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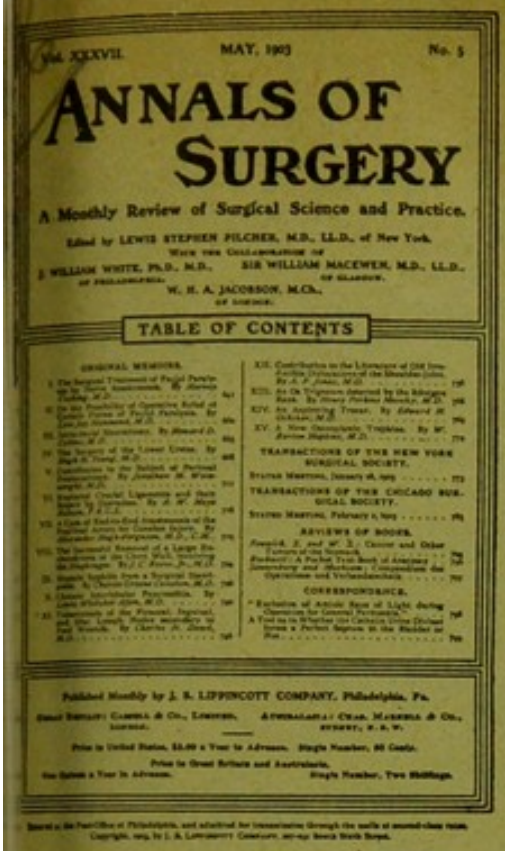

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With the Collaboration of

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TABLE OF CONTENTS

ORIGINAL MEMOIRS.

I. The Surgical Treatment of Facial Paralysis and Its Various Complications. By Charles Ballance, M.Ch. 641

II. On the Possibility of Operative Relief of Certain Forms of Facial Paralysis. By Lewis McMunn, M.D. 676

III. Intestinal Anastomosis. By Edward D. Fisher, M.D. 693

IV. The Surgery of the Lower Colon. By Hugh H. Young, M.D. 698

V. Contribution to the Subject of Perineal Prostatectomy. By Jonathan M. Wainwright, M.D. 711

VI. Ruptured Cystic Ligaments and their Repair by Operation. By J. M. Mason Knox, F.R.C.S. 716

VII. A Case of End-to-End Anastomosis of the Popliteal Artery for Complete Injury. By Alexander Hugh Ferguson, M.D., C.M. 725

VIII. The Successful Removal of a Large Portion of the Liver With Recovery of the Patient. By J. C. Munn, Jr., M.D. 734

IX. Stones Impacted in a Surgical Opening. By Thomas Evans Comstock, M.D. 748

X. Chronic Intussusception Permanently Relieved by Operation. By Lewis Wardner Allen, M.D. 760

XI. Tuberculosis of the Peritoneal, Inguinal, and Other Lymph Nodes secondary to Foci Elsewhere. By Charles H. Lloyd, M.D. 768

XII. Contribution to the Literature of Gall-Stone Ileus. By A. P. Lobdell, M.D. 778

XIII. An Os Trigonum Injured by the Ethogene Band. By Henry Perkins Merrill, M.D. 788

XIV. An Answering Tremor. By Edward H. Clarke, M.D. 794

XV. A New Osteoplastic Triphine. By W. Barton Hopkins, M.D. 774

TRANSACTIONS OF THE NEW YORK SURGICAL SOCIETY.

Stated Meeting, January 18, 1903 772

TRANSACTIONS OF THE CHICAGO SURGICAL SOCIETY.

Stated Meeting, February 2, 1903 785

REVIEWS OF BOOKS.

Hospital, E. and W. D.: Cancer and Other Tumors of the Stomach 792

Rochester: A Pocket Text-Book of Anatomy 792

Lehrenberg and Hartmann: Compendium der Gynäkologie und Verhandlungen 792

CORRESPONDENCE.

"Exclusion of Artificial Rays of Light during Operations for General Peritonitis." 796

A Tool to do Whatever the Carbolic Oiled Thread Serves a Perfect Purpose in the Hands of Man 796

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PUERPERAL GENERAL PERITONITIS,

REPORT OF ELEVEN CASES.

BY ELLICE McDONALD, M.D.,

OF NEW YORK.

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PUERPERAL GENERAL PERITONITIS,

REPORT OF ELEVEN CASES.

BY ELLICE McDONALD, M.D.,

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THE problems of puerperal infection are still unsolved and, since the introduction of aseptic methods into obstetric practice, no great advance has been made in the prevention and treatment of the condition. While women continue to lose their lives in such large numbers from this condition, little help has been offered by investigation. This disease annually accounts for thousands of deaths in this country, and is more worthy of study than many more abstruse but less important problems investigated by special committees and under special grants.

The course of puerperal infection shows but little difference from that of severe infection in other parts of the body. The great severity of the condition is due to several factors. The pregnant woman is more susceptible to infection, in general, than the non-pregnant, and is prone to disturbances of metabolism which lessen resistance and decrease elimination. The traumatism of labor causes a local disturbance of circulation and the raw surface of the uterus bares a huge surface for the entrance and growth of micro-organisms. The anatomical relations and blood supply of the organs have been temporarily altered and an easy portal of infection is presented by the genital parts of the recently pregnant woman.

Puerperal infection shows varied manifestations and, in itself, is not a sufficient diagnosis of a condition whose varied phases require different treatment. The clinical evidences of puerperal infection are those of a rapidly advancing genital infection. The endometrium, in the majority of cases, is the point of entrance of the micro-organisms and their products. Wounds of the perineum and vagina may become infected but, as a rule, the infection here remains local. If the endome-

trium is the seat of the infection, the micro-organism spreads into the veins and lymphatics and may gain entrance to the general circulation. This is the common course of streptococcus puerperal infection. In this form of infection, the essential lesion is a peri-uterine lymphangitis. This lymphangitis follows a similar course to the common and well known streptococcus infection of the arm, save that it is modified by the more generous lymphatic and blood supply and an adjacent serous membrane—the peritoneum.

This lymphangitis is more commonly associated with thrombosis than with the presence of actual pus in the lymphatics, which is a result only in severe cases, and accounts for Trendelenberg's [1] findings of thrombosis of the uterine and spermatic veins in 21 out of 43 fatal cases, and lymphatic infection in only four. Grossman,[2] in a study of 51 post-mortem examinations in puerperal infection, found peri-uterine thrombo-phlebitis alone in fourteen instances, and associated with lymphangitis in 13 other cases. However, the frequency of peritonitis, which must be from direct lymphatic extension, shows that the condition is more common than the post-mortem records show. The thrombosis of the peri-uterine veins is a matter of secondary consideration and a comparatively unimportant complication of the disease. The essential lesion is a spreading lymphangitis following the course of lymphangitis elsewhere in the body.

Cuff [3] has recorded a case in which laparotomy was done, of a severe puerperal infection with rigors and very high temperature. There was, on vaginal examination, a mass on the right side of the uterus which proved to be the broad ligament, " the thickness of three fingers stretching from the uterus wall internally to the pelvic wall externally." The ovarian vein was tied and the patient recovered. While assisting Noble of Philadelphia, I saw two such cases of puerperal infection in which the broad ligament was thickened, respectively, to one and to two and half fingers in breadth. Both cases got well after closure of the abdomen without further surgical treatment. There was no abdominal pus in either Cuff's case or the two referred to. This condition is, however, a preliminary state

before peritoneal infection. However, the condition may subside before peritoneal infection results, and become spontaneously cured.

This form of peritonitis is usually streptococcic and is marked by severe clinical manifestations, with little evidence of effort at repair on the part of the peritoneum. The local seat of the disease is about the broad ligaments and the pus is found generally distributed within the abdominal cavity, with little or no evidence of repair in the distant parts. Intestinal paralysis soon follows and the streptococcic form of the disease is almost always fatal. Sargent [4] believes that no recoveries follow this type of peritonitis, but I have seen two cases of general peritoneal infection with streptococcus recover after a simple laparotomy and drainage.

While this type of peritonitis is a most common one in the puerperium, there are other forms in which the infecting organism does not pass through the genital canal, but is the result of the lighting up of a previous focus of infection. Into this class fall all cases of infection from bruising of tumors, rupture of pus tubes, or purulent collections about the adjacent pelvic organs. Any variety of a pelvic tumor may be bruised or have its blood supply cut off by torsion at the pedicle and become inflamed and gangrenous, thus setting up a peritonitis. The important point to recognize is that, in this form, the lymphatics are not involved, and that the treatment is that of a non-puerperal peritonitis from a similar cause. However, it frequently happens, particularly in infection from pus tubes, that there may be associated two infections, one of which is of the nature of a lymphangitis, and the other is the old infected focus lighted up. Thus, as occurred in one of my cases, an old purulent gonorrhœal salpingitis may become reinfected with streptococci by lymphatic extension and form pus collections. This may rupture, causing a general peritonitis. In this way it will be seen that it is difficult to make hard and fast subdivisions between lymphatic puerperal peritonitis and peritonitis from previous infection.

While the streptococcus is the most common cause of lymphatic peritonitis, other organisms also have a share in its

production. The streptococcus is the most frequent cause of puerperal infection, and in a previous paper [5] it was estimated to be the causative organism in 40 per cent. of all cases.

In more recent communications on this subject, Lloyd,[6] in 159 cases, says streptococci were found in 33 cases, staphylococci in 30 cases, pneumococci in 17 cases, gonococci in 21, colon bacilli in 22 cases and bacillus capsulatus ærogenes in 2 cases. While these statistics are based upon a study of the infection in the puerperium, it has also been shown that the streptococcus is a frequent cause of infection in premature labor and abortion.

The frequency of the incidence of the various infecting organisms, is shown by the following table of eleven cases, in which they occur in about the usual ratio:

TABLE OF CASES.*

Case.	Local Lesions.	Character of Peritonitis.	Complications.	Organisms.
I	Acute hæmorrhagic endometritis.	Acute suppurative.	Acute suppurative endocarditis; septic pneumonia.	Staphyloçoccus aureus.
II	Abortion; acute suppurative endometritis.	Acute purulent.	Acute suppurative salpingitis.	Pneumococcus.
III	Premature labor.	Acute purulent.	Bilateral gonorrhœal salpingitis.	B. coli; streptococcus; gonococcus.
IV	Abortion; gangrenous endometritis.	Acute purulent, with petechial hæmorrhages.	Acute yellow atrophy of liver.	Streptococcus; B. capsulatus ærogenes.
V	Acute exudative endometritis.	Acute plastic.	Salpingitis; hydrosalpinx.	Streptococcus.
VI	.....	General plastic.	Pyosalpinx.	Streptococcus.
VII	Purulent endometritis.	Acute purulent.	.....	Streptococcus; B. coli.
VIII	Acute endometritis.	Acute purulent.	Abscess of cul-de-sac.	Streptococcus.
IX	Acute purulent endometritis.	Fibro-purulent.	Pericarditis.	Staphylococcus aureus.
X	Acute purulent endometritis.	Acute purulent.	.....	Streptococcus.
XI	.....	Acute purulent, with multiple local collections.	Bilateral purulent salpingitis.	Gonococcus.

* Some of these cases have been referred to before in other communications. A number of them were studied in the Bender Laboratory and some are from the records of that place. Others were studied at the N. Y. Lying-in Hospital and Kensington Hospital for Women.

These cases show that the streptococcus was the infecting organism alone in 3 cases, and associated with other bacilli in 3 cases. Thus, it will be seen that the streptococcus occupies a greater share as a cause of puerperal peritonitis than it does of puerperal infection in general. Also, Sargent [4] states that, in a study of 258 cases of peritoneal lesions, the staphylococcus albus was found in 108 cases; and he states that this and the colon bacillus form the most important and frequent causative organism concerned in peritonitis, outside of pregnancy. In the cases in which streptococcus was concerned, there were very acute symptoms and marked clinical disturbances. The course was fulminating, and was associated with marked anatomical disturbances in other parts of the body.

Case III, a case of premature labor at the sixth month, showed few symptoms until the seventh day, when there was a high temperature of 104°, with abdominal tenderness and rigidity. The uterus seemed well involuted and there was profuse purulent discharge. The physical examination showed a moderate sized fixed mass on the right side of the uterus. No swelling or mass in the cul-de-sac. The uterine discharge contained the gonococcus and streptococcus. The leukocyte count at this time was 29,000, with 90.5 per cent. polynuclears. On the ninth day after delivery, the patient was taken with sudden severe abdominal pains and marked tenderness and rigidity. The pain was peristaltic in character, intermittent and intense. The tongue was dry and there was slight vomiting. The physical examination showed fulness in the cul-de-sac, with marked tenderness and a disappearance of the mass on the right side; leukocyte count was 11,000, with 90 per cent. polynuclears. Laparotomy showed general distribution of sero-purulent fluid with marked congestion and redness of the viscera. This is most marked in the pelvic region. The right tube had formed part of the wall of an abscess beside the uterus and pus exuded from the fimbriated end and from the cut surface of the broad ligament. Death occurred nine hours after rupture of the abscess. The gonococcus, streptococcus and colon bacillus were recovered from the abdominal fluid.

Case IV was one of criminal abortion at the seventh month, and was seen five days after labor. The pulse was 140 and temperature 102°. There was slight jaundice. The leukocyte count was 19,000, and the polynuclears 86 per cent. Vomiting was continuous until stupor intervened and increased to coma. The patient died on the fifth day after the operation. The autopsy showed acute yellow atrophy of the liver, with general peritonitis. In the fluid was found the streptococcus and B. capsulatus ærogenes.

Case V was one in which streptococcic infection was associated with an old pyosalpinx; this caused an acute peritonitis, more marked in the lower part of the abdominal cavity, but which invaded the upper part as well. There was little pus. The hydrosalpinx on the left side was uninfected.

Case VI was a similar case to Case V, save that the peritonitial infection was not as acute, and there had been some attempt at repair by adhesions.

Case VII showed general peritonitis with a large amount of pus containing colon bacilli and streptococci. This followed upon an acute purulent endometritis. Death occurred on the tenth day.

Case X was a similar case, being marked by severe vomiting, a high temperature of 105° and intense pain and rigidity. Death occurred on the ninth day.

While the cases of streptococcus infection showed severe symptoms, the cases of infection with staphylococcus aureus also showed acute clinical manifestations with marked anatomical lesions. In this series the staphylococcus aureus was present in two cases.

In the study of puerperal infection, the staphylococcus aureus has been seldom isolated. Fullerton and Bonney [7] have found this organism in one out of 54 cases. Lloyd,[6] in a study of 159 cases, found staphylococcus in 30. The type of infection with this organism seems to be one of great severity. with a tendency toward pyemia and the production of metastatic abscesses.

Such was the result in Case I where, after an apparently

normal pregnancy, the patient was delivered by a midwife. She was seen on the eighth day of the puerperium, profoundly infected. There was marked tenderness and rigidity. Temperature was 104.3°. Death occurred on the next day. No operative measures were attempted. Post-mortem examination showed a large quantity of peritoneal pus with a dull injected peritoneum covered with a small amount of fibrin. The pericardial cavity also contained pus. There were areas of septic pneumonia. Bacteriological examination of the organs and pus showed staphylococcus pyogenes aureus.

Case IX was somewhat similar but ran a more prolonged course. The patient entered the hospital on the eighteenth day and succumbed to the profound infection on the same day. Autopsy showed a pericarditis, empyema and general peritonitis with little evidence of repair. The broad ligaments were thickened and firm and there was macroscopic evidence of lymphangitis, pus exuding from the cut surface of the broad ligament. In these two cases, the tendency of staphylococcus infection toward extension to the neighboring cavities and metastatic abscesses is well shown.

Gonorrhœal infection is one of the most common varieties of infection in the puerperium, and is the least frequently discovered. It usually causes little or no rise of temperature, but may cause high fever, serious morbid disturbances and death. Stone and myself [8] found the gonococcus in the lochia of puerperal women in 17 out of 171 cases. This organism was associated with peritonitis in only one case of that series. This is Case III, in which there was a bilateral purulent salpingitis from gonorrhœal infection and, following upon this, lymphatic peritonitis from the streptococcus, rupture of an abscess and general peritoneal infection.

Case XI was one of gonorrhœal infection of the uterus with extension to the tubes and peritoneum. In this case, there was a lesion of gonorrhœal infection before pregnancy, and the disease followed its usual course of rapid extension in the puerperium. The temperature went to 101° and the third day of the puerperium, the pulse was 96. The gonococ-

cus was isolated from the vaginal discharge. Pain, rigidity and a temperature of 103.6° appeared on the fifth day. On the sixth day, indefinite masses in the abdomen and pelvis were made out on abdominal and vaginal examination. Pulse at this time was 120. Pulse and temperature kept high until the seventeenth day when death occurred. Operation was refused. Autopsy showed bilateral purulent salpingitis and a general peritonitis of some duration. Collections of pus were found encapsulated by intestines and adhesions in various parts of the abdomen. The gonococcus was isolated in pure culture. Mann[9] has reported a somewhat similar case, in which the symptoms came on the tenth day. Temperature was 107°, pulse 120. Death occurred and the post-mortem examination showed a peritonitis from pure gonococcus infection.

Gonorrhœal infection usually extends by direct continuity of mucous membrane, but may, in the soft condition of the genitalia, penetrate the uterine muscle and extend into the broad ligament. Salpingitis is a frequent complication of this infection in the puerperium, as is shown by the study of Stone and myself,[8] in which we found clinical evidence of extension of the infection beyond the uterus, in 7 out of 17 cases. This extension of the disease may continue, as in Cases III and XI, to cause a peritonitis which is, however, usually localized in the cul-de-sac. The late complications of gonorrhœal infection are more to be feared than is the earlier uncommon general peritonitis. Salpingitis and pelvic peritonitis are the most common results. This is the cause of "one child sterility."

Pneumococcus infection is one of the more uncommon forms of puerperal peritoneal infection, and one of comparatively slight severity, save in isolated cases, as in Case II. This condition resulted in a woman four months pregnant, following an induced abortion. She was seen five days after the induction and was curetted for retained secundines. The pneumococcus was isolated from smears and cultures taken at the time of curettage. The temperature was 102 and continued high until death three days afterwards from peritonitis.

Post-mortem examination showed all the peritoneal sur-

faces to be covered by a sticky, greenish-yellow purulent exudate which was thin between the adherent intestines, but thick between surfaces held apart by collections of fluid. All the dependent parts of the abdomen contained yellowish turbid fibrino-purulent exudate. There was an acute suppurative salpingitis and endometritis. The pneumococcus was recovered from the peritoneal exudate.

While pneumococcus puerperal infection is uncommon, a number of cases have recently been reported. Weichelbaum [10] as well as Bar and Tissier,[11] have reported cases; and Cohn one similar to this. Fullerton and Bonney [7] found six cases of pneumococcic infection in 54 cases of puerperal fever; and Lloyd [6] found the pneumococcus in 17 out of 159 cases of puerperal infection. The pneumococcus is being more frequently isolated in such conditions, as the bacteriological methods of study improve. That the condition is by no means rare as a cause of infection of the peritoneum, is shown by Annand's and Bowen's [12] collection of 91 cases of pneumococcus peritonitis in children. They found that in half the cases the pus was encysted and that the peritoneal infection was usually secondary to some remote pneumococcal lesion. The exhaustive discussion by Jensen [13] of this form of peritonitis, gives a thorough idea of the subject. He reports several very interesting cases and gives a list of 143 references.

The character of this form of peritoneal infection is usually that of Case II, reported here. The infection is characterized by a plastic exudate, very rich in fibrin, which causes adhesions and encapsulation of the exudate; quite rarely, in the severer forms, the entire mass of the intestines adhere together and are surrounded by pus. In typical cases, the clinical picture is quite striking; the onset is that of an acute peritonitis, followed, very soon, by a chronic stage with mild symptoms and indefinite masses on abdominal palpation. The diagnosis is never certain without bacteriological examination, although one might suspect this infection from the thin, odorless, greenish-yellow pus and the abundant fibrinous adhesions. The prognosis is, as a rule, favorable; but recovery without

operation is rare. Simple evacuation of pus and drainage of the collections are all that is usually required.

There is, in this series of cases, no instance of peritoneal infection resulting from direct traumatism or necrosis of a tumor from pressure or torsion of the pedicle. A fibroid or ovarian cyst may take on rapid growth during pregnancy, and from torsion of the pedicle or sudden loss of nourishment from lessening of the blood supply after labor may become necrotic and infected. Lepage and Mouchotte [14] have collected a number of such cases. Similar causes lead to infection from ovarian cysts; and Getter [15] has reported 21 cases where, in spite of normal labors, infection of the cyst has occurred and led to fatal peritonitis. The infection is commonly due to the colon bacillus and is usually widespread and severe. Lawrence has reported ten such cases sent to the hospital as peritonitis following a puerperal genital infection. Patton [17] has collected 321 cases of ovarian cysts in pregnancy. In 95 cases treated expectantly until labor, torsion of the pedicle occurred 29 times—4 times during labor and 25 times during the puerperium. Rupture happened 13 times—3 before and 10 after or during labor. There were 25 deaths in the 95 cases, only 4 of which occurred in patients who had operations after labor, and 21 in those who were treated wholly expectantly. General peritonitis occurred in 7 of the 95 cases. There were 184 cases treated by operation, with a mortality of 8 (4.3 per cent.). Infection of ovarian cysts is especially likely to happen in the early puerperium. The teratoid ovarian tumors are particularly liable to be aroused from quiescence to rapid growth during pregnancy. This is well shown by a review of 35 cases by Neuhauser.[18]

Appendicitis is another lesion which may cause an extensive and fatal form of peritonitis in pregnancy and the puerperium.

The lessened resistance to infection of the pregnant woman has added to it the local abdominal disturbances of the presence of the mass of the gravid uterus and the increased vascularity of the pelvic viscera. Futh,[19] in his recent papers, up-

holds Waldeyer's statement that the cæcum and appendix are pushed up during pregnancy. This displacement begins about the fourth month, when the uterus rises out of the true pelvis. It then opens up the broad ligaments and the ovarian vessels are enormously increased in size. This elevation of the cæcum is of clinical significance as, in addition to predisposition to disease by reason of the alteration of position, the focus of inflammation is thereby placed in a more dangerous position—*i.e.*, higher in the abdomen, where adhesions are more easily torn and where inflammatory processes spread with greater ease. The cæcum returns to its proper place after labor; but, if the appendix is adherent to the uterus or adnexa, it is dragged into the true pelvis by the involution of the uterus. This may cause rupture of an abscess and increase the extent of the inflammation to the general peritoneum.

Seven casts of the abdominal cavity of women, dying during pregnancy, are described by Futh [20] to uphold this view. Five cases of appendicitis in pregnancy are also reported, and he states that appendicitis is much more dangerous after the fourth month, on account of the size of the uterus influencing the position of the cæcum. He has divided Boije's series into two groups. In the first, under four months, there were 10 cases, with three deaths. In the second, from the fourth to the ninth month, there were 32 cases with 19 deaths—a much higher mortality. This seems to be clear proof of the effect of the continuance of pregnancy upon the mortality of this condition. Hlawecek,[21] in 1897, collected 13 cases of peritonitis from this cause with 11 deaths; but under early operative treatment, the prognosis is brighter.

The diagnosis of generalized peritonitis in the puerperium is by no means easy. The extension of an infection in a woman already severely infected, shows few additional symptoms and small increase of pathognomonic signs. The diagnosis of puerperal genital infection is in itself not always easy, and the presence of pathogenic micro-organisms in the lochia is not proof of infection. Bumm and Sigwart [22] found that, by very careful examination, the streptococcus was isolated in the

vaginal discharge of 38 per cent. of women in the later months of pregnancy. Leo,[23] in an examination of the lochia in 38 normal women, in the puerperium, found the streptococcus in the vagina in 50 per cent. and in the uterine lochia in 17.6 per cent. In the later days of the puerperium, streptococcus is more commonly found, as was shown by a study of Schenk and Schieb,[24] who found this organism four times more frequently late in the puerperium than at the beginning. They found that streptococci existed in the lochia of one-third of all normal women. Also the discovery of a certain organism in the vagina, although an indication is not pathognomonic of the cause of infection. Sargent [4] reports a case of gonococcus infection found in the vagina while the peritoneum was infected with the pneumococcus. Stone and myself [25] have shown, in a study of the gonococcus, that this organism may exist in the uterine lochia without causing temperature. However, the presence of certain bacteria in the uterine lochia, as shown by smears and cultures, is some indication of the cause of infection.

The time at which the extension of the infection to the peritoneum occurs seems to be very variable. Lymphatic peritonitis from streptococcus usually appears from the third to the tenth day of the puerperium. It is seldom earlier but often later. The time of onset of symptoms of peritonitis from previous lesions also varies within a wide limit, but is usually later in appearance than the lymphatic form.

Pain is usually a prominent symptom, and occurred in all my cases. The pain of peritonitis is, I believe, fairly characteristic, and is a great aid in the diagnosis of the condition. It depends upon two conditions: First, it is now recognized that most of the pain in peritonitis is due to an accompanying lymphangitis. This, in part, causes the crampy pains of peritonitis—the lymph vessels of the intestines press upon the sensory nerves as the lymphangitis extends. The pain in the lymphatic peritonitis is not usually localized, as in appendicitis, but is sometimes referred, like that of appendicitis, to the epigastrium. This is supposed to be due to the infection in the peri-lymphatic tissue and the lymph glands around the aorta.

There seems to be but little pain from lymphangitis of the broad ligament.

The second source of pain is from the exterior of the gut itself, and is due to the presence of an irritant causing an inflammation of the peritoneum. The movement of the intestine causes severe crampy pains. That this pain is due somewhat to the irritant, and not directly to the inflammation, is shown by a case of abdominal hæmorrhage following Cæsarian hysterectomy upon which I did a laparotomy to control the bleeding. After clean hysterectomy there was a sudden flow of blood into the peritoneal cavity. The patient, who had been resting quietly, immediately complained of intense pain in the epigastrium and in the diaphragmatic region, generally. There was difficulty in breathing and nausea. The pain was intermittent and intense. Immediate laparotomy showed that the unclotted blod was generally distributed throughout the abdominal cavity. The character of the pain was similar to that of Case III, in which there was a sudden rupture of a pus collection, causing intense intermittent pain, crampy or peristaltic in character. In addition to the direct irritation and inflammation of the peritoneum, the pain is further caused by the rubbing of the inflamed intestinal covering against the parietal peritoneum. The visceral serosa has comparatively few sensory nerves while the parietal peritoneum is exceptionally well supplied. The pain in the diaphragmatic region, in the two cases cited, was probably due to irritation of the parietal serosa in the area. It is also recognized, however, that peritonitis may exist in the center of the belly, beneath the colon and above the pelvis, amongst the coils of the small intestine, for some time and become widespread without causing marked pain.

That acute abdominal symptoms may be caused by a lymphangitis alone, is shown by a report of Rowland,[26] of two cases of operation upon supposed perforation in typhoid fever. Masses of enlarged lymphatic glands were found in the mesentery of the gut without any evidence of perforation or peritonitis. The pain was intermittent and peristaltic. There was

localization of the tenderness with little or no rigidity. Armstrong [27] has reported a similar case in typhoid fever; and McCrae,[28] in his study of the pain in typhoid fever, cites two cases in which the explanation for the acute symptoms was the enlarged mesenteric glands.

In peritonitis, while the intestines are quiet, pain is not usually a marked feature; but, during peristalsis, it is usually present. For this reason, it is often useful for diagnostic purposes to set up peristalsis and elicit the pain by giving a purgative enema. This usually also gives a clue to the location of the point of greatest inflammation.

Rigidity is usually an early and trustworthy sign. It is present in all cases of peritonitis which were seen at their inception. It was present in the 7 cases of localized gonorrhœal peritonitis, before referred to. Rigidity, however, depends, in some measure, upon the suddenness of the onset of the peritonitis and sometimes does not last long. Intestinal distension usually overcomes it and causes it to disappear. To the educated hand of the surgeon, it is the most reliable early symptom.

Tenderness on palpation is not usually a marked symptom unless there has been marked effort at repair and the formation of much exudate and many adhesions. It may sometimes be produced vaginally by movement of the uterus. It can also be elicited after peristalsis has been set up.

Vomiting is a fairly constant symptom and occurs at two different periods in the disease. First, at the onset, there is usually vomiting, regurgitative in character, and later there is more persistent bile-stained vomiting, often fæcal or hæmorrhagic in character.

The temperature was rather variable in character. In the infection with the more virulent organisms, it went very high and was intermittent in character; but in the last days of the disease, this intermission was not present. In the less virulent infection by gonococcus and pneumococcus, the temperature while high, did not rise above 103°. The pulse in these two instances was also lower than in the more virulent

form. There was, however, nothing characteristic about the less virulent peritonitis. In Case III, after rupture of the abscess, the pulse suddenly rose from 90 to 120 and became of high tension and thready. The pulse is usually a better indication of the condition of the patient than is the amount of fever.

The blood changes of general peritonitis are interesting and instructive. There is usually a diminution in the red cells. This is more marked in puerperal peritonitis, and, in puerperal infections generally, than in infection in the non-pregnant. The leukocyte count is increased as it is in all septic conditions. This depends upon many conditions, as the patient's resistance and the virulence of the infection. It may be said that, as a general rule, in streptococcus infection, the leukocyte count is less in the pregnant than in the non-pregnant. A sudden fall in the leukocyte count (as in Case III, from 29,000 to 11,000) is suggestive of an overpowering of the system by toxins of the infective organisms. The polymorphonuclear leukocytes are usually increased in percentage.

Iodophilia is another useful sign and gives reliable evidence in all septic conditions. After staining with weak solution of iodine (Ehrlich's method), the blood, in cases of septic infection, usually shows a reaction in the cytoplasm of the leukocytes. This iodophilia usually occurs in the polymorphonuclear neutrophile cells and sometimes in the lymphocytes. It does not bear a definite relation to the leukocytosis; but depends upon the amount of toxemia, not upon the leukocyte range. Thus, the blood of a profoundly septic person may show intense iodophilia with a fall in the leukocyte count; while a high leukocytosis, without iodophilia, is not incompatible with an infection exciting a toxæmia sufficient to stimulate the cells to overproduction, but not of a character to affect them structurally. This fact makes the reaction especially valuable in cases of puerperal peritonitis, where there is often sudden overpowering of the system by toxins. An interesting review of the work of Cabot, Locke,[29] Dunham,[30] Dunn [31] and Keen [32] upon this subject is given by Da Costa,[33] with a report

of 100 cases, including 30 cases of sepsis in which the iodophilia was present.

It must be remembered, however, that the diagnosis of peritonitis can never depend upon the blood changes which only give confirmative evidence of infective processes. No hard and fast rule in regard to the degree of leukocytosis can be laid down, in spite of efforts of some of the camp followers of scientific investigation to have us diagnose our cases of sepsis in their private laboratories.

The bacteriology of the blood and lochia is of importance in forming an exact idea of the extent and character of the infection. Smears of the uterine lochia often give immediate and useful information in regard to the possible cause of the peritonitis.

Careful physical examination should be made upon all cases of suspected puerperal peritonitis, as it is the exception and not the rule for all patients with severe puerperal peritonitis to be without complications and infection in other organs. Care should be taken to recognize lung and heart complications, and search should be made for metastatic collections. Pyelitis and pyonephrosis are also not uncommon results of infection in pregnancy. The use of the Pravaz or Hollenbeck [34] needle has been suggested for the detection of free pus in the abdominal cavity, but should be used with great care, as there may be adhesions which would cause the bowel to be punctured.

The treatment of uncomplicated puerperal peritonitis is that of peritonitis in the non-pregnant. Immediate laparotomy and evacuation of the purulent matter with removal of the focus of infection, if possible, is recognized by all surgeons to be the proper procedure. While this is true in all cases in which peritoneal infection has resulted from previous foci as pus tubes, abscesses or necrosis of tumors, there is more difference of opinion as regards the value of operation and the procedure in lymphatic peritonitis.

Trendelenberg has advised, in this condition, resection or ligature of the veins of the broad ligament. It is decidedly

questionable whether this operation has any great value, as cases are noted in which recovery has taken place after simple laparotomy. The thrombosis of the veins is merely an incident of the infection, and offers no excuse for the laceration of the broad ligament and the division of the lymphatic channels, to allow the escape of micro-organisms. Hysterectomy, an operation which has been practically abandoned unless there is a local lesion, has more raison d'etre, for it removed a greater part of the infected surface and allowed of better drainage.

In order to thoroughly discuss operative measures in the lymphatic form of general puerperal peritonitis, a proper knowledge of the processes of infection with the most common causative organism, the streptococcus, is necessary. Streptococcus infection differs from most other infections in that the blood serum does not acquire streptococcidal properties, but the destruction of the cocci and relief from the infection is brought about by the leukocytes. There is a relatively small amount of toxin developed and no bactericidal properties in the serum, and it is reasonable to suppose that phagocytosis is an important factor in recovery. In addition to a leukocytosis, there must be an increase in the opsonin, *i.e.*, an increase in the power which prepares the leukocytes to engulf the bacteria. Any substance, then, which aids the body defenses to resist the infection is of use.

Anti-streptococcic sera have been tried for many years with poor results; but recent reports show that properly prepared polyvalent serum has given more satisfaction. Bumm [35] reviews his results in 32 cases of severe infection, and in four particularly striking cases of high fever, but without phagocytosis. Hyperleukocytosis occurred 12 hours after the injection of the serum and was intense, the leukocytes incorporating the steptococci with great avidity. Rau [36] has reported similar good results, and a reduction of his mortality from 60 to 36 per cent. in streptococcus infection. Escherich [37] has also reported beneficial effects in scarlet fever with the Moser polyvalent serum. Anti-streptococcus serum is, however, more of a prophylactic measure against peritonitis and it is doubtful

whether it has much effect after the infection has passed the confines of the uterus. It should, however, be used.

Other substances, however, are used to enhance the resistance of the peritoneum and to increase the activity of the leukocytes. Mickulicz [38] used intra-peritoneal injections of nucleic acid and performed operations as soon as the leukocytes began to increase. In 45 cases, the increase varied from 9 per cent. to 452 per cent. Seven cases died, but none from peritonitis, although two recovered in spite of this complication. Diez [39] also used nucleic acid and advocates the injection of a 2 per cent. solution as a preliminary to operation.

Hanner [40] reports 51 cases in which 50 c.c. of the same acid was injected in the form of sodium nucleate 13 hours before operation. Marked hyper-leukocytosis was present in every case. The reaction was always striking, the temperature rising one to two degrees C., with a chill in some instances. He also, as did Gray,[41] used quantities of dead organisms as vaccine, but the effect was doubtful.

It would, therefore, seem that the injection of nucleic acid is a useful adjunct to early operation in generalized peritoneal infection, particularly in infection from the streptococcus.

Operation should remove, if possible, any focus of infection with as little traumatism and handling of the intestines as possible. Lavage should not be performed, as the risk of spreading the infection is too great. The treatment should be after the method of Murphy and consist of making a small opening with the introduction of drainage tubes. Vaginal drainage is often useful. Evacuation of the intestinal contents in cases with paralyzed intestinal walls is sometimes of use. A high rectal tube may answer when the sigmoid is involved. If paralysis be higher than this, enterostomy is necessary.

Peritonitis characterized by abundant sero-purulent exudate and unaccompanied by signs of deep inflammation is a comparatively benign affection provided that operation is timely; but that form characterized by little or no exudate and with the gut wall red, dry, distended and paralyzed, gives an almost hopeless prognosis.

The prognosis, as a whole, in puerperal peritonitis is bad unless early operation is done. However, results are improving, as is shown by 121 cases collected by Jeannin.[42] These histories extend back to the early days of abdominal surgery, yet there were 60 recoveries and 61 deaths. This showing will be improved with more modern methods of treatment and earlier operation. In cases of puerperal general peritonitis treated expectantly, the result is almost invariably fatal, while in those cases treated surgically there are frequent successes, hence every case of puerperal general peritonitis should be operated upon as soon as diagnosed.

In the after-prognosis of such severe puerperal infections, it should be kept in mind that the late appearance of metastatic abscesses is not an uncommon condition.

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