CLINICAL AND PATHOLOGICAL NOTES ON PUERPERAL INFECTION *

(A Lantern Demonstration)

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I shall talk to you very simply tonight, and the bulk of my talk will consist in the demonstration of a number of lantern slides which illustrate certain factors and phases in puerperal infection. After I finish the demonstration, I shall say a few words concerning puerperal infection in general, when Dr. Polak will probably dwell upon certain points which I have omitted.

(Demonstration) This slide represents a section through the uterus of a woman who died from hemolytic streptococcus infection following a criminal abortion. The reason I show it to you is to demonstrate that the lesions in the interior of the uterus are extraordinarily slight. Here you see the muscle and here the thin decidua. The latter is perfectly smooth, so that it is apparent that nothing could have been scraped away by a curette; so that the only effect of such a procedure would have been to spread the infection.

(Demonstration) Here is a section from the uterus of a woman who died from beta hemolytic streptococcus infection following fullterm labor. Death occurred from general peritonitis nine days after delivery. Here again you see that the lining of the uterus is very little thickened, and all that is visible under the low power is a narrow zone of leucocytic infiltration; and yet from this little area of infection streptococci made their way through the lym-

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phatics and uterine wall and gave rise to the general peritonitis which caused death.

(Demonstration) This represents a section from the same uterus, much more highly magnified; and here you see that the zone of leucocytic infiltration is made up of a number of layers of polymorphonuclear leucocytes. Toward the uterine cavity is a thin layer of necrotic tissue containing streptococci. The protective leucocytic wall was not sufficient to prevent their invasion, and they passed through the uterine wall, giving rise to the peritonitis from which the patient died. I think that it is extraordinarily important to consider this slide carefully and to realize how slight the local lesion may be in certain fatal cases.

(Demonstration) Here is a section from the uterus of another woman who died following a criminal abortion. This section represents the interior of the uterus, the upper part representing the decidua and the lower, uterine muscle. You will note that the entire decidua is infiltrated with leucocytes, and these can be traced as strands of cells through the entire muscularis. In this instance, the process extended through the entire thickness of the uterine wall and gave rise to the general peritonitis.

(Demonstration) This section represents the muscular wall of the uterus of the same patient. Note the muscle fibres and the veins and the spaces between them, and you will see that the spaces which represent the lymphatics are filled with round cells and leucocytes. In properly stained sections these can be traced from the interior of the uterus to the peritoneum and show chains of streptococci making their way to the peritoneum, where they gave rise to the fatal peritonitis from which the patient died.

(Demonstration) Here is a section of a great deal of interest because it gives us a clue as to how an infectious process is transmitted from the uterine cavity to the peritoneum. This section shows the interstitial portion of the fallopian tube just after leaving the uterine cavity, and you will notice that it is perfectly normal, shows no signs

of inflammation, and there is nothing about it to indicate that the peritoneal infection had resulted from the transmission of infectious material to the peritoneal cavity by means of the tube. On the other hand, if we go back to the specimen which I just showed you, it is clear that the infection had traversed the uterine muscles and reached the peritoneum by way of the lymphatics. Too much stress cannot be laid upon this point. The great majority of writers will tell you that in many cases the infection is conveyed from the uterus to the peritoneum by means of the tubes; yet in the many autopsies which I have seen upon women dead from puerperal infection, I have never seen one in which there was any evidence that infection had occurred in this manner.

(Demonstration) This section which was obtained from a woman dying from puerperal peritonitis shows that while the tube offers signs of inflammation, it has not been the means by which the infection had transmitted. In this case, the interior of the folds of the tubal mucosa was filled with leucocytes, round cells and streptococci, and yet the lumen was free from infectious material. In this instance, the involvement of the tube was not the result of direct extension from the uterine cavity, but was due to the extension of the process through the lymphatics; and this again proves the correctness of what I have just said.

(Demonstration) This is a section from the uterine wall of one of the cases we have already demonstrated, and in it we see chains of streptococci which are making their way through the lymphatics and the uterine wall to the peritoneum. I wish to make it perfectly clear that the extension occurred in this way, and not through the lumen of the tube. When you think of the large number of streptococci which are visible, and recall that they only constitute a fraction of those actually present, it becomes clearly apparent that any attempt at intrauterine treatment would have been of no avail.

(Demonstration) I shall now present a section of a

great deal of interest, illustrating a protective mechanism, which develops in the uterine wall, against the occurrence and spread of infection. Several years ago, my associate Hofbauer said to me that he had an idea that there must be some mechanism in the lateral part of the uterine wall and the inner margin of the broad ligaments which played a part in preventing the occurrence of infection and in lessening its spread; for, if this were not the case, serious infections must occur more frequently than they do, when we take into consideration the defective technique employed by so many obstetricians. With this in mind, he studied microscopically all of the pregnant uteri which we had in our museum; and after about a year's work, he felt that he had discovered such a protective mechanism, and pointed out that it consisted in a process of rejuvenation of the connective tissue in those localities in which large numbers of peculiar connective cells were present—clasmatocytes. He pointed out that in the non-pregnant uterus the connective tissue is much more dense and fibrous; but that under the influence of pregnancy it became much looser in structure and more juvenile in type. Furthermore, that when infection occurred, the number of clasmatocytes rapidly increased and they could be seen ingesting the offending organisms. He held that this constituted an important protective mechanism which is always present, and that when the infecting organisms are not virulent, it usually suffices to arrest the process; but that when they are virulent it is unequal to the task and extension of the infection occurs. Since then, Stieve has pointed out essentially the same thing. In the section before you, you see isolated muscle cells and between them the loose connective tissue scattered through which are numerous large cells with a small nucleus and vacuolated protoplasm. The latter are the clasmatocytes which Dr. Hofbauer believes, and I agree with him, play so marked a part in preventing infection in the first place, and in limiting its extension when it occurs.

(Demonstration) As you know, when women die from infection, death may occur in a number of ways. There

are, however, three great groups: first, general peritonitis; second, thrombophlebitis; third, generalized infection or sepsis foudroyant. We have already dealt with the first group, and I have shown you how peritonitis occurs, and when the outcome is to be fatal, death usually occurs within ten days after delivery. On the other hand, in women suffering from thrombophlebitis, symptoms do not occur until the end of the first or the beginning of the second week, and when death occurs, it is not until after the expiration of six or more weeks. In cases of this kind, we find that the process originates at the placental site and spreads outward through the veins, being marked by the development of thrombosis. This process extends beyond the uterus into the veins and the broad ligaments and thence into the common iliac and ovarian veins; and in advanced processes may even involve the vena cava. In my experience, this is the second most common cause of death.

The third cause of death is generalized infection, or sepsis foudroyant. In such cases, the woman is overwhelmed by the access of large numbers of virulent streptococci to her blood stream and these form toxins before local lesions become manifest. At the present time, this constitutes a rare occurrence; but in times past, it was very frequent. At autopsy in such cases, no gross lesions are found; but when cultures are made, we find that the blood stream is loaded with virulent streptococci, and in such cases death often occurs within eighteen to thirty-six hours after delivery.

(Demonstration) The section which I am showing was obtained from a woman dying from the thrombophlebitic type of infection, and inspection shows that the vessels of the uterine wall are filled with thrombi; and it is from the extension of this process that the fatal issue occurs. As I have already told you, death in such cases does not occur until many weeks after delivery and then is the result of pyemic processes in other parts of the body, and here is a section from the uterus of a patient who died ten weeks after the original infection; and yet the endome-

trium is perfectly normal and shows no signs of inflammatory reaction. How do we account for this? Simply that the portal of entry for the infection had undergone resolution; and yet the process beyond the endometrium was sufficient to kill the woman. Here in the section we see a vein in the uterine muscle which is partially filled with thrombi, and it is evident that from it the process had extended outward and given rise to the condition from which the woman died. It is apparent that in this case local treatment would have been without avail, as all that the curette could have removed would have been normal endometrial tissue.

(Demonstration) This section represents another portion from the same uterine wall and shows a vein which is completely thrombosed. In this instance, it would appear that the thrombosis is doing no particular harm and it will continue to be inert until it undergoes liquefaction and its contents pass into the general circulation. I shall come back to this a little later; but in the meantime, bear in mind that in thrombophlebitis, when we see the patient late, all trace of infection may have disappeared from the uterine cavity so that there is nothing there to treat; but out in the wall of the uterus are the thrombosed vessels from which the process may extend.

(Demonstration) Here is another very interesting form of infection. This slide represents the half of a uterus which was obtained under the following circumstances. A colored woman with a markedly scoliorhachitic pelvis so greatly contracted that the only possible means of delivery was by Cæsarean section, had been in labor in her home for six days after rupture of the membranes, and came into my hands with a high temperature, a rapid pulse and apparently a general peritonitis. The child was dead, but even in spite of that, we did a Cæsarean section and removed the uterus unopened, as the easiest way of coping with the situation, although we did not feel that she had more than one chance in five; and she died the next day from generalized peritonitis which was

present at the time of operation. After hardening in formalin, the uterus was opened and the child removed, when we found that its entire interior had been converted into an abscess cavity. Except at the placental site, the entire interior of the uterus had been denuded of its membranes and decidua, and scattered through it, as shown in the slide, were deep ulcerated areas going down into the muscularis. In other words, the patient had a beta hemolytic intrapartum infection which spread to the peritoneum while the child was still in utero, and I am showing you the specimen to indicate how useless any form of treatment would have been which was undertaken with the idea that it would cure the patient.

(Demonstration) I shall now show several sections from a very interesting case. Several years ago, a patient whose membranes ruptured before she entered the service was admitted with fever. The following day she gave birth to a premature stillborn child, without a vaginal examination. The day after delivery, jaundice appeared and aroused the suspicion that we might have to deal with acute yellow atrophy of the liver. Chemical examination of the blood rather confirmed that opinion. There was a marked increase in the non-protein nitrogen, a definite increase in sugar, and large amounts of leucin and tyrosin in the urine. Death occurred within twenty-four hours, without any attempt at local treatment on our part, and I shall never forget the impression the autopsy made upon me. As soon as the abdomen was opened, it was apparent that she was suffering from a general peritonitis, and a peculiar crackling sound was heard as of a child blowing soap bubbles. On closer observation, it was found that this was due to bubbles oozing out from the surface of the uterus; and to make a long story short, she had a generalized gas bacillus infection, while the clinical diagnosis had been acute yellow atrophy. I am showing you a section of this uterus under an ordinary high power and not under an oil immersion lens, and yet you see myriads of gas bacilli lying between the muscle fibres. The next slide shows this in an even more striking way; and on looking

at it carefully, we find that it presents a honey-comb appearance due to the disassociation of the muscle fibres by gas cysts, and again all through it are numerous gas bacilli.

(Demonstration) The following slide is from a portion from the liver of the same patient, and here you see that the central vein is almost entirely clogged with the same organism; and it is apparent from the study of these sections that nothing that could have been done would have saved the patient's life; and that any attempt at treatment would have only led to self-deception.

(Demonstration) This slide is not a microscopic section, but represents a horizontal section through a three weeks' puerperal uterus. The patient in question had a normal spontaneous delivery followed by mild infection. After ten days or so the temperature course became hectic in character, rising to 103°, 104° and 105° in the evening and dropping to normal or subnormal in the morning. We examined her repeatedly in the expectation that we might demonstrate thrombosed veins, but we could not do so. In view of the clinical history, as well as of the fact that her uterus was about twice as large as it should be for that stage of the puerperium, I made a tentative diagnosis of multiple abscesses of the uterine wall and opened the abdomen and removed the uterus. Prompt recovery followed. The slide shows that the uterine wall is studded by a number of small abscesses, and closer study shows that they were due to a breaking down of thrombi. Streptococci were found in large numbers. They had not given rise to the peritonitis but merely to the local process which yielded to surgical treatment.

I have shown you these specimens with a definite purpose, and that is, to impress upon you that in severe puerperal infection there is almost nothing you can do; that medication does no good, as far as I know the various serums are useless, and only occasionally, as I shall point out in a moment, is surgical treatment of any avail; and consequently, the best results are obtained by those who

do the least. Indeed, I am fond of saying to my students that in such cases prayer is the best treatment, and the better prayer a doctor is, the better his infected patients do. At the same time, it must be remembered that there is a great number of cases, of which I have shown you a number of examples, that will not recover even under prayer; but it is my experience that in the less serious cases the more radical the treatment the higher the mortality. This is a very important point to get into your minds, and the first uterus I showed you is a good illustration of the fact that had it been curetted, nothing would have been brought away, and all you would have done would have been to stimulate the infection and thus hasten the fatal outcome.

Likewise in the case of the woman with thrombophlebitis, I showed you that the interior of her uterus was lined by normal decidua and that the lesion lay in the thrombosed vessels out in the uterine wall and beyond; and accordingly, nothing that might be done to the interior of the uterus could do good, but might do great harm; and furthermore, it might spoil one's own mental processes, because the worst thing that a doctor can do is to fool himself. Generally speaking, it may not be bad to fool one's patients, because it is sometimes good for them; but as soon as a doctor begins to fool himself, he becomes dangerous to his patients and to the community. Again, I may state that in my experience all the mild cases of puerperal infection do very well provided, as I am fond of expressing it, we do not be-devil the patient. On the other hand, the very severe cases tend to die, and the specimens which I have shown you illustrate that they would have died no matter what was done, and probably death would occur sooner the more energetic the treatment. Consequently, I hold that in treating puerperal infection we are facing a very serious matter, and that the mild cases take good care of themselves, and that the severe cases as a rule die.

Now, if this is the case, where does surgical treatment come in? In my estimation it plays a very restricted part,

but as I shall indicate to you, it sometimes saves life. For example; once in a while when we can make a diagnosis in the early forms of thrombophlebitis, after we open the woman's abdomen and tie off the veins beyond the point of thrombosis, we sometimes effect a surprising cure; and in such cases, the woman, who had been having a hectic temperature suddenly has her temperature return to normal and makes a perfectly satisfactory recovery. This, however, is a relatively rare indication and is in great part due to the fact that our diagnostic means are faulty, for if we operate too early the intervention proves unnecessary, whereas, if we operate too late, interference is generally useless. On this account, I find an indication for such an operation only once in every few years.

The most common surgical intervention which I employ is in cases in which the infection is limited to the broad ligaments; in other words, when we have to deal with so-called parametritis, where a pelvic abscess develops and bulges over Poupart's ligament. In such cases, if we open and drain the abscess extraperitoneally, recovery nearly always ensues. It is probably in this type of case that Hofbauer's defensive mechanism plays an especially important part, and as a result of its activity limits the infection to the broad ligaments and prevents its spread to other parts. Again, as in the case of multiple abscesses of the uterine wall, which I have just demonstrated, we occasionally get an indication for surgical intervention; but in general, such intervention is rarely called for. I shall not, however, labor the point, and Dr. Polak will probably continue the discussion as to the propriety of such intervention.

To recapitulate, I may say that puerperal infection is relatively common, fortunately the great majority are mild cases which take care of themselves if they are not made worse by the ministrations of the physician; while many of the severe cases die no matter what we do. And this reinforces once more the necessity for prophylaxis upon which Dr. Kosmak has laid such stress. I shall not

discuss this question further than to say that it consists in the most meticulous observation of aseptic technique and the consideration of the various factors concerned in the production of immunity.

Dr. Kosmak has brought out a point of a great deal of importance, and that is, that we ordinarily fail to take into consideration as much as we might the individual patient; and I consider this a point of capital importance.

In my clinic, we have a material almost equally divided between whites and blacks, although the latter form only one-sixth of the population of Baltimore. You may ask why the latter have such disproportionate representation, and the answer is that the colored woman in general is in much greater need of hospital care, as in my experience she has a much harder time having her babies than the white woman; and this is due to several factors. In the first place, she has nearly four times as much contracted pelvis as the white woman, and indeed, she does everything obstetrical worse than the white woman except that she appears to have less placenta prævia. Moreover, we find that many more deaths occur in the colored patients, notwithstanding the fact that they are treated in the same clinic, in the same operating rooms, and by the same staff. In general, febrile puerperia are twice as common in them as in the whites, and when severe infection develops, it is much more fatal. For example: in the 11 cases of fatal infection for which my service has been responsible during the last few years, 3 occurred in white and 8 in black women. I cannot tell you why the colored woman is so much more prone to infection and succumbs to it more readily; but this observation may have an important bearing upon the general question of the high maternal mortality in this country, which as you know, is higher than in any other country of the world except New Zealand and Chile. I have no facts to prove it, but I have gotten the impression that the results are not always due to poor obstetrics but may be due to some peculiarity of American women themselves; and this raises the question as to

whether the product of the "melting pot" is as good medically as most persons are inclined to believe it is in other respects. This is a mere supposition, but I am not at all sure that further investigation may not indicate that the mixture of races in this country has brought about a type of woman who stands childbirth less well than the women in other countries where the racial strains are purer. With this in mind, you can see why I appreciate what Dr. Kosmak has said, and how I am hopeful that more extensive observations may bring us eventually to some more definite conclusion.

Thus far, I have talked a good deal as a pessimist and possibly have given the impression that there is nothing we can do for puerperal infection except to prevent it. I am, however, something of an optimist, and this optimism is founded in part upon observations made in my service. At the end of last year, I analyzed the causes of death in 12,000 patients admitted to my service and found that 47 of them had died from infection. This is a large number and at first glance would appear excessive; but when I came to analyze them, I found that we could eliminate a considerable number at least so far as our responsibility was concerned. In the first place, we found that 22 of the 47 women were admitted to the service with general peritonitis following criminal abortion. Nearly all of these were admitted in extremis and all died. We assume no responsibility for them because the condition existed before they came into our hands. Likewise, 7 of the 47 women succumbing were admitted postpartum and were brought to the hospital to die from an infection contracted elsewhere. Furthermore, 6 other women were sent into the service as neglected cases, and when they entered, were already profoundly infected; and cultures made before we undertook any operative intervention showed the presence of beta hemolytic streptococcus. Likewise, we can assume no responsibility for these women, as the infection was firmly established before they came into our hands. After deducting these cases, we have left 11 cases for which we must assume a more or less per-

sonal responsibility; and I want to say a few words about them.

Eleven deaths out of 12,000 admissions is, as I figure it, a little less than 1/10 of 1 per cent; in other words, a little less than 1 death per 1,000 from infection among women who came into our hands uninfected. I shall not go into them in detail except to say that in several cases the fault was clearly ours, while in others, the mode of infection was not clear. For example: one woman died after an easy spontaneous labor; she had not been examined vaginally, but when we came to inquire as to how the fatal infection occurred, we found that the man who had delivered her had handled a woman admitted suffering from the results of criminal abortion, and who later died from a hemolytic streptococcus infection. Although no vaginal examination was made, it would appear fair to assume that in this case the infection should be attributed to us. In another instance, the responsibility for the infection was not so clear. In this instance, the patient was admitted to the service with fever and signs of tonsillitis and otitis media before she fell into labor. Twenty-four hours later she had an easy spontaneous labor during which no vaginal examinations were made. There was no perineal tear, yet two hours afterwards her temperature was 104.6 and a blood culture taken at that time showed the presence of hemolytic streptococcus. She died on the eighth day with general peritonitis, and at autopsy the same organism was isolated. I am inclined to believe that in her case the fatal infection was a metastatic one, that it originated from her throat infection and was not due to anything that we did or failed to do. If this supposition is correct, it is apparent that death from similar causes cannot be prevented.

After making all allowances, it would appear that even in a well regulated lying-in hospital a small amount of fatal infection is inevitable, which ordinarily should not exceed 1 case in 1,000. Some of these deaths are unavoidable, as in the case of metastatic infection just mentioned.

while others are due to errors in technique, which might be avoided. At the same time, I do not wish to be understood as belittling the occurrence of infection in lying-in hospitals, as it is only by the utmost attention to the details of technique that we can expect to get and maintain good results; but in the present state of our knowledge we are extraordinarily impotent in the presence of actual infection. Finally, the great majority of infections are mild and do little harm except to increase the length of the patient's stay in the hospital. On the other hand, a certain number are severe, and in those we are able to do very little more than our predecessors, except in a small number of cases in which radical surgical intervention seems indicated.