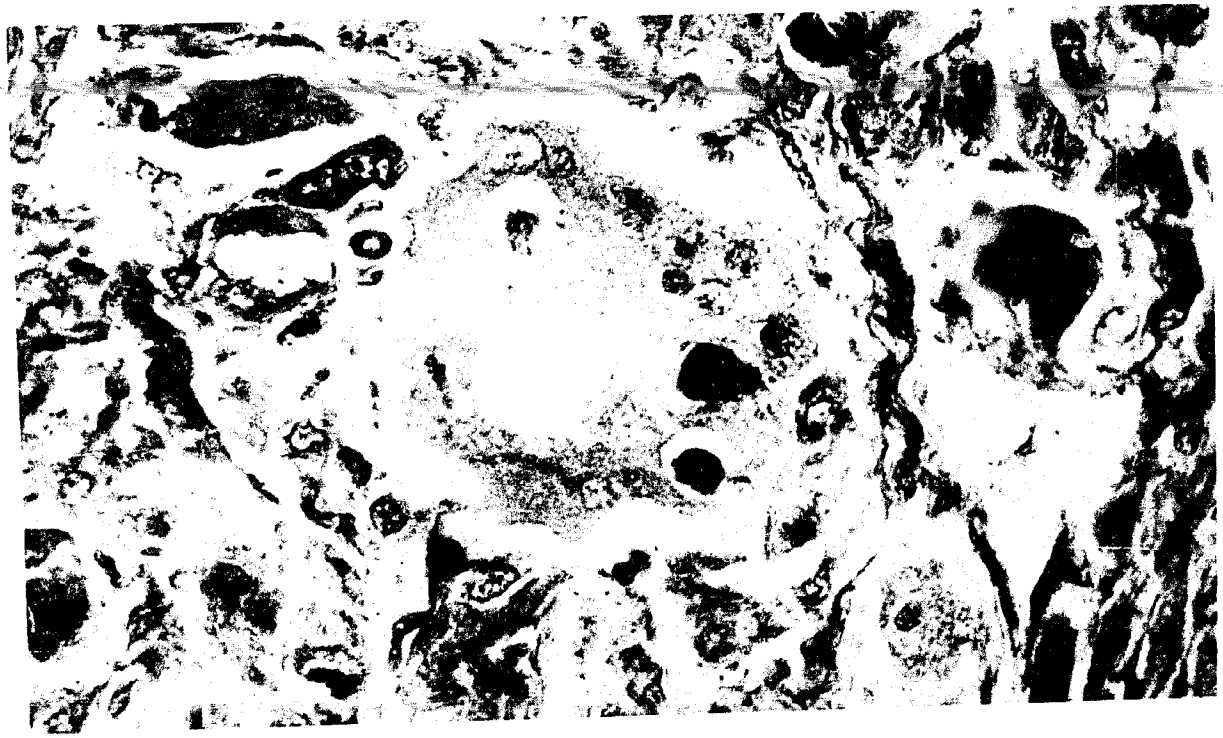
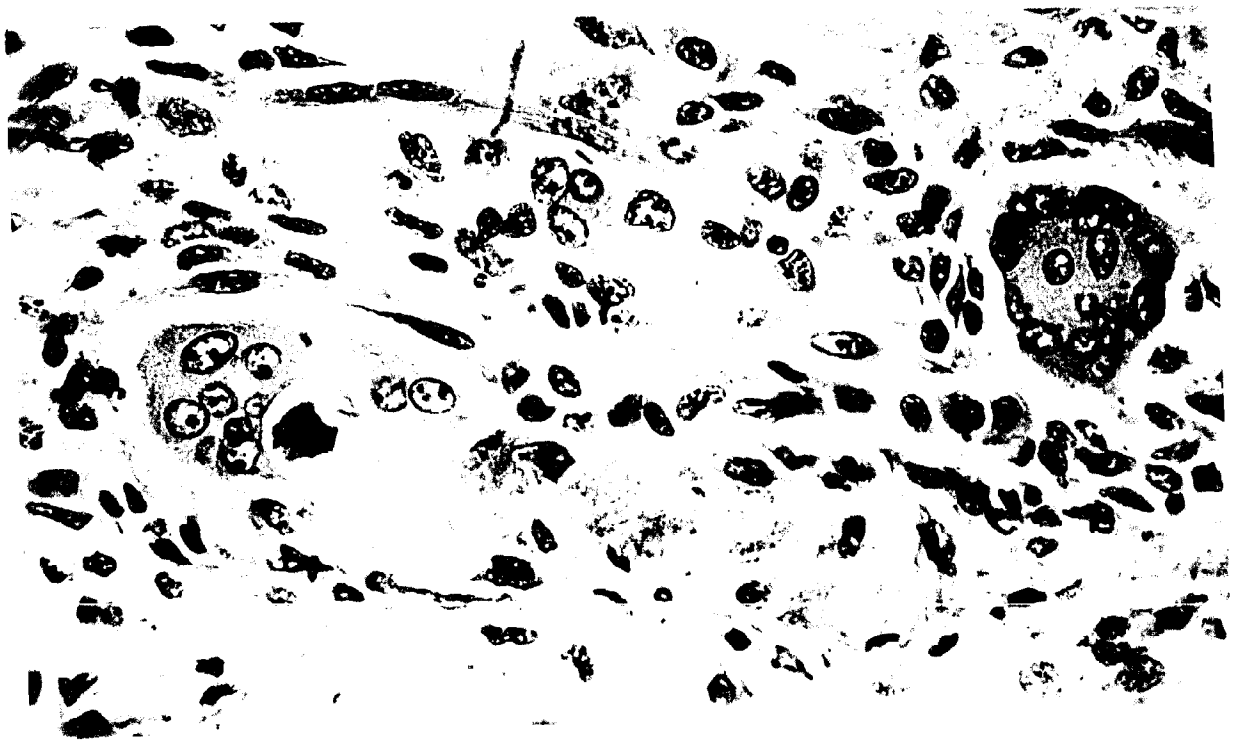


Fig. 11. Case 7356.

Muscle showing giant cell with food vacuole. Some regenerating muscle fibers were scattered around. A group of cells like this has been called "Muskelzellenschlauche" of Waldeyer. (H & E), X 780.



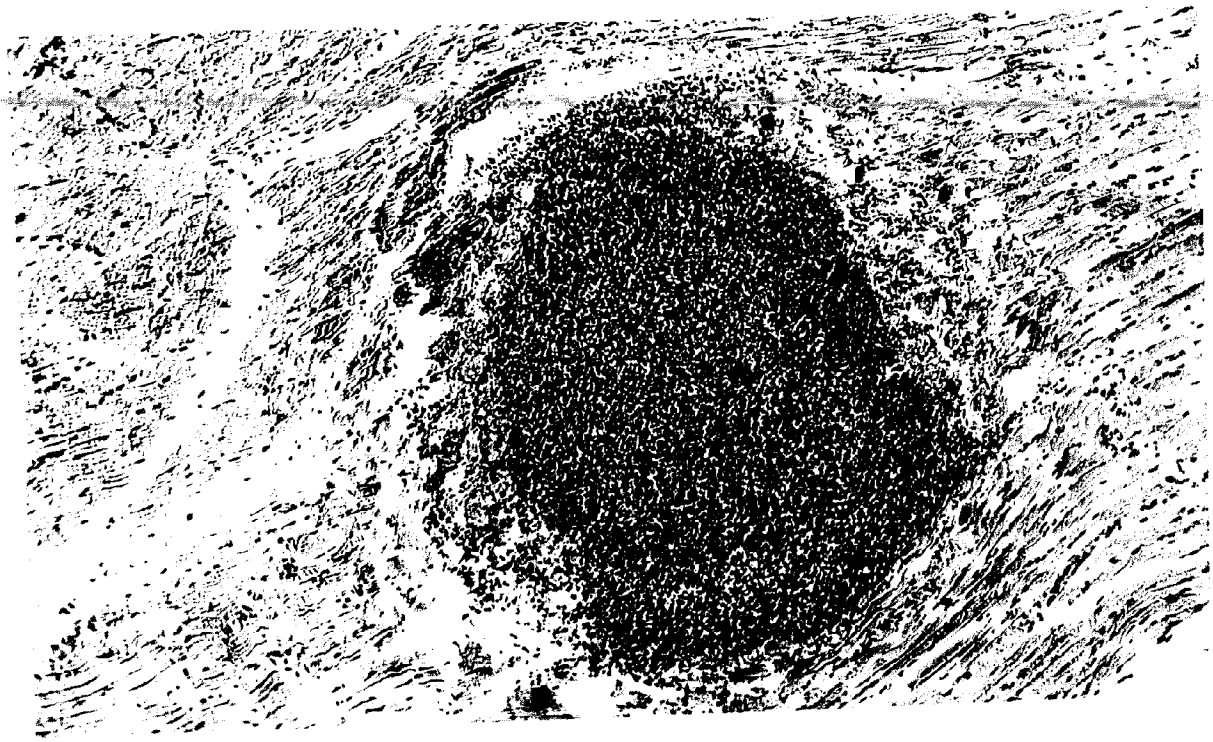
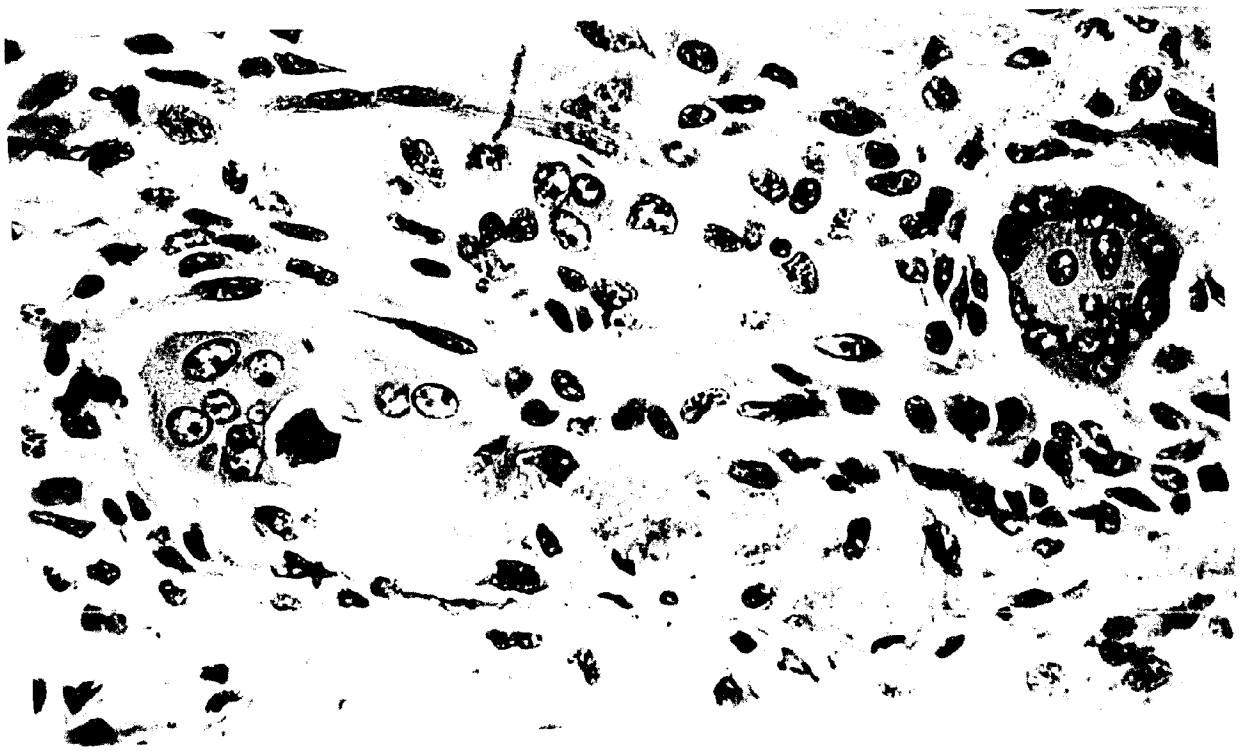


Fig. 12. Case 7358.

Muscle showing giant cells and regenerating muscle fibers. One ofthe muscle giant -cells is in the resting stage while the other is still actively phagocytic. (H & E), X 750.

Fig. 12a. Case 7358.

A recent abscess in the myocardium. (H & E), X 116.



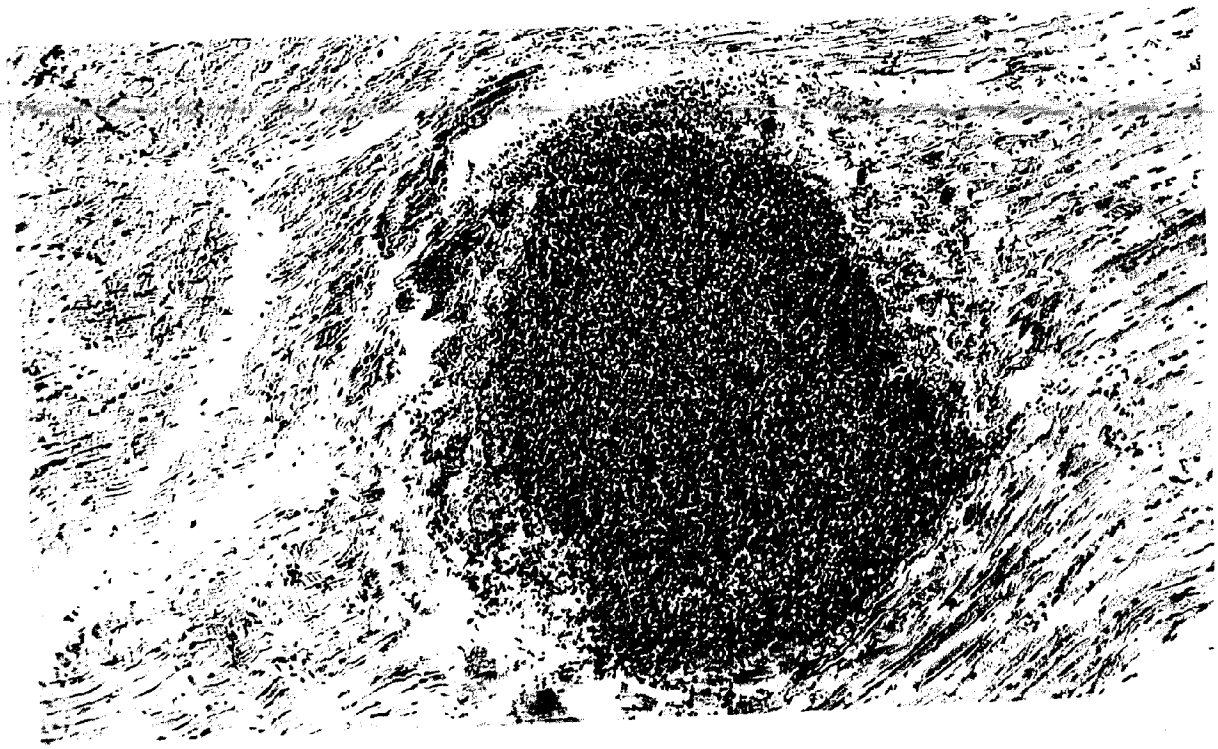


Fig. 13. Case 7356.

Muscle showing the formation of a giant-cell by fusion. Note the different stages of nuclear transformation in the fusing cells. (H & E), X 780.

Fig. 14. Case 7356.

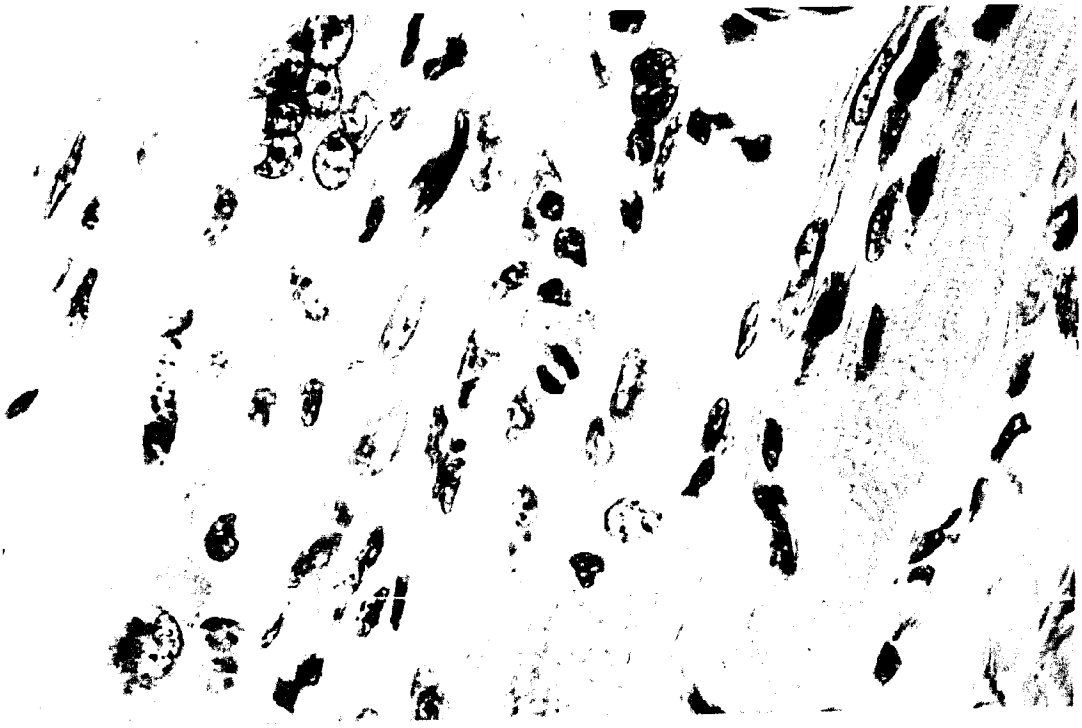
Same but different giant cell. (H & E), X 780.

Fig. 15. Case 7356.

Muscle showing the formation of giant cell by mitosis without cytoplasmic division. (H & E), X 780.

Fig. 16. Case 7337.

Liver showing focal necrosis. (H & E), X 609.



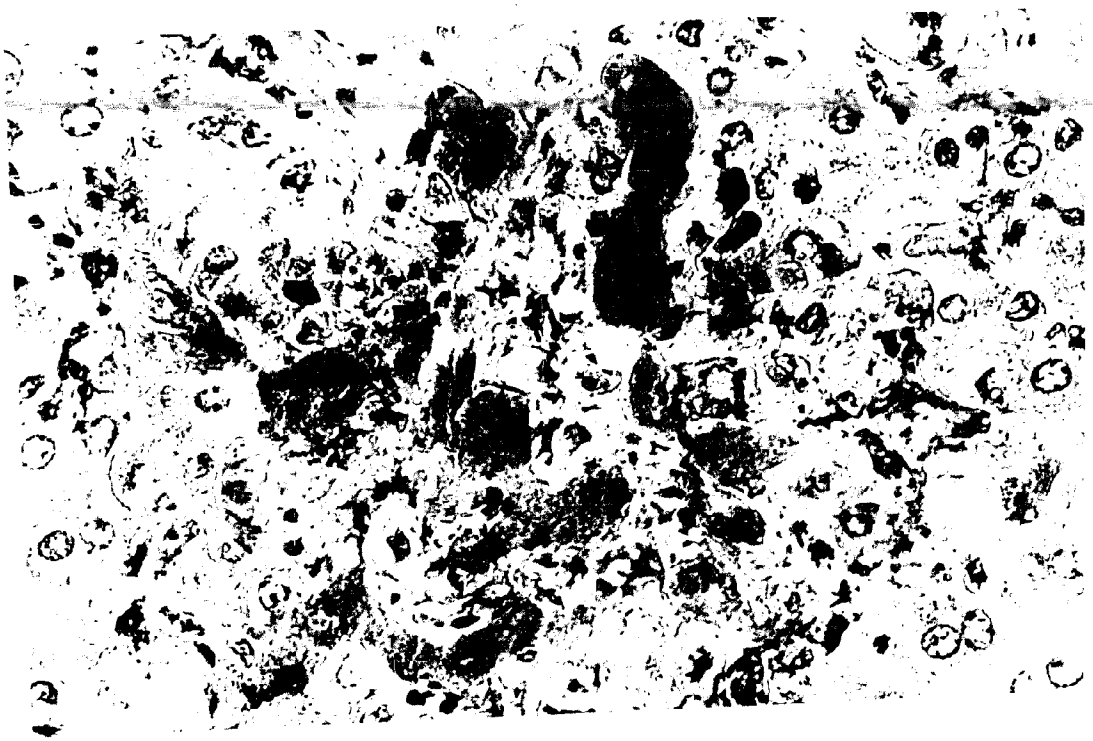


Fig. 17. Case 7338.

Blood smear showing immature neutrophils.
(Wright's stain), X 516.

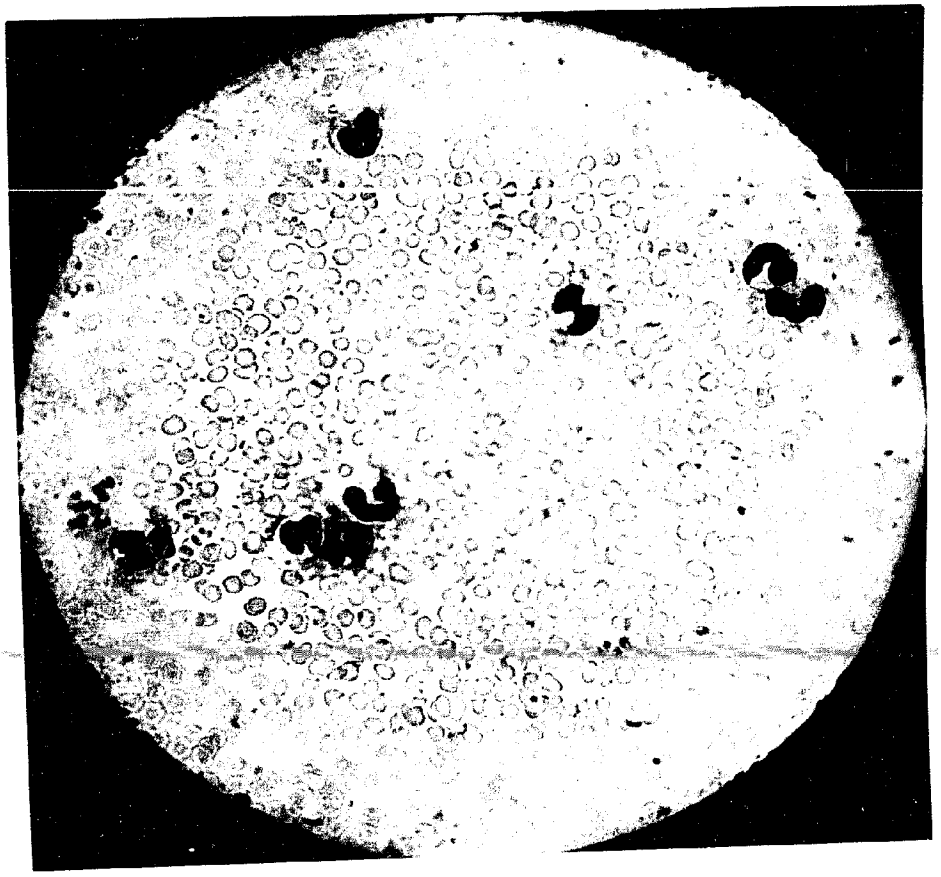


Fig. 18. Case 7338.

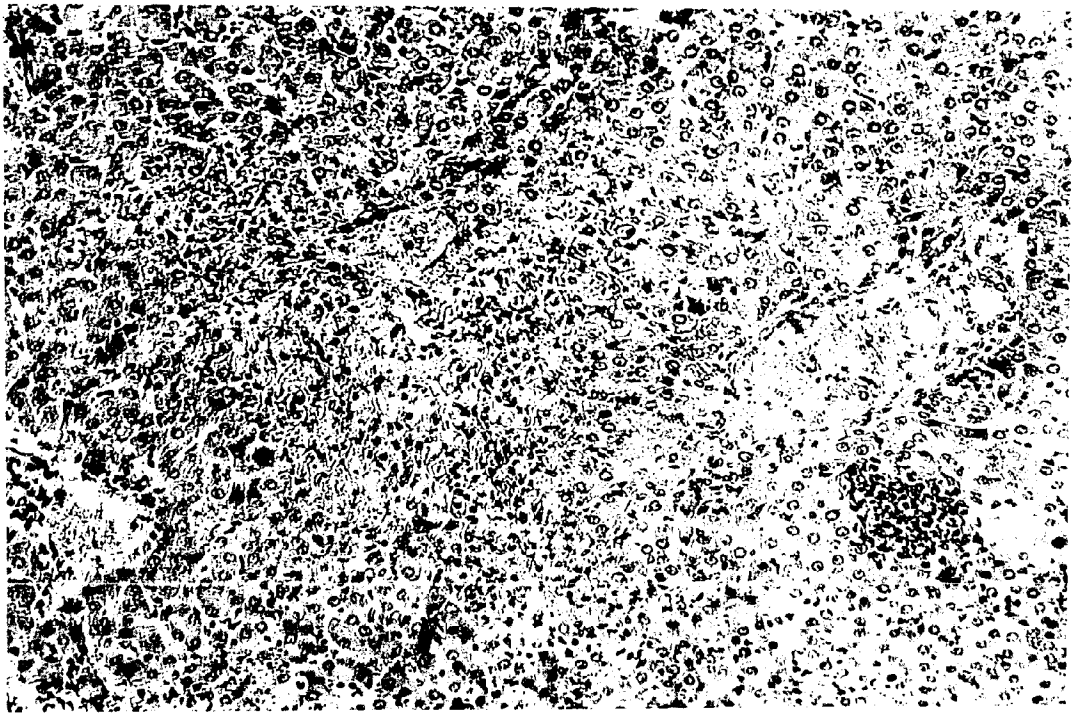
Staphylococcic abscesses in muscle, lungs, kidneys and heart.

Fig. 19. Case 7338.

Liver showing necrotic foci. (H & E), X 160.

Fig. 20. Case 7338.

Liver showing necrotic foci and hemorrhage. (H & E), X 160.



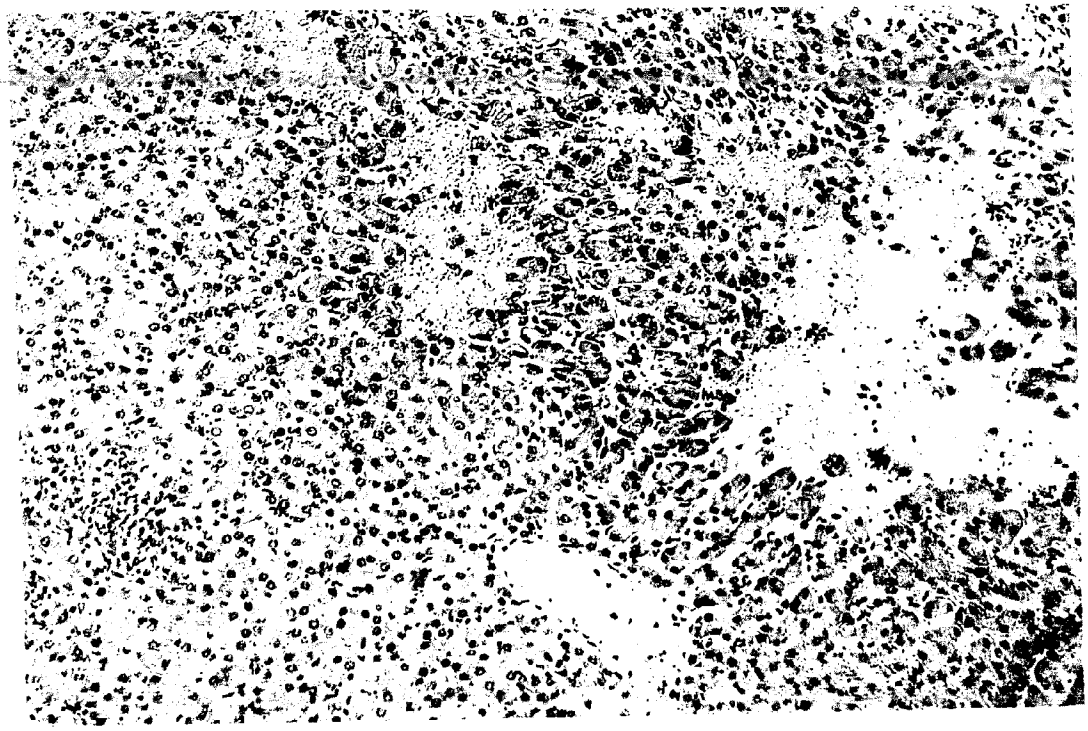
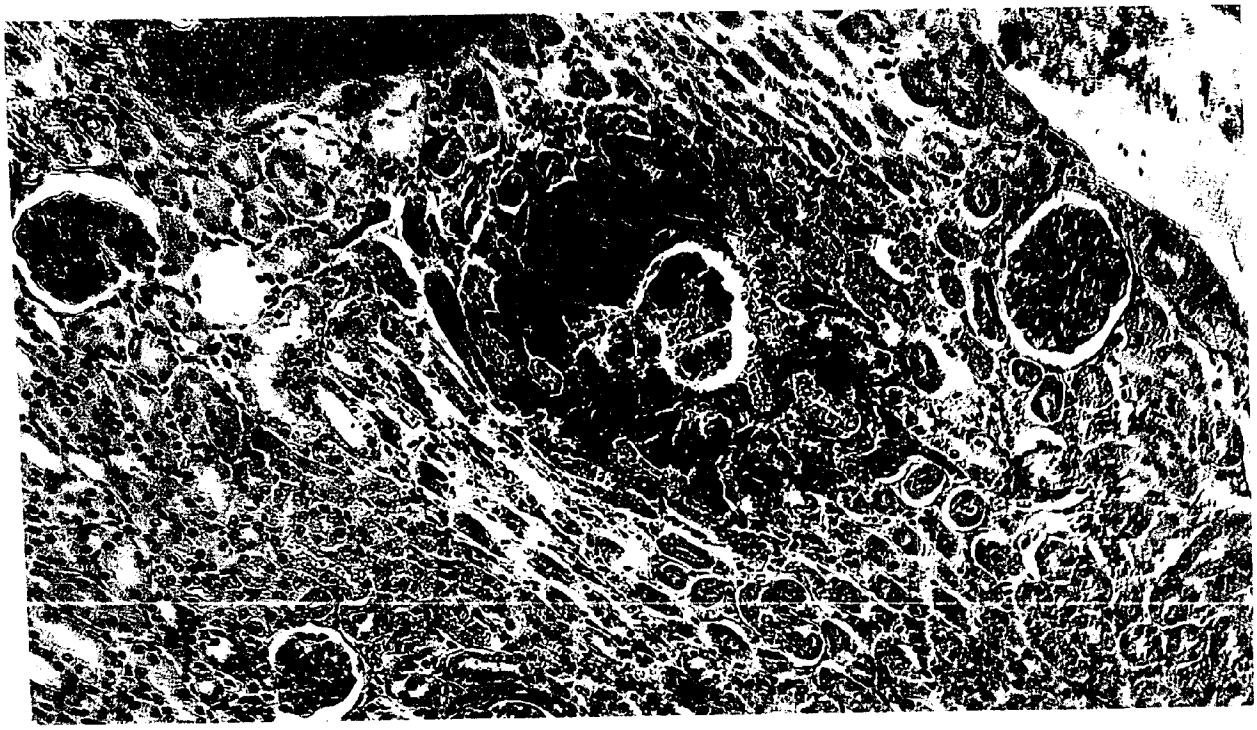


Fig. 21. Case 7338.

Kidney showing bacterial emboli in a glomerulusand focal necrosis. (H & E), X 160.

Fig. 22. Case 7338.

Kidney showing interstitial focal necrosis. (H & E), X 630.



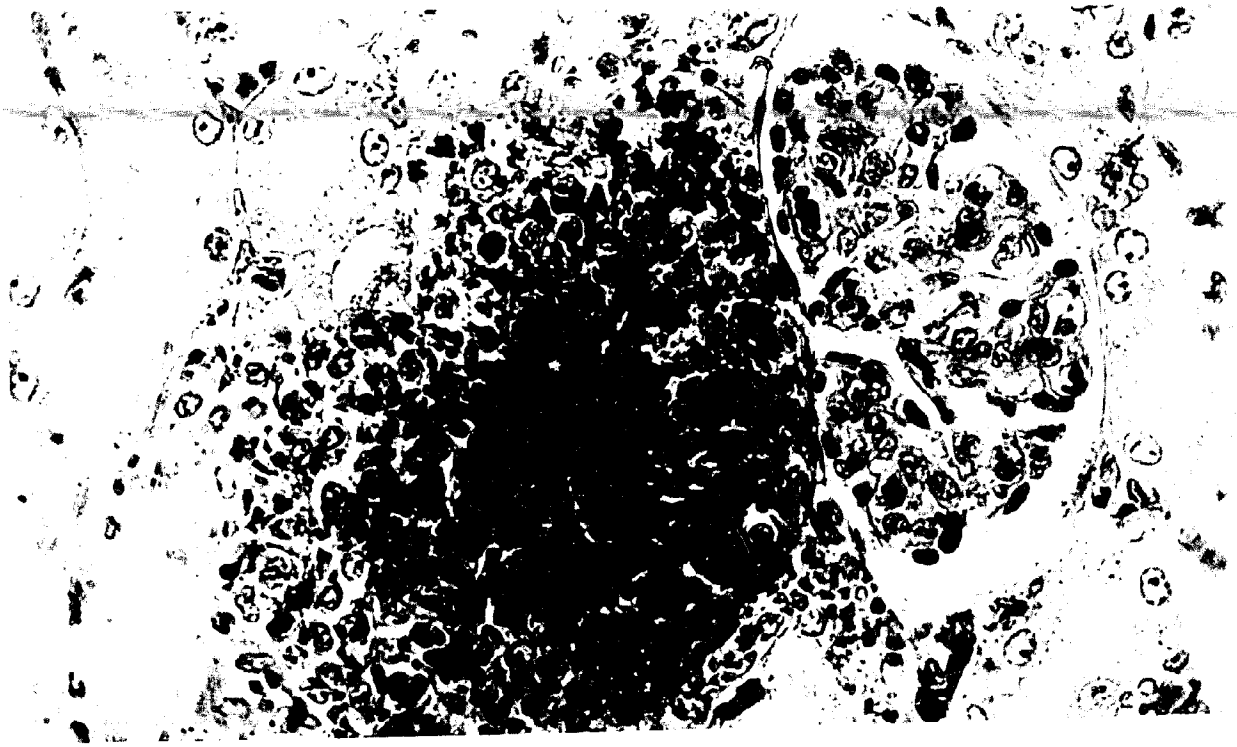


Fig. 23. Case 7338.

Staphylococcic abscess involving renal cortex and capsule. The black area in the glomerular space was a mass of staphylococci. (Gram-Weigert), X 95.

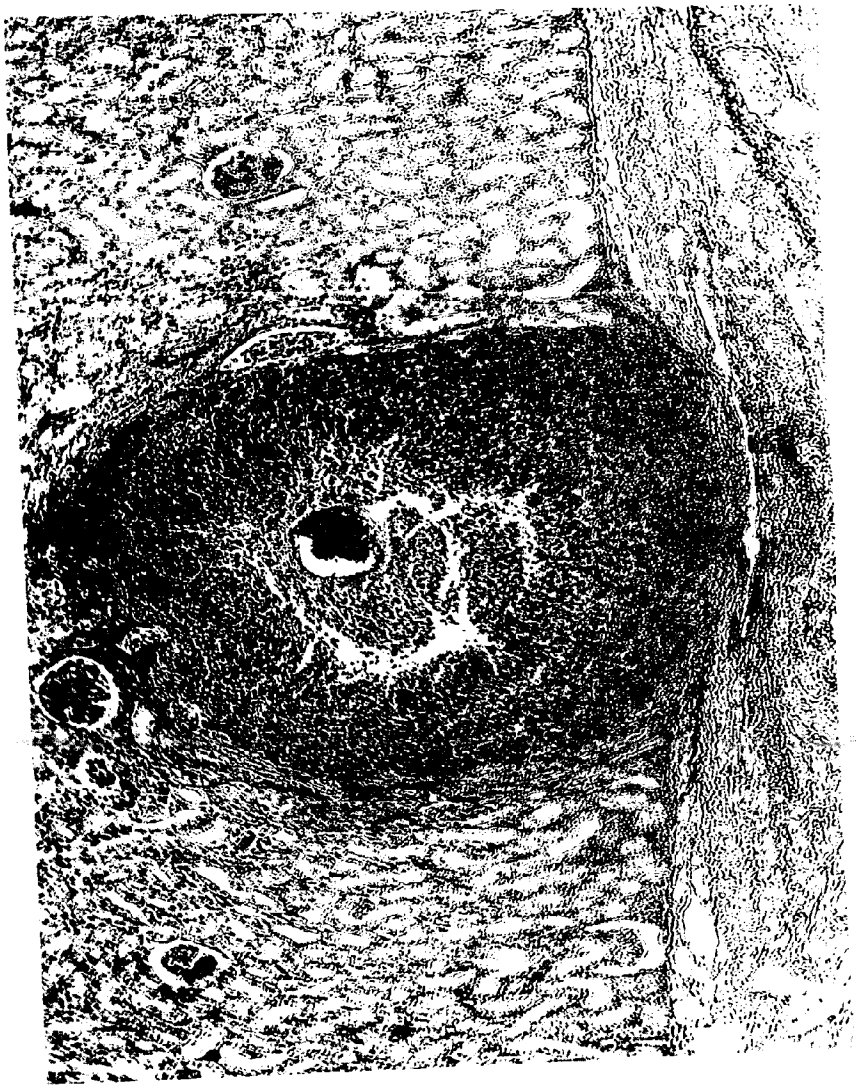


Fig. 24. Case 7338.

Kidney showing cortico-medullery abscess. (H & E), X 3.5.

Fig. 25. Case 7338.

Kidney showing staphylococci in glomerular space. (Gram-Weigert), X 860.

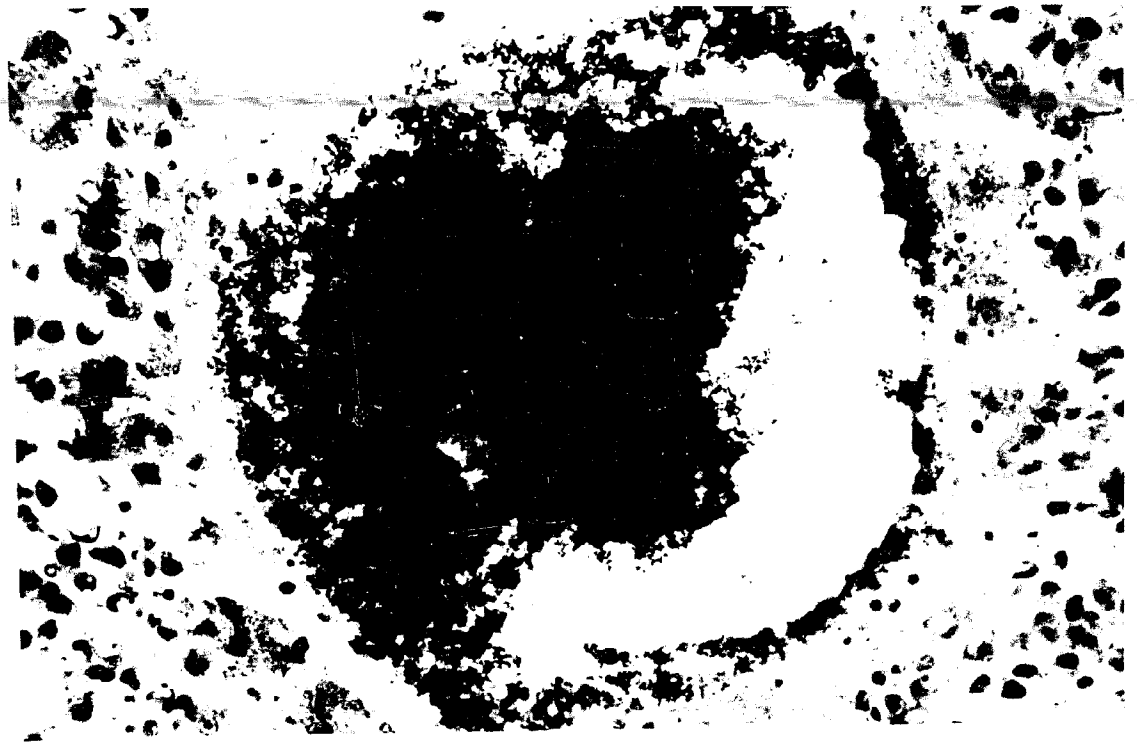
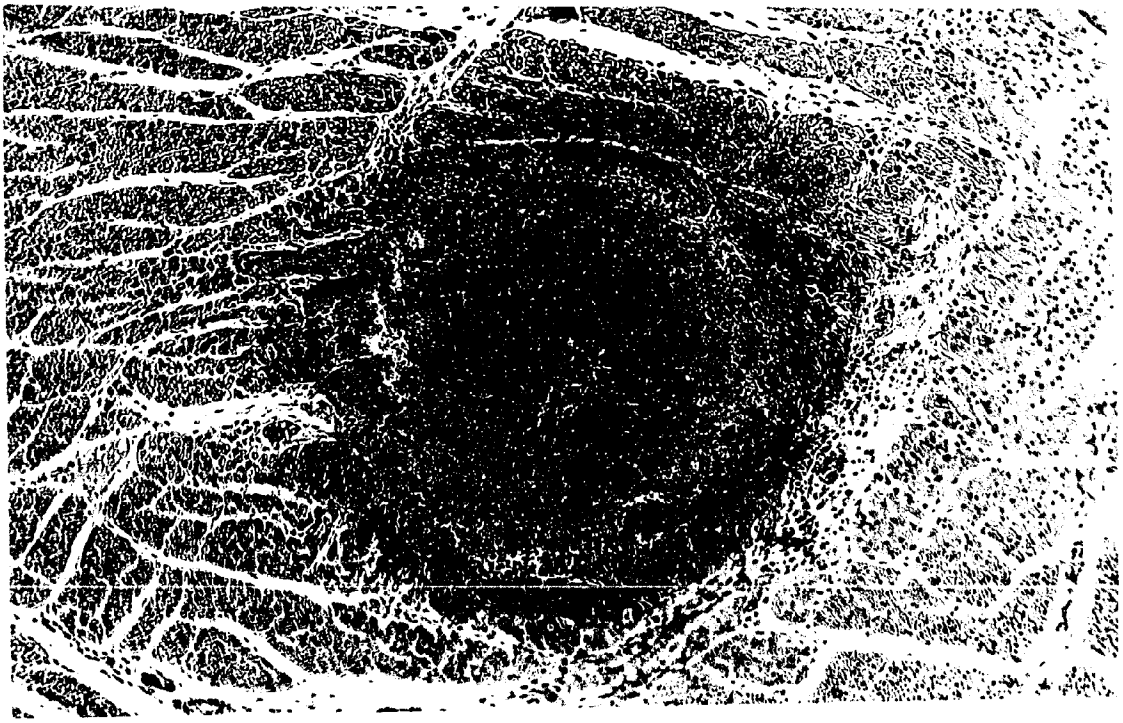


Fig. 26. Case 7338.

Myocardial abscess. (H & E), X 116.

Fig 27. Case 7338.

Abscess in the diaphragm. (H & E), X 116.



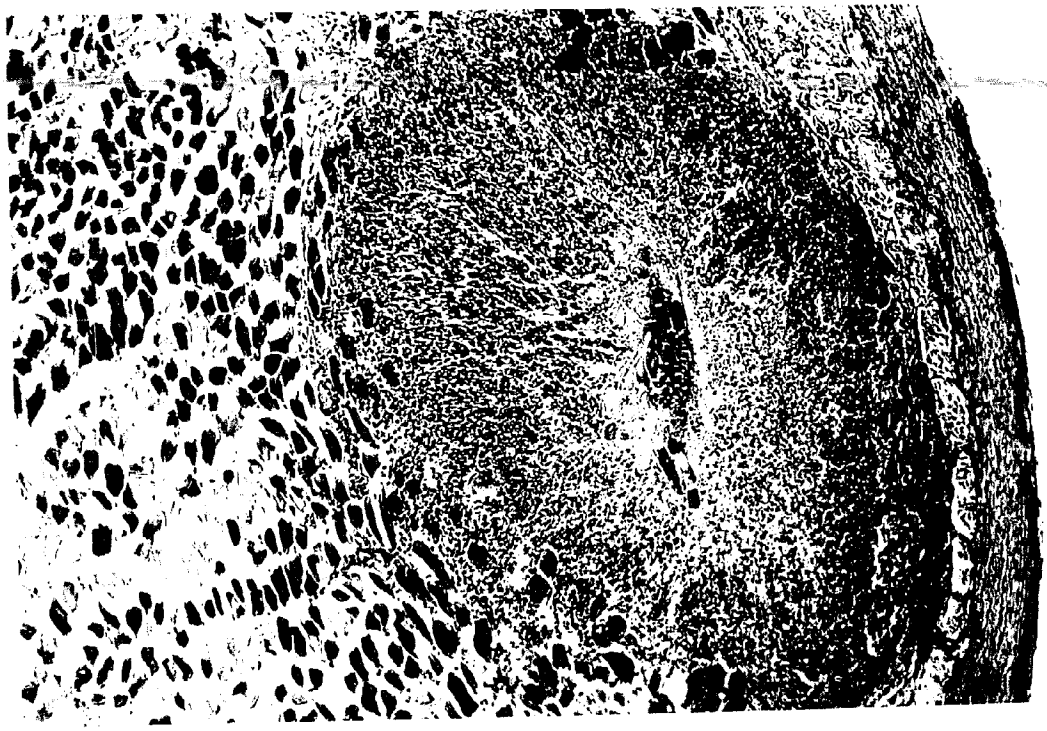


Fig. 28. Case 7343.

Actinomyces necrophorus abscess in liver, lungs, heart and ribs.

Fig. 29. Case 7343.

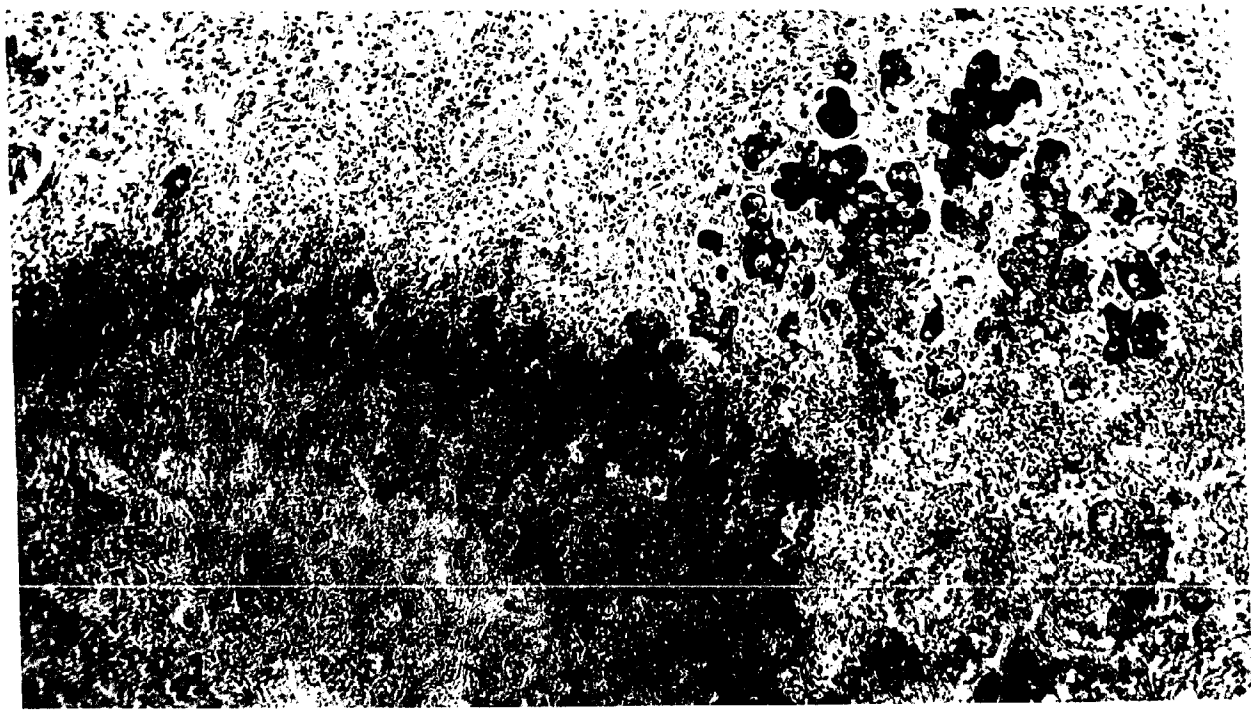
Multiple abscesses of the liver (H & E), X 50.

Fig. 30. Case 7343.

Lung showing calcified areas adjacent to an abscess. (H & E), X 116.

Fig. 31. Case 7344.

Brain showing infiltration with polymorphonuclear leucocytes. Note the necrotic nerve cells and disorganized neuroglia. (H & E), X900.



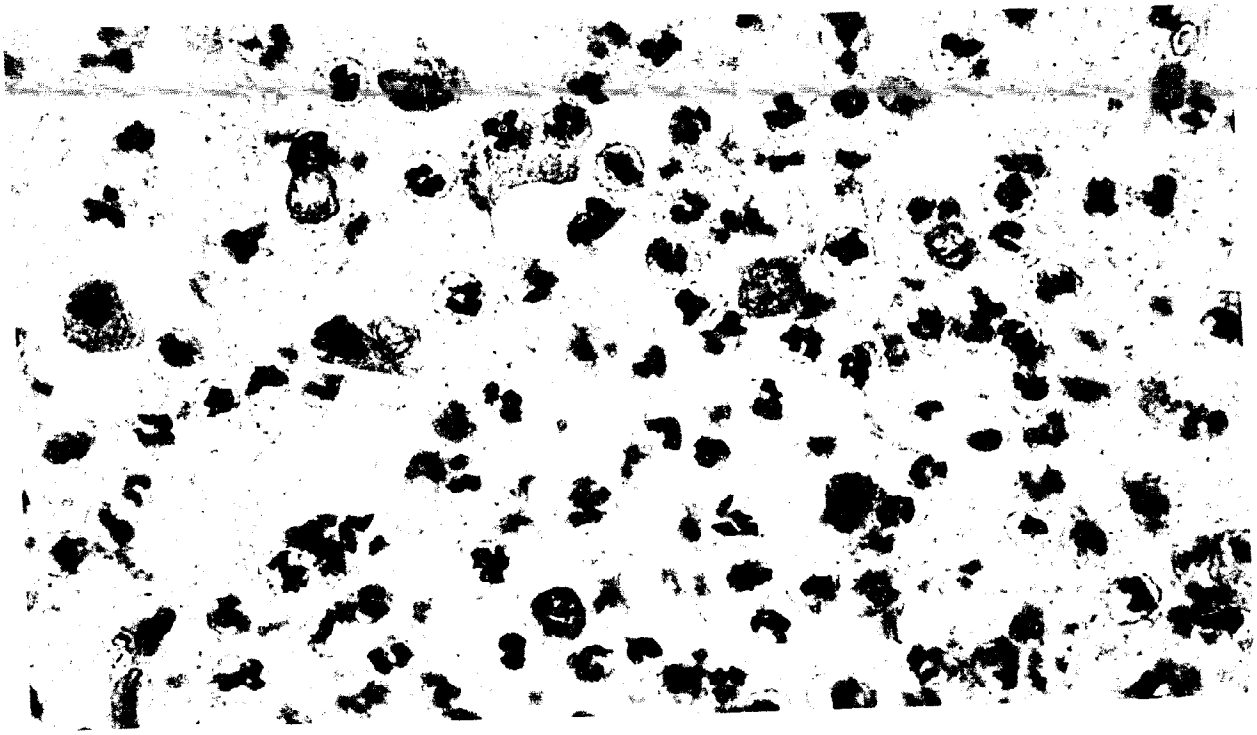
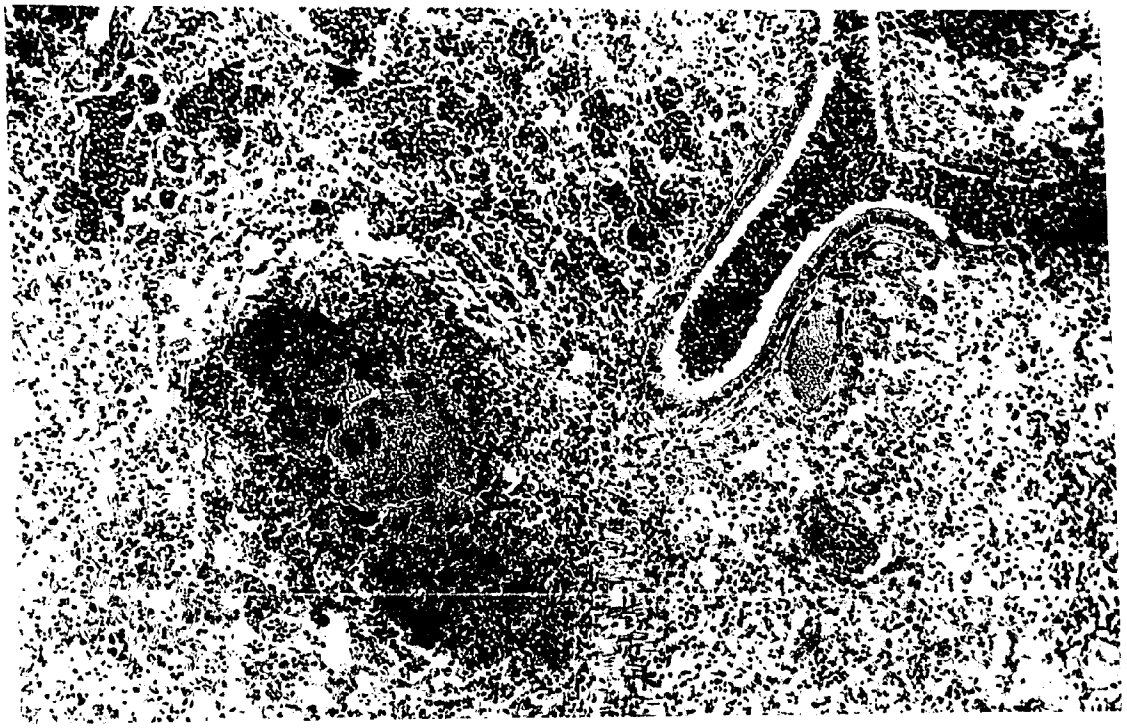


Fig. 32. Case 7372.

Lung showing *Actinomyces necrophorus* abscess. (H & E), X 116.

Fig. 33. Case 7394.

Smear from carpal joint showing phagocytosis of staphylococci. (Wright's stain), X 1640.



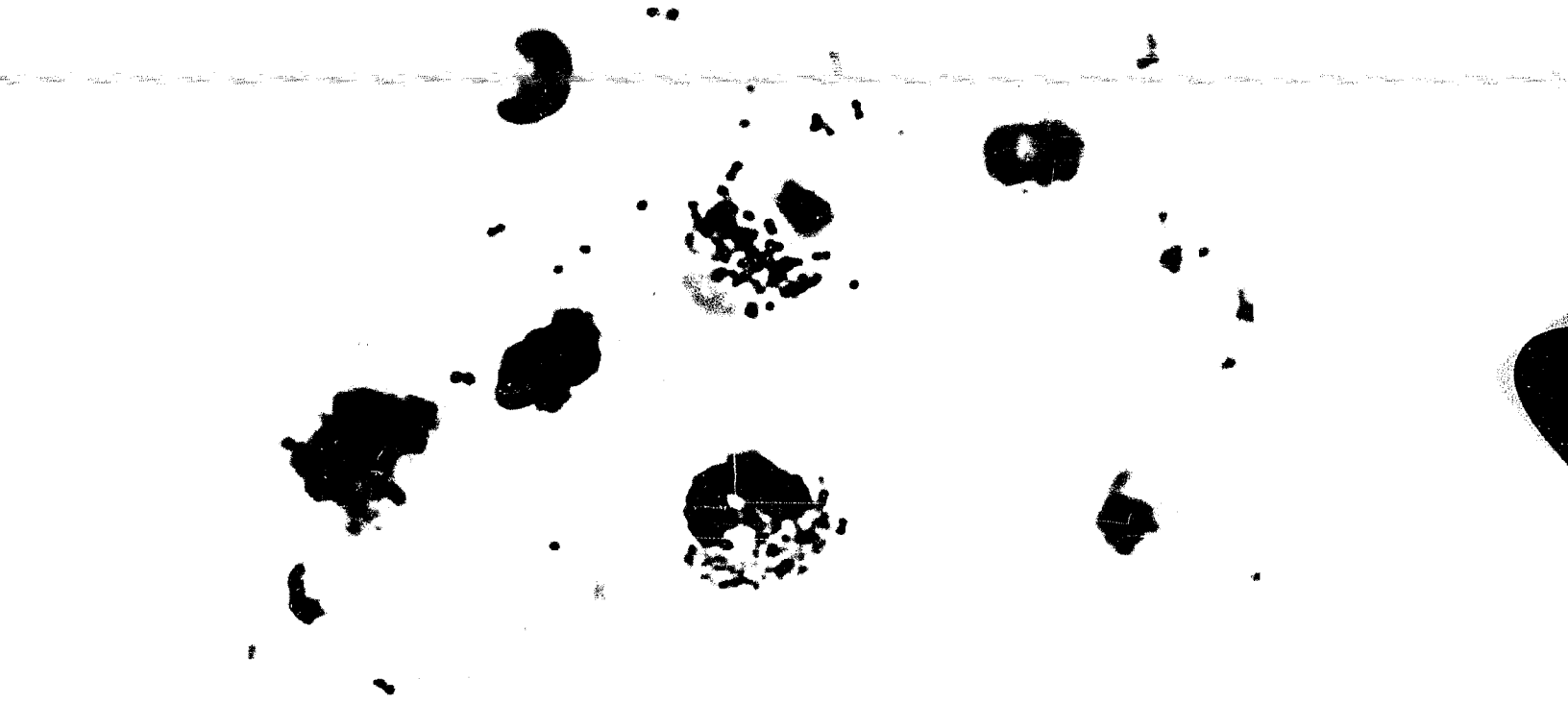


Fig. 34. Case 7394.

Suppurative myocarditis. (H & E), X 106.

Fig. 35. Case 7394.

Heart showing chronic suppurative lesion.
(H & E), X 106.

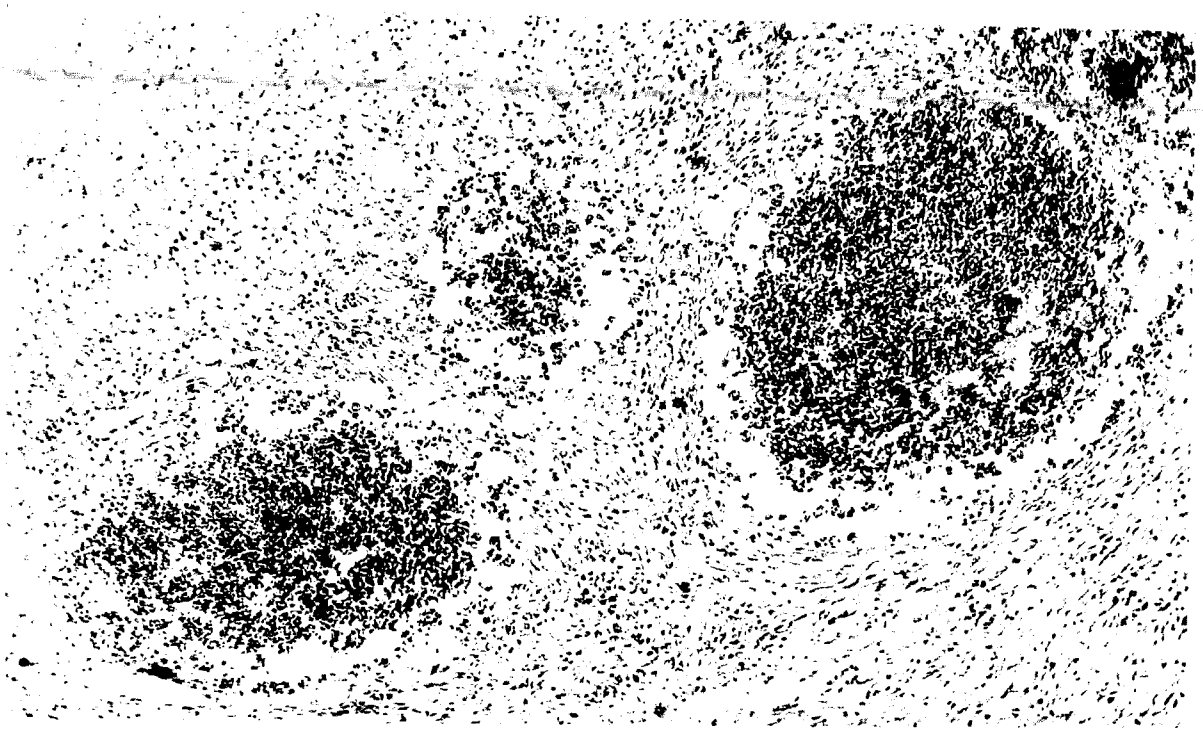


Fig. 36. Case 7362.

Spinal cord showing abscessation.

Fig. 37. Case 7362.

Cross section of spinal cord showing abscessation. (H & E), X 6.

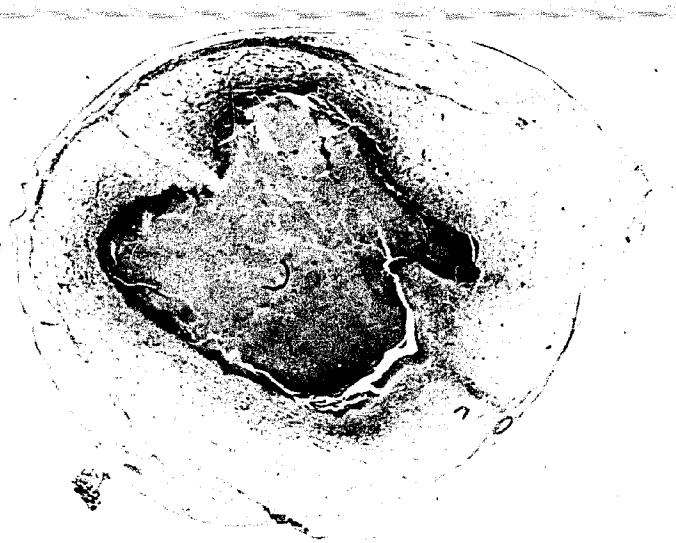


Fig. 38. Case 7365.

Cross section of spinal cord showing abscessation and hemorrhage. (Gram-Weigert), X 6.

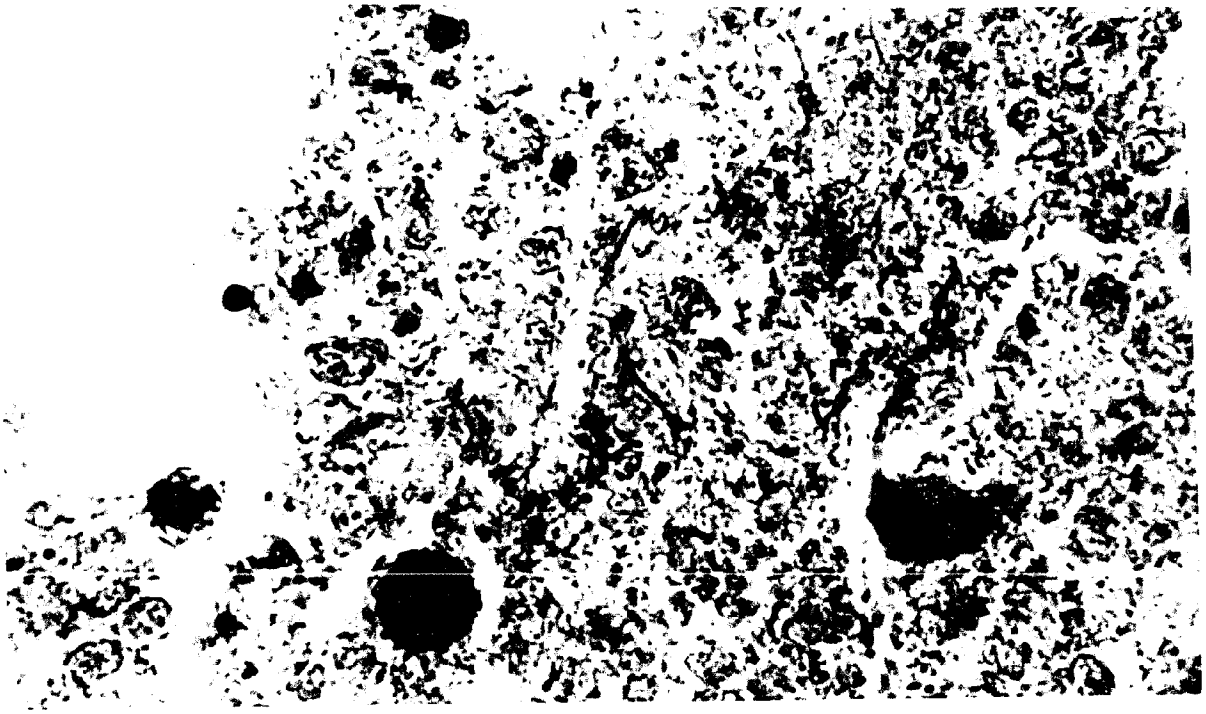
- Plate 22a -

Fig. 39. Case 7365.

Spinal cord showing lesion containing Actinomyces necrophorus and a diphtheroid. (Gram-Weigert), X 1440.

Fig. 40. Case 7308.

Liver showing fatty degeneration and necrosis around central vein. (H & E), X 590.



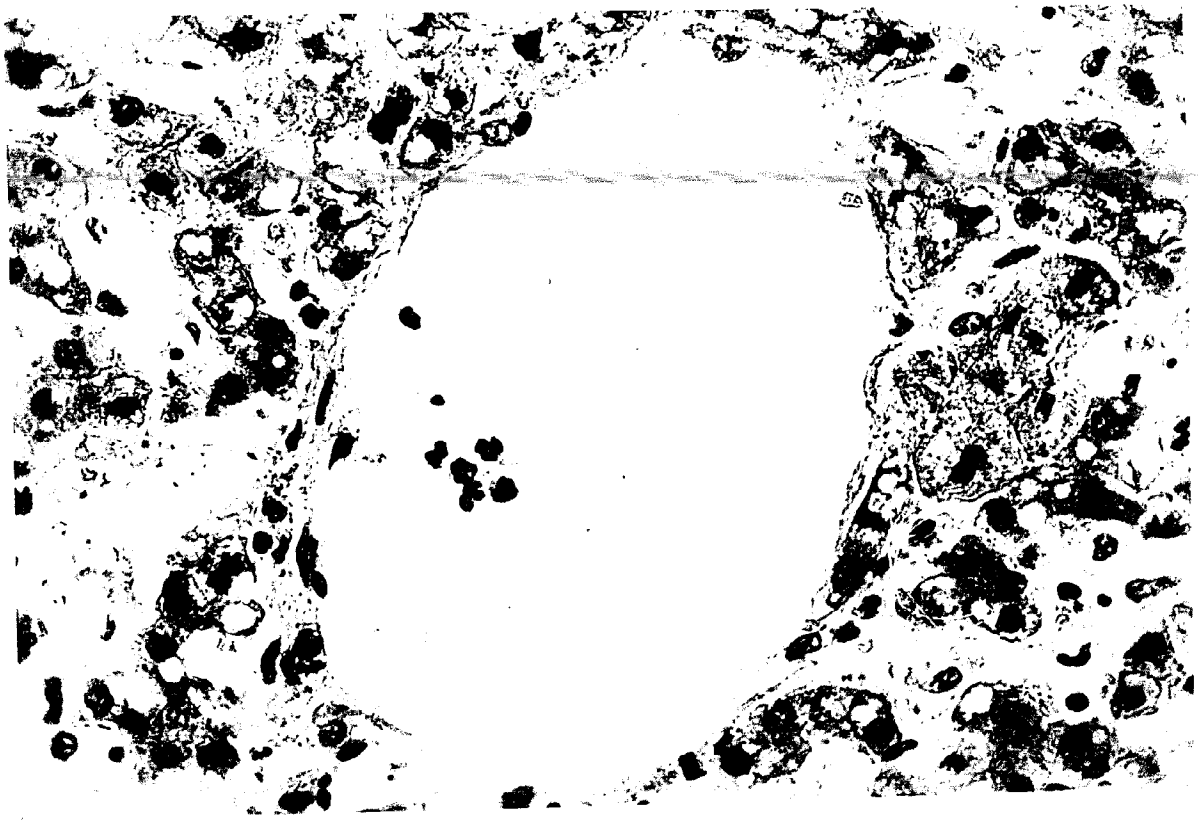


Fig. 41. Case 7329.

Lung showing interstitial bronchopneumonia. Note the tubercle- like lymphocytic infiltration. (H & E), X 97.

Fig. 42. Case 7329.

High power of the same. (H & E), X 600.

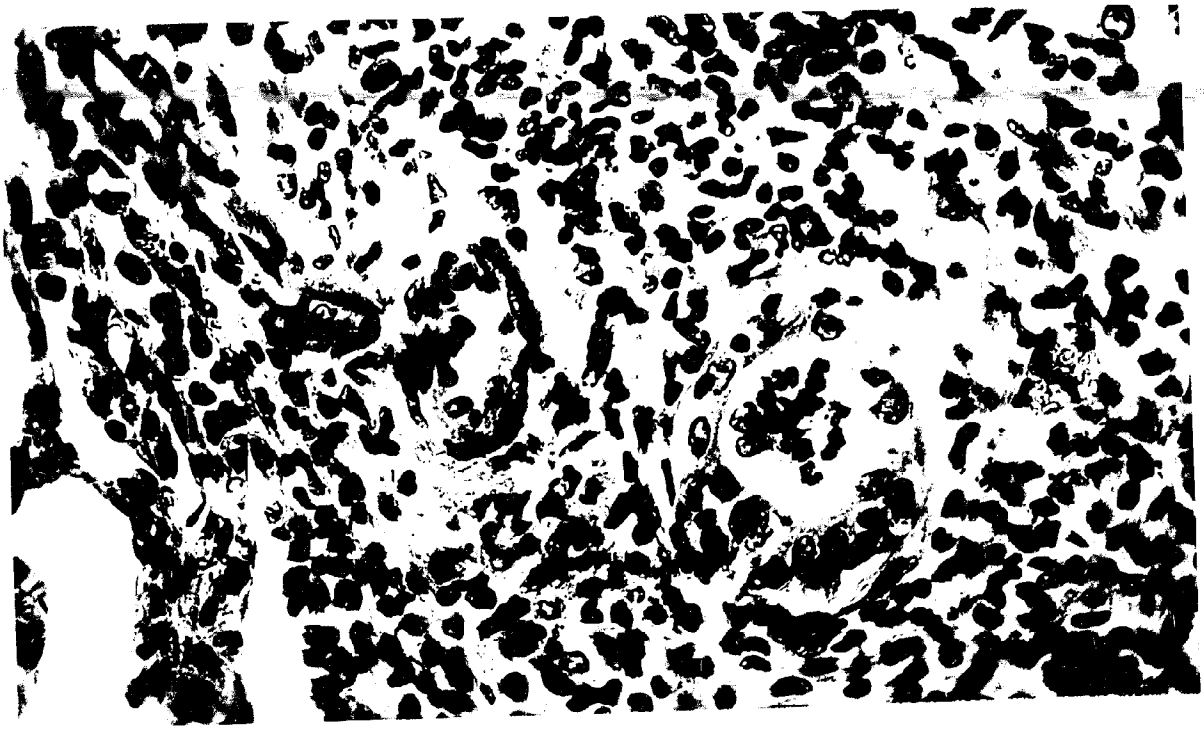


Fig. 43. Case 7353.

Lung showing hemorrhagic bronchopneumonia. (H & E), X 160.

Fig. 44. Case 7415.

Brain showing perivascular cell infiltration. (H & E), X 630.

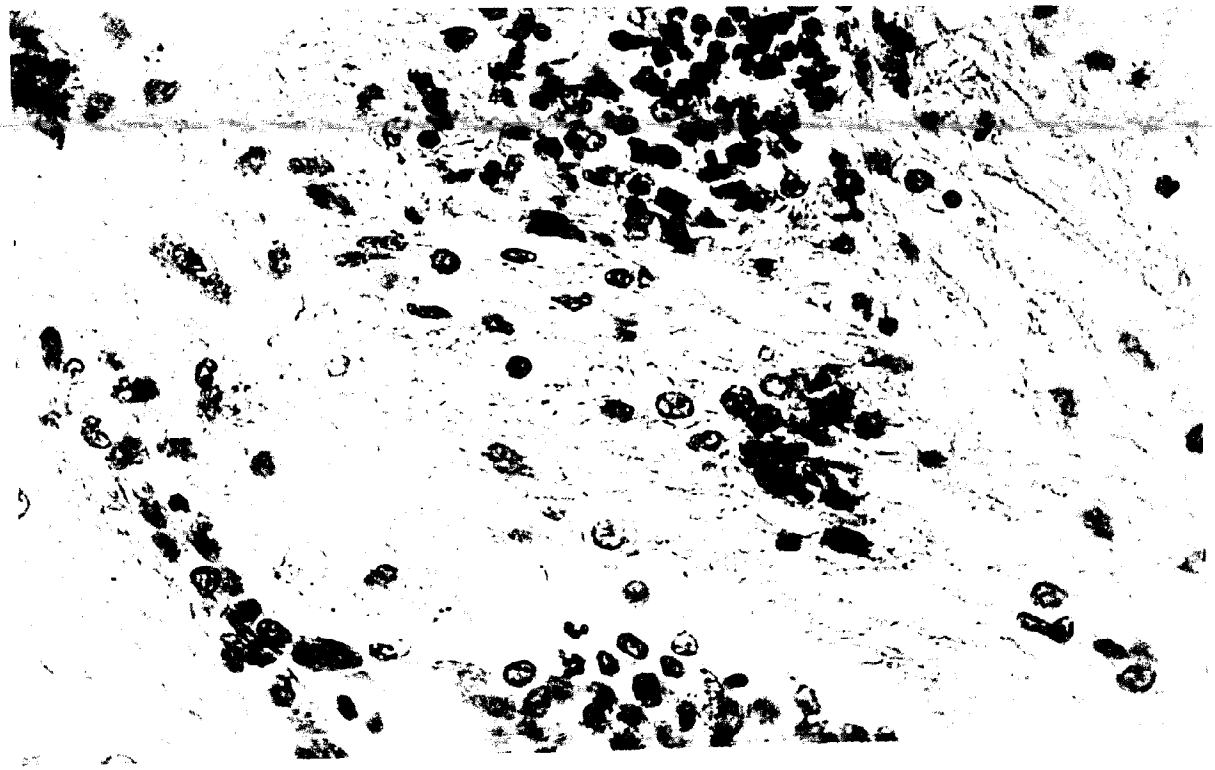


Fig. 45. Case 7334.

Spleen showing serosal congestion and hemorrhage into capsule. (H & E), X 116.

Fig. 46. Case 7399.

Intestine showing hemorrhage due to enterotoxemia.

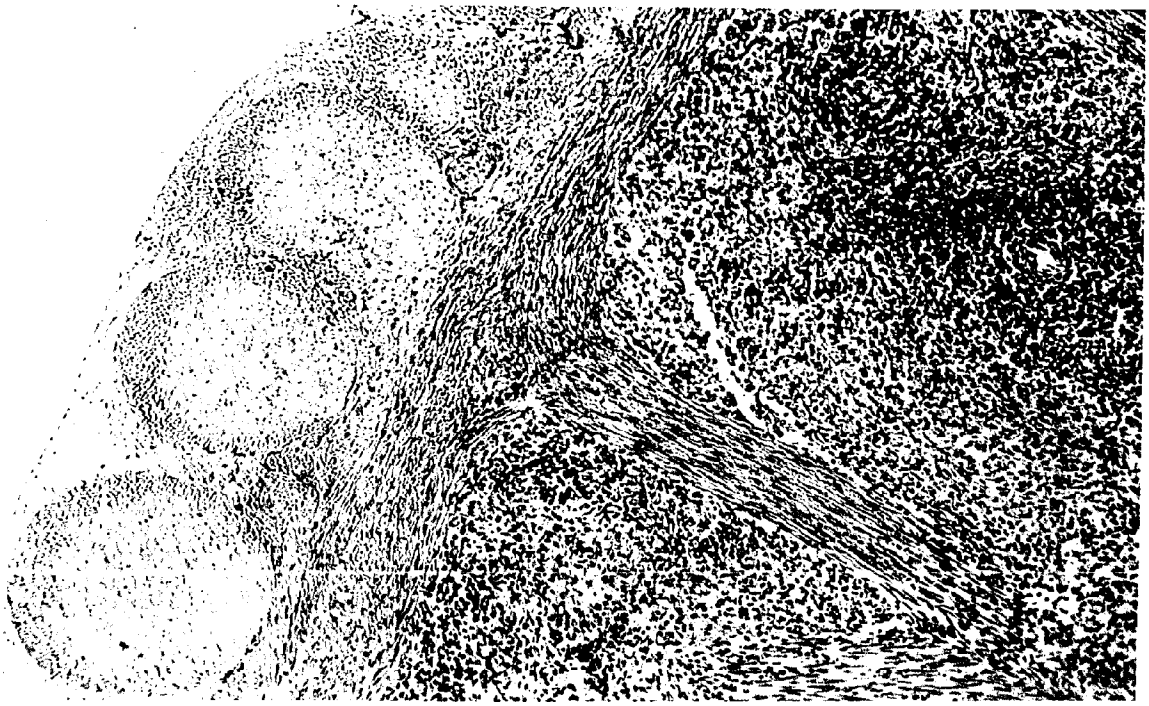


Fig. 45. Case 7334.

Spleen showing serosal congestion and hemorrhage into capsule. (H & E), X 116.

Fig. 46. Case 7399.

Intestine showing hemorrhage due to enterotoxemia.

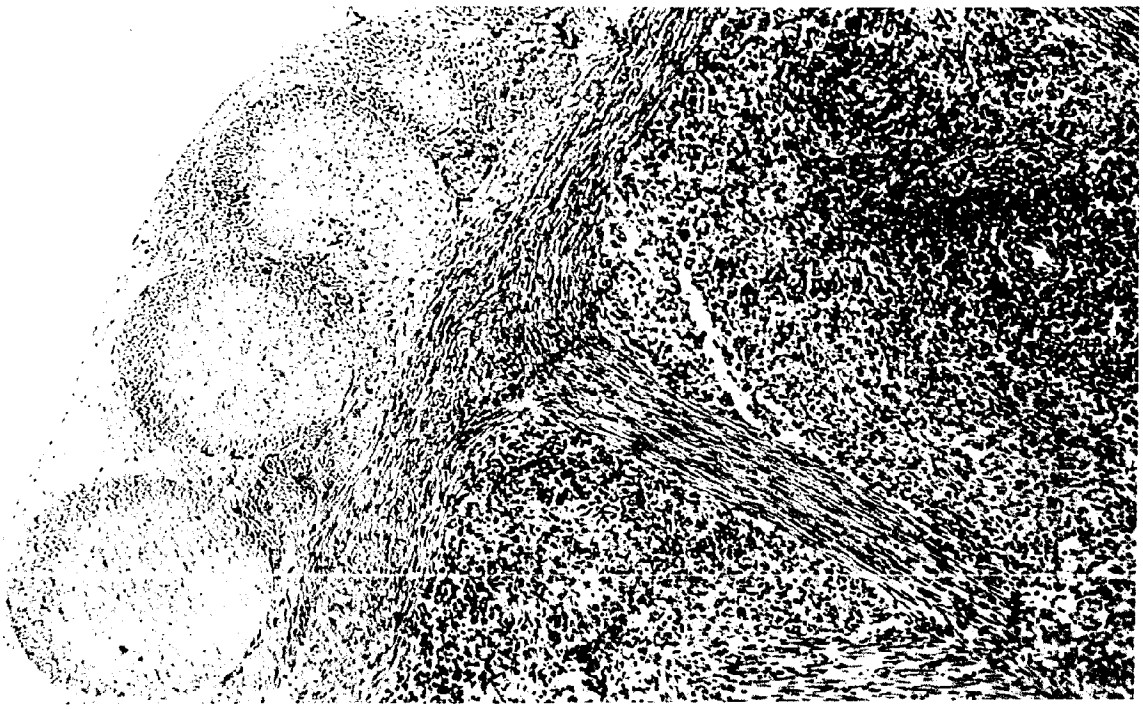
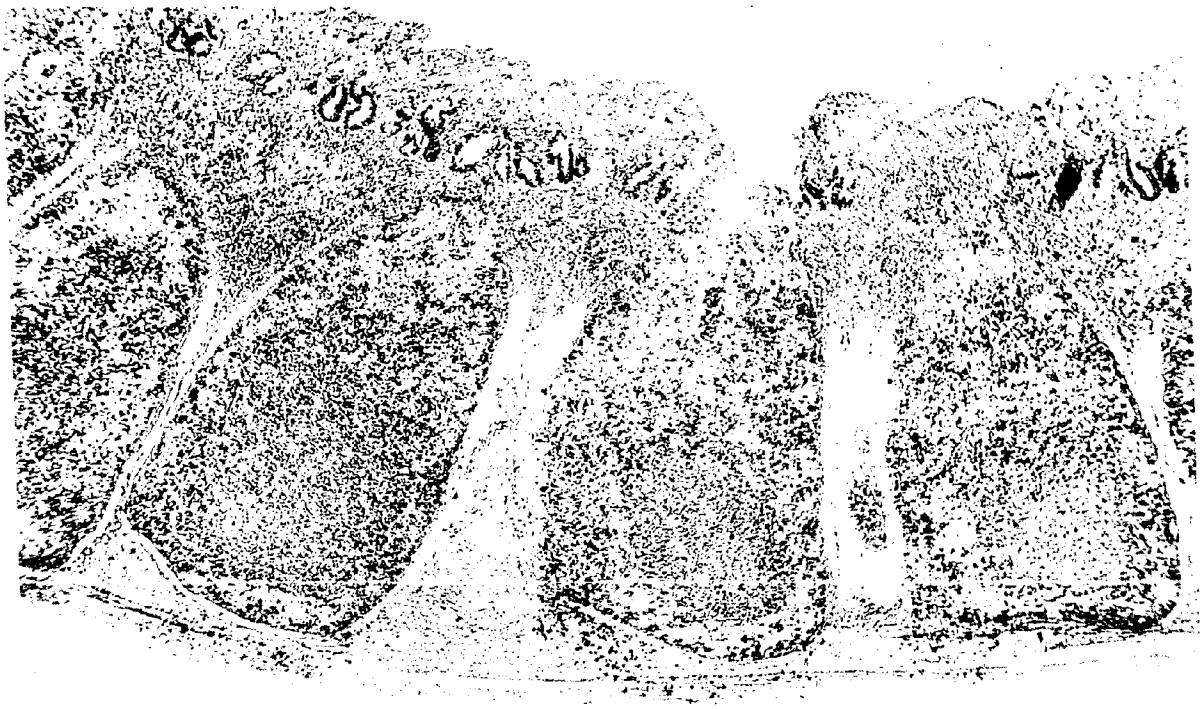


Fig. 47. Case 7399.

Section of intestine showing marked submucosal hemorrhage. (H & E), X 80.

Fig. 48. Case 7400.

Brain showing meningitis. (H & E), X 630.



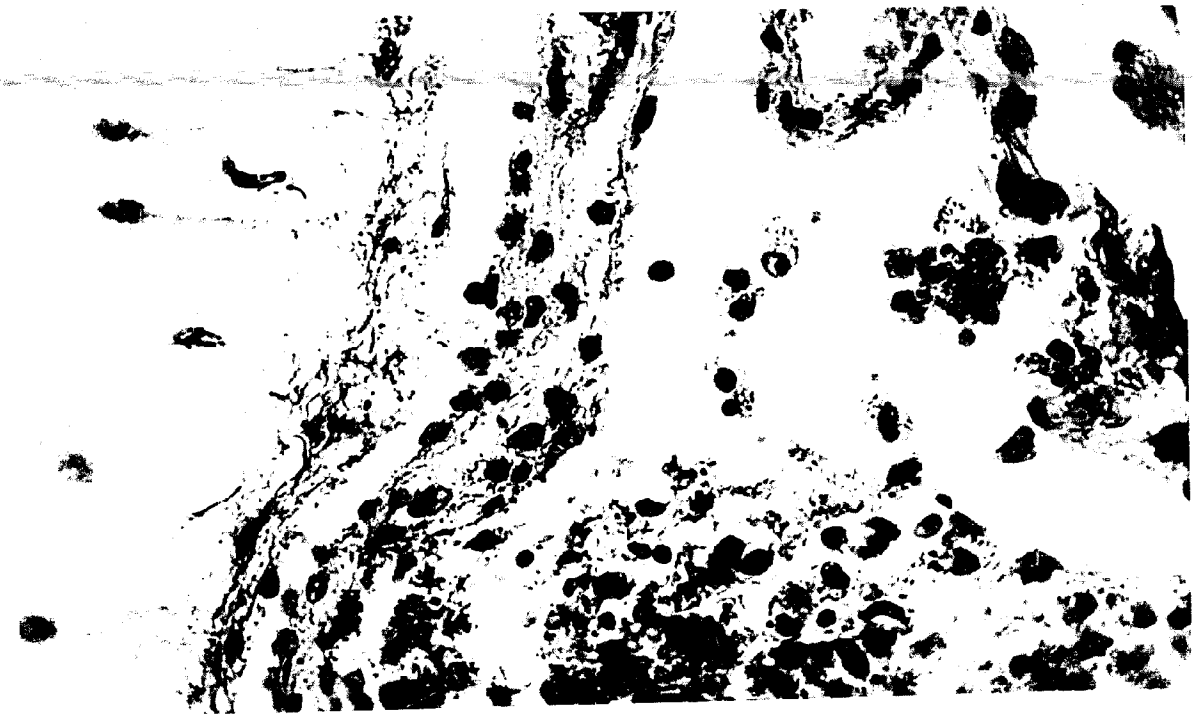
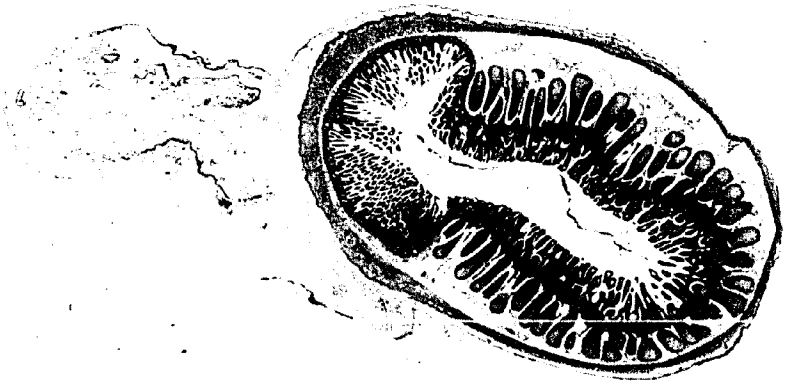


Fig. 49. Case 7406.

Intestine showing serosal involvement due to peritonitis.

Fig. 50. Case 7406.

Liver showing necrosis near capsule. A case of acute peritonitis. (H & E), X 116.



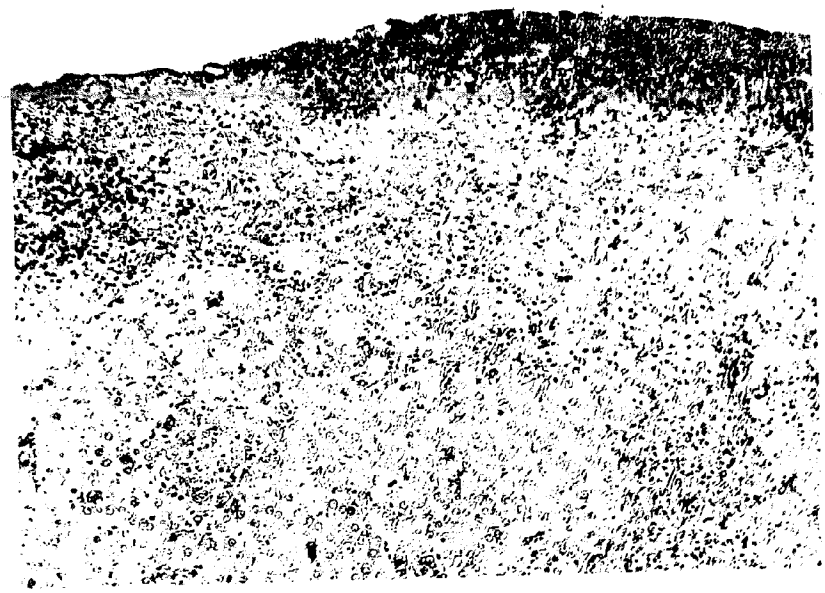
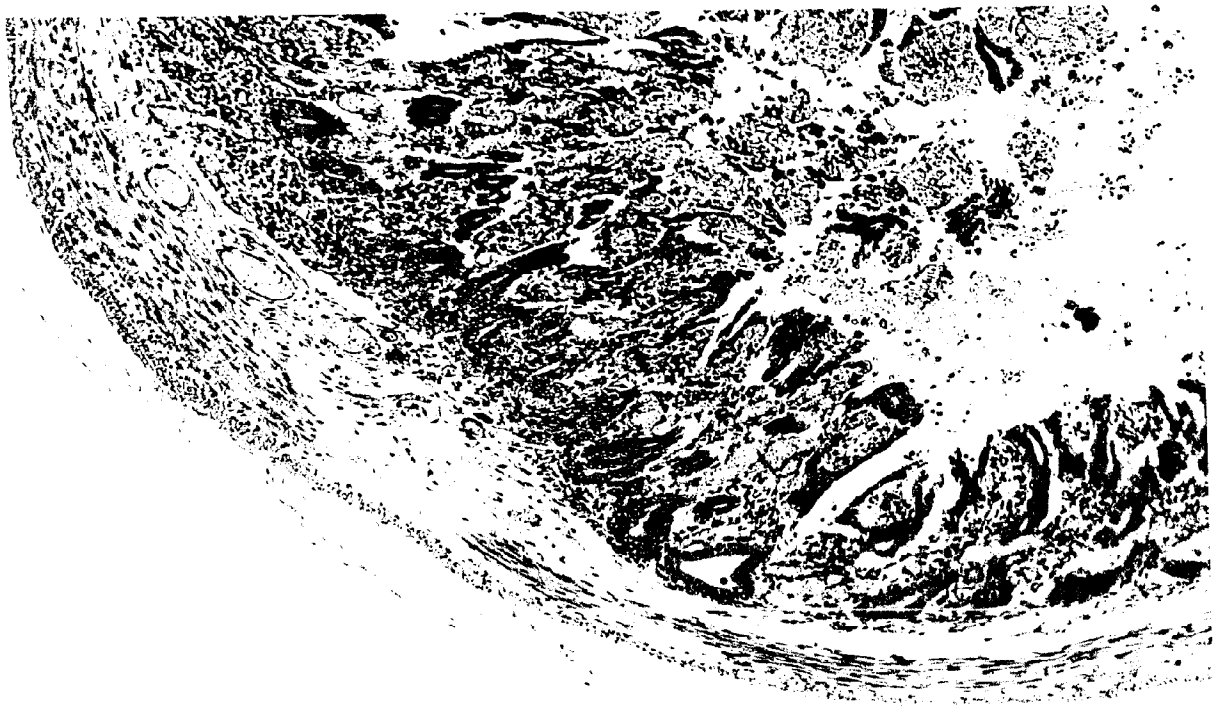


Fig. 51. Case 7336.

Intestine showing hemorrhagic enteritis. (H & E), 60.

Fig. 52. Case 7344.

Stomach showing ulceration. (H & E), X 15.



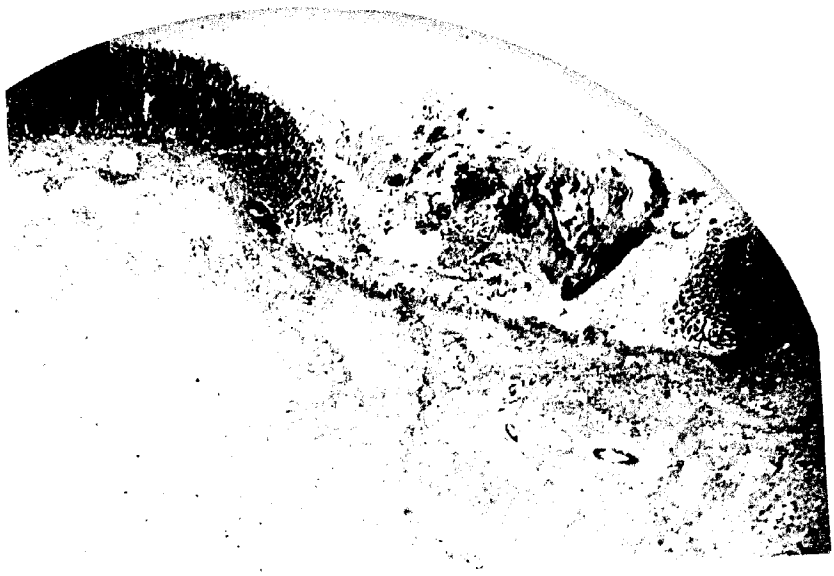


Fig. 53. Case 6598.

Regenerating cardiac muscle fibers with various stages of mitosis. Cell of this kind has been called "Anitschkow myocyte". (H & E), X 900.

Fig. 54. Case 6598.

Heart showing cardiac muscle giant-cell. Note the cell with two or three nucleoli in each nucleus. Cell of this kind has been called "Aschoff cell". (H & E), X 900.