ACUTE PERITONITIS.

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The mass of confusing and contradictory literature that one encounters in the study of peritonitis renders the subject as difficult to treat from the essayist's standpoint as it is in the therapeutic sense. If one will take the trouble to analyze the hundreds of reported cases, and critically read the more pretentious articles, he must be impressed that as yet there is no very well defined idea, no generally accepted teaching upon the nature of this affection. A prevailing fault with the general contributions to this subject is a failure to correctly classify the different forms of the disease. It is all-important to recognize the wide difference between localized infection, circumscribed area of inflammation, if you so choose to style it, and the contamination of the general peritoneal membrane with its necessary systemic intoxication.

In the short time allotted me I shall deal with acute peritonitis in its broadest sense. Tubercular peritonitis, chronic peritonitis in all its forms, and localized peritonitis, are not in any way referred to. In the report of cases which I append, the character and extent of the pathology was determined by the disclosures of the operating table. Nothing has been taken for granted.

Is it not now generally conceded that peritonitis in all its protean types, depends almost, if not entirely, upon micro-organisms? There are those who believe in the idiopathic form of this affection. Conspicuous among them is Senn. I can not presume to assert that the disease is absolutely an entity, of germ origin, yet such is my belief founded upon close study of the researches of bacteriologists. All surgeons are willing to accept the infective origin of the disease in those cases occurring in the presence in a parietal wound, or in

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the progress of a neighboring suppuration. There still remains a large number of cases dying of so-called idiopathic peritonitis, the true pathology of which we could not grasp, until it became an established fact that certain germs did under favorable circumstances pass through the intestinal wall, and by their presence and action produce peritonitis. This was a decided advance and before it vanished the material support of the adherents of the idiopathic idea. Cornil found bacteria actually in the substance of the wall of a partly necrosed intestine (Treves). In general septic peritonitis following intestinal wounds, perforating ulcer, etc., a special bacillus was found to be constantly present, the *bacillus coli communis*. It was known to inhabit the intestine under normal conditions, therefore its presence in the peritoneal effusion, after intestinal perforation, was considered of no ætiological significance. Further study of peritoneal exudates demonstrated the existence of this bacillus in peritonitis not due to perforation. Its pathogenic significance was then deemed of more importance, and finally when this anaerobic bacillus was actually discovered on its journey through the wall of a necrotic gut (Cornil and Treves) it at once assumed rank as a potent factor in the production of peritonitis. Observers were not slow to grasp the essential fact that so long as the intestinal tube remained in normal condition this bacterium showed no disposition to escape from its natural habitat, but when disordered circulation, strangulation, extreme distention, undue pressure or mechanical injury impaired the integrity of the bowel, and lowered tissue resistance a migratory spirit was at once incited in these bacteria. The behavior of this colon bacillus of Escherich after it reaches the peritoneal cavity depends more upon the resistance there met with than upon any characteristic of the special germ. It is well known that these bacteria are facultative anaerobics, that they are pyogenic in character, having been found in man in a state of almost pure culture in the pus from an ischio-rectal abscess (Treves) and in a general sense they are pathogenic by reason of the toxic ptomaines produced by them, and because of the local inflammatory process which they produce (Sternberg). They also do, perhaps, multiply in the blood, hence, producing both septicæmia and toxæmia. The conditions under which the *bacillus coli communis* is found as the predominating organism are in all types of peritoneal inflammation of intestinal origin. I can not better illustrate my meaning than by borrowing from Mr. Treves Macaigni's table of thirty-five cases of peritonitis of intestinal origin, in which the colon bacillus was practically always present.

Macaigni's Table.

With troubles in the appendix	10 cases
With typhoid fever	9 "
With ulcerative enteritis	6 "
With perforation	3 "
With cancer of colon	3 "
With hernia	2 "
With thrombosis of mesenteric vessels	1 "
With ulceration of the gall bladder	1 "
Total	35 cases

Appreciating the condition under which the colon bacillus may escape from its natural habitat, and become actively pathogenic, and knowing the supply is unlimited, the dose being governed alone by the integrity of the bowel, naturally we accord to this bacillus the first place in the causation of peritonitis. One can scarcely conceive of an intra-abdominal disease or injury that does not furnish the physico-chemical conditions essential to the action of this germ, yet in according all proper distinction to this, we must not be unmindful, for clinical reasons, if for no other, that long before we were acquainted with the morphology of Escherich's organism the potency of certain pyogenic bacteria of the streptococcus type had been demonstrated by Bumm, Fränkel, Renne and their work corroborated by a host of followers. These organisms were always introduced from without, and their source could generally be ascertained, and fortunately the supply being often limited, the results were not as destructive. I would argue, however, the disturbance consequent upon the infection of the peritoneal envelope of the viscera establishes at once Grawitz' ideal condition of lessened tissue resistance, which rallies to the field of battle the hitherto neutral colon bacillus, so that in the shortest possible time your case becomes one of mixed infection, the toxic features being largely due to the intestinal organisms. Yet, it is quite well known that puerperal peritonitis is almost always originally due to one germ—the streptococcus. In obedience to the teachings of experimental work, the surgeon must accept the classification of Pawlowski of two forms of peritonitis: 1. That produced by chemical agents, with which we are not concerned. 2. That produced by infection. The latter is more tangible. It is fully in accord with our idea of the genesis of the disease. It harmonizes with the clinical work. With Mordecai Price, I must agree that every case of general peritonitis has a demonstrable cause, and that cause is septic in character. Pathological manifestations of peritoneal infection are subject to many variations, which, in a great measure, indicate the violence of the poison, and guide us in forming a prognosis, but to

simplify matters, we may consider it under two heads, which are fairly illustrative of the microscopic and macroscopic changes—the results of general peritonitis.

First: The peritonitis mycotica is, I take it, synonymous with the septic peritonitis of Mikoulowlitz. This form of inflammation usually follows sudden outpouring of intestinal contents into the peritoneal cavity. Death is likely to occur at once from intoxication. If the cavity is examined very slight changes will be remarked. A yellowish green ichor may be the only manifestation. Should, however, the patient survive the onset, there quickly forms a sanguino-purulent fluid with but little tendency to fibrinous deposit. There is slight injection of the entire peritoneal membrane, no very gross changes.

The next type observed—the fibrino-purulent—is a much milder infection, slow in its onset, and is therefore met by decided structural changes. There is great congestion of the peritoneal membrane; flakes of fibrinous deposit are found throughout the peritonæum. A strong effort is made by Nature to circumscribe by adhesions the infected areas, thus retarding the progress of the disease. Ultimately, however, the cause still acting the adhesions yield, and general purulent peritonitis follows. To the inexperienced observer fibrino-purulent peritonitis appears by far the more malignant, when quite the reverse is true. The purulent exudate and fibrinous deposits are only evidences of Nature's resistance. Clinically speaking, we are frequently forced to content ourselves with the surgical diagnosis of the secondary condition of peritonitis, relying upon the revelations of the operation to establish the true pathology. It is, however, an indisputable fact that the type and virulence of the inflammation is largely dependent upon the origin—hence in our bedside work, we may consider the subject under the following ætiological classification:

Extraneous Infection.	Immediate.	This is direct infection of the peritoneal membrane through penetrating wounds of the abdomen, either accidental or surgical.
	Mediate.	This form embraces all cases of contamination of the peritonæum occurring from extension of adjacent infected areas, as leakage from mural abscesses, or puerperal infection.
Intestinal Infection.	Immediate.	Visceral perforation or rupture and direct inoculation of the peritoneal membrane with escaping contents, as perforating typhoid or gastric ulcer, appendicitis, or rupture of intestine.
	Mediate.	Infection from emigration by micro-organisms through visceral wall of impaired resistance, as in incarcerated hernia, intestinal obstruction, ruptured ovarian cyst.

It is proper to refer here to those rare cases of peritonitis occurring with attacks of pneumonia, rheumatism, etc., but having no acquaintance with such types, and very seriously questioning their ætiology, I will not devote further time to their consideration. In my abdominal work, I have met with peritonitis sufficiently often to warrant a record of a brief summary of a few illustrative cases, the treatment employed, the results, and endeavoring, as the cases are called, to assign them to their proper class.

Case I.—Mrs. K., aged twenty-four. April 28, 1893, was submitted to operation for supposed intra peritoneal rupture of ectopic gestation, accident occurring eleven hours before operation. The clinical history and physical signs warranted the diagnosis. At the time of operation she was *in extremis*, temperature 99°, pulse 140, hurried, thoracic respiration, the face was dusky, cyanotic, features drawn, great anxiety and restlessness depicted in her countenance, abdominal muscles rigid, sensitive to hyperæsthesia, slight tympany. This condition of shock was attributed to hæmorrhage, therefore operation was undertaken before thorough reaction was established. The cavity opened, a quantity estimated as one quart of brown sero-purulent effusion escaped. It was horribly offensive, and well it might be, for on examination it was found to contain fæcal matter. The seeds of strawberries, eaten two days before, were scattered about within the peritoneal cavity. Not an adhesion was seen. The general contents were bathed with this septic material. Immediate search was made for the appendix. To my surprise, it was entirely gone, leaving a round ulcer, the size of a dime, perforating the caput coli, through which escaped the intestinal contents. This escaping material entered directly the peritoneal cavity, contaminating everything. The edges of the ulcers were trimmed, and perforations carefully folded in and sutured. The cavity was thoroughly irrigated with gallons of hot water; the intestines and omentum were washed, ample drainage—glass tube and gauze—was used. The operation was done in great haste owing to the extreme condition of the patient. When taken off the table I feared she would not react. Under strychnine and other stimulants she revived, and the case progressed uneventfully until the third week, when symptoms of intestinal obstruction developed. The cavity was promptly opened, and a coil of intestine was found twisted upon an adhesion. This was freed. The patient now went on to slow, but absolute recovery, and she remains a perfectly well woman to-day with none of those post-operative symptoms, which are so likely to follow where there has been peritoneal inflammation.

Remarks.—The operation in this case was undertaken upon an erroneous diagnosis. Not by way of excusing myself, but with the hope that it may be profitable to some, I will indicate the sources of error. A young healthy woman passed six weeks without menstruating, then irregular uterine hæmorrhage, appearing suddenly and as quickly ceasing, paroxysmal colicky pains in the lower abdomen, a swelling in the right broad ligament, sudden onset of abdominal symptoms, intense abdominal pain, a quick, soft, almost imperceptible pulse, rapid respiration, cold limbs and extreme prostration were the misleading features that called forth the diagnosis of ruptured tubal pregnancy. The ætiology and pathology of this case assigns it at once to the class of the *diffuse septic peritonitis.* The infection was from within, of immediate intestinal origin. The perforation in the cæcum permitted the escape of contents of the gut directly into the cavity. It certainly was *diffuse septic peritonitis.* The dose of infection was so large, the toxæmia so profound, I mistook the shock it produced for hæmorrhage. That it was general can not be denied—not an adhesion was found, yet this woman recovered. Mr. Treves says: "I am doubtful if a single human life has been saved by surgical interference in a genuine case of peritoneal toxæmia," and he is an honorable man—an indisputable authority. Will you, gentlemen, indicate my error? Am I improperly recording a case? I consider the recovery of this case to be due to three things: First, prompt operation; second, multiple incision; third, thorough irrigation and ample drainage.

Case II.—Miss R., prostitute, patient of Dr. James Stephens and Dr. Menees of Nashville. Pyosalpinx following criminal abortion. One of her physicians, while conducting an examination, ruptured the pyosalpinx. The patient appreciated the accident and said something had burst within her. In twenty-four hours there were general symptoms of suppurative peritonitis. I urged operation, but it was declined. The patient appeared in fairly good condition, pulse well sustained, and temperature marking 100° and 101°. On the fourth day after the accident, we obtained consent to operate. When the cavity was opened several pints of thick, purulent, but not offensive fluid escaped. Strong adhesion bound coils of intestines in pelvis together, yet these barriers had been broken down and septic matter had invaded the general surface of the peritonæum. Thorough irrigation and drainage constituted the treatment. Decided temporary improvement followed the operation, but she died at the end of the third day of septic intoxication.

Remarks.—There is but little to remark upon this case, except to

cite it as another illustration of the fatality of delay. Our experience teaches us all that temperature is no guide in peritonitis. Dalton of St. Louis has written at length upon this point. In this particular case, in the four days following rupture, temperature remained under 101°, and the pulse of good volume, varying between 90 and 110. All of this with a belly full of pus and not one adhesion to fence it off. Does it not show that certain individuals enjoy a comparative immunity, or does it prove that the *streptococcus pyogenes,* the active micro-organism in puerperal infection, does not produce such violent systemic intoxication as the colon bacillus. This case is one that should be classed as purulent peritonitis. Mediate infection from without through the genital tract.

Case III.—Miss W., patient of Dr. Swaney of Gallatin, Tenn. History of intestinal obstruction of six days' duration. When first seen by me she was *in extremis*, temperature 97°, pulse almost imperceptible, and too rapid to count. Appreciating the gravity of the case, and recognizing that her only hope lay in a surgical operation, I resolved to hazard the procedure. The cavity was quickly opened: there gushed forth a quantity of pea-soup-like fluid, and coils of distended intestines bulged through the wounds. Seizing the presenting coil, it was drawn out and freely incised, thus relieving the gaseous and fluid distention. This wound was carefully closed, and now with flaccid gut and empty cavity, I sought and found the source of trouble. It was a band of adhesion stretching from the omentum to the gut under which had become ensnared several feet of small intestine. Flakes of fibrinous deposit were patched about the peritoneal surface. There was no matting of the intestine, nor effort on the part of Nature to limit or circumscribe the inflammation. The cavity was thoroughly irrigated, a quantity of water left in and drainage established. The condition of patient improved constantly during operation. Her recovery was uneventful. Her subsequent history is one of perfect health.

Remarks.—The recovery of this patient was a source of great gratification to me. It makes a striking reality of the metaphor "taken from the jaws of death." There is nothing unusual in the case. It merely furnishes an illustration how a purulent peritonitis may develop by auto-infection through tissue of impaired resistance. The strangulated and distended gut was the medium through which the colon bacillus passed. Fortunately her vital forces resisted the invasion, the plastic exudate marked the battle-ground, finally Nature succumbed to the functional disturbance and to the gradual dose of

infection, and the patient was dying of exhaustion and toxæmia. Perhaps in this case it was not necessary to irrigate. Dry gauze might have cleansed just as thoroughly—so say some—I do not think so.

Case IV.—Mr. E. H. I found him lying in my office bathed in cold perspiration, almost pulseless, short, shallow respiration, cyanotic, screaming with intense abdominal pain. The muscles of the abdomen were as hard as a board, and strongly retracted. McBurney's point was no more pronounced than any other point. From pain and shock he was slightly delirious, yet I elicited that he had had pain in the right inguinal region for two or three days, though not sufficient to confine him to his home. From this meager history and the present condition of patient Dr. Wilson and myself diagnosticated perforated appendicitis. He was moved to his home. The intensity of the shock seemed so great that I did not deem it wise to operate at once. I resorted to proper stimulation. That night at eleven o'clock, nine hours after the accident, his condition was considered suitable for operation. The usual lateral incision was made. On opening the peritonæum a little greenish-yellow fluid with fæcal odor escaped. The appendix was found, small, gangrenous and perforated. This was removed and stitched over. The head of the colon was unhealthy and a point of perforation just above the attachment of the appendix was found and closed. Believing the general cavity infected, a free incision in the median line was made for irrigation and drainage. The patient did well for twenty-four hours. Reaction was thorough. Septic peritonitis developed later and he died in fifty-six hours.

Remarks.—The unfortunate issue in this case does not deprive it of its interest or lessen its teaching value. You will please note that this gentleman engaged in his daily occupation was seized while walking along the street with violent abdominal pains which proved to be due to the perforation in the cæcum at the base of the appendix. This in itself is worthy of record. Abdominal rigidity is incidentally alluded to by all as one of the phenomena of intra-abdominal trouble. In this robust muscular patient the rigidity amounted to a clonic spasm of the abdominal muscles. These were absolutely so resisting that you could not depress them. They were so much retracted that they appeared to lie flat against the spine. There was also general abdominal hyperæsthesia. This reflex nerve phenomenon is due to the sudden impression made upon the nerve centers within the belly, the solar, cœliac and superior mesenteric plexuses. And it is noteworthy that the contribution they receive from the spinal nerve is de-

rived in whole, or in the greater part, from the lower seven dorsal nerves through the splanchnics (Treves), and as you are aware these same dorsal nerves supply the integument and the muscles of the belly. This muscular rigidity is never seen in old chronic cases. It is only marked when a sudden impression is made upon a peritonæum, previously healthy. So says Treves, and it was certainly apropos of this case. The shock in this case was extreme. Certainly so slight a lesion does not explain it. It is attributable to the quick absorption by the healthy peritonæum of the septic matter. This, and this alone, could produce such depression of the vital forces. The poison of the most venomous reptile could not have acted with greater rapidity and with more virulence. The operation was done as quickly as his condition would admit. Too soon to note any macroscopic changes except at the point of infection. A little greenish fluid was the only exudate. This case should be classed an immediate infection from within resulting in diffuse septic peritonitis.

Case V.—Master D., aged seven, patient of Dr. Woodson of Gallatin, Tennessee, for three days had suffered with symptoms of appendicitis. When I saw him, his temperature was 99°, his pulse 140. The general and local symptoms were those usual to this disease. I advised immediate operation, found sero-pus, a gangrenous appendix and a free communication with the general cavity. No effort at adhesion. I carefully cleansed the part locally, left the incision wide open, packed with gauze and did not irrigate. Patient died in forty-six hours with symptoms indicating septic peritonitis. No post mortem.

Remarks.—Without discussion we can assign this case to the category of immediate infection from within with the *Bacillus coli communis.*

Case VI.—Mrs. H. Diagnosis of fibroid tumor with some acute complication, probably torsion of pedicle and consequent peritonitis. When the peritoneal sac was opened a quantity of sero-hæmorrhagic fluid escaped. The tumor, larger than an adult's head was black, almost gangrenous. It had rotated on its pedicle, completely strangulating circulation. There was no accessory adhesion for nutrition, hence death of the mass was inevitable. The parietal and visceral peritonæum throughout was of a deep red color, flakes of fibrino-purulent material were deposited here and there. Supravaginal hysterectomy, with ventral fixation of stump, irrigation and drainage completed the operation. Uninterrupted recovery. Unusual good health since.

Remarks.—This patient, although the subject of a large uterine fibroid had enjoyed the most remarkable health. There was no insanity here, Dr. Price, no pus tubes, but the slender pedicle in an unfortunate moment became twisted. Sero-fibrinous peritonitis followed. In this, as in Case III, the germs of infection were invited by the favorable condition of the tissue.

Case VII.—Mr. B. Patient of Dr. T. G Shannon. Taken with appendicitis Friday night at five o'clock. Case proved to be a very rapid one. I first saw him Sunday at 2 P. M. His rapid pulse, low temperature, greatly tympanic abdomen, rigid muscles, thoracic respiration, and Hippocratic countenance bore convincing evidence that perforation had occurred and that he was laboring under the toxæmia of general peritonitis. Immediate operation was determined upon. Peritoneal cavity was found full of sero-purulent matter. There were no adhesions nor fibrinous deposit, the septic material was generally disseminated throughout the cavity. Irrigation and multiple incision for drainage were employed. Patient did remarkably well until the end of the third day. He grew suddenly worse, abdomen became greatly distended, and he died with all the symptoms of sepsis.

Remarks.—I shall only remark upon this case to express the belief that I committed an error. Had I made multiple incision after the method of Witzwell, had I incised the distended bowel, evacuated its contents and irrigated the intestine, the efficiency of which procedure was accidentally discovered by Reibel in 1883, had I more thoroughly irrigated the general cavity, the patient's chances for recovery would have been greatly enhanced. He died of general purulent peritonitis produced by immediate infection from within.

Case VIII.—Mr. S. Patient of Dr. W. G. Black. Eighteen months ago I operated on this man for appendicitis ; found quite a collection of pus and a rotten appendix. Ligated stump but did not invert it into cæcum. The silk ligature came away, the patient made a good recovery. On August 31st he had a chill followed by high temperature, excessive nausea, vomiting and constipation. I saw him September 1st at 11 P. M. Advised immediate operation. He refused to be moved to the hospital, and we decided to defer it until the following morning. At that visit all symptoms appeared relieved and we temporized. On Wednesday, September 3d, while carefully examining the abdomen, using I am quite sure, but little force, he complained of a sudden pain in his penis and a great desire to pass his urine. The pain was intense. He quickly passed into a condition of

extreme shock. His pulse became almost imperceptible, thinking that rupture of the abscess into the bladder had occurred, a catheter was passed with negative result. He did not react from the shock for several hours. Indeed so ill did he seem to be that I abandoned all thought of his reviving sufficiently to justify surgical interference. During Thursday the 4th his condition was very bad. Thursday night he rallied, his pulse became stronger, though the belly was still very tympanitic. He seemed to be in a fair condition the next morning and operation was determined on. The stomach was well washed out before giving the anæsthetic as a preparatory measure. The abdomen was freely opened in the median line, a quantity of fœtid purulent matter, at least a quart in quantity gushed forth. It came from every part of the cavity, from diaphragm to Douglas' pouch. I washed and rewashed the intestines, especial care being given to the omentum ; and fearing insufficient irrigation I made a second lateral incision at the site of the old operation. Carried it well back so as to drain the lumbar region. Gallons of hot sterilized water were used in this cleansing process. Ineffectual search was made for the point of perforation. The old adhesions about the caput coli so fixed that gut that I could not discover exactly the source of the infection, and I must confess that I was not very diligent in my search. The condition of the cavity did not warrant hope of recovery. I completed the operation with the one idea that if anything would purify him he should die with clean insides. Strange to relate I was repeatedly assured during the operation by Dr. Fort, the anæsthetizer, that the general condition of the patient was as good, if not better, than when first put on the table. Free gauze drainage and the ordinary dressings finished the operation. There is nothing more to say. His bowels moved that night and continued to act freely. He is well today, now ten weeks since the operation.

Remarks.—I am quite well aware that the report of quarts of pus being evacuated from the peritoneal cavity will be accepted *cum grano salis.* One of our distinguished fellows is on record as skeptical upon this point, yet I aver that in this case the pus bathed every abdominal viscus. There was no adhesion except those remaining from preceding inflammation. Perhaps the opponents of irrigation could have cleaned this cavity by dry mops. I did it effectually with water and under similar circumstances should employ the same treatment with renewed confidence. The recovery of this case of general purulent peritonitis was due entirely to the free incision, thorough irrigation, and ample drainage.