Kober (Geo. M.)

Prevention of Puerperal Fever.

By GEO. M. KOBER, M. D.,

WASHINGTON, D. C.,

Professor of Hygiene, Medical Department,
University of Georgetown.

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Puerperal fever has been defined by the College of Physicians of London,[1] as "a continued fever, communicable by contagion, occurring in connection with child-bed and often associated with extensive local lesions, especially of the uterine system."

This *definition* was characterized by our own Dr. Fordyce Barker, who crossed the Atlantic purposely to join in the discussion, as "absolutely correct." This is not surprising, when we remember that this author believed it to be a fever which is peculiar to puerperal women, that the symptoms of this disease are essential, and are not the consequence of any local lesion, and that it is as much a distinct disease as typhus fever, typhoid fever or relapsing fever. Dr. Barker also held that "any of the local inflammations may occur in the puerperal woman without puerperal fever; and on the other hand, puerperal fever may be so severe as to destroy life without sufficient local disease to account for the symptoms or explain the cause of death."[2]

Before entering upon the dominant feature of this paper—viz., the prevention of puerperal infection—it will be well to point out

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briefly the recent views as to the etiology of this affection, and that the above definition requires some modification; and, lastly, that whilst the disease is most frequently communicated by the attendants, they cannot be held responsible for every case of puerperal fever.

I believe the sooner we abandon the idea that puerperal fever is a specific disease, peculiar to lying-in women, and adopt the view that a parturient woman is precisely in the same condition as a patient after a surgical operation, and therefore liable to all the local and general infections, the better it will be for the cause of humanity.

As regards the *etiology of puerperal fever*, there has always been, and is even now, considerable difference of opinion. Some authors insist that the virus of this disease and of erysipelas, are practically the same. Gusserow,[3] supported by clinical and experimental evidence, denies this; whilst Winckel[4] and Hartmann[5] are prepared to assert that in a large number of cases, the disease is brought about by the agency of Fehleisen's micrococcus erysipelatos, which at least was present in all their cases of purulent peritonitis, diphtheritic endometritis, and the ulcerative lesions accompanying puerperal fever. The evidence presented by Hartmann leaves little room to doubt that these organisms not only invade the lymph-spaces and vessels of the skin, but also the mucous membrane and internal organs, and may enter the blood-vessels directly through the abraded surfaces of the mucous membrane of the genital organs. Whilst Hartmann is careful not to assert that puerperal fever is invariably due to Fehleisen's micrococcus, he is quite positive that a large number of cases are thus caused. There are also a goodly number of physicians who believe that the virus of scarlet fever and of diphtheria may induce puerperal fever, whilst others insist that it is simply due to blood-

poisoning from the absorption of decomposing blood or tissue either in the uterine or vaginal cavity—the absorption having been rendered possible by abrasions or lacerations occurring during delivery.

As a matter of fact, this difference of opinion is by no means as great as would appear at first sight, and I believe the conflicting views will be fully reconciled in future. In the first place, a large number of competent bacteriologists believe that the streptococcus pyogenes and Fehleisen's streptococcus erysipelatos are identical. We also know that the streptococcus and staphylococcus are generally present in scarlet fever, diphtheria, and especially in the so-called pseudo-diphtheritic affections, and in septic wounds.*

From this standpoint, puerperal fever, erysipelas, septicæmia, pyæmia, pseudo-diphtheria and certain throat complications of scarlet fever, are all caused by septic germs, chief of which are the streptococci and staphylococci. But it must be remembered that over thirty other organisms have been isolated and described in connection with the suppurative, pyæmic or allied processes in man or the lower animals; and the point we wish to emphasize is, that any germ capable of producing the various manifestations of wound infections, is capable of causing puerperal infection. Puerperal fever is, therefore, nothing more nor less than one of the many phases of septic infections.

In addition to the germ, we must have a suitable soil for its proliferation and pathogenic effects; hence the condition known as predispo-

* Prudden reports twenty-four cases of diphtheria, in which in all but two he demonstrated a streptococcus, probably identical with the streptococcus pyogenes and streptococcus erysipelatos; and Baginsky reports that of 154 cases of diphtheria treated under his supervision, in thirty-six only streptococci and staphylococci could be demonstrated. These same germs are found in the septic throat affections of scarlatinous patients.

sition plays an important rôle; this we find in the average civilized woman, by a lowered vitality, the result, perhaps, of long suffering from headaches, neuralgia and indigestion during pregnancy, and not infrequently an abnormal loss of blood, great fatigue or exhaustion during labor. Moreover, we have a local predisposition in weak sexual and pelvic organs, and possibly in injuries, in the form slight tears and abrasions. After infection has taken place, no organ affords a better cultivating chamber than the uterus filled with a suitable medium like the lochia of a parturient woman. The blood, too, owing to an excess of waste products, the result of great muscular exertion and the return of the womb to its non-pregnant state, affords the best possible papulum for disease germs. While in the majority of cases the germs are introduced into the vagina or uterus by the hands, or instruments of the attendants, still we shall presently allude to evidence tending to show that a puerperal woman may be infected with germs which were in her genital organs before delivery. In any event, it is reasonable to assume that the germs, however introduced, remain for a time at least at the point of invasion and vicinity; here they grow, and as they grow they evolve the so-called ptomaines, some of which are so deadly that even small quantities may prove fatal, whilst many of them act as irritants and cause an inflammation; this, as in the case of erysipelas, may go on without suppuration. Other bacterial forms cause the white blood cells to gather about the parts; and as they proliferate in overwhelming numbers the leucocytes die, and we have inflammation with suppuration, as in purulent metritis, salpingitis or peritonitis; whilst still others cause inflammation with necrosis, as seen in the instances of diphtheritic endometritis and the ulcerative lesions.

An exact classification would be premature at this time. The most we can say is, that the

effects of these septic germs may be local and constitutional, but always the result of absorption of their toxic products. Take, for example, a case which begins within twenty-four hours after delivery with an intense and long chill, but with little or no pain in the abdomen; the face is anxious, skin pale or purplish, tongue dry or brown, pulse rapid and extremely weak, features pinched, the temperature may be high, or even subnormal, there may be vomiting, but more frequently involuntary evacuations, with delirium, suppressed or scant urine, and the patient dies within twenty-four or thirty-six hours. If these symptoms, which are also seen in other cases of acute septicæmia, are not the result of a ptomaine intoxication, it will be difficult to account for them in any other manner. We need not be surprised if in just such instances we fail to find any great pathological changes, for the simple reason that the proliferation of the germs has gone on with such a rapidity as to overpower the system before marked lesions could be produced. A similar condition is observed in some malignant forms of scarlet fever and diphtheria, but just as we see various shades and gradations in all infectious diseases, so we may expect to observe differences in degree in puerperal infections, conditioned upon the amount of the poison, the power of resistance of the patient, and the character and seat of the resulting pathological lesions.

We have already pointed out how different forms of bacteria may cause different types of inflammatory lesions, and we can readily surmise how the seat of these lesions may be influenced by the channels of infection. In some cases, the invaders may not be carried beyond the next lymphatic glands, there to be arrested or disposed of by the vis medicatrix naturæ; in other cases, the same class of germs may find their way through the lymphatics into the abdominal cavity, and from there invade the

pleura or pericardium; in some patients, the ravages of the microbe may be confined to the vagina, uterus or appendages, resulting in chronic lesions; while in still others, they may be carried in a detached piece of a uterine thrombus to the heart and through the pulmonary artery and cause infarctions and abscesses in the lungs, from whence again infection may spread to other parts or organs of the body, as the liver, spleen, kidneys and brain, and cause metastatic abscesses, and perhaps various derangements of the nervous system, such as puerperal mania, convulsions, delirium, insomnia, or paralysis. While it is possible that some of these affections may develop simultaneously in the puerperal state, in the majority of instances they are the result of septic infection, not suspected until the autopsy reveals a thrombosis of the veins of the brain, or purulent meningitis.

It is generally held that the lochia of a healthy woman, after confinement, contains no micro-organisms, and that the vaginæ of women, who have never used injections, or been subjected to digital examinations, is free from pyogenic organisms. Credé,[6] therefore, claims that every case of puerperal fever is the result of an infection from without, while others jump to the conclusion that the germs are invariably introduced by the physician or nurse.

While it is true that no puerperal infection can take place without the entrance of germs, it has been shown by Velits,[7] and the experience of nearly every accoucheur, that infection has taken place without a digital examination having been made; and we also know that many of the pathogenic germs may be present in the vagina or exposed surfaces of the body, as the skin and mucous membranes of perfectly healthy individuals.

Steffeck[8] (1892) examined the vaginal secretions of twenty-nine pregnant females, and

found staphylococcus pyogenes albus in nine, staphylococcus pyogenes aureus in three, and streptococcus pyogenes in one.

This, however, affords no argument to the opponents of the "germ theory," for it simply shows that disease-germs may exist within the body and the disorder they give rise to may be absent; and we maintain that an infectious disease does not exist merely because some noxious micro-organisms have taken their abode in the system, but because the host has furnished a suitable soil for their proliferation, and functional and structural changes have been brought about by the agency of bacterial products. The question of soil also explains why a woman may be infected with septic germs which were in her vagina long before delivery, but remained dormant or harmless until the blood and other conditions offered a suitable environment for their proliferation, and why some women, with a decomposing fœtus or placenta, do not infect themselves, and yet are the cause of a profound septicæmia in others.

In the majority of cases, however, the disease is communicated by the attendants, and in order to point out briefly the various sources of the poison and the manner in which it is conveyed, and also for the purpose of illustrating the identity of puerperal and wound or septic infection, we adduce the following data:

Semmelweis[9] was the first to point out that the poison could be brought on the hands of students going from the dissecting-room to the lying-in ward and examining the women in labor. In 1841, an epidemic of puerperal fever occurred in the Maternity of the General Hospital of Vienna and lasted for twenty months. Of 5,000 parturient women, about 800, or 16 per cent., died. From 1841 to 1846, the Maternity was divided into two departments—one for students and the other for midwives; the patients were sent to each of the two divisions

on alternate days. The fatality in the students' wards of 20,042 patients was 1,989 deaths, or 9.92 per cent., and in the midwives', it was only 3.38 per cent., or 691 deaths in 17,791 deliveries. Whilst Semmelweis was endeavoring to solve this mystery, he became convinced, by the death of a friend who fell a victim to phlebitis and secondary abscess after an operation wound, that the pathological lesions in this case were exactly what he had so often noticed in fatal puerperal cases, and he reasoned that the cause in both were blood-poisoning by decomposed particles of a dead body. He therefore ordered every student, before examining a pregnant woman, to wash his hands with chlorinated lime water. At this time, in the middle of 1847, the percentage of mortality was about 12 per cent.; six months later, it was reduced to 3 per cent., and in 1848, the second year of this precautionary measure, the death-rate fell to 1.27 per cent.

Semmelweis,[10] in 1847, also called attention to the fact that puerperal infection may arise from suppuration; and Sir Spencer Wells refers to the fact that the death of several women has been distinctly traced to midwives, who have inoculated the germs of purulent ophthalmia with a fatal result to one woman, and who have carried the poison and fever to a succession of other parturient women who afterwards died of puerperal peritonitis—both streptococci and staphylococci having been found in the pus.

Such cases, of which the medical annals supply a large number, will serve to explain the mystery of occurrences like that of Dr. Rutter, of Philadelphia, who had, in 1842, a large number of cases of puerperal fever, whilst his brother obstetricians had none. He bathed, changed his clothes, shaved off his hair, left the city for a week, and visited his next confinement case with an entire change of clothing; still the patient perished from the dreadful fever. It was not until 1875,[11] that a contemporary practi-

tioner of Dr. Rutter mentioned as a possible explanation, the fact that his collegue was suffering all this time from an obstinate ozæna, acquired from a neglected pustule, following inoculation upon the index finger.

Apart from digital or instrumental inoculation, there is no question that the germs may be conveyed in the air because many of the pyogenic germs have been demonstrated in the air and dust of hospitals, and they may of course cling to the walls, floor, furniture, bedding, clothing, etc. Dr. Garrigues[12] refers to the fact that whilst he was connected with the Maternity Hospital, a new building was erected for the lying-in women; it had scarcely been opened before a violent epidemic of puerperal fever broke out, compelling the evacuation of the building. In the opinion of one of the surgeons, this outbreak was due to guano, with which the adjacent grounds has been covered as a fertilizer.

The cause of another epidemic of puerperal fever in one of the New York Asylums[12] was found to be a dead rat undergoing decomposition in the cellar, and Fehling[12] observed an outbreak of puerperal fever, diphtheria, and erysipelas, which was caused by the leakage of a waste-pipe, the drainage polluting the soil upon which the hospital was erected, and as soon as this was corrected the epidemic stopped.

We know, from clinical experience, that just such unsanitary surroundings, as also bad food, impure air and water, absorption of putrid gases formed in the intestinal tract, etc., predispose to wound infection, and hence also to puerperal infection; but we cannot say whether such conditions actually increase the virulence of otherwise harmless bacteria, or simply diminish the power of resistance, or whether they operate by causing an alteration of the blood, and thus render it a suitable soil for their proliferation. There is good reason

for assuming that puerperal infection may take place through the mucous membrane of the lungs. Depaul, quoted by Garrigues,[12] reported to the French Academy, in 1858, the case of a student midwife, who nursed a very severe case of puerperal fever. While she was washing the genitals, she felt an unpleasant sensation; in the evening she was taken sick, and died on the third day "with all the symptoms of the most characteristic puerperal fever."

The post-mortem confirmed the diagnosis, and she was furthermore found to be a virgin and not in a menstrual period; there was no evidence to show that infection had taken place through abrasions, etc.

That there is a systemic infection in puerperal fever is shown by the fact that Karlinsky[13] demonstrated staphylococci in the milk of a woman suffering from this fever; her infant was attacked with septicæmia, neonatorum in the form of gastro-enteric catarrh, peritonitis, pleuritis, double parotitis, and lobular pneumonia; and the same bacteria were found in the blood and intestinal contents of the child as were present in the milk of the mother.

Sirédy[15] reports another striking case in point. A man suffering for six months from a stercoral abscess was nursed by his two nieces. Both were pregnant, and were delivered with an interval of two months; and, in spite of easy labors, both developed severe puerperal infection, and the child of the first had, a few days after its birth, a whitlow, a large abscess on the buttocks, and erysipelas all over the body.

We have pointed out the various sources of the poison, and the possibilities of even auto-infection are frequent enough, when we recall the fact that Steffeck found pyogenic organisms in the vaginal secretions in 13 out of 29 pregnant females examined by him. This very fact shows that it is not sufficient for us to simply

render our person, fingers and instruments aseptic, but that it is equally essential to remove or destroy any germs which may be present in the vagina before delivery; for no one can know how soon the system may offer a suitable soil for their proliferation and jeopardize the life of the patient.

The accoucheur who neglects the rules of modern midwifery certainly ignores established scientific truths; and though he may flatter himself with having had few if any cases of puerperal fever, he does not deny that many of his patients suffered from some one of the numerous forms of puerperal infection, which he naively calls, however, mere idiopathic affections.

My own experience with antiseptic midwifery has satisfied my conscience and professional pride, for even the old grannies of California appeared to think that I was either very lucky or painstaking in the management of my cases, and, as a result, my obstetric practice increased.

I mention this, not as a matter of egotism, but to impress the importance of preventive measures so aften deprecated by older men. Apart from adhering strictly to the rules of antiseptic midwifery, I have endeavored to place my patients, when engaged sufficiently early, in the best possible condition as regards the general health, before delivery.

Most of the lying-in hospitals have adopted strict rules for the management of cases of labor, whereby all students or physicians are excluded from attending a case of labor, if they have come in contact within the last thirty-six hours with septic germs, whether in the dissecting-room or in the treatment of septic wounds or erysipelatous and diphtheritic patients. In some of the University Hospitals, the attendants are required to register in the "control book," for the purpose of tracing the source of infection, but no enlightened medi-

cal man or student would wish to attend a labor case, under the circumstances mentioned, without taking a full bath with 2 drachms of bichloride of mercury, and devote special attention to the disinfection of his hair, beard, hands, and finger-nails.

As soon as labor sets in, the patient receives a full bath with warm water and soap, and is dressed in clean clothes and placed in a clean bed; a rubber sheet, previously disinfected with a solution of bichloride of mercury (1:1000), is put under the sheet; she then receives an enema of soap suds, and the abdomen, thighs, buttocks, and especially the hair and folds of the skin, are carefully disinfected with solution of bichloride of mercury (1:2000), whilst the vagina is douched either with 2 quarts of this solution, or with a 2 per cent. solution of creoline.

The attendants should be surgically clean—this involves a careful toilet of the hands, and arms, and especially of the finger-nails with brush, soap, and water, after which the hands should be rinsed in water, and immersed in a bichloride of mercury solution (1:2000) for at least three minutes. The vaginal exploration should be as delicate as possible, and not extend, unless necessary, beyond the external os uteri, and of course should not be unnecessarily repeated, and never without previous disinfection of the hand in the bichloride solution. Lint, cotton, or napkins should likewise be wrung out in a warm bichloride solution before application to the vulva, whilst instruments had better be disinfected by immersion in a 5 per cent. solution of carbolic acid. The best lubricant is glycerine with 3 per cent. of carbolic acid or mollin, with 5 per cent. of the same. In tedious labor, the vaginal antiseptic douches are repeated every three hours, and when the presenting part appears at the vulva Garrigues recommends the application of a piece of lint wrung out in the bichloride solu-

tion, which serves other useful purposes besides presenting a too free access of air to the vagina as the fœtus moves to and fro.

In every case where the uterus has been invaded by the hand or instruments of the accoucheur, or after still births with evidence of decomposition, intrauterine injections are called for. About 1 quart of a bichloride of mercury solution (1:4000) or preferably a 2 per cent. solution of creoline of a temperature between 110°–115° F., should be cautiously injected whilst the patient lies on her back; the porcelain jar should not be held more than a foot above the fundus uteri. After the injection, the fluid should be squeezed out from the womb, and permitted to escape from the vagina by placing the patient on her side.

In any case, after the removal of the afterbirth, the patient is again washed with the bichloride solution (1:2000), employing absorbent cotton for the vulva and clean muslin rags for the skin; especial attention should be paid to the removal of the blood-clots from the hairy parts, which is often most quickly accomplished by cutting part of them off. In the Maternity Hospital of New York, it is the rule to compress the womb with the hand for half an hour before the binder is put on, and to the latter is fastened an antiseptic occlusive dressing, recommended and described by Dr. Garrigues[12] to obstetric practice, and consisting, according to his directions, of—(1) a piece of lint, 12x8 inches, folded twice lengthwise, so as to be 3 inches wide; (2) a piece of oiled muslin, 4x9 inches; (3) a large pad of cotton batting, and (4) a piece of muslin half a yard square. The lint is wrung out of the solution of bichloride (1:2000), and carefully applied over the vulva and anus. The oiled muslin is washed with the same solution and placed over the lint, turning the edges forward against the inside of the thighs. These two constitute the antiseptic part of the dressing. The pad of

cotton outside serves only to keep the compress in opposition to the entrance of the genital canal, and is itself held by the muslin kerchief, which is folded like a cravat and fastened to the binder with four pins in front and two behind. A good binder should go down beyond the trochanters, and a /\-shaped opening left at the genitals, which is closed by the pad just described. This dressing is changed every six hours, or oftener, if the patient has a movement or passes her urine in the meantime. Before the fresh dressing is applied, the genitals and nearest parts are irrigated with the bichloride solution (1:2000), the patient lying on a bed-pan. No injection is given; nay, the genitals are not touched." The cost of these dressings averages about one dollar for the whole lying in period, but it has paid me in more than one respect to adopt these precautions.

The results of strict antisepsis, of which Dr. Garrigues' method is of course the type of perfection, have been most beneficial. We have already learned what Semmelweis accomplished by compulsory disinfection of the hands in the Vienna Maternity; the results have been more striking since we have correct ideas of germicides. Whilst a century ago the mortality at the Lying-in Department of the Hotel Dieu amounted to 10 per cent., it has been reduced since 1881 to 1.1 per cent. In Berne,[14] it has been reduced from 4.5 per cent. to 0.83 per cent., and at Basle,[15] the mortality in 1872 still reached 3.33 per cent., whilst in 1886 it was reduced to 0.98 per cent. Of 29,098 deliveries by prominent obstetricians in Great Britain and Ireland,[16] there were 251 deaths, or 0.86 per cent. The statistics of the Maternity at Lyons before and after the introduction of antiseptics, furnished by Dr. Vincent, are extremely suggestive. In the 8 years before 1878 the deaths varied from 79 to 21 per 1,000, or 7.9 to 2.1 per cent. In the following 6 years to

1883, when carbolic acid was used as an antiseptic, the mortality fell to 9 per 1,000, or 0.90 per cent.; and after 1884, when the bichloride was introduced, the mortality has gradually fallen until it is less than 1 in 1,000; in 1887, but one death occurred among 1,231 births. Dr. Vincent informed Sir Spencer Wells that the bichloride solution of the strength only of 1 to 2 to 4,000, no harm has in any case been traced to the mercury. The average mortality in the Maternity Hospital of New York, between 1875 and 1883—*i. e.*, prior to the introduction of strict antisepsis with bichloride of mercury, was 4.7 per cent., or 146 deaths in 3,504 deliveries; from 1884 to 1888 inclusive, there were 2,271 deliveries with 24 deaths—a total mortality of 1.06 per cent., and that from sepsis was only 0.27 per cent., and Dr. Garrigues,[12] who deserves special credit for his devotion to antiseptic midwifery, informs us that the change in regard to morbidity is no less remarkable; whilst formerly nearly 1 out of 4 women delivered was seriously sick, and 1 in 5 from puerperal inflammations; since the change there have been very few sick puerperal, and, with few exceptions, the cases have been very mild, and he says: "We have every year had cases, which, from all symptoms, such as pain, tenderness, and swelling, had to be diagnosticated as cellulitis, and still the thermometer showed no rise in temperature, a phenomenon which I can only account for by supposing that the inflammation was of purely traumatic origin, due to the bruising of the genital canal by the passage of the child, and that the aseptic way in which labor is conducted, as well as the precautions we take during the lying-in period, excludes all infectious germs, which develop so easily in bruised tissues."[12]

1819 *Q Street N. W.*

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