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ON THE

NATURE, ORIGIN, AND PREVENTION

OF

PUERPERAL FEVER.

BY

W. T. LUSK, M.D.,

PROFESSOR OF OBSTETRICS AND DISEASES OF CHILDREN IN THE BELLEVUE HOSPITAL MEDICAL COLLEGE, NEW YORK.

EXTRACTED FROM THE TRANSACTIONS OF THE

INTERNATIONAL MEDICAL CONGRESS,

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ON THE NATURE, ORIGIN, AND PREVENTION OF PUERPERAL FEVER.

The total number of deaths in the city of New York for the nine years from 1868 to 1875, inclusive, was 248,533. Of these, 3342 were deaths either from diseases complicating pregnancy, from the accidents of child-bearing, or from diseases of the puerperal state; or, in other words, very nearly one in seventy-five of all the deaths occurring during that period, was the result of the performance of what we are in the habit of regarding as a physiological function.

A careful study of the records preserved by the Health Department of the city has enabled me to classify these 3342 deaths into: —

(1) Those due to child-birth proper, under which head I would place deaths from shock, from protracted and unusually severe labor, from convulsions, from flooding, from rupture of the uterus, and, for the sake of convenience, those from extra-uterine pregnancies;

(2) Deaths from eruptive diseases, from phthisis, and from the various non-puerperal, inflammatory disorders complicating child-birth. A few cases recorded as typhus, typhoid, and intermittent, I have included under puerperal fever, on account of certain inherent difficulties in the differential diagnosis. Should exception be taken to this action, I have only to state that the entire number thus recorded is much too small to palpably affect any conclusions which this report may contain;

(3) Deaths from a variety of diseases, occurring during pregnancy, in which the pregnant state was, in all probability, an active cause of the unfavorable result;

(4) Deaths from miscarriage (I use the term as I find it employed by the majority of physicians, in their reports, to designate all cases of interrupted pregnancy during the first six months), in which either intercurrent affections, the lack of proper medical assistance, actual want, or criminal malpractice, have been the chief factors in determining the fatal issue. To be sure, in many of these instances, death is due to the supervention of peritoneal complications, septicæmia, and pyæmia, assimilating them closely to cases of puerperal fever. I do not, however, include them under puerperal fever, because their origin is not involved in mystery. Two words tell the whole story, viz., neglect and violence. The explanatory term, retained placenta, testifies to the one, and the certificate of the coroner to the other. We all know how common fatal cases of metria are, in large hospitals, after delivery at full term. After abortions, on the contrary, they are rare. Thus the uterine wards of the Bellevue Hospital have always contained a large complement of women, who have entered the hospital in a very low state induced by some of the sequelæ of abortion. Of these, in nine years, two only have died. Dr. George K. Johnston

reports in seven years, in the Rotunda Hospital in Dublin, but one death in 234 cases of abortion, and that the result of mitral disease of the heart. I have, for the sake of convenience, exhibited the precise proportion of deaths due to each of these several causes in tabular form. Collectively they will be seen to have numbered 1395 cases, or about 42 per cent. of the entire number.

TABLE I.—*Deaths occurring during pregnancy, confinement, and the puerperal state, in New York, between the years* 1867 *and* 1875, *inclusive, excluding those from metria.*

	1867.	1868.	1869.	1870.	1871.	1872.	1873.	1874.	1875.	Total	Total from all causes.
Childbirth[1]	18	13	20	20	37	38	22	19	16	203	
Convulsions	55	36	47	46	54	62	30	51	27	408	
Uræmia[2]	...	4	8	3	5	5	7	29	22	83	
Flooding	16	35	13	25	20	24	20	25	26	204	
Placenta prævia	...	4	10	5	11	9	14	11	18	82	
Protracted & instrumental labors	4	5	4	2	...	...	7	10	20	52	
Rupture of uterus	4	4	3	4	5	5	6	5	11	47	
Extra-uterine pregnancy	1	1	...	5	3	...	1	4	...	15	
Miscarriage[3]	13	10	12	19	36	27	24	30	26	197	
Pregnancy[4]	4	2	4	1	5	5	3	2	5	31	
Phthisis	3	2	1	2	6	8	8	4	5	39	
Smallpox	...	...	...	...	1	9	2	...	7	19	
Scarlatina	...	...	...	...	2	4	1	4	2	13	
Erysipelas	...	...	...	...	...	...	1	...	1	2	
Total	118	116	122	132	185	196	146	194	186	1395	3342

[1] Under *childbirth* are included deaths from shock, from intercurrent non-puerperal diseases, from complications, such as tumors, cancer, disease of the heart, and the like, and all cases reported under childbirth which proved fatal within twenty-four hours of delivery, not elsewhere mentioned.

[2] A large number of deaths from *uræmia* are reported, in which it is not stated whether convulsions existed or not.

[3] Under *miscarriage* are reported all cases in which pregnancy was interrupted previous to the seventh month.

[4] Under *pregnancy* are given deaths, from non-puerperal causes, in which pregnancy existed and probably contributed to the fatal issue. The number of deaths from phthisis is specifically given in the report, as are those from smallpox, scarlatina, and erysipelas, subsequent to 1870.

(5) There thus remain 1947 cases which, variously reported as puerperal fever, puerperal peritonitis, metritis, metro-peritonitis, phlebitis, phlegmasia dolens, pyæmia, and septicæmia, furnish the subject for special inquiry in this paper. All these varieties we will term briefly puerperal fever, attaching to the term, however, no special significance. The diagnosis of anatomical lesions from clinical symptoms alone, unverified by post-mortem examination, is, in the most experienced hands, only approximative. As usually reported by the practitioner, these lesions reflect for the most part his individual theory, or early training. As a matter of convenience then we propose to employ the name puerperal fever as a general term to cover all the febrile conditions peculiar to the puerperal state.

In the 1947 cases of puerperal fever, I have included cases of patients reported to have died of puerperal mania, the non-febrile form rarely proving speedily fatal. That this rule is not universal, I readily admit. Thus, I remember a case occurring in the Bellevue Hospital, in which the

maniacal patient was confined in one of the cells of the hospital. During the night, she got out of bed and broke the windows. The night was bitterly cold. The patient sat up in her night-dress with the cold air streaming in upon her. On the next morning she was seized with pneumonia, and died. A few cases of death, too, reported from insane asylums do not belong in this category. Giving to the term, puerperal fever, then, this broad significance, we find that in nine years it was the cause of nearly one one-hundred-and-twenty-seventh of all the deaths occurring in the city. It is to be regretted that the returns of births in the city are imperfectly rendered. The actual number of births during the last nine years may, however, be roughly set down at 284,000. This estimate is based upon the assumption that the natural birth-rate is thirty-three to the thousand,[1] an estimate which errs upon the side of liberality. The total number of deaths then to the entire number of confinements would be at least in the proportion of one to eighty-five, or, from puerperal fever alone, in the proportion of one to one hundred and forty-six. These figures proclaim more eloquently than any words can do, the necessity of frequently reviewing the state of our knowledge as to the nature of puerperal fever, and of inquiring as to how far it is due to causes which are amenable to human control.

At the outset, it is proper to recall that, as the various names applied to puerperal fever indicate, it is, with rare exceptions, associated at some period of its progress with certain inflammatory processes which have their starting-point in the generative apparatus. The lesions most commonly found in post-mortem examinations consist of catarrhal inflammations of the mucous membrane lining the vagina and uterus, with simple ulcerations, or those presenting a diphtheritic character, inflammation in the pelvic cellular tissue, metritis, local and general peritonitis, inflammation of the lymphatics and veins, thrombosis, pleuritis, pericarditis, valvular affections of the heart, abscesses, and other destructive processes in the parenchymatous organs, and purulent collections in the joints. As a rule, the occurrence of one or other of these inflammatory processes is synchronous with the first outbreak of fever.

When we come to analyze our cases of puerperal fever, we find two general groups, the one *infectious*, the other *non-infectious*.

I. *The Non-infectious Group.*—Every one who has seen much of hospital practice, has of necessity observed instances of puerperal inflammations and febrile conditions, often of a severe type, which have possessed this distinctive peculiarity, that they have in no wise visibly affected the health of puerperal patients in their vicinity. They possess this feature in common, viz., that in them the symptoms of blood-poisoning are absent, or are present only to a subordinate extent, and as a late feature of the disease. The nature of cases belonging to this group may be best expressed by an examination of the known causes which call them into existence. I give the following from a vast number of recorded cases, without pretending, however, that they exhaust the list:—

(1) Inflammations may arise from traumatic injuries, such as rupture of the cervix uteri during labor, those resulting from unskilfully performed operations, and from the bruising or crushing of the soft parts of the mother in long labors, especially in cases of moderate pelvic deformity.

[1] The census for 1870 gives to New York a population of 942,292. The average of annual increase from 1860 to 1870, was 15,000. I have taken 15,000 as the probable average increase since 1870. The inaccuracy in this calculation is not considerable enough to materially affect the proportions.

(2) The imprudence of patients or their nurses. Thus I have frequently been able to trace, in hospital practice during the winter season, severe cases of cellulitis, pelvic peritonitis and general peritonitis, to the patient's getting out of bed, dripping with perspiration and clad only in a night-dress, and going thus bare-footed over a cold uncarpeted floor to the water-closet. Even in private practice, like instances of wilfulness are not unknown; nurses too, especially those who are reputed by the fair sex to know more than any doctor, are fertile sources of trouble.

(3) Moral causes are frequently effective in producing serious disturbance. In studying the records of Bellevue Hospital for the year 1873, I was greatly impressed by the fact that in 240 deliveries in married women there were but two deaths from puerperal fever, while in 209 in non-married women there were thirteen deaths. From the Bureau of Vital Statistics I learn that one-eighth of the deaths from puerperal causes occur among unmarried mothers. Dr. Johnston dwells much upon a similar state of affairs existing during his mastership at the Dublin Rotunda Hospital. In a paper on "The Genesis of Puerperal Fever,"[1] I reported an instance in which five women were confined in the same ward, by the same attendants, and under absolutely the same conditions, with the exception that one chanced to witness the sudden death of a patient from rupture of the uterus. While the other four women did well, this one, after a period of great mental agitation, developed peritonitis and died. It has been suggested that such cases occur only in the records of hospital practice. This is not so. During the past summer I saw in consultation, in a remote and very healthy rural district in which puerperal diseases were unknown, a case of severe fever, lasting eight days, which was traceable to the mother's anxiety and fret over the illness of one of her children. Dr. Alfred Wiltshire, of London, after writing to me an account of a poor woman who died in less than twenty-four hours in collapse, brought on by a rough remark from her husband, adds, "I have seen many other similar though less severe cases, and I confess that my sympathies are deeply touched whenever I have to attend women who have cause for depression during labor."

(4) I have, in a number of cases, had occasion to notice the influence of old adhesions existing between the serous coverings of the pelvic organs, due to a by-gone peritonitis, upon the pregnant and puerperal states. The two following instances will serve as illustrations:—

The first was that of a young wife, who, a few months after her marriage, had a miscarriage. Unfortunately a portion of the ovum remained in the uterus, where it became the source of protracted hemorrhage and some febrile disturbance. A couple of months later, however, the offending body was removed, after preliminary dilatation of the os, by a distinguished physician who was summoned in consultation. A sharp attack of pelvic peritonitis followed, from which, however, the patient so far recovered as to enjoy a period of reasonable health. Then she became again pregnant. In the sixth month of gestation she began to lose flesh rapidly, and had repeated chills, with profuse perspirations, and a continuously high temperature with evening exacerbations. Living at the time, however, in a New Jersey rural district, her symptoms were attributed to malarial influences. I was consulted concerning the case in the eighth month, and on hearing the history predicted an inflammation of the kind under consideration; and found, as I had anticipated, on examination, a large and distinct tumor reaching from the left iliac fossa to the upper margin of the kidney, which was evidently the product of chronic

[1] American Journal of Obstetrics, vol. viii., Nov. 1875, p. 371.

cellulitis and peritonitis. Soon after, she gave birth to a premature but living child. In a few days subsequent to her confinement, the lady died. Every circumstance in the clinical history pointed to the connection between the slowly developing inflammation during pregnancy and the earlier acute attack of peritonitis.

In the second case I was called to see a patient suffering from puerperal convulsions, in whom gestation was complicated by a large fibro-cystic tumor of the uterus. After administering chloroform, I dilated the os, and delivered the child by version. Hemorrhage followed, which was, however, promptly arrested. The three days following, my patient had no bad symptoms. On the fourth, she complained of pain on the left side, which gradually extended over the abdomen. Meantime the temperature rose, the pulse became rapid, and on the following day she died. The post-mortem examination showed no lesions of either the uterine cavity, the cervix, or the vagina, which were all normal in appearance. That portion of the uterus, however, which had undergone fibro-cystic degeneration, and which was found to weigh between six and seven pounds, was covered with fibrinous flakes. Everywhere the tumor was glued to the adjacent serous surfaces by adhesions of long standing, and strong, organized bands, many of them the size of a goose-quill, connected it with neighboring organs. There could be no doubt, on examining these appearances, that the peritonitis had been caused by the dragging of the heavy tumor upon these adhesions, after the evacuation of the uterus and the loss of the support of the abdominal walls.

(5) It is proper to take into consideration the vulnerability of individuals. All puerperal women are said to tremble in the balance between a physiological and a pathological condition, and it is no unfounded assertion to say that trivial causes which would be inoperative in many cases will turn the scale in others. We see this exemplified in first births, and in women who have borne children in excess. In this connection, the question started by Dr. Nathan Allen, of Lowell, as to what is "The Normal Standard of Woman for Propagation,"[1] becomes of real interest.

(6) It is to be borne in mind that the zymotic fevers, not peculiar to child-bed, may provoke in the puerperal woman the same inflammatory lesions that are commonly associated with puerperal fever.[2] This is in conformity with the well-known surgical experience that a febrile paroxysm from any cause exerts an unfavorable influence upon a wounded surface. Olshausen[3] has, however, recently shown that pelvic inflammations and peritonitis are somewhat rare in cases of scarlatina complicating the puerperal state. He likewise adds affirmative evidence to the view advanced by Braxton Hicks,[4] that scarlatina, after a long period of latency or incubation during pregnancy, can make its appearance two or three days after delivery, and, as a rule, with slight throat symptoms. The discussion in the London Obstetrical Society[5] has done much to draw attention anew to the effects of infective diseases upon the puerperal state. I should prefer, however, to exclude well-defined cases of typhus, typhoid, and scarlatina, complicating child-bed, from the

[1] Amer. Journ. of Obstetrics, April, 1876, p. 1.

[2] Vide Hervieux, Traité Clinique et Pratique des Maladies Puerpérales; Art. Maladies Accidentales, pp. 1073 *et seq.*

[3] Untersuchungen ueber die Complication des Puerperium mit Scharlach und die sogenannte "Scarlatina Puerperalis;" Archiv für Gynækologie, Bd. ix. Heft 2, 1876.

[4] Contribution to our Knowledge of Puerperal Diseases; Obstetrical Transactions, vol. xii. p. 44.

[5] Obstetrical Transactions, London, vol. xvii.

category of puerperal fever, unless coexistent local lesions should involve the pathogeny in doubt.[1]

Now as regards the foregoing dangers to the puerperal woman, it is rare that the physician is enabled to employ preventive measures; and these are necessarily not general, but those suggested by his tact, skill, and wisdom, for independent application to each case.

II. *The Infectious Group.*—The infectious form of puerperal fever is characterized by blood-poisoning. The poison is of a septic nature. The usual points of introduction of the poison are the lesions of the parturient canal. This does not, however, exclude other points of entry, and clinical experience renders it highly probable that, under certain conditions, the poison may be primarily introduced into the blood through the respiratory and digestive organs. This form is contagious, but all cases of infection are not contagious in an equal degree. It is customary for many to limit the term puerperal fever to the members of this group; as, however, there is as yet no common understanding of authors among themselves, or among practitioners in general, as to the precise limitation of the term; as, indeed, hardly any two physicians mean precisely the same thing when they speak of puerperal fever, I shall endeavor to avoid confusion by not using the term in a specific sense.

That the infectious diseases of child-birth are of septic origin, there is now abundant evidence. The question of the identity of puerperal fever and septicæmia is largely one of definition. It is a matter of ordinary experience that the retention of a small bit of the membranes within the uterus will produce fetid lochia; and, as the result of infection, a febrile condition, which, as a rule, subsides with the expulsion of the offending body and the use of disinfectant washes. A virulent form of fever is not unfrequently occasioned by retained coagula, or placental *débris*, which have undergone decomposition. I was once sent to see a puerperal patient, suffering from fever, on the fourth day following her confinement. On entering the room I found the stench intolerable; turning down the sheets, I discovered that the patient was lying in a decomposing mass, and learned that her doctor had forbidden, after the birth of her child, the removal of the soiled linen and blankets. The patient died in the third week from pyæmia multiplex.

Haussmann[2] reported a case of auto-infection in the rabbit, which terminated fatally. A portion of the membrane, retained in the left cornu, led to diphtheritic losses of substance in the lower portion of the vagina, to hemorrhagic enteritis, and to peritonitis. The same author produced death from septicæmia by injecting into the gravid uterus of the rabbit serum from the abdomen of a rabbit which had died from infection. The post-mortem examination showed the muscles filled with granules, and the peritoneum injected, but no fibrino-purulent exudation. Injections into the uterus of pus from the abdomen of a woman dying from infectious puerperal disease, produced no effect upon rabbits two weeks gravid, while in the second half of pregnancy premature delivery and

[1] There does not appear to be any very good reason why a well-marked case of scarlatina occurring in a puerperal patient should be termed puerperal fever, more than one of smallpox. Dr. George K. Johnston informs me that the cases of scarlatina occurring in the Rotunda Hospital of Dublin have never started epidemics of puerperal fever among the lying-in patients. I remember but one case of the kind in the Bellevue Hospital, and this did not affect unfavorably patients in the vicinity.

[2] Entstehung der übertragbaren Krankheiten des Wochenbettes; Beiträge zur Geburtshülfe und Gynækologie, Bd. iii., Heft 3, S. 345.

death occurred, in one case in one and a half, in another in two and a half days. In the animal which died in thirty-six hours there was commencing perimetritis and peritonitis, while in the one dying after the lapse of sixty hours the abdomen was found to contain fibrin and pus.[1] D'Espine injected into the uterus of a rabbit, which had just produced her young, pus from the abdomen of a woman who had died from puerperal disease two days before. This was subsequently followed by other injections of fetid fluids during the four days following. On the twelfth day the animal died. The autopsy revealed peritonitis, most marked in the pelvic cavity, inflammatory alterations in the vagina, uterus, and tubes, small abscesses in the body of the uterus, softened clots in the veins of the broad ligaments, and infarctions of the liver.[2] Schüller found that subcutaneous injections of septic material in female animals, during pregnancy, produced a diphtheritic, ulcerative process on the uterine surface, which determined the separation of the placenta; diphtheritic patches, likewise, were found in the cornua of the uterus.[3]

Thus we find that in the human subject, and in experiments made upon animals, septic poisons introduced into the system following or near delivery produce lesions similar to those found in puerperal fever. As a further coincidence, we notice that, as in puerperal fever, the lesions from direct, septic poisoning have nothing characteristic about them, producing in one case pyæmia, in another partial peritonitis, in another general peritonitis, in another diphtheritis, while in others the lesions are comparatively trivial—these differences being due to differences in conditions which are but imperfectly understood.

My friend, Prof. Barker, in his magnificent work on "The Puerperal Diseases," which is, as every one knows, a masterpiece in the way of clinical observation, mentions, as a distinction between septicæmia and puerperal fever, that, while puerperal fever is contagious, septicæmia is a non-contagious affection. I have the record of the following case which militates against this theory. A physician in New York delivered a woman of a putrid fœtus. The woman died of septicæmia. Two days after attendance upon the first case, he attended a second patient, who likewise died of septicæmia. Dr. Barker states that by inquiry among surgeons he cannot learn that septicæmia follows in their wake as does puerperal fever in that of the obstetrician, and yet Sir James Simpson asserts it to have been the case in his younger days.[4] The vital statistics of New York, for the last nine years, show that a sequence of deaths following confinement in the practice of individuals has become very rare, and, let us hope, that if the enlightenment of these later days has enabled the surgeon to avoid such a misfortune, in the end the same profit may accrue to the practitioner of midwifery.

But it is to be remembered that the conditions in a puerperal patient, and in one upon whom a surgical operation has been performed, are not identical. Samuel, in speaking of immunities and dispositions to septic poisoning, says:—"The statistical frequency of septic, puerperal

[1] Ibid., p. 394.

[2] Contribution à l'Étude de la Septicémie Puerpérale; Paris, 1873, p. 28.

[3] Experimentelle Beiträge zum Studium der septischen Infection; Deustche Zeitschrift für Chirurgie, Bd. vi., S. 141.

[4] "I have repeatedly heard of instances of a rapid succession of surgical fever cases and disasters in the practice of the same surgeon, while the other surgeons in the same locality had their patients recovering as usual." On the analogy between Surgical and Puerperal Fever; Simpson's Obstetric works, edited by Priestley and Storer, vol. ii. p. 19.

diseases is due to the length of the parturient canal, to the fact that through this long passage there must pass all the pathological and physiological excretions, and to the soiling of these parts with fingers, instruments, and secretions, which have become the bearers of sepsis."[1] He found, on the other hand, that it was extremely difficult to produce a progressive ichorous condition by daily painting an open stump with a septic fluid,[2] though the same was readily obtained when an infinitesimal quantity of septic fluid was injected underneath a fascia. Dr. Barker is, however, much too keen an observer to ever go far astray, and it is true, as will be mentioned hereafter, that there is a form of septicæmia which is not contagious. The only question to be raised is as to whether it is allowable to restrict the term to this form. As usual in all questions concerning puerperal fever, there is practical agreement as to facts, with divergence of opinions concerning definitions.

Until very recently the whole subject of septicæmia has been in a state of wellnigh hopeless confusion. From Gaspard and Panum, through a long list of experimenters, hardly any two have arrived at precisely similar results. Something like an approach to order has, however, been produced since it has begun to be understood that the effects produced by septic fluids vary with the quality of the poison and the method of experimentation, and that to obtain identity in the result, there must be identity in all the conditions. Thus Samuel has shown that the same organic substance produces different effects at different stages of decomposition; again that the enteritis which is commonly quoted as characteristic of septic poisoning, occurs, as a rule, in animals, when the septic fluid is injected directly into the blood, and is rare when it finds its way into the circulation through the lymphatics, as is the case usually in clinical experiences.[3] There is one experimental point of extreme practical importance too in connection with puerperal septicæmia, viz., that if the injection of a septic fluid be made directly into a vessel, toxic effects speedily follow, but are transitory, unless the amount of the fluid be large, or its virulence exceptional, or the animal very young,[4] whereas very small amounts injected subcutaneously, by developing rapidly spreading phlegmonous inflammation, resembling malignant erysipelas in man, are capable, after a period of incubation, of producing fatal results; or they may, if injected into a shut cavity or underneath a fascia, lead to the development of an inflammation of an ichorous character. In other words, the eliminating organs suffice, under ordinary conditions, to remove from the blood the same amount of septic fluid which would prove fatal if injected into the tissues.[5] To produce similar results the injections into the blood need to be repeated at intervals. This experience leads us to the conclusion that, in the tissues, septic poison possesses the capacity of self-multiplication, and that, in the local inflammation set up, a reservoir is formed from which poison is continuously poured into the circulation.

This capacity of self-multiplication, which septic fluids possess, has

[1] Ueber die Wirkung des Faülniss Process auf den lebenden Organismus; Arch. f. exp. Pathologie, Bd. i. S. 343.

[2] Loc. cit., p. 339.

[3] Loc. cit., p. 349.

[4] Traube und Gscheidlen, Versüche ueber Faülniss und den Widerstand des lebenden Organismus. Schles. Ges. f. vaterländische Cultur, Febr. 13, 1874.

[5] In some instances in which absorption from the tissues is very rapid, the effects of subcutaneous injections may be similar to those produced by injections made directly into the circulation, and the local lesion be insignificant.

recently been found to be coincident with the presence of certain organic bodies, termed variously micrococci, microspores, or sometimes, less specifically, bacteria. All carefully made experiments serve to show that, if a septic fluid be deprived of these organic bodies by boiling, or filtration, while it continues capable of producing inflammation, the inflammation is usually of diminished intensity, and remains local in its character;[1] whereas the microspores, retained upon the filter, possess all the virulent properties of the original fluid.[2] This does not alone necessarily prove that the virus resides in the microspores, for it does not exclude the possibility that both the virus and the microspores remain upon the filter.

So far, attempts at isolating the microspores and cultivating them separately in vehicles, composed of water holding in solution certain inorganic constituents necessary for their healthy nutrition, have been only partially successful in proving them to be the sole source of infection. Some experiments of Tiegel and Klebs[3] were attended with positive results, but Hiller arrived at different conclusions. He found that bacteria washed in pure water were innocuous.[4] But pure water had long before been proven by observers to be inimical to the well-being of the organisms in question. Schüller says that Hiller's experiments prove apparently that while a putrid fluid may be in the highest degree poisonous, its component parts, viz., either the fluid or the bacteria singly, are neither deadly nor poisonous.[5] The fact is that all isolation experiments are subject to what seems an unavoidable source of error. As Davaine noted, early in his observations, the physiological action of bacteria is very dependent on the constitution of the medium in which they are developed, which is in entire harmony with what is known of organisms much higher in the scale. "Many plants," says Burdon Sanderson,[6] "containing active principles, become inert when transplanted from an appropriate soil." Bucholtz, in a series of experiments designed to test the influence of antiseptics upon the vitality of bacteria, found not only a difference between those taken directly from the infusion and those cultivated in artificial fluids, but between bacteria derived from the same source and cultivated in modifications of the nutrient medium.[7] Under these circumstances, all evidence of a positive character is to be regarded as of more value than that which is purely negative.

It is, however, from the constant presence of the round bacteria in infected wounds, and their distribution through the tissues, that the argument in favor of connecting septic symptoms with the bacteria has been mainly deduced. Here the ground is sufficiently solid, and, judged by ordinary laws of scientific evidence, the pathological importance of the microspores may be regarded as established. To be sure, we find them

[1] In filtration through porous earthenware cylinders, the filtrate possesses no phlogogenic properties.

[2] Tiegel, Correspondenzblatt für Schweizer Aertze, 1871, S. 1275. Klebs, Archiv für exp. Pathol. und Pharmakol., Bd. i. Heft 1, S. 35.

[3] Klebs, Archiv für exp. Pathologie und Pharmakologie; Beiträge zur Kenntniss der pathogenen Schistomycetin; Band iv., Heft 3, S. 241 und ff. Tiegel, loc. cit.

[4] Exp. Beiträge zur Lehre von der organisirte Natur der Contagion und von der Faülniss; Archiv für klinische Chirurgie, Bd. xvii. Heft 4, S. 669 u. ff.

[5] Exp. Beiträge zum Studium der septischen Infection; Deutsche Zeitschrift für Chirurgie, Bd. vi. S. 162.

[6] Lectures on "The relations of bacteria to disease;" British Med. Journal, March 27, 1875. See, also, Klebs, Beiträge zur Kenntniss der pathogenen Schistomycetin; Arch. für Pathol. und Pharmakol., Bd. iii. S. 321.

[7] Antiseptica und Bacterien; Arch. f. exp. Pathol. und Pharmakol., Bd. iv., Heft 1 und 2.

in tongue-scrapings of healthy individuals, but tongue-scrapings are poisonous if injected into the tissues. That they do not ordinarily prove so in the mouth, is no more singular than that woorara can be swallowed with impunity. Tiegel has endeavored to show that the round bacteria are found normally in the internal organs of the body.[1] If his experiments should in fact stand the test of criticism, they would only show that a few bacteria may be found in health in the liver and in the pancreas, but never in anything like the same numbers or the same general distribution, nor with the same characteristic groupings, that have been proven for a number of infectious diseases.[2] It is stated that they are sometimes absent from the blood taken during life in septic diseases. As, however, their constant presence has been confirmed in the vessels and glomeruli of the kidney, it is fair to assume that they are filtered out by those organs when the conditions favorable to their development do not exist in the blood. Again it is an open question, which awaits confirmation, whether it be not true, as Hueter claims, that the microspores do not disappear, but are taken up by the blood-globules, rendering the latter adhesive, and predisposing the blood to the stases characteristic of inflammation.[3] Zahn has shown that the inflammation in the mesentery of the frog, in the Conheim experiment, does not take place if the air is first filtered through diluted carbolic acid.[4]

As to the exact manner in which these minute bodies exercise their pernicious influence, whether they operate mechanically, or whether they produce a virus in the process of nutritive activity, or whether, as is probable, both suppositions are correct, we may safely leave as questions to be decided by subsequent investigations. It is enough for us to note that the connection between sepsis and the round bacteria is intimate and vital. Panum, who is often quoted as opposed to what is known as the Bacteria theory, admits as probable that the microsporon septicum is inoculable, appears in the blood during life, multiplies in the tissues, and, in part by production of a special poison, perhaps, and in part by mechanically irritating the tissues, excites inflammation, suppuration, and fever.[5] Bergmann, who once thought that he had found the secret of sepsis in a crystallizable substance derivable from putrid fluids, which he termed sepsin,[6] now squarely accepts the modern doctrine. Virchow has so far given in his adhesion to the new school as to say: "Especially in this connection are to be mentioned the diphtheritic process and the erysipelatous, especially erysipelas malignum. The granular deposit in diphtherically affected tissues, of which I formerly spoke, has more and more proven to be of a parasitic character. What we formerly regarded as simple, organic granules, as infiltration, or exudation, has since proven to be a dense aggregation of micro-organisms which penetrate into the tissues and cells to compass their destruction."[7] Even Billroth, who contends that what he terms a zymoid ferment is the first thing developed in the line of cau-

[1] Virchow's Archiv, Bd. lx. S. 453.

[2] Klebs., Arch. für Path. und Pharmakol., Bd. iii S. 319.

[3] Allgemeine Chirurgie, Cap. xvii.; Der fieberhafte Process. So, too, Schüller, loc. cit. pp. 168, *et seq.* Birch-Hirschfeld likewise found bacteria in the white globules of pyæmia; Schmidt's Jahrbücher, Bd. 166, No. 5, S. 187.

[4] Arbeiten an der Berner pathol. Institut., 1871.

[5] Das putride Gift, die Bacterien, die putride Infection und Intoxication, und die Septicæmie; Virchow's Archiv, Bd. lx. S. 348.

[6] I have not been able to obtain access to Bergmann's original paper, but make this statement on the authority of Hueter, Allgemeine Chirurgie, S. 543.

[7] Die Fortschritte der Krieg's Heilkunde; Berlin, 1874.

sation, and that the bacteria are a sort of epiphenomenon, concedes that the organisms, by their migrations, may become the carriers of the virus into the interstices of the tissues.[1] I mention the less willing witnesses to the importance of bacteria in disease: I need not recapitulate the names of a host of active advocates of the germ theory.

I have been thus explicit regarding the evidence concerning bacteria in septic diseases, because it places the question of the infectious group of puerperal fever-cases in the following position:—Experiences occurring clinically, as well as those produced upon animals, teach us that certain lesions and symptoms, similar to those we are accustomed to regard as characteristic of puerperal fever, result from septic poisoning. In a large class of cases, however, the connection between child-bed fever and sepsis has been deduced rather from analogy than direct proof. For those who chose to regard such as due to a specific poison peculiar to the puerperal state, there was really no objection. If, however, round bacteria are characteristic of septic poisoning, the question presents itself in a different light, and we have to inquire whether, in the less obvious cases, bacteria are present in puerperal fever in the proportions and groupings that we find them in other diseases due to putrid infection. Now it is precisely proof of this nature that has recently been abundantly rendered.

Waldeyer,[2] Orth,[3] Heiberg,[4] and Von Recklinghausen, found the tissues and lymphatics of the parametria filled with pus-like masses, which consisted, in addition to pus-cells, chiefly of bacteria. Bacteria swarmed in the fluid of the peritoneal cavity. In one case examined by Waldeyer, six hours after death, while the body was still warm, the peritoneal exudation was like an emulsion, and furnished an abundant deposit which consisted almost entirely of bacteria. Orth injected ten minims of peritoneal fluid from a woman dead of puerperal fever into the abdomen of a rabbit. As the animal was dying he broke up the medulla oblongata, and found in the peritoneal fluid enormous quantities of these organisms. In puerperal fever, round bacteria have been likewise found, though in less quantities, in the lymphatics of the diaphragm, and in the fluids of the pleura, the pericardium, and the ventricles of the brain. In post-mortem examinations of fresh subjects, the serous fluids, withdrawn under proper precautions, do not contain round bacteria except in cases of septic infection.[5] Orth found in the purulent contents of the vessels of the funis, in children who died of sepsis, precisely the same formations as existed in the exudations of the mother.

The presence of these germs in puerperal fever serves not only to fix cases hitherto considered doubtful in the category of septic diseases, but it affords the best explanation of the protean phenomena of puerperal fever itself. Steurer, formerly *interne* at the Bellevue Hospital, where he witnessed the epidemic which prevailed in that institution in the year 1874, afterwards made, under the guidance of Prof. Von Recklinghausen, a special investigation of the pathological changes in a similar

[1] Untersuchungen über die vegetaliens Formen von Coccobacteria septica; Berlin, 1874, S. 200.

[2] Ueber das Verkommen von Bacterien bei der diphtheritischen Form des puerperal Fiebers; Archiv für Gynækologie, Bd. iii. S. 293.

[3] Untersuchungen ueber puerperal Fieber; Virchow's Archiv, Bd. lviii. S. 437.

[4] Die puerperalen und pyæmischen Processe; Leipzig, 1873.

[5] Klebs, Beiträge zur Kenntniss der pathogenen Schistomycetin; Archiv für exp. Pathol. und Pharmakol., Bd. iv. S. 441 u. ff.

epidemic which occurred in Strasbourg. From a written communication received by me from Dr. Steurer, many of the following facts concerning the pathogeny of the disease have been derived. These facts, I may add, are fully supported by the investigations of others, and form a most valuable contribution to our knowledge of puerperal fever.

Steurer's cases all presented diphtheritic patches about the vulva, or upon the mucous membrane of the vagina and uterus. These patches were always associated with a loss of substance, and were composed of disintegrated fibrin, white and red blood-corpuscles, and colonies of round bacteria in great abundance. From the patches, the bacteria could be traced between the muscular fibres, and deep down into the canalicular spaces of the connective tissue, where their presence gave rise to cellulitis. From the canalicular spaces they entered the lymphatics, with resulting lymphangitis. In many cases the lymphatics could be traced along the broad ligaments to the ovaries (puerperal ovaritis), and into the subperitoneal tissue of the lumbar region. By perforation of the walls of the lymphatics which directly underlie the peritoneum, they made their way into the peritoneal cavity and excited pyæmic peritonitis, an affection which differs from traumatic peritonitis, and for which the claim has been set up that it is peculiar to puerperal fever. The wide stomata upon the abdominal surface of the diaphragm allowed the facile entrance of the organisms into its lymphatics. Waldeyer found in diaphragmitis the lymphatics of the diaphragm filled with bacteria. And thus following the lymphatic system, if we only admit that the round bacteria are the carriers of sepsis, a fact which hardly admits of dispute, the frequency, in severe types of puerperal fever, of inflammations of the serous membranes—of the peritoneum, the pleuræ, the pericardium, and the joints—finds an easy explanation. We can understand, too, how it is not always altogether accident which determines in different cases the precise serous membranes which are affected.

The ductus thoracicus is the principal channel through which the poison enters the blood. Bacteria are not usually found in the blood during life. A few hours after death they swarm in that fluid. Possibly the rapidity of the blood-currents during life does not favor the multiplication of bacteria. That the bacteria do, however, enter the general circulation during life, is incontestable. Steurer writes, "As the kidneys are the great filters of the human system, I never neglected to examine them, and almost invariably found the glomeruli and arterioli filled with micrococci (round bacteria)." This is in correspondence with what occurs in other septic diseases, and accounts for the albuminuria and interstitial nephritis which often supervene in the advanced stages. We have seen already that, in consequence of septic poisoning, the white blood-globules have a tendency to adhere to the walls of the vessels. This leads to stases in the capillaries, to congestion of the deep-seated organs, and to an increase of blood in the large veins of the trunk. Finally death takes place from apnœa, partly from the inability of the blood-corpuscles to carry oxygen to the tissues, and partly from paralysis of the respiratory nerve centres.[1] Sometimes the bacteria pass directly into the veins, where they give rise to phlebitis. Prof. Von Recklinghausen recognizes three ways in which this may take place: (1) Through a thrombus; and here let me call to mind that it is very common in

[1] Vide Schüller. Exp. Beiträge zur Studium der septischen Infection. Deutsche Zeitschrift für Chirurgie, Bd. vi. Heft. 1 und 2, S. 149 u. ff.

uterine phlebitis to find the uterus large, and the vessels at the placental site filled with soft thrombi; (2) through direct perforation of the venous walls; (3) by being taken up by white corpuscles and by them conveyed into the vessels in the manner described by Cohnheim.

When the bacteria enter directly into the circulation, they sometimes, in passing through the heart, adhere to the endocardium and the valves, causing exudation, ulceration, and decomposition, and thus give rise to the so-called endocarditis ulcerosa puerperalis.[1] In the cases studied by Waldeyer and Steurer, there were diphtheritic patches, serving as the starting points of the puerperal processes. Whether these so-called diphtheritic patches are identical with those which appear in the throat, is an open question. Morphologically, they are so, but, in hospitals, epidemics of puerperal diphtheritis are not associated with throat diphtheritis.

To avoid misapprehension, let me distinctly state that diphtheritic patches are not necessary to the infectious form of puerperal fever. They indicate an unwholesome atmospheric condition, and are somewhat rare outside of public institutions. Orth and Heiberg noticed the same, general, post-mortem changes in those cases in which the patches were absent, as in those in which they were present. My own observations show that they are rarely developed in the early stages of a hospital epidemic of puerperal fever, nor are they to be found in all cases when such an epidemic is at its height. In some of the lying-in hospitals in Europe, puerperal diphtheritis appears, however, to be endemic.

The question as to the extent to which erysipelas and puerperal fever are cognate diseases, is in a fair way to be solved by recent investigation. Orth took the contents of a vesicle, from an erysipelatous patient, which contained bacteria in great abundance, and employed the same for injections under the skin of rabbits. In this way he succeeded in producing in these animals a species of erysipelas malignum. In the subcutaneous œdema, and affected portions of the skin, he found enormous masses of bacteria, so far exceeding in quantity the amount introduced as to prove an abundant new production.[2] Samuel produced similar results by the injection of ordinary putrid fluids containing round bacteria. An affection resembling simple erysipelas he obtained most frequently by the application of fluid to a wound torn open after the second or third day.[3] Lukowski found that erysipelas could be produced by fluid containing micrococci even when putrefaction did not exist. The contents of erysipelatous vesicles containing no micrococci, excited no morbid manifestations. Where the erysipelatous process was fresh and progressing, micrococci were found in great abundance in the lymphatics and canalicular spaces. Where the process was retrogressive, there were no micrococci to be found, even in cases in which inflammation existed to an intense degree.[4] Virchow's testimony we have already given.

Thus we find in surgical fever, in puerperal fever, in diphtheria, and in erysipelas, the presence of a common element which links them together, and which establishes the relationship which has long been recognized as existing between these various processes. Experiments made by competent men, with care and intelligence, serve continually

[1] Heiberg, Die puerperalen und pyæmischen Processe; Leipzig, 1873, S. 22 und 34. Gives references to cases reported by Wirge and Eberth.

[2] Untersuchungen über Erysipel.; Arch. für exp. Pathol. und Pharmakol., Bd. i. S. 81.

[3] Arch. für exp. Path. und Pharmak., Bd. i. S. 335 u. ff.

[4] Untersuchungen über Erysipel.; Virchow's Archiv, Bd. lx. S. 430.

to increase the probability that the bacteria are no chance products, but that they have a vital connection with the diseases designated. Whether these organisms are identical in the different infectious diseases in which they have been recognized, is another question. Billroth complains of the monotonous appearance of always the same forms.[1] When we bear in mind, however, that our best instruments fail to enable us to distinguish the ovum which is to produce a mouse from one that will produce a tiger, though the ovum is at least one hundred times larger than the micrococcus, the argument loses something of its value. Whether identical or not, they all possess the common property of penetrating the tissues, under favorable conditions, of multiplying, and of producing, by their migrations, local inflammations and general infection.

I cannot refrain, in conclusion, from quoting entire the following statement of Panum, which appears to reconcile certain differences in the definitions of the term septicæmia by different authors:—

"The putrid poison may, during life, enter the blood with or without bacteria, especially from wounds, and occasion all the symptoms of septic poisoning, whereas, however, the *bacterium termo* does not appear to occur in the blood during life. *This simple putrid infection does not appear to be inoculable.* Another, as it appears, distinct, specific, pathogenic fungus, the microsporon septicum of Klebs, developed especially in pus (and blood?), perhaps under the predisposing influence of the putrid poison, when the air (as in overcrowded hospitals) contains the latter, or when it is transferred by inoculation, seems on the other hand during life to increase in the blood and tissues, and, in part perhaps, by production of a special poison, in part, perhaps, in a more mechanical way, by penetration, and, under circumstances by its irritative action on the tissues, excites inflammation, purulence, and fever."[2]

Causes of Puerperal Fever.

The Atmosphere.—The effect of a poisoned state of the atmosphere is best observed in the so-called nosocomial malaria of hospitals. At Bellevue I have had frequent occasion to witness febrile outbreaks among the patients in the lying-in service, which were instantaneously arrested by closing the tainted ward and transferring the inmates to a healthy locality. As at these times the nurses, the bedding, and the utensils, remained unchanged, it is fair to assume that the previous, unhealthy condition was not due to the transfer of a poison from patient to patient by the attendants, but to something residing in the air of the vacated apartment. In the inquiry as to the production of this condition, it can be assumed that it is not caused by aggregation alone. The medical wards of Bellevue, always crowded, have been in time of need safe receptacles for lying-in patients. It certainly is not due to the presence in excess of what are generally regarded as the ordinary constituents of the atmosphere. We must, therefore, look for some additional element capable of unfavorably affecting the economy. When the disturbance produced by nosocomial malaria is not arrested by change of locality, and the golden moment is allowed to slip by, the secretions of the patients affected become inoculable. Under such circumstances the epidemic spreads rapidly, and assumes continuously a more and more severe type.

[1] Untersuchungen über die Coccobacteria septica, S. 3.

[2] Das putride Gift., etc., Virchow's Archiv, Band lx. S. 349. I have translated literally. The meaning of the sentences, in spite of the involved construction, is sufficiently clear.

If, during such an epidemic, the external genitals be carefully watched, diphtheritic patches may now and then be observed. At first these patches may not be of any special clinical importance. It is possible that they may rapidly clear off, and thus come to be regarded as of little consequence. When, at length, the epidemic has assumed a pestilential form, these patches, which may make their appearance in isolated cases at any time in a hospital, are rarely absent. I have already dwelt upon the composition of these patches, not because I believe that they are essential to puerperal fever, but because their presence tells the tale of what it is in the atmosphere which accomplishes the charnel-house work. Conditions have been present in the air to favor the multiplication of bacteria, and have fitted them to become the active producers of disease. Can we doubt this? First, the epidemic was mild. If a patient, however, died, her tissues and secretions were filled with bacteria, as has been described. Then the epidemic became virulent, and the lesions of the generative apparatus, especially of the external organs which were most exposed to the air, became covered with patches which were found to swarm with micrococci. I cannot, under the conditions named, but consider it more in accordance with ordinary scientific reasoning to conclude that the micrococci played an important part in the production of puerperal fever, than that the puerperal fever produced the micrococci.

To be sure, bacteria or their spores are always present in the atmosphere, and it may be fairly asked how patients are ever spared from their perverse industry. The answer is that they are not always equally active for evil. Bucholz found that the same bacteria, developed in Cohn's fluid, offered more resistance to carbolic and salicylic acids than those cultivated in an analogous fluid which he had adopted. Distilled water renders the action of bacteria extremely feeble. In experiments upon animals, the results obtained with septic fluids depend in no ordinary degree upon the age of the fluid, the material from which it is formed, and the conditions under which it is generated. Micrococci multiply in hospitals when organic materials favorable to their growth are present in sufficient quantities. Robin and others have demonstrated the existence of albuminoid matters in water condensed upon vessels containing freezing mixtures, and placed in overcrowded wards of hospitals. When the results of crowding become manifest, these albuminoid matters not only impart a peculiar fetid odor and putrefy with great rapidity, but rapidly impart putrefaction to normal blood and healthy muscle with which they are brought in contact.[1] Micrococci both cause putrefaction, and serve as the carriers of septic virus. Hueter found putrid blood a most favorable fluid for septic experiments. It was noticeable in Bellevue Hospital that febrile outbreaks always arose in, and were usually confined to, the ward in the hospital which, by a bad arrangement, was assigned to patients for the first four or five days following confinement, *i. e.*, during the period of the lochia cruenta. As puerperal fever is rare after the fifth day, this at first sight would seem natural. But if a patient was transferred directly after confinement, during one of these unhealthy periods, to the ward containing the patients who had passed the first five days, but had not completed the ten days, she would escape the fever. It was always the same ward that required to be disinfected. In a communicating apartment all the confinements took place; and at all times, therefore, the conditions were present for loading the atmosphere with

[1] Leçons sur les Humeurs; Paris, 1867, p. 195.

the products of decomposing blood. In the summer months, as long as the windows were open all the time, the patients enjoyed immunity from nosocomial malaria. In the early fall, as soon as it became necessary to close the windows partially on account of the cool nights, it was not uncommon for the more trivial disturbances, such as so-called milk-fever, the hospital pulse, and catarrhal affections of the genitalia, to manifest themselves. Through the months of February, March, and April, the mortality was usually greatest. During the winter months there was, as a rule, crowding of patients, insufficient ventilation, the saturation of the air with albuminoid materials chiefly derived from blood, which, under the furthering influence of the heat requisite to make the wards comfortable, entered readily into decomposition. That the latter winter months should prove the most perilous, is in accordance not only with the theory of continuous accumulation, but with the experimental fact that weeks sometimes elapse before a decomposing substance acquires the highest degree of virulence.

Apart from the nosocomial malaria of hospitals, there is reason to believe in the influence at times of certain general wide-spread atmospheric states which affect the entire community. In the year 1871, the mortality from child-bed in New York was 399; in 1872, 503; in 1873, 431; in 1874, 439, and in 1875, 420. Now the excess in the deaths for 1872 was due wholly to an increase in the cases of metria, those from ordinary accidents remaining nearly the same as in the preceding years. The disease certainly did not extend into the city from the hospitals serving as foci, for the mortality that year at Bellevue Hospital was hardly more than half the usual average. There was no especial mortality that year from either diphtheria, erysipelas, or scarlatina, but the aggregate mortality was the largest known in the history of the city. There are no positive data connecting the civil deaths from puerperal fever in 1872 with parasiticism, but the prevalence of epizootics, of epidemic catarrhal affections, of peculiarly fatal forms of pneumonia, and other diseases which are now attributed to the presence of minute organisms in the atmosphere, renders such a source highly probable.

It is proper to say here that, though the argument is very strong in favor of regarding the genitalia of puerperal women as the exclusive point of entry into the system of infectious materials, it seems impossible at the present time to make all the facts coincide with such a theory. I have the records of a number of cases occurring during an epidemic of puerperal fever, in which patients were either attacked with fever previous to parturition, or in whose cases the unusual length of labor, the frequency of post-partum hemorrhage, and the imperfect contraction of the uterus, immediately after confinement, were signs of some abnormal influence exercised upon the economy at an early period of labor, previous to the existence of traumatism. That deleterious materials may find other channels for entering the system than a wounded surface, is evidenced by the cachectic condition not unfrequently produced in physicians by too assiduous attendance in dissecting-rooms and places in which post-mortem examinations are conducted. One severe and rapidly fatal case of puerperal fever, which occurred in Bellevue Hospital, I find it impossible to attribute to any other cause than that the woman, for five months previous to her confinement, served as a helper in a lying-in ward. The post-mortem examination disclosed no special local lesions, but her symptoms were those of intense septicæmia. It does not yet seem quite time to give up the idea that, under exceptional circum-

stances, the respiratory, and probably the digestive, tracts, may allow the passage of materials of a septic character.

Inoculation.—Another and frequent source of puerperal fever is by direct inoculation. Any material of a septic character, introduced into the genital passages of a woman during or after confinement, may produce a general infection of the system. But the point upon which I wish especially to dwell is that it is possible to trace epidemics of puerperal fever directly to carrying puerperal poison from patient to patient, through the medium of attendants. In such cases, changes in wards and the most rigid sanitary precautions avail but little, as long as the affected *personnel* is continued in charge. Unless this fact is fully recognized, all the cleverest devices in hospital construction will fail to prevent the occurrence of disasters. In epidemics this source of danger is especially to be guarded against, as septic poison is increased in intensity by successive inoculations. Davaine[1] showed that when a number of animals were poisoned, the one from the other, while from ten to fifteen drops of putrid blood were required to produce death in the first animal, one ten-trillionth part of a drop was sufficient in the twenty-fifth animal of the series, and in puerperal fever epidemics a similar augmentation in the deadliness of the poisons generated by patients is observed.

The nurses in hospitals and in private practice are usually the carriers of contagion. In studying the records of New York City for nine years, I find, however, that the occurrence of two deaths from puerperal disease, following one another so closely as to lead to the suspicion of inoculation, occurred to thirty physicians; a sequence of three cases occurred in the practice of three physicians; one physician lost three cases, and afterwards two, in succession; one physician had once two deaths, once three deaths, and twice four deaths following one another; finally, a physician reported once a loss of two cases near together, then of six patients in six months, and then of six patients in six weeks. Thus in the practice of more than 1200 physicians, in nine years, I find, excluding cases occurring in hospitals, that the experience of thirty-six only lends color to the idea that puerperal fever is due to criminal neglect on the part of the medical profession. Undoubtedly in many of these cases, too, the responsibility is only apparent, as when a practitioner has, for example, had the misfortune to lose in one week a woman from puerperal convulsions, and another in the following week from placental hemorrhage. Singularly enough, not one of the sequences mentioned occurred in the practice of a physician connected with a lying-in hospital. In face of the charge that the physicians holding obstetrical appointments in public institutions are active disseminators of puerperal fever through populous communities, I find that the total loss from all puerperal causes occurring in the private practice of ten physicians intimately associated with such institutions, numbered, during the nine years, but twenty-one cases. Of these, thirteen were the result of ordinary accidents, and only eight cases of metria proper, of which one was developed before the physician was called in attendance; whereas a single physician, holding no hospital appointment, lost during the same time twenty-seven cases, of which twenty-one were cases of metria.

I have been interested in endeavoring to ascertain how far experience corresponds with Semelweiss's theory that puerperal fever owes its origin to poisonous materials obtained from dissecting-rooms, and introduced into

[1] Report before the Académie de Médecine, Sept. 17, 1872.

the genital canal by the hands of physicians attending cases of labor. With this view I have made personal application to a number of gentlemen who have engaged in midwifery practice while performing the functions of demonstrators of anatomy in our medical schools. Dr. H. B. Sands, of the College of Physicians and Surgeons, reports that in the five years during which he held the office of demonstrator, he attended about sixty cases of labor. All did well. He lost his first patient, from child-bed, a short time after he had resigned his position in the dissecting-room. Dr. J. W. Wright, the present Professor of Obstetrics in the Medical Department of the New York University, who held for one year the position of Demonstrator in the Woman's College, writes me that "during the year, I attended one hundred and four cases, including twenty-two forceps cases, two of craniotomy, two of podalic version, and four of breech presentation. Of this number I lost two cases, one from phlegmasia dolens complicating uræmia, from both of which troubles the patient had suffered during her previous labor, and one from double pneumonia, the result of unusual exposure following confinement. Out of these one hundred and four cases, I can recall but three or four cases of metritis, and those of a mild character; I have never thought they had any special connection with my duties in the dissecting-room. I may add that for ten years I have attended a pretty large number of confinements each year, and that during the whole of this time I have been in the habit of making autopsies as occasion has offered, and of handling and examining pathological specimens both in and out of the dissecting-room, notwithstanding which, my death record among this class of cases has been unusually low." Dr. Samuel B. Ward, formerly Demonstrator at the Woman's College, at present Professor of Surgery in the Medical School at Albany, writes: "While I was daily in the dissecting-room, during the winter sessions of the school from 1868 to 1872, I attended thirty-two confinements of which I have notes. All of the patients recovered, nor did any of them suffer from any complication that could be traced to infection." It is familiarly known that after Semelweiss had introduced the practice, among the physicians attending patients at the large lying-in hospital in Vienna, of washing the hands in a solution of chloride of lime, there was a great diminution in the mortality which prevailed, notwithstanding which, G. Braun reports, however, that in 1857, in the month of July, in two hundred and forty-five deliveries there were seventeen deaths. The following month, Prof. Klein gave orders to suspend the use of disinfectants. By chance, in August there were only six deaths out of two hundred and fifty confinements, and in September, of two hundred and seventy-five patients, none died. From 1857 to 1860, the mortality was slight, though disinfectants were not used, while during the three following years, in spite of the systematic and persistent employment of these agents, the death-rate once more assumed formidable proportions.[1]

Of course I do not wish to underrate the importance of Semelweiss's labors. There is no question but that it is a perilous experiment to pass from the dissecting-room to a patient in labor, without employing rigorous measures to disinfect the hands and all parts of the person brought into contact with the dead body. But it is well to call attention to the fact that puerperal fever is not due to any single, simple cause, nor is to

[1] Rückblicke auf die Gesundheits Verhältnisse unter den Wöchnerinnen, u. s. w., S. 32, 33.

be effectually guarded against by a single precaution; and again that cadaveric poison does not of necessity exist in every cadaver examined. Haussmann found that injections into the vagina of gravid rabbits, in the latter half of pregnancy, of serum from the corpse of a person not dying of septicæmia, produced no fatal results, while rapid death resulted from injections, under the same conditions, of pus from the abdomen of a woman dying from puerperal infectious disease.[1]

TABLE II.—*Deaths occurring in New York, between the years* 1867 *and* 1875, *inclusive, from metria, excluding other causes which proved fatal during the puerperal period.*

Year.	Jan.	Feb.	Mar.	April	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Total deaths from metria.	Total from all causes.
1867 . . .	16	10	14	16	12	5	6	8	5	5	9	14	120	238
1868 . . .	17	14	27	16	18	9	12	8	7	3	8	12	151	267
1869 . . .	25	26	18	10	15	14	12	15	8	12	16	19	190	312
1870 . . .	12	17	37	27	20	10	12	16	10	12	11	17	201	333
1871 . . .	21	30	22	25	26	17	11	13	7	7	12	23	214	399
1872 . . .	26	37	41	40	27	21	14	24	13	14	18	32	307	503
1873 . . .	33	40	32	39	29	22	16	15	13	11	23	12	285	431
1874 . . .	19	32	24	33	34	21	18	15	10	7	7	25	245	439
1875 . . .	28	28	40	30	22	17	10	10	5	11	14	19	234	420
Total . .	197	244	255	236	203	136	111	124	78	72	118	173	1947	3342

Season of Year.—I have in a special table (Table II.) classified all the deaths from metria in New York, during nine years, according to the months in which they occurred, and from this it appears that more than twice as many deaths took place between the six months from December to May inclusive, as between the six months from June to November inclusive. The greatest mortality occurred in February and March, amounting to 499 cases, or rather more than one-fourth of the entire number. The smallest death-rate occurred in September and October, in which months but 150 deaths, or one-thirteenth of the entire number, took place.

[1] Untersuchungen und versuche über die Entstehung der übertragbaren Krankheiten des Wochenbettes; Beiträge zur Geburtshülfe und Gynäkologie, Bd. iii, Heft 3, S. 374, u. ff.

TABLE III.—*Deaths from puerperal causes in the different city wards of New York.*

	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.
1867 . . .	4	0	0	10	6	3	7	17	4	15	2	13	2
1868 . . .	4	1	1	12	3	4	9	11	9	8	8	28	7
1869 . . .	7	0	0	5	2	5	20	24	8	19	13	20	16
1870 . . .	4	0	1	5	6	5	6	20	4	19	13	21	16
1871 . . .	6	2	4	4	8	3	17	17	11	21	21	27	10
1872 . . .	6	1	4	5	4	11	13	19	10	26	14	47	11
1873 . . .	8	0	2	5	4	12	12	19	8	20	14	40	14
1874 . . .	7	0	1	7	7	14	14	26	8	23	11	24	13
1875 . . .	9	2	2	3	4	7	17	14	13	17	20	25	18
Total . . .	55	6	15	56	44	64	115	167	75	168	116	245[1]	107
Estimated number of births[4] . . .	4284	387	1089	7053	5049	6237	12,855	10,395	14,137	12,325	18,975	14,107	9898
Ratio of deaths to births . . .	1 : 78	1:64.5	1 : 72	1 : 126	1 : 115	1:97.5	1 : 112	1 : 62	1 : 188	1 : 73	1:163.5	1 : 57	1 : 92

	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.	XXIII.	XXIV.	Total.
1867 . . .	9	9	8	38	15	22	18	24	12	....	....	238
1868 . . .	7	5	6	32	18	17	21	35	21	....	....	267
1869 . . .	10	4	9	33	21	37	22	27	10	....	....	312
1870 . . .	2	5	9	41	28	34	19	53	22	....	....	333
1871 . . .	8	6	18	41	21	38	37	54	25	....	....	399
1872 . . .	14	11	23	45	24	85	45	43	42	....	....	503
1873 . . .	5	7	14	40	24	51	40	54	38	....	....	431
1874 . . .	7	6	20	36	8	56	39	66	34	12	....	439
1875 . . .	14	12	18	51	27	53	19	30	39	5	1	420
Total . . .	76	65	125	357	186	393[2]	260	386[3]	243	17	1	3342
Estimated number of births[4] . . .	7850	8197	14,363	28,323	17,701	25,542	22,394	16,840	21,191			
Ratio of deaths to births . . .	1 : 103	1 : 126	1 : 113	1 : 79	1 : 95	1 : 65	1 : 86	1 : 45.6	1 : 87			

[1] The twelfth ward includes Ward's Island Hospital and the Colored Home, which furnished together 102 deaths; deducting these the ratio would be 1 : 99.

[2] The nineteenth ward contains the Nursery and Child's Hospital and the Charity Hospital, which furnished together 83 deaths; deducting these the ratio would become 1 : 82.

[3] Includes 189 cases from Bellevue Hospital, deducting which the ratio would become 1 : 85.

[4] The number of births in each ward is roughly estimated by assuming the population of 1870 to be the average of the nine years, and then allowing 33 births to the thousand. This method makes no pretence to accuracy, but it is not likely that exact returns would materially alter the relative proportions given. It furnishes a total of 279,000, or 5000 less than that obtained by assuming an annual increase of 15,000 to the entire population of the city.

Influence of Locality.—I have likewise prepared a table showing the number of deaths from puerperal causes occurring in each year in the several wards into which the city is divided, and the ratio of deaths to

the estimated number of births in each ward. The average ratio is, as has been already stated, about 1 to 85. In the 9th Ward, however, it was 1 to 188; in the 11th Ward, 1 to 163.5; and in the 15th Ward, 1 to 126. These wards make the most favorable showing in the city. By reference to the map, they are all found to occupy the same territorial zone. In the 17th Ward, situated between the 11th and 15th Wards, the ratio of deaths to births, it is true, was 1 to 79; but the 17th Ward is the most densely populated section of the city. As neither the 9th nor the 11th Wards are otherwise especially favored, both containing a large and crowded tenement-house population, it is hard to understand their relative immunity from deaths due to puerperal diseases, unless upon the assumption that local causes, probably superior drainage, render them exceptionally free from endemic sources of disease.

TABLE IV.—*Deaths from puerperal causes in the 18th, 19th, and 21st wards, with the relative proportion of deaths, not occurring in Hospitals, among the population east and west of Third Avenue.*

Year.	Total.	Died in hospital.	Deaths upon the west side of Third Avenue.	Deaths upon the east side of Third Avenue.	Relative proportion of deaths on west and east side, expressed in percentages.
1867 . . .	61	20	8	33	24 per cent.
1868 . . .	70	28	8	34	23.5 "
1869 . . .	85	23	12	50	24 "
1870 . . .	115	34	9	72	12.5 "
1871 . . .	113	32	15	66	22.7 "
1872 . . .	173	43	10	120	8.3 "
1873 . . .	129	37	15	77	19.5 "
1874 . . .	130	45	14	71	19.7 "
1875 . . .	110	15	8	87	9.2 "
Aggregate . .	986	277	99	610	16.2 "

Social State.—I have, too, sought to ascertain whether the wealthy and well-to-do classes enjoy special immunities from puerperal disease. The district bounded by Fourth and Sixth Avenues, and Twelfth and Fifty-ninth Streets, contains the great proportion of those who make up the privileged classes. In this portion of the city, the proportion of deaths from puerperal causes to the deaths from the same sources throughout the city, is as 1 to 43. The precise population of the district I cannot obtain, but it is safe to say that it contains at least one-thirtieth of the entire population of the city.

The eighteenth, nineteenth, and twenty-first wards, included between Sixth Avenue and the East River, and extending from Fourteenth to Eighty-sixth Streets, contain in a marked degree the two extremes of wealth and poverty. Third Avenue separates the territory into two equal parts, and forms a fair dividing line between the upper and the lower social strata. The population upon the west side of Third Avenue is, it is true, less than that upon the east side,[1] possibly not more than half as great, but the mortality of nine years from puerperal causes was in this favored region less than one-sixth, even after deducting

[1] I am informed by politicians that the vote in the ward mentioned is about 50 per cent. larger on the east side of Third Avenue.

deaths occurring in hospitals, which for the most part lie upon the east side. In 1872, the fatal year, the proportion was one to twelve.

TABLE V.—*Deaths from scarlatina, diphtheria, and erysipelas, and from all causes, by quarters, compared with deaths from metria, for the nine years ending December* 31, 1875.

Causes of death.	1867.					1868.					1869.				
	1st quarter.	2d "	3d "	4th "	Total.	1st quarter.	2d "	3d "	4th "	Total.	1st quarter.	2d "	3d "	4th "	Total.
Scarlatina	264	194	89	103	650	318	351	102	90	861	299	285	159	223	966
Diphtheria	79	56	53	63	251	92	87	43	55	277	87	89	66	86	328
Erysipelas	40	32	20	22	114	31	45	13	13	102	54	55	14	29	152
All causes	5197	5121	7516	5325	23,159	5953	5523	8658	4755	24,889	5919	6065	7390	5793	25,167
Metria	....	....	....	....	120	....	....	....	....	151	....	....	....	....	190

Causes of death.	1870.					1871.					1872.				
	1st quarter.	2d "	3d "	4th "	Total.	1st quarter.	2d "	3d "	4th "	Total.	1st quarter.	2d "	3d "	4th "	Total.
Scarlatina	418	258	106	193	975	274	196	137	184	791	301	367	132	190	990
Diphtheria	94	69	67	78	308	89	49	46	54	238	93	86	87	180	446
Erysipelas	50	42	4	25	121	54	50	18	23	145	64	73	23	25	185
All causes	6517	6293	8416	5949	27,175	6622	6621	7833	5900	26,976	7406	8737	10,025	6479	32,647
Metria	....	....	....	....	201	....	....	....	....	214	....	....	....	....	307

Causes of death.	1873.					1874.					1875.				
	1st quarter.	2d "	3d "	4th "	Total.	1st quarter.	2d "	3d "	4th "	Total.	1st quarter.	2d "	3d "	4th "	Total.
Scarlatina	242	275	202	326	1045	322	239	166	152	879	186	161	63	104	514
Diphtheria	169	184	316	482	1151	334	314	320	697	1665	608	549	489	683	2329
Erysipelas	83	60	30	31	204	49	62	25	33	169	66	51	21	29	167
All causes	6815	6583	8983	6703	29,084	6328	6548	8509	7342	28,727	7840	7065	9257	6547	30,709
Metria	....	....	....	....	285	....	....	....	....	245	....	....	....	....	234

Relations to Zymotic Diseases.—I prepared, some time ago, tables to answer the inquiry as to whether there was any relation between the frequency of cases of scarlatina, diphtheria, and erysipelas, and those from metria proper. Previous to their publication, however, I was anticipated in my deductions by a paper upon the same subject by Dr. Matthews Duncan.[1] I therefore simply submit the tables with the comment that

[1] On the alleged Occasional Epidemic Prevalence of Puerperal Pyæmia, or Puerperal Fever, and Erysipelas; Edinburgh Medical Journal, March, 1876, p. 774.

they show no relation to exist between the statistical frequency of puerperal fever and the zymotic diseases mentioned. The tables do not, however, invalidate any direct testimony which goes to show that, in individual instances, a real connection between puerperal fever and the zymotic diseases may exist.

Prevention of Puerperal Fever.

Of the 3342 deaths from puerperal causes, 420 died in hospital, or one-eighth of the entire number. Of the 1947 cases of metria, about 300, or not quite one-sixth, were contributed by the hospitals. Upon such a showing the first impulse would be to cry out loudly for the suppression of maternities. But a wiser policy suggests the inquiry whether the large mortality in hospitals is an evil necessity. To this the answer is "No." It is possible, in the present state of our scientific knowledge, to so control the conditions that favor the generation of puerperal diseases in large hospitals, as to make them safe asylums for the needy.

Thus in the Charity Hospital, Dr. Kitching reports the number of confinements between July 1, 1874, and May 1, 1876, at 1149; of which 20 ended in death, or 1 in 57. But only six were cases of metria, or 1 in 191 deliveries, a better record than that afforded by the average of the city at large. In the Nursery and Child's Hospital, there was but one death from metria in 1874, and one in 1875. In 1872, the number of confinements was 205. I regret that a request for the number of confinements in the succeeding years has been refused me. The Marion Street Lying-in Asylum, an excellent institution for married women, reports two deaths from metria in nine years. Dr. Goodell reports that, at the Preston Retreat, in 756 cases of labor there have been but two deaths from septic disease; Winckel,[1] of the Lying-in Institution in Dresden, reported, in 1873, eighteen deaths from metria, or 1.8 per cent., but from the 10th of January to the 7th of July, in 570 deliveries there was but one case of septic disease; in the year 1872 the death-rate exceeded 5 per cent. The reduction in the mortality was no fortuitous circumstance, but was due to rigid measures for the prevention of disease.[2] Stadfeldt reduced the mortality from puerperal fever, in the Maternity Hospital of Copenhagen, from 1 in 37, the proportion between the years 1865 and 1869, to 1 in 87, between the years 1870 and 1874.[3] Dr. Johnston reports in the Rotunda Hospital of Dublin, during seven years, 7860 deliveries (I have excluded the cases of abortion) with 169 deaths, of which 85, or 1 in 91, were from metria.[4]

Prof. J. J. Bischoff reports that in the Maternity of Basel, between the years 1862 and 1867 inclusive, in 514 confinements there were 33 deaths, or in about the proportion of one to sixteen. In 1868, of 75 patients confined, there died three, or one in twenty-five. In 1869, of 86 patients confined, seven died, or one in twelve. In 1870 effective measures of prevention were introduced, and, that year, in 80 confinements there were no deaths.[5] In 1871, two out of 124 patients died, or one in sixty-two. In 1872, in 153 confinements there were four deaths, a proportion of one

[1] On the Means employed at the Preston Retreat for the Prevention and Treatment of Puerperal Diseases, p. 13.

[2] Berichte und Studien, u. s. w., Leipzig, 1874, S. 183.

[3] Les Maternités, leur Organisation et Administration; Copenhague, 1876.

[4] Series of Clinical Reports, from 1870 to 1876 inclusive.

[5] J. J. Bischoff, Zur Prophylaxis des puerperal Fiebers; Basel, 1876, S. 12, 13.

to nearly forty. The next year, 1873, strict measures were again put in force, and in 184 labors there were two deaths, or one in ninety-two. In 1874, in 214 labors there were three deaths, one to seventy-one, but one of the patients entered the hospital with endocarditis, of which affection she died. In 1875, in 204 confinements there were two deaths, one from hemorrhage, and one from pyelitis which existed at the time the patient entered the hospital.

Surely these figures do not support the idea that it is better for a woman to be confined in a street gutter than to enter the portals of a lying-in asylum. Dr. Goodell's experience shows that a hospital for respectable married women may be so conducted that its inmates may enjoy absolutely a greater degree of safety than do women in their homes, surrounded by all the aids that wealth can command. Equally good results are not to be obtained in hospitals which are open to unfortunates of every class. But there is much misapprehension and confusion of ideas respecting the fate of these women, when no charitable provision is made for them. In Copenhagen, the Maternity Hospital is closed for from six to eight weeks in the summer time. During this period, unmarried, parturient women receive pecuniary assistance from the hospital to enable them to obtain a place in which to be confined. Now Stadfeldt reports a larger mortality among this class than among those delivered in the hospital. Yet they are confined at a favorable season of the year, without any communication with the furniture, the *sage-femmes*, or the physicians of the hospital. As they fortunately receive nothing but money, that can hardly be suspected of communicating contagion. What their fate would be in New York City, perhaps, may be judged from the following facts: Excluding cases confined in hospitals, nearly one-thirtieth of all the deaths and one-twenty-fourth of the cases of metria are reported by four practitioners. Ten practitioners out of 1200 signed the death certificates of one-fifteenth of the women dying from puerperal causes, and one-tenth of the cases of metria. But it is not to be supposed that these deaths were all the result of malpractice and incompetence. The true history of most of them probably was that the doctor was engaged to attend the case of confinement for a small fee, with the understanding that he should make no calls subsequently, unless specially summoned by the friends of the patient. The latter, left to ignorant care, or perhaps without any assistance whatever, and exposed to all the pernicious influences bred by poverty, when illness supervened probably did not call the physician to her aid until the time for help had passed, so that in the end his professional functions were confined to procuring the requisite permit for burial.

Humanity demands that charity should furnish places of refuge in which poor outcasts can receive assistance during the perils of childbearing. If we must then have maternities, we should make them safe, and this can be in great measure accomplished by remembering the twofold source of danger arising from a poisoned atmosphere and direct inoculation. A hospital must be clean, spacious, and well ventilated, or its atmosphere will become charged with decomposing albuminoid substances, and produce nosocomial malaria. But the experience of the Hôpital Cochin,[1] a costly, palace-like structure, with every appliance of art, proves that fresh air alone does not protect patients from the consequences of inoculation. On the other hand, the most rigid sanitary pre-

[1] Billet, Réforme des Maternités, p. 75.

cautions observed by the attendants will not prevent a badly ventilated ward from becoming unhealthy, unless unoccupied wards are kept to which patients can be transferred upon the first admonition of danger. At the Charity Hospital, the service consists of two large wards, occupied for three weeks at a time alternately. Dr. Goodell states that at the Preston Retreat the wards are used invariably in rotation. In connection with the Maternity at Copenhagen, there are a number of small supplementary hospitals scattered through the city, which serve as safety-valves for the central institution. Artificial methods of ventilation render the task of keeping the wards healthy, comparatively easy. They do not need, however, to be complicated and expensive. The good repute of the Rotunda Hospital, it seems to me, is in large measure due to the natural ventilation afforded by open fireplaces.

The testimony is very general to the advantages of frequently washing the wards with carbolic acid. At the Charity Hospital, in addition, the wards are fumigated three times a week by burning a mixture of carbolic acid, chloride of lime, and sulphur. During parturition, Stadfeldt exposes the woman to a fine spray of carbolic acid. If, after confinement, vaginal washes of carbolic acid should be used, every woman should be supplied with her own nozzle, which can be made of glass tubing, and attached in turn to the irrigator. When not in use, these should be kept in solutions of carbolic acid.

A liberal supply of metallic catheters should be always kept on hand and, when not in use, placed in a disinfectant fluid. Sponges, in a hospital, are an abomination. Oakum, lint, or old cloths should be employed to receive the discharges, and, when removed, should be placed at once in a vessel containing disinfectants, and then, instead of going into the washtub, should be burned. As regards all prophylactic measures, cheapness is not to be consulted.

As puerperal patients require much attention, lying-in wards should possess a sufficiency of attendants, best, perhaps, by making them training schools for nurses. Nurses should be intelligent enough not only to comprehend their duties, but their responsibility. Physicians should wash their hands with disinfectants before and after making examinations, and nurses should do the same whenever their hands are brought into contact with vaginal secretions. D'Espine has shown that the lochia of the third day of a healthy patient will poison a rabbit.[1] A nurse employed in the puerperal wards ought not to have access to cases of labor. A patient attacked with fever should be immediately removed to another part of the building, and the nurse in attendance should go with her. Special wards for the recently confined should never be established. As enforcement of details is essential to success, a lying-in hospital should have a resident medical head with plenary power to enforce the most rigorous discipline. The adoption of Lister's principles in maternity hospitals has, when carried out with the requisite discipline, served to dissipate the well-founded objections to their existence. It makes no difference whether they are old hospitals or new, cottages or pavilions. All that is required is space, light, air, intelligent nursing, an active head, and generosity in needed supplies, to keep them as free from infectious diseases as are the homes of the better classes. I have already shown, in another paper, how, through limited room, ignorant nurses,

[1] Contributions à l'Étude de la Septicémie Puerpérale, p. 18.

lax discipline, and niggardly management, an epidemic, costly in life, could be generated.

So far from tearing down the maternity hospitals now in existence, new maternities of small size, for married women, modelled after the Preston Retreat, are urgently called for to meet the present waste of life among the very poor. I cannot but hope that such institutions, intelligently constructed, ordered, and maintained, may yet be the outcome of private charity. A small maternity has the advantage over a large one that, when a dangerous element is introduced, repressive measures can be more rapidly and effectively carried into execution. I have already taken up so much time that I will not dwell upon the duties of the physician toward his private patients: how he should avoid conveying to them zymotic diseases, or septic poisons; how he should understand that the proper management of labor involves a normal puerperal state; how he should guard lest the antecedents of his patients should unfavorably affect the period of parturition and child-bed. I have only to add that, among the wealthy and refined, there is an annual sacrifice of lovely women, due, not to want of skill or care, not to infection, but to the fact that the enervating influences of civilization and the pressure of social life, have apparently unfitted them for offering effective resistance to any form of traumatism. Dr. E. H. Clarke's little work entitled "Sex in Education," strikes the key note of a subject requiring on the part of obstetricians the profoundest consideration.

In closing this paper, I beg to offer the following conclusions:—

I. There is no specific, puerperal fever, and, as long as, when the term is limited, each physician uses it in some sense personal to himself, it is best to employ it as a general term for all the febrile affections peculiar to the puerperal state.

II. Puerperal fever, the term being used as above, may be either a non-infectious or an infectious disease.

III. The non-infectious form is due to traumatic inflammations of a simple character, to old peritoneal adhesions, to moral causes, and to the vulnerability of the patient.

IV. The infectious form, on the other hand, is a septic disease, intimately associated with the existence in the tissues of minute organisms which form the connecting link between puerperal fever and erysipelas and diphtheria.

V. Its causes are certain atmospheric conditions (nosocomial malaria of hospitals), and infection from septic materials. It prevails most in certain winter months, and finds its victims chiefly among the very poor.

VI. Prevention is best accomplished in hospitals by the adoption of Lister's principles.[1] Maternity hospitals are no longer necessarily foci

[1] The following regulations, enforced by Bischoff, in Basel, will serve as an example:— Removal from the genitalia, after confinement, of *débris* capable of undergoing putrefaction. Protection of the wounded surfaces from all air not impregnated with carbolic acid. Hands of attendants, and all instruments used in labor, to be disinfected with carbolic acid. A full bath to be given upon the advent of labor, and the vagina to be washed out with a two per cent. solution of carbolic acid. In cases of premature rupture of membranes, of protracted labor, artificially induced premature labor, and of dead fœtus, a repetition of the injection every two hours. Hands and instruments to be lubricated when necessary with a ten per cent. preparation of carbolized glycerine. In cases of version, of artificial removal of the placenta, of putrid fœtus, of post-partum hemorrhage with relaxation of the uterus, employment of intra-uterine injections of two to three per cent. solutions of carbolic acid. After the end of labor, the external genitalia to be inspected, and a ten per cent.

of disease. Small maternities, if well managed, meet a need among the very poor, and their establishment by the charitable should be encouraged. In private practice the physician should never, and need never, be the carrier of contagion. The antecedents of nurses should be investigated. Auto-infection should be very rare in cases properly managed during the period of labor.

VII. The question of personal responsibility cannot be too strongly impressed upon the medical profession.

carbolized oil to be applied to all lesions. Rupture of the perineum to be closed with carbolized silk or catgut sutures. The parts to be washed clean, and picked lint soaked in carbolized oil (ten per cent.) to be laid upon the vulva. Vaginal injections of a two per cent. solution of carbolic acid to be used twice a day—or, in cases of difficult labor, putrid fœtus, or retained portions of ovum, to be repeated every two hours. In cases of retained portions of placenta, hindering the contraction of the uterus, intra-uterine injections of a two or three per cent. solution of carbolic acid in water twice a day. The nurse to wash at each visit the genitalia and inner surface of the thighs, with a lukewarm solution of carbolic acid. I have already given the results of this treatment.