SEPTIC PELVIC THROMBOPHLEBITIS.

BY
K. I. SANES, M. D.,
Pittsburg, Pa.

THE frequency of septic pelvic thrombophlebitis and its high mortalitiy makes this subject of sufficient interest to bring it up for discussion before our society. To intelligently discuss this subject, it is necessary for us to understand the meaning, formation, etiology and progress of a thrombus. Thrombus is frequently used synonymously with clot. To understand what is meant by thrombus it is best to differentiate it from a clot, for even men like Virchow not many years ago, considered thrombus formation and blood coagulation to be identical processes(54).

Thrombus and Clot Differentiated.—A thrombus is not a clot. A clot forms in a stagnant circulation, while thrombus can form only in the current blood(54). While in clot formation the fibrin ferment is a chief factor, in thrombus formation it does not play any primary rôle at all (Aschoff and Lubarsch (54)). A clot formation is based on the entangling by fibrin of red and white blood cells while a thrombus formation is based primarily on agglutination of blood platelets,[1] the fibrin and blood cells becoming deposited in the thrombus secondarily. A clot is primarily independent of the vessel wall while a thrombus is always adherent to the wall of the vessel.

Formation of Thrombus.—Observing types of thrombi in their early stages we find them to consist almost entirely of coalesced platelets, the leucocytes, fibrin and erythrocytes (that we usually find in thrombi at the margins or between the platelet masses) appear later. It is upon the degree of the prevalence of these secondary blood elements in the thrombi that their classification into hyaline, white and red depends.

The hyaline thrombus is very small. It is found chiefly in

[1] According to Wright the most typical platelets are derived from the megacaryocytes of the bone-marrow. Arnold claims that they originate from the red blood cells.

The platelets are about 1/3 micron in size, very fragile, round or oval, have no hemoglobin, no nucleus, and no chromatin. They have a tendency to agglutinate outside the body and under the microscope they are seen in clumps between strands of fibrin. They number between 200,000 to 860,000 per cubic centimeter and are frequently mistaken for the plasmodium malariæ, especially when they chance to lie on red cells.(1)

capillaries and very small vessels. It may be composed of a mass of ordinary blood platelets, showing only here and there a leucocyte, or of platelets resembling colorless erythrocytes, or again it may consist of a number of layers, shading off one into another, beginning with a hyaline colorless layer, gradually passing into a layer of blood platelets, then changing into a layer of blood cells poor in hemoglobin.

The white thrombus is much larger in size. It is the most common and therefore the most important one. It is found in the large vessels and is composed of masses of blood platelets, separated by a close network of fibrin which may or may not hold leucocytes and erythrocytes.

The red thrombus is found in aneurysms and large veins. It begins as a platelet thrombus but the fibrin appearing later enmeshes erythrocytes in such large numbers that it give the thrombus a red appearance.

The so-called acute red thrombus occurs only in a completely arrested blood current as in a ligated vessel. The column of stagnant blood above the ligature forms a clot and only at the point of ligation of the vessel on account of injury to its wall can a collection of platelets be made out(1).

ETIOLOGY OF THROMBUS FORMATION.

Let us consider now what factors bring about the agglutination of the platelets into masses and make them adhere to the vessel wall, in other words let us consider the causative factors of thrombus formation.

Slowed Circulation.—One of the most important factors in thrombus formation is slowed circulation. While the platelets under normal conditions circulate with the corpuscles in the axial current, they appear in the outer zone when the circulation is sufficiently slowed, thus coming in contact with the vascular intima. Moderate retardation, however, may not be sufficient; it may favor accumulation of only the white corpuscles in the outer zone of the vessel, while to bring the platelets into the outer zone considerable slackening of the stream is necessary. But it must not be a complete blocking[1], for a blood current

[1] Baumgarden(54) tied a vessel off in two places and found the blood between the ligatures fluid after a week.

Schwalbe claimed to have found a blood platelet thrombus in a doubly ligated vessel but Derevenko found that the reason for Schwalbe's thrombus was the puncture in the vessel wall, which allowed some of the blood to flow out. The blood platelets accumulated at the point of puncture during the slow outflow of the blood(11).

is necessary to bring sufficient number of platelets to any given point for thrombus formation. In a completely blocked circulation this cannot occur. Of course the larger the platelet count, the greater is the number of platelets brought in contact with the vessel wall. This view is held by Eberth, Schimmelbusch, von Recklinghausen, Aschoff, Lubarsch, Ferge, Zurhelle and others. Since for thrombus formation the platelets must be brought in contact with the vessel wall (for a thrombus must be adherent to a vessel wall) retardation of blood current must be considered an important factor in thrombosis.

This characteristic ability of retarded circulation to predispose a vessel to thrombosis is more frequently seen in the venous system than in the arterial. According to Lubarsch venous thrombi are met with four times as frequently as the arterial.[1]

The explanation of this greater predisposition is easy. The slower speed of the blood current, the lower blood pressure, the absence of pulsation, the fixation of the venous wall in certain situations to the fascia and bones, the presence of valves an the flow of the venous blood from smaller into larger vessels, all these physiological pecularities of the venous system tend unquestionably to retard the blood current and thus render the venous system a more favorable locality for thrombus formation than the arterial system.

The same relation between retardation of blood current and greater tendency to thrombosis is noticed in individual vessels. Such are the vessels, the blood current of which is subject to a more than normal resistance, as the femoral vein passing over the elevated point under Poupart's ligament, such are the vessels that are under constant external pressure as the left common iliac under the pressure of the overlying arteries[2] and the pelvic vessels in general under pressure of the fetal head, such are also dilated blood channels that receive their blood from smaller vessels as the cerebral sinuses and the cardiac chambers.(27) Thus we see that wherever we find in the circulatory system physiological conditions interfering with the normal rapidity of the blood current, we find also in the same place a predisposition to thrombus formation. If this holds good in physio-

[1] Ferge, in the Freiberg Path. Institute, found 76.3 per cent. of venous thrombi to 23.7 per cent. of arterial(27).

[2] The left iliac vein as it is known, is under pressure of the right common iliac (almost at right angle), left hypogastric and median sacral arteries and according to Kistler (Thrombophlebitis of the Left Leg., *J. A. M. A.*, Aug. 10, 1912)(31) also the iliolumbar artery. The right common iliac vein is under pressure of the right common iliac artery only and then under an acute angle.

logical retardation it should be found to be true also in pathological, and it is a clinical fact, well recognized by many that in diseases inducing unfavorable cardiac changes or causing in any way retardation of blood flow, thrombosis is frequently met with. The most striking example of ths is the uterine fibroid, a disease accompanied with unfavorable heart changes[1] and strikingly predisposed to thrombosis. Klein in 12,923 laparotomies found 270 thrombi, Black in 3000 cases, thirty-five thrombi, Mayos in 1788 cases, eighteen thrombi and Herff in 2679 cases eighty-one thrombi or in a total of 20,440 cases, 404 thrombi or 1.9 per cent. After laparotomies for myoma it was found as follows: Zurhelle 2.5 per cent., Fritsch 2.75 per cent., Kelly and Cullen 3 per cent., Werthelm 3.3 per cent., Bouchardt 4.6 per cent., Albanus 4.6 per cent., and Wenczel 5 per cent(27).

Interesting clinical observations have been made by reliable authorities which may be utilized here to confirm the relationship existing between retardation of the blood current and thrombosis. Thus, a long rest in bed weakening the heart muscle and thus slowing the circulation(22) is found according to the observations of Hoffman and Kummel to favor thrombosis(24). Trendelenburg's position with knees strongly bent over the table, slowing, by pressure on the popliteal veins, the circulation in the lower extremities, predisposes to thrombosis according to Olshausen and Zweifel(23).[2] Pressure with abdominal retractors over the branches of the epigastric vein may cause in them thrombosis according to the observations of Clark.[3] Prolonged mental depression weakening the heart force is claimed by some to predispose to thrombosis. Ether narcosis producing secondary heart weakness (Reidell) favors thrombosis the first day after anesthesia according to the observations of Lennander.

Changes in Blood-vessels.—The mere slowing of circulation will not suffice to cause thrombi, for it only brings the platelets into the outer zone of vessels, thus allowing them to come in contact with the endothelium(10). But to get a thrombus we must have an agglutinated mass of platelets adherent to a vessel wall. This can occur only in the presence of a diseased intima of the vessel wall.

[1] It is generally admitted that there is a clear relationship between myoma and fatty degeneration of the heart. Strassman and Lehman found anatomical lesions of the heart in 40.8 per cent. of their myoma cases(27).

[2] Zweifel had no emboli in 1600 laparotomies done in the horizontal position nor in vaginal panhysterectomies, while he had twenty-one cases of embolism in laparotomies in the Trendelenburg position(23).

[3] In a case laparotomized by Clark a second time, three months after the first operation the epigastric vein was found thrombosed(23).

It is universally recognized that the normally smooth[1] endothelium of the vascular wall has a great influence in maintaining the fluid state of the blood, but an abnormality in the vascular endothelium may induce the viscous metamorphosis of the platelets coming in contact with it and permit a platelet mass thus formed to adhere to the abnormal part of the vessel wall. These abnormal changes in the vessel may be extensive, when induced by injury, inflammation, degeneration, or neoplasm or it may be very slight and even microscopic, when induced by impaired nutrition of the endothelium as noticed in anemia, toxemia and chilling of blood-vessels during operations.

The relative importance of blood-vessel disturbances and retarded blood current in thrombus formation is best shown by the fact that while in arteries pathological changes are much more commonly met with than in veins(27), yet, because of the greater current force, arterial thrombi are very rare in comparison with the venous ones. In fact, Lubarsch and Zurhelle studying this question came to the conclusion that, so long as the circulation is strong and the blood cells are not injured, the changes in the vessel wall will not cause thrombus formation.

3. *Alteration in Character of Blood.*—In studying the question of the etiology of thrombus formation, the blood changes met with during the course of the disease must be considered, and here again we want to emphasize the point that the increase or the reduction of fibrin has no bearing on this question and that blood coagulation time has no constant relation to thrombus formation(54).[2] But while coagulation time is of no importance in thrombosis, the increased viscosity or viscous metamorphosis is quite generally admitted to be an important factor. Bachman(27) found that in diseases with high viscosity thrombi are frequently met with. Its relative value, however, at present is not settled.

The blood change that is universally admitted to be present in thrombophlebitis is the increase in the platelet count. While their number per centimeter even in healthy people is not uniform (from 200,000 to 860,000), yet there is a well recognized

[1] In his experiment No. 16, Guthrie of Pittsburg implanted in the course of a rabbit's carotid artery a segment of vena cava hardened with formaldehyde, dehydrated with alcohol and impregnated with paraffin, and this dead piece of vein functionated for twenty-two days with no sign of thrombosis (Adami).

[2] According to Welch, there may be very extensive bacterial inflammation of the venous wall even with bulging of the intima into the lumen without thrombosis(52).

[3] Schimmelbusch succeeded in inducing a thrombus in a dog whose blood he rendered incoagulable by injection of albumose(52).

parallelism between their numbers and the predisposition to thrombosis, *i.e.*, an increase in platelets is found in diseases predisposed to thrombosis and a decrease in diseases in which thrombosis is very rarely or never met with. As an interesting example we will cite chlorosis and pernicious anemia, the first with a high platelet count and a pronounced tendency to thrombosis (Muir), the other with a very low count and the striking absence of thrombosis (Hayem, Birch Hirschfield(54–52). In connection with our subject it is important to remember that there is an increase of platelets at the end of pregnancy, after delivery, during septic infection, in post hemorrhagic anemias and in states of bad nutrition in general [1](52).

4. *Infection.*—We see then, that for thrombus formation the necessary factors are: 1. Blood changes leading to increased number of platelets and viscous metamorphosis; 2. retarded circulation, and 3. vascular changes in the intima. There is another causative agent in thrombosis that must be considered and this is infection. According to Fromme,[2] von Jackowsky, Latzko, Veit and others, infection is the only factor in thrombosis, the factors referred to above being secondary. They cite the experiments of Fromme, von Jackowski and others to prove this view. But their view cannot be adopted by us. Their experiments were either proven to be wrongly interpreted or were entirely discredited by such authorities as Zurhelle, von Bardeleben, Rubesch, Heller [3] and others. Besides, their view cannot

[1] Other examples of the parallelism are: Increase of platelets in erysipelas, meningitis and influenza. In pneumonia the number is increased but in severe cases the number is diminished occasionally. There is also an increase in tuberculosis, various cachectic conditions and during the subsidence of, and early convalescence from infectious diseases such as typhoid fever and pneumonia. On the other hand there is a decrease or entire absence of platelets in purpura hemorrhagica (Denys, Hayem, Erlich), a decrease in malaria and in typhoid during progress of the disease.

[2] Fromme reported in 1908 experiments on rabbits in whose jugular veins he inserted sterile and infected silk threads. He found thrombi only in the ones with infected threads. A year later, Zurhelle disproved it. He, under most careful asepsis, repeated Fromme's experiments. In a large number of cases he found typical blood platelet thrombi whether he used sterile or infected silk threads(54).

Von Jackowsky tied blood-vessels of a rabbit with gum bands; twenty-four hours later he injected into these vessels bacterial cultures and toxins. He discovered afterward thrombi in vessels but the tying of vessels brought about considerable vascular engorgement, injury and alteration to blood-vessel wall. Therefore, there is no indication that the bacteria and thrombi have any cause and effect relation. It is very likely that the bacteria appeared in this case later with the blood stream into the already formed thrombi.

[3] Experiments of Heller(27) with colon bacilli injections into the blood stream and that of von Bardeleben(54) with living streptococcic injections and of Rubesch(45) with staphylococcic gave negative results, *i.e.*, did not produce septic thrombi. If Welch is correct that bacteria by themselves in the blood cannot cause changes in the vessel wall, this alone would explain the reasons for the results obtained by these careful investigators.

explain why we meet thrombosis more commonly after the hysteromyomectomy than after operations for septic adnexa, why thrombi are met with in the venous circulation more frequently than in the arterial, why the left lower extremity is more subject to thrombosis than the right one, why thrombi may develop as late as three and six weeks after an operation and why, in spite of the most careful asepsis in the hands of the best surgeons, thrombosis cannot be eliminated. Again it has been clinically proven that nonseptic diseases with low platelet count, low heart force and badly disturbed nutrition, bring about thrombosis without the aid of sepsis. Such are, for instance, the "quiet" thrombi of Bennett in legs of old people(5), the marantic thrombi[1] met with in wasting diseases, the thrombi of pregnancy before delivery, the afebrile puerperal thrombi[2] and experimental aseptic thrombi.

The presence of aseptic thrombi must, therefore, be admitted. But it should also be admitted that infection is an important factor in formation of thrombi. In fact, local infection by itself may, under favorable conditions, slow the circulation, injure the vessel wall, introduce the necessary blood changes and thus bring about thrombosis. As a rule, however, infection acts only as a contributory factor. If we have, for instance, a retarded blood current and an injured vessel, infection, by its increased blood platelet count and viscous metamorphosis, can supply the missing factor (blood changes) for thrombosis, but it should be remembered in this connection that thrombosis thus formed while usually septic, is not always so. Klein(27) in 215 autopsies on infected puerperal thrombosed cases found microorganisms only in 10 per cent. of thrombi. Here it might not be out of place to call attention to a predisposing factor of thrombosis, named by Felix Mendel(40) thrombophilia or an

[1] Marantic thrombi are met with in infectious diseases and in wasting, cachectic and anemic conditions. True, a large number of so-called marantic thrombi are found on careful examinations to be of infective origin (Vasquez demonstrated bacteria in such thrombi as far back as in 1894), but in very many instances repeated bacteriological examinations yielded negative results in such careful hands as that of Welch.(52)

[2] Bumm(11) speaks of the aseptic thrombus as follows: "There is an aseptic thrombus of the uterine veins in the puerperium that begins at the placental site and extends to the spermatic veins, pampiniform plexus, the hypogastric and iliac veins. If the uterine retraction is normal the venous sinuses are closed by simple contraction of the vessel walls, but in an atonic uterus they are closed by thrombi usually without symptoms. Aseptic thrombi are also found in varicose veins of the leg during pregnancy and the puerperium. Neu, in the Heidelberg Clinic, found seventeen out of thirty-five cases of puerperal thrombosis without fever.(27) Welch has found marantic thrombi without any visible alteration in the vascular intima at the site of thrombus.(52)

individual predisposition to thrombosis. He has noticed thrombi develop in the lower extremities and pelves of some patients from time to time after attacks of slight general infection. He attributes this predisposition to a peculiarity of blood opposite to the one found in hemophilia. His observations are purely clinical and he offers no experimental proof. Nevertheless it is worth while bearing in mind, for it may account to a great extent for some of the unexplainable puerperal and postoperative thrombi.

The Progress of a Thrombus.—A thrombus usually beginning as mural, may either remain as such or by continued growth may fill the vessel and become an occluding thrombus. If the thrombus is sterile it may go on to absorption,[1] organization,[2] canalization,[3] calcification,[4] or bland softening with or without embolism.[5]

In septic thrombi the changes depend a great deal on the virulence of the infecting organism. In mild infections the termination may not be different from that of the sterile ones.

In the severe infections, the bacteria finding a good culture medium in the thrombus, may gradually change it into a purulent mass and give rise, in bad streptococcic cases, to suppuration and even gangrene of the blood-vessel. If an embolus be

[1] A thrombus of moderate size may, through leucocytic activity, undergo resolution and absorption, thus allowing the vascular channel to be restored to its natural lumen. This, of course, is the most favorable termination of a thrombus if embolism does not occur during the resolution.

[2] Organization is a substitution of vascularized connective tissue for the thrombus. As a result of chemotactic irritation leucocytes and so-called migratory cells are attracted into the site of the adhesion. Through their activities the thrombus is slowly disintegrated and absorbed. While this absorption is going on, granulation tissue forms in the thrombus, the cells being derived from the leucocytes, from the endothelium and other fixed cells of the vessel wall, and the new vessels being derived chiefly from the vasa vasorum. This new tissue gradually contracts, converting the vessel into a small contracted mass of fibrous tissue, a fibrous cord with complete occlusion. Such organization ends the danger of thrombus.

[3] The organized thrombus does not always tend to complete occlusion of the vessel. The blood-vessel may only be narrowed at the site of the thrombus. It may present a number or channels of a cavernous structure. These cavernous septa or channels may disappear, restoring the lumen of the vessel with perhaps a few fibrous bands as seen in the normal cerebral sinuses.

[4] In certain regions of the body notably in the uterine plexuses, thrombi frequently become the seat of calcareous deposits and are converted into phleboliths.

[5] Instead of organizing into connective tissue or calcareous substances, the thrombus may soften and liquefy. This process is called also puriform softening because of the mistaken idea about its contents, which was supposed to have been pus. It results from cell digestion by the thrombotic leukocytes or by the migratory cells outside of the thrombus and consists of a brittle shell the interior contents of which are composed of necrotic fatty leukocytes, albuminous and fatty granules, blood pigment and altered red corpuscles. This unorganized condition makes the thrombus a starting-point for bland emboli, with the degree of the danger depending upon the size of the embolus and the importance of the organism to which it is carried.

detached from such a thrombus it may cause metastatic infection, the gravity of which depends on the virulence of the infecting agent, the location of the embolus, and the general resistance of the patient. So much for thrombus formation.

Now, before we take up septic thrombophlebitis, a few words must be said about phlebitis and its relation to thrombosis. A thrombus may exist without a phlebitis, for as referred to above, vascular changes caused by slight injuries or nutritive disturbances are sufficient to permit the platelet masses to adhere to the vessel wall. A phlebitis even when septic may exist without a thrombus for unless the inflammation is very severe and is accompanied with periphlebitis, it supplies only one of the three factors necessary for thrombus formation, *i.e.*, the diseased vessel. When a thrombus is followed by a phlebitis or *vice versa*, a phlebitis followed by a thrombus, a condition known as thrombophlebitis appears, which may be sterile or septic. A nonseptic phlebitis in presence of other necessary factors may give rise to a sterile thrombus thus forming a nonseptic thrombophlebitis.

A septic phlebitis or septic thrombus may respectively induce a septic thrombus or a septic phlebitis and thus give rise to a septic thrombophlebitis. But a mildly septic thrombus may, by its irritative properties, cause a nonseptic phlebitis(52), and again, a mildly septic phlebitis may, in presence of other favorable factors cause a nonseptic thrombus. We see then that not every case of thrombus nor every case of phlebitis is necessarily followed by thrombophlebitis, and not every case of thrombophlebitis is necessarily septic.

Omitting from our further consideration the thrombus and phlebitis as separate entities and excluding as far as possible the sterile thrombophlebitis we shall now take up the septic pelvic thrombophlebitis and attempt to bring out the points that may throw light on the present status of its treatment.

Pelvic Septic Thrombophlebitis.—The most important and to us the most interesting cases of pelvic septic thrombophlebitis are those of puerperal and postoperative origin, and of these the more common are the puerperal ones.

Septic Puerperal Pelvic Thrombophlebitis.—The striking frequency of septic pelvic thrombophlebitis in the puerperium is not difficult to explain. The condition of the blood, the dilated blood-vessels, the torn and gaping sinuses, the presence of bacteria in the vagina and the cervix, the extensive pelvic traumatism,

the prolonged dorsal position and the after-effects of functional disturbances of pregnancy, all these conditions are most favorable for thrombophlebitis.

As mentioned above, not every case of thrombophlebitis is septic, but there is no question that the great majority of cases of pelvic puerperal thrombophlebitis in septic cases are septic. The bacterial agent of this form of thrombophlebitis is most commonly the streptococcus. The rapidity and manner of the extension of this form of thrombophlebitis depends on the degree of the bacterial virulence and the degree of the patient's resistance. If the streptococci are highly virulent, especially if the patient is of low resistance, the infection usually begins as phlebitis. The streptococci enter from the endometrium into the endothelium of the veins and rapidly ascend along the endothelium of the vessels involving in a short time the uterine plexus, the spermatic, hypogastric, iliac, femoral and even the vena cava. Here the septic phlebitis is the predominant primary feature, the thrombus formation being secondary. If suppuration occurs, it begins at the vessel wall extending up and along the intima, the thrombus undergoing purulent changes later. (24–29–11)

In the development of milder forms of puerperal sepsis the veins are found thrombosed prior to the appearance of the infection. The bacteria invading the sinuses attack the thrombus first, and if suppuration takes place it begins in the center of the thrombus, extends toward its periphery, setting up a secondary phlebitis(11).

There is a form of puerperal septic thrombophlebitis that is secondary to lymphangitis. The bacterial causative agent of the lymphangitis may bring about a thrombophlebitis by local extension, *i.e.*, the bacteria found in the area of the septic lymphangitis may attack the neighboring vessels, causing septic phlebitis and then a thrombus. This same bacterial agent may cause thrombophlebitis through the general circulation, *i.e.*, the bacteria may be carried from the lymphatic system into the general circulation, cause the necessary blood changes, weaken the heart force and thus bring about a thrombus in a pelvic vessel that happens already to be injured or diseased. While such a thrombus primarily is usually not septic, it generally becomes so secondarily on account of the presence of the bacteria in the blood. The septic thrombophlebitis thus formed is of course secondary to the lymphatic infection and should be considered only as a complication of still graver an infection, *i.e.*, lymphatic septicemia.

Postoperative Septic Pelvic Thrombophlebitis.—The postoperative pelvic thrombophlebitis, while found in about 2 per cent. of laparotomies, is not as commonly met with in our practice as the puerperal. We do not believe that the postoperative thrombophlebitis is as frequently septic as the puerperal and we certainly cannot adopt the view that pelvic thrombophlebitis is always septic. The higher percentage of thrombophlebitis after myomectomies than after salpingooöphorectomies for infected adnexa, is sufficient to disprove it. Besides, a patient with a weak heart, in a bad state of nutrition, who always shows a high platelet count has all the factors necessary to give rise to aseptic thrombophlebitis if one or more of his veins is injured during operation (by separation of adhesions, by pressure with retractors or by simple chilling). There is no need to add infection as a requirement for postoperative pelvic thrombophlebitis.

But while we cannot consider infection as the only cause of postoperative thrombophlebitis, it is unquestionably the most common one and is almost always local. An infection carried to the cellular tissues around a vein may give rise to a phlebitis and periphlebitis, which limiting the vascular elasticity causes local slowing of circulation. An endophlebitis with consequent damage of the intima usually follows in such cases, bringing about a vascular condition necessary for the attachment of the platelet masses. If now the necessary blood changes are present in the circulation, from one of the many possible causes septic thrombophlebitis develops.(23)

It should be remembered here that in the postoperative just as in the puerperal septic thrombophlebitis, bacteria are not always found in the thrombus. In fact it is not always caused by direct action of the bacteria. For, while streptococci enter the lumen of the vessel to bring about a thrombus, the staphylococci and colon bacilli bring about a thrombus through the actions of their toxins and the products of decomposition. Here we may find an explanation for postoperative thrombophlebitis in clean cases with mild general symptoms. In separating bowel adhesions in such cases, the colon bacilli may find entrance into the perivascular tissues and by their toxins cause thrombophlebitis in the manner shown in Rubesch's experiments.[1]

[1] Rubesch's(45) experiments on animals with staphylococci and Heller's with colon bacilli show that even if these bacteria are deposited near the surface of the vessel wall, thrombi are found on the intima before the bacteria have a chance to invade the entire vessel wall. In such thrombi no bacteria can be discovered, their formation having been caused by bacterial toxins permeating the venous walls.

Speaking of septic thrombophlebitis we must distinguish between the acute on the one hand and the subacute and chronic on the other. This distinction is based on the extent of the gravity of the infection. The graver forms show an earlier onset, severer symptoms, more rapid progress and higher mortality. The milder forms show a later onset, milder symptoms, slower progress and a much lower mortality. The first type is the acute one, the second the subacute or chronic.[1]

Acute Pelvic Septic Thrombophlebitis.—In our study of septic pelvic thrombophlebitis we shall discuss chiefly the acute type, referring only here and there to the chronic type wherever its distinctive features demand it.

Symptomatology.—If we bear in mind the rapid progress and high mortality of acute septic thrombophlebitis, we can easily understand the importance of recognizing it early, for the energetic treatment such á disease demands, must be undertaken early if results are to be expected. To do this we must have an early diagnosis. But can we do it? Mahler speaks of a prodromal symptom consisting of a climbing or step-ladder pulse with a temperature remaining normal. Michaelis(32) speaks of a subfebrile temperature, meaning low rise of temperature between 99 1/2° and 100°. Each of them considers his symptom pathognomonic of oncoming thrombosis. If such symptoms could be found they would be of great value but unfortunately only a small number of authors confirm Mahler's and Michaelis's claims, most of them discussing these symptoms either deny their existence entirely or consider them rare. These prodromal symptoms therefore cannot be relied upon, although it is worth while bearing them in mind and to take advantage of them when they do appear.

The clinical phenomena of thrombophlebitis after its onset vary with the degree of the severity and the rapidity of the extension of infection. In mild forms, the subacute and chronic types, the symptoms may be so mild as to make the early diagnosis by clinical symptoms impossible.

The characteristic symptomatology of thrombophlebitis is best seen in acute cases. In the acute cases observed by us, all early cases, the clinical symptomatology at the time of the pelvic examination was not sufficiently characteristic to determine the diagnosis. It was the detection of the thrombosed

[1] The sterile thrombophlebitis is mild and often unrecognizable and does not come under the subject of our diagnosis.

veins of the uterine plexus at the pelvic examination in conjunction with the absence of any other palpable pelvic pathology, with the presence of general symptoms of infection, that settled in our minds the diagnosis. Of the clinical symptoms that are now considered more or less characteristic and that were found in our acute cases to be more or less constant, the most valuable ones are the severe, frequent and irregularly repeated chills, some lasting as long as fifty minutes. The frequency of the chills in the beginning at least, did not in our cases necessarily indicate a grave outcome. A case that within one week after the first chill reached a normal pulse and temperature had nine chills during the seven days.

Another feature in the symptomatology that is considered rather characteristic and was found to be so in our cases is the temperature with its irregular pronounced remissions. A sudden rise of temperature accompanying the chill is followed within a short time by a rapid fall to its previous level where, with slight remissions, it remains until the appearance of the next chill. It is not uncommon for the temperature to go up during the chill to 106° and go down to about normal in half a day.

As to the pulse rate in our cases, it was found rather lower than one would expect judging from the temperature. In a case of ours that terminated favorably, the highest pulse was 120 and when the temperature registered 105 and 105.2° the pulse was 96 and 108 respectively. In this particular case, the pulse, in spite of the good behavior during the height of the disease, became intermittent during convalescence.

An interesting symptomatic feature of the disease is the absence of pain. This is not due to mental dulness, for clearness of mind is rather a general symptom of septic thrombophlebitis. The patient as a rule does not suffer and feels comfortable except for a short time during and immediately after the chill when she is desperately ill.

Edema of the lower extremities was always met with in cases of extensive thrombophlebitis, the extent of the edema depending on the severity of the pelvic thrombophlebitis. In one case that was fatal the right leg was swollen to almost three times its normal size and in another case the upper parts of both thighs, both hips, the lower part of the abdomen and the vulva were involved.

The blood picture was not found to be characteristic. Our blood cultures were most frequently negative. Such findings

can easily be explained. The bacteria only entering the blood stream at intervals, are rapidly destroyed in the blood and the various organs of the body. Examinations, therefore, made more frequently and in more carefully selected moments should give better results. In fact Lenhartz claims to have obtained thirteen positive cultures in sixteen cases, his success, however, as far as our knowledge based on literature is concerned, has not been duplicated. So far as leucocytosis is concerned it does not seem to depend so much on the severity of thrombophlebitis itself as on its complications. The erythrocytes and the hemoglobin are always low on account of the hemolysis. The influence of septic thombophlebitis on the kidneys does not seem to be pronounced. Albuminuria was found strikingly absent except after the appearance of septic involvement of the kidney.

The complete symptomatology of the thrombophlebitis as given above is not found in every case. If the thrombophlebitis can be mapped out by palpation,[1] a diagnosis can be made no matter how incomplete this symptomatology may be (lymphangitis being the only condition to be excluded) but if it cannot be mapped out, a sufficient number of characteristic symptoms, above referred to, must be had before a diagnosis of septic thrombophlebitis can be made.

There is a symptom-complex which if found, may make us feel reasonably certain of the diagnosis of puerperal or post-operative septic pelvic thrombophlebitis even in absence of palpable pelvic veins. This symptom-complex we may describe as follows: A fairly good general condition, with slightly elevated pulse and temperature, with clear mind and good appearance, interrupted at various intervals by irregular intermissions of desperate disturbances consisting of a severe chill, with a pronounced rise and characteristically rapid fall of temperature, with a comparatively slight rise of pulse, and with a pronounced general depression.

Prognosis.—If a case is diagnosed as that of septic thrombophlebitis, how are we to take care of it? What mode of treatment should we choose? In order to obtain a better under-

[1] In our experience the palpation of thrombosed veins of the uterine plexus is easy, that of the ovarian is not easy. We are not speaking of the chronic ovarian thrombophlebitis with extensive periphlebitis that one may feel through an abdominal wall of moderate thickness, we are speaking of its acute form. We must say here in a general way that a thrombus of the uterine plexus is easy to be diagnosed early by pelvic examination even before the appearance of characteristic chills, while thrombosis of ovarian plexus is difficult to diagnose early; the early diagnosis must be made chiefly by symptomatology. If our view is correct, thrombophlebitis of the uterine plexus should and will be diagnosed earlier than that of ovarian plexus.

standing of the value of the different modes of treatment, let us first discuss the prognosis of the expectant and operative treatment, for on the prognosis must, to a great extent, depend our judgment as to the choice of treatment.

The prognosis of thrombophlebitis of course depends on the severity of the infection and the resistance of the patient. In mild cases, subacute and chronic, the infection may remain localized and the thrombophlebitis may undergo one of the favorable changes mentioned above, with or without embolism. In severe cases, the acute ones, the cases that we are mostly interested in, the mortality is high. Arnold W. W. Lea(32) collected 566 nonoperative cases of thrombophlebitis pyemia with 327 deaths. If we add to this Latzkos' own nonoperative 199 cases of septic thrombi, with sixty-eight deaths, we get a total of 765 cases with 395 deaths or a mortality of 51.6 per cent. Of course if the statistics of the milder cases that now pass unrecognized would be obtained, the mortality of nonoperative thrombophlebitis would in all probability be found to be much lower.

Now the mortality of operative cases, no doubt, must be higher at present than it will be in the future when the diagnosis will be made earlier. But we have no other way to judge rightly statistics except as they appear in their cold figures. The number of operations at our disposal at present may be too small to make deductions from them, but if we are to use them at all we must use them as we do in any other diseases or operations, that is, count all we have. Now counting them all, we find the following:

Williams' collected cases including his own five(53)	52[1]	with	28 deaths.
Latzkos' own cases	37	with	21 deaths.
Huggin's collected cases including his four with one death(25)	25	with	13 deaths.
The writer's collected cases	8[1]	with	2 deaths.
Total	122	with	64 deaths.

		Cases.	Deaths.
J. W. Taylor(49), 1905.	Vaginal	3	0
A. A. Landin(28), 1906.	Transper. lig. of rt. ovar	1	0
W. B. Bell(4), 1909.	Trans. excis. of rt. ovarian	1	1
H. D. Bishop(6), 1909.	Trans. excis. of rt. ovarian	3	1

Thus we get a mortality in operative thrombophlebitis cases of 52.4 per cent.

If these statistics based on only 122 cases are of any value, they show the mortality of operative thrombophlebitis to be higher than the nonoperative. While the lack of experience

[1] We deducted from Williams' statistics four of Latzkos' cases with three deaths. that we give under Latzkos' own statistics

and the unwillingness on the part of the patient and the hesitancy of the profession to adopt early surgical measures account to a great extent for the high mortality of the operation, yet at the present stage of our experience it is difficult to say what the mortality will be in the future. Cases may be operated on early and die, in whom under expectant treatment the thrombus might have undergone absorption, organization, calcification or even softening with favorable termination.

With such a prognosis in operative and nonoperative cases we cannot help but believe that the prophylactic and expectant treatment must for the present occupy a very prominent position in the care of this disease.

Prophylactic Treatment.—We saw that in the formation of thrombus, the necessary factors are: 1. increase in number and agglutinability of blood platelets; 2. retardation of the blood current, and 3. pathological changes in the intima of a blood-vessel. We saw also that in septic thrombophlebitis, in addition to the above, infection must be added as a very important factor. It is easily seen that the prophylactic treatment of septic pelvic thrombophlebitis should consist in measures directed toward avoiding or correcting these factors. So far as the blood changes are concerned, we have at the present state of our knowledge, no measures to control them. "If it were possible, says Zurhelle, "to reduce the agglutinability of the platelet, as great deal of good could result from such measures, but we are not in possession of such measures."(54) We doubt, however, that measures directed to diminishing the increased number and agglutinability of platelets are desirable because platelet changes are beneficial when abnormal conditions need their protection. In this connection it should be remembered that even the thrombus is a natural conservative process, for a septic parietal thrombus is a temporary shield plastered on an injured vessel wall to prevent either the bacteria and their toxins from entering through it into the general circulation, or to prevent the bacteria and their toxins already in the circulation from attacking it. The same is true of the occluding thrombus. It is also protective in character. It is a temporary cork in the vessel lumen keeping the circulation away from the diseased vessel or corking up the infection in a limited area for the protection of the general circulation. There is no reason therefore to look for measures to combat this increase in numbers and agglutinability of platelets. Prophylactic here consists in correcting the abnormal

conditions calling for this increase, such conditions being chlorosis, posthemorrhagic anemia, convalescence from infectious diseases, cachexias and bad states of nutrition in general. Therefore if any of these conditions be present before delivery or before serious operative procedures, they must be taken care of by such prophylactic measures as good nourishment, tonics, rest, fresh air, etc.

So far as preventing the retardation of the circulation is concerned, the prophylactic treatment consists in adopting measures to increase the weakened heart force and to avoid anything tending to diminish this force. If a patient before delivery or operation shows a lessened heart force it should be toned up and strengthened by rest, therapeutic agents, etc. While Jaschke(26) may be right that endocarditis with good compensation, especially in young people with normal blood count, needs no preoperative attention, it must not be overlooked that a diseased heart cannot accommodate itself to the strain and shock of an extensive operation as well as a good heart. For this reason it may in such cases be a part of good prophylaxis to avoid performing extensive operations at one sitting, also to avoid prolonged anesthesia and to make every effort to allay preoperative fear and excitement. As a prophylactic measure against lowering heart force may be considered the free movements after operation and labor, especially the early getting out of bed. This allows the organs to resume earlier their normal functions, the circulation to become more active, the blood pressure to reach a more normal level and all this to increase the heart force. There are plenty of clinical facts to prove this statement. While statistics give 1 to 2 per cent. of postoperative thrombophlebitis, the surgeons allowing their operative patients out of bed early show a much lower percentage in their cases. Thus Reese gives .4 per cent., Boldt .3 per cent., Mayos 1/3 per cent., Kroenig and Doederlein none(32). Of course this early sitting up after operation cannot always be carried out. Just as good judgment must be used in carrying out this prophylactic measure as in other lines of treatment. It would be poor judgment for instance to allow out of bed early a septic case with a high temperature, an acutely anemic case after a severe hemorrhage or a case with drainage through an abdominal incision, but in the great majority of cases this prophylactic measure can be enforced with great benefit. So much for prophylactic measures against retardation of blood flow.

The prophylactic measures against the third factor of thrombophlebitis (the injury of the vessel wall) are but few and in general this factor is beyond our control. Care, however, can be practised during operation to avoid injuries to vessels wherever it is possible. Profiting by the observations of Clark and Zweifel referred to above, we may be careful to avoid pressure by abdominal retractors on the epigastric veins and also pressure on the popliteal veins, the result of prolonged Trendelenburg position with bent knees.

The most important, most practicable and most efficient measures against septic thrombophlebitis are the ones directed against infection. They consist of course in careful observance of asepsis in obstetrical and pelvic operative procedures. With the present state of development of asepsis, and especially with the easy access of surgeons to the aseptic technic of the operating room, this prophylactic measure can be easily enforced. When one considers that in 6000 consecutive labor cases of von Herff's clinic there was no death from sepsis and that in 8000 cases in Ahlfeld's clinic there was only one death(32), the infection in this case being carried in by the patient herself, there can be no doubt that this prophylactic measure of thrombophlebitis can be successfully carried out.

It should be remembered, however, that in septic puerperal thrombophlebitis the infection is not necessarily carried in from the outside for the vaginal lochia according to Bumm and Seigert(32) was found to contain streptococci in from 50 to 75 per cent. of cases and the cervical lochia in 17.6 per cent. of cases. If therefore retained membranes or placental tissue project into the cervix or vagina they may carry infection into the uterus without any external aid. The same may be true of lacerations. Bathed in septic lochia, they may carry infection into the circulation without outside interference. As a prophylactic measure against the infective agent of septic pelvic puerperal thrombophlebitis must also then be given the complete evacuation of the uterus and the repair of lacerations.

Expectant Treatment.—The comparison between the mortality of the expectant and the surgical treatments of thrombophlebitis led us to state that the expectant treatment at present must be favorably looked upon. Now the chief dangers of septic thrombophlebitis are embolus, secondary organic changes and exhaustion.

Emboli become detached most commonly from thrombi that

undergo purulent softening. Not all septic thrombi so changed give off emboli and not all emboli are fatal. Again, not all septic thrombi undergo purulent softening. We do not know what percentage of septic thrombophlebitis undergoes the favorable thrombotic changes discussed above, but we have every reason to believe that it is high. Just as a septic exudate frequently terminates in absorption or in an aseptic connective-tissue formation, so may a septic thrombus terminate in absorption or in one of the favorable formations seen in the sterile thrombi. The frequency with which we meet phleboliths in the pelvic *x*-ray pictures should prove this point. In more than 50 per cent. of *x*-ray plates taken in Mayo's clinic for diagnosis of renal or urethral stone, phleboliths were found. If we could add to the number of patients in whom phleboliths were found the number in whom the thrombi were absorbed or had undergone organization, canalization, and softening the percentage would be a great deal higher. It would appear from these observations that thrombi in the pelvic cavity are almost general. Of course most of these thrombi must be sterile, but there can be no question that quite a number of them at one time or other were septic.

Not only the thrombus but the phlebitis as well may undergo favorable changes. There is no reason why it should not undergo the reparative changes that are commonly seen in the different tissues. The progress then of thrombophlebitis may under favorable conditions, lead to a favorable termination. If it were possible for us to find and induce these favorable conditions, a successful expectant treatment could easily be outlined. Let us see then what are the favorable conditions that can be induced.

In our expectant treatment we have to take into consideration the infecting bacteria, the thrombus and the phlebitis. If we could eliminate the infection a great element of the danger could be excluded. Infection could be excluded by the destruction of the bacteria, but in the present state of our general therapeutics, even of vaccin and antitoxin therapy, the bacterial agents of thrombophlebitis cannot be destroyed. While this is true, yet, we can to a certain extent increase the bac ericidal powers of the patients' blood, (phagocytosis, opsonins, etc.) by increasing her body resistance. Herein lies the first principle of the expectant treatment of thrombophlebitis, for by increasing this resistance, we may increase greatly the reparative power

of the inflamed veins and thus reduce or eliminate the danger. Of course this does not eliminate the possibility of embolism. Emboli can be detached at any time but this detachment is especially favored by increase of pressure behind the thrombosed portion of the vessel. Such increase of pressure may be brought on by disturbance of the organs involved in the thrombophlebitis, as by motion, pressure, rubbing, etc., or by sudden increase of heart action, as by stimulation, excitement, exercise, or inrease of blood pressure from any cause. Herein lies the second principle in the expectant treatment of septic thrombophlebitis. For by keeping the diseased parts at rest, the heart action and blood pressure at their even and normal level, we can easily avoid the disturbance of the thrombus, allowing it under the increased body resistance to undergo the possible favorable changes without detachment of emboli. Having these two principles in view, we can outline an expectant treatment which, while not ideal is at least rational. It consists in adopting measures to increase body resistance, to keep the parts involved at perfect rest and to keep the circulation and blood pressure at as even and normal level as possible. These measures are: A nutritious diet, tonics (cardiac stimulants to be avoided as much as possible), careful attention to the eliminating organs, sleep, avoidance of all excitement, perfect rest of the part involved in the thrombophlebitis and strict avoidance of pelvic examinations, of treatments such as douches, enemas and of such nursing at that attended with rough handling of the patient.

Such expectant treatment should give fairly good results, but septic thrombosis in spite of this treatment must show a high mortality, for a virulent type of streptococcic infection especially in a puerperal uterus, spreads so rapidly along the endothelial surface of the pelvic veins that neither ordinary body resistance nor perfect rest can control it. It can easily be understood, therefore, why the surgical mind turned to surgical procedure for remedies to combat septic thrombophlebitis.

Surgical Treatment.—Stimulated by the success the otologist attained in lateral sinus pyemia by ligation of the internal jugular veins, gynecologists introduced a similar procedure in pelvic septic thrombophlebitis. This procedure has so far not been received enthusiastically by the pelvic surgeons. Eighteen years have passed since its introduction to the gynecological mind by Freund and only 122 cases could be collected by the

writer from a rather extensive reading of the literature on this subject. As at present practiced the operation is performed through the abdominal and vaginal routes and consists in the ligation of the infected veins (with or without a phlebotomy) or in the excision of the vein. We shall first consider these different methods and then discuss their remedial value in the treatment of thrombophlebitis.

Abdominal Route.—The extraperitoneal abdominal route has been recommended highly by Trendelenburg, von Herff, Lenhartz and others. This method has recommended itself especially in purulent thrombi because of the possible avoidance of peritoneal infection from septic thrombi. Even Bumm, who is strongly in favor of the transperitoneal route speaks favorably of this method for unilateral septic ovarian thrombus.(24)[1] But this route has objections. It requires two incisions in a bilateral involvement. It necessitates extensive dissection with danger of infection of the lymphatics. It gives us a deep wound making the veins inaccessible and therefore difficult to expose, differentiate and inspect. For this reason mistakes are not infrequent. Williams(53) in his collection of fifty-six cases shows that Lenhartz in his eighth extraperitoneal operation tied off a ureter while ligating the left spermatic vein, ligated twice the pudic for the hypogastric vein and in one of these two cases ligated also a small vein for the spermatic. Another disadvantage of this route is its high mortality. C. J. Miller(39), cites 40 per cent. mortality for the extraperitoneal and only 21.66 per cent. for the transperitoneal route. Williams collected fifteen extraperitoneal cases with a mortality of 80 per cent.(53.)

The intraperitoneal route is the one strongly advocated by Bumm(11) and has more followers than any other route.[2] The

[1] An incision is made about 1/2 inch from a parallel to Poupart's ligament, dividing the skin, muscle and fascia. All the structures including the peritoneum are then reflected inward until the brim of the pelvis and the iliac veins are exposed. The exposure of the ovarian veins is easy, but that of the hypogastric veins, in case of inflammatory exudate in the broad ligament is extremely difficult. The possible injury to the ureter is also extremely great.

[2] A median incision is made and search is made for thickening or infiltration along the course of the ovarian and hypogastric veins. If found, the presence of thrombosis at the seat of infiltration is almost certain. In case of ovarian thrombosis the peritoneum is incised over the vessel, an aneurysm needle is passed under it and the vessel is ligated as far above the thrombus as practical. In case of thrombosis of the hypogastric vein it is necessary to draw the uterus firmly over to the opposite side, finding the hypogastric vein on the inner side of the artery. The peritoneum is divided exposing the vein which is then ligated close to the entrance into the common iliac. The median iliac, if present, should be ligated at the same time. If the ligation is high, it may, in order to expose the field of operation, demand considerable eventration with its consequent shock.

advantages of the transperitoneal method are: The vessel can be readily inspected, traced and isolated, making it comparatively easy to decide which vessels and how far they are thrombotic and where and how to ligate or excise. Mistakes, however, are possible even by this method, for such an experienced operator as Leopold ligated the hypogastric artery for the iliac vein (33). By this route the ureter can be kept in sight better than the extraperitoneal route and therefore injury to it can better be avoided during the operation. By this method a thorough inspection can be made of the pelvic organs, a bilateral ligation or excision and if necessary operations on the pelvic organs can be performed at the same time. Retroperitoneal infection or infection of the peritoneum itself can be discovered, that might be overlooked during an extraperitoneal operation. The mortality does not seem to be as high as in the extraperitoneal route. Williams(53) analyzed forty-one cases done by the transperitoneal route, with a total mortality of 43 per cent. while as mentioned before, in fifteen extraperitoneal cases there was a mortality of 80 per cent.

Ligation with panhysterectomy for early acute thrombophlebitis cases had been advised on the grounds that in such cases a septic endometritis and metrophlebitis usually exist. Ligation alone, by not removing the dangerous pathology, cannot relieve the condition. Opitz and Lenhartz(42) found pus in the uterus in most cases of chronic pyemia, especially in thrombophlebitis of the hypogastric vein, and therefore advise in acute cases hysterectomy with the ligation of the hypogastric vein. Latzko(30) reports thirteen hysterectomies (three without ligation) with eight deaths or a mortality of 61 per cent., which compares favorably with that of simple ligation in acute cases. Bumm(11) speaks against hysterectomy for acute cases stating that the operation is useless, but a 39 per cent. recovery makes the operation seemingly worth while to undertake.

The Vaginal Route.—Thrombosed veins of the uterovaginal plexus can sometimes be reached by the vaginal route. Usually only a phlebotomy is performed and drainage is established through the vaginal incision.[1] Taylor of Birmingham(49), who

[1] An incision is made over the thickened thrombosed vein, felt through the vagina and carefully avoiding the uterus. The veins are exposed, ligated if possible, the blood clots are turned out and drainage is established through the incision A search is made through the incision for any pockets of pus about the vessel and if found an additional drainage is established. As Latzko puts it, the effect of this incision is similar to the one the otologist achieves by making the jugular skin fistula of Alexander, (49).

suggested the operation, reported three cases with three recoveries (1905). Latzko reported ten cases with five deaths, a total of thirteen cases with five deaths, or a mortality of 38.4 per cent.

Ligation and Excision Compared.—A question of great importance in the operation is whether ligation of the vein above the thrombus is sufficient or excision of the vein between two ligatures must be practised.

The choice between these two methods depends a great deal upon the length of time from the beginning of the disease and on the seat of the disease. Ligation is the simpler, safer and easier. The results obtained by ligation are: 1. The checking of the extension of the infection; 2. prevention of metastasis; and 3. drainage of bacteria in a local area(53).

In acute cases according to Bumm(11) ligation is of no use. It should be remembered that in septic thrombophlebitis the venous wall may be involved by septic changes for a considerable distance above the thrombus, and if the vessel is tied a short distance from it, the clot that forms on the proximal end of the ligature will surely be septic(5).

As to the vessel to be ligated, some advise the ligation of the vessel involved only, others advise the ligation of all four vessels (Bumm(11) and von Bardeleben(2)).

There can be no question that excision is the better procedure in case of purulent softening of the thrombus (the excision to be done with the cautery knife). It prevents the passing of septic material into the collateral circulation, for as G. R. Noble(41) puts it, "A ligature is a poor barrier for a flood of pus dammed up behind it." But excision is exceedingly dangerous and difficult in thrombophlebitis of the hypogastrics and common iliacs. Literature does not report such cases as far as we know. Both Bumm and Trendelenburg(29), while considering the extirpation of the veins desirable, state that it is possible only with the spermatic veins.

In a number of the ligation cases phlebotomy was performed. The phlebotomy after ligation of course is possible only in the ovarian thrombophlebitis cases and then only through the extraperitoneal route, but as Haeckel(20) justly says, "The danger of retroperitoneal infection and the imperfect drainage makes it a poor procedure." Latzko performed fourteen phlebotomies after a preliminary ligation. Ten of these cases died, giving a mortality of 71.4 per cent.(30).

It is interesting in this connection to call attention to the operation suggested by Trendelenburg for removal of emboli from the pulmonary artery. He operated twice. With the first case he failed completely. The second case lived fifteen minutes. Sievers reported a case in which he removed two emboli from the pulmonary artery. The patient lived fifteen hours though there was no heart action noted during the operation. Kongers reported a case that lived five and one-half days and died of infection.

Comparing the mortality of the expectant and operative treatment of septic pelvic thrombophlebitis, one might justly be led to look with disfavor on the present operative procedures. But the last word on this treatment has not yet been said. If we, as a profession will give up every new surgical procedure that starts out with a high mortality, the progress of surgery will be very slow. The early mortality of fibroid operations was more than 30 per cent. and yet it is now considered one of our greatest surgical triumphs. Let us take the view that a surgical procedure that is rational must not be given up in its beginning because of its mortality. The ligation of a vein above the seat of a septic thrombophlebitis, checking its septic extension, preventing its metastasis and changing it into a local infective process is to a great extent a logical and rational procedure. But while it is logical and rational and justifies us in giving it a trial, in spite of the high mortality, we must always bear in mind its objectionable features, of which the most serious are the following.

1. Lymphatic infection may accompany thrombophlebitis or may even be its causative factor. The ligation or excision of the veins will not stop such pelvic infection.[1]

2. Early in the disease and even later there may be in the pelvis a number of slightly inflamed veins with small unrecognizable mural thrombi. Left undisturbed these septic foci will keep up the infection in spite of the operation. The postoperative histories and autopsies prove this.

3. In severe infections the septic phlebitis may extend far above the thrombosed portions of the veins. The ligation or excision of the vein even at a distance from their thrombotic portions may not include all the infected part or parts of the vein and therefore may not check the progress of the disease.

[1]Grossman in fifty-one septic cases found fourteen thrombophlebitis cases, twenty-four lymphangitis and thirteen mixed. Thus in twenty-seven cases of thrombophlebitis there were thirteen or about fourty-eight per cent. accompanied by lymphangitis. This percentage is rather high but it indicates how frequently in one's experience lymphangitis may complicate thrombophlebitis.

4. The operation itself presents difficulties that lead to serious consequences even in the hands of experienced operators. The operation requires handling of the thrombosed vessels making it possible for emboli to detach themselves from the thrombus, with all the dangers incident to such occurrences. The veins cannot always be recognized and isolated even by experienced operators. Wrong veins were ligated, ureters were included in ligation, an artery was tied for a vein by such an expert operator as Leopold, and the same surgeon was compelled to give up the ligation because of the difficulty of separating the iliac vein from the artery. Again, ligation alone is frequently an incomplete procedure, and excision is impractical in the iliac vessels. To the operative difficulties we may add the fact that usually the patient herself is a very bad subject for operation. These are the disadvantages that we must bear in mind in deciding our choice of treatment. At the same time we must remember that if the thrombophlebitis be left alone the septic vein may undergo favorable reparative changes and that the thrombus itself may undergo resolution, organization, canalization or phlebolithic transformation and thus become harmless.

Conclusion.—As we have said above the operative statistics do not compare favorably with that of the expectant treatment[1], but better acquaintance with the early symptoms of thrombophlebitis and lymphangitis, better recognition of the pathological characteristics of the septic thrombosed vessels, and improvements in technic may in the near future give the operative treatment a great many advantages over the expectant treatment as presently practised.

With the hope of contributing to the development of the diagnosis and surgical technic of septic pelvic thrombophlebitis, the gynecologists might feel justified in utilizing this procedure in the treatment of these cases, but in what class of cases one should adopt this procedure is not easy to definitely state. So far as the writer is concerned he feels at present that one is justified in undertaking the operation under the following conditions.

1. When a thrombophlebitis is diagnosed by vaginal examination before the appearance of chills, especially in the absence of pain (lymphangitis is usually accompanied by pain). Ligation by the transperitoneal route of the two veins of the side involved or of all the four veins if both sides are involved, should be the

[1] The gynecologists therefore have at present sufficient reasons for being slow to give up the expectant for the doubtful surgical treatment.

operation of choice, for in such cases the venous infection may be localized and by ligating the veins the extension of infection may be overcome.

2. In chronic septic thrombophlebitis when the general condition of the patient between chills is fair, when there are no coincident or complicating conditions, making the operation useless. In such cases the operation may consist of a transperitoneal ligation and in case of purulent softening and periphlebitis, an additional extraperitoneal incision and drainage. This with our present state of technic is practical only in the chronic ovarian thrombophlebitis.

3. As a procedure of last resort in any acute case unless it be definitely known to terminate fatally. While this class of cases will give a high mortality an occasional favorable result may be expected. The operative procedure will of course depend on the extent of the disease and the condition of the patient.

4. In acute cases with palpable veins, that come under observation after a number of chills with general condition between chills fair but getting gradually worse. Here, ligation of all four vessels with or without a panhysterectomy depending upon the patient's general condition, might be indicated.

5. In acute cases without palpable veins in presence of the symptom-complex above referred to and with a fair general condition. Here, for the present during the experimental stage of the operation, ligation with or without hysterectomy might be practised.

BIBLIOGRAPHIC REFERENCES.

1. Adami and Nicholls. Principles of Pathology, vol. ii.

2. Bardeleben. *Arch. f. Gynäk.*, lxxxiii. Strept. und Thrombose.

3. Bell, W. Blair. *The Practitioner*, London, Aug., 1911. Puerperal Septic Thrombophlebitis of Pelvic Veins with Report of a Case Treated Successfully by Operation.

4. Bell, W. B. *J. Obs. and Gyn. British Empire*, 1909, vol. xv, 396–401. Puerperal Septic Thrombosis of Pelvic Vessels with Report of an Acute Case in which Infected Ovarian Vessels were Excised.

5. Bennett. *British Med. Jour.*, London, i, 1–4. A Post-graduate Lecture on Treatment of Venous Thrombosis mainly from the Surgical Aspect.

6. Bishop, H. D. *Cleveland Med. and Surg. Reporter*, vol. xvii, p. 467–470. Treatment of Thrombosis of Pelvic Veins.

7. Brettauer. *Surg. Gyn. and Obs.*, vol. ix, 608–612. Operative Treatment of Puerperal Thrombophlebitis.

8. Brooks. *Jour. Exp. Med.*, N. Y., 1908, p. 271. Concerning the Coagulation Time of the Blood to Thrombosis in Phlebitis.

9. Buerger, L. Internat. Clinics, Phil., 19 s. 84–105. Association of Migrating Thrombophlebitis with Thromboangitis Obliterans.

10. Buhlig, W. H. *Surg. Gyn. and Obs.*, vol. vii, 51–58. So-called Phlebitis of the Left Leg in Appendicitis not Operated upon. With Theoretical Consideration of Etiology of Postoperative Phlebitis.

11. Bumm, E. *Berliner klin. Woch.*, July, 1905. Zur operativen Behandlung der Puerperal Pyæmie.

12. Bumm, E. *Zentralblatt f. Gynäkologie*, Oct., 1909. Behandlung des Puerperalen Fiebers.

13. Cragin. Am. Jour. Obs., 1906. Treatment of Puerperal Infection.

14. Cuff, A. *Jour. Obs. and Gyn. British Emp.*, 1906, vol. ix, p. 317. A Contribution to the Operative Treatment of Puerperal Pyemia with Report of a Successful Case.

15. Doederlein and Kroenig. *Operative Gynecologie.*

16. Dorman. Am. Jour. Obs., 1906. Pathology of Puerperal Infection.

17. Duncan, H. A. *N. Y. Med. Jour.*, 1907, 86, 1168–70. Etiology and Treatment of Thrombosis based on Studies of Coagulation Time of Blood.

18. Faix, A. *Bull. Soc. d'obst. de Paris*, 1906, lx, 325–335. Quelques mots sur la ligature des veins du bassin dans la pyemie puerperale.

19. Fothergile and Gordon. *British Jour. Obs. and Gyn.* Puerperal Thrombosis of the Inferior Vena Cava.

20. Haeckel. *Deut. med. Woch.*, 1905, 1637–1638. Unterbindung der Venæ Spermaticæ und Hypogastricæ bei puerperaler Pyæmie.

21. Harris, J. A. Amer. Jour. Obst., 1911, lxiii, 599–614. Puerperal Infection, Clinical Varieties and Treatment.

22. Hell, L. Aus der Clinic des Prof. Dr. von Herff. Ueber die Aetiologie und Prophylaxe der postoperativen Thrombophlebitis.

23. Hoffman, Adolph. *Monat. der Geburtsh. und Gyn.*, Band xxx, in Greiswald, 1909. Die klinische Bedeutung der

Thrombose und der derzeitige Stand ihrer chirurgischen Behandlung.

24. Howard. *Lancet*, 1906, i, 645–655. Hunterian Lecture on Phlebitis and Thrombus.

25. Huggins, R. R. *J. A. M. A.*, July 20, 1912. The Ligation or the Excision of the Ovarian or Deep Pelvic Veins in the Treatment of Puerperal Thrombophlebitis.

26. Jaschke, R. Th. *Therapeutische Monatsh*, 1908, xxiii, Jan. und Feb. Hertzmuttelle vor und nach gynäkologischen Operationen.

27. Klein, H. V. *Arch. für Gyn.*, 1911, xciv, 117–262. Puerperale und Postoperative Thrombose und Embolie.

28. Landon, A. A. *Australian Med. Gaz.*, Sydney, 1907, vol. xxvi, p. 120–122. Puerperal Infection Thromboses, Ligature of Right Ovarian Vein. Recovery.

29. Latzko, W. *Wiener klin. Wochenschrift*, xix, May, 1907. Die chirurgische Therapie des Puerperalen Prozesses.

30. Latzko, W. *Medizinische Klin.*, xxiv, 1910. Zur chir. Therapie des Puerperalen Prozess.

31. Kistler, H. W. *Jour. A. M. A.*, Aug. 10, 1912. Thrombophlebitis of Left Leg.

32. Lea, Arnold, W. W. Puerperal Infection.

33. Leopold. Thrombophlebitis Purulenta Puerperalis.

34. Macan. *Jour. Obs. and Gyn. British Empire*, Oct., 1908. Operative Treatment of Puerperal Fever.

35. Michaelis, Hans. *Münch. med. Wochenschrift*, Jan., 1911, p. 73–76. Prodromale Erscheinungen der puerperalen und postoperativen Thrombose und Embolie.

36. Michels, F. *Lancet*, 1903, 1025. The Successful Treatment of Puerperal Pyemia.

37. Michels, F. *Lancet*, 1909, Jan. 16, vol. i, 1656–1658. Further Contribution to Treatment (Surgical) of Puerperal Pyemia.

38. Miller, C. J. *New Orleans Med. and Surg. Jour.*, 1910–11, vol. lxiii, p. 583–590. Successful Case of Ligation of Ovarian Vein for Thrombophlebitis of Puerperal Origin.

39. Miller, C. J. *J. A. M. A.*, July 20, 1912. The Present Status of the Ligation of the Pelvic Veins in the Treatment of Septic Thrombophlebitis of Puerperal Origin.

40. Moore, W. *Intercolonial Med. Jour. of Australia*, Melbourne. Operative Treatment of Puerperal Pyemia.

40*a*. Mendel. *Münch. med. Woch.*, October, 1909. Ueber

Thrombophilie und das Frühaufstehen der Wöchnerinnen und Laparotomierten.

41. Noble, G. H. AMER. JOUR. OBST., 1907, vol. lvi, p. 189. The Treatment of Puerperal Infection.

42. Opitz. *Deutsche med. Wochensch.*, Dec., 1905, vol. xxxi, p. 2048. Zur Behandlung der Puerperal Pyemie.

43. Parke, W. E. AMER. JOUR. OBST., vol. lxiii, p. 638–644, 1911. Remarks on Treatment of Puerperal Septic Infection, Report of a Case of Septic Thrombophlebitis of Long Duration.

44. Roberts, H. D. *Phys, and Surg. J.*, Detroit, 1909, vol. xxxi, p. 495. Thrombophlebitis and its Relations to Phlegmasia Alba Dolens.

45. Rubesch, Rudolph. *Beiträge z. klin. Chir.*, Tübingen., 1911, 75. Experimentale Untersuchungen über die Beziehungen der Staph. Pyog. Aureus zur Infectiosen Venenthrombose.

46. Sinclair, Sir. W. J. *Surg. Gyn. and Obs.*, Nov., 1907. Surgical Treatment of Puerperal Fever.

47. Stowe, H. M. AMER. JOUR. OBST., Aug., 1909. Puerperal Sepsis.

48. Sutton, Blaud. *Lancet*, 1909; Jan. 16, vol. i, 147–152. Hunterian Lecture on Thrombosis and Embolism after Operation on Female Pelvic Organs.

49. Taylor. *British Jour. Gyn.*, vol. xxi, 1905. On Value of Colpotomy in Thrombotic Form of Puerperal Fever.

50. Veinberg. AMER. JOUR. OBST., 1909, vol. lix. Ligation of Pelvic Veins for Puerperal Pyemia with Report of a Case.

51. Veinberg. Trans. of Am. Gyn. Soc., 1910, vol. xxxv. When shall We Operate in Puerperal Septic Infection.

52. Welch, Wm. Allbutt's System of Medicine.

53. Williams, J. W. *Am. Jour. Obs.*, May, 1909, vol. lix. Ligation and Excision of Thrombosed Veins in Treatment of Puerperal Pyemia.

54. Zurhelle, Erich. *Beiträge zur Pathol.; Experimentelle Untersuchungen in der Anatomie; und Zeit. Allg. über die Beziehungen der Path.*, June, 1910, xlvii. Infectionendes Fibringerinung zur Thrombenbildung im Stroma des Blutest, zugleich ein Beitrag zur Morphologie.

PARK BUILDING.

DISCUSSION ON THE PAPERS OF DRS. FINDLEY AND SANES.

DR. R. R. HUGGINS, Pittsburg, Pa.—This subject is too interesting to let it pass without some discussion, and for fear

that I may appear too enthusiastic about operation in certain instances, I would like to say that I believe there are a large number of cases that recover without operation; that we find many mild cases of septic thrombophlebitis that recover under palliative treatment. On the other hand, my own personal experience shows six cases of the severe form in the last twenty years with five deaths under palliative treatment. Having this record, it seemed I could not make it much worse, and I decided two years ago that in the next case of septic thrombophlebitis I would explore the abdomen, see what was the matter, and if possible ligate the vessels. I did this, and the patient recovered. Since that time I have had three other cases, a total of four with three recoveries.

There are only three points for discussion: the first is the diagnosis, the second, the indications for operation, and third, the technic. In regard to the diagnosis, there seems to be a good deal of doubt on the part of the medical profession as to our ability to make a diagnosis in these cases. I believe there is a picture which is almost typical clinically of thrombophlebitis in the vast majority of instances. If there is a temperature such as is portrayed on these charts, with marked remissions, varying from a high point to almost normal or below, accompanied by chills and corresponding variations in the pulse rate, when the temperature is low the patient feels well and looks fine, but when the temperature goes up, there is a corresponding rise in the pulse and an increase in the severity of her symptoms, we are justified in suspecting the presence of this variety of infection.

How are we able to differentiate this form of infection from septicemia, which is the one thing with which it is most likely to be confused? As a rule, in septicemia, we do not find the wide fluctuations of temperature and frequent recurrence of chills. We do not have the intervals of apparent improvement, and the symptoms in every way seem more grave than in the early days of thrombophlebitis. The blood cultures in the latter are almost always negative, especially in the early stage, unless complicated by a septicemia. I believe that in nearly every case of septicemia, with the employment of good technic, the bacteria are found in the blood stream. To sum up in a case of this kind, going on day after day, with recurrence of chills, negative blood cultures, and the absence of local signs pointing to a lymphangitis or peritonitis, my belief is that an exploratory operation should be done. As to the technic of the operation, the abdomen should be opened in the midline which will give a good view of the veins and abdomen. In my experience, this had not done the patients any harm. If an accompanying lymphangitis is found, I see no reason why its presence forms an objection to operation, because there is no place in the body where infection is more dangerous, if allowed to progress, than in the lymphatics in the retroperitoneal space. If the veins are found thrombosed they can be ligated from within more easily than by the extraperitoneal

method. The abdomen can then be closed and if drainage is necessary, it is not difficult to make an extramedian incision, push the peritoneum forward, and drain behind the peritoneum to the region of the kidney, or downward into the pelvis, behind or between the layers of the broad ligament.