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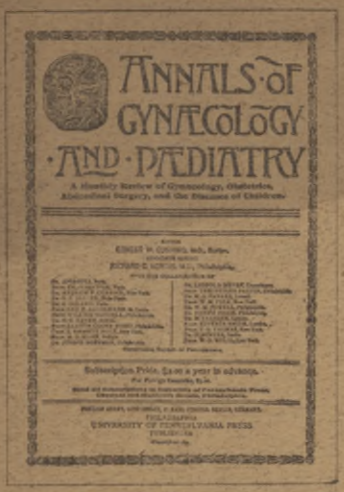

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UTERINE THROMBOSIS FOLLOWING POST-PARTUM HÆMORRHAGE, AND ITS RELATION TO PUERPERAL INFECTION.

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Uterine Thrombosis Following Post-Partum Hæmorrhage, and Its Relation to Puerperal Infection.[1]

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POST-PARTUM hæmorrhage is followed by a series of consequences dependent upon thrombosis. The most conspicuous of these are phlegmasia alba dolens, and pyæmic infection, although a general infection dependent upon the same cause is to be observed, as demonstrated by the histological study of the subject. In normal involution the contraction and retraction of the uterine muscle is sufficient to prevent bleeding from the sinuses, by causing an approximation of the vessel walls. In the absence of normal uterine contractions dependent upon want of muscular development, or upon loss of blood, as in placenta prævia, or upon over-distention from twins, or hydramnios, hæmorrhage is prevented by the formation of thrombi. On the part of the blood itself the increase of fibrin, consequent to the loss of blood, is an important factor in thrombosis. This natural means for controlling hæmorrhage approaches a pathological condition, in that it admits of an extension of the thrombi into the veins surrounding the uterus, namely, those of the parametrium and broad ligaments. In this way a direct communication between the endometrium and pelvic veins is set up.

In active involution the blood current is diminished, and the absorptive power of the veins and lymphatics is decreased, whereas in defective involution the amount of blood in the uterus is increased, and the lymphatic circulation called more actively into play. According to Winckel (1) the outcome of physiological thrombosis is described as a conversion of the thrombi, by the immigration of wandering cells, probably with the aid of the endothelium and vasa-vasorum, into a firm connective tissue cord, which at times becomes canaliculated, possibly by the passage of red blood corpuscles, so that the blood current is restored. Thus, under the conditions in which uterine inertia exists, we have hæmorrhage giving rise to increased tendency to inertia, and to the formation of thrombi, which serve as a dangerous means of communica-

[1] Read before the Philadelphia Obstetrical Society, May, 1893.

tion with the central venous circulation, and as a stimulation of the lymphatics surrounding them. Where elements of infection are absent the thrombi shut off the uterine cavity from the circulation, but where septic material is present they offer, when once affected by the putrefactive changes about them, a means of entrance into the system for the micro-organisms which attack them.

Bacteriological research has shown that the normal uterine lochia contain no germs, and may be injected into the body of any animal in any amount without injury. Doederlein (2) found that after a normal labor with a temperature not exceeding 98.4° there were no germs, but when fever was present, bacilli and cocci were found until the temperature fell, being eliminated by the very abundant secretion, especially when this was purulent. Micro-organisms may find entrance into the uterine cavity by various means, and when in contact with the endometrium give rise to infection. It has been positively shown that the endometrium is the usual source of infection, for in puerperal ulcers of the vagina we have only a mild form of infection accompanying the local signs, and although the same micro-organisms are present as those which are found within the uterus in puerperal endometritis they occur only at the seat of infection, and are not found penetrating into the neighboring tissues (3).

Having, therefore, a case of hæmorrhage with the occurrence of dilatation thrombosis, and the presence of septic material, we have the liability of infection, the process attacking the endometrium, and spreading by means of the disorganization of the thrombi along the course of the veins, especially at the placental site, and invading the general circulation.

It will be of interest to study the means by which infecting germs find entrance into the uterus. These may be present before the occurrence of labor in cases in which hæmorrhage is likely to occur, their presence and the liability to hæmorrhage being dependent upon the same cause. Namely, in cases of endometritis we have, as has been so forcibly maintained by Pozzi, the presence of pathogenic organisms, the prevailing species being staphylococci (pyogenes aureus, albus and citreus), and various kinds of streptococci. As to the part played by the uterus in cases of hæmorrhage due to metritis and endometritis with the presence of the usual pathogenic organisms, Winckel states that a limited metritis, or premature fatty degeneration of the muscle of the pregnant uterus is likely to interfere with the contractile power of the affected area. Endometritis having existed during pregnancy, and present at the time when uterine contraction and retraction are essential to the arrest of hæmorrhage, predisposes to bleeding; first, on account of the hyperæmia; secondly, by reason of erosion of already occluded vessels from the presence of mycotic elements; and thirdly, by interference with involution. The question arises, what determines the presence of micro-organisms within the uterus in endometritis, and why, if in any such case their existence is proven, should puerperal endometritis and its consequences be the exception rather than the rule? The answer to this lies in the fact that the tissues of the genital tract possess, under normal con-

ditions, a power of resistance to the pathogenic action of the germs which may be present. The vitality of these germs becomes more and more attenuated as they are acted upon by the normal secretions and cellular elements of the tissues. This antagonism of the tissues against the invasion of pathogenic germs continues as pregnancy advances, up to the time of the beginning of labor. The completion of labor, marked by the expulsion of the placenta and discharge of liquor amnii, affords the natural means by which the genital tract is flushed out, and the possibility of the lodgment of germs is prevented. In pathological conditions, on the other hand, that is, in simple endometritis, in contra-distinction to puerperal endometritis, the mucous membrane becomes infected by the invasion of germs which are indigenous to the genital tract. According to Pozzi (4) there exists in the genital tract of the female a zone rich in micro-organisms, situated at the level of the internal os. The activity of this zone is increased by the general debility of all the tissues, which reduces cellular vitality, or by traumatism. In endometritis the mucous membrane becomes infected from this source, and the ordinary lesions and symptoms follow. In puerperal endometritis, in cases where hetero-infection can be excluded, we may ascribe the condition to an ante-partum infection dependent upon an earlier endometritis, the earlier pathological changes in the uterine mucosa and connective tissue predisposing to hæmorrhage, by interfering with contractions, with infection of the resulting thrombi by the germs which are already present. In cases of atony from other causes (want of muscular development, over-distention) the treatment which is used to avert the hæmorrhage, and the necessary manipulations, may be responsible for the infection. Frequent examinations during the course of labor, hasty and careless manipulations at the time when the patient is bleeding, and carelessness of the principles of asepsis owing to the loss of self-control on the part of the attendant, and the introduction of infected instruments, all contribute to the risk of infection. There exist, therefore, under these circumstances, ample opportunities for the invasion of bacteria.

What, on the other hand, are the natural means of resisting these bacteria in cases of non-infection, and what are the local changes in septic cases, resulting from the action of micro-organisms? Immediately after the expulsion of the placenta the uterus contracts and obliterates the cavity recently occupied by the ovum. This contraction is influenced largely by the nervous condition of the woman, and may be considered as an active process. Under normal circumstances the innervation of the organ produces the active power of contraction irrespective either of the elasticity of the fibres or of the diminution due to shortening of the fibres by retraction. The blood supply is lessened by this contraction, and the vessels at the placental site are compressed by the uterine fibres and emptied of blood. Both the free contents of the uterus, namely, blood and the remaining amniotic liquid, and the adherent shreds of decidua are expelled. As soon as the tonic contractile power of the uterus is estab-

lished, retraction of the muscle (fatty degeneration of the muscular fibres) and regeneration of the mucous membrane take place. Together with the lessening in size of the uterus by retraction, there is an increase in the development of intraglandular tissue and a reconstruction of the mucosa from the epithelium springing from the remaining glands. The exudation which accompanies this process, together with the migration of white corpuscles and the secretion from the cervical canal, constitute the lochial discharge. As to the local changes occurring in the course of infection, we have these normal processes modified as follows: First, as a predisposing cause of infection we have the absence of uterine contraction. As a result, the hæmorrhage from the sinuses is controlled not by pressure, but by thrombosis; secondly, the uterus contains, also incident to the absence of contraction, remnants of decidua or placental débris, these, together with the thrombi projecting from the placental site, act as foreign bodies, and are prone to putrefactive changes; thirdly, the reconstruction of the mucous membrane is replaced by necrosis of the epithelium and basement membrane; fourthly, the normal constituents of the lochial discharge are replaced by the putrefactive débris of disorganized thrombi, the remnants of decidua and necrotic mucous membrane, mixed with the various micro-organisms which accompany these putrefactive changes. In order to appreciate the relation of such changes to the devolopment of infection, it will be necessary at this point to study the histology of puerperal endometritis.

According to Bumm we have commonly to deal with the following forms:

Putrid Endometritis.—In this form putrefaction occurs from the presence of saprophytic organisms. The bacteriology of this condition is still undeveloped; as to the histology, we find that the necrotic decidua is cut off by a zone of cellular infiltration, by which the various micro-organisms present are prevented from penetrating into the underlying tissues. Invasion of the thrombi, however, at the placental site, is not prevented by any such zone of reaction on account of the want of organization of the thrombotic tissue.

Septic Endometritis, occurring in two forms.—First, a localized septic process in which a granulation zone occurs (~~contrary to what is~~ found in the form mentioned above) shutting off the necrotic endometrium and preventing the invasion of germs. The uterine lymphatics are not actively involved. The placental site, as in the putrid form, is most markedly affected. Secondly, a septic endometritis, accompanied by a general infection. Bumm has studied five cases belonging to this class, and has found in three instances that infection has occurred by invasion through the lymphatic system, and in two instances along the course of the veins. In the first set of cases the placental site is free from micro-organisms and thrombi, so that it is not likely that this pathological condition bears upon that form of infection resulting from hæmorrhage in which thrombi, especially at the placental site, occur. In the second set of cases the smaller lymphatic branches surrounding the sinuses are marked by colonies of cocci, which extend into larger lymphatics underlying the peritoneal covering of the uterus. The decidua is disorganized and in-

filtrated with a fibrinous exudate, presenting a diphtheritic appearance. In this class of cases, as well as in that about to be described, the granulation zone is absent. This fact has evidently an important bearing upon the function of such a zone of demarkation, in combating the progress of micro-organisms into the underlying tissue.

Thirdly, a *thrombotic form of infection;* and this is the form which concerns us principally in the discussion of post-hæmorrhagic infection. This is characterized by both a putrid and septic endometritis. It is described by Bumm as follows:

"The decidual layer of the uterine cavity, in a state of necrosis, is beset with micro-organisms. In the neighborhood of the colonies of streptococci, outlined by the staining process, are scattered innumerable colonies of putrefactive germs. The histological relation of the tissues, that is, the decidual, glandular and muscular tissues, in the necrotic area, is unrecognizable."

The zone of reaction is marked. The placental site presents no remains of the placenta, but is marked by the projection of thrombi. The latter are found to be infected by various pathogenic germs, are disorganized, and offer, by reason of their disorganization, a direct means of entrance for the septic products into the current of the blood. The disorganization occurs first in the axis of the thrombi. The endothelium and the vessel wall become rapidly affected and break down into a mass of necrotic tissue mixed with white corpuscles and infected with cocci and bacilli.

In conclusion, we may summarize the development of infection as a result of thrombosis by noting the following events: First, a predisposition to infection arising in cases of hæmorrhage the result of atony of the placental site; secondly, the formation of thrombi which offer, on account of their want of vital organization, an improper means of resistance to infecting germs; and thirdly, the presence of infecting material either from the pre-existing endometritis or from contamination at the time of delivery by careless or frequent examinations. When these factors are present we have a resulting infection occurring in accordance with the histological changes described above.

Clinically, we are apt to consider pyæmia as the type of infection occurring as a result of thrombosis. Such a view is based upon—first, the frequency of the occurrence of phlegmasia following phlebitis, either by extension from the veins of the broad ligament or by the lodgment of coagula washed from the placental site and carried into the hypogastric veins and obstructing the flow of blood through the crural veins; and secondly, upon the occurrence of embolism from the detachment of thrombi from the placental site or the parametrium. But it is more likely, from the histology of endometritis in the puerperal state, that the thrombi act more as a channel by which pathogenic germs find entrance into the organism than as a direct means of conveyance by their detachment and circulation in the blood current. We have observed in the thrombotic form of endometritis that the disorganization of the thrombi is a pathological change dependent upon the action of bacteria, and that the natural barrier to the entrance of infecting elements is removed by this process of disor-

ganization. According to this, the blood current is likely to be contaminated, not by the remnants of uterine coagula, but by the presence of pathogenic bacteria and their chemical products. These, carried along in the blood current, may be reasonably supposed to set up inflammatory changes, causing phlebitis, and, especially, to produce the development of a general septicæmia. There is no doubt that the formation of emboli is a common result of the detachment of thrombi from the placental site, but in the study of the subject from a histological point we are not warranted in accepting the occurrence of pyæmia as the universal clinical associate of thrombotic infection the result of hæmorrhage, and, on the other hand, we *are* warranted in assuming the possibility of a marked state of septic endometritis occurring after hæmorrhage without the early association of pyæmic symptoms.

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(2) Doederlein: Centr. f. Gynæk., 1888, Nos. 23 and 28.

(3) Bumm: Archiv. f. Gyn., Bd. XXIII, p. 237.

(4) Pozzi: Medical and Surgical Gynæcology, translated by Brooks H. Wells, M.D., 1891.

DISCUSSION.

DR. EDWARD P. DAVIS:

As a clinical illustration of some of the points made by Dr. Wilson may be mentioned the not infrequent occurrence of hæmorrhage in syphilitic women after labor. When such hæmorrhage occurs septic processes are apt to follow unless special care is taken. We recall such a case recently. A woman with syphilis had hæmorrhage after labor. Following this a septic process developed, with dissemination of micro-organisms through the body. I feared a tubal abscess and opened the abdomen, but found nothing warranting removal of the tubes. They were allowed to remain and the abdomen closed. There then occurred multiple thrombosis of one lower extremity, thrombosis of the popliteal vein and some of the smaller veins. The patient recovered under careful nursing, and illustrated well the clinical course of a case complicated as described by Dr. Wilson.

In regard to treatment of hæmorrhage, the use of the intra-uterine iodoform gauze tampon is based upon the fact that after hæmorrhage the thrombi are apt to become infected; the use of the antiseptic or aseptic gauze is an attempt to maintain an aseptic condition of the thrombi at the placental site for twenty-four or thirty-six hours after delivery.

DR. T. RIDGWAY BARKER:

I hoped that Dr. Wilson, in treating of this subject, would add something to our knowledge of the causes of thrombosis, as this disease is one in which I am much interested. I do not think that there is any doubt that thrombosis of the uterine veins is due in a large measure to a hyperinotic condition of the blood incident to pregnancy.

While the exact cause is often doubtful, the predisposing are unquestionably, first, early rupture of the membranes leading to premature separation of the placenta, with the formation of soft coagula in the sinuses; second, sudden fall of blood pressure, usually the result of hæmorrhage; and third, uterine inertia.

The question arises, how shall we combat by prophylactic measures these predisposing causes? We always have associated with gestation an increase of the fibrin in the blood, and it becomes anæmic in character.

By sunlight, fresh air, and exercise we may counteract this tendency to deterioration.

With reference to premature separation of the placenta we have only to delay rupture of the membranes, while in the case of uterine inertia a timely application of the forceps will usually prevent its occurrence.

I would say in conclusion with regard to the avoidance of puerperal infection of the thrombosis, that I believe ergot administered after labor is a very important measure, given either hypodermatically or in the form of tablet triturate by the mouth.

It tends to produce firm tonic uterine contraction and retraction, and insures obliteration of the uterine sinuses, and, moreover, causes complete expulsion of all debris and clots, which otherwise would remain within the cavity.

DR. J. PRICE:

I may say a word on the practical side of this subject. Some of you may have read the interesting researches in regard to the presence of micro-organisms in the uterus, of Dr. La Place, whose investigations have been republished in the *British Gynæcological Journal.* Speaking purely from the clinical side of the question, I would state that my experience has been rather peculiar, and differs somewhat from that of others. The Germans have condemned the expression method of delivering the placenta, and I believe that it has been condemned by a few teachers in this country. In my own experience I value the expression method above all others. With me it begins with the delivery of the child. When the head is delivered I anticipate delivery of the shoulders, and place my left hand on the uterus, and follow the extremities. My practice is that of early delivery of the placenta, and securing early firm contraction and retraction. This I hold on to for some time, not permitting the relaxation that so commonly follows slow or delayed delivery of the placenta. I have no recollection in the Retreat work of a single bubble of air or clot that you find in late delivery of the placenta. You are called to deliver the placenta because some one has failed. You find the uterus large and relaxed, and it takes some time to stimulate a contraction. If you succeed, you succeed in delivering the placenta without introducing the hand, but with it you have a great quantity of clot, and a great blubber of air.

I am satisfied that tardy delivery of the placenta, and the slow contraction and retraction of the uterus, is at the bottom of much mischief that the reader has dwelt upon. At the Retreat I have no knowledge of a post-partum hæmorrhage in 1300 cases. I have not had a post-partum hæmorrhage for ten years in my own work. I find the practice of early delivery of the placenta, and following the uterus down, very valuable. It makes me very comfortable to leave a woman after such a practice. Following this practice the statistics of the Retreat strongly fortify its value—1300 deliveries without a death from any cause.

DR. RICHARD C. NORRIS:

Dr. Wilson's remarks are of great scientific interest and value, but the clinical aspect of this question is of especial interest to me. It is an interesting problem to practical obstetricians to arrive at any conclusion as to when infection of the uterine thrombus takes place. We know that it is the custom with many when elevation of temperature occurs to begin local disinfection, and some would resort to the use of the curette. There have been a few cases in which after this curetting prompt elevation of temperature has occurred. When I find this elevation of temperature I fear the possibility of infection of the thrombi, and that the curette may have done harm. It breaks up the clots, and, perhaps, aids in the absorption of more septic material. In one case this symptom of secondary rise of temperature occurred, and not long afterward septic pneumonia appeared, probably from embolism. I believe that in the absence of local signs of disease of the tubes, ovaries, or cellular tissue, and where we curette the uterus and find debris, and this is followed by a prompt rise in temperature, we should look upon the case as one of infection of the thrombi in the uterus, and desist from further use of the curette.

DR. W. REYNOLDS WILSON:

I think that perhaps the most important bearing of this subject is as to the existence of pathogenic germs within the uterus—the existence of these germs even before the chance of distribution and infection following labor. I think, as I said in the paper, that the researches mentioned by Pozzi as to the cause of endometritis and the general infecting inflammatory troubles of the uterus are of especial interest at this time. I think that in obstetrical and gynæcological work we are coming to a time when this subject should be looked into.

The remarks of Dr. Norris are very practical. We find general rapid infection occurring principally through the lymphatics. In the case mentioned by Dr. Norris, it is probable that there was beginning a general infection through the lymphatic system, and that the disturbance of the thrombi added a local septic process to the general infection already developed.