THE FALLACY OF PERITONEAL DRAINAGE

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DURING the first two years of the first World War peritonitis from gunshot wounds of the abdomen caused a mortality of close to 100 per cent. Then British Army surgeons began to close the peritoneum without drainage, after carefully suturing all perforations of the hollow viscera. The mortality immediately dropped to about 50 per cent.

In the quarter of a century since this epochal discovery, relatively few American surgeons have profited by it, as is shown by the almost universal mistreatment of appendiceal peritonitis. Our new army is staffed by these same civilian surgeons. Unless they are aware of the deadly results of drainage in general peritonitis, it seems probable that about one-half of our battle casualties involving the abdomen will result in unnecessary deaths. It is the purpose of this paper to prove the truth of this seemingly extreme statement.

In 1905, Yates[1] proved that the peritoneal cavity could not be drained by any method whatsoever. The drains were always walled off within a few hours. His findings have been confirmed by many investigators and refuted by none. Why then do the majority of writers of the subject advocate drainage in peritonitis? If they are at all familiar with the literature, they know that they advise and attempt the impossible except in the case of walled-off abscesses. Their self delusion in the case of diffuse peritonitis is an example of the perpetuation of a fallacy for nearly half a century after the fallacy was exposed.

In 1897, John G. Clark analyzed 1,700 cases of abdominal section at Johns Hopkins Hospital. He concluded that not only is drainage useless in the great majority of cases in which it had been used, but that it was frequently productive of harm.

In 1908 Robert T. Morris,[2] impressed by Clark's study, reported that in cases of appendicitis with pus and peritonitis he had closed without drainage for about a year with no deaths and no increase of peritonitis.

In 1931, Buchbinder and his associates,[3] experimenting on dogs, produced a widespread peritonitis by opening a loop of bowel and leaving it open for twenty-four hours. They then reopened the abdomen and removed the source of infection by closing the bowel. In fifty-three animals so treated thirty-three were closed without drainage, and nineteen or 57.5 per cent died. In the remaining twenty animals two drainage tubes were inserted, one in the upper abdomen and one in the pelvis; all died.

These experimental results are in conformity with surgical experience. For the sake of brevity only a few typical reports will be quoted.

Haggard and Kirtley[4] quote the experience of Giertz who in patients with purulent appendiceal peritonitis operated upon in the first forty-eight hours, reduced the mortality from 22.2 to 3.5 per cent by primary closure of the peritoneum. These authors also state that the mortality of generalized peritonitis "averages 41.2 in some of our best clinics."

In 1939, Kelly and Watkins[5] reported that of 171 patients treated by drainage forty-four died, 25.7 per cent, while of nineteen patients treated without drainage only one died and that from postoperative pneumonia.

Convincing evidence is furnished in a remarkable paper of Storck,[6] who reported forty-six cases of penetrating wounds of the abdomen of which thirty-five were gunshot and eleven were stab wounds. The number

of perforations in the patients who survived averaged 5.44, with a maximum of twenty-five in a single case. These perforations included practically every viscus in the abdomen. The mortality in the cases of stab wounds was 27.2 per cent; and in gunshot cases 40 per cent; and the combined mortality was 37 per cent.

In spite of hemorrhage, shock and gross spillage of intestinal contents from multiple perforations, these figures compare favorably with the mortality of simple perforative appendicitis as reported by some of our best clinics. If one asks how this can be possible, he may find the answer in this sentence from Storck's paper: "Because of the impossibility of draining the peritoneal cavity and because of the danger of intestinal obstruction resulting from the introduction of drains into the peritoneal cavity, the intraperitoneal introduction of drains at the time of operation for penetrating wound of the abdomen is now considered futile." This report should be "must" reading for every military surgeon.

Space does not permit even a summary of the many reports of improved results following the abandonment of the drain, but a few of the writers are: Shipley and Bailey, H. C. Miller, E. P. Hall, Sr., R. D. Kirk, Jr., Stanley Raw, B. Banks-Marchini, J. G. Andrew. It is significant that in a rather extensive review of the literature I have failed to find a single report of a surgeon's return to the use of drainage after he has discontinued its use.

Failure to grasp the significance of all this experience is exemplified by two of many similar recent reports.

In July, 1940, King[7] in a study of 804 cases of acute appendicitis at the Binghamton City Hospital during the years 1934–1937, states, " . . . the records show a definite trend toward less frequent drainage in acute appendicitis. Almost without exception, however, drainage was used in peritonitis cases. . . . In all, 277 cases were drained. Of these 216 were cases of peritonitis (only four peritonitis cases were not drained) while sixty-one drained cases were uncomplicated by peritonitis at the time of operation. . . .

"The cases complicated by diffuse peritonitis had appalling mortalities. In 1929–1930 the mortality in this group was 63.6 per cent, in 1934–1935 it was 33.3 per cent; in 1935–1936, 75 per cent, and in 1936-1937, 85.7 per cent. The four-year average for thirty-two cases was 65.6 per cent, or roughly two deaths out of three cases."

As late as August, 1940, Jackson and Perkins[8] advocated not only drainage through the wound in all cases in which pus is present, but rubber tube drainage through a stab wound in the flank, and a drain to the pelvis if there is generalized contamination. Although their mortality rate for 100 cases was only 12 per cent, thirty-two of the one hundred developed complications including:

6 patients with general peritonitis; all died
4 patients with fecal fistula; one was associated with intestinal obstruction and died; one was associated with repeated hemorrhages and died
1 patient developed subphrenic abscess and died
1 patient developed septicemia and died

None of these complications has occurred in any of our cases of diffuse peritonitis treated by closure without drainage in the past thirteen years with the exception of one death from peritonitis reported below.

In any discussion of peritonitis, confusion results from ambiguous terminology. For the purpose of this paper peritonitis is either localized or diffuse (some authors prefer the term "spreading" for this type). By localized peritonitis is meant a collection of pus surrounded and limited by a definite wall of adhesions. It may be an abscess the size of a walnut or a cavity containing several ounces of purulent fluid. The essential point is that the pus is definitely walled off from the rest of the peritoneal cavity. Whatever its size, it is still an abscess. If the walls of this abscess are lined with intact peritoneum, it may be treated in the same way as diffuse peritonitis without drainage. If the walls are necrotic and the integrity of the peritoneal lining is impaired, or if any part of the wall

is not lined with peritoneum, the treatment does not differ from that of an abscess elsewhere in the body. It must be either loosely packed with gauze or adequately drained.

By diffuse peritonitis is meant an extension of purulent inflammation beyond the immediate source of infection with no definite limiting wall. It may consist of purulent exudate between loops of bowel agglutinated by fresh fibrin deposits, or a pelvis full of pus or an entire abdomen full of infective fluid. The term general peritonitis is properly applied only to the latter condition and it is of rare occurrence. In nearly all cases some part of the peritoneal cavity is kept free from infection by protective adhesions. It is in diffuse peritonitis that drains do the greatest harm.

In untreated peritonitis the cause of death is almost always overwhelming toxemia. After this toxemia has developed operation of any kind is usually futile. For that reason the nondrainage treatment is effective during the first forty-eight hours, after which time the mortality increases rapidly regardless of the form of treatment used. It is in these delayed cases that conservative treatment is indicated, because the defense mechanism of the peritoneum has been overcome by the invading bacteria and general body resistance must be reinforced. We are no longer dealing with a peritoneal battlefield, but a total war involving the entire body. At this stage local treatment is less important than the measures necessary to combat the effects of toxemia, exhaustion, dehydration, disturbance of the acid base balance, and paralytic ileus. No elaboration of these technics is necessary here because they are not germane to the question of drainage. The use of transfusions, sulfonamides, Miller-Abbot tubes, saline infusions, etc., when indicated are taken for granted.

On the other hand in the case of deaths following operation with the insertion of deep drains, autopsy usually shows that peritonitis at death is largely limited to the region of the drain. In these cases a frequent cause of death is intestinal obstruction and the obstruction is nearly always found in the region of the drainage tract.

Sir Samson Handley[9] believed that in generalized peritonitis death is never due to the peritonitis itself, but always to intestinal obstruction.

Shipley,[10] in describing the experiences which led to his discarding drainage says, "But there were a certain number who came to grief and in almost every instance intestinal obstruction was the complication present. Even when this condition did not actually exist there was often a period of uncertainty because of the presence of paralytic ileus or incomplete obstruction due to angulation. In the spring of 1930, within a few weeks, four patients on whom I had operated for peritonitis following appendicitis and drained, developed mechanical obstruction and two of them died. At operation all four were completely obstructed by angulation of a loop of small intestines *in the drain tract. The remainder of the peritoneum was free of adhesions or any evidence of infection.*"

In neglected cases in which a retrocecal abscess has formed, intestinal obstruction is also a frequent factor in the mortality but the obstruction is more often due to mesenteric thrombosis than to mechanical obstruction.

An analysis of our last 374 cases of acute appendicitis follows:

In 216 patients the appendix was acutely inflamed; none were drained; none of the patients died

97 patients had gross perforation and frank peritonitis, either localized or diffuse; 54 were drained; 13 died; mortality 24 per cent; 43 were not drained; 4 died; mortality 9.53 per cent

Of the four deaths in the undrained series only one was due to peritonitis. The other three deaths were all instructive.

In the first, the tip of the gangrenous appendix was retroperitoneal under the root of the mesentery. An abscess formed under the root of the mesentery and fatal mesenteric thrombosis resulted.

The second patient was a woman seven and one-half months pregnant. She was

convalescent with a normal temperature when on the seventh day miscarriage occurred with fatal collapse.

The third patient was a man of sixty whose whole lower abdomen was full of thick, foul smelling pus. He had complete suppression of urine and died of uremia on the seventh day. When on the fourth day severe vomiting caused a disruption of his midline incision (made because of a mistaken diagnosis) the peritoneum was everywhere normal, no adhesions had formed, and no pus was found. The absence of adhesions and the complete disappearance of pus within four days has been a startling and constant finding in the few cases which we have had an opportunity to explore. Shipley reports similar experience.

If we eliminate these three cases, we have one death from peritonitis in forty-three patients treated by nondrainage, a mortality of 2.33 per cent. Furthermore our mortality of 24 per cent in drained cases was largely due to the complications which made drainage necessary. They included delayed operations on patients who were extremely ill on admission, with large abscesses which were simply opened and drained without removal of the appendix. All the patients in the fatal cases had been ill for periods of from forty-eight hours to five weeks.

The important point is that most of our undrained cases with the low mortality were those of diffuse peritonitis while practically all of the drained cases with much higher mortality had walled-off abscesses. The advocates of delay and the Ochsner treatment base their position on the assumption that mortality is less if the pus is allowed to become localized. This assumption is perhaps true if drains are used in cases of diffuse peritonitis. It certainly is not true if these patients are treated by early operation, removal of the appendix, aspiration of the purulent fluid and closure of the peritoneum without drainage.

Neither the amount of pus, the character of the pus, nor the area involved has anything to do with the question. If the source of infection can be removed and if the peritoneum is intact, a drain is not only unnecessary but harmful.

To avoid residual abscesses the free pus should be removed with a Poole suction tube, especially from the pelvis. In order to do this effectively it is often necessary to separate loops of bowel agglutinated by fibrin. In the first forty-eight hours this fibrin is not organized into true adhesions. If a wet, gloved finger is gently used, loops can safely be separated without injury to the visceral peritoneum and no permanent adhesions will form. If this is not done the fibrin will exercise its function of forming a true adhesion to wall off the pus, with resultant abscess and permanent adhesions.

Diffuse peritonitis treated by nondrainage within forty-eight hours is no longer a serious problem. Every trace of pus has disappeared within four days and no adhesions occur provided that the source of infection is removed with no traumatizing of the peritoneal endothelium. (This implies the McBurney incision for direct approach and simple ligation without burial of the stump.)

Drainage is contraindicated in all types of diffuse peritonitis when the source of the infection can be eliminated, whether this be a pyosalpinx, perforated intestine or gunshot wound. We have closed without drainage more than fifty consecutive wounds in patients with acute salpingitis or tuboovarian abscess with no deaths, and no serious complications. Also, we have the satisfaction of knowing that few if any of these patients will suffer later from crippling adhesions.

SUMMARY

The nondrainage treatment is based not on theory but on overwhelming experimental and clinical evidence. Both experiment and experience have demonstrated beyond any doubt these facts:

1. It is physically and physiologically impossible to drain the peritoneal cavity by any means whatsoever.

2. In all types of peritonitis, removal of the source of infection, aspiration of pus, avoidance of injury to the endothelium and closure of the peritoneum is the only treatment required. If treated within forty-eight hours, the temperature will usually reach normal in three or four days, the exudate will be absorbed and permanent adhesions will rarely, if ever, be formed.

3. With such treatment deaths from peritonitis *per se* will seldom occur. Most deaths result from delayed operation, allowing abscesses to invade retroperitoneal tissues, from overwhelming toxemia, from mesenteric thrombosis, or from intestinal obstruction resulting from adhesions produced by the drainage tube.

4. Retroperitoneal tissues and the fascia and fat of the abdominal wall have none of the resistance of the peritoneum. Hence they should be drained or left wide open until healthy granulations form. Many such wounds can then be closed by secondary suture.

5. Since the success of nondrainage depends on the integrity of the peritoneum, a drain is indicated if the walls of an abscess are necrotic or lined with a shaggy gray membrane, if the retroperitoneal space is opened, or if bleeding cannot be completely controlled.

6. There need be no hesitation in gently separating fresh fibrinous adhesions in order to reach all collections of pus with the aspirator. Failure to do this may result in a localized abscess.

7. The more widespread the peritonitis, the less the indication for drains.

8. The time to operate is when the diagnosis is made. No surgeon lives who can tell with certainty what is going on inside the abdomen. With nondrainage there is nothing to be gained by delay and everything to be gained by removal of the source of infection as early as possible.

9. Appendiceal peritonitis has been used to illustrate the principles underlying the treatment by nondrainage only because of the great mass of evidence available in that particular field. Removal of the source of infection and omission of the useless and harmful drain is equally important in the treatment of battle casualties. The validity of the method was established beyond question in the last two years of the first World War.

With a long war facing us the life of every fighting man who suffers a penetrating wound of the abdomen will depend largely on three things: Control of shock, sulfonamides and nondrainage of the peritoneal cavity.

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