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THE ETIOLOGY, MORBID ANATOMY AND BACTERIOLOGY

OF SCARLATINA.

by

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OF SCARLATINA.

Scarlatina has been known to the medical profession for upwards of six-hundred years, and one is almost tempted to say that our present knowledge of the cause of the disease is as limited as it was in the time of Michael Scotus ⁽¹⁾ (1250 D), Ingrassia (1560 D), Gregor Horst (1624 D), and other physicians of the early periods of medical science.

According to Hirsch ⁽²⁾ the disease must have been known as a separate one for many years before it was described by the medical profession, and this seems not improbable. In the earliest description Measles and Erysipelas were classed with the disease; and those diseases occurring in conjunction with it were looked upon as types. It is to Sydenham and Morton that we are indebted for its earliest description, and there can be little doubt that it is a disease which possesses characters entirely its own.

Locality.

Europe may almost be called the home of the disease, and in no other continent except the



northern portion of America is it so prevalent.

Sex.

Infection seems more common in the female than the male sex, but when one considers that there are more women than men, and that there are slightly more girls than boys after infancy, that would greatly account for the greater number of female patients one comes across. According to statistics mentioned by Johannessen⁽³⁾ the relations are males 49.2%, females 50.8%. The Metropolitan Asylums Board statistics for 1905 give males 8021, females 8937. On the whole one may say that the sexes are affected equally and the disease is slightly more fatal to the male (3.4) than to the female (2.9) sex.

Age.

The disease may be said to be confined to childhood, youth, and adolescence, so rarely do we come across cases in advanced life. The question then arises: is not this due to the fact that a large proportion of the population are affected in early life? To answer this question one is compelled to refer to an outbreak occurring in some country in which the disease had not previously appeared. Much light may be gained from the figures

quoted by Hoff⁽⁴⁾ in an outbreak in the Faroe Isles. "Out of 343 inhabitants of Thorshavn up to 20 years 193 were attacked with Scarlatina (56.3%): out of 582 inhabitants of Thorshavn over 40 years of age 44 suffered from the disease (7.6%)." These figures are reliable and seem conclusive that immunity is conferred by age. In my series of cases I have had no one over 40 who had genuine Scarlatina, but this is of little importance when one takes into consideration the large proportion of the population of this country who are infected in childhood and youth. Much interest attaches to the age in fatal cases. In an infectious hospital it is very rare indeed to see the death of an adult from scarlatina. Of 274 patients over 7 years of age, under my care, there was one death. The case was that of a woman aged 25 who at the time of her admission was so ill that death was expected within 24 hours. The highest mortality is among children under 5 years of age. And the older the child, the greater chance does it seem to have of recovering.

Race.

Of over 1000 cases under my observation I have had various nationalities, and as regards the susceptibility of races there is general agreement that all are equally liable to infection.

Wounds.

The picture of his illness, from a post mortem wound, and described by Von Leube himself, is of great interest. The post mortem was conducted on a case of Scarlatina, and ten days after having had a wound during the operation the symptoms of Scarlatina were manifest. Over and over again has my attention been drawn to the importance of burns as an indirect cause of the disease. It seems very probable that by producing a raw surface, the natural protection afforded by the skin is lost, and the infection gains an entrance into the system by the wound. In one of my cases infection occurred three days after an incision had been made at a general hospital for inflamed cervical glands.

Surroundings.

When one considers the insanitary conditions under which the poor live, and the lowered vitality of their tissues, it seems remarkable that the disease should make itself felt no more among them than among the richer and well to do classes. The evidence on this point from the report of the London epidemic of 1868 is quite definite. And though other opinions have been advanced, they have not altered its importance. The report states "The officials of the City Sanitary Department unite in

the statement that the wealthy suffered as much and more than the poorer portion of the population."

Incubation period.

The common figure given is from 2 to 7 days, but this is certainly far from being accurate. In investigating cases with the Medical Officer of Health for Willesden we had definite proof of the period covering 9 days and sometimes 10. In hospital, it is difficult to know the exact period, for patients are admitted with histories which are mostly very indefinite. But one occasionally comes across cases which are wrongly certified and contract the disease in hospital. One such case I shall mention. The patient, a girl aged 15, was certified as suffering from Scarlatina and was admitted during the night and sent to a Scarlatina Ward. On examining her on the following morning I found the fauces and tongue negative and absence of an Erythema or temperature. She was a very emotional girl and the blushing on her face when approached might very likely have led to her condition being wrongly diagnosed. On the nineteenth day, and two days after being up, she had a temperature of 102, sore throat, vomiting and a general Erythema. In other cases I have observed the symptoms occurring a few days before being up, and about the end of the

third week. It is impossible in these cases to give an opinion as regards the period of incubation, for the beginning of the infection is unknown. According to Murchison's figures he has been able to demonstrate the period as varying from one day to six days, and Hagenbach from one day to over twenty. I have seen no record of the histories of the cases by which they arrived at their conclusions, but as far as my experience goes, the periods of over twenty days, which are given, are doubtful. It is quite permissible that, since infection depends on the reaction of the tissues and the virulence and quantity of the poison, one person may receive a more virulent dose of the poison or may have a lower tissue reaction and so take a shorter time to manifest the symptoms than another whose tissue reaction is high and whose dose of poison is small and less virulent. But it is most unlikely that the periods would differ so much as from one day to six weeks as some writers have tried to make out. If the cause of the condition is due to an organism, or more than one organism, then it would be more reasonable to expect a certain amount of uniformity of the variation of the period, although the germs may vary in their virulence and tissue reactions of individuals vary. After looking through the writings of various authorities the period that seems

to me conclusive is the one of ten days recorded by Von Leube, and in his case the evidence is definite since we have proof of the poison gaining an entrance into the system by a wound on the finger when performing an autopsy on a case of Scarlatina.

Some writers are of opinion that wounds shorten the period: others that the period is shorter when infection is caught from the beginning of a case of Scarlatina, than during convalescence: and, further, that the period in epidemics is shorter. These statements might all be true and they may be explained wholly on the ground of a lowered vitality of the patient and an increased virulence of the organism rather than the mere presence of a ready means of entrance for the poison by a wound, or the mere fact of an epidemic being present.

Pregnancy and Puerperium.

In quite a recent text book on Midwifery I find it stated that "pregnancy seems to prolong the incubation period, but this may be because germs remaining about a house may attack the patient only when she reaches the puerperal state - a very dangerous state". It seems evident that this statement is due to the confusing by Malfatti, and even the London Obstetrical Society of 1876, of Puerperal Septicaemia with Scarlatina. What proof is there

that pregnancy confers immunity? for that must be gathered by its supposed power to lengthen the incubation period. Rather than believe that pregnancy has an immunising influence on the condition, I cannot but agree with those who look on Scarlatina as dangerous to pregnancy, and that given a severe case abortion is the rule. As regards its dangerous character in the puerperium genuine Scarlatina never influences the course of childbed illness unfavourably, and there is little doubt that the cases mentioned by Malfatti and others were Septicaemias: thus "the more marked the odour of the Lochia was in the beginning, the more serious proved the disease"; again "it was not rare for the Scarlatina which was then epidemic in Vienna to assert itself by no other symptoms than sore throat and fever, without any evident eruption". It may be doubted whether there ever was such a condition as Epidemic Puerperal Scarlatina, and certainly the number of persons affected in our day with Scarlatina during the puerperium are very few. Of 1600 puerperal patients in the ⁽⁷⁰⁾Berlin Obstetric Clinic there were only three cases, and of 1461 women who died in child-bed in Norway only 0.2% had Scarlet Fever. In my series of 184 adult females suffering from Scarlatina, 2 cases occurred during the puerperium. One patient, Mrs S., was probably infected by her children who

were admitted in hospital some days before herself. On the fourth day after labour she had headache, sickness, vomiting and sore throat, and on the fifth day she was admitted with a typical punctate erythema, angina, and rise of temperature. Her lochia was at no time offensive, was never increased in amount, and disappeared at the usual time. She was discharged at the end of the seventh week after having desquamated in the usual way. The infant who was in the ward with her was not affected. The history of the other case differed in no way from the one mentioned except that the illness began on the ninth day after child-birth.

Fomites.

It has been strongly affirmed that clothes, letters, and even books, are sure conveyors of infection, and, though I do not doubt the accuracy of many of the cases mentioned as proof, yet I am strongly of opinion that infection is not as easily carried as is generally supposed. I have had doubtful cases of Scarlatina remaining in Scarlatina wards over three weeks which proved eventually not to have been cases of Scarlatina, and which were discharged without having contracted the disease. Cases of Diphtheria, Typhoid, and other diseases have been received by nurses and physicians receiving

scarlatina cases: no gowns were worn and no attempts at disinfection made, and there was not one case in 1,000 which contracted scarlatina. Patients suffering from influenza and other diseases have remained in scarlatina side-wards, have been attended by doctors working in scarlatina wards, and have not been infected. Nurses and doctors have worked in scarlatina wards and have not contracted the disease. It is very rare indeed that the general practitioner carries infection from his fever cases to his other patients, and in the few isolated cases that have occurred it is questionable whether the infection was carried by them. Until one knows more about the poison of scarlatina one should be guarded in their opinion as regards what is or what is not a means by which infection is carried, but I am prepared to say in the meanwhile that the fomites which should be considered infectious are those which are part of the apparel worn by the infected patient or such fomites as were in direct contact with him. The risk of a healthy person whose clothes had not been in actual contact with a scarlatina patient, carrying infection to another, is infinitesimal.

Milk.

We are greatly indebted to R. G. Freeman for
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the way in which he has shown the means by which

milk may carry infection, and though he failed to find an organism in milk relating to scarlatina, yet his contribution to our knowledge on the subject is none the less valuable. According to Freeman most of the epidemics traceable to milk were caused by milkers or persons having care of the milk.

⁽¹²⁾ Hall three years later (1899) found that infection did not occur to any extent in countries where children were not given cow's milk as food or where children lived on the breast until three, four, or more years old. He also mentioned that where ass or goat milk is used infection is unknown. These investigations show plainly that milk, and certainly cow's milk, is a carrier of infection: but the statement made by Klien, that lesions on the udders of cows may be considered as the primary cause, is to be looked upon as most unreliable.

Transmition of Infection. When? How?

⁽¹³⁾ Vogl in his experience in the Munich garrison epidemic considered that direct contact during the incubation period, and before the actual appearance of the disease, caused infection. ⁽¹⁴⁾ Trousseau reported on a case in which infection occurred a few hours after scarlatina had developed. Given then that a case is infectious from the incubation period, when may it be considered to be free from infection?

According to many one is infectious from the onset of symptoms to the tenth week; but recent investigations show that this is anything but correct. Of 153 cases discharged from the Northern Hospital and which were responsible for "return" cases, 16 were detained over twelve weeks. And further, one case, who during illness had mucous discharge, gave rise to infection after being detained in hospital 18 weeks. (1905 Reports - Metropolitan Asylums Board.) Various opinions have been advanced as to when the possibility of infection is greatest, but before one should make any positive statement they should at least know something about the nature of the infecting agent. If the condition is due to an organism then one would expect that the germ would have its greatest virulence during the height of the attack or before convalescence, and after that it would gradually lose its toxic power until arriving in a suitable host. Certainly it cannot be the desquamating period for of 2,213 cases discharged desquamating from the Northern Hospital in 1905, 2.21% were productive of "return" cases, and of 1,931 discharged after desquamation 2.12% gave rise to "return" cases. Desquamation should be considered of importance to us only as a helpful sign in doubtful cases in concluding that the patient had an erythema, which together with other signs and symptoms, point strongly

to the case as probably one of scarlatina: but in no way should it be made the determining factor as to whether a case is infectious or not. ⁽¹⁵⁾ Rupperecht considered that infection was got from the secretion from the Pharynx or Bronchial passage. After performing a tracheotomy for Diphtheria and Scarlet Fever he resorted to artificial respiration. Through the tracheal canula he forced his own breath, and receiving some of the expressed air and mucus from the patient in his own mouth was himself affected. In Von Leube's case infection was got from a wound. If Von Leube's case is true, and there is no reason to doubt it, from what source did the poison come? I do not think one is justified in saying that it did not come from the blood because we know of no case in which the mother had transmitted infection to the foetus in utero. The very similarity in the erythema of scarlatina and the colour of the skin in the new born child, together with the desquamation common to both, may often lead to a failure on the part of the physician to diagnose the one from the other, and though it is quite true that the cases put on record of scarlet fever in the new born child, contracted when in utero, are absolutely unreliable and of little consequence, yet I feel convinced that it is to the blood that we must look to find the

cause; granted that we are of opinion that the nature of the infection is an organismal one. Of 6,493 cases discharged from the Northern Hospital in 1905, 2,349 had mucous discharges at some time during their illness, which included rhinitis and otorrhoea, and although in-patients of the hospital for three months they gave rise to 2.68 "return" cases as against 2.21% and 2.12% from cases desquamating and finished desquamating which were in hospital six weeks. This points to mucous discharge as a definite agent of infection, for even though detained twice as long in hospital the mucous cases gave rise to 20% more "return" cases than did the others. It is probable that infection is carried by the dust of dried up secretions, and though this may be reduced to a minimum in well kept hospitals, yet it is possible that it plays an important part in infection in ordinary dwelling houses. Most are agreed that the clothes of a patient suffering from the disease are infectious; but the origin of the infection no one knows. Probably it may be due to mucous secretion which contaminated the clothing; but I do not see why the patient's own sweat may not be looked upon as a probable cause, for it is more likely that contamination of clothing will occur through sweat than from the dripping of mucous secretion on them: and certainly this is true for adults.

Absorption of infection.

At the outset one may say that the channel by which the infection gains the body is not definitely known. Sore throat has long been looked upon as one of the most important symptoms of scarlatina, and in other words one may say that whenever infection occurs symptoms and signs point to the presence of an irritant in the faucial membranes. Since, therefore, in the mildest cases one sees a congestion of the posterior part of the soft palate and of the uvula, and in the more severe cases the fauces are covered with a pultaceous exudate under which is often ulceration, it is reasonable to expect that the poison gains access to the system from the fauces in the majority of cases. The Pharynx also may play an important part in the passage of the irritant into the system, for like the fauces the mucous membrane there is often congested. Some writers have also mentioned the intestines as a probable means of entrance, and this is not improbable since Gastro-Intestinal symptoms are commonly present in the early manifestations of the disease. That when there is destruction of the skin by burns the irritant may gain access to the system, I feel quite confident, for I have observed many instances of the kind. One such case I received but a few days ago.

A girl G received a scald on the 25th and on the 1st she had angina, a temperature of 101, and a bright punctate erythema generally over the body.

Susceptibility.

I have already mentioned that all races are equally liable to infection, but in my experience fatal cases are commoner among the fairer races. The Park Hospital, in which a large number of Hebrews are treated, brings this out quite clearly, and their report for 1905 shows a Jewish death rate of 1 in 40 to 1 in 23 among other races. Thomas brought forward many instances to prove that some families are more susceptible than others, and I shall mention the observations of Trousseau who was a supporter of family susceptibility which will at a glance prove that the theory exists on very unsubstantial ground:- "The malignancy limits itself in a measure to a single hearth and in such cases the disease is malignant for all persons that live within its circle". "Scarlatina epidemics may assume this character only for a single family." "I wish to take the opportunity to recall the sad case, recently reported in the English papers, of a clergyman of York who, within one week, lost all of his six or seven children." I have often had whole families in my wards, and in some instances the type

was more severe than others, and further I have had the domestic servants along with the families. This seemed only to prove that some homes encountered the irritant in more virulent form than others, and the domestics, being members of the homes, were themselves equally attacked. Observations by Thomas and others prove that susceptibility varies, and that this is really so, ⁽¹⁷⁾ R. Forster reports a case in which a man nursed his child through a severe attack and was not affected, but was infected later on calling to inquire of a friend's child who was ill with the disease. That this is quite possible is evidenced by the fact that often it is months after Nurses begin their duties in scarlatina wards that they are infected.

Immunity.

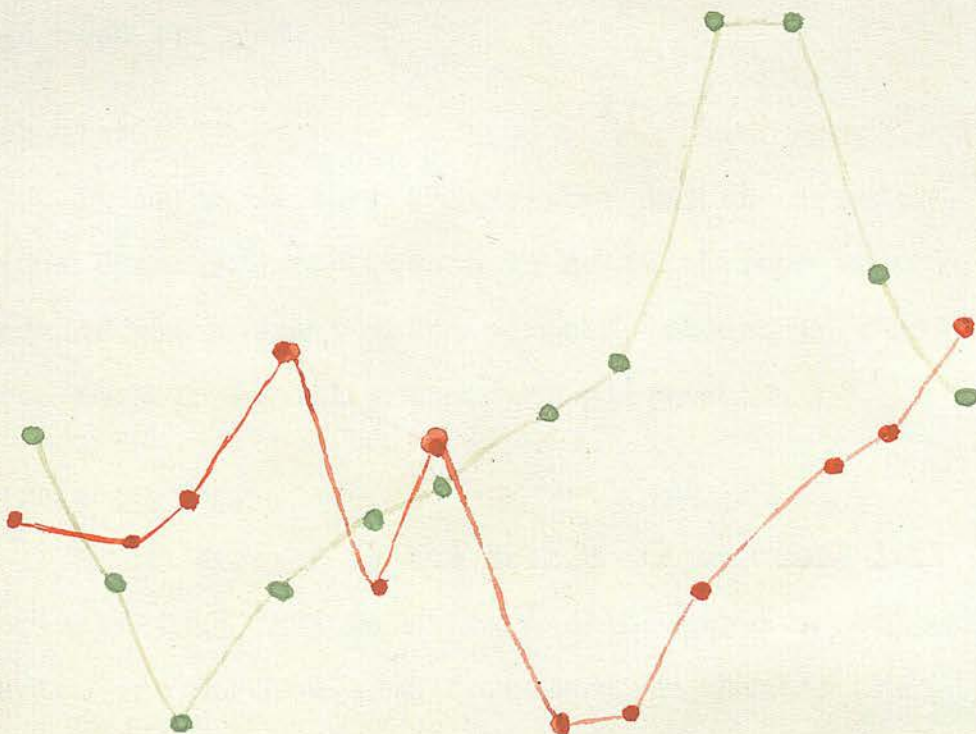
This is confirmed on one by years, and secondly by a previous attack. I have already mentioned statistics and have given my own experience, which more or less show that scarlatina is a respecter of old and middle age. It is confined almost entirely to individuals of from six months to thirty-six years of age. Many cases of second attacks have been mentioned by Thomas, ⁽¹⁸⁾ Murchison, ⁽¹⁹⁾ and others, but in those cases which have come under my observation the patient invariably had an indefinite previous

attack or the third or second attacks were not scarlatinal at all. Before venturing on a definite opinion I would prefer having more evidence one way or another; and I am now only prepared to say that second attacks may occur and less probably third ones, but that they are extremely rare.

Season.

The disease is at its lowest from about the middle of February to the middle of March, and at its highest from the middle of October to the middle of November. The percentage of mortality is highest in mid-spring and early winter and lowest in June and July. Represented in curves the appearance is as follows:-

Jan, Feb, Mar, Apr, May, June, July, Aug, Sept, Oct, Nov, Dec.



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 Some writers have tried to prove a relationship between temperature and mortality and also temperature and number of cases of infection. I cannot myself see how temperature can influence either, and it seems very probable that the greater number of cases occurring in Autumn are due not so much to the temperature prevailing as to the dryness of the soil. The soil is at its driest in autumn and so far as we know dust is the most likely medium by which infection is conveyed to the individual; therefore we may say, though with some caution, that the period of greatest infection is probably proportional to the dryness of the soil and the amount of dust that circulates in the air. Why the percentage of mortality should be greatest in winter we do not know and so far as I can gather no reasonable explanation has been offered.

Schools.

It is there that the greater number of infections occur and the reason is quite obvious when we know of the contact which normally occurs in children both in school and on the playground.

Morbid Anatomy.

In no disease is the morbid anatomy more indefinite than in scarlatina and the appearances post mortem are more or less dependent on whether the

patient developed complications during illness or not. Whether there has been lack of some definite description of the appearances after death, both in cases with complications and cases without, the fact remains that physicians whose daily work is among scarlatina patients seem even to-day to err in their treatment of such cases through a want of pathological knowledge. I shall consider each organ separately giving firstly the appearances in non-complicated cases or those which are simply overwhelmed with toxæmia and which die within a few days without any other symptoms than rise of temperature, sore throat, and unconsciousness, and secondly the appearances in those cases in which there may be one or more complications.

Brain and Meninges.

The dura is never involved and all that is seen in the pia-arachnoid is a distention of the veins with dark fluid blood. The veins of the gray and white matter are also usually distended. The cortex is glistening, pale, soft and oedematous. The white matter is at times so soft and oedematous that it might well be likened to whipped cream. The basal nuclei are pale, soft and oedematous. The lateral ventricles are not dilated and one would expect that, when they are never greatly distended with fluid.

The choroid is pale. The ependyma is usually quite smooth and healthy. In neither the subdural nor the pia-arachnoid spaces is there an excess of fluid and the diffluence of the brain tissue is to be regarded as a general inflammation resulting from a profound toxæmia. In all grave cases of scarlatina stupor is a fairly early manifestation and the more profound the toxæmia the more marked is the stupor. When death occurs in such cases one finds on examining the brain a general softening of the tissues, both gray and white. The greatest attention should therefore be paid to this organ in the severe types of the disease and the slightest tendency there is to stupor should be the index of the morbid process beginning in the brain, and the index, therefore, of the urgency of appropriate treatment. The white and gray matter of the cerebellum is affected just like that of the cerebrum, being usually soft and oedematous. In complications of scarlet fever one may find in the brain abscesses and meningitis. These are so common that they should certainly have a prominent place in the brain pathology of the disease.

Abscess.

In giving an instance of abscess in the brain, I shall mention a few facts in the clinical history before describing the morbid process. Albert W.

was admitted on August 1st with a temperature of 102.6, congested fauces and a marked erythema. On the 4th he had Rhinorrhoea. On the 8th marked adenitis of the cervical glands; 9th pain in both ears; 10th double otorrhoea; 12th otorrhoea scanty; 22nd emesis and profuse otorrhoea; 23rd slight retraction of head, left internal squint, profuse ear discharge, flexed right arm, and twitching of left side of face; 25th lumbar puncture made, fluid clear, no microbes on smear or culture; 26th coma; 29th right arm and leg less contracted, and paralysis of left arm and leg; September 1st left hemi-plegia and right ptosis; 7th death. On examining the brain the meningeal veins were distended, the dura healthy, and the inter-spaces showed neither excess of fluid nor turbidity of fluid. The pia-arachnoid was intact and gave way only on removal of the brain from the skull. The whole of the right temporo sphenoidal lobe was a huge cavity filled with pus. A small abscess was also present in the cerebellum, and occupied the vermis at about its middle. The roof of the middle ear was necrosed and perforated, and the chamber was filled with pus. Abscess in the brain in scarlatina is always due to middle ear disease and the common sites are the temporo sphenoidal lobes and the lateral hemispheres of the cerebellum.

Illustrative of two cerebellar abscesses:-

Victor R. was admitted on January 15th with all the signs and symptoms of scarlatina. On the 23rd he had double otorrhoea and rhinorrhoea; February 8th temperature subnormal and emesis; 24th temperature rose to 102; 26th lateral nystagmus and double otorrhoea; March 3rd coma; 5th death. On removal of the vault of the cranium there was a large flow of fluid from the pia-arachnoid space. The meningeal vessels were distended, and a large quantity of purulent fluid escaped on incising the tentorium. The right cerebellar hemisphere was excavated and filled with pus, and the outer and inferior boundaries of the cavity were formed by meninges; so great was the destruction of the brain tissue. The roof of the right middle ear was destroyed, the cavity was filled with pus, and a firm decolourised clot present in the lateral sinus.

H. R. was admitted on January 8th with the usual signs of scarlatina. On the 13th left otorrhoea developed and the temperature fell from 103 to 101; 20th right and left otorrhoea and temperature 103 to 104; 22nd acute mastoid disease on both sides; 25th swelling over left mastoid subsided; February 19th emesis and double otorrhoea; 20th delirium; 21st antrum of right side was opened, pus found, and cavity syringed. Death occurred the

same day. The autopsy revealed thrombosis in left lateral sinus near the jugular foramen. Complete excavation of left cerebellar hemisphere. No pus in either middle ear, and no obvious necrosis of either roof.

Meningitis.

I have placed it in a position secondary to abscess, firstly because it does not occur as often, and secondly because in the instance I am about to mention the most I can say is that scarlatina was only a remote cause. In other cases on which I have made post mortem examinations I have been even more perplexed to find an origin in some diseased process than in the case I am about to mention.

Grace C. was admitted on August 11th; 13th right cervical glands greatly enlarged, and from that date to the 20th the temperature varied from 101 to 103; 21st abscess incised and temperature normal; 30th temperature 100.6, emesis, convulsions, unconsciousness, irritability and restlessness; 31st pupils did not react to light, eyes fixed and turned to left, twitching of muscles of left side of face, twitching of limbs of right side, kernig positive, no head retraction, wound in neck healed; Sept. 2nd convulsions, twitching of face on right side and limbs of left side; 3rd general convulsions, coma,

and death. On examination the entire pia-arachnoid space was filled with a thick creamy exudate about $\frac{1}{2}$ inch thick and which enveloped the entire brain. The gray and white matter were soft and there was no obvious middle ear disease. Cultures taken from the brain showed a mixed infection of streptococci and staphylococci. The only inference I could draw in this case - I had only charge of it when convulsions began - was that meningitis was due secondarily to the coccal affection of the neck, and that this suppurative adenitis was brought about by the passage of the streptococci along the lymph passages from the primary focus of inflammation in the fauces. It is not unreasonable to suppose that the organisms reached the meninges by passing from the inflammatory lesion in the neck along the lymphatic paths, or probably by means of the venous system. Though how many hundreds of cervical abscesses are evacuated in cases of scarlatina without any further infection of an organ?

Pericardium.

Inflammation of the pericardial sac is rare, and neither clinically nor post mortem have I come across an instance of pericarditis complicating scarlatina. The most I have seen are minute haemorrhages in the sac, and these are usually situated in the epicardium.

Heart.

In the types of scarlatina in which there is a high temperature and delirium, and in which death occurs in a few days, this organ is never the seat of any greatly marked muscular degeneration. I shall give a clinical picture of the circulatory system and shall then describe the appearances post mortem.

Florence P., aet 24, was admitted on January 6th with temperature 102, partially suppressed rash, ulcerated fauces, dry and dirty brown tongue, offensive odour of breath, rapid, thready and irregular pulse, inaudible heartsounds, slightly dilated right heart, delirium and unconsciousness. On the following day coma was marked and death occurred during the night. On examination the right auricle was distended and filled with dark fluid blood. The tricuspid orifice admitted four fingers readily. The right ventricle also was distended and contained fluid blood. The left chambers were empty and not dilated. The mitral orifice admitted two fingers. The valves were quite healthy. Minute haemorrhages were present in the endocardium of both right and left chambers. The muscular tissues showed loss of tone and were pale and flabby. Probably fatty degeneration had occurred.

In cases of scarlatina in which toxaemia is short and profound, or extends over a long period, cloudy swelling is present but never extensive fatty degeneration. Loss of tone in the heart muscle is rare and in this case it is rather the exception to the rule. The muscular tissues are usually firm and of good tone. Haemorrhages in the endocardium are present in about one half of the cases.

The danger signals in these cases are always irregularity and feebleness of pulse, together with the appearance at intervals of an ash grey colour of the face. How is the heart responsible for this? The myocardium never shews extensive degeneration: the muscular tone is usually good, and dilatation is usually confined to the right chambers and is never very marked in the left. The explanation is that the heart has to act so rapidly to aerate the blood that it has no time in which to fill up thoroughly before driving its blood through the system. Both the quantity of blood and the force of the stream are as a consequence diminished. As breathing becomes shallower and aeration of the blood more imperfect, the heart in attempting to make up for the deficiency pumps faster and smaller until it is perforce obliged to give up the unequal struggle. The pulse corresponding to this heart is a small one and one which tends to grow smaller. Then some of the

beats become imperceptible and finally all are imperceptible. The colour of the face is a warning by the tissues that their life is endangered by the growing shortness of nourishment. It is clear, therefore, both from a pathological and a clinical point of view, that in these cases the need of the heart is that the toxaemia, together with the great burden thrown on it by the respiratory system, should be removed, and that cardiac stimulants are of little value, if not absolutely worthless.

Endocarditis.

As a complication of scarlatina it is rare, and in over 1000 cases under my observation it was absent. In one case I thought that the double mitral murmur that I heard was brought on by the scarlatina toxins, but I was not long in doubt for I obtained later a definite history from the parents of a previous rheumatic attack with heart trouble. I am quite convinced that I was dealing with an acute attack on a chronic one, and it is very probable that many of the scarlatina endocarditis cases recorded would in no way differ from mine, if only one could obtain a correct previous history.

Myocarditis.

Myocarditis is uncommon and I have seen no instance of it in any of the post mortem examinations

I have made on cases of scarlatina.

Fauces and its surroundings.

The tongue is either parched and brown or thickly coated. The posterior part of the soft palate and uvula may be swollen, congested and oedematous, coated with exudate, or ulcerated, or all combined. The same may be said for the pillars of the fauces and the tonsils. The tonsil of Luscha is often enlarged, but never sufficiently to block respiration. The mucous membrane of the pharynx is deeply injected and swollen. The mucous membrane of the larynx may be pale, slightly swollen and oedematous, or there may be simply a slight injection. In no instance have I observed involvement of the vocal cords, and though there is usually a slight swelling of the mucous membrane in these parts, it is never sufficient to seriously influence respiration.

Lymphatics.

There is general involvement of the lymph system and according to some writers the glandular enlargement precedes the appearance of the rash. In the great majority of cases glandular enlargement is present with the appearance of the rash, but it is also present in erythemas which are not of scarlatina origin. Taken together with other signs and symptoms of scarlatina, the involvement of the lymph

system is of diagnostic importance, but taken by itself it is of worthless value.

Pleurae.

Usually healthy but occasionally minute haemorrhages are present. Pleurisy is present in a small percentage of cases and is to be looked upon as a complication and a rare one.

Lungs.

Acute congestion is common; sometimes it involves all the lobes, and at other times the lower lobes only. This together with areas of collapse are all that one finds in the uncomplicated type of cases. The collapse is usually slight, and affects few or more areas of about $1\frac{1}{2}$ ins. in diameter, but it may assume large proportions and involve a whole lobe. Among pulmonary complications of scarlatina broncho-pneumonia ranks high. The areas of consolidation are usually haemorrhagic in variety and may be so numerous as to extensively involve both lungs. Acute bronchitis is also common and the bronchi affected are usually the medium sized and smaller ones, but there may be general involvement of them all. Lobar pneumonia is rare. Involvement of the lung and bronchi occur in the great majority of deaths from scarlatina, heart failure

in all such cases being purely a secondary condition to the respiratory inefficiency. The heart suffers whether there is toxæmia and only acute congestion of the lung, or whether there is pneumonia. In the first case from improper expansion of the air vesicles, inefficient aeration and shallow breathing, and the necessity of rapid compensatory cardiac action, and in the second from the want of air space in the lung and the necessity of rapid action to accomplish a sufficient aeration of the blood. In those cases in which the air hunger is intense, either through broncho-pneumonia, bronchitis, or collapse, it is marked in children by flapping of the alae nasi and recession of the ribs. These clinical signs should not be mistaken for obstruction of the higher air passages, and any attempt to relieve such a patient by tracheotomy must only bring on death earlier than it would otherwise have occurred.

Cellulitis.

In scarlatina the site of such inflammation is usually below the inferior maxilla, but it may be at the side of the face superficial to the parotid. It may occur early in the disease or it may occur late. The inflammatory process usually begins in the fauces, and from there spreads by the lymph

channels to the submaxillary lymph glands of the neck. The inflammation then pervades the deep tissues and might eventually find its way to the larynx. I shall mention the clinical history of a case and then give the appearances post mortem.

F. R. was taken to a general hospital because of his having a swollen neck. When the child was undressed it was found to have a scarlatina rash and an operation was not performed. It was admitted here on that day with a brawny swelling in the submaxillary region and the temperature rose from 99 on admission to 103 during the day. The temperature fell to 99 during the next two days and on the latter day the swelling showed signs of fluctuation. An incision was to be made on the following morning, but during the night the temperature flew up to 102.4 and there was marked laryngeal obstruction. Tracheotomy was at once performed but death ensued an hour after. On examination post mortem there was necrosis and pus formation in the deep tissues of the neck. The aryteno-epiglottidean folds were greatly swollen and there was marked tumefaction of the ventricular bands.

Liver.

This organ is usually normal in size though flabby and somewhat anaemic. Often scattered over

the surface of the organ are yellowish areas of about the size of a bean, superficially situated, and not extending in the liver tissue for more than one eighth of an inch. Cloudy swelling with some slight fatty change may occur in long continued fever, or in short profound toxaemia, and congestion is not infrequent.

Spleen.

It is commonly found to be about one third larger than the normal size, of normal colour and firm consistence. Occasionally there is acute congestion or haemorrhages in the splenic substance and an enlargement of the follicles is almost always present.

Kidneys.

The capsules usually strip quite readily. The organs are generally somewhat larger than normal and flabby, and the superficial veins under the capsules congested. On section a striking feature is occasionally the marked anaemia of the pyramids which one finds. More often one gets a congestion of the veins of the cortex and in some cases it may be very marked. The tissues of the cortex are usually pale, and at times may be markedly so, swollen, and opaque.

Other organs.

The stomach, intestines and other organs have been grouped together, for they give rise to no pathological changes which are constant or which are characteristic of the disease, e.g. the stomach may show swelling of the mucosa, catarrh and petechial ecchymoses, or it might show no morbid condition. The intestines might show congestion of the veins in some areas and swelling of the Peyer's patches, or there may be nothing to be seen.

Bacteriology.

From the time that bacteria were known to give rise to disease scarlatina has been looked upon as one of the diseases which were most likely to be due to an organismal infection. And it is not from lack of observation that the direct cause of the disease is as little known now as it was in the remote ages, for workers on the subject have been numerous, and among them may be numbered some of the best known bacteriologists of the present day. In this branch of the subject my desire is not to lay claim to the finding of the cause of the disease but simply to show the characters of the organisms which are found in the blood during life and post mortem, and to discuss generally their bearing on life in cases of scarlatina.

Blood was removed from 50 scarlatina cases. Eleven specimens were taken from the usually mild variety, and the remaining 39 from severe cases. In none of the 11 mild cases did I obtain an organismal growth, but growth occurred in 35 of the remaining 39 cases. The method of procedure was as follows:-

The arm of the patient, from whom a sample of blood was to be taken, was thoroughly sterilized in the region of the bend of the elbow. This was done about an hour prior to the operation, and fomentations left on for the remaining time. A metal syringe to which was attached a hypodermic needle was thoroughly sterilized and the hands of the operator were scrubbed in weak antiseptic solution. The arm of the patient was grasped by the left hand of the operator, and the needle was inserted obliquely into the median cephalic in a direction opposite to the flow of the blood current. On withdrawing about 5 to 10 c.c. of blood, portions were allotted to the media selected. The cultures were then placed in an incubator at 37 C. On taking samples after death the instruments were thoroughly sterilised and the blood removed from the right heart.

Of the 35 growths obtained there are no less than three families and eight distinct types. I shall place them in tabulated form and then discuss each type separately.

		Broth	Blood Serum	Agar	Gelatin	Potato	Milk	Gram Stain
Type A.	Small cocci arranged in bunches, also occurring in chains of 2, 3 or 4 cocci.	Turbidity	Greyish white growth	Greyish white growth	Not liquefied	Greyish white growth	Not changed	Stains
Type B.	Characters same as A.	Turbidity	Greyish white growth	Greyish white growth	Slowly liquefied	Greyish white growth	Coagulated Distinctly acid	Stains
Type C.	Arranged almost entirely in bunches of cocci	Turbidity	Greyish white growth	Greyish white growth	Rapid liquefaction	Greyish white growth	Coagulated Distinctly acid	Stains
Type D.	Arranged in chains of four or five cocci			Minute white colonies	Not liquefied	No growth	Coagulated Distinctly acid	Stains
Type E.	Short thick rods				Not liquefied	Thick yellowish grey growth	Not changed	Partially stained
Type F.	Same as E.				Liquefied	Thick yellowish grey growth	Not changed	Partially stained
Type G.	Same as E. and F.				Not liquefied	Greyish white growth	Coagulated acid	Not stained
Type H.	Rods varying in length but mostly short and thick				Not liquefied	Greyish white growth	Unchanged	Not stained

Type A.

This type, with type B., were present in the great majority of the 35 growths. The microscopic appearances of both of these types can be distinguished from the streptococcus only with great difficulty. The chains are small ones of two, three, four, or five cocci, and together with these are minute cocci clustered in small bunches. Sometimes the bunches are rather hard to discover and all that one sees are the small chains.

Type B.

This differs from A. only by its milk and gelatin reactions. Of the two types this one occurred in slightly more cases than B.

Type C.

This type differs considerably from A. but only slightly from B. Types A. B. C. are undoubtedly of one family, viz. the staphylococcus. A. may be represented as the weak, B. as the slightly stronger, and C. as the virulent branch of the family. I am not prepared to make any distinction between the three types by referring to one or other by special names; sufficient that they belong to the albus group and that A. and B. are attenuated forms of C. A. and C. could not have been obtained by accidental

infection from the skin for they were found in the blood ante mortem as well as post mortem.

Type D.

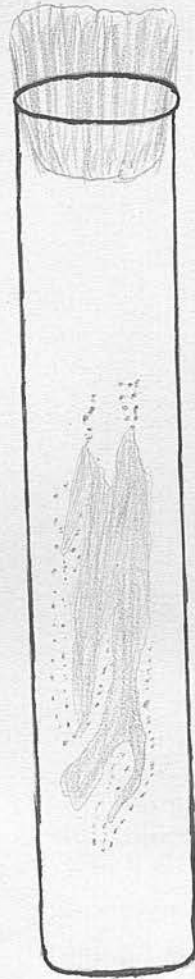
In only two of the 35 cases was this variety present and its occurrence was in pure growth. In both cases the organism was obtained from the heart blood post mortem. This type undoubtedly belongs to the streptococcus family, though the spread of growth on the surface of the gelatin was by no means typical of the streptococcus.

The Diphtheroids: Types E. F. G. H.

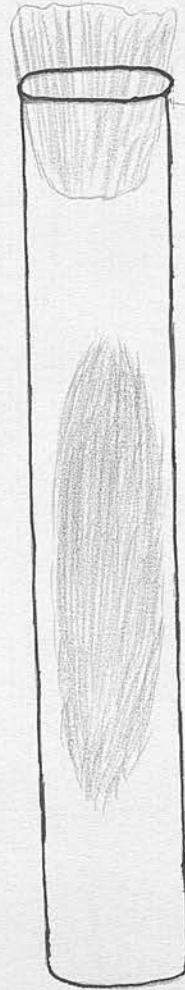
This group occurred in all the cases in which types A. and B. were present. The cultural behaviour of this group is not unlike that of the Diphtheria Bacillus, hence the term diphtheroid. This group has yielded four cultural types, though their variance from one another is so slight that a relationship is easily seen. They have been obtained from the blood ante mortem and post mortem from vaginal discharges and from cervical abscess.



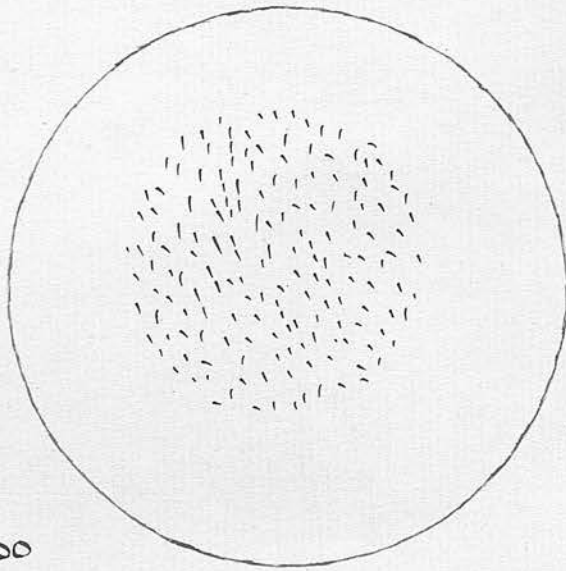
Type E Agar.



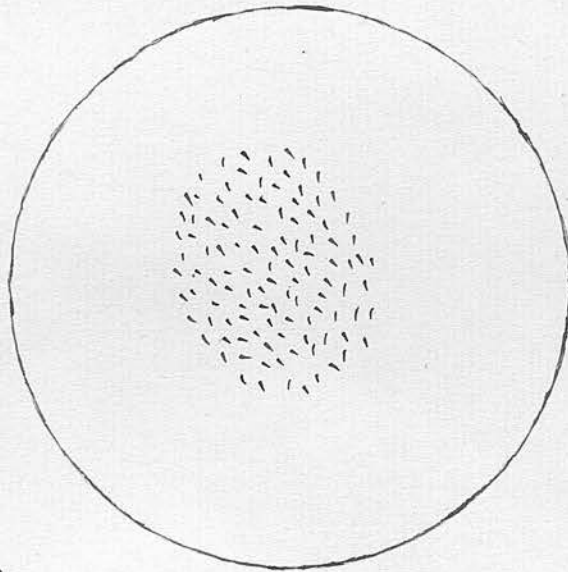
Type F Agar



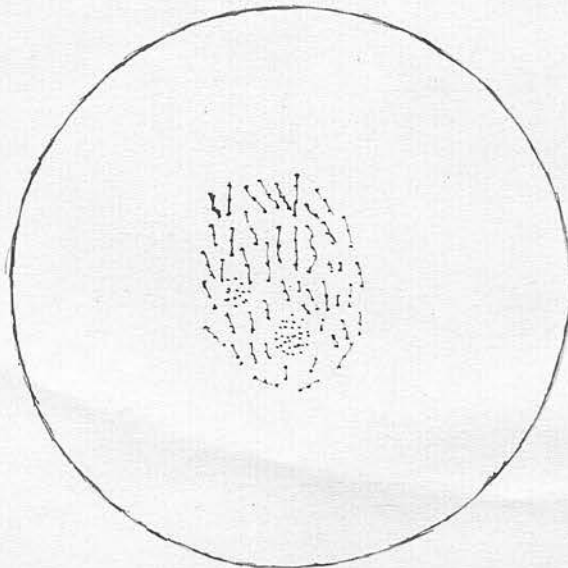
Type B Agar



Type E
x 1000



Type F
x 1000



Type B x 1000

ANIMAL EXPERIMENTS.

The inoculating experiments in

what one might expect in attenuated forms of

Staphylococcus Pyogenes Albus.

The experiments are not devoid of

diphtheroid group.

which a pathogenic effect is

injected subcutaneously or intraperitoneally

for as the

Type F. also

white mice when injected intraperitoneally

when injected subcutaneously in guinea pigs

observed in

guinea pigs

no instance did they seem affected by injected

from this group.

The relative

well to divide

relationship of the various types of organisms

of the scarlatina toxins.

In four such cases no

were found, either ante mortem or

mortem, and death in these cases might

attributed entirely to the specific

of the scarlatina toxins.

	<u>Guinea Pigs.</u>	<u>White Mice.</u>	<u>Rabbits.</u>
<u>Type B.</u>	Injected subcutaneously = negative result second month.	Injected subcutaneously = negative result second month.	Injected subcutaneously = negative result second month.
<u>Type E.</u>	Broth culture subcutaneously = no change in weight fifth day. Broth culture intraperitoneally = increase in weight 15 grammes fifth day.	Broth culture subcutaneously = decrease in weight 1/6th dram third day. Broth culture intraperitoneally = decrease in weight 1/3rd dram third day.	
<u>Type F.</u>	Broth culture subcutaneously = increase in weight 30 grammes fifth day. Broth culture intraperitoneally = no change in weight fifth day.	Broth culture subcutaneously = increase in weight 1/3rd dram third day. Broth culture intraperitoneally = decrease in weight 2/3 dram third day.	
<u>Types C. and D.</u>	Known to be pathogenic.		

The inoculating experiments in type B. prove what one might expect in attenuated forms of the *Staphylococcus Pyogenes Albus*. On the other hand the experiments are not devoid of interest in the diphtheroid group. Type E. is seen to have produced a retrograde effect on white mice, whether injected subcutaneously or intraperitoneally, in so far as their weights were decreased in both instances. Type F. also is shown to have had a morbid effect on white mice when infected intraperitoneally, though when injected subcutaneously no morbid effect was observed in the animals. So far it would seem that guinea pigs are not influenced by either type for in no instance did they seem affected by injections from this group.

The relationship of the various types to Scarlatina.

Scarlatina for bacteriological purposes might well be divided into four great groups and the relationship of the various types of organisms to each considered.

1. Cases in which the patient is overwhelmed with the scarlatina toxine and dies within a few days from the commencement of symptoms. In four such cases no organisms were found, either ante mortem or post mortem, and death in these cases might be attributed entirely to the specific action of the scarlatina toxines.

2. Cases which are of a severe type and which usually succumb to a general toxaemia after about seven, ten, or more days of illness. In these cases the streptococci are found in the blood.
3. Mild cases in which no organisms are found and in which the course of the disease might be attributed solely to the scarlatina germ.
4. Cases of moderate severity which assume a septic type, and in which manifestations of septicity are present in the form of otorrhoea, rhinorrhoea, and suppurating adenitis, etc. In these cases the diphtheroids and staphylococci are present in the blood. There is usually a long illness but recovery often takes place.

Group 1.

The absence of organisms in the blood either ante mortem or post mortem, the collapse of the patient even from the onset of illness, and the rapid pathological changes in the organs of the body seen after death, are sufficient proof that in these cases infection must be due to organisms or toxins of some specific kind and not to the ordinary pyogenic species. Whether the organism passes into the blood stream or liberates its toxins into it from the faucial membranes, one does not know; but that the faucial inflammation and ulceration due to the streptococcus and others of the pyogenic series

is only part of the role played by the specific germ, seems only too evident, since in the worse varieties of streptococcal throats the clinical picture is unlike that of scarlatina. What then is the relationship between the pyogenic series and this specific germ in cases of infection? A lowered vitality of the tissues is certainly the essential factor, for without that infection can be withstood (vide "susceptibility"). With a lowered tissue reaction, therefore, it is probable that the germ passes into the tissues, and then either continues onwards into the blood stream or liberates into it its toxins. The early sore throat is most probably due to the specific organism and not to the streptococcus or we would not have that uniformity of sore throat and rash which are so characteristic of one common cause. But the further destructive changes occurring in the fauces might well be placed to the credit of the pyogenes group. If on the other hand the early invasion of the throat was due to cocci and by their action a way was made for the entrance of the specific germs, then sore throat would be a symptom complained of days or probably weeks before the appearance of the rash. In this group the part played by the coccus is quite a local one for in none of the four cases from which blood was removed both

ante mortem and post mortem was it found, and it is especially this variety of case which compels one to believe that scarlatina is due to a specific germ common to itself and not to a septicaemia due to the streptococcus or some mixed infection.

Group 2.

These cases are commoner than those in group 1. Here the streptococci pass into the blood stream and the case eventually becomes one of septicaemia. In these cases the pathological changes in the organs are not so marked as they are in cases in group 1, and this points to the changes as being proportional to the virulence of the scarlatina toxins and not to the presence of the pyogenes group. In two cases in this group I obtained the streptococcus in pure growth from the heart blood post mortem. Whether staphylococci and diphtheroids may be found in the blood of such cases I am not prepared to say, but certainly they were not present in that of those cases which I examined.

Group 3.

Here the great majority of scarlatina cases are to be found. Failure to demonstrate organisms of any kind in the blood in these cases showed me conclusively that I was dealing with an infection

which was not due to the known pyogenic cocci. The probable reason why there has been failure in discovering the specific germ is probably owing to the requirements for their growth being different from those yet devised.

Group 4.

Cases falling in this group are of less severity than those in groups 1 and 2, and though the illness is usually a prolonged and rather severe one, the prognosis in hospital is hopeful. In the great majority of the cases in which growths were got, both ante-mortem and post mortem, the organisms belonged to the diphtheroid and staphylococcic types. The streptococcus seems either not to invade the blood in this group or does so in such small numbers that it is not easily found. The diphtheroids were found in all the cases in which the staphylococci were present and the role they play in this group may be of a kind similar to that on white mice, i.e. they sap the vitality of the patient and help in the constitutional disturbance produced by the staphylococci.

CONCLUSION.

After a considerable amount of labour I have failed to demonstrate the specific germ, and it is

my desire to openly acknowledge defeat, and not to veil my failure by ambiguity. I make no hesitation in saying that the streptococci - whether they be called pyogenes or scarlatinae - are no more the cause of the disease than are the staphylococci. And in fact I think one would be more justified in naming the latter, for they occur in considerably more cases than the former.

What about the diphtheroids? They are my own discovery, but so far I know little about them, though the fact of their not occurring in groups 1, 2 and 3 compels me in the meantime to class them as secondary in their effect to the scarlatina germ. Their effect on animals is not a very toxic one, but it is very probable that they might be more toxic to man, and that they might have some important significance in the large percentage of scarlatina cases in which they occur.

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(Cultures and Slides of Types E.F.
(Diphtherous) will be forwarded if
required.