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## THE ETIOLOGY AND THE CLASSIFICATION OF PERITONITIS.

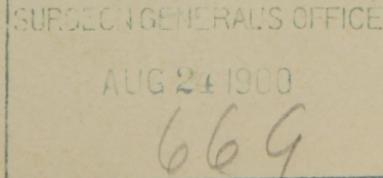
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THE causes of peritonitis have been investigated in various ways. The condition, as found in man, at surgical operations and at the autopsy-examinations, has been studied anatomically and bacteriologically. Numerous experiments have been made upon the lower animals with the view of reproducing the disease encountered in human beings; and there is at present amassed a number of important data that throw much light upon the conditions under which acute inflammations of the peritoneum tend to arise.

So far as I am aware, the only large series of observations in which systematic bacteriologic examinations were carried out has been published by Tavel and Lanz.<sup>1</sup> Their material had the advantage of being obtained at surgical operations. Of course, the literature contains many reports of examinations of single and several cases; but the advantages to be derived from the study of a large number of cases of peritonitis under quite uniform conditions are considerable as compared with the results to be achieved by a comparison of those reported in the literature. This consideration will be found of importance when we attempt to answer the question whether the bacterial flora of the inflamed peritoneum bears any definite relation to the several conditions under which the inflammation arises.

<sup>1</sup> Ueber die Etiologie der Peritonitis. Mittheilungen aus Kliniken und medizinischen Instituten der Schweiz, 1893.



The material at my command was derived exclusively from postmortem examinations. It has been thought that this fact might make the results less reliable and convincing than if it had been obtained from the living subject. I do not think that we are prepared to give a final answer to this question at the present time; indeed, I am of the opinion that the complication supposed to be introduced by this factor may have been overestimated. This belief is based upon the fairly sharp lines of demarcation that come out in a study of the several classes of peritonitides.

I am constrained to believe that the importance of the cases presented depends in part upon the fact that in their bacteriologic study plate-cultures were always employed. By this procedure mixtures of bacteria are separated, so that the relative numbers of the different kinds present may be estimated, and the weakly and slightly growing forms are not quickly overgrown and obscured by their more vigorous associates. The cases are taken from the pathologic records of the Johns Hopkins Hospital. It will be noted that all forms of tuberculous peritonitis have been omitted from consideration.

At the first glance it would seem improbable that the mere study of the bacteriology of fatal cases of peritonitis could be held capable of throwing additional light upon the etiology of the disease. Indeed, it is by no means certain that any essential advance can be made along these lines. Medical literature contains the records of many cases of peritonitis studied in this way by different observers, and we have already begun to associate certain microorganisms with certain kinds and sources of inflammation. Notwithstanding this fact it has seemed to me that a number of distinctions can be drawn more closely than has yet been done. This conclusion has been borne in on me by the study that forms the basis of the present paper.

One has not to go very far into the earlier literature of experimental peritonitis to be impressed with the fundamental importance of the work of Wegner and Grawitz and later Halsted, Pawlowsky, and others. This work has defined accurately the conditions that permit of the development of peritonitis in animals. Grawitz has, moreover, shown how nearly allied are the results thus obtained and the natural conditions found in man. The conceptions brought forward by Grawitz need very little alteration to make them fit into the scheme that we should propose to-day.

Grawitz<sup>2</sup> divides peritonitis into primary and secondary forms. The first agrees with the so-called "idiopathic" or "rheumatic" inflammation of the peritoneum. Grawitz follows Leyden<sup>3</sup> in regarding all these as provoked by microorganisms; but he expresses more definitely the fact that besides the pyogenic bacteria (*causa proxima*) some favoring condition (*causa occasionalis*) is demanded. The latter he finds in wounds communicating with the abdominal cavity or the accumulation of fluid in such quantities that it cannot be absorbed in the natural manner and thus tends to stagnate in this situation. The pyogenic microorganisms may be introduced directly (through the wound, upon a trocar in paracentesis) or be brought by the blood-current or the lymph-current. Secondary peritonitis is a more common affection and occurs under a great variety of conditions, the pyogenic bacteria being introduced by contiguity. This group includes instances of puerperal infection, gangrene, and perforation of the intestine, etc.

If we examine critically the cases of primary peritonitis cited by Grawitz we shall have to dissent from a portion of his conclusions regarding what belong in this

<sup>2</sup> Charité Annalen, xi, 1886.

<sup>3</sup> Cited by Grawitz, *op. cit.*

class. His first case, for example (diphtheric angina, ovarian abscess, peritonitis), is really of secondary nature. The reasons for considering it primary is stated to be the existence, in a recently ruptured Graafian follicle, of a *locus minoris resistentiæ*, in which the bacteria entering from the throat found an opportunity to develop, producing an abscess, which later infected the peritoneal cavity. Of great interest are the several cases of chronic renal, cardiac, and hepatic disease, associated with ascites, in which peritonitis developed. The infection-atrium was sometimes made out—an osteomyelitic focus; diphtheric dysentery; a leg-ulcer, and sometimes it was not discovered. Indeed, these cases, in which chronic disease preexisted, are alone, of the primary examples, convincing. The other examples of primary peritonitis given are either unverified or of secondary origin. Bacteriologic examinations seem not to have been made or they are too imperfect to help us at this time. This is true of all the cases reported by Grawitz.

The cases that are classed as secondary belong clearly to this group. They are such as leave no doubt that the infectious microorganisms reached the peritoneum from a neighboring diseased viscus.

There can, I think, be little doubt that the subject of the etiology of peritonitis has been much cleared up by the work of Grawitz and his demonstration of the essential identity of the conditions that lead to it in human beings with those discovered in experiments upon animals. Grawitz did not, however, consider the subject in its entirety; nor did he or Leyden fully appreciate the significance of the cases of primary peritonitis that they so accurately describe. It is only because in the lapse of time since his writings up to the present some additional points of view have been secured that I feel justified in presenting the matter from the human pathologic side again.

Tavel and Lanz have, along with other things, attempted to answer the question whether peritonitis is a single infection or a multiple infection. Their conclusion is that the hematogenous varieties are mono-infectious, while those that proceed continuously from a diseased organ are usually poly-infectious. They found the majority of cases of circumscribed and diffuse peritonitis to be of the latter kind, although in both mono-infection was encountered. It may be observed that Tavel and Lanz tend to doubt their four cases of diffuse peritonitis in which a single microorganism was found. They are inclined to regard these as chemical forms, a class of which they make much, in which the bacteria played little part and perhaps entered secondarily.

Tavel and Lanz<sup>4</sup> have adopted a rather complex etiologic scheme of classification in which each portion of the gastro-intestinal tract, from the stomach throughout, is taken separately, and special kinds of peritoneal infection are derived from the gall-bladder and liver, the kidneys and urinary bladder and the female genitalia, while still other forms are supplied by operative procedures and hematogenous infections.

The statistics upon which the present report is based comprise 106 cases of peritonitis, which came to autopsy in the Johns Hopkins Hospital and in which bacteriologic studies were carried out, including examination of cover-glass preparations and the making of aerobic cultures. Only exceptionally were anaerobic cultures made. The isolated microorganisms were tested upon the different culture-media and identified as far as possible. Any discrepancies between the appearances observed upon cover-slips and obtained in cultures were noted. Only rarely were the bacteria isolated tested for pathogenesis.

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<sup>4</sup> *Op. cit.*, p. 7.

It soon became evident that several classes of peritonitis could be distinguished. If it were desirable to retain the older names these might be divided into primary and secondary forms. Practically all were bacterial in origin; in our experience chemical peritonitis is of extremely rare occurrence.

By secondary peritonitis we should understand such conditions as follow operations upon the peritoneum or contained viscera, and those in which the abdominal cavity becomes inflamed through the mediation of diseased contained viscera. The numerous experiments made upon animals and many observations upon man, tend to prove that pathogenic microorganisms may find their way into the healthy peritoneum and even remain there for a time without setting up inflammation. Indeed, Tavel and Lanz think that they have shown that only an already inflamed peritoneum can be provoked to peritonitis through the agency of pathogenic microorganisms. For this reason I prefer to call all such inflammations in which the microorganisms are brought by the blood-current or the lymph-current, without the mediation of some diseased neighboring organ or part, as primary. Some metastatic forms of peritonitis would then be primary in nature; and as it is probable that under normal conditions the same microorganisms might have reached this serous cavity without setting up inflammation it becomes necessary to inquire into the cause for this difference.

By primary peritonitis, according to this etiologic definition, is understood a condition in which an inflammation, usually diffuse, of the serous cavity takes place without the mediation of any of its contained organs, and independently of any surgical operation upon these parts. Such a primary peritonitis may arise as an independent affection, or it may develop in the course of infectious processes in distant parts of

the body. The microorganisms associated with them, and to which they are chiefly due, may be brought by the blood-current or the lymph-current. It is conceivable that they may wander through the intact intestine, although convincing proof has yet to be supplied in support of this possibility. The portal of entry of the bacteria into the circulation may be at any distance from the peritoneum; but inflammations of this cavity that arise by extension, as in certain phlegmonous infections of the abdominal walls and perhaps certain acute pleurisies, are not included in this definition.

Of the 106 cases studied, 12 presented the characters of primary peritonitides. These cases will be recognized as corresponding in part with the idiopathic form of some writers. The protocols are abstracted so as to indicate the associated pathologic conditions.

- No. 2.<sup>5</sup> Healed dysentery; chronic nephritis; sero-fibrinous peritonitis. Microorganisms: Staphylococcus aureus; streptococcus.
- “ 364. Chronic nephritis; sero-fibrinous peritonitis. Microorganism: Proteus vulgaris.
- “ 482. Chronic endocarditis; heart-hypertrophy. Circumcision; acute endocarditis. General serositis. Sero-purulent peritonitis. Microorganisms: Streptococcus pyogenes; B. coli com.
- “ 515. Cirrhosis of the liver; chronic endocarditis; arteriosclerosis. Acute endocarditis; general serositis. Fibrino-purulent peritonitis. Microorganism: Streptococcus pyogenes.
- “ 517. Arteriosclerosis; hypertrophy of the prostate gland; purulent cystitis. Fibrinous peritonitis; very slight exudate. Microorganism: Staphylococcus albus.
- “ 519. Cirrhosis of the liver; chronic nephritis. Sero-fibrinous peritonitis. Microorganism: Micrococcus lanceolatus.
- “ 534. Chronic nephritis; cirrhosis of the liver; arteriosclerosis; chronic endocarditis; shallow ulcers of the large intestine. Sero-fibrinous peritonitis. Microorganism: Bacillus pyocyaneus.
- “ 542. Arteriosclerosis; carcinoma of the stomach. Sero-

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<sup>5</sup> The numbers given refer to the autopsy numbers in the records of the Johns Hopkins Hospital.

- purulent peritonitis. Microorganism: Streptococcus pyogenes.
- No. 555. Carcinoma of the stomach and general carcinosis. Sero-fibrinous peritonitis. Microorganism: Unidentified bacilli.
- “ 570. Chronic nephritis; chronic endocarditis; heart-hypertrophy. Fibrino-purulent peritonitis. No bacteria found.
- “ 577. Chronic endocarditis; sero-hemo-fibrinous peritonitis. No bacteria found.
- “ 626. Amyloid disease; cirrhosis of the liver. Fibrino-purulent peritonitis. Microorganism: Streptococcus pyogenes.

An analysis of the foregoing cases brings out the following facts: In all there was previous chronic disease, at one time involving one important organ, at another several organs. The peritoneum was the only serous membrane affected in 10 cases, while in two instances the pleura and the pericardium were involved at the same time. In 2 of the cases no bacteria were demonstrable upon films or in cultures. These would doubtless be regarded as examples of chemical peritonitis by Tavel and Lanz. I think that more evidence than we have at present is needed before this explanation can be accepted. There is at least a possibility that microorganisms were present at an earlier time. The microorganisms represented are both cocci and bacilli, the former predominating. In 9 instances single infection and in 1 instance multiple infection occurred. The organisms found were streptococcus pyogenes 5 times, 4 times alone and once in association with the bacillus coli com.; staphylococcus aureus and albus twice; while the micrococcus lanceolatus, bacillus proteus, bacillus pyocyaneus and an *unidentified* bacillus occurred once each.

The portals of entry were several, including a circumcision-wound, acute endocarditis and slight lesions of the intestinal mucosa.

If we turn our attention to secondary forms of peri-

tonitis we shall find not only that these are far more common than the primary, but also that at least two classes of cases can be distinguished. These classes depend upon the mode of entrance of the pathogenic microorganisms, rather than upon their kind or the character of the inflammatory reaction. Yet there are differences in the kinds of bacteria found in each, or, at least, differences of predominance and of combination.

The first class of secondary peritonitides that I shall consider may be designated "exogenous peritonitis." In it the infectious microorganisms have, in large part, entered from without. They actually are examples of wound-infection. This fact is brought out by the clinical histories of the cases and is supported by the bacteriologic findings, which agree with those of ordinary surgical infections. In our series of 106 cases I found 34 belonging to this group :

- No. 45. Exploratory celiotomy; carcinoma of the liver. Suppurating abdominal wound. Microorganism: *Staphylococcus aureus*.
- " 52. Myomectomy. Microorganism: *Staphylococcus aureus*.
- " 89. Carcinoma of the kidney. Celiotomy. Microorganism: *B. coli com.*
- " 108. Ovariectomy. Microorganism: *Staphylococcus aureus*.
- " 144. Ovariectomy. Microorganism: *Staphylococcus aureus*.
- " 160. Ovariectomy. Suppurating external wound. Microorganism: *Staphylococcus aureus*.
- " 214. Chronic peritonitis; exploratory celiotomy. Microorganism: *B. coli com.*
- " 218. Ovariectomy. Microorganism: *Staphylococcus aureus*.
- " 219. Chronic peritonitis; celiotomy. Microorganisms: *Micrococcus lanceolatus*; coarse liquefying bacillus.
- " 221. Ovariectomy. Microorganism: Unidentified peptonizing bacillus.
- " 243. Ovariectomy. Microorganism: *Streptococcus pyogenes*.
- " 273. Hysterectomy for carcinoma uteri; ligated ureter; Microorganism: *Staphylococcus albus* (perhaps epidermidis.)

- No. 277. Ovariectomy. Microorganisms: *Staphylococcus aureus* and *streptococcus pyogenes*.
- “ 288. Myomectomy. Slight peritonitis. Peritoneum sterile. *B. coli* com. in organs.
- “ 292. Ovariectomy. Microorganism: *Staphylococcus albus*.
- “ 380. Myomectomy. Purulent and hemorrhagic peritonitis. Microorganism: *Staphylococcus aureus*.
- “ 382. Ovariectomy. Microorganism: *Staphylococcus aureus*.
- “ 383. Ovariectomy. Microorganism: *Staphylococcus aureus*.
- “ 385. Hystero-myomectomy. Fibrinous peritonitis. Microorganism: *Staphylococcus aureus*.
- “ 446. Perforating carcinoma of the uterus. Fibrino-purulent peritonitis. Microorganisms: *B. pyocyaneus*; *streptococcus pyogenes*.
- “ 487. Ovarian cystoma. Tapping abdomen. Sero-fibrinous peritonitis. Microorganism: *Streptococcus pyogenes*. Few bacilli in films; did not grow.
- “ 549. Traumatic rupture of the bladder. Celiotomy. Fibrinous peritonitis. Microorganisms: *Streptococci*; *B. coli* com.
- “ 635. Chronic pelvic peritonitis; celiotomy; fibrinous peritonitis. Microorganism: *Micrococcus lanceolatus*.
- “ 644. Fistula between bladder and abdomen; chronic tuberculous peritonitis; acute fibrinous peritonitis. Microorganism: In cultures, *streptococcus pyogenes*.
- “ 653. Sloughing myoma uteri; celiotomy. Fibrino-purulent peritonitis. Microorganism: *Streptococcus pyogenes*.
- “ 666. Hysterectomy; fibrino-purulent peritonitis. Microorganism: *Staphylococcus aureus*.
- “ 671. Hysterectomy; fibrino-purulent peritonitis. Microorganisms: *Streptococcus* and *staphylococcus albus*.
- “ 700. Sloughing myoma uteri; celiotomy; purulent peritonitis. Microorganisms: Large unidentified coccus; films, bacilli resembling *B. aërog. capsulatus*.
- “ 812. Hysterectomy; infection of an external wound; fibrino-purulent peritonitis. Microorganisms: *M. lanceolatus*; *B. coli* com.
- “ 876. Perforating carcinoma uteri. Microorganisms: *Streptococcus pyogenes*. In films a few bacilli.
- “ 895. Ovariectomy for carcinoma; fibrino-purulent peritonitis. Microorganisms: *B. coli* com.; in films a few cocci.

- No. 914. Hysterectomy; fibrinous peritonitis. Microorganism: *Staphylococcus pyogenes aureus*.  
 " 958. Suprapubic cystotomy; fibrinous peritonitis. Microorganisms: *Streptococci*; *B. pyocyaneus*; *B. coli com.*  
 " 1015. Suprapubic cystotomy; fibrino-purulent peritonitis. Microorganisms: *Staphylococcus aureus*; *B. coli com.*

This group comprises 34 cases in all. Of these 25 are of the nature of single and 9 of multiple infections. The bacteria concerned in their causation are chiefly the pyogenic cocci, either alone or, more rarely, associated with bacilli. The following tables give the microorganisms found and their combinations:

TABLE OF MICROORGANISMS FOUND IN CASES OF EXOGENOUS PERITONITIS.

	Total No. Cases.	Alone.	Combined.
* <i>Staphylococcus aureus</i> .....	15	12	3
" <i>albus</i> .....	3	2	1
<i>Streptococcus pyogenes</i> .....	10	5	5
<i>Bacillus coli communis</i> .....	7	2	5
<i>Micrococcus lanceolatus</i> .....	3	1	2
<i>Bacillus proteus</i> .....	1	0	1
<i>Bacillus pyocyaneus</i> .....	2	0	2
Unidentified organisms.....	3	0	0

TABLE OF COMBINATIONS.

	No. Cases.
<i>Staphylococcus aureus</i> and <i>streptococcus</i> .....	1
" <i>albus</i> "    ".....	1
"    "    " <i>B. proteus</i> .....	1
"    "    " <i>B. coli com.</i> .....	1
<i>Streptococcus</i> and <i>B. coli com.</i> .....	1
" <i>B. pyocyaneus</i> , and <i>B. coli com.</i> .....	1
"    and <i>B. pyocyaneus</i> .....	1
<i>Micrococcus lanceolatus</i> and <i>B. coli</i> .....	1
"    "    "    liquefying bacillus.....	1

What is especially striking in these tables is the similarity of the bacterial flora with that of ordinary surgical infections. The only important difference is afforded by the part played by the *Bacillus coli*. This feature is, however, at once explained when the particular cases in which this organism occurred are considered.

In 4 cases there was inflammation of the genito-urinary tract prior to operation, in 2 the operation (autopsies 958 and 1015) being directly upon the urinary bladder. As this bacillus is so commonly found in inflammations of these parts its entrance into the peritoneum is easily accounted for. Of the remaining three cases two were ovariectomies for carcinoma. It is extremely probable that adhesions to the intestinal coils, which necessitated handling of and perhaps more or less injury to these parts, opened the way for the penetration of this organism and for the provocation of an inflammatory reaction.

The cases in which the bacillus coli was the sole organism found in the exudate should be mentioned especially. There has been a radical change of opinion regarding the pyogenic activities of this organism in peritonitis. Undoubtedly the great majority of cases in which it is found are examples of multiple infection. That the bacillus coli may, however, be the only bacterium present in the inflammatory exudate must be admitted in the light of our present knowledge. In this group one certain instance only has been encountered (autopsy 214). In that case an exploratory celiotomy had been performed, the condition found being a chronic peritonitis. The exact character of the acute process is not given in the notes. The second instance (autopsy 89) is less convincing, as it dates from a period in which the same minute care was not exercised in searching for other bacteria, especially streptococci. In it there was also a general invasion of the organs with the bacillus coli communis.

The next class of secondary forms of peritonitis may, I think, be termed "endogenous peritonitis." In it the bacteria come in part or wholly from the intestinal tract. This fact determines them to be chiefly multiple infections. The variety of microorganisms is greater than in the other classes. In our experience, and the same

is generally true, the endogenous form is the commonest form of infection encountered in surgical practice and at autopsy. It is quite certain that our examination by cultures and films give us a more imperfect result in this group than in the others, for the reason that not all of the intestinal bacteria are capable of cultivation upon artificial media. This fact has been dwelt on by Tavel and Lanz.

This class comprises 60 cases, as follows :

- No. 55. Myoma uteri; volvulus of the intestine. Celiotomy. Fibrino-purulent peritonitis. Gelatin-culture sterile. No others made.
- “ 84. Ovariectomy; myoma uteri; celiotomy; wound of the intestine. Fibrino-purulent peritonitis. Bacillus coli only isolated.
- “ 176. Strangulated hernia; resection of the intestine. Bacillus coli alone isolated; present in large numbers.
- “ 181. Extra-uterine pregnancy; perforation of the vermiform appendix; celiotomy; fibrino-purulent peritonitis. B. coli obtained in great numbers at operation and autopsy.
- “ 229. Typhoid fever; perforation of the intestine. Microorganisms: B. typhi and coli com.; staphylococcus aureus; streptococcus and unidentified bacillus.
- “ 234. Amebic dysentery; perforation of the intestine; hepatic abscess. B. coli in pure culture.
- “ 250. Diphtheric dysentery; fibrino-purulent peritonitis. No perforation. Microorganism: Micrococcus lanceolatus.
- “ 251. Removal of ovaries, oviducts and vermiform appendix. Microorganism: Streptococcus.
- “ 255. Strangulated hernia; circular suture of the intestine. Fibrinous peritonitis. Microorganisms: In cultures, streptococcus; in films, a small number of bacilli also.
- “ 275. Ovariectomy; volvulus; ulceration of the intestines. Microorganism: Staphylococcus albus (perhaps epidermidis) cultivated from wound and peritoneum.
- “ 295. Perforation of the vermiform appendix. Microorganisms: B. coli and streptococcus.
- “ 365. Typhoid fever; perforation. Microorganisms: B. coli and streptococcus.
- “ 371. Perforation of the cecum; carcinoma of the uterus. Infection of the abdominal wound. B. coli isolated from wound and peritoneum.

- No. 372. Typhoid fever; perforation of the vermiform appendix. Microorganisms: *B. coli* com.; *B. aerogenes capsulatus*; streptococcus.
- “ 397. Paresis of the intestine; hemorrhagic peritonitis. Microorganisms: *B. pyocyaneus* and *B. coli* com.
- “ 400. Intussusception; celiotomy. Streptococci and liquefying bacillus.
- “ 408. Resection of the pylorus; fibrino-purulent peritonitis. Microorganisms: Streptococcus and *B. coli* com.
- “ 411. Resection of the rectum for carcinoma; recurrence. Fibrino-purulent peritonitis. Microorganism: Streptococci.
- “ 419. Suppurative appendicitis; purulent peritonitis; celiotomy. Microorganisms: *B. coli* and *M. lanceolatus*.
- “ 422. Perforating carcinoma of the stomach; purulent peritonitis. Microorganisms: Streptococci and *B. coli*.
- “ 431. Perforation of a tuberculous ulcer of the intestine; fibrino-purulent peritonitis. Microorganisms: Streptococci and *B. coli*. Fine bacilli in films failed to grow.
- “ 454. Typhoid fever; perforation of the vermiform appendix. Streptococcus in pure culture.
- “ 468. Perforation of the vermiform appendix; fibrino-purulent peritonitis. Microorganism: *B. pyocyaneus* and *coli* com.
- “ 500. Vaginal hysterectomy; hemorrhagic infarction of the small intestine; hemorrhagic and fibrinous peritonitis. Microorganisms: *Staphylococcus albus*; *B. coli*; orange sarcina.
- “ 502. Traumatic rupture of the intestine; fibrino-purulent peritonitis. Streptococcus; *staphylococcus aureus*; *B. coli*. Other bacilli on cover-slips.
- “ 529. Diffuse carcinoma of the stomach; peritoneal metastasis; no perforation; serous peritonitis. Streptococci in general cavity; *B. coli* also in pelvis, where fibrin was present.
- “ 532. Chronic peritonitis; ovarian abscess; perforating ulcer of the intestine; sero-fibrinous peritonitis. Microorganisms: *B. pyocyaneus* and *coli* com.
- “ 548. Strangulated hernia; sero-fibrinous peritonitis; celiotomy. Cultures negative.
- “ 557. Typhoid fever; perforation; fibrino-purulent peritonitis. Microorganisms: Streptococci; unidentified bacilli.
- “ 569. Gangrene of the vermiform appendix; fibrino-purulent peritonitis. Microorganisms: *B. coli* and unidentified cocci.
- “ 571. Typhoid fever; perforation; sero-purulent periton-

- itis. Microorganisms: *B. coli*; streptococci; *B. aërogenes capsulatus*.
- No. 591. Perforation of the vermiform appendix; fibrino-purulent peritonitis. *B. coli* alone found.
- “ 622. Perforating carcinomatous ulcer of the duodenum; fibrino-purulent peritonitis. *Bacillus aërogenes capsulatus*.
- “ 637. Hernia; operation; necrosis of the mucous membrane of the intestine; perhaps  $\text{HCl}_2$  poisoning. Microorganisms: Streptococci and *B. coli*.
- “ 649. Thrombosis of the portal and mesenteric veins; infarction of the intestine; sero-fibrino-purulent peritonitis. Microorganisms: *B. coli*; *aërogenes capsulatus*.
- “ 660. Strangulated hernia; necrosis of the intestine; celiotomy. Microorganism: Streptococcus.
- “ 672. Artificial anus; fibrino-purulent peritonitis. *B. coli*.
- “ 674. Chronic adhesive peritonitis; artificial anus; fibrinous peritonitis. *B. coli* in culture; cocci in films.
- “ 730. Intestinal obstruction; celiotomy. Microorganism: *Staphylococcus aureus*.
- “ 732. Strangulated hernia; gangrene of the intestine; celiotomy. *Staphylococcus albus* only isolated.
- “ 735. Amebic dysentery; necrosis of the vermiform appendix; fibrino-purulent peritonitis. Microorganisms: *M. lanceolatus*; *B. coli* com.
- “ 737. Intestinal anastomosis; fibrinous peritonitis. *B. coli* in cultures; diplococcus in films.
- “ 747. Suture of the gall-bladder. *B. coli* alone isolated.
- “ 752. Intestinal anastomosis. Microorganisms: *Staphylococcus aureus* and *B. coli*.
- “ 803. Appendicitis; fibrino-purulent peritonitis; celiotomy. Streptococcus alone isolated.
- “ 814. Appendicitis; fibrino-purulent peritonitis; celiotomy. *B. coli* in cultures; other bacilli and cocci in films.
- “ 825. Appendicitis; fibrino-purulent peritonitis. On films cocci and bacilli. Only *B. coli* in cultures.
- “ 837. Strangulated hernia; fibrinous peritonitis. *B. aërogenes capsulatus*.
- “ 838. Gangrene of the intestine; fibrino-purulent peritonitis. Microorganisms: various bacteria on cover-slips; in cultures *B. aërogenes capsulatus*.
- “ 850. Thrombosis of the mesenteric veins; infarction of the intestine. Fibrino-purulent peritonitis. *Proteus vulgaris* in pure culture.
- “ 854. Teratoma of the ovary perforating into the rectum; purulent peritonitis; celiotomy. *B. proteus* in cultures; cocci in films.

- No. 889. Typhoid fever; perforation. Microorganisms: Streptococci; *B. typhosus*.
- " 869. Periproctal abscess, fibrino-purulent peritonitis. Pelvic abscess, *B. coli* and streptococci; general peritoneum, streptococci.
- " 909. Perforating tuberculous ulcer of the intestine, fibrino-purulent peritonitis. Films: various bacteria; culture: *B. proteus*.
- " 911. Appendicitis; perityphilitis; celiotomy. Abscess. *Streptococcus pyogenes*; *B. aërogenes caps.*
- " 947. General melanotic sarcomatosis; necrosis of the intestine; celiotomy. Bacilli in films; could not be cultivated.
- " 948. Perforating ulcer of the stomach; purulent peritonitis. Microorganisms. *B. aërog. caps.* *B. coli com.*
- " 954. Hysterectomy; removal of the vermiform appendix; perforation of the sigmoid flexure; fibrinous peritonitis. Microorganisms: *B. coli com.*; streptococci.
- " 974. Perforating ulcer of the intestine; purulent peritonitis; celiotomy. *B. coli com.* alone isolated.
- " 1028. Perforation of the vermiform appendix; celiotomy; fibrino-purulent peritonitis. Microorganisms: *B. coli*; streptococci.

Of the 60 cases in this list two only (autopsies 55 and 548) yielded negative bacteriological results. As No. 55 was tested in gelatin only, and no record was made of the examination of films, it should be excluded. The number of cases in which positive results were obtained is therefore 58. Single infections occurred in 21 and multiple infections in 37 instances. The following tables give the kinds and combinations of microorganisms met with:

TABLE OF BACTERIA FOUND IN CASES OF ENDOGENOUS PERITONITIS.

Bacteria.	No. of Cases.	Alone.	Combined.
<i>B. coli com.</i> .....	47	9	38
<i>Streptococcus pyogenes</i> .....	39	7	32
<i>Staphylococcus albus</i> .....	4	2	2
" <i>aureus</i> .....	3	1	2
<i>Micrococcus lanceolatus</i> .....	4	1	3
<i>Bacillus proteus</i> .....	4	2	2
" <i>aërogenes caps.</i> .....	8	2	6
" <i>pyocyaneus</i> .....	3	0	3
" <i>typhosus</i> .....	3	0	3
Unidentified .....	3	0	3

## TABLE OF COMBINATIONS.

	No. Times.
Streptococcus and B. coli com.....	16
“ “ B. aërog. caps. and B. coli .....	2
“ “ and B. aërog. caps.....	1
“ “ “ staphylococcus aureus.....	1
“ “ “ B. typhosus.....	2
“ “ S. aureus, B. typhi, proteus and coli.....	1
“ “ and B. proteus .....	1
“ “ “ unidentified organism.....	1
Bacillus coli and M. lanceolatus.....	3
“ “ “ B. pyocyaneus .....	3
“ “ “ B. aërog. caps.....	2
“ “ “ S. aureus, and B. aërog. caps.....	1
Staphylococcus albus and orange sarcina .....	1
“ “ “ unidentified organism.....	1

In comparing this group with the secondary form of exogenous origin, several differences become apparent. Not only are single infections relatively more infrequent with the endogenous variety, but the microorganisms found differ widely. The streptococcus in the latter plays the part of the staphylococci in the former, and the bacillus coli communis, as might be expected, assumes a much more important role. I do not intend to renew the discussion as to whether, after all, the bacillus coli may not in all cases have been associated at some stage of the pathological process with more usual pyogenic organisms. The question has been answered both ways by the studies of Barbacci,<sup>6</sup> who found in his experiments upon dogs that the colon-bacillus often overgrew the pyogenic cocci, and by French writers, Laruelle and Vendrick,<sup>7</sup> and in this country by Welch,<sup>8</sup> who believes in a primary bacillus-coli peritonitis. My own opinion is in favor of conceding this bacillus the possibility of acting as a pyogen, just as we have come to do for the bacillus typhosus.

What is further remarkable is the small part that the pathogenic staphylococci seem to play in the cases of

<sup>6</sup> *Centralbl. f. Pathologie*, iii, p. 129.

<sup>7</sup> Quoted from Tavel and Lanz, *op. cit.*, p. 5.

<sup>8</sup> *Medical News*, Philadelphia, 1891.

intestinal origin. That this fact may have clinical significance is not improbable, especially in view of the fact that in several of the tabulated cases, a general streptococcal infection (bacteremia) had taken place.

The observation that the cases of primary or idiopathic peritonitis in our series were all examples of terminal infection suggests the question as to how far chronic disease may have been responsible for the results in the exogenous secondary infections. I have considered, in a previous communication,<sup>9</sup> the relations that exist between chronic disease and infection; and in view of the resistance offered to infection by the normal peritoneum it is necessary to recognize general as well as local predisposing causes. Of course, in instances of chronic disease it is a general rather than a local disturbance that makes these parts unduly vulnerable. It is, to say the least, remarkable that of the 35 cases of exogenous peritonitis in 26 (autopsies 45, 89, 144, 160, 214, 219, 273, 288, 383, 422, 446, 486, 635, 644, 700, 876, 895, 914, 929, 950, 1015) there should have preexisted more or less chronic disease, consisting of malignant tumors, chronic renal and cardiac disease, arteriosclerosis, cirrhosis of the liver, pulmonary tuberculosis, chronic peritoneal tuberculosis, and proliferative peritonitis.

The classification attempted in this paper is etiological only. Further subdivisions are possible in each class, according to the presence or absence of microorganisms, their relative numbers, and especially the kinds of reaction provoked. It remains to be seen whether the limits of the so-called "aseptic" peritonitis of Bumm and the "chemical" peritonitis of Tavel and Lanz have been correctly drawn. Our experience is somewhat at variance with that of the latter writers,

<sup>9</sup> A Statistical and Experimental Study of Terminal Infections, *Jour. Exper. Med.*, Vol. I, 1896, No. 3.

which, however, may be due to the fact that the examinations recorded here were made after death. According, then, to the indications supplied by a rather large material, the primary or idiopathic forms of peritonitis are restricted to a definite and small number of cases of terminal infection, and unless the resistance of the peritoneum is broken down through local lesions or general disturbances this cavity is eminently capable of protecting itself against injurious chemical and living agents. A second variety of peritonitis conducts itself in every way like surgical infections; and the conditions that protect the tissues generally from, or predispose them to, infection may be seen in operation here. Finally, a third variety is dependent upon disease in an intraperitoneal organ that brings pathogenic microorganisms and other extraneous (chemical?) substances directly or indirectly into the abdominal cavity, thus breaking down its resistance and exposing it to infection from within.

