The Hunterian oration on the etiology of puerperal fever : delivered at the Annual Meeting of the Hunterian Society / by Alfred Lewis Galabin.

With the Author's Compliments.

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THE HUNTERIAN ORATION

ON

THE ETIOLOGY OF PUERPERAL FEVER.

Delivered at the Annual Meeting of the Hunterian Society.

BY

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THE ETIOLOGY OF PUERPERAL FEVER.

I NEED hardly remind you that the genius of Hunter was not limited to the field of surgery and general pathology, but that he devoted some of his attention also to subjects connected with obstetrics. Thus his name is immortalised in the description of the several parts of the decidua of the gravid uterus, and especially in that term decidua serotina, whose late Latinity is apt to perplex students having minds imbued with classical models of a purer age. No one can comprehend this nomenclature without mastering the Hunterian theory of decidual formation.

In undertaking, therefore, to address you, I venture, as an obstetrician, to select an obstetrical subject, and to direct your attention to some of the problems connected with Puerperal Fevers. I do so the more readily, because many of these problems are yet unsolved, and their solution demands especially three things in which Hunter was pre-eminent—painstaking clinical observation, scientific experiment, and sound reasoning. While I cannot hope to bring before you novel facts, I shall be repaid if I can obtain the aid of some members of the Hunterian Society in investigating these questions by Hunterian methods.

Modern discoveries as to the connection of microscopic organisms with disease, which have thrown such a flood of light on the nature of spreading inflammations, of several infectious diseases, and on some diseases not hitherto considered infectious, have not failed considerably to modify opinions as to the nature of puerperal fever.

No other rational theory has, indeed, ever been proposed to account for the existence of an infectious or contagious disease, which may spread indefinitely from one individual to another, but is limited by time in the individual, except the theory that it is dependent upon parasitic organisms. A merely chemical ferment may indeed suffice, although present in very minute quantity, to kill an individual. A second may even be inoculated from the first, and suffer in minor degree, as when the hand is wounded in dissecting an animal dead from snake-bite. But all experiment shows that such a chemical ferment, if free from organisms, is incapable of causing a disease spreading from one animal to another progressively.

The Darwinian theory of pangenesis, indeed, asserts that the cells of the body give off germs of a minuteness far beyond the reach of any present or any future microscope; and that it is owing to the presence of such germs, derived not only from the parent, but from further ancestors, in the ovum and the spermatozoa, that the progeny grows to resemble its parents, and even reproduces traits of its long-past ancestors. However incredible such a theory may appear at first sight, it will hardly be possible, in any easier and simpler way, to ac-

count for the extraordinary powers of ovum and spermatozoon. If we accept such a theory, we may suppose that cells having, for some reason unknown, taken on a morbid action, may throw off germs which reproduce such morbid action not only in other cells of the same animal, but in those of another animal, if in any way they get access to its tissues. We can hardly dispute the existence of some such spermatic influence of one cell over another in malignant growths —as, for instance, when we find that the glands of the rectum or the uterus are reproduced in a secondary deposit in the liver or the lung.

It is conceivable, then, that an infectious disease might exist, dependent on such a spermatic influence of cells through their own germs, and not on any parasitic organisms. But, on this theory, it appears impossible to imagine any explanation, either for the tendency of the animal to recover from the disease after a certain time, or for the immunity, more or less complete, which is generally conferred by one attack against future attacks of the same disease. The disease ought to be ever spreading into a wider circle and increasing, as is the case in the advance of cancer.

On the parasitic theory of infectious disease, on the other hand, it is possible to account for both of these facts. In artificial cultivations of microbes it is not an uncommon experience that the organism may cease to multiply and die after a certain time—not merely when it has exhausted the pabulum upon which it feeds, but also, apparently, from the effect of some chemical substance which itself produces. Some similar chemical substance may be produced by the growth and multiplication of pathogenic organisms within the body, either by their own power or by some reaction which they set up in the cells of the body.

The immunity conferred by one attack against future attacks of the same disease is not quite so easy to explain. It is well known that three modes of accounting for it have been suggested.

1. That the pathogenic organism exhausts some material in the body necessary for its subsistence; and that this material is never reproduced, or reproduced only after an interval of years. If this were so, then the material in question must be different for each zymotic disease, since an attack, for instance, of whooping-cough does not protect from measles. Men and animals must certainly be singularly unfortunate if they have in their bodies so many substances capable of nourishing zymogenic organisms, but entirely useless to themselves, as they must be if the body is able to go on for years without appreciable loss, and without ever reproducing the substance in question. This explanation is, moreover, directly disproved. An animal which has suffered from anthrax is thenceforth proof against inoculation by anthrax bacilli, which produce the disease with much certainty in unprotected animals of the species which are subject to it; but as soon as the animal is dead the anthrax bacilli will thrive luxuriantly in its tissues, as they will in most organic substances. There is, therefore, no want of pabulum, but the resistance to the growth of the organism depends upon the vitality of the tissues.

2. The second explanation is that the disease is terminated by the production by the pathogenic organism in the body of some chemical substance inimical to its own future growth; and that this substance remains in the body for years, or for life, and prevents any future attack of the same disease, or modifies that attack by hindering the development of the microbes. This explanation is approved by Klein, amongst others, as being probably the correct one. But it seems almost as improbable that this series of substances—one for each zymotic disease from which the animal has suffered—should be retained in the body for an indefinite time, and never excreted nor destroyed, as that there should exist at first in every animal a pabu-

lum specially suitable to each zymotic disease to which it is liable, which is never reproduced when once consumed. The theory appears to me, moreover, to be refuted by the same fact that as soon as the animal is dead the anthrax bacilli will grow in the tissues of an animal protected from anthrax by a previous attack. Is it conceivable that a protecting substance once produced should remain for years intact in the animal's body, and yet disappear immediately upon death, and before decomposition has occurred?

3. The third explanation ascribes the acquired immunity to an altered vital action in the cells or other tissues of the body transmitted by inheritance, more or less completely, to all generations of future cells which succeed the first. If Pasteur's theory be correct as to the mode in which his inoculations for hydrophobia confer an immunity against that disease, we must conclude that this altered vital action can be brought about not only by the multiplication of microbes within the body, but by the repeated introduction by inoculation of some chemical substance produced by the microbes. There is little doubt that the tissues of the body have more or less power of resisting the growth of all organisms within them. An organism is innocuous as regards which the power is complete; that organism is pathogenic as regards which it is incomplete or absent. It may be regarded as essential to the existence of an animal that it should have the power of resisting the entrance into, or multiplication within it, of those organisms to which it is constantly or most frequently exposed; and therefore as certain that such a power would be developed to adapt the animal to its environment. The mode of resistance may be that some chemical substance inimical to the multiplication of the organisms is either produced continuously by the tissues, so as to be constantly present in the body, or else is produced only by the reaction between the cells and the microbe which is assailing them.

May it not be essential to the continuance of the species, not only that this resisting quality in the tissues should exist, but that it should increase in the presence of pathogenic organisms which put it to the test? The mode of increase might be the preferential survival by natural selection of those nuclei in the body which are best adapted to destroy the assailing organisms rather than be destroyed by them. The hypothesis of such a faculty of increase of the resisting power of the tissues of the body in the presence of assailing organisms has the recommendation that it explains by one cause three facts which call for explanation. First, it accounts for the fact that, after a certain time, if the animal attacked by infectious disease does not die, the tissues of the body gain sufficient power to destroy the organisms and put an end to the disease. I think that the explanation is even a better one than that of supposing that the microbes produce some chemical substance by an inherent quality of their own, and that this substance accumulates in the body until it is sufficient in quantity to prevent their further development. For, take the case of syphilis; if the accumulation of this hypothetical substance is so slow that the disease still exists after months or years, it is difficult to suppose that it would not be continuously excreted or destroyed, and fail ever to accumulate at all to the required total amount. On the other hand, we can conceive that a struggle for existence may be prolonged even for years and yet end in the victory of the tissues of the body over the assailing organisms. Secondly, all future cells of the body, the progeny of those which acquired the improved resisting power, may be expected to inherit this property to some extent, but probably not to the full degree. The further removed are the generations of cells the weaker would be the inheritance. Thus we have an explanation of the fact that an immunity, more or less complete, is generally conferred against future attacks of the same disease, but that this immunity is apt to become impaired in the course of time.

Thirdly, the improved resisting power in the tissues is likely to be transmitted, to some extent, from the animal which suffered from the disease to its descendants; and, if many animals in the same lineal descent suffer from the disease, such improved resisting power is likely to become a permanent acquirement in the race. Several well-known facts are capable of explanation on this ground. A zymotic disease introduced to countries where it does not generally prevail usually has a high mortality. Diseases such as measles and whooping-cough, which are constantly present, and from which almost everyone suffers, have a very small mortality. The very same diseases introduced into an island whose population has been free from them for generations become very fatal.

I think that considerations such as these, together with the facts which have been proved as to the dependence of certain diseases upon parasitic organisms, are sufficient to induce many to consider that, if not absolutely demonstrated, it is probable in the highest degree that every contagious or infectious disease is due to the multiplication in the body, or upon some surface, of a pathogenic organism. But whether or not this be admitted as regards all infectious disease, few will deny that it is true of the infectious forms of puerperal fever. Organisms have been found in abundance in the peritoneal exudations, in diphtheritic patches on the genital tract, in the pus of parametric abscesses, and that of metastatic abscesses in different parts, in the lymphatics, and in thrombi within the vessels. In necrotic patches in the walls of veins associated with thrombosis, the spread of microbes has been found coterminous with the necrosis. Microbes have also been found in emboli in distant organs, in the Malpighian tufts of the kidney, in the fluids of the pleura, the pericardium, and the ventricles of the brain, and on the valves of the heart in endocarditis. Both fresh fluids from the peritoneum, or elsewhere, and cultivations of microbes derived from such fluid, have proved fatal to animals when injected into the cellular tissue or peritoneum. Some, indeed, of the animals experimented on have proved able to resist the virus, but animals in the puerperal state have appeared to be more susceptible. In some cases, although comparatively rarely, microbes have been visible in the blood during life; artificial cultivations of microbes have been obtained from the blood, and these have proved fatal to animals in the same way as those derived from the peritoneal exudation.

Both evidence as to the occurrence of organisms in puerperal fever and the importance assigned to it have rapidly increased within the last few years. So recently as 1875, in the discussion on puerperal fever at the Obstetrical Society of London, although the relation of bacteria to the disease was one of the points specially put down for consideration by Sir Spencer Wells, who introduced the debate, only one speaker touched at all considerably on this subject, and he was of opinion that the bacteria are quite innocuous, and rather beneficial in their action than otherwise.

This modern evidence has tended to bring towards unity the various opinions which have been held as to the nature of puerperal fever. The chief of this opinions have been (1) that it is a specific zymotic disease to which puerperal women alone are liable; (2) that it consists primarily in an inflammation of some one or more of the organs or tissues connected with the process of parturition, and that the fever and general symptoms are secondary to and the consequence of these local inflammations; (3) that it is due to absorption from organic matter in a state of decomposition, and is identical with surgical septicæmia and pyæmia; (4) that the name includes affections of different causation, some of which originate from decomposing organic matter, and some from other morbid poisons. Of these, it is scarcely necessary now to consider any except the last two; for it is clear that the in-

fectious form of puerperal fever cannot be merely dependent upon a local inflammation. And I know of no authority of the present day, except Dr. Fordyce Barker, of New York, who still maintains the view that puerperal fever is a specific zymotic disease.

The application to puerperal fever of the germ theory of disease has introduced a new classification—more essential than the old classification into autogenetic and heterogenetic forms of disease.

There are three possible sources of poison in the body of the puerperal woman :

1. The poison may be produced within the body itself. For the resulting condition the term "endogenetic toxæmia," proposed by Dr. Barnes, may be adopted. Even in the normal puerperal woman the excessive liability to septic disturbances and zymotic diseases which exists is doubtless due to the presence in the blood of an excess of waste products. If the excretory organs (especially the kidneys and liver) are acting inefficiently, a greater proportion of such waste products is apt to be retained in the blood. An unusual excess of waste products may also arise in another way. It is known that the bodies of over-driven cattle decompose very rapidly. Again, the flesh of the coursed hare is noted among epicures for its tenderness. It may be inferred that excessive muscular exertion produces such a condition of the tissues that the microbes of the ordinary decomposition develop in them more rapidly than usual after death. Probably, therefore, they may be less protected than usual from other septic germs even during life. We may see here one of the reasons why, after a very severe labour, women are specially prone to septic disturbance. I know of no evidence to prove that endogenetic toxæmia can by itself produce a febrile disease. At any rate, it cannot be clinically distinguished as a separate variety, and is to be regarded rather as a condition predisposing to puerperal fevers proper.

2. A chemical poison—the so called sepsin—may be produced by the action of the bacteria of decomposition on organic matters in the genital canal, and may be absorbed into the system. Experiments made on animals by injection of such fluid into their tissue after sterilisation show that the effect is proportional to the amount of the fluid injected ; that a comparatively considerable amount, such as a cubic centimètre in the case of a small animal, is requisite to cause death, and that, if it does not die, the animal soon recovers if no more poison is introduced. The condition so produced is called septic intoxication or sapræmia. Septic intoxication may play an important part in many cases of puerperal fever, in which, so long as some decomposing material is retained within the uterus, there may be a continuous entry of poison. But it is hardly ever possible clinically to decide that any case belongs purely to this variety ; only, if the symptoms subside very rapidly and completely after the removal of the decomposing matter, it may be presumed probable that the case has been one of septic intoxication only.

3. Parasitic organisms may gain access to the body, and multiply in the tissues, the blood, or the lymphatics, or in all of them. This constitutes septic infection, or septicæmia proper, and is the variety of puerperal fever upon which almost the whole interest is concentrated ; for it is hardly ever possible to be certain that a given case is not of this nature ; and without doubt this class comprises the great majority of cases which occur, including many, if not all, of apparently local affections, such as pelvic cellulitis, and phlegmasia dolens, which have not generally been included under the title of puerperal fever.

It is evident that, by the definition I have given of the third class, zymotic diseases are included, although this is not generally understood to be implied by the term septicæmia. The class may, therefore, be divided into two subdivisions, septicæmia, and zymotic diseases.

Even before the germ theory of disease had been worked out in reference to puerperal fever, most German authorities had adopted the view that puerperal fever is identical with surgical septicæmia, and due to no other cause. This was largely due to the labour of Semelweiss, who showed that dissecting-room poison conveyed by the hands of students is a fertile source of puerperal fever, and that the danger may be averted to a great extent if the hands are carefully washed with antiseptics. American authorities mostly adopt the same view. Many British authors, on the other hand, have long held that certain zymotic diseases, especially erysipelas, scarlatina, and typhus fever, may produce a disease indistinguishable from puerperal fever due to septic poison.

This is now the most doubtful point connected with the disease. I propose especially to consider what evidence on the subject may be obtained, both from observations and experiments in reference to pathogenic organisms, and from clinical experience. The simplification attained by the theory that puerperal fever is identical with surgical septicæmia is not so great as may at first sight appear. It does not reduce puerperal fever to a single disease, but leaves it still a group of diseases.

I will first consider the evidence which has been obtained about septicæmia by experiments on animals. It has been shown that the common organisms which produce putrefaction in organic matter (mainly the bacterium termo and a common bacillus) are not capable of multiplying in living tissues, although they produce the sepsin which may be the cause of septic intoxication. They are aerobic, that is to say, they require the presence of oxygen for their growth, while most, at least, of the pathogenic organisms are anaerobic, and multiply and flourish without oxygen. If, therefore, septic infection is produced, this is due not to the organisms chiefly concerned in putrefaction, but to others, the germs of which chance to be conveyed to the genital canal, and which are capable of multiplying in organic fluids, as are also the organisms of several known zymotic diseases. Each species of these must produce a disease which is essentially different.

An acquaintance with the subject which I confess with sorrow to be very limited enables me to enumerate nine diseases in animals, to be included under the term of septic infection.

1. *Davaine's Septicæmia in Rabbits.*—Davaine injected into the subcutaneous tissue of rabbits a small quantity of putrid ox blood; this produced rapidly fatal septicæmia, and the infection was transmitted through twenty-five animals in succession. For the last effective transmission of the putrid infective material, only a trillionth part of a drop of blood was used.[1] The blood contained actively moving bacteria in great numbers. Most diverse fluids were found effective for the first infection. Coze and Feltz[2] had previously produced a similar septicæmia in rabbits by injecting blood from a puerperal patient, and also continued the transmission by gradually diminishing quantities of blood. They observed in the blood not only bacteria, but micrococci in chains, and long moving rods, and probably, therefore, were dealing with a mixed infection. Davaine's septicæmia cannot always be produced. Koch failed to obtain it. It must, therefore, depend upon organisms not always present in the air. These experiments attracted great attention, because they were supposed at first to show a gradual increase in the virulence of the infection during its transmission from one animal to another. Later experiments, especially those of Gaffky and Dowdeswell, appear to have shown that

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[1] *Med. Jahrb.*, vol. clxvi. Koch on *Traumatic Infective Diseases*, New Sydenham Society's Translation, p. 10.

[2] Virchow und Hirsch, *Jahresbericht für* 1866, i, p. 195.

this was a fallacy, due to the absence of sufficient control experiments, and that the extreme of virulence is already attained in the second, or at the most the third, generation. A greater quantity of the blood of the first animal is required because it dies from the combined effect of the first injection of putrid fluid, and before the bacteria have multiplied to such an extent in the blood that some of them are contained even in a very minute fraction of a drop.

2. *Koch's Septicæmia of Rabbits.*[3]—By injecting into rabbits an infusion of putrid meat, and also water from a rivulet, Koch produced a rapidly fatal septicæmia, which could be transmitted through a series of animals like the last form. Everywhere the blood contained bacteria about 14 μ[4] in length and 6 μ in breadth. The disease is fatal to mice and birds, but guinea-pigs, dogs, and rats resist it. It thus differs from Davaine's septicæmia, which is easily transmissible to guinea-pigs, but not to birds.

3. By injecting a putrid infusion of meat into rabbits, Koch[5] on two occasions obtained a form of septicæmia transmissible like the last, but dependent on large oval micrococci, whose greatest diameter was .8 to 1 0 μ.

4. *Koch's Pyæmia of Rabbits.*[6]—A piece of mouse's skin was macerated two days in distilled water, and a syringeful of the fluid injected into the back of a rabbit. Metastatic deposits were produced in the lungs and liver, and the disease was inoculable through the blood. The organism was a micrococcus, spherical, .5 μ in diameter.

5. *Koch's Septicæmia in Mice.*[7]—This was obtained by injecting putrid fluids. It was transmitted through as many as seventeen animals in succession. The blood was full of very minute bacilli. They are only .8 to 1.0 μ long, that is about one-tenth of the average size of bacillus anthracis or the tubercle bacillus. From their minute size they could only be seen after careful staining and illuminations by a condenser of very large angular aperture.

6. *Koch's Progressive Necrosis in Mice.*[8]—The organism is a micrococcus, spherical, .5 μ in diameter, occurring chiefly in chains and zooglœa. This occurred first in association with No. 5, and could only be separated from it by transmission of the infection to field-mice, which possess an immunity from that septicæmia.

7. *Klein's Pyæmia of Mice.*[9]—This was obtained from an accidental contamination of a culture in pork-broth. The organism is a micrococcus, larger than that seen in No. 4, but forming smaller clusters.

8. *Koch's Spreading Abscess in Rabbits.*[10]—This was produced by injection of putrid blood. The micrococci are spherical, and very minute, only .15 μ in diameter.

9. *Koch's Erysipelas in Rabbits.*—This was produced by inoculation with mouse's dung softened in distilled water. Local inflammation like erysipelas was produced. The blood was not infective, but material from the site of inflammation was not tested. The organism was a bacillus, about half the length (10 μ) and a quarter or a half of the thickness (.3μ) of bacillus-anthracis. The disease, therefore, although producing a similar local effect, is not the same as ordinary erysipelas, the organism of which is a micrococcus.

To these may be added two forms of septicæmia in which a special organism has been observed in the human subject.

10. In several cases of human septicæmia, Klein found in the blood-

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[3] Koch, *Mittheil. Aus. d. k. Gesundh.*, 1881.
[4] μ = a micromillimètre, or .001 millimètre.
[5] Koch, *Traumatic Infective Diseases*, p. 53.
[6] *Op. cit.*, p. 47.
[7] *Op. cit.*, p. 33.
[8] *Op. cit.*, p. 40.
[9] Klein, *Micro-organisms and Disease*, p. 56.
[10] *Op. cit.*, p. 44.

vessels of the swollen lymphatic glands large numbers of minute bacilli, slightly thicker than those of Koch's septicæmia of mice, their length about 1 to 2 5 μ.

11. A number of persons were poisoned by eating ham at Welbeck in 1880. In the fatal cases, bacilli were found in the vessels of the kidney. In experiments on animals, the disease proved to be transmissible, and the same bacilli were found in the blood and exudations. The bacilli are from 3 to 9 μ long, their thickness about 1.3 μ

After a very small research, therefore, eleven distinct diseases can be enumerated, and probably many more exist. In five of these the organism is a micrococcus, in two a bacterium, in four a bacillus. Experiments cannot, of course, be made upon the human subject, and it is therefore not known to how many of these man is liable. But it may be inferred that in him also there are at least several distinct forms of disease, each having a special microbe, included in the term septicæmia. A similar inference may be drawn from the knowledge, imperfect as it is, which has been obtained as yet about the microbes which occur in puerperal fever.

So long ago as 1878 Pasteur found in the blood of puerperal patients who died from a rapid form of septicæmia a microbe, which has the form of a vibrio. This could sometimes be obtained by a culture from the blood, even though it could not be seen in the blood itself. Further researches, both by Pasteur himself and others, have shown that the organisms most generally present are micrococci, and these are always found, even if bacteria and bacilli are present also. They form a main part especially of the peritoneal exudation, and of any diphtheritic patches formed in the genital canal.

The micrococci are found most frequently in chains and chaplets, especially when propagated in cultures. They thus differ from the micrococci commonly found in ordinary abscesses, which are usually arranged in pairs. The chain-like arrangement, however, affords no distinctive character, for it is a very common mode of growth of different species of micrococci. Micrococci similar to those seen in puerperal fever are found in small-pox, erysipelas, diphtheria, pneumonia, scarlatina, as well as in some of the forms of septicæmia in animals already described. Although it has been contended that some of these diseases, as, for instance, scarlatina and diphtheria, may be transformed one into the other, yet there can be no doubt about the absolute clinical distinctness of small-pox, and thus it is proved that micrococci morphologically similar may be of different species, and have absolutely distinct qualities.

Although micrococci are the most usual organisms found in puerperal fever, yet in secondary abscesses, especially if offensive, bacteria and bacilli may be found. Doléris professes to be able to classify the forms of puerperal fever according to the organisms found, in such a way as to be in agreement with the clinical symptoms. He makes the following classification of puerperal fever:

1. Rapid, acute septicæmia, which kills without any notable local lesions. In this form he finds the chief microbe to be a moving bacillus, which in rare cases appears early, both in the lymphatics and the blood. In the cases observed, micrococci also were always found in the lymphatics.

2. In a less rapid form of the disease, there is a tendency to suppuration, the lymphatics and the peritoneum being chiefly affected. A micrococcus growing readily into chaplets is the chief microbe found abundantly in the lymphatics. The same septic bacillus as that seen in the last form is found also in the lymphatics, and appears in the blood only shortly before death. This septic bacillus appears to correspond with the microbe observed by Pasteur in rapid forms of puerperal septicæmia.

3. In forms characterised by thrombosis and phlebitis, and tending

to pyæmia, micrococci were found in the blood. These were in small quantity, and could frequently only be obtained in certain parts of the body, not everywhere throughout the vascular system.

4. In slow forms of disease associated with phlegmon, or tendency to metastatic abscesses, and accompanied by great anæmia, micrococci were also found in the blood. In cases where the hæmatic lesions existed alone, cultures from the blood showed only double points and zoogloea. When it was accompanied by phlebitis and infarctus, the almost constant form in cultures was that of double points in enormous quantities. Chaplets appeared late, or not at all. When there was also lymphatic lesion with development into abscesses, cultures of the blood gave rise almost constantly to long chaplets, similar to those found in the lymphatics.

It is to be observed that during life, as a rule at any rate, the microbes can be seen in the blood only in very small quantities. It appears that the movement of the blood hinders their development. When, however, some corpuscles become agglutinated and form an embolus in a small vessel, the microbes multiply rapidly in and around the embolus. Very shortly after death, in some acute septic-æmic cases, Doléris found the blood swarming with bacilli, which, after such a short interval, he considered could not be the result of the multiplication of the ordinary bacilli of decomposition.

By other authors forms of disease have been recorded somewhat differing from those described by Doléris in reference to the appearance and distribution of the microbes. Thus Lomer[11] relates the case of a woman, aged 37, in whom symptoms of peritonitis commenced twenty-four hours after delivery. Phlegmonous swellings were formed in various parts. She died five days after delivery. Shortly before, some blood was drawn with antiseptic precautions, and was found to be crowded with chain-like micrococci, each chain being composed of from two to twelve members. To every blood-corpuscle two or three chains were present. On *post mortem* examination, performed only one hour after death, the same micrococci were traced in the peritoneal cavity pus, in the liver and kidneys, and in lymph from the phlegmonous swellings; a variety of bacteria were also found. This case is very unusual from the large number of organisms seen in the blood during life. It also proves that, in a rapid form of disease, the microbes in the blood may be micrococci and not bacteria.

The following case, related by Fränkel,[12] is of interest, because it shows, first, a mixed form of disease, and, secondly, an apparent relation to erysipelas. A puerpera died with embolism, suppurative parametritis, and pleuritis. The pleural exudation was found to be an almost pure cultivation of fine bacilli. Some of the micrococci usually seen in suppurations were also present. In the spleen was found a special form of micrococcus, very minute, chiefly occurring in pairs. Cultivations of these cocci, injected subcutaneously into mice, caused death. In rabbits there was no reaction to subcutaneous injection, but injections into the pleura caused pleuritis and pericarditis, the exudation containing the same micrococci. An injection of this exudation into the ear of another animal caused a local inflammation resembling phlegmonous erysipelas. A similar erysipeletous inflammation was produced at the site of the wound where a pure cultivation of the micrococci was injected into the jugular vein of another rabbit.

The following interesting experiment by Pasteur[13] seems to show the passage of a microbe through the blood of the mother to that of

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[11] *Amer. Journ. of Obstet.*, xvii, p. 676.
[12] *Deutsch. Med. Wochenschrift*, No. 14, 1884.
[13] Pasteur, *Comptes Rendus*, 1880.

the fœtus. From the blood of a rather sickly new-born child, whose mother was suffering from supposed puerperal fever, he cultivated a microbe having the form of a vibrio. From the mother's blood no cultivation could be obtained, and at her death no lesion of the genital organs was found. She had, however, an old hepatic abscess, and in pus taken from this he discovered the same vibrio.

The following observation, communicated by Pasteur to the Academy, June 10th, 1879,[14] is also of interest in reference to the question whether it is possible for a zymotic disease in the puerperal state to simulate puerperal septicæmia derived from decomposing organic matter. A woman died at Nancy, under the care of M. Feltz, having all the classical symptoms of puerperal fever. Two days before death, M. Feltz discovered the presence in the blood of bacilli; these were cultivated according to Pasteur's method, and the cultivations, inoculated in animals, killed them by infection. Feltz thought that he had discovered in these bacilli the special septicæmic poison of puerperal fever, and showed them to Pasteur. Pasteur recognised in them the bacilli of anthrax. To test the matter, animals were inoculated with the bacilli and with cultivations derived from anthrax. All died with the same lesions, and the aspect of the blood was identical. It was then discovered that the woman had lived during her pregnancy close to a stable, whence she might have acquired anthrax, although it was not known that the disease had been present in it.

Examination of the lochia has not thrown much light upon the nature of the microbe concerned in puerperal fever. As might be expected from the vicinity of an external surface, the lochial discharge is never absolutely aseptic. Even normal lochial discharge has been found to produce poisonous effects when injected into animals, these effects increasing, the later the date after parturition.[15] The organisms which may be found even in the case of women suffering from no morbid symptoms are of the most various kinds, including bacilli, bacteria, and micrococci, varying in size and form as much as the several species which have been shown to be concerned in different forms of septicæmia in animals. As a rule, however, when the women are free from febrile symptoms, only a very few organisms are found, or it may be impossible to see any, especially if antiseptic injections have been used, and the fluid is obtained high in the vagina. When febrile disturbance is present, the microbes are generally present in abundance. It is thus clear that examination of the lochia does not solve the question how many species of puerperal septicæmia there may be, or whether the microbes causing them are those whose germs are generally present in the air, or must be derived from some special source of contagion.

Experiments on animals have been equally inconclusive. Cultivations from the blood or pus of patients suffering from puerperal fever have frequently caused death when inoculated upon animals, and the blood of the animal has sometimes shown micrococci, generally taking the form of chaplets; but in many cases also the animals have been able to resist the infection, especially when not in the puerperal state.

On the whole, the observations on the organisms present in puerperal fever confirm the view that there are at least several pathogenic organisms capable of producing different forms of the disease. It is obvious that the micrococcus growing in the form of chaplets need not have been of the same species in all cases, since a similar form is observed in such various diseases as small-pox, diphtheria, scarlatina, erysipelas, and pneumonia. There are also apparently harmless

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[14] Ardenne, *Les Microbes, les Miasmes, et les Septicémies*, p. 287.
[15] Kehrer, *Beitr. z. vergleich. Geburtsk.*, No. 6, 1875.

micrococci, such as those which have been found even on the surfaces of wounds running an apparently aseptic course under Listerian dressings.

It is probable that much more may yet be discovered as to difference of species of the microbes in puerperal fever by cultivation and experiments on animals. The various forms of the disease—some marked mainly by rapid septicæmia, some by peritonitis, some by thrombosis and phlebitis, some by slow pyæmia—may depend upon the mode in which the microbes find entry, but may also depend on a difference in their species or their virulence.

If the observations be confirmed, showing that the organism concerned may sometimes be a bacillus, the point may prove to be of some practical significance. For, while micrococci, bacteria, and bacilli themselves are killed by boiling water, and by some antiseptics in general use, the spores of bacilli—of some known species, at any rate—have been found to resist boiling for fifteen minutes, and solutions of carbolic acid 1 in 10, and even corrosive sublimate 1 in 100.[16] Possibly this may afford some explanation of recorded cases in which practitioners, after a series of cases of puerperal fever, have given up practice for weeks, and used antiseptics with diligence, and yet have found the disease recur on resuming midwifery practice.

In the absence of any positive method of distinguishing between micrococci of similar morphological appearance, the question whether zymotic diseases, such as erysipelas, scarlatina, and diphtheria, can give rise to a disease indistinguishable from puerperal septicæmia, must rest at present upon clinical evidence. In reference to this, I desire to call attention to some evidence which appears in the Report on Puerperal Pyrexia, issued by the Collective Investigation Committee, especially as this and other similar reports, owing to their having been published only in the *Collective Investigation Record* and not in the BRITISH MEDICAL JOURNAL, have probably been seen by very few persons. It has been objected to collective investigation that, for deciding an obscure question, no advantage can be gained from an indiscriminate collection of reports from observers, on many of whom no reliance can be placed for scientific observation. It is found, however, that, although some reports are of little value, many are evidently the work of careful observers.

In reference to puerperal fever, the evidence of general practitioners is the best available. Physicians in charge of lying-in hospitals may, indeed, throw much light on one aspect of the subject. But the great mortality of lying-in hospitals up to the date of the most modern improvements in antiseptic precautions proves that in them the risk of puerperal infection is of such a special kind that no inference can be drawn from their experience as to the etiology of sporadic cases occurring in private practice. Again, obstetric physicians, who see cases in consultation, can only form a judgment as to their causation, unless some obvious local cause exists, through the report of the practitioner in charge.

In the case of those zymotic diseases, such as erysipelas and diphtheria, the organisms of which can be easily cultivated in organic fluids at ordinary temperatures, there is every probability in the supposition that the organisms frequently multiply in nature, apart from the human body. It is confirmed by the fact that it is precisely these diseases which often appear to arise spontaneously, in the presence of insanitary conditions, and without its being possible to trace the infection to any previous case of the disease. If it be correct, then such organisms are likely to become the source of traumatic infectious diseases in the same way as any one of the various microbes, the result of whose action is included in the term septicæmia.

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[16] Klein, *Micro-organisms and Disease*, p. 190.

The zymotic disease which has most generally been considered as a possible source of puerperal fever is erysipelas. Erysipelas appears to occupy an intermediate position between zymotic diseases and those diseases which are included in the term septicæmia. It is generally considered a zymotic disease, and it corresponds to one in the fact that it may be caught by anyone not suffering from a wound, in the ordinary sense of the term. No doubt some slight abrasion of surface may be necessary for the entrance of infection. On the other hand, it differs from most zymotic diseases in the fact that one attack does not confer a protection against future attacks.

Recorded instances in which a series of cases of puerperal fever have arisen in the practice of a medical man while he was attending a patient suffering from erysipelas, generally of the phlegmonous form, are so numerous that it is needless to quote any. When a lying-in ward has existed in a general hospital, erysipelas has often prevailed in the surgical wards, and puerperal fever at the same time in the lying-in ward. A case of erysipelas, accidentally introduced into a lying-in hospital, previously in perfect sanitary condition, has sometimes been followed by a series of cases of puerperal fever, as in an instance at the Rotunda Hospital, recorded by Dr. Atthill.[17]

Even by the German authorities and their disciples, who maintain that all puerperal fever is identical with surgical septicæmia, it is scarcely denied that erysipelas has a very close relation with puerperal fever. To be consistent in denying that a zymotic disease can assume a form in the puerperal woman indistinguishable from septicæmia, they are obliged to separate phlegmonous erysipelas from superficial erysipelas, and to regard the former as merely a septic inflammation. But clinically it is impossible to draw an absolute line between superficial and phlegmonous erysipelas ; and experience points rather to the existence of a virus varying in intensity, than to that of two or more distinct viruses. The following case, related by Doléris,[18] may serve as an illustration.

A man, aged 40, suffered from a facial erysipelas which underwent a normal development, phlyctænæ being formed on the affected skin. Cultures of fluid from these showed micrococci in chaplets similar to those seen in puerperal fever. After the subsiding of the erysipelas a series of abscesses associated with distended lymphatics were formed in the substance of the derma over the affected surface during a period of about three weeks. The pus contained micrococci in abundance. Culture of the blood drawn during the acute stage of the erysipelas proved negative. Blood drawn at the time when the patient was suffering from sympathetic fever in consequence of the suppuration gave a cultivation of micrococci, mostly in pairs, like those commonly seen in suppuration.

Moreover, erysipelas does not seem to be exactly identical with septic inflammation, although it is apt to give rise to it, for its microbe has been traced with greater certainty than that of any other ordinary infectious disease, and appears to be very definite in its effects. Felhleisen[19] found micrococci in the lymphatics of erysipelatous human skin. These he cultivated artificially for fourteen generations, during a period of two months. He then, by inoculation in the ear of rabbits, produced an erysipelatous rash which spread to the head and neck, but led to no general septicæmia. He also produced the disease again in man, by inoculating certain tumours with the cultures. Science here gained the unusual benefit of experiments on the human subject, from the fact that the operator expected, and attained, a beneficial influence upon the tumours.

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[17] *Medical Press and Circular*, April, 1877.
[18] *Op. cit.*, p. 249.
[19] *Die Aetiologie des Erysipels*, Berlin, 1883.

A strong point in favour of a close relation between puerperal fever and erysipelas is the fact that when the mother has suffered from puerperal fever, showing its ordinary symptoms, the child has sometimes died with erysipelatous inflammation starting from the umbilicus, often leading to suppuration, and micrococci in chaplets have been found in the affected part. In one instance, in Italy, a man had connection with his wife, who afterwards died of puerperal fever. The husband also died with phlegmonous erysipelas of the penis, extending to the abdomen.

The question is a more difficult one, whether scarlatina poison can give rise to a disease resembling ordinary puerperal septicæmia, and not showing the ordinary symptoms of the disease. In favour of the affirmative, the evidence of Dr. Braxton Hicks,[20] who, out of sixty-eight cases of puerperal pyrexia seen in consultation, found twenty cases of actual scarlatina, and seventeen others in which there might have been infection from that disease, either through the medium of the accoucheur or in other ways, has justly been regarded as of much weight.

Scarlatina may undoubtedly sometimes, like erysipelas, take the form of a traumatic infective disease. In proof of this I may refer to an account by Mr. Howse in the *Guy's Hospital Reports*, vol. xxiv, of an outbreak of scarlatina in Guy's Hospital, in which patients having wounds specially suffered, and the rash often commenced in the neighbourhood of the wound.

Scarlatina in the puerperal woman has three peculiarities. 1. The puerperal woman has a special susceptibility to the disease, while in pregnancy there appears to be unusual immunity. Olshausen[21] found 134 recorded cases within a week after delivery, as against only seven throughout the whole of pregnancy. 2. While the mortality of the disease is much greater than usual—generally from 20 to 50 per cent. —this mortality is not due to the throat-affection, which is almost always slight. 3. It is not unfrequently accompanied by local lesions of the pelvis and peritoneum. This is denied by some authors, but appears in the reports of the Collective Investigation Committee. In 354 cases of puerperal pyrexia there were thirteen of undoubted scarlatina. In all but two of these thirteen cases some symptom of local inflammation of pelvis or peritoneum was noted, and in all the fatal cases the abdomen became distended. In four of the thirteen cases the lochial discharge was scanty, and in five it was offensive. Thus undoubted scarlatina in the puerperal woman shows diminution of some of its own special symptoms, greater fatality, and the addition often of some of the symptoms usually present in puerperal septicæmia.

Again, cases are not uncommon of puerperal pyrexia in which a general scarlet rash occurs with little or no sore-throat, often with no desquamation, and as to which it is very difficult to say whether they should be classed as scarlatina or not. Thus Guéniot, who regards such rashes as symptoms of septicæmia, recognises the resemblance by calling them puerperal scarlatinoid. The following, related by Aulas[22] as an example of this, strongly suggests true scarlatina. A woman, aged 21, two days after delivery had a scarlet rash on the trunk and inner part of the thighs, with pyrexia. Next day it had extended to the whole body except the face. The urine was albuminous. There was slight sore-throat, and a little redness of fauces was visible. There were no abdominal symptoms throughout. The rash began to fade after eight days, and was not followed by desquamation. There was still slight albuminuria when the patient left the hospital.

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[20] *Obstet. Trans.*, vol. xii.
[21] *Obstetrical Journal*, vol. iv.
[22] *Des Eruptions Septicémiques.* Paris, 1878.

There is one point which appears to tell somewhat in favour of the view that such an eruption should be interpreted as indicating a slight attack of scarlatina. This is that, while both in puerperal women and in surgical septicæmia the other forms of rash which are likely to occur—namely, limited erythematous blotches, a vesicular or papular rash running on to form pustules, or a petechial rash—are of very grave import, this is not so with the scarlatinoid rash in puerperal women. Guéniot even regards it as being rather a favourable sign than otherwise in a case of puerperal pyrexia.

In the 354 cases reported in the *Collective Investigation Records*, besides the thirteen of undoubted scarlatina, there were six, not including a case in which a rash followed pneumonia commencing before delivery, in which there was a general scarlet rash. This rash was generally of short duration, and not followed by desquamation in the patients who recovered. In all six cases there were marked local symptoms in pelvis or peritoneum. There was slight sore-throat, with congestion of fauces, in four of the six cases. Two of the patients died—a mortality of 33.3 per cent.

If these cases be regarded as scarlatina, it is not impossible to suppose that there might be a still further modification of the disease, in which the rash might be entirely suppressed, and pelvic and peritoneal symptoms similar to those which occur in ordinary puerperal septicæmia form the main feature of the complaint. Among patients who showed the other forms of rash which I have enumerated, combined with the ordinary lesions of puerperal fever, not including cases of mere sudamina or miliary eruption, the mortality was much higher. Sixteen such cases were reported, and the mortality was thirteen, or 81.2 per cent.

There is one point in which the *Collective Investigation Records* appear to me to afford evidence of some importance in favour of the view that the contagion of erysipelas and scarlatina may produce a form of puerperal pyrexia not having distinctive signs of the original disease, namely, the relative mortality of the several classes. The records were divided into a number of sections, according to probable causation, as far as this could be judged from the report, by myself in conjunction partly with Dr. Herringham, partly with Dr. O. A. Browne. In making the classification, we had no idea in our minds with regard to the mortality. The result is that in the following classes the mortality is decidedly above the average: (1) cases ascribed to infection from puerperal fever itself, mortality 70.8 per cent.; (2) cases occurring after exposure to the infection of erysipelas, mortality 70.5 per cent.; (3) cases occurring after exposure to infection of scarlatina, mortality 62.5 per cent.; (4) cases in which the pyrexia commenced before delivery, and thus indicated that the disease arose from special poison absorbed, and not from any decomposition or other morbid process occurring after delivery, mortality 83.4 per cent. Among the cases not classed as probably due to any special infection, a division corresponding to the old class of autogenetic cases, including, also, the rather numerous cases in which no cause could be assigned, the mortality was only 37.7 per cent. The probability would be very strongly against such a large excess of mortality appearing merely by chance in every one of the four classes above mentioned. The reasonable inference is that in each of them the infecting poison was of unusual virulence.

It is rather singular that, in actual cases of scarlatina and erysipelas, the mortality was less—37.5 per cent. for erysipelas, 30.7 per cent. for scarlatina. It would seem that the excessive danger as compared with average cases of puerperal fever only exists when the stress of the poison falls upon the pelvic organs and peritoneum, and the usual symptoms of the zymotic disease are not manifested.

The strongest evidence which has been adduced tending to show that zymotic diseases cannot be the source of puerperal fever is that of Matthews Duncan,[23] who collected statistics proving that the epidemic prevalence of either erysipelas or scarlatina in any place was not accompanied by any increase of the deaths ascribed to puerperal fever. This is at any rate satisfactory as a proof that zymotic contagion is not often conveyed by the accoucheur. It does not appear to me to prove that it may not be conveyed in some instances, especially when it is remembered that, in time of epidemic, accoucheurs would be likely to use extra precautions. There is no doubt that puerperal women are very liable to take scarlatina; and the evidence would be more conclusive if it were proved that while there was a marked increase of puerperal scarlatina when scarlatina was prevalent in any locality, there was none of puerperal fever in its ordinary form.

Other authorities, on the contrary, have reported unusual prevalence of puerperal fever as coincident with epidemics of erysipelas. A series of such instances, occurring in America, in small villages, has been collected by Minor.[24]

The old division of puerperal fever into the autogenetic and heterogenetic class must clearly be regarded as a less radical and scientific division when it is remembered that in every case of true traumatic infective disease the microbes or their germs must come from without. Yet it may still be to a certain extent a useful practical division, since it is as yet impossible clinically to recognise each disease according to the microbe which produces it. The autogenetic class then includes those cases in which the microbes are only those whose germs are generally present in the air, and in which the essential part of the causation consists in a nidus being provided for them to multiply in, in the shape of some bruised or damaged tissues, or in some solids or secretions too long retained in the genital canal. Yet even so the line of distinction is not clear between the two classes. For there may be every gradation in the sanitary surroundings of a patient, as regards the quantity and quality of the germs present, from a pure mountain height to a bedroom where sewer-gas is laid on abundantly to the room itself or its vicinity. We cannot as yet define what the germs are which are generally or constantly present.

This consideration leads us to a question which is of great importance as regards the causation of puerperal fever, namely, whether it it is possible for a morbid virus to become intensified. The experimental proofs by which such an intensification was not long ago supposed to have been established are stated all to have broken down. These were the case of Davaine's septicæmia in rabbits, already described; the supposed conversion by Buckner of the hay-bacillus into the anthrax bacillus; the poisonous properties said to be acquired by a bacillus subtilis when growing in jequirity infusion; similar properties said by Grawitz to be assumed by an aspergillus, when grown on neutral and alkaline material at about body temperature.[25]

There is no doubt, however, that a virus may be attenuated, and this fact affords a strong presumption that it may also be intensified. Indisputable examples of attenuation are found in vaccinia, and in Pasteur's attenuated virus of anthrax. Although experimental proof is yet wanting, clinical experience is strongly in favour of the view that virus may become intensified. Thus the fatality of infectious diseases varies in different epidemics, and in different stages of the same epidemic. The special dangers of hospitals in general, and lying-in wards in particular, are less easily explained by supposing

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[23] *Edinburgh Medical Journal*, March, 1876.
[24] *American Journal of Medical Sciences*, 1875.
[25] See Klein's *Micro-organisms and Disease*, p. 152, *et. seq.*

that rare microbes find their way thither than by the view that common microbes find a congenial soil of organic matter and acquire there unusual vigour. If the popular view be correct that a common cold is infectious, the only explanation of this quality is that a common micrococcus is first able to multiply in the morbid secretion, and then to acquire thereby the faculty of establishing itself even in normal secretion, and so setting up inflammation of the mucous membrane of another person.

It appears probable that a similar modification may take place in the micro-organisms of pneumonia. The disease is commonly produced by cold as an exciting cause, and not by infection from another person. It is associated with a microbe which can be easily cultivated, and is probably, therefore, capable of multiplying in nature outside the body. But there is evidence that the disease sometimes assumes an infectious form, and spreads like a zymotic disease.

Even as regards Davaine's septicæmia in rabbits, though a progressive intensification of the virus by transmission through a succession of animals has been disproved, there does appear to be some intensification at the outset. The bacillus in question cannot be of extreme rarity, since, on several occasions at any rate, it has been developed in putrid blood during hot weather. But apart from these experiments it is not known to have caused infective traumatic disease. On the other hand, when once it has been transmitted from one rabbit to another, so that a pure cultivation of it in the living blood is obtained, touching an abrasion with a very minute fraction of a drop of this blood is sufficient to produce certain death in another rabbit.

In the case especially of septic organisms, which commonly multiply outside the living body, and only exceptionally in its tissues, it might be expected *a priori* that increased power of growing in the living tissues, that is to say, an increase of pathogenic virus, would be developed. For if the view which I have contended for be correct, that it is an increase in the resisting power of the blood and tissues which terminates a disease dependent upon pathogenic organisms, and protects against its recurrence, it may expected that, on the side of the microbes also, when they are introduced into novel surroundings in the living body, there may be an improvement by natural selection in their power of maintaining the struggle for existence against the tissues. It will be still more likely that this has occurred if the microbes gain the victory, and the patient dies.

One of the problems in puerperal fever is the following. Micrococci, similar to those seen in puerperal fever, normally or commonly reach the vagina, and do no harm, if there is no undue retention of organic matter. On the other hand, in some forms at any rate of puerperal fever, the organisms are so virulent that the accoucheur is apt to carry fatal infection to other women who have had perfectly normal labours, notwithstanding careful ablution, and even considerable use of disinfectants. Must these virulent organisms have been originally derived from some special source, such as a transformed erysipelas, or a micrococcus which dwells in sewers, or may they be the organisms whose germs are commonly present in the air, but possessed of acquired virulence?

The contagious virulence of puerperal fever, in some cases, probably exceeds that of any other disease commonly met with. Practitioners are now so well aware of the danger that a wide spread of infection is rare, though sometimes we still meet with two or three deaths among patients attended by one practitioner, within a few days, before he had become aware that he was likely to be a source of danger. It is necessary to go back nearly fifty years for instances like the following. In the town of Sunderland, within one year, there were forty-three cases of puerperal fever. Of this number, forty occurred in the prac-

tice of one medical man and his assistant.[26] A midwife delivered a woman who shortly died with symptoms of puerperal fever. Within one month, the same midwife delivered thirty other women, of whom sixteen caught the disease, and all died.[27] I know of no such propagation of surgical septicæmia in the private practice of a surgeon, although something similar has occurred when wounded men have been crowded in an insanitary hospital.

It has generally been held that the so-called autogenetic form of the disease is little, if at all, contagious. The following case, which I have met with, appears to me to indicate the probability that organisms commonly present may acquire an increased infective virulence. A woman miscarried about the third month, and a portion of the ovum was retained. This decomposed, and produced an offensive discharge from the uterus. Pyrexia and general peritonitis were developed, and the patient died in a few days with symptoms resembling those of ordinary puerperal septicæmia. Notwithstanding free use of carbolic acid, this case appeared to be the starting-point of a series of cases of puerperal septicæmia in women attended within the next few days by the practitioner in charge, and his assistant who had also seen the patient. Two of these proved fatal, and both the practitioner and his assistant had to give up midwifery practice for some time.

The point which chiefly comes out in the *Collective Investigation Record* as to the danger of contagion is, that it is from fatal cases that danger is most to be feared. Out of the 356 cases, there were nineteen in which there was a probable conveyance of contagion. In eight, this was to other puerperal women ; in nine, to the infant. All but one of the nineteen cases which were the source of contagion proved fatal. In only two of them was there a probable autogenetic source for the original disease.

In conclusion, I have to say that the Collective Investigation Committee still hope for more evidence, and will be grateful to any who may furnish it.[28] I may indicate two points of special interest : 1. In what forms of the disease is contagion most to be feared ? 2. Can, not only erysipelas and scarlatina, but other zymotic diseases—typhoid fever, diphtheria, small-pox, pneumonia—give rise to a disease in the puerperal woman closely resembling septicæmia ? Of special interest would be an instance, if any such occur, in which an apparent puerperal septicæmia is itself traced to the contagion of a zymotic disease, and reproduces the original zymotic disease again by contagion to the infant, or to some other person.

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[26] Armstrong, *Facts and Observations Relative to Puerperal Fever*. London. 1814.
[27] Robertson, *London Medical Gazette*, January, 1840.
[28] Forms of return can be obtained from Dr. Isambard Owen, Secretary to the Collective Investigation Committee.

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