Original Articles.

A STUDY OF TWENTY-FOUR CASES OF TYPHOID FEVER WITH SYMPTOMS OF PERITONEAL INFECTION; LAPAROTOMY.¹

SUMMARY.—SEVENTEEN CASES OF PERFORATION OF INTESTINE; TWO CASES OF PREPERFORATIVE NECROTIC AREAS; ONE CASE OF RUPTURED MESENTERIC GLAND; THREE CASES CAUSE OF SYMPTOMS UNDETERMINED, DIAGNOSIS NOT CONFIRMED.

GROUPING AND ANALYSIS OF CASES;
GENERAL CONCLUSIONS; DETAILED REPORT OF CASES.

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Committee of the Boston Society for Medical Improvement.

THE whole number of cases studied by this Committee is 27, all coming in the years between 1895 and 1900; 9 cases from the Massachusetts General Hospital; 16 cases from the Boston City Hospital; 1 case from St. Elizabeth's Hospital, and 1 case from the Boston Children's Hospital.

In the year 1895 there are 3 cases, but only one of them will bear analysis as a case of genuine typhoid fever; 1 case in 1896, 4 cases in 1897, 5 cases in 1898, and 14 cases in 1899.

This is a striking illustration of the increase in the operative tendency. The data from which this analytical report has been made are the complete medical, surgical and post-mortem records of the several hospitals, together with the summarized statements and opinions sent in by the physicians and surgeons in attendance. From this available material a concise view of each case has been made with reference to all the important points bearing in any way on the diagnosis and treatment of peritoneal infection in typhoid fever.

The Committee would deprecate its critical comments of to-day being interpreted as fault-finding with the practice of earlier years; neither does the Committee forget that the ideal is not always, or under all conditions, to be realized in practice. Doubtless a nearer approach to the ideal should be aimed at and would now be possible than is found in most of the cases here reported. And the Committee is still further quite aware that the ideal itself—for example, in regard to such a matter as the value and application of the white blood count—is subject to further changes; the value of pain as a warning symptom also merits further study.

Of the 27 cases A, B and C must be thrown out of our consideration, because of no proof that they were genuine cases of typhoid. Cases B and C were both cases of advanced general peritonitis when first seen, and in both of them there was no proof that typhoid was the origin of the peritonitis.

Case A was brought to the City Hospital in a moribund condition, and showed general peritonitis at the operation. Cultures from the interior of the gall-bladder showed the presence of the bacillus typhosus. This, however, cannot be accepted as conclusive proof that the case was one of typhoid peritonitis. Cultures from the peritoneal fluid showed only the staphylococcus albus. It is an accepted fact that typhoid bacilli can and do remain active in the gall-bladder for

months and years after an attack of typhoid fever. For example, Miller² has reported a case in which the bacillus typhosus was found in the gall-bladder seven years after an attack of typhoid fever. There is no proof, therefore, that the peritonitis in Case A might not have been of other origin than typhoid fever, and, therefore, it cannot be included in our list of cases for study. This series, then, embraces 24 cases, which are all undeniable cases of typhoid; there can be no controversy on this point.

Eighteen cases had a positive Widal serum reaction in addition to the evidence furnished by the clinical course of the case and the positive pathological evidence furnished by the operation or post-mortem examination. In 3 cases the Widal test was not made; in 3 cases the Widal test was negative. Two of the cases in which it was not made occurred in the years 1895 and 1896, a period of time before the use of the test.

Of the 6 cases in which the Widal test was not made or was negative there can be no doubt of the diagnosis of typhoid, because of the findings at the operation or the post-mortem which fully corroborated the clinical diagnosis. In 2 of the cases in which the Widal test was not made the diazo test was positive.

All of the 24 cases had symptoms upon which a diagnosis of intestinal perforation was made and for which an operation was performed.

Three of the cases (XXII, XXIII and XXIV) presented symptoms strongly suggestive of intra-abdominal infection, and accordingly were operated upon with a diagnosis of intestinal perforation, but at the operation no positive cause for the symptoms was found. Two cases recovered from this exploratory operation, and 1 died. These cases are of the greatest importance from the diagnostic point of view, and will be considered carefully when the subject of "Symptoms and Diagnosis" is taken up.

Case XIX had characteristic symptoms of acute sudden peritoneal infection, and operation discovered a general septic peritonitis, but did not determine the cause; therefore this case should not be classed with those in which the cause of the peritonitis is certain, although it certainly seems rational, on considering the history and the symptoms and the fact that several different thin places in the intestinal wall were found at operation, to consider it a case of perforation.

In Cases II and XVI the classic severe symptoms of perforation were simulated, but operation discovered the cause of the symptoms to be a general septic peritonitis originating in damaged and necrosed areas of peritoneum over the bases of one or more typhoid ulcers. These cases might be called cases of progressive peritoneal infection from threatened perforation.

In Case XXI the symptoms amply justified the diagnosis of intestinal perforation, but operation proved that the symptoms, which were identical with the usually accepted symptoms of perforation of the bowel, were due to a general septic peritonitis arising from a ruptured mesenteric gland.

So that out of the 24 cases of true typhoid only 17 were cases of peritoneal infection from actual perforation of the intestinal wall. Two cases were of general infection from threatened perforations or areas of necrotic peritoneum; 1 case of general infection from

¹ Report presented at a meeting of the Boston Society for Medical Improvement, April 16, 1900.

² Johns Hopkins Hospital Bulletin, May, 1898.

a ruptured mesenteric gland; 1 case of general infection from an unknown cause, presumably intestinal perforation; 3 cases had no certain cause for the symptoms which demanded operative interference.

Since it will be shown that in many cases no differentiation between the usually accepted symptoms of actual perforation and the symptoms of general septic peritonitis can be made, this series of 24 cases gives us, for further study, 21 cases of grave peritoneal infection in typhoid fever and 3 cases of mistaken diagnosis, but with symptoms of peritoneal infection.

Diagnostically and from the point of view of treatment all of these cases should go in under the head of acute peritoneal infection in the course of typhoid fever. It may be well, however, to analyze the two groups of cases separately, dividing the perforations from the other cases.

Of the 24 cases presenting acute abdominal symptoms, with or without discoverable cause, 18 were males and 6 were females. Of the 18 males 15 were under thirty years; 1 was forty-five years; 1 was thirty-eight years; 1 was thirty-two years; 14 cases were between the years of nineteen and thirty and 1 case was sixteen years. Of the females, 6 in number, 4 were between twenty and twenty-six years, 1 was forty years and 1 was sixteen years.

The total number of cases shows, therefore, that acute abdominal infection occurs most often in young adults between the ages of eighteen and thirty years; that it is most frequent in men; that it is rare in children. It is noteworthy that 2 of the cases presenting acute abdominal symptoms without discoverable cause occurred in young women, aged twenty and sixteen years, respectively.

These facts in regard to age and sex are in general accord with the investigations of Dr. R. H. Fitz and others, although the percentage of those under thirty years of age happens to be somewhat larger than in Dr. Fitz's tables.³

As to the question of typhoid perforation in children under twelve years of age, we have been able to find but 4 cases of operation for perforation in children under that age in all the cases reported in literature.

Cushing:⁴ Boy, nine years old, on which case 3 laparotomies were done; the case recovered. Three cases are given in Keen's⁵ tables of cases of operation for typhoid perforation.

All the cases but one were white in color. This proportion of blacks to whites would very likely be different in other parts of the country, where the proportion of the colored population to the white is greater.

In 18 of the 24 cases the clinical nature and course of the typhoid was mild, and in 15 of these 18 mild cases perforation or general septic peritonitis was present. The 3 cases which presented symptoms without certain cause were mild cases.

One case, XVII, was a "walking typhoid"; 6 cases, II, VI, VIII, XII, XIV and XX, were *severely sick* typhoids. All of these 6 cases had general septic peritonitis from actual or threatened perforation, and all died.

These facts are in accord with the hitherto accepted knowledge that intestinal perforation in typhoid fre-

quently occurs in those cases of a mild type, or, in other words, there is no relation between the severity of the course of the fever and the likelihood of intestinal perforation.

The difficulty of the diagnosis of the peritoneal infection in cases of the severe type will be touched upon later.

The sudden fulminating symptoms in the severely sick cases will be found in the majority of instances to be due to a general septic peritonitis, which was often unsuspected and had slowly progressed. On the other hand, a sudden, acute onset in the mild type more often meant sudden intestinal perforation with extravasation. In certain of the severely sick cases in this series it would seem as if a diagnosis of the onset of the peritoneal infection was practically impossible.

The organic complications were very few: Three cases had mitral regurgitation amply compensated; 1 case in which operation discovered no cause for the symptoms had an especially feeble heart, with mitral regurgitation, yet this case recovered from the exploration; in 9 cases intestinal hemorrhages are noted; in 2 cases it occurred three weeks before the severe symptoms of perforation appeared; in 2 cases it occurred forty-eight hours before the severe abdominal symptoms, but there was a distinctly comfortable period between the occurrence of these hemorrhages and the sudden onset of abdominal pain. In 3 cases intestinal hemorrhages preceded and coincided with the beginning of peritoneal infection. Case XIX had daily intestinal hemorrhages for three days before sudden abdominal symptoms. Case XII had a severe hemorrhage five days before the symptoms for which operation was done; but this case was so sick and stupid as to justify the supposition that perforation probably took place long before the foul and evidently long-established general peritonitis was diagnosed. Case VI had two severe hemorrhages just preceding and coincident with the perforation. In 2 cases intestinal hemorrhages are noted after the operation. In Case I a severe hemorrhage five days after the operation was undoubtedly the cause of death.

Intestinal hemorrhage, inasmuch as it must come through extensive ulceration of the intestinal wall, and since this may mean proximity to the peritoneal coat of the intestine, must be to a large extent a danger signal of perforation. The occurrence of intestinal hemorrhages, therefore, should properly demand a close watch for the symptoms of beginning peritoneal infection, frequent examination of the abdomen to detect tenderness or abnormal rigidity and muscular spasm, associated with frequent leucocyte counts. The occurrence of acute abdominal pain following and coincident with intestinal hemorrhage should lead to the suspicion of peritoneal infection.

THE WEEK IN THE DISEASE IN WHICH PERITONEAL INFECTION TOOK PLACE.

Of the cases in which perforation and general peritonitis existed the acute abdominal symptoms appeared at the following times: In 5 cases at the end of the second or first of the third week; 8 cases at the end of the third week; 1 case during the fourth week; 4 cases during the fifth week; 1 case during the sixth week; 1 case during the ninth week; 1 case during the eleventh week in a relapse.

Of the cases operated upon under a mistaken diag-

³ Transactions of the Association of American Physicians, vol. vi, p. 200.

⁴ Johns Hopkins Hospital Bulletin, 1898.

⁵ Surgical Complications and Sequels of Typhoid Fever.

nosis 1 was in the fourth week, 1 in the fifth week and 1 in the sixth week. The diagnostic symptoms of intestinal perforation in typhoid as they are usually understood, namely, sudden abdominal pain, fall of temperature, collapse, general abdominal rigidity and tenderness, with or without vomiting, anxious facies, coming on without warning, do not present themselves as a complete picture in any one of this series of cases. The majority of the cases show records of definite warning symptoms, or, in other words, present evidences of a gradual onset of peritoneal infection.

It is, in fact, rational to conclude from a study of the symptoms in these cases, together with the findings at operation, that the severe symptoms corresponding to the ordinary text-book picture of intestinal perforation in typhoid were, in the majority of cases, caused by the general septic peritonitis resulting from a perforation or a threatened perforation, the occurrence of which (the perforation or local area of infection) was indicated more or less plainly by symptoms of less severity, which antedated the severe or so-called diagnostic symptoms by a definite number of hours.

If it is one thing to operate for intestinal perforation, and a decidedly different thing to operate for a septic peritonitis resulting from a perforation, the fact should be thoroughly understood that the severe and usual diagnostic symptoms in this class of cases meant that septic peritonitis had already existed for some time, and that the perforation took place at a previous time and was evidenced by milder and more localized symptoms. It is a fact that in the majority of these cases definite premonitory symptoms are recorded at a time more or less remote from the severe symptoms which first led to the diagnosis, and for which the physician called the surgeon in consultation.

Seven cases of the 21 in which operation found cause for the symptoms, namely, I to VII, inclusive, presented a sudden, acute picture of grave abdominal infection without *any* premonitory symptoms. Of these 7 cases 5 (I, III, IV, V and VII) were mild typhoids, whose general and mental condition was favorable to the noting of abdominal pain. Two cases (II and VI) were very sick, stupid and semi-conscious; a class of cases in which the occurrence of considerable localized abdominal pain might not be noted. In Case II the symptoms, sudden though they were, were due to a general peritonitis from the damaged peritoneum over the base of one or more ulcers, and not due to a sudden perforation. In Case VI the sudden symptoms were caused by the presence of a general peritonitis which had extended from a localized abscess originating from the intestinal perforation which at its appearance had given no symptoms, so that of the 7 cases which presented fulminating symptoms and which most closely conformed to the hitherto established idea of the symptoms of intestinal perforation only 5 proved to be due to *actual sudden perforation*, while 1 was not a case of perforation at all, and in the other the perforation and progress of the localized peritonitis with abscess formation did not cause any symptoms.

The sudden onset in these 7 cases presents a different picture in the 5 cases in which the symptoms were proved to be due to actual perforation. In these cases we have the symptoms of sudden severe pain

coming on without warning, more or less localized in the right iliac region, accompanied by localized muscle spasm and tenderness, with the varying amount of shock. In the 2 cases in which the symptoms were due to general peritonitis, we have a picture suggesting general peritonitis, marked collapse, great distention, and general tenderness and rigidity. It might be concluded from a study of these 7 cases that in mild typhoids the appearance of sudden severe abdominal symptoms without any previous abdominal pain or tenderness may most often mean sudden intestinal perforation with extravasation into the general peritoneal cavity, and that the symptoms may vary, the chief symptoms being sudden pain, tenderness and muscular spasm localized, attended with more or less marked shock; but in very sick and lethargic cases the appearance of severe abdominal symptoms without warning most often means the presence of general septic peritonitis originating in some one of the possible sources of peritonitis in typhoid, the gradual onset of which it has been impossible to diagnose because of the sick and stupid condition of the patient.

It has been stated that in the above 7 cases there were no warning symptoms. By this is meant direct abdominal symptoms — pain, tenderness and muscular spasm. It should be stated, for the sake of completeness, that 3 cases had one or more intestinal hemorrhages on the two days preceding the acute abdominal symptoms. One case had several hard chills without hemorrhage during the twenty-four hours preceding the onset of the peritoneal symptoms. The occurrence of chills as a symptom of peritoneal infection in typhoid is probably rare. It can only be a symptom leading to suspicion of infection when it is unassociated with hemorrhage.

J. F. R. Gairdner⁶ states that rigor as an initial symptom of peritoneal infection in typhoid is uncommon. He noted it in only 2 cases out of 47 studied by him. It is noteworthy that in 1 of his cases and in the case of Jenner the peritonitis ushered in by rigors was due to the rupture of a softened mesenteric gland. In Case XXI, the only case of our series of peritonitis from the same cause, no chill occurred. In the absence of intestinal hemorrhage to account for rigors it might not be irrational to suppose that it might occasionally be present in the early stages of peritoneal infection.

In those 3 cases in which no cause for the symptoms was found at operation the onset of the same was gradual. These cases will be referred to again in the subject headed "Symptoms and Diagnosis."

We have 14 cases, namely, Cases VIII to XXI, inclusive, with more or less gradual onset of the abdominal symptoms. Eleven of these are cases of intestinal perforation and resultant general septic peritonitis; 2 are cases of general peritonitis without perforation; 1 a case of general septic peritonitis of unknown origin, though probably due to a perforation.

One of the perforative cases (Case XVII) was a "walking typhoid," and had probably had general peritonitis for at least thirty-six hours before entering the Massachusetts General Hospital.

In all of these 14 cases *distinct warning symptoms* antedated equally distinct severe and diagnostic symptoms. The milder symptoms, which we must conclude marked the beginning of the peritoneal infection, were chiefly various degrees of abdominal pain and

⁶ Glasgow Medical Journal, 1897, vol. xlvii, p. 103.

tenderness, either localized or general, complained of by the patient. All of these 14 cases were mild types except 4. In only 1 of the 14 cases was any complaint recorded of abdominal pain until the complaint ushering in the severe symptoms, and in the single exception to this the complaint of abdominal pain antedated the actual perforation by some weeks.

In 4 cases abdominal distention throughout the course of the fever is noted. In 9 cases there is no record of any abdominal complaint or abnormality until the peritoneal infection was under way. In every one of these cases recorded *complaint by the patient of abdominal pain* was followed by peritoneal infection. The severe symptoms followed these warning symptoms at various intervals in every case, and there can be little doubt that the severe symptoms, when they appeared, meant that the peritoneal infection had become general and alarming.

An analysis of these premonitory symptoms shows that in 6 cases the pain complained of was general abdominal pain; in 3 cases localized in the left iliac region; in 4 cases in the right iliac region; in 1 case in the lower abdomen; in 1 case general abdominal pain, and also pain in the rectum and bladder; in 1 case the pain began in the epigastrium and later became marked in the right iliac region. It is much to be regretted that the importance of this abdominal pain was apparently not appreciated, as shown by the case records.

In 3 cases *comparatively* complete physical examination is recorded as made at the time. In no case was there a record of as careful an examination of the patient in every detail as the importance of the symptoms demanded, so that our knowledge of the accompanying conditions of tenderness, muscular spasm, leucocytosis, pulse and temperature is insufficient.

In 2 cases the complaint of pain was preceded by vomiting. In only 2 cases was the record of a leucocyte count made, and in these cases it appears that the blood count was only done once. As will be shown under the head of "Leucocytosis as a Symptom," an isolated blood count is of little or no value. In 1 case general abdominal tenderness and rigidity were noted, and in 2 cases there was general tenderness. In 4 cases there is no record of any physical examination, but a record that *morphine was given*. In 3 cases it is noted that the pulse and temperature were rising. This warning abdominal pain preceded the severe symptoms by hours and days; it appeared in 1 case sixty hours before; 1 case forty-eight hours before; 1 case twenty-four hours before (Case XVII); 4 cases twelve to fifteen hours before; 4 cases eight hours before; 3 cases three to five hours before. It is noteworthy that the 2 cases in which no actual perforation but general peritonitis from other causes existed had equally marked advanced pain.

Cases XVI and XXI, where the severe symptoms followed just as in the cases of perforation, point plainly to the reasoning that the early pain in these cases was due to local peritoneal infection, and that the severe symptoms were due to the establishment of a general infection.

The severe abdominal symptoms in every case determined the diagnosis and the decision to operate. In general these symptoms were those of general septic peritonitis, namely, general abdominal pain, severe; general tenderness and rigidity, with distention, collapse, poor pulse and anxious facies.

In only 3 of the cases was there any vomiting. The classical sudden fall of temperature is not present in many of the cases. The temperature fell somewhat in a few cases. In 2 cases it fell to normal, and began at once to rise; in 1 case it fell to and remained subnormal; in 3 cases a slight drop of temperature is noted; in 6 cases there was no fall of temperature, and in 3 cases it is recorded as rising.

In 8 cases a leucocyte count was taken at a time corresponding to these severe symptoms; in 4 cases it showed leucocytosis; in 4 cases it showed no leucocytosis. The leucocyte counts are, however, of only relative value, as will be considered later under the heading of "Leucocytosis as a Symptom."

In only 2 cases was a record of obliterated liver dullness made.

The constant symptoms in every case are abdominal pain, general tenderness and rigidity, with distention, associated with collapse and rapid, feeble pulse.

A comparison of the severe abdominal symptoms in the two classes of cases—the 7 cases where warning symptoms were not present and the 14 cases with warning symptoms—shows *distinct differences* in the severe symptoms in those in which the sudden intestinal perforation and extravasation came on without warning. As has been stated, the severe pain, more or less localized, with shock and muscular spasm and tenderness, characterized the first set of cases, and the shock varied in the different cases. In 1 case the pulse was not over 100. There was no vomiting, no general abdominal tenderness or rigidity, and no distention in any case.

Contrasted with this first set of cases, the severe symptoms in the second set present a much graver picture. It is noteworthy that in the 2 cases in the first set, in which general peritonitis was the cause of the symptoms and not intestinal perforation, the picture corresponds to the severe picture in the second class of cases.

It should be noted that if the signs of actual perforation with extravasation are local to a great extent, as in the 5 cases of our first set, a careful examination of all cases at the warning complaint of pain might in many of them give us definite diagnostic signs, as in Case III, for example.

A comparison of these two sets of cases—the 7 with sudden symptoms without perforation and the 14 cases with severe symptoms following premonitory pain—bears out the point that in most of them no positive diagnosis of perforation was made, but only a diagnosis of the general peritonitis resulting from the perforation.

THE TIME OF OPERATION AFTER SYMPTOMS APPEARED.

In the 7 cases with no warning symptoms operation was done within twelve hours in every case; 1 case within an hour and a half; 3 cases within four hours; 1 case within seven hours; 1 case within eight hours; 1 case within twelve hours.

At the operation general septic peritonitis was not evident to the eye in Cases I, III and VII. In Case V it is noted as just beginning and not far advanced. In Case IV the perforation was protected by weak adhesions, and no general infection had taken place up to the date of operation. In Cases II and VI general peritonitis was farther advanced and was evidently the cause of the sudden symptoms, so that only 5

operations can be said to have been for perforation itself and not for general peritonitis; and of these 5 cases 2 cases, namely, Cases I and IV, may be said to have recovered from the operation, although Case I died at the end of five days from excessive intestinal hemorrhage. Of the 14 cases which had warning symptoms 9 were operated upon within five hours from the severe symptoms, or from the time of positive diagnosis of perforation; 1 case within eight hours; 1 case within nine hours; 3 cases within twenty-four hours; 3 cases, however, were not operated upon for twenty-four hours after general peritonitis was plainly evident.

Reference to the annexed table will show that the average time of the operation after the early warning symptoms was twenty-three hours.

NUMBER OF HOURS ELAPSING BEFORE OPERATION IN THE 14
CASES WITH PREMONITORY SYMPTOMS.

After first pain and warning symptoms	.	.	.	.	.	20 hours.
" " " "	.	.	.	.	.	12 "
" " " "	.	.	.	.	.	48 "
" " " "	.	.	.	.	.	11 "
" " " "	.	.	.	.	.	15 "
" " " "	.	.	.	.	.	24 "
" " " "	.	.	.	.	.	20 "
" " " "	.	.	.	.	.	12 "
" " " "	.	.	.	.	.	60 "
" " " "	.	.	.	.	.	8 "
" " " "	.	.	.	.	.	48 "
" " " "	.	.	.	.	.	6 "
" " " "	.	.	.	.	.	12 "
Case of Bloom, XVII .	.	.	.	.	.	?
After severe symptoms	.	.	.	.	.	34 hours.
" " " "	.	.	.	.	.	8 "
" " " "	.	.	.	.	.	24 "
" " " "	.	.	.	.	.	3 "
" " " "	.	.	.	.	.	3 "
" " " "	.	.	.	.	.	9 "
" " " "	.	.	.	.	.	5 "
" " " "	.	.	.	.	.	34 "
" " " "	.	.	.	.	.	24 "
" " " "	.	.	.	.	.	3 "
" " " "	.	.	.	.	.	4 "
" " " "	.	.	.	.	.	2 "
" " " "	.	.	.	.	.	34 "
Case of Bloom, XVII .	.	.	.	.	.	?

If it is granted that the early pain, etc., meant perforation or beginning peritoneal infection, and the severe symptoms meant general septic peritonitis, then it must be granted that operation took place from six to sixty hours after it was first indicated.

In all of these 14 cases the operation should be considered as primarily directed against the septic peritonitis and not the intestinal perforation. The recoveries from the operations in this class of cases are Cases XVI and XVII. Case IX is reported to have recovered from the operation for one perforation and to have lived eight days, until a second perforation necessitated a second operation, followed by death. It has been seen in the analytical report of this case that such a view of it cannot be permitted. It seems just to decide that this case did well after the first operation, until the extensive necrotic changes in the cecum involved the first sutured area and caused giving way of the sutures and a second extravasation.

SITUATION OF THE PERFORATION AND SITUATION OF THE DAMAGED AREAS.

We have in this series of cases 17 cases of true intestinal perforation and 2 cases of threatened perforation, with damaged and necrosed peritoneum. In every case the perforation or impending perforation was in the ileum.

In 7 cases it was within one foot of the cecum; in 4 cases it is noted as close to the cecum; in 1 case two feet from the cecum; in 1 case three feet from the cecum; in 1 case four feet from the cecum; in 5 cases

the site is not recorded; in 3 cases it is recorded to have been the size of a "pin's head"; in 2 cases the "size of a dime"; in 1 case the size of a "split pea"; in 1 case to have been a large, irregular hole; in 1 case two perforations close together, near the cecum. In only 1 case (Case XV) was a second perforation found at the post-mortem.

In Case XIV the perforated coil of intestine was found situated low down in the pelvis, and in this case the initial pain complained of was referred to the region of the rectum and the bladder.

The method of closure of the perforation in every case but one consisted of inversion of the hole with silk sutures. In 1 case (Case XI) the ulcer was resected by a V-shaped incision. In Case X two rows of silk Lambert sutures were used, causing extreme inversion of the intestinal wall. This case died within thirty-six hours, with extreme abdominal distention and obstinate vomiting. At the autopsy it was found that the use of the two rows of sutures had reduced the lumen of the intestine to such an extent that it only took the top of the little finger, and that it was obstructed by a moderate amount of hard fecal matter. It seems legitimate to raise a query as to whether this case died from peritoneal infection or from intestinal obstruction, from the great inversion of the wall of the gut.

In 3 cases, in addition to suturing perforations or threatened perforations, additional suspicious thin places in the intestinal wall were inverted. This procedure, provided the condition of the patient justifies the expenditure of the extra time, is certainly advisable in all operations for typhoid perforation. It has been advocated by Sifton⁷ and also by Hill in a case reported by Keen.⁸

In 7 cases out of the 21 the abdominal incision was median; in 2 cases the incision is not recorded; in 12 cases the primary incision was in the right iliac region. In only 2 cases are counter openings for irrigation and drains noted. The right iliac incision proved most capable of allowing speedy access to the infecting lesion. In 2 of the cases where the median incision was used it is especially recorded that long overhauling of the intestines was needed before the perforation was found. In 1 of the cases in which the median incision was used the time of operation is recorded as one and a half hours. Certainly the site of the infected areas in this series of cases points to the routine employment of a right lateral incision.

PERITONITIS: KIND, AMOUNT, WITH THE BACTERIOLOGICAL FINDINGS.

Unfortunately, the value of this series of cases is much impaired by the lack of bacteriological data concerning the nature of the general infection. If this series of cases furnished complete records of a careful physical examination at the appearance of the first symptoms in every case, and also complete bacteriological evidence concerning the virulence of the peritonitis resulting from the initial infection, we should be much better equipped to arrive at some definite statistical basis for establishing an approximate prognosis in such cases.

Three cases, namely, Cases I, III and VII, operated on one and a half, three and eight hours after the sudden onset of the perforation, had no visible

⁷ Chicago Clinical Review, vol. iv, p. 368.

⁸ *Surgical Complications and Sequels of Typhoid Fever.*

macroscopical signs of general peritonitis. Case I lived nearly a week, and died from intestinal hemorrhage, and the autopsy showed no general peritonitis. Cases III and VII both died within sixty hours, one with the symptoms of general peritonitis and the other without such symptoms. There were, however, no bacteriological studies of the peritoneal infection in these 3 cases at the time of operation. Two of the cases may very probably have died from a virulent infection, which did not cause any visible signs.

Vincent⁹ experimentally has called attention to the fact that a streptococcus peritoneal infection in typhoid is usually a fatal complication. Vincent's studies have been corroborated by Flexner and Fraenkel.¹⁰ Cushing¹¹ reports a case in which operation was done within an hour or two after perforation, and at the operation no visible signs of general peritonitis were present. Cultures and cover-slip preparations from the fecal fluid pouring out of the perforation in the intestinal wall showed large quantities of streptococci, and the case died of streptococcus peritonitis.

It is perfectly within reasonable bounds to believe that one or more of the supposedly favorable cases in our series may have had a streptococcus infection. Certainly we have no evidence to the contrary.

Cases II and V show a beginning general peritonitis, as evidenced by gas and serous fluid free in the abdominal cavity, but indicated no injection of the peritoneal coat of the intestines. Both of these cases, so far as the macroscopical appearance went, seemed favorable, yet both died. Cultures taken in Case V showed a pure infection of the bacillus typhosus.

Case IV had no general peritonitis, as has been stated. Case XVI was one of general peritonitis arising from the same cause as in Case II, namely, damaged peritoneum; yet in Case XVI peritonitis was much more extensive macroscopically than in Case II, being evidenced by seroturbid fluid with fibrin flakes and with green fibrin patches on the intestines. Case XVI recovered; Case II died. Although Case II was a more severe typhoid, presumably the general peritonitis in one case was caused by a virulent infection, and in the other by a non-virulent; yet no bacteriological proof is available in either case.

General septic peritonitis in typhoid arising from threatened intestinal perforation from the necrosed and damaged areas of peritoneum over the base of ulcers must be accepted as a not very uncommon occurrence. J. F. R. Gairdner¹² has reported 5 such cases.

To sum up in regard to our first class of cases, namely, 7 with sudden onset of symptoms, 3 had no general peritonitis evident to the eye; 1 case had limiting adhesions around the perforation, with no general peritonitis; 2 cases had slight evidence of general peritonitis. These are all of them the most favorable cases for operation, for it must be said of them that operation was actually directed against the infecting cause and not against the resulting general infection. The bacteriology was studied in only one of these cases, and in this one a pure culture of the typhoid bacillus was found.

It is a fact that the typhoid bacillus has been very rarely found in the peritonitis of typhoid fever, possibly because, through delayed operation, cultures

have not been taken until the typhoid bacillus was overgrown.

It is possible, and probable, that the others of these early and seemingly favorable cases for operation may have had a general virulent infection either with typhoid bacilli or streptococci. It is past controversy that fatal peritoneal infection can have taken place without causing visible macroscopical signs at operation or autopsy. Of the 14 cases of gradual onset or with early warning symptoms, in every one visible peritonitis was found at operation, and in the majority of cases it was evidently of comparatively long standing.

Comparing the two sets of cases, namely, the 7 cases with sudden onset and the 14 with gradual onset, we find that in operations done on the cases of sudden perforation within one to eight hours after the first symptoms there was found no general peritonitis, but at most a very slight beginning peritonitis; but operation done on those of gradual onset, within one to eight hours after the severe or diagnostic symptoms, found extensive general peritonitis—so extensive that in several cases the operator noted that this peritonitis must have existed many hours longer than the symptoms would lead one to suppose. The conclusion is plain that in these cases the perforation or the beginning peritoneal infection took place at a time more or less remote from the severe and diagnostic symptoms.

These 14 cases are cases of operation for general peritonitis which had existed for a longer or shorter time—in 1 case originating in a ruptured mesenteric gland; in another, threatened perforation; in 1 from an unknown cause; in 11 from intestinal perforation.

The prevailing type of peritonitis noted was the seropurulent, with fibrin flakes in the turbid fluid and adherent fibrin patches on the intestine. Nine cases are noted that had feces or fluid fecal matter in the intestinal cavity or fluid with a fecal odor. All of these cases were intestinal perforations. Two cases of intestinal perforation note a cloudy, turbid fluid without fecal odor.

Case XXI was a case of general seropurulent peritonitis originating in a ruptured mesenteric gland. Cultures showed this to be a pure infection of bacillus typhosus. In Case XVII punctate hemorrhages on the intestinal wall are noted, and brownish fluid in the peritoneal cavity without odor.

It is greatly to be regretted that in only 4 of these 14 cases were any bacterial cultures taken, and in certain of these the question arises as to their value, because of our incomplete knowledge as to how they were taken and because of incomplete reports of the differentiation of the various bacteria found. Flexner¹³ and Cushing¹⁴ dwell upon the necessity of making cover-slip preparations as well as plate cultures at the operation from the abdominal contents in cases of peritonitis, because of the well-known fact that the bacillus coli communis overgrows the other organisms in the mixed infection. Cushing says: "Undoubtedly the colon bacilli, being more in evidence, were frequently held responsible for peritoneal infection due to more virulent but culturally less active organisms."

Case XIV is recorded as showing an infection with colon bacilli and "other varieties of bacteria." Case

⁹ *Annales de l'Institut Pasteur*, vol. vii, p. 141.

¹⁰ *Johns Hopkins Hospital Reports*, 1895, vol. 1.

¹¹ *Philadelphia Medical Journal*, March 3, 1900.

¹² *Glasgow Medical Journal*, vol. xlvii, p. 97.

¹³ *Johns Hopkins Hospital Reports*, 1895, vol. 1.

¹⁴ *Philadelphia Medical Journal*, March 3, 1900.

XX is recorded as having turbid peritoneal fluid with fibrin flakes, infection with colon bacilli. Case XXI, resulting from a ruptured mesenteric gland, gave a pure culture of bacillus typhosus. Case XV is reported to have been a specially foul-appearing general peritonitis, in which the infection was colon bacilli, and "many others not differentiated." The 2 cases (Cases V and XXI) in which the peritonitis was found to be due to a pure infection of bacillus typhosus died—1 within twelve hours and 1 on the fourth day.

The finding of the bacillus typhosus as the infecting organism in cases of peritonitis is exceedingly rare. At most, but 5 cases, including the 2 cases reported in this series, have been published. It may be suspected, reasoning from the evidence furnished by Case V and Case XXI, that when it does occur it is probably virulent and fatal.

DIAGNOSIS.

The diagnosis of the peritoneal infection within a sufficiently reasonable time from its beginning is a question of the greatest importance to physicians and surgeons. So far, our study of these cases has shown us that the diagnosis in 14 cases out of 24 was apparently made later than it might have been, and in 7 cases only once was it made as soon as possible, and that in 3 cases a mistaken diagnosis was made.

It seems fair to suppose that the majority of cases of intestinal perforation present a slow intestinal infection or a gradual onset, or that actual perforation may take place without fulminating symptoms. Cushing has referred to what he calls a "preperforative stage," or a "pre-extravasation stage," and has attributed the early complaints of pain to a slight involvement of the serosa and possibly to omental adhesions. He understands by his "preperforative stage" the "whole period between the first involvement of the serosa, with the formation of customary adhesions at this point, until these adhesions (which may for a time constitute the floor of the ulcer after the serosa has given way) have themselves become broken down, and general peritonitis has taken place." This condition of things is well illustrated in our Case IV, which is a typical case of pre-extravasation, because of delicate protecting adhesions.

It would seem, however, from a study of our cases, that the formation of protecting adhesions in perforation or threatened perforation of the ileum is exceedingly rare. Monad and Van Verts¹⁶ state that in their study of this condition, adhesions, however slight, almost never occur when the perforation is situated in the ileum, and they further state that peritoneal infection is not always a rapid fulminating affair; that it may occur slowly from what they call "propagation"—that is, migration of the bacteria through the necrosed and damaged intestinal coats—and that this form of infection is no less worthy of consideration than the rapid infection from sudden intestinal rupture and extravasation of intestinal contents.

Intestinal ulcers certainly may have a preperforative stage, and by this we mean a stage of local peritoneal infection and bacterial involvement of the peritoneal coat—whether or not adhesions exist. This local infection may cause general infection without adhesion formation and without perforation. The

pre-extravasation stage properly means a period of time after local peritonitis has taken place in which there is no extravasation of intestinal contents, either because of the necrosed portion of the intestinal wall remaining in place without dropping out, as in Case XVI or in Case II of our series, or because of adhesions, as in Case IV of our series. Local infection of the peritoneum causes pain of greater or less severity. This pain may not be appreciated and complained of in very sick and stupid typhoids, but in mild cases may be just as prominent a feature as the pain from the local peritoneal infection of appendicitis before perforation has taken place.

Our series of cases shows that this early pain was absent in only 7 cases out of 21; 5 of these 7 cases were mild cases, in which sudden rupture of the serosa and extravasation took place; 2 cases with equally sudden symptoms had a septic peritonitis already under way, which cases were so sick and stupid that it was impossible to diagnose the beginning of the general infection.

Fourteen cases, as has been seen, had early warning pain, earlier by a definite number of hours than the severe symptoms, and all these cases were found to be far advanced in general peritonitis at the time of operation. These early symptoms undoubtedly meant beginning peritoneal infection, as is shown by the after development, and the severe symptoms meant general septic peritonitis then existing. Whether these early symptoms meant a necrosed serosa without perforation, or a pin-point perforation protected or not protected by delicate adhesions, it is positive that general septic peritoneal infection started at a time coincident with these early symptoms and not coincident with these severe ones.

It must be admitted that these 14 cases had advanced general peritonitis at the time of operation; that they had signs and symptoms antedating the symptoms of peritonitis; that the severe symptoms in the cases of actual perforation differed in no way from the severe symptoms in the cases of general peritonitis from other causes of infection; and if this statement is true, then the diagnosis of these cases should have been made before these severe symptoms set in, and, therefore, a decision as to what symptoms are of importance in making a sufficiently early diagnosis of peritoneal infection in these cases is of the utmost necessity.

From a study of these cases and also of the cases reported by Cushing it would seem that complaints of abdominal pain in any typhoid case, especially if localized, and especially in cases of mild type, should be regarded as serious danger signals. We have already seen that in our sick and septic cases the condition of the patient may be such that moderate pain may not be noted, and that therefore a complaint of abdominal pain on the part of such a sick case may mean an established general peritonitis. (See Cases II and VI.)

In the mild cases of typhoid complaint of abdominal pain should lead to a strong suspicion of beginning peritoneal infection through a preperforative stage, or of a slow peritoneal infection through perforation, with or without temporary adhesions; or of a beginning infection from some other source than the intestine, as in Case XXI. This plan may vary in severity and locality. It is most frequent in the right iliac region, and is most often associated with

¹⁶ *Revue de Chirurgie*, 1897, p. 169.

localized tenderness and spasm. To confirm the suspicion of beginning peritoneal infection a leucocytosis, steadily rising, during a few hourly counts, at least, should accompany this pain. Vomiting is rare. The temperature generally rises and the pulse also.

It cannot be too strongly emphasized that abdominal pain, especially if localized, complained of by the patient in the mild or moderately severe typhoids, is not a frequent occurrence unless it means peritoneal infection, localized or general. By pain we do not mean the discomfort or uneasiness accompanying meteorism or distention by gas.

We have made a study, bearing upon this point, of 70 cases of typhoid fever at the Massachusetts General Hospital occurring in the last two years, and have found, according to the records, that in the majority of cases of the mild and moderately severe typhoid no genuine abdominal pain whatever was complained of, even in the presence of distention, diarrhea and hemorrhage. Seventy cases: 20 severe typhoids and 50 mild; 7 cases died. In only 8 cases was any abdominal pain recorded as a complaint (11.4 per cent.), and 4 of these cases died (1 case with probable certain perforation or peritonitis, 1 of pneumonia, and 2 cases with suspicious symptoms of peritonitis). The other 4 cases had slight and transient abdominal pain, and recovered. Abdominal distention of more or less extent was noted in 10 cases, without any pain. Intestinal hemorrhages are noted in 8 cases, but pain accompanied these in only 1 case. Diarrhea is recorded as marked in 3 cases, but in only 1 was any pain, and in this slight.

With reference to this same point the records of the Boston City Hospital have been gone over for a year. Pain is recorded as complained of in 22 cases out of a total of 160 cases; of these 22 cases 5 are included among those operated on, which leaves 17 cases with pain, or about 10.6 per cent. of the total.

Localized muscular spasm and localized tenderness are diagnostic signs of the utmost importance associated with pain and leucocytosis.

The question as to whether the site of the perforation or the region of the beginning peritoneal infection materially affects the prognosis both of unoperated and operated cases is one that needs discussion. The fact that the appendix may be the seat of the typhoid perforation was first noted by Dr. R. H. Fitz, and the relation of this fact to the prognosis in cases of typhoid perforation was thus stated by him: "The probability of its occurrence (perforative appendicitis) furnishes the best solution to the prognosis of intestinal perforation in the latter disease (typhoid fever). Most cases of recovery from symptoms of perforation of the bowel in typhoid fever are those in which an attack of appendicitis is most closely simulated, while the fatal cases of perforation of the bowel in typhoid fever are, in the great majority of instances, those in which other parts of the bowel than the appendix are the seat of the perforation."

This set of cases does not furnish any case in which the perforation was appendicular. Fitz found this variety of perforation in 2 per cent. of 167 cases. In a series of cases operated upon — that is, our own set, 24; Cushing's, 8, and Finney's, 3; 35 in all — no instance occurs. Gairdner's¹⁶ 47 cases included only one of *perforated appendix*. Perforation in the appendix can only have a more favorable prognosis

because of its occurrence in a fixed portion of the bowel in a remote region where the chances of the peritoneal infection being shut up by protecting and life-saving adhesions prevail.

It is certainly misleading to state that most cases of recovery from symptoms of perforation are those in which an attack of appendicitis is closely simulated, because — as can be plainly seen from a study of our list of cases — many of them had beginning symptoms which suggested appendicitis, and yet the perforation was in the ileum, and the peritoneal infection became general and fatal. It is imperative that we should not be prevailed upon to wait operation in the hope that the perforation may be in the appendix, considering that the chance of localizing the infection by delay offers more hope to the patient than speedy operation, because the attack began with right iliac pain and tenderness and muscular spasm. The better teaching is that perforation of the appendix may occur in typhoid, although *rarely*; that when it does occur the infection may be localized, as in any cases of perforating appendicitis; but the great majority of cases of perforation of the bowel in typhoid fever may simulate an acute appendicitis.

A study of our whole list of 27 cases shows the following: Eleven cases had initial symptoms referred to the right iliac region, with one or more of the symptoms of localized pain, tenderness and spasm. Any one of these cases might have been suspected as the appendicular variety, and delay counselled because of the apparent localization of the signs; but 9 cases were perforations of the ileum, followed in all but 1 by general infection, and 2 had no cause for symptoms. In only 3 cases pain and tenderness, etc., were initial in the left iliac region. One was a case of general peritonitis from a suppurating gland, and the other 2 cases were very sick cases, in which the peritonitis had become extensive and general before any symptoms were noted. In 7 cases the pain and tenderness with rigidity were general. In 1 case locality of pain is not given. Therefore, we may conclude that the majority of cases of perforation have initial symptoms referred to the right iliac region; that in a very small percentage of all such cases is the perforation in the appendix; that general abdominal pain and symptoms, or symptoms and signs referred to the left side, point to the existence of a general peritonitis, the onset of which has been gradual and not definitely noted.

LEUCOCYTOSIS AS A DIAGNOSTIC SYMPTOM.

A careful comparative white blood count at frequent intervals in the presence of abdominal pain in typhoid may furnish the most valuable evidence of beginning peritoneal infection. A full and complete knowledge of leucocytosis in typhoid peritonitis has not been reached. Thayer, in 1892, first showed that in typhoid fever the white blood corpuscles are less than normal, or, in other words, that a condition of hypoleucocytosis exists, and that the smallest number of leucocytes is usually found at a period of time corresponding to the height of the fever. Cabot, in 1897, reiterated these facts and pointed out that a low leucocyte count in typhoid meant freedom from septic complications. Our study of 70 typhoid cases at the Massachusetts General Hospital is merely corroborative of the statements of Cabot and Thayer. The majority of cases at entrance had a hypoleucocytosis

¹⁶ Glasgow Medical Journal, 1897, vol. xlvii.

of about 4,000; the lowest count in an uncomplicated case was 1,800. Septic complications, as phlebitis and otitis media, caused a leucocytosis. In nearly all the cases, however, only one white count—the one at entrance—is recorded; and even when this early count was high—over 9,000—no other record is available. Certainly more frequent counts should be made in the future. The fact that leucocytosis in typhoid means inflammatory complications in the majority of instances, and that leucocytosis was present in peritoneal infection, was noted by Cabot (1897); and Finney, in his paper on "Typhoid Perforation" (1897), considered the most important symptoms of perforating typhoid ulcer to be "marked increase in the number of white blood corpuscles in association with continued abdominal pain." It does not appear that a full realization of the importance of a falling leucocyte count or the absence of leucocytosis in general peritoneal infection is held by a majority of medical men. Cabot stated that certain cases of general peritonitis would show no leucocytosis because of their being too sick to react against the infection.

Cushing, in his two papers on this subject, seems to have most carefully studied leucocytosis as a symptom, and his work strongly suggests that a white blood count, steadily rising from the first complaint of abdominal pain (which is confessedly the first warning of local or general infection), with certain fluctuations, reaches a maximum point, and then as steadily falls. This he aptly calls the wave of leucocytosis, and raises the question as to whether a falling count or an absence of leucocytosis (other symptoms being present) may not be diagnostic of a well-established general septic peritonitis. He considered it rational to suppose that the enormous migration of leucocytes to enter into the seropurulent exudation was a sufficient reason for the falling or deficient white blood count in general peritonitis. This point is a very important one to settle, for diagnostic reasons. Cushing's conclusions from a careful study of leucocytosis in his cases are here given:

"If this septic complication, namely, complication causing leucocytosis, is a peritonitis which remains localized, associated possibly with a preperforative stage of ulceration or with a circumscribed, slowly forming peritonitis after a perforation, it may be and usually is signalized by an increase of leucocytes in the peripheral circulation. If, however, a general septic peritonitis follows, the leucocytosis may be transitory and overlooked, as it disappears concomitantly with the great outpouring of leucocytes into the general peritoneal cavity.

"In order that the white blood count be of any diagnostic value whatever in the presence of abdominal symptoms it is essential that, for the purpose of comparison, previous counts have been made. An individual count at such a critical time may be misleading—in the first place, because a condition of hypoleucocytosis often is a characteristic of the fever, and a count comparatively within normal limits, of from 8,000 to 10,000, may actually represent a double increase in the number. Secondly, because by a single count the transient wave of leucocytosis may entirely escape notice.

"The flood-tide of this increase is a transient affair, lasting but a few hours in some cases, and may in consequence entirely escape observation, since there

ordinarily is a following rapid diminution in number, corresponding to the spread of peritoneal infection. In some instances, when the process for the time remains localized, and in cases in which the preperforative stage exists, the leucocytosis may be of longer duration, the condition being analogous to that associated with acute appendicitis (typhoid or otherwise) before perforative peritonitis has occurred. Suppurative complications of typhoid, here or in other situations where they remain localized processes, have ordinarily a persisting leucocytosis, as is well known."

These conclusions of Cushing are reinforced by careful observations, and seem to give the best interpretation of leucocytosis in this class of cases. Whether absence of leucocytosis in the face of symptoms of peritonitis means well-advanced general peritonitis in every case has not been proven absolutely. Certainly one sure thing is that a single isolated blood count on the appearance of abdominal symptoms may be of little or no value. In one of Cushing's cases of perforation an hourly blood count showed that the white corpuscles rose in two hours from 4,800 to 10,400, and in an hour and a half later dropped to 7,000. In another of Cushing's cases of intestinal perforation a drop of 20,000 in three hours after the probable time of perforation is noted. The appearance of leucocytosis in the presence of abdominal pain may, however, be misleading as a diagnostic symptom of peritoneal infection.

As is well known, the existence of a septic focus, an abscess, or a septic phlebitis elsewhere might furnish cause for the leucocytosis coincident with abdominal symptoms. One or more cases have been reported in which the symptoms of peritoneal infection, including leucocytosis, led to an exploratory laparotomy, with negative findings. Cushing has had such a case. Case XXIII in our series is also such a one. Finney has been led to an exploratory laparotomy in which the symptoms suggesting intestinal perforation, including a leucocytosis, were probably due to an iliac thrombophlebitis. This case recovered. It has been found by Cushing that a post-operative leucocytosis occurs after all abdominal operations.

With these facts and theories in mind as representing the latest opinions in regard to leucocytosis as a symptom, reference to our series of cases with a view to find corroboration of these facts furnishes very little of value because of the lack of comparative counts. In almost all of the cases only one blood count was made.

To take our cases in detail, the leucocyte count is only recorded in 11 cases of the 21 in which perforation or peritonitis was found. In the first set of cases, namely, those without warning symptoms, the leucocyte count is recorded in 4 cases. In Case IV (the case of perforation protected by local adhesions) a leucocytosis of 14,300 was found at an unknown number of hours after the onset of abdominal symptoms. No other count is recorded. In Case V one count at the onset of symptoms showed 6,000; no other count. Case VI showed a leucocytosis of 13,200. This case was one in which perforation and localized abscess formation took place without any diagnostic symptoms at a time probably remotely preceding the severe symptoms which demanded operation. Occasional comparative leucocyte counts as a matter of medical routine might have led to the suspicion of a septic complication. Case VII showed a white count of 8,600 at the onset

of the symptoms; no other count is recorded. The other 3 cases of our first set have no record of any leucocyte count.

Taking our second set of cases, namely the 14 in which premonitory symptoms existed, as we have seen, it would have been of the *greatest importance diagnostically* to have known what the white blood count had been for several days before the symptoms, and also to have known what the white blood count at early intervals after the occurrence of abdominal pain was; yet in only 2 cases was there any leucocyte count taken at this time, and then only one isolated count in each case is recorded.

Case XVI had a white blood count of 6,900 two days before he complained of abdominal pain; at the first complaint of pain a count of 8,000. Then no further leucocyte count was taken for twenty-four hours; but when severe symptoms (which meant general peritonitis) set in a white count equalled 17,200. Certainly in this case frequent and comparative blood counts might have contributed much to our knowledge of a rising and falling count. Case XV, seen after the warning complaint of pain, had a white blood count of 31,000. Twenty-four hours after, when general peritonitis had set in, the white count had dropped to 23,000 and 17,000. Only these three counts are recorded.

It is much to be regretted that in this series of cases in which 14 had premonitory symptoms, such slight knowledge in regard to the diagnostic value of the white blood count is available. In Case XIV the blood count at the time of the first abdominal symptoms showed 10,000 whites, but no other count was taken. Six cases of the 14 record no blood count at the occurrence of either mild or severe symptoms. In 6 cases the only blood count was made when severe symptoms appeared. In Case XII, a case of extensive peritonitis at operation, the white cells were 3,800. In Case XIII, also a case of extensive peritonitis, the white cells were 5,400. In Case VIII, also a case of general peritonitis, the white cells were 7,300. In Case II they were 8,000 just before operation.

Here are 4 cases in which an isolated blood count, taken when peritonitis had become general, showed no leucocytosis. Whether a previous leucocytosis had existed, and whether the absence of leucocytosis in the presence of general peritonitis is a diagnostic sign, cannot be determined because of the absence of comparative counts in all of these cases.

Case IX had a leucocytosis of 16,000 after general peritonitis was evident. Ten days before the count was 14,000. No interval counts are recorded.

Case X had a white count of 14,000 with the symptoms of general peritonitis. At entrance to the hospital, ten days before, his white count was 6,500. No other counts are noted.

It is not possible to establish anything new and valuable in regard to leucocytosis as a symptom from the study of these cases, because of the lack of sufficient data. The one conclusion is that the leucocyte counts in typhoid cases should be sufficiently frequent to be of comparative value; that any complaint of abdominal pain or the appearance of abdominal symptoms demand frequent counts.

The question of diagnosis and the relative value of abdominal symptoms and leucocytosis as deciding for operation is illustrated in a negative way in Cases

XXII, XXIII and XXIV. Cases XXII and XXIII were mild or moderately severe typhoids, practically convalescent in the fourth and sixth weeks of the disease. Case XXII had no leucocyte count, and it seems questionable whether it would be justifiable to operate upon a case with such abdominal symptoms without the corroborative evidence of a leucocyte count. It has been done, however, in other cases (the case reported by Herrington and Bowlby¹⁷). Case XXIII had practically the same abdominal signs and symptoms as Case XXII, except that there is no record of the temperature at this time, which in Case XXII was rising from a normal curve, and Case XXIII had also a leucocytosis of 12,000 one hour before the operation. While this leucocytosis is not large, the white count had risen from 9,000 twelve hours before the operation. Neither of these 2 cases seems to have been very sick or to have had any great amount of shock. Case XXIV seems to have been a case of typhoid septicemia, in which no certain cause for the abdominal symptoms was found. No leucocyte count and no bacteriological cultures are recorded in this case. Probably he died from typhoid fever, although the presence of a green, necrotic fibrin patch found on a loop of the intestines at the operation leaves room for the thought that the abdominal symptoms and death may have been caused by a virulent peritoneal infection.

RESULTS.

Of the 21 cases in which the local or general peritoneal infection was found at operation 3 cases recovered absolutely—that is, 14.3 per cent. recovered. In Case IV—a case of operation in the pre-extravasation stage—it is noted by Dr. F. S. Watson that the protecting adhesions were very slight and delicate, and that in Dr. Watson's opinion they would not have long resisted fecal extravasation.

The 2 other cases which completely recovered are Cases XVI and XVII, both cases of general septic peritonitis. Together with Abbe's¹⁸ case they prove that it is not impossible to save cases of general peritonitis in typhoid fever. The bacterial infection, however, in both of these cases, as well as in the case of Abbe's, was not determined.

Case I properly should be classed as a recovery from an operation for intestinal perforation in typhoid, for death occurred from intestinal hemorrhage five days after the operation, and the autopsy showed the suture tight and no general peritonitis. If this case can be classed as a recovery, we have four recoveries from operation from acute peritoneal infection in typhoid, or 19 per cent.

Case IX cannot be considered as recovering, but it is a case of death caused by giving way of the intestinal sutures put in at the first operation.

Taking our 24 cases of typhoid fever which were operated on, 21 with abdominal infection and 3 with no infection found at operation, we have 6 cases of recovery from laparotomy in typhoid fever, or 25 per cent.

Cases XXII and XXIII are noteworthy as showing that the mere abdominal operation in a slight or moderately severe typhoid is not a very fatal thing when no peritoneal infection is present. Case XXIII was also not a very favorable subject for any opera-

¹⁷ Medico-Chirurgical Transactions, London, vol. lxxx, p. 127.

¹⁸ Medical Record, 1895, vol. xlvii, p. 1.

tion, because of cardiac and renal complications, yet both these cases made uneventful recoveries from the operation. In Case XXII an appendectomy was done for an old chronic process in the appendix. In Case XXIII the operation was simply exploratory. Two of Cushing's reported cases further show that an exploratory laparotomy or a simple appendectomy in a mild or moderate typhoid is usually well borne. We have seen that the 5 cases (Cases I, III, IV, V and VII) were especially favorable cases for operation. Three of these died, and they were all cases that could have stood laparotomy just as well as Case XXII or Case XXIII had it not been for the peritoneal infection, the nature of which was undetermined in 2 cases and found to be the bacillus typhosus in 1. These 3 fatal favorable cases point to the fact that a fatal peritonitis may set in within from one to two hours after perforation. Of these 3 cases Case III died on the third day, Case V on the fourth day and Case VII on the second day.

Of the 14 cases in our second set, in all of which operation found more or less extensive peritonitis, and in regard to which it may be said that the operation was directed against the peritonitis primarily, 12 died; 1 lived eight days and died from secondary extravasation. All the others died within fifty hours after the operation; 2 within six hours, 7 within thirty-six hours, and 1 on the operating-table.

If we grant, for the sake of argument, that the sick typhoids, 6 in number, might have died from their typhoidal condition complicated by an abdominal operation, even if peritonitis had not been present, we still have 8 mild cases in which the cause of death was probably a peritoneal infection, and not the operation itself.

Calculating statistics of the operative treatment of general peritonitis from any cause is foolish business. The chances for success in such treatment depend on so many factors: the amount and virulence of the infection, the condition and resisting powers of the patient, the differing operative technique of different men, the time of operation, the kind and amount of anesthetic used, etc. In trying to find out why some of these cases lived and some died it would be well to take only the 15 mild cases, throwing out for the time being the 6 severely sick typhoids as doubtful cases for standing the shock of the abdominal operation, leaving ourselves 15 mild typhoids for consideration. Of these, 5 had perforation without any warning symptoms, and were operated upon very speedily thereafter; 2 recovered, 1 because the operation was done in the pre-extravasation stage—a rare event; 3 died from peritoneal infection from the intestinal extravasation, although no macroscopical signs—or, at most, slight signs—of general peritonitis were present at the operation. Ten of these mild cases had general septic peritonitis at the time of operation; 2 recovered.

When we consider the reasons for the recovery of 2 of these cases and the death of 8 we are brought face to face again with insufficient recorded data. The time of operation is recorded in only 3 of the fatal cases: 1 case, thirty minutes; 1 case, thirty-five minutes; 1 case, one and a half hours.

The time of operation in the cases that recovered is not noted. The kind of bacterial infection in the cases that recovered is unknown. In 1 case that died it was an infection of bacillus typhosus; in 1 case infection of the colon bacillus and other doubtful bacteria.

Ether was the anesthetic in every case; but the amount given, and the time the patient was kept under it, and the effect on the pulse are in no case recorded.

Cushing has enthusiastically endorsed the use of cocaine anesthesia in exploratory laparotomies in acute abdominal infection in typhoid. He has not, however, proved in his article that the majority of his cases were any better off than the cases operated upon under ether. It is not the feeling of this Committee that operation under cocaine anesthesia is advisable in cases of mild typhoid whose general condition of mind and body is fairly good, because rationally the shock to the mental and physical organism from an abdominal operation, with all its attendant manipulation, to a conscious patient must be greater than the judicious administration of ether. In certain cases of advanced peritonitis in which the patient is sick or nearly moribund, stupid and unintelligent, the use of local anesthesia may be advantageous.

Irrigation of the peritoneal cavity with hot salt solution or hot sterile water was practised in every case of general peritonitis but 1, namely, Case XVI. In this case careful wiping out of the cavity with gauze was done. Drainage with gauze or with tube and gauze was used in every case but 2, both of which died. It does not appear from available data that these variations in technique affected the result. The surgical opinion is practically unanimous the world over, that the operation for septic peritonitis must include free incisions, careful cleaning of the abdominal cavity by wiping, irrigation and free drainage.

It is impossible to work out by percentages the chances of recovery in cases of intestinal perforation and peritoneal infection in typhoid fever. It is a question of peritoneal infection (kind, amount and virulence), together with the condition and resisting powers of the patient, who has already contended with another disease. It is not the mere fact of operating in these cases that kills them, and it cannot be said that the typhoidal condition forbids an operation. It has been shown that a majority of the cases of perforation and infection are mild typhoids, and it has also been shown that abdominal operation in the absence of infection is not necessarily fatal, but, on the contrary, is well borne. The peritoneal infection is the thing that kills in the majority of cases.

A peritonitis from a perforation of the intestine in typhoid may be caused by virulent or non-virulent organisms just as in any other perforation of the bowel. Most of the typhoid perforations operated upon in this series of cases and all cases hitherto published have had a general peritonitis that would kill a majority of the men who had contracted it from other causes than typhoid, namely, perforated appendix or wounds. General peritonitis of the intestinal variety in a strong man, without typhoid fever, is a very fatal affair, and the chances of a recovery from operation in any case cannot be established by statistics except in a general way.

CONCLUSIONS.

This series, unfortunately, leaves much to be desired in the way of accurate and carefully reported study of many of the cases in the light of the most recent experience; but from the foregoing analysis of the material we can definitely conclude that:

(1) In many very sick typhoids perforation or

peritoneal infection cannot be diagnosed until the results are already widespread and of fatal extent. The chances of a fatal issue from an abdominal operation in such cases are overwhelming.

(2) In mild typhoids of fair general condition an abdominal operation is readily borne, provided no peritoneal infection is present.

(3) A small number of mild typhoids may have sudden perforation with free extravasation. In these the symptoms are fulminant, but localized to a great extent, and in these

(4) Operation must be done at once, for general infection may become past relief in from one to five hours, and walling off of the perforation by protecting adhesions is so rare as not to be counted upon.

(5) In the majority of mild cases, beginning infection (whether from perforation or not) is marked by comparatively slight symptoms—local pain, tenderness, spasm and leucocytosis. The severe following symptoms mean general peritonitis.

(6) These warning symptoms demand serious consideration and study, but in many cases are either not rightly understood or not acted upon.

(7) Complaint of abdominal pain in a case of typhoid should always lead to a suspicion of beginning peritoneal infection.

(8) Frequent leucocyte counts are needed in every case of typhoid. In the presence of abdominal pain an hourly count is necessary.

(9) Pain associated with local tenderness and muscular spasm and a rising white blood count points in most cases to an operation; in all cases to a surgical consultation.

(10) In not a few of this series of cases operation was imperative a varying number of hours before it was done.

If it can be appreciated that the severe symptoms more often mean general peritonitis, it must be understood that the milder and earlier symptoms are the important ones.

REPORT OF CASES.

CASE I. Seeley, male, age thirty-two years, white. Drs. Bowditch and Thorndike, Boston City Hospital, September, 1899. Sudden perforation. Operation within three hours from first symptoms. No general peritonitis. Death five days after from intestinal hemorrhage. Uneventful until the sudden abdominal symptoms. Mild course. Widal positive. Diazo (?). No complications. No abdominal complaints. Not a sick man. End of sixth week, forty-second day, operation. Ether. Time not known. Incision, right iliac. Irrigation. Gauze drainage. Sudden intense abdominal pain, not localized. Marked shock. Pulse 150. Temperature 102.6°. No leucocyte count recorded. No fall of temperature. No vomiting. Operation three hours after. No general peritonitis. Intestinal contents in abdominal cavity; fluid fecal matter. Perforation in ileum, two feet from cecum. Closed with silk. No cultures. Excellent result for five days. Operation for the perforation successful. Five days after, excessive intestinal hemorrhage causing death. The autopsy showed that the operation was a success. No general peritonitis. The suture was intact and protected by adherent intestines with slight local peritonitis, from which the colon bacillus and staphylococcus albus were grown. Cultures from rest of abdominal cavity negative.

CASE II. Bowler, male, age twenty-five years, negro. Drs. G. B. Shattuck and F. B. Lund. Boston City Hospital, October, 1899. A case of general septic peritonitis from necrosed patches of peritoneum, *without perforation*. Symptoms of perforation, however. Moribund at opera-

tion. Death. Severe course. Widal positive. Diazo (?). Abdominal pain constant. Fourth week, twenty-second day. Operation. Ether. Time (?). Incision, right iliac. Irrigation and drainage. Very sick case. Temperature high. Delirious. Abdominal pain and tympanites at frequent intervals. Two intestinal hemorrhages before operation (two days before). Sudden chill, shock, collapse, rising temperature and pulse. Distention. No vomiting. Operated on seven hours after, when in moribund condition. No leucocyte count. No perforation. Two necrosed patches in wall of ileum over ulcers. General peritonitis. No cultures made. Gas and serous fluid escaped. Intestines clean and not injected. Several necrotic areas. Necrosed places, inverted with silk. Death in eighteen hours. Cause, peritoneal infection and typhoid state. Compare this case to Case XVI. This case had sudden severe symptoms from peritonitis without perforation. An unfavorable case from all points of view. Peritoneal infection from necrosed patches, probably virulent, whereas in Scappacci's case infection from necrosed patches probably non-purulent, with the abdominal pain and distention marked. Specially associated with hemorrhages. Careful white blood count at frequent intervals might have been suggestive.

CASE III. McCann, male, age thirty years, white. Dr. F. C. Shattuck and Dr. Warren. Massachusetts General Hospital, January, 1899. Sudden perforation without warning. Operation within two hours. Death in three days. Mild course. Widal positive. Diazo positive. Bronchitis. No abdominal complaints. Fifth week, thirtieth day, incision, right iliac. Irrigation. Drainage, gauze. Ether. Time of operation (?). Mild fever complicated by hemorrhagic bronchitis. Intestinal hemorrhage two days before perforation. No warning. Patient comfortable, when sudden sharp pain in right iliac region with great tenderness and muscular spasm. No white count at this time. White count two days before 3,800. White count at entrance, ten days earlier, 10,000. No vomiting. Temperature subnormal, but began soon to rise. Pulse below 100. Operation one and a half hours after. Perforation in ileum, site not mentioned. No general peritonitis. Some free fluid. No bacteriology. Death on third day, in sixty hours, with symptoms of general peritonitis. No autopsy.

CASE IV. Scott, male, age twenty-eight years, white. Drs. Withington and Watson. August and September, 1895. Perforation. Localized peritonitis. Operation in pre-extravasation stage. Recovery. Mild course. No Widal test made. Diazo positive. Rose spots. Eleventh week in a relapse. Operation. Ether. Time (?). Incision right iliac over induration. No irrigation. No drainage. Long course, one or two relapses. No special complications. Twenty-three days before perforation, two or three hemorrhages. No abdominal pain recorded, but some tympanites. Without warning sudden severe pain in right iliac region at 2 A. M. Temperature did not fall, but rose. Pain steadily worse. Pulse rapid and weak. Some cyanosis and prostration. Muscle spasm on right side of abdomen. Dulness and indurated feeling to right of umbilicus, "size of hand." No distention. No vomiting. "The symptoms of local pain and rigidity increased while patient's general condition improved." Leucocytosis 14,350. Operation twelve hours after first pain. A loop of ileum adherent to parietal peritoneum around a perforation size of a dime; well-defined area of local peritonitis. Limiting adhesions delicate. "Every probability that these adhesions would not have resisted fecal extravasation long and that a general peritonitis would have resulted if case was left to itself." (Watson.) Perforation closed. Recovery uneventful. No cultures.

CASE V. Dolan, female, age twenty-five years, white. Drs. George B. Shattuck and Lund. Boston City Hospital, November, 1899. Perforation. Beginning general infection with typhoid bacillus. No definite warning symptoms. Death. Mild course. Widal positive. No complications. No abdominal complaints. In second week, twelfth day. Operation. Ether. Time, thirty min-

utes. Incision, median. Irrigation. Drainage. Uneventful till twenty-six hours before operation, then hard chill (no other record at this time). Next A. M. (twelve hours after) several chills. Twenty-two hours after first chill and four hours before operation abdominal pain above pubes. Tenderness and spasm (more on right). Temperature fell to 98°, but rose at once. No distention. Pulse 120. No vomiting. Nausea. Leucocyte count, recorded as taken when pain complained of, 6,000. No other count. Perforation eight inches from cecum in ileum. Gas and turbid fluid in general abdominal cavity. General peritonitis not far advanced. Infection, pure typhoid bacillus. Perforation closed with silk, also two thin places enfolded. Death on fourth day, with symptoms of general peritonitis. Perforated coil of intestine was lying low down in pelvis. (Note locality of pain above pubes.) Perforation probably when pain was first noted. Operation reasonably soon. Typhoid infection, probably a virulent one. The general peritonitis was not far advanced at operation, yet was undeniably a general infection.

CASE VI. Devlin, male, age twenty-four years, white. Drs. F. H. Williams and Munro. Boston City Hospital, November, 1899. Perforation and local abscess formation, with no diagnostic symptoms, followed by general peritonitis with severe symptoms. Death. Severe course. Widal negative. Rose spots. Large spleen. No complications. In fourth week, twenty-sixth day, operation. Ether. Time (?). Incision, right iliac. Irrigation. Drainage. Case stupid and sick. Fever high. No abdominal complaints till special ones. Two severe intestinal hemorrhages on day before in A. M. and P. M., accompanied by hard chill and collapse. Very near death at this time. Unconscious twenty-four hours after this. Abdominal pain. Tenderness, mainly right iliac. Distention. Rigidity. No vomiting. Leucocytes 13,200. Temperature (?). Operation twenty-eight hours after hemorrhage. Operation four hours after pain, etc. General peritonitis with cloudy fluid; pus and feces in left pelvis free. In right iliac region an abscess cavity walled off by fairly strong adhesions, filled with pus, blood and feces; large ragged hole in ileum near cecum. No cultures recorded. Perforation closed. Death in forty-eight hours from general peritonitis. No autopsy. This case was one of perforation and forming of a walled-off abscess with no symptoms complained of by a very stupid, sick man. Time of extension to a general peritoneal infection doubtful. No cultures from general peritoneal fluid, as well as from local abscess, were taken.

CASE VII. Marchiono, male, age thirty years, white. Drs. Henry Jackson and Munro. Boston City Hospital, July, 1898. Perforation. Sudden onset. Peritoneal infection not known. Quick operation. Death. Mild case. Widal positive. Diazo (?). No complications. No abdominal complaints. In third week, twentieth day, operation. Ether. Time, one-half hour. Incision, right iliac. Irrigation. Drainage. Ten A. M. pain complained of in abdomen, with no special tenderness, nothing localized. No spasm. Leucocyte count then 8,640, not taken again (?); 1.30 P. M. severe pain and chill, collapse. Abdomen everywhere tender and rigid, but especially on right side. Fall of temperature recorded to 98°, rising soon to 105°. No vomiting. Operation eight hours after first pain, five hours after collapse. One perforation in ileum ten inches from cecum. No free fluid. "No extensive signs of peritonitis." Perforation closed. No cultures recorded. Death in forty-eight hours without symptoms of peritonitis. Partial autopsy through wound. Slight microscopic signs of general peritonitis, but no bacteriology. Cause of death (?). Infection not known. In this case quick operation after first symptoms. Beginning peritonitis.

CASE VIII. Schick, male, age sixteen years, white. Dr. Gannett and Dr. Mixer. Massachusetts General Hospital, August, 1897. Case of perforation. Symptoms of peritonitis. Perforation probably took place some hours before first symptoms. Death. Severe course. Widal positive. Diazo positive. End of third week, twen-

ty-first day, operation. Ether. Time (?). Incision, median. Irrigation. Drainage, gauze. Albumin, a trace in urine. Mitral regurgitation. Compensated. White count on entrance 4,000. High temperature and great abdominal distention all the time in the hospital, but no pain till August 20th (the day before severe symptoms) — that is, twenty hours before operation sharp pain in the left iliac region. No white count. Temperature falling gradually to 99.4° the following evening. No record of close watch of case. Next A. M. severe symptoms. Very great pain. Great distention, "board-like abdomen, most tenderness left iliac," bad pulse, anxious facies, no vomiting. White count 7,300. Perforation eight inches from cecum in ileum. Virulent general peritonitis, pus and feces. Intestines covered with fibrin. No cultures. "Peritonitis of longer standing than twenty hours." Death in fifty hours. Peritonitis, spreading from a local area of infection or from perforation which took place without being noted. Small leucocyte count, possibly due to extensive purulent peritonitis. No comparative counts except one at entrance.

CASE IX. Hanahan, male, age twenty-seven years, white. Drs. Fitz and Beach. Massachusetts General Hospital, November, 1897. Perforation. General peritonitis. Operation. "Second perforation" or giving way of sutured area eight days after second operation. Death. Mild course. Widal positive. Diazo positive. First of third week, operation. Ether. Time, thirty minutes. Incision, median. Irrigation. No drainage. Slight bronchitis. Trace of albumin and hyaline and granular casts in the urine. Nothing pointing to the abdomen save a little distention. No intestinal hemorrhages till after the first operation. December 6th, distention and tenderness to the right of the umbilicus. Temperature rising, pulse steady, no white count then. Leucocytosis at entrance, November 27th, 14,100; November 29th, 14,200; December 8th, 16,000. December 7th, tenderness increasing and temperature rising. No white count. December 8th, gradually falling temperature and great increase in tenderness and distention of abdomen. Liver dullness obliterated. White count 16,000. General tenderness, pulse 130, temperature 104°. No vomiting. No complaint of pain. Operation next day (December 9th), about sixty hours after first warnings and after twenty-four hours after symptoms of general peritonitis. The infection was a slowly spreading one; the severe symptoms on December 8th were probably due to the general peritonitis. The condition on December 6th and 7th strongly suggests a pre-extravasation stage, but comparative blood counts are not recorded. Operation would certainly have been justified by these symptoms of peritonitis fully fifteen hours before it was actually performed. Perforation one-eighth inch in diameter, one inch from cecum in ileum (Beach). Closed. Foul gas and fecal fluid in belly. Yellow fibrin on intestines. No bacteriology. Did well after operation save for the continuation of typhoid temperature, averaging 102°, and three or four moderate intestinal hemorrhages. Some distention. Eight days after first operation pain all over belly, followed in three hours by collapse. Second operation found large necrotic area and ragged hole in the cecum which was, for a great space around ulcer, soft and gangrenous — "shows patches of necrosis." Peritoneal cavity contained much dark fluid, feces and blood. Died five hours after. Autopsy: Much foul fluid in the peritoneal cavity. Many ulcers in last three feet of ileum and in ascending colon. In cecum large necrotic areas, two small perforations, and two ragged large openings. In ileum no ulcer through to peritoneal coat, and no sutures found. Although this case has been considered a case of second perforation after recovery from the first perforation, the facts do not permit this view of it. The first perforation was said to be one inch from the cecum; the second operation and the autopsy found great necrotic softening and ragged holes in the cecum, and the autopsy found no ulcers in the ileum which were through to the peritoneal coat, and no trace of the silk sutures. The localities are practically coincident. It seems just to add that the case did well after the first operation until the

extensive necrotic changes in the cecum involving the sutured area caused giving way of sutures and second extravasation. Certainly eight days is too short a time in which a sutured ulcer could heal and vanish, and the silk sutures would not have disappeared.

CASE X. Hoyt, male, age nineteen years, white. Drs. F. C. Shattuck and C. B. Porter. Massachusetts General Hospital, October, 1896. Perforation. General peritonitis. No full study of early and warning symptoms recorded. Death. Mild course. No Widal test made. Diazo. No complications. No abdominal complaints. Thirteenth day, last of second week, operation. Ether. Time (?). Incision, median. No irrigation. Wiping. Gauze drains. No hemorrhages. Uneventful till October 26th. Pain in abdomen growing steadily worse. No collapse, no fall in temperature then. Pain steadily increasing. Legs drawn up. No records of any examination for spasm, etc., but records of morphine given. No white count. Eight hours after pain first noted symptoms of general peritonitis. Pulse 112, sweating, temperature down somewhat, pinched face, belly rigid, tenderness general. Liver dullness not obscured. No vomiting. White count 14,700. Operation eleven hours after first symptoms. Perforation in ileum "near cecum." This is called in record "a sloughing area from which gas was escaping, but no fecal matter." General peritonitis, seroturbid fluid of fecal odor. No bacteriology. Perforation infolded with two rows of sutures. Death in thirty-six hours after great distention with obstinate vomiting. The autopsy raises a speculation as to the cause of death — whether due to obstruction from too much infolding of intestines by the two rows of sutures. The lumen of intestine at the point of suture admitted the little finger-tip, but was obstructed by a hard, impacted mass of feces. Whites at entrance 6500. Eight hours after first pain 14,700.

CASE XI. Bresnahan, female, age forty years, white. Dr. C. A. Porter and Dr. Thompson. St. Elizabeth's Hospital, August, 1899. Perforation. Definite early symptoms, which were not considered important. Operation six hours after severe symptoms. Death. Mild type and course. No Widal test made. No diazo. Rose spots, etc. No complications. No abdominal complaints. In third week, operation. Ether. Thirty-five minutes. Incision, right. No second incision. Irrigation. Drained with gauze. Uneventful case. No intestinal hemorrhages. Twenty hours before operation, pain in abdomen with some tenderness. No white count. No examination through the day. Six and one-half hours before operation pain severe, pulse 135. Three and one-half hours before operation — that is, sixteen and one-half hours after first complaint of pain — symptoms of collapse (general peritonitis). Temperature 106°. Muscle spasm and tenderness in right iliac region. No vomiting. Leucocytes 8,000. One perforation six inches from cecum in ileum. V-shaped resection, silk sutures, one thin place inverted. General peritonitis. Free fluid of fecal odor. Injected intestines, with fibrin flakes. No cultures taken. Infection unknown. Death thirty-seven hours after. Partial autopsy through wound. Existence of other perforations unknown. Cause of death, peritoneal infection. Beginning infection probably when first pain, twenty hours before operation. No importance attached to early symptoms. Severe symptoms due to general peritoneal infection.

CASE XII. McDonald, female, age twenty-six years, white. Drs. Buckingham and Munro. Boston City Hospital, September, 1899. Case of perforation with excessive general peritonitis. Perforation and peritonitis much earlier than diagnosed. No symptoms save of general peritonitis. Death. Severe course. Widal positive. Diazo (?). No complications. No abdominal complaint. First of fifth week, thirtieth day, operation. Ether. Time of operation (?). Incision (?). Entered the Boston City Hospital in the fourth week. Two intestinal hemorrhages five days before "perforation." After these very sick, stupid. Pulse and temperature high. Some abdominal distention. No abdominal pain until the morning of September 18th, then pain and tenderness, requiring mor-

phine. Four hours after this great distention, general tenderness, chill, collapse, poor pulse, temperature fell to normal, but began at once to rise. No vomiting. Leucocytes 3,800 (note, general peritonitis with excess of fluid). No other count recorded, and no time of this given. Operation six hours after pain, two after collapse. Case "moribund" at operation. Perforation site not recorded. "Excessive general peritonitis, evidently of long standing; appearance of intestines and abdominal cavity showed general peritonitis of much longer standing than six hours" (Munro). No bacteriology. Death on operating table. No post-mortem. This case was "stupid" and sick, and illustrates that perforation and general peritonitis may be gradual, and in a sick and stupid patient unnoticed until extreme septic infection.

CASE XIII. Starnar, male, age twenty-five years, white. Drs. Gannett and C. B. Porter. Massachusetts General Hospital, October, 1899. Perforation gradual. General peritonitis of long duration. No very definite diagnostic symptoms. Death in two hours. Mild case. Widal positive. Diazo positive. No complications. First of fourth week or end of third, operation. Ether. Time, one and one-half hours. Incision, median. Irrigation. No drainage. In the hospital ten days before operation. Abdomen distended most of the time, but no pain. Patient dull and stupid. Temperature high, pulse good. Four A. M. intense pain, abdominal distention. No examination. Eight A. M., September 18th, sudden vomiting. No pain complained of, but abdomen much distended and knees drawn up, face anxious, general rigidity, abdominal and general tenderness. No fall of temperature recorded. White count, 8.30 A. M., 5,400; 10 A. M., 4,400; 2 P. M., 6,300. Operation twelve hours after first pain. General peritonitis, cloudy fluid, gas in large amounts. Intestines much injected, distended, covered with fibrin. Perforation just above ileocecal valve in ileum, "pin-head," closed. No culture. "Condition on leaving table very bad." Death in two hours. No autopsy.

CASE XIV. McKenna, male, age thirty-eight years, white. Drs. George B. Shattuck and F. B. Lund. Boston City Hospital, December, 1898. Perforation. General peritonitis. Definite warning symptoms. Death. Severe course. Widal positive. Diazo (?). No complications. No abdominal complaints. End of third week, twenty-first day. Ether. Time (?). First incision in right iliac, second in left. Irrigation. Drainage, gauze and tubes. Seven P. M. of the day before operation general abdominal pain, also pain in rectum and bladder. General abdominal tenderness and rigidity. No change in pulse and temperature. Leucocyte count 10,000. Query as to why this was not repeated. No record of any observation of case till next day. The next morning (twelve hours after warning) symptoms of peritonitis recorded, namely, distention, anxious facies, vomiting, no fall of temperature, pulse rising to 120. Operation fifteen hours after first symptom. Perforation in ileum one foot from cecum. Closed. Seropurulent general peritonitis, pus and feces, fibrin. Infection, colon bacillus and other bacteria. Death in forty-two hours. Autopsy showed general peritonitis. No other perforations. Beginning of infection when first pain, fifteen hours before operation. Definite warning. No record of case for twelve hours. Leucocyte count day before, when suggestive symptoms appeared.

CASE XV. Mahr, male, age thirty years, white. Drs. Bowditch and Watson. Boston City Hospital, February, 1898. Perforations. General peritonitis. Early symptoms definite. Operation delayed forty-eight hours. Death. Mild course. No Widal. No diazo. Post-mortem proof. End of third week, operation. Ether. Time (?). Incision, median, and two counter incisions in flanks. Irrigation. Drainage tubes and gauze. No special complications. Temperature normal for two days. No abdominal complaints till last. September 15th, A. M., vomited and complained of sudden pain, first in epigastrium, then all over abdomen and shooting into testicle. Tenderness in abdomen general but most marked in right lower region and in epigastrium. "Shock not marked." Temperature

rose from normal to 102°. Pulse 150. In a few hours rigidity of abdomen and tympanites. Leucocytes on September 15th, 31,550; next day, 17,000 to 23,000 (September 16th). In spite of these marked warning symptoms no operation for forty-eight hours. Then symptoms of fatal peritonitis, case nearly moribund, temperature subnormal, cold extremities, marked prostration, cyanosis, marked distention and rigidity. Operation found perforation in ileum near cecum, size of "split pea." Found general peritonitis, pus, fecal fluid, dirty-gray lymph, coils adherent, pelvis full of turbid fluid, perforated coil fastened in abdominal wound. Infection, colon bacillus and great variety of other bacteria (?). Death in six hours. Autopsy: Numerous typhoid ulcers in lower ileum. A second perforation (pin-point) close to valve. General peritonitis. A repeated white count in this might have contributed much to the knowledge of a falling count in peritonitis.

CASE XVI. Scappacci, male, age nineteen years, white. Drs. Cutler and Elliot. Massachusetts General Hospital, June, 1899. General peritoneal infection from a necrosed and gangrenous patch over an ulcer. Threatened perforation. Definite warning symptoms of infection. Recovered. Mild course. Widal positive. No complications. No abdominal complaints. Last of second week, operation. Ether. Time (?). Incision, median. No irrigation, but wiping. Gauze drainage. Uneventful till June 4th, then pain in lower abdomen with general tenderness. Leucocytes June 2d, 6,900; June 4th, 8,000. No complaints or examination recorded for June 5th. June 6th, severe pain, abdomen tender and rigid, but more on right side; tympany, anxious facies, pulse 120, fall of temperature. Leucocytes 12,200. No vomiting. Operation two days after first abdominal pain. Four hours after, symptoms severe. Four feet from cecum a necrotic patch of peritoneum, the base of intestinal ulcer. General peritonitis, seroturbid, purulent fluid and fibrin flakes. Intestines injected, with green fibrin patches occasional. No cultures. Patch inverted. Recovery after relapse of typhoid. Infection not determined. A case of perineal infection from ulcer not yet perforated. Symptoms of perforation all present. Infection probably non-virulent (?). Compare Case XXI.

CASE XVII. Bloom, male, age forty-five years, white. Dr. Brooks. Massachusetts General Hospital, September, 1898. Extensive general peritonitis from perforation in walking typhoid. Infection not determined. Recovery. Widal positive. Ambulatory case. In second week, operation. Ether. Time (?). Incision, right iliac. Irrigation. Drainage, gauze. Case of typhoid as shown by Widal and by finding of typical ulcer indurations in ileum. Had been sick for a week or ten days, with pains in abdomen. Up and about out of bed most of time (walking typhoid). Brought to accident-room 3 A. M. in bad condition. Temperature 103.4°, pulse 134, respiration 60. History of pain in region of appendix, severe for two days, with nausea. Operation found brown, purulent fluid in general peritoneal cavity, with no odor; portions of small intestine covered with hemorrhagic spots, "size head of tack." Perforation found one foot from cecum. "Indurated area, size of quarter, in centre of which pin-point opening." The appendix removed, incidentally, for old thickening. No active process in it. Perforation took place two days at least (?) before entrance. No white count. Recovered. No return of typhoid state, though Widal positive after operation. Discharged, well, in six weeks. No bacteriology.

CASE XVIII. McCarron, male, age thirty years, white. Drs. Bowditch and Thorndike. Boston City Hospital, October, 1899. Perforation. Beginning general peritonitis. Operation eight hours after first symptoms. Death. Mild course. Widal positive. Diazo (?). No abdominal complaints. In the fifth week, thirty-third day, operation. Ether. Time (?). Incision, right iliac. Irrigation. Drainage, tubes and gauze. Cardiac murmur, systolic, not affecting compensation. No other complications. Early warning symptoms: 2 A. M., sudden, sharp pain in right iliac

region. No fall of temperature, good pulse. Vomited. No blood count in this case. Seven A. M. (five hours after) severe symptoms. General severe abdominal pains and rigidity, especially right iliac. Pulse 120. Vomiting. Operation eight hours after first pain. Operation three hours after severe symptoms. Perforation in ileum three feet from cecum. Perforation closed with silk. Straw-colored fluid and fecal matter in abdominal cavity. Intestines injected and fibrin flaked — that is, beginning general peritonitis. No cultures recorded. Death eighteen hours after. No autopsy. This case perforated eight hours before operation, when pain noted. If the case had been carefully watched with hourly blood counts between the hours of 2 A. M. and 7 A. M. operation possibly could have been decided on three or four hours sooner. Severe symptoms at 7 A. M. due to general spreading infection.

CASE XIX. Ellam, female, age twenty-one years, white. Drs. Townsend and Burrell. Children's Hospital, October, 1899. General peritonitis. No perforation found. Symptoms of perforation or of peritonitis. Warning pain sudden and not reported. Death. Moderately severe. Widal positive. No complications. No abdominal complaints. Fifth week, thirtieth day, incision, right iliac region. Irrigation. Gauze and tube drainage. Intestinal hemorrhages daily from three days preceding severe symptoms. No abdominal pain with them. Sudden sharp abdominal pain in the middle of the night; no examination. At 9 A. M. (eight hours after) severe pain, abdominal tenderness and resistance, marked in right upper quadrant, slight distention. No leucocyte count recorded. No vomiting. Pulse 130. No fall in temperature noted, but rise to 104° at 9 A. M. May have fallen in the night. Operation eleven and one-half hours after pain. General peritonitis. Injected fibrin flaked intestines. No perforation found. "Thin place," but not necrosed; turned in. "Condition forbade search" for cause of peritonitis. No bacteriology. Death in twelve hours. No autopsy. This case could have been operated sooner. Severe symptoms came suddenly and were not reported for seven or eight hours.

CASE XX. Buckley, male, age thirty-four years, white. Drs. Jackson and Munro. Boston City Hospital, September, 1899. Signs suggestive of perforation in third week. Very sick after this. Perforations in ninth week. General peritonitis. Early warning symptoms. Death. Severe course. No Widal. No diazo. Rose spots. Cardiac enlargement. Systolic murmur. In ninth week, fifty-ninth day, operation. Ether. Time (?). Two incisions, first median; second right flank. Irrigation. Drainage, gauze and tube. In the third week, while pulse and temperature were high, sudden fall of temperature. Collapse. Distended abdomen. Operation advised and refused. Leucocyte count then 6,800. No other count recorded. From this attack very sick until ninth week. Abdomen distended. Pulse and temperature high. One small intestinal hemorrhage in fourth week. Twenty-four hours before operation abdominal pain, especially on left side. Pulse and temperature rising still higher. Twelve to fifteen hours after pain general and severe. Vomiting. Temperature subnormal. Two perforations in ileum three inches apart (where?). Also in ileum (site?). "Recent scar, possibly healed perforation of third week." Perforations closed. General peritonitis, cloudy fluid with fibrin flakes. Infection, colon bacillus. Death ten days after. No autopsy. A frequent leucocyte count would have been very valuable.

CASE XXI. Powell, male, age twenty-seven years, white. Drs. Ames and Munro. Boston City Hospital, September, 1899. No perforation. General peritonitis from ruptured mesenteric glands. Symptoms of perforation exactly simulated. Infection, pure typhoid bacillus. Death. Mild course. Widal positive. Diazo (?). No complications. No abdominal complaints. Last of third week, twenty-first day, operation. Ether. Time (?). Incision, median. Irrigation. Drainage. Pain, abdominal, more on left side at 11 P. M. This required morphine. No further record till 7.30 A. M. (eight and one-half hours later), when collapse, anxious facies, tympanites, general

abdominal tenderness, and rigidity. Temperature rising. (Temperature had been normal for twenty-four hours.) No vomiting. No leucocyte count. Operation twelve hours after pain. No perforations. Two suspicious thin places of inflamed peritoneum over ulcers. Seropurulent general peritonitis. Infection, pure typhoid bacillus. Death in twelve hours. Autopsy: No perforations. General peritonitis from two softened and ruptured mesenteric glands. This case strongly suggests the idea that symptoms of general peritonitis are identical with the "supposed symptoms" of perforation, and suggests the virulence of bacillus typhosus in peritonitis.

CASE XXII. Sawyer, female, age sixteen years, white. Drs. G. B. Shattuck and Watson. Boston City Hospital, December, 1897. No perforation. Nothing to account for symptoms of infection. Leucocyte count not taken. Recovered. Mild course. Widal positive. Diazo (?). No complications. No abdominal complaints. In sixth week, thirty-sixth day, operation. Ether. Time (?). Incision, right iliac. No irrigation. No drainage. Uneventful case. Temperature falling steadily. At no time especially sick. The afternoon before operation, severe abdominal pain and tenderness in right iliac. Temperature rising to 104°. Pain local in "lower abdomen," gradually increasing, not sudden. Next morning pain and tenderness general, but more on right side. Temperature 102°, pulse 128. No vomiting. Abdominal muscles rigid. No distention. No leucocyte count. "Absence of marked shock." Operation seventeen to eighteen hours after first symptoms. No perforations. No peritonitis. Appendix not actively inflamed, but bound by old adhesions; removed. Recovery uneventful. Important case from diagnostic side. Pain, tenderness, rigidity, rise in temperature and pulse. No great shock. No vomiting. The leucocyte count is all important, but is not recorded.

CASE XXIII. Eagles, female, age twenty years, white. Drs. Vickery and Mixer. Massachusetts General Hospital, August, 1898. No perforation found at operation. No cause for symptoms of perforation, which were definite. Recovered. Moderately severe. Widal positive. Complications, cardiac and renal. Abdominal complaints frequent. Fourth week, operation. Ether. Time (?). Incision. No irrigation. No drainage. Moderately severe case. Complicated by mitral regurgitation, with poor compensation and by acute renal congestion. Both cardiac and renal complications improved in hospital. Frequent complaints of moderate abdominal pain, which was due to retention of urine and relieved by catheter. Probably cystitis. Temperature lower and sitting up in bed in fourth week, when pain in abdomen complained of; white count at this time 9,200. Leucocyte count at entrance 4,200; twelve hours before operation 9,200, when pain first complained of; one hour before operation 12,000. Twelve hours before operation, pain, abdominal tenderness and spasm, mainly on the right side; slight distention; pulse 118. Temperature (?). No vomiting. Operation twenty-four hours after pain first noted. Nothing found. Recovery. Symptoms of gradual perforation, including some leucocytosis. No marked shock recorded. Query as to how to account for leucocyte count. Compare this case with Sawyer, Case XXII. Same symptoms, but in Sawyer's case no leucocyte count. The comparative value of leucocyte count in this case is questionable.

CASE XXIV. Seile, male, age twenty-seven years, white. Dr. Balch. Massachusetts General Hospital, September, 1897. Symptoms of peritonitis of gradual onset. Operation found necrosed patch, but no microscopic general peritonitis. No leucocyte count. No cultures. Death. Mild course. Widal positive. No complications. Fifth week, operation. Ether. Time (?). Incision, median. Irrigation. No drainage. Slight intestinal hemorrhages at intervals for three weeks before operation. Ten days before, abdominal distention, but no pain or rigidity. Temperature rising. Distention only transient. None two days before operation. September 25th, at noon, pulse 140; some sweating in the afternoon. Pain below and to left of umbilicus. Abdomen distended (spasm,

vomiting and white count not recorded). (Temperature not recorded.) September 26th, picture of peritonitis, anxious face, pain, restlessness. Liver dullness obliterated. Operation twenty-four hours after first symptoms. No perforation, one Peyer's patch, one and one-half feet from cecum, threatening perforation, with fibrin area on intestine, "green, necrotic area," this inverted. No extravasation in peritoneal cavity. No bacteriology. Death in ten hours. Autopsy showed no general peritonitis. Only local necrotic patch. Death from typhoid.

CASE A. Eladed, male, age fifteen years, white. Drs. Bowditch and Munro. 1899. Case of general peritonitis. No perforations. Gall-bladder infected with typhoid bacilli. Cause of peritonitis unknown. Sick case. Widal negative. Diazo negative. Large spleen. Sick for four weeks. Came into hospital with general peritonitis. "Nearly moribund at entrance" (Bowditch). Delirious; distended belly; distended, rigid muscles; temperature 104°; pulse 120. Leucocytes 5,200 (note, purulent peritonitis of long standing). No symptoms of perforation. At operation for general peritonitis no perforations found. Gall-bladder opened, but no evidence of origin of peritonitis. Cultures of free peritoneal fluid, staphylococcus albus. Cultures from gall-bladder, bacillus typhosus. Exploration only half satisfactory (Munro). No anatomical evidence of typhoid found. Death in twenty-four hours. No autopsy. No conclusive proof that this was a case of general peritonitis during typhoid. Moribund case of general peritonitis, cause of which doubtful. Should not be counted with other cases.

CASE B. Collins, male, age twenty-five years, white. Drs. Jackson and Gavin. Boston City Hospital, September, 1895. Case of general peritonitis. No evidence of typhoid fever. No Widal. No rose spots. No spleen. Case of no value. This case had general peritonitis, was operated on and died. No reason to call it typhoid. No post-mortem. Leucocytes 32,000 before operation.

CASE C. James, male, age (?). Dr. Munro. Boston City Hospital, 1895. Case of general peritonitis. No evidence of typhoid or of perforation. Case of no value. Case entered the Boston City Hospital with general peritonitis. Operated on and died. No cause for peritonitis found. No autopsy.

SEPARATION OF THE EPIPHYSIS OF THE OLECRANON.

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SEPARATION of the epiphysis of the olecranon is classified as one of the rarest of the epiphyseal separations. Poland, in his recent book, gives no instance of this separation save as a complication of other injuries of the elbow, unless one accepts a doubtful case by Bryant, and a case of Hamilton's where he produced the injury by forced flexion in attempting to reduce an old dislocation of the elbow. Nor has the writer, in a short search of the literature, found any case recorded where this separation occurred without other injury.

There is experimental evidence enough (Poland, Guerin and Salmon) to show the possibility and ease of separating this epiphysis, but curiously enough, all the experimenters seem to have used forced hyperextension to produce it. Muscular action in resisting flexion, the apparent cause in the following cases, cannot, of course, readily be imitated on the cadaver.

Both the cases here reported came under the care of the writer while he was acting out-patient surgeon to the City Hospital.

The first case was in a naval apprentice, a lad of sixteen years. He gave a history of having had a fall on the hand without any blow on the elbow.