

perhaps applying an occasional detergent or disinfectant, would be to inflict needless distress. It may also become a question how far, when lung mischief has become revealed, it is desirable to persist in treating the uterine catarrh. It should not I think be always given up. The principle of curing as far as we can, every component part in a chain of morbid complication obtains here. But often it will be found the most judicious course to abandon local treatment, and to address all our care to the general system, and the alleviation of the lung-distress.

*Anomalies of Consistency.*—One of the most remarkable is the *pulpi-ness* of advanced age, coming on after long-continued mucous secretions, which disposes to ecchymosis or uterine apoplexy. Another form of softening is that ensuing upon childbearing, when involution is arrested by marasmus. The mucous membrane may also become soft from repeated hemorrhage. From constant infiltration or maceration it swells and disintegrates.

*Abnormal hardness* of the uterus affects chiefly the vaginal-portion in consequence of the predominance of connective tissue in hypertrophy.

*Lesions of Continuity of the Uterus.*—Lacerations may occur in the non-pregnant uterus. I have carefully described the lacerations of the pregnant uterus in my *Lectures on Obstetric Operations*, 3d edition, 1876. Under excessive distension from collections of blood or mucus, laceration or perforation has occurred. The uterus also has ruptured from the pressure of a polypus in its cavity.

The *tumors* of the uterus will be described in a separate chapter.

## CHAPTER XX.

PELVIC CELLULITIS (PARAMETRITIS); PELVIC PERITONITIS (PERIMETRITIS):  
GENERAL DISCUSSION ON PATHOLOGY AND CAUSES; PERIMETRIC INFLAM-  
MATION (PERI-UTERINE INFLAMMATION); METRO-PERITONITIS.

THE subject of inflammation of the pelvic tissues connected with the uterus and its appendages has been worked out, of late years, with great clinical skill; and, I may venture to add, with superfluous critical acumen. There is a natural tendency to embody or condense the new views we arrive at as to an essential pathological condition, by assigning to this condition a new name. If this name be tolerably precise and descriptive, it is often readily accepted as the last expression of science. Hence a name is apt to impose upon the learner the belief that he has caught the true clinical idea. And then in accordance with another tendency, the mind, satisfied with the seeming fulness of the idea embodied in a

new term, proceeds to eject every other term hitherto associated with the condition under discussion as false. Unable to entertain two ideas at the same time, hastily concluding that one or the other must be false, the one which is presented in the most attractive or authoritative manner is accepted, to the absolute exclusion of the other.

This reflection is remarkably illustrated in the history and varying nomenclature of inflammations of the pelvic structures. These inflammations of course remain, or continue to be reproduced, as they always have been. New names may represent new theories, but the clinical facts are unchanged. It is these which it is important to understand. It is to be feared that new names have tended rather to obscure these facts than to elucidate them. In attaching too much importance to names, that is, in allowing the mind to be dominated by the theories that names represent, we are apt to lose sight of the truth which lies in the rival names and theories. A true theory and a false theory are antagonistic. If we accept the one we must, logically, reject the other. But there is no antagonism between two true theories. These must be reconcilable, however widely observation and reasoning carried on in different lines may place them in opposition.

I will now endeavor to state the case plainly, divested of all theory or school-doctrine. All the structures in the pelvis are liable to inflammation. It is conceivable, and true in fact, that any one of them may be alone the seat of inflammation. It is conceivable, and true, that two or more of them may be inflamed together. We have already endeavored to trace the history of inflammation of the ovaries, Fallopian tubes, and uterus. It remains to fill up the account by tracing the history of inflammation of the other adjoining structures. What are these structures? We are not called upon in this place to consider inflammation of the rectum, bladder, or vagina, otherwise than incidentally. The structures with which we are now concerned are the cellular or connective tissue, the peritoneum, and the broad ligaments. No one disputes that each of these structures may be the principal focus of inflammation. For example, however we may cavil at the term "pelvic cellulitis," we cannot deny that the pelvic cellular tissue, that is, the connective tissue in relation with the uterus and broad ligaments, is liable to inflammation. Paris, Frasier, Courty,<sup>1</sup> E. Simon, Alph. Guérin, each relates cases of distinct pelvic cellulitis. "Pelvic cellulitis" expresses this fact, and nothing more. Again, however we may cavil at the term "pelvic peritonitis," we cannot deny that the peritoneum which invests the organs in the pelvis is liable to inflammation. "Pelvic peritonitis," then, is a good term, as expressing this fact. So again, at the bedside we are often called upon to speak of inflammation of the broad ligaments. When we so speak we do not pretend to define rigorously which of the constituents of the broad ligaments—connective tissue, vessels, muscular fibres, or peritoneum—is especially the seat of inflammation. Although undoubtedly inflammation may *begin* in the vessels, or in the connective tissue, or in the peritoneum, we shall rarely find an instance in which inflammation does not involve the proximate tissues more or less. We are there-

<sup>1</sup> *Maladies de l'Utérus*, etc. 1870



fore generally compelled to speak of inflammation of the broad ligaments in the aggregate. It is scarcely possible for the vessels and connective tissue inclosed in a thin lamina between the folds of the peritoneum to be inflamed, without involving the peritoneum. Now, the cellular tissue is chiefly situated on either side of the uterus surrounding the vessels and nerves at the line of ingress and egress: in front of the lower third of the uterus where its cervix is attached to the bladder; behind the uterus and vagina, where a stratum connects these organs with the peritoneum and rectum; and between the peritoneal folds of the broad ligaments. All this cellular tissue may be described as continuous; and therefore inflammation beginning at one part may spread to the rest. The part which seems most isolated from the rest is that mass of tissue which connects the cervix uteri with the base of the bladder. Accordingly this part is occasionally the seat of inflammation, which may run its course without spreading beyond its own limits, and without implicating the peritoneum, at least in any important degree. But the like limitation can hardly be predicated of any other part of the cellular tissue. The vessels which so often carry the cause of inflammation, and the peritoneum, are in such intimate relation with the cellular tissue at the sides of the uterus, and with that in the broad ligaments, that the serous investing membrane can rarely escape.

We thus come to the general conclusion, one amply borne out by clinical observation, that pelvic cellulitis, pure and simple, is a rare affection. Thus when we use the term "pelvic cellulitis," in the great majority of cases we use a term which only expresses a part of the morbid process. So far then the term is open to objection.

Again, the peritoneum covering in the pelvic organs being continuous with the abdominal peritoneum is subject to inflammation spreading to it from the abdomen. With this secondary inflammation we are not now concerned. The pelvic peritoneum is far more frequently the primary seat of inflammation, which, beginning here, may spread to the abdominal portion of the serous membrane. Now, if we ask how it is that the pelvic peritoneum is so prone to inflammation, we shall find a plain answer in the clinical and pathological facts. There is not—I say this with some confidence, yet not without reserve—evidence to show that inflammation begins in the pelvic peritoneum; inflammation in this membrane is excited by a morbid condition of the structures which it invests. That is, where the uterus, tubes, ovaries, vessels, and cellular tissue are healthy, scarcely any cause, other than extension of inflammation from the abdominal peritoneum, can produce inflammation of the pelvic peritoneum. The chief exceptions to this proposition are those cases where inflammation is kindled by some irritating matter poured into the peritoneal cavity, and which by gravitation or proximity to the seat of injury is chiefly concentrated in the dependent pouches of the pelvic peritoneum. And even in these cases where the peritonitis takes its rise in conditions independent of inflammation or disease of the pelvic organs, the cellular tissue, at least in some degree, soon becomes involved.

We come then to the further conclusion, that pelvic peritonitis, pure and simple, is also a very rare affection.

Disease rarely consents to the limits which medical nomenclature would

assign to it. All terms, therefore, which profess to be precise definitions are pretty sure to be fallacious. If then we conclude that the terms "pelvic cellulitis" and "pelvic peritonitis," which have been for some time in use, imperfectly, and therefore inaccurately, represent what we find at the bedside, the more recent terms, which are equally exclusive, must be equally fallacious.

The more recent terms to which reference is made are those introduced by one of the greatest of living pathologists, one from whose authority no one can dissent without hesitating long. Virchow<sup>1</sup> proposes to substitute the terms "Perimetritis" and "Parametritis." He bases the first term on the analogy with "Pericarditis." Just as the serous investment of the heart may be the special seat of inflammation, so may the serous investment of the uterus. Perimetritis, then, may be taken as the equivalent of pelvic peritonitis. It is difficult to see any sufficient grounds for preferring the new to the older term. The analogy with the heart is surely strained. The heart is completely, solely, and everywhere closely invested by its own exclusive serous membrane; the pericardium reflected upon itself forms a special bag, within which the heart alone is inclosed. The pericardium then enjoys absolute immunity from inflammation extending from serous investment of other organs; and comparative immunity from inflammation extending from any other source than the heart itself. Pericarditis probably, like pleurisy, chiefly owes its origin to offending matters carried in the blood. Peritonitis undoubtedly often owes its origin to like conditions. But the pelvic peritoneum, whilst not free from liability to inflame under general toxicohæmic influences, is exposed to inflammation from other causes. These are, as we have seen, metritis, salpingitis, oophoritis, pelvic cellulitis, inflammation of the vessels carrying septic matter from the uterus, irritating matters poured into the peritoneal sacs of the pelvis, and extension of inflammation of the abdominal peritoneum. The serous investment of the body of the uterus forms a very small part of a membrane which has numerous other relations, and which is therefore exposed to numerous sources of disease. If the term "perimetritis" be limited to the few square inches of peritoneum which covers the body of the uterus, it only expresses a very small part of the clinical case. Inflammation so limited is extremely rare. If the term be made to embrace inflammation of the serous membrane of the tubes, ovaries, and broad ligaments, then it is strained beyond its etymological meaning, and is wholly inadequate for clinical purposes. The correlative terms, "perisalpingitis" and "perioophoritis," proposed to supplement "perimetritis," prove the inadequacy of this latter term. Themselves, they are hardly worthy of discussion.

I submit, then, that there is no sufficient reason for adopting the term "Perimetritis."

The term "Parametritis," intended to describe inflammation of the cellular tissue in the neighborhood of the uterus, is also open to objection. It is less comprehensive than "pelvic cellulitis." It is not alone the tissue immediately surrounding the uterus which is exposed to inflammation. Indeed, inflammation of the cellular tissue is rarely so limited. Nor does it even always begin in this part. The inflammation in the

<sup>1</sup> Archiv. für Pathol. Anat. und Phys. 1862.



broad ligament often begins from disease of the Fallopian tubes or ovaries, and may never reach the cellular tissue near the uterus.

We are driven then alike by etymology and by clinical observation to reject both the terms "Perimetritis" and "Parametritis" as being inadequate, and not justified by scientific necessity.

The truth being that the pelvic peritoneum and the pelvic cellular tissue being each liable to inflammation, we want the terms "pelvic peritonitis" and "pelvic cellulitis." And it being also true that in a great, perhaps the greater, proportion of cases, both peritoneum and cellular tissue are inflamed together, we want a term which shall express this common affection. The term "peri-uterine inflammation," adopted by Courty, answers to this want. It is indeed open to an objection, which not seldom meets us in medical nomenclature, namely, that it is a discordant compound of Greek and Latin. This may be avoided by substituting the term "perimetric inflammation." In adopting this term I mean to include inflammation involving the broad ligaments and their contents.

This term carries us back somewhat to the term "Phlegmonous intrapelvic abscesses," adopted by Marchal (de Calvi), to whom science is indebted for the most important of all modern contributions to this subject.<sup>1</sup> His is the great merit to have shown that the chief seat of the puerperal and many other abscesses in women was in the pelvis. He thus exploded the erroneous ideas which connected these inflammations with the iliac fossæ.

And, since perimetric inflammation is so frequently consequent upon, and therefore complicates, inflammation of the uterus or its appendages, to express this compound condition we want the term "metro-peritonitis."

The researches of Bernutz<sup>2</sup> and of MM. Bernutz and Goupil,<sup>3</sup> conducted in the most admirably philosophical spirit have been pre-eminently useful in extending and in correcting our knowledge of the subject. These researches have demonstrated that what had hitherto commonly passed for pelvic cellulitis was often pelvi-peritonitis. The phlegmonous masses rising out of the pelvis, and extending into the iliac fossæ, so frequently met with after labor, are shown to be, strictly speaking, not pelvic cellulitis, but peritonitis. This much, with some qualification, may be granted. But it is to be feared that here again is an instance of one idea making good its way by driving out another from ground where both have a common right. In these post-puerperal cases, with which we are the most familiar, because they most frequently come under clinical and post-mortem observation, there is almost always a complication of cellulitis and peritonitis; and it might also be added, of metritis as well. The peritonic element will, it may be admitted, generally predominate. But the other elements coexist. It appears to me then that Bernutz, whilst rendering incontestable service in calling attention to the important part played by peritonitis in these cases, has rather undervalued the other factors of the disease.

<sup>1</sup> Des abcès phlegmoneux intra-pelviens. 1844.

<sup>2</sup> Archives générales de Médecine. 1857.

<sup>3</sup> Clinique médicale sur les maladies des femmes: Mémoire de la pelvi-péritonite et de ses diverses variétés. Paris, 1862.

*In post-puerperal perimetric inflammation*, which must serve for a type or illustration of other orders of cases as well, there may be distinguished three kinds:—

1st. That kind which, as far as we can judge, is simply inflammatory.

2dly. A kind in which septicæmia plays a conspicuous part. The inflammation is of a low type. There is a tendency to diffuse, or erysipelatoid inflammation and to a general systemic empoisonment.

3dly. There is a kind intermediate between the two preceding, in which there is a septic factor, but which is held in abeyance by the superior vigor of the blood. In these cases the septic matter is blocked out by the healthy blood coagulating in the efferent pelvic vessels, and intercepted by the lymphatic glands. In this way the system escapes, and the morbid influences are mainly concentrated in the pelvis.

*In the first, or purely inflammatory kind*, the action is chiefly spent upon the peritoneum. These may properly be called cases of pelvic peritonitis. They are analogous to the cases of pericarditis and of pleurisy, which supervene on a sudden impression of cold, when the pericardium and pleuræ have undergone unusual strain, the blood being also modified, under violent bodily exertion. The violent perturbation of parturition induces a peculiarly susceptible condition of the uterine and pelvic peritoneum, and an alteration of the blood which is favorable to the development of inflammation, if an exciting cause, such as cold or emotion, be applied. These are the cases to which the description of Bernutz more strictly applies.

In this order of cases the peritonitis, in many instances, does not break out until two or three weeks or more after labor. Thus, a young lady of delicate organization, suckled imperfectly for seven weeks; whilst menstruating she went to town, undergoing great fatigue, and came home with intense abdominal and pelvic pain and fever. Pelvic peritonitis had been produced. This is not an uncommon history. In another case a young woman was doing well until the fourteenth day after labor, when she was subjected to sexual intercourse. Shivering set in a few hours afterwards, and perimetric inflammation ensued.

But not even in all these essentially inflammatory cases is the inflammation chiefly expended upon the peritoneum. There is a sub-order of cases which appear to be essentially of traumatic origin, in which the chief, or at least the primary, seat of the inflammation is in the perimetric cellular tissue. During the passage of the child's head through the cervix uteri there is commonly laceration of the margin of the os, bruising of the mucous membrane, and of the whole substance of the neck, attended by a dragging or gliding movement of the structures in most immediate contact with the head upon the deeper parts. The cellular tissue around the cervix, where cellular tissue most abounds, is especially contused, stretched, vessels in it are torn, effusion of serum and ecchymosis take place in it. All this I have frequently verified by actual inspection. Everything is prepared for inflammation. There is the local injury, the effusion. There is the altered blood charged with effete matters, hyperinotic, under the influence of pregnancy and labor. An exciting cause alone is wanting. A chill is often sufficient. The chief seat of the inflammation in this case will be the wounded cellular tissue. In this tissue



it may run its course, ending in resolution or in abscess; and affecting the peritoneum slightly, if at all.

*In the second order of cases, characterized by the predominance of a septic factor, the inflammation of the pelvic tissues is universal. The uterus itself, its bloodvessels and lymphatics, the cellular tissue around and in the broad ligaments, and the peritoneum are all involved in a low kind of inflammation. The inflamed pelvic peritoneum throwing out unhealthy lymph which rapidly breaks down into pus, sets up the like inflammation in every part of the abdominal peritoneum with which it comes into contact. The poisonous matter may be generated in the woman's system under the strain of labor, her blood becoming overcharged with noxious materials, resulting from tissue-changes. To this order of cases I have given the name, now generally adopted, of "autogenetic puerperal fever."*

*In another order of cases the poisonous matter is inoculated, it comes from without. The woman, whilst in a highly susceptible state, takes in the poison of scarlatina or some other zymotic. The general infection of the blood here acts as the exciting cause of inflammation; and the inflammation will naturally, in the first instance, break out in the pelvic tissues, rendered susceptible by traumatic action. In these cases again the inflammation will not be limited to the peritoneum. It will invade the uterus, cellular tissue, and peritoneum alike. It must, however, not be forgotten that patients seized with this, the "heterogenetic" form of puerperal fever, not seldom die of the fever before any marked local inflammation declares itself. A feature distinguishing cases of this order from the first or simple inflammatory kind, is that the disease commonly breaks out much earlier, that is, within three or four days after labor.*

*In the third, or mixed order of cases, in which there is a septic factor, controlled by a comparatively healthy state of the blood, the inflammation begins in the uterine sinuses and lymphatics. Under the combined influence of traumatism, and of blood somewhat impaired by the tissue-changes of pregnancy and labor, and sometimes of decomposing débris of placenta, membranes, and blood-clots in the uterus, foul matters form in the uterine cavity, get into the uterine sinuses and lymphatic, and, not arrested there, either from want of contractile energy of the uterine fibre, or because, being as yet too abundant for the blood it meets in its course to segregate by coagulation, it invades the vessels in the broad ligaments, where further progress may be stayed by the formation of clots. This thrombotic process is generally attended by inflammation of the peri-vascular tissues, and of the broad ligaments, which is pretty sure to involve the peritoneum. If the lymphatics be concerned as well as the veins, then the phenomena of phlegmasia dolens are developed.*

That the broad ligaments are chiefly involved in the majority of these cases, seems proved by the seat of the tumefaction being in the sides of the pelvis; by the inflammation being in many cases unilateral; and by the uterus itself remaining in many cases apparently free from inflammation. That is, in this rather considerable order there is not necessarily inflammation of the peritoneal investment of the body of the uterus; that is, there is no perimetritis.

Trousseau may be cited as insisting upon the frequent complication of

phlebitis with inflammation of the broad ligaments. He believed that phlebitis was the most frequent cause of this inflammation.

A very similar description will apply to the perimetric inflammations supervening on abortion. It applies often very closely to inflammation of the broad ligaments leading to phlegmasia dolens beginning in cancer of the uterus.

Hemorrhage at the time of labor or abortion powerfully predisposes to perimetric inflammation. The parts being so predisposed, comparatively slight causes set up inflammation. A frequent cause is cold, usually so freely applied by ice or cold water injections, or by cold douche to the abdomen. Exposure to chill and getting about too soon are common causes.

It deserves to be remembered that pelvic peritonitis is not uncommon in the foetus; and that in after-life the pelvic organs may remain bound by persistent adhesions. This condition, it is highly probable, renders the subject unusually liable to new attacks of peritonitis when the organs are called into functional exercise.

*Perimetric inflammations occurring in the non-pregnant states, present features which it is interesting to compare with those which follow labor. They follow the same laws. When metritis is set up from the retention of decomposing matters in the body of the uterus, from traumatism, as from injury by the sound or other instruments, or from an intra-uterine pessary, the primary inflammation being in the body of the uterus, the secondary inflammation will attack the uterine peritoneum, at least chiefly. On the other hand, when the cervix is first attacked by inflammation, resulting from operations performed upon it, by the irritation of tents or other causes, the nearest tissue external to the cervix—that is, the cellular tissue in which the vessels run—will first catch the inflammatory process, and perimetric cellulitis will be the chief, perhaps the exclusive, secondary affection.*

Another illustration of this proposition may be found in the history of epithelioma of the cervix. This disease in its progress long respects the body of the uterus; as it extends, it involves the perimetric cellular tissue, and it is often late before the peritoneum is attacked.

Perimetric inflammation, apart from pregnancy, is not uncommon as the consequence of suppressed or disordered menstruation. During this function, we have in miniature the conditions of pregnancy and labor. The gorged organs, caught in a state of intense susceptibility, are exceedingly prone to become softened. Probably, too, in many of these cases there is some exudation of blood into the peritoneal cavity as a primary factor in the inflammatory process. In these cases, dissections of Bernutz and Goupil prove incontestably that it is the peritoneum which is the chief seat of inflammation. They found the cellular tissue perfectly free. This is no subject for surprise. Abortion and arrested menstruation differ from labor in this particular: the cervix escapes all traumatic injury; the seat of functional activity, and therefore of susceptibility, is the body of the uterus, the tubes, and the ovaries. Hence the body of the uterus, the tubes, and ovaries are primarily subject to inflammation, and inflammation of these organs is readily followed or attended by inflammation of their investing membrane.



Bernutz and Goupil affirm, and, if I may be permitted to express my own opinion, prove, that pelvic peritonitis, acute or chronic, takes its origin, in a vast proportion of cases, in disease of the uterus, tubes, and ovaries; that peritonitis is, therefore, secondary, symptomatic of other disease. They further maintain that inflammation of the pelvic peritoneum proceeds more frequently from inflammation of the tubes and of the ovaries than from inflammation of the uterus.

Although facts enough exist to prove that metritis, acute or chronic, may excite inflammation of the peritoneum, yet it is a remarkable clinical fact that, common as chronic metritis is, the uterus rarely becomes fixed, as it would be, were its peritoneum to become inflamed.

Although pelvic peritonitis in a large proportion of cases is caused by disease of the uterus, tubes, or ovaries, or is secondary upon pelvic cellulitis, it is nevertheless true that there is a large class of cases in which this membrane is the seat of primary inflammation. For clinical purposes it is important fully to recognize this distinction. The history of the two orders of cases is often strikingly contrasted. The secondary form following upon diseases of the pelvic organs is of course preceded by the symptoms which belong to those diseases; the peritonitis is an epiphenomenon, declaring itself in the course of another disease; its special characters often make their appearance gradually, even insidiously, being for a time masked by those of the original disease. On the other hand, the primary peritonitis makes its appearance suddenly; it is ushered in by acute and severe symptoms, often by shock or collapse, and other signs of traumatism or local injury. Such is the history of peritonitis caused by the escape of offending matter from the Fallopian tube, either running from its fimbriated extremity, or from bursting or perforation of its walls; from bursting or perforation of an ovarian cyst or abscess; from rupture of the uterus, or of an extra-uterine gestation-cyst; from effusions of blood into the peritoneum; from perforation of the intestine; from perforation of a dermoid cyst.

Even in some cases of this class the symptoms are not marked by suddenness of invasion or by great severity at first. For instance, when an ordinary ovarian cyst or a dermoid cyst undergoes perforation, the amount of irritating matter escaping into the peritoneal cavity may be small and the consequent peritonitis will be limited and subacute.

Pelvic peritonitis may, like inflammation of the peritoneum of the abdominal intestines, arise from 1, tubercular; 2 cancerous; or 3, traumatic affections.

Encysted serous peritonitis may appear to be, and sometimes is, associated with antecedent pelvic disease.

The connection of some unilateral pelvic or abdomino-pelvic abscesses with a pelvic origin is sometimes obscure. We make out clearly enough an abscess, and even define its limits; but dissection only can reveal the cause of the peritonitis, the products of which envelop and shut out from observation the offending disease. This is illustrated in a case related previously, in which a small ovarian cyst was found embedded in a peritoneal abscess.

Perimetric inflammation is rare after the menopause. This fact confirms the modern view that this inflammation takes its rise almost invari-

ably from the inflamed uterus tubes or ovaries. This proposition, although generally true, is however often affirmed too absolutely. Malignant disease, especially of the body of the uterus; chronic metritis, depending upon stenosis or flexion; the various forms of hypertrophy of the mucous membrane attended with hemorrhage, are very reliable, especially on rough surgical treatment, to lead to perimetric inflammation. It may also result from local violence, such as too frequent subjection to sexual intercourse.

Bernutz gives a summary of the cases observed. Of 99 cases of pelvi-peritonitis—

43 were puerperal	} 35 after delivery at term 8 after abortion
28 were blenorragic	
20 were menstrual	} 3 after venereal excess 2 after syphilitic disease of cervix 2 after use of the sound 1 after the use of a vaginal douche
8 were traumatic	

But to these causes must be added, the effects of diseases of the uterus, tubes and ovaries, and of the other organs of the pelvis; the accidents arising during the growth and degenerations of tumors, ovarian, dermoid or other.

*Peritonitis meretricum; gonorrhœal peritonitis.*—When gonorrhœal infection is the starting-point, the course is usually as follows:—The poison, acting first at the point of contact, lights up inflammation of the vaginal and cervical mucous membrane. This spreads to the mucous membrane of the body of the uterus, thence along the Fallopian tubes. The ovaries are very commonly engaged. In the case of gonorrhœa, Dr. Matthews Duncan says “he has never seen pelvic inflammation come on without the presence of ovaritis in addition, and as the ovaritis follows the endometritis, so the latter is itself a consequence of the original vaginitis.” In some of these cases, proof has been obtained that the peritonitis was immediately caused by the escape of infected pus from the fimbriated ends of the tubes. But in many cases, probably, the peritoneal coat of the tubes and ovaries becomes inflamed, consequent upon the inflammation of these organs.

Mr. Giles<sup>1</sup> relates three cases in which peritonitis, the result of gonorrhœa, broke out after childbirth. It must not, however, be concluded that the peritonitis of prostitutes is always traceable to infection. In many instances there can be little doubt that it is due to the wilful suppression of menstruation by the local application of cold, and to other forms of exposure and violence.

In this place reference must be made to the doctrine of “latent gonorrhœa” set forth by Dr. Emil Næggerath.<sup>2</sup> He submits that gonorrhœa apparently cured, may persist in certain sections of the organs of generation, in the male as in the female, for life, constituting “latent gonorrhœa;” that in this form it may infect a healthy person with acute

<sup>1</sup> British Med. Journal, 1871.

<sup>2</sup> American Gynæcological Transactions, 1876.



gonorrhœa or gleet; and that in the female it may pass from the latent into the apparent form, and give rise to acute, chronic, or recurrent perimetritis or ovaritis. It is obviously difficult to prove or to disprove how far this doctrine can be substantiated. I cite it, believing that it has at least an apparent basis in facts; and that the subject is worthy of further investigation.

*Course.*—When the opportunity occurs of examining the subject of pelvic cellulitis in the early stage, we may find a lax condition of the connective tissue; its meshes infiltrated with serum, lemon-colored, and limpid, or turbid and brownish, from being stained with blood or mixed with pus. When the affection is the result of labor, there is commonly ecchymosis from the rupture of small vessels.

At a later stage, the watery part of the serous effusion has disappeared; there is a firm, more or less circumscribed tumefaction, which on section exhibits reddish points, and evidence of hyperplasia.

In some cases, comparatively rare, pus is found in the phlegmonous swelling. But almost always when this is the case the peritoneum is involved, and the appearances are lost in those characteristic of peritonitis.

The chief character of peritonitis, of course, is plastic effusion. But this is preceded by intense vascular injection of the membrane. It is bright with punctate, stellate, and arborescent injections, and it is often uniformly red.

The membrane has lost its glistening smoothness; it looks villous or granular. This condition probably lasts only a few hours. Plastic lymph is quickly thrown out over the whole inflamed membrane, and glues opposing surfaces together. It is common to find the ovaries and tubes enveloped in a mass of yellowish lymph, more or less solid, and united to the peritoneal lining of the iliac fossæ, the summit of the bladder, the anterior wall of the lower part of the abdomen, and the front of the rectum. The fundus of the uterus is the part that most frequently escapes. As in life this part can often be felt and made out distinct from the firm tumefactions on either side or behind it, so after death we often find it cropping out comparatively unaffected from the fibrinous conglomerations of the sides and hollow of the pelvis.

At a stage more advanced in progress, but often even earlier in point of time, evidence, more or less extensive, of suppuration will be found. Where there was a septic factor, the lymph may be found in flakes, dirty red or yellow, adhering loosely to a dull-red peritoneum; easily breaking down, this lymph will be seen pultaceous, semi-fluid, purulent. In the half-circumscribed cavities formed between the imperfectly-adhering peritoneal surfaces, a dirty turbid serum collects; or, in some cases, the lymph seems to have no plastic property at all; then on being opened streams of dirty serum and pus flow out from the general peritoneal cavity.

In the stage of recent effusion, the parts can be separated by breaking down the still soft agglutinations. The ovaries or tubes or uterus, in which the inflammation probably began, will then be seen red or dull on their peritoneal aspect, generally swollen beyond their normal bulk, and fuller of blood. At a later stage, but still not remote from the beginning,

the effusions found will be increased in bulk and solidity. The organs, especially those of the pelvis, will be so buried in the consolidated masses of effused matter, that only by tedious dissection can they be traced and isolated; often the ovaries will be glued to the posterior wall of the uterus.

If a case in life correspond to the above description, recovery by resolution may still occur. The swelling seems to melt away, and the only post-mortem evidence of what has gone before are dull-white strings or bands tying the ovaries and tubes to the sides or posterior surface of the uterus, or to surrounding structures.

*Perimetric or Pelvic Abscess.*—In many cases suppuration takes place. Various estimates, fairly open to all the objections that invalidate most statistical operations performed upon pathological histories, have been made to express the proportion of cases which end in suppuration. It is certainly large, probably much exceeding those which end in resolution. McClintock, out of seventy-seven cases of puerperal pelvic cellulitis, found thirty-seven end in suppuration, with discharge of pus; twenty-four burst or were opened externally; six discharged through the vagina; five through the anus; and two burst into the bladder. The termination in suppuration is liable to be overlooked. Pus escaping into the rectum, or even into the vagina, may not be noticed, or, if observed, may not always be set down to the right source. These undetected suppurations naturally go in a statistical table to swell the number of cures by resolution.

The clinical physician will form a much more correct prognosis as to the advent or not of suppuration, by weighing the characters of the case before him. If there be septicæmia; if the patient be of strumous or lymphatic diathesis; if she be reduced by hemorrhage; if, in short, the individual conditions be of a depressing kind, the probability of suppuration is vastly increased.

Generally, but not always, in this event a fresh increment of the febrile symptoms is observed. Shivering or rigor occurs; the pulse is subdued in power; sometimes vomiting is excited. These mark the first entry of septic matter into the circulation, and constitute the stage of shock. The characteristic is depression. Then come the signs of reaction. The pulse is accelerated, the temperature rises. If the amount of septic empoisonment be great, signs of attempt at elimination appear. The poison, carried like almost all poisons to the intestines, irritates the mucous membrane and causes diarrhœa; and perhaps vomiting is again excited. This is the stage of irritation or elimination. If only one moderate dose of the poison is imbibed, the signs of constitutional irritation quickly subside; but if, as often happens, fresh doses continue to be imbibed, the symptoms of shock, reaction, and elimination will recur in regular order. This dependence upon the repeated dosing with, or accumulation of poison, is remarkably proved by the cessation of these signs when the purulent collection bursts or is artificially opened, or the case merges into chronic septicæmia. The aspect becomes dull, earthy; the patient emaciates; loses appetite; she is harassed with irregular attacks of shivering and flushes; prostration advances. This state may be protracted through many weeks.