# ANNUAL LECTURES DELIVERED BEFORE THE ALUMNI ASSOCIATION OF THE COLLEGE OF PHYSICIANS AND SURGEONS OF BALTIMORE, APRIL 11TH AND 12TH, 1892

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Annual lectures delivered before the Alumni Association of the College of Physicians and Surgeons of Baltimore, April 11th and 12th, 1892 by W. E. B. Davis



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Two years ago the ALUMNI ASSOCIATION OF THE COLLEGE OF PHYSICIANS AND SURGEONS OF BALTIMORE, with the view of rendering their annual reunion more interesting and profitable, invited Dr. Lewis S. McMurtry, of Louisville, to give two lectures upon the Pathology, Diagnosis and Treatment of Extra-Uterine Pregnancy. Dr. McMurtry's lectures gave so much satisfaction to the members and invited guests that at the succeeding meeting Dr. W. E. B. Davis, of Rome, Ga., one of the most distinguished surgeons of the South, was invited to address the Association. He chose as his subject "Local and General Peritonitis," a subject of vital interest to every practitioner. Dr. Davis's lectures as printed in the following pages form a valuable monograph of Peritonitis from the most modern standpoint.

The subject of the lectures for 1893 has not yet been decided upon. Due notice will be given through the medical press.

# LOCAL AND GENERAL PERITONITIS.[1]

By W. E. B. DAVIS,

President of the Tri-State Medical Society of Alabama, Georgia and Tennessee; Secretary of the Southern Surgical and Gynecological Association; Fellow of the American Association of Obstetricians and Gynecologists; Honorary Member of New York State Medical Society, etc.

*Gentlemen:*—The subject I have selected is a very familiar one. So much has been written on it during the past few years that any further contributions would seem supererogatory, but it must be remembered that a large majority of the profession do not regard this disease as do the abdominal surgeons. Those who are opening the abdomen constantly and seeing its diseases as they are have learned that peritonitis is not a disease distinct, as taught by Bichat, but that it is nearly always due to some well-recognized lesion of the abdominal viscera. A large proportion of the profession, and of those who see these cases first, still regard the disease as a distinct affection, and therefore their treatment is based on this conception of the disease and is usually followed by a fatal issue. Too much praise cannot be given Habershon, who showed so conclusively, by his post-mortems in Guy's Hospital, that a cause could nearly always be found for peritonitis,—that there was a perforation of the appendix, a rupture of an abscess, a leakage from a pus-tube, a perforation of the gall-bladder, of the intestine, or a tubercular disease, or some other well-recognized lesion at the bottom of nearly every case of peritonitis. Of course there are certain constitutional affections which predispose to the disease. Diseases of the kidney may cause peritonitis. We may also have a peritonitis as a result of a malignant growth in the abdomen.

There has been much confusion over the terms septic and simple peritonitis. Especially has the general physician been confused over the teachings of the surgeon, inasmuch as the surgeon too often fails to differentiate between these two diseases and fails

[1] The "Alumni Lectures" for 1892 of the Alumni Association of the College of Physicians and Surgeons, Baltimore.

to teach that they are very different inflammations, due to different causes.

A septic germ may cause a local inflammation which does not go on to pus formation, and on the other hand a simple inflammation, with very slight septic infection, may go on and result in pus formation. We may have salpingitis, due to septic germs passing up through the uterine canal into the tubes, but the germs are in such small number and the tubes in such a healthy condition as to resist suppuration, and there may result inflammation which will simulate a local, simple peritonitis. On the other hand, traumatism to the uterine canal may cause such inflammation that, should there be any septic germs in the tubes from an old inflammation, or infection, that this new inflammation of the uterus will result in suppurative inflammation of the tubes. A simple, irritative, non-suppurative inflammation of the peritoneum may result, or terminate, in a septic peritonitis by producing a *locus resistentiae minoris* in the peritoneum, or by producing paralysis of the intestinal walls, thus allowing septic germs to pass from the intestine into the general peritoneal cavity. Obstruction of the bowel thus gives rise to a septic peritonitis.

In a simple peritonitis there is thrown out an exudation lymph which offers a splendid nidus for the development of septic germs, and in the presence of a simple inflammation a very few septic germs will cause septic inflammation of a very fatal character.

That there may be no confusion over the terms septic and suppurative peritonitis, as used in this paper, it may be well to state that it is conceived that their etiology is the same, and that every septic peritonitis would become suppurative if time permitted; for both are due to the same microbe. Many cases of septic peritonitis are cut short by the power of the organism to take up the germs and destroy them. In other cases the infection is so profound that death is produced in a few hours from toxaemia, before there has been any collection of fluid in the peritoneal cavity; still others die from hemorrhagic peritonitis in from twelve to forty-eight, or even sevnty-two hours, before the fluid has become purulent; but, when life is prolonged till after seventy-two hours, and sometimes after forty-eight, the fluid in the cavity will be purulent.

When quarts and gallons of pus are reported as having been removed from the general peritoneal cavity and recovery followed, I believe that the pus has usually resulted from a local collection

which has ruptured into the general cavity, and the operation has been done before sufficient time had elapsed for this amount of pus to result from the septic inflammatory process in the general cavity. It is easy to understand how a gallon of pus which has been shut off from the general cavity by inflammatory exudations and adhesions, and which has only recently ruptured into the peritoneal cavity, can be removed and recovery follow; and it is not difficult to comprehend how this condition might be mistaken for acute, general, suppurative peritonitis, with a gallon or a quart of pus as a result in the cavity; as the pus, by its irritating properties, will produce an inflammation which would be misleading: but the condition is quite different from what would be had if the pus had been the result of a general inflammation. True, there would be many grave symptoms, but not that extensive local inflammation and fatal toxaemia which would result from a peritonitis which had existed long enough to give rise to so large a quantity of free pus in the cavity.

Pathological and clinical study, combined with bacteriological and experimental research, has demonstrated conclusively that by the time a general peritonitis has become purulent there have resulted such destructive local effects and so profound a general infection that the condition must be considered fatal.

A suppurative, or purulent, peritonitis is usually localized; in other words, a septic inflammation that goes on to pus formation is, as a rule, a localized inflammation, and the pus is limited by inflammatory adhesions so as to protect the general cavity from infection. A septic inflammation that involves the general peritoneal cavity will generally produce death before pus has had time to form, and hence septic inflammation, as a rule, must be localized in order for the patient to survive long enough for pus formation. Fortunately the most frequent class of septic peritonitis is the localized purulent form, which is due to protective adhesions being established before the rupture of the tubes, a perforation of the appendix, the gall-bladder, or of the bowel, etc.

Furthermore, small quantities of fluids will be shut off by rapid formation of protective adhesions when there have been no preparatory adhesions. However, when gas escapes without preliminary protective adhesions a peritonitis of the gravest form usually results, the gas being so diffusible that it rapidly involves the whole cavity. I have seen cases of death from this form where there was no lymph thrown out from the inflammation.

A few months ago I operated on a man at Collinsville, Ala., who had all the symptoms of approaching death from obstruction of the bowel, and found a long appendix with a small opening which had permitted the passage of gas, and as a result there was a diffused peritonitis without the presence of fluid formation in the cavity. I have also noticed this several times in my experiments on animals.

In recent years much has been learned about the etiology and treatment of this disease from bacteriological, experimental and clinical study. The following points have been pretty well settled by this class of work:

A simple peritonitis may produce death from the extent of the inflammation, which is dependent upon the amount of traumatism or of the chemical irritant, and upon the condition of the peritoneum.

A septic inflammation is always produced by pus germs, either directly or indirectly, and it has been shown that in inflammation produced by pus germs, cultures made from the products of this inflammation would produce a similar inflammation in other animals, while the products of a simple inflammation would not produce cultures capable of causing an inflammation.

The peritoneum in a healthy condition can take up a moderate number of septic germs and destroy them, no peritonitis resulting; while a peritoneum weakened by some antecedent trouble would succumb to the same number of germs and a fatal peritonitis would be developed. The number of germs introduced into the cavity with a chemical irritant which would cause the pouring out of lymph, and cause a fatal peritonitis, without a chemical irritant, would be taken up and destroyed.

Much depends upon the condition of the peritoneum and upon the manner in which germs are admitted into the peritoneal cavity. For this reason many cases died from septic peritonitis when it was the custom to use chemical solutions for irrigating the abdominal cavity.

Where there are sufficient germs to produce a general septic peritonitis, if death results in twenty-four to forty-eight hours, there will be a hemorrhagic inflammation. I have often seen this occur in my experiments on animals. If the animal lives more than three days there will be a purulent peritonitis. If it dies in twenty-four hours there will frequently be an intense congestion without much fluid in the cavity, which would show that the in-

fection had been so great that the animal had succumbed early to the poison and shock.

Some of the most rapid forms of infection produce death without a very marked localization, the system being overwhelmed with the septic germs.

A simple inflammation must be very severe in order to produce death. I have seen some very violent simple inflammations recover, and it is just here that the general physician, and also the surgeon, get the treatment of peritonitis confused. The general physician thinks that most cases can be cured by opium and rest, or that all cases that can be cured at all can be cured in this way; while the abdominal surgeon claims, in a general way, that when you get peritonitis you have a disease for the surgeon, and so the confusion goes on and the patient is the sufferer. What is necessary is to convince the general physician of the nature of the disease, and to show him that the surgeon does not consider that in all cases surgical procedures are necessary, but, above all things, that the disease is a surgical disease, and that a surgeon should be sought as a consultant in these cases, and that he should be called early and not after the patient is practically dead. It may be said with truth that the surgeon is to blame, in a large measure, for the general physician's failure to properly regard peritonitis. The contributions on this subject have been too general in their tone. In other words, they have generalized too much and particularized too little. They have not distinguished, nor has there apparently been a great effort to draw the line, between the cases in which surgery should be resorted to and those cases in which it is not necessary.

It is impossible, in many cases, to determine the nature of the inflammation. I recollect a case in point which I saw at Alice Furnace in Birmingham, Ala., three years ago. The man had been caught between the furnace elevator and the wall of the building and mashed severely and then dropped thirty feet on a stone floor. He had retention of urine and involuntary passage of feces for the first twelve hours. After that time his bowels would not move and his legs became very markedly paralyzed. His abdomen became more and more tympanitic until he could scarcely breathe. His pulse on the third and fourth days ran from 135 to 150. It looked as if death was inevitable. By the persistent use of calomel and enemas of salts and glycerine his bowels were finally induced to move several times, and then his