tained in pure culture on serum agar, and also demonstrated in cover slip preparations stained with methylene blue, being found both free and contained in the pus cells.

Bujivid,[12] in 1895, reported a case of abscess due to the presence of the gonococcus, unassociated with infection of any tendon sheath. The patient was suffering from a chronic gonorrhea, and two days after catheterization had a severe chill, after which abscesses developed in the right popliteal space, in front of the elbow, over the right external malleolus and on the inner side of the left leg. The author states that all of these had their origin in the muscles, and none in the cellular tissue or neighboring articulations. He therefore classes the gonococcus with the pus-producing organisms.

Young[13] reports five cases of subcutaneous abscesses in the course of gonorrhea, in all but one of which the gonococcus was found alone. In all of these the abscess was adjacent to the urethra or to some synovial membrane.

Deutschmann[14] found organisms which were characteristic gonococci with regard to form, size, grouping, staining reactions and cultural characteristics, in the synovial secretion of an affected knee of a child suffering from ophthalmia neonatorum.

The patient in the case studied by the writers is a woman of about 40 years of age, married, and a housewife by occupation. Her present trouble began July 25, 1903, as sharp shooting pains around the base of the right thumb and index finger, which after 24 hours became so severe that medical aid was summoned. The hand was found on examination to be slightly swollen, especially over the location of the pain. Her temperature was found to be 101 F. Any motion of the index finger was extremely painful, especially movements of the metacarpophalangeal joint. The patient's husband had been under treatment for gonorrhea during the previous month, and consequently the present trouble was suspected to be a complication of a gonorrhea which the patient had acquired from her husband; but close inquiry failed to elicit any history of such a condition. She was therefore placed on antirheumatic treatment, and the pain controlled by sedatives, but without beneficial result.

The swelling and redness became more pronounced, and August 2 an incision was made over the proximal phalanx of the index finger, both above and below, resulting in the evacuation of a small amount of purulent material. After this the temperature fell to normal, but in the course of a few days rose again, and the swelling extended to the palm and dorsum of the hand. August 16 free incisions were made into the diseased area under chloroform anesthesia, and a considerable amount of pus was evacuated. The pus cavities were curetted and washed out thoroughly with a hot carbolic solution. The subsequent treatment consisted of hot bichlorid irrigations and antiseptic dressings. Recovery was uninterrupted, but the involvement of the flexor tendon resulted in its shortening, which prevents the complete extension of the index finger. There was no ankylosis of any of the neighboring joints. This fact, together with the evident involvement of the flexor tendon, indicates that the condition was purely a tendovaginitis.

BACTERIOLOGIC INVESTIGATION.

The freely flowing pus, obtained at the second operation, was received on a sterilized platinum loop and transferred to liquefied gelatin and agar tubes (45 C.). The gelatin tubes were kept at room temperature, and the agar tubes in the incubator at 37 C., for some days, without, however, showing any growth. Unfortunately, tubes of serum agar were not at hand, and growth on this medium was not attempted. Thin spreads of pus obtained as above were made on cover glasses, dried in the air, and fixed lightly in the flame. A portion of these spreads were stained with Loeffler's methylene blue, the balance with a 1:10,000 solution of neutral red. On examination, these preparations showed the presence of groups of "biscuit-shaped" diplococci, although not in very large numbers —not more than one or two such groups to a field (1/12-inch oil immersion). Similar diplococci were also found within the cell bodies of the leucocytes, and in all respects as regards form, size, arrangement, staining reaction, and grouping were typical gonococci. They were readily decolorized by Gram's method.

Although the organism could not be cultivated on the media employed, the characteristic appearance of the flattened diplococci, the grouping, the presence of the organisms in the pus cells, the decolorization by Gram's stain, and, above all, the inability to grow on the ordinary media, on which the ordinary pus germs readily thrive—leaves no doubt that the organism in question was none other than the gonococcus of Neisser.

The fact that such metastatic abscesses, due to the gonococcus alone, can occur in various parts of the body, even in the absence of symptoms on the part of the urethra or other portions of the genito-urinary tract, being established, the importance of routine microscopic examinations of pus from such abscesses, especially those connected with tendon sheaths, is apparent. A patient such as the above becomes an especial danger, not only to herself, but to those with whom she is associated, unless the true condition is understood. It is readily seen that the liability of such a patient to contract a gonorrheal ophthalmia or to infect the eyes of others, is infinitely greater than when the disease is localized in the urethra. The transference of purulent matter to the eyes of such a patient or to those of her young children, from an abscess on her hand, is indeed more of a probability than a possibility, unless especial precautions are taken to prevent.

Aside from the danger to which the ignorance of such a condition subjects the patient and her associates, the clinical discovery of the gonococcus in such an abscess affords a clear indication as to subsequent treatment. It is probable that in all cases the organisms growing in such metastatic abscesses have migrated from the mucous membrane of the urethra, this locality being, par excellence, the most suited for their growth. It is also probable that here they will be found in greatest numbers, whether the patient presents symptoms of urethritis or not. Proper treatment directed to the urethral condition should, therefore, be employed in all cases. First, for the purpose of curing the primary condition; and, second, as a prophylactic measure against other possible metastatic complications, endocarditis or synovitis, and especially to forestall the more serious dangers to which every sufferer from a chronic gonorrhea is liable.

12. Centrabl. f. Bac. u. Parasit., Jena, 1895, xviii, 435.
13. Jour. of Cut. and Gen. Urin. Dis., 1900, xviii, 241.
14. V. Graefe's Arch. f. Ophthal., xxxvi, 1.

TREATMENT OF TYPHOID PERFORATIONS.

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While the modern methods of treatment by the general practitioner have done much to reduce the percentage of deaths due to typhoid fever, the rôle played by the surgeon in lessening the death rate occupies a conspicuous place.

The tubbing and proper feeding do much to keep down temperature and maintain strength, but no medicinal agents nor the most careful management can overcome the great danger of intestinal perforation by ulcer-

ation. From a careful perusal of the literature and consultation of authorities we find that about 3 per cent. of all cases present this most dangerous complication, i. e., it is the cause of one-fourth to one-third of all deaths in typhoid fever.

In 3,686 cases, Schultz found peritonitis from perforation in 1.2 per cent., Liebermeister in over 2,000 in 1.3 per cent., while Hölscher found it in 6 per cent. in 2,000 cases, and Murchison in 11.38 per cent. in 1,721 cases. Fitz shows the mortality to be 6.58 per cent. from perforation in 4,680 cases tabulated up to 1891. In more recent times, since the subject has been more thoroughly studied, we find that Armstrong reports 932 cases, with 3.66 per cent. perforations, and McCrea and Mitchell cite 8 cases occurring in 275 patients. This, in comparison with the present-day mortality, would mean a very large proportion of deaths in typhoid fever due to perforation, for under former methods of treatment these cases practically all died and to-day the death rate from this cause continues large.

In 1898, Keen reported a total of 83 cases of typhoid perforations operated on with 19.3 per cent. recoveries, to which a year later, he added 47 cases with 28 per cent. recoveries. This work was largely done in this country, due probably to the doubt as to the efficacy and advisability of operation existing in the minds of the European surgeons, as expressed by Eulenberg in his paper in 1891.

In Keen's monograph, which caused the profession to awaken to this subject, he says that the accident is a very rare one in children, but Elsberg, in a very recent communication, thinks it almost as frequent as in the adult. Basing the frequency of cases in children as seen by the surgeon, i. e., 48 cases in a total of 315, we must conclude that, though not occurring as often, it can not be considered as an infrequent complication in children. We would especially call attention to these figures, in view of the fact that typhoid fever is said to be not only much more seldom seen in children, but that it also runs a far milder and shorter course. It may be of interest to know that one of Chevalier's cases was in an infant of 11 months of age, and that this author, in a large experience with the young in the hospitals of Paris, believes it to be a source of great danger in them.

Recognizing these facts, we should then be constantly on the alert for the very first symptoms indicative of perforation. It may take place at almost any time, even during the first week of illness, though most often after the second week. This is contrary to the older teachings and also the opinions which are held by many practitioners, it being popularly thought that the end of the third and beginning of the fourth week of illness is the time when this danger may be expected. It is not always an easy matter to determine the date of the illness, even after careful inquiry, as many patients are ill for a variable time before a physician is consulted or before they take to bed. In some of the reported cases the signs of perforation were really the first warning the patients had that they were very ill and were also the occasion for consulting medical advice.

It is also a mistake to believe that after the fourth week this danger is passed, for as many cases have been reported as taking place in the tenth as in the first week. Of 193 cases of perforation, tabulated by Fitz, the time of occurrence was as follows:

First week 4, 2d week 32, 3d week 48, 4th week 42, 5th week 27, 6th week 21, 7th week 5, 8th week 3, 9th week 2, 10th week 4, 11th week 3, 12th week 1, 16th week 1.

SEAT OF PERFORATION.

In 85 to 90 per cent. of cases perforation takes place in the last twenty inches of the ileum, though it may occur in the colon, and several cases of appendiceal ulceration and perforation have been recorded. The sigmoid may be the seat of the opening or the jejunum, and in the table of Fitz above quoted, it occurred four times in a Meckel's diverticulum.

SYMPTOMS AND DIAGNOSIS.

No case of typhoid should be so mild as to cause us to lose sight of this dreaded accident. We believe that the symptoms are such that a diagnosis can be made correctly in the majority of cases, if one is on the lookout in the least. These symptoms are those due to the insult to the peritoneum, and are not dependent on the perforation *per se*. The classical signs are pain, rigidity, tenderness. With these we have thoracic instead of abdominal respiration, which means fixation of the diaphragm, inhibited peristalsis, absence of liver dullness, tympany, vomiting, increase in blood pressure as shown by the sphygmomanometer in all cases, leucocytosis, increased pulse rate followed by rising temperature and altered facial expression and shock. Let us very briefly analyze the symptoms:

Pain is not a necessary accompaniment of typhoid, and while it may be present during the course of the disease, it is not continuous or persistent, but is temporary. Nor is the onset sudden as a rule, unless as an indication of some severe and dangerous complication. Ordinarily it is relieved by an enema. On the contrary, the pain of perforation is sudden and may follow a stool or manipulation of the patient. It begins in the hypogastric, though more often in the right iliac region, and spreads thence gradually over the entire abdomen. Not infrequently a stool soon follows, giving some temporary, slight relief. The pain may increase and be continuous or may after a time cease entirely, which means that a general toxemia has supervened. Even in the smallest pin-hole openings this pain is present. Persistence of pain without the slightest evidence of shock, if accompanied by rigidity, is almost sure to mean perforation. Acute or sudden or continuous pain should always be a danger signal and cause the attendant to redouble his care and vigilance, making more frequent visits and getting into touch with a surgeon so as to be prepared for the emergency. It may denote, in the absence of perforation itself, an ulceration through to the serous coat of the bowel and the approach or early possibility of extension through all the coats. It is the peritoneal layer alone which possesses nerves of sensation, hence the pain at this stage. Ulceration, no matter how extensive so long as the peritoneal coat is uninvolved, causes no pain.

Rigidity is one of the most valuable signs of peritoneal damage we have. It is noticeable almost at the beginning and may be present even before the perforation. Just as pain may denote an impending perforation, so may rigidity be found before the damage has extended through the peritoneal coat of the bowel. Localized at first to the affected area, it soon spreads over the entire abdomen, indicating spread of infective material. Unlike pain, which may be well borne and not cause much complaint in certain stolid individuals or in those profoundly septic or which may grow less and even cease as toxemia advances, rigidity is constantly present. The patient can not voluntarily overcome it. Its presence in the diaphragmatic muscle

causes the alteration in type of respiration from abdominal to costal.

Tenderness usually exists most markedly in the right iliac region, i. e., over the seat of perforation, and over the now most violently affected serosa. It soon becomes more or less general, but may be slight over the abdomen, except in the region above mentioned or the hypogastric region. Osler says the attendant must be constantly on the watch for the sudden occurrence of the above three symptoms.

Inhibited peristalsis is, according to Taylor, a very important symptom. Not infrequently we find, however, that the inhibition has been preceded by a very active peristalsis immediately following the perforation as indicated by the two or three copious evacuations, which at times follow the onset of the very earliest manifestation of pain. These stools may contain blood from the perforating ulcer. That inhibition follows we can readily understand, and as the infection progresses, it becomes complete, resulting in paresis, which, in my opinion, indicates absolute hopelessness of recovery in this as well as in other peritoneal involvements.

Tympany and absence of liver dullness with pain, are, to our mind, the only really indicative symptoms of the perforation *per se,* all other symptoms being those of the infection rapidly following the peritoneal insult. This symptom, however, is so rarely present early that it is difficult to see how one is to differentiate the symptoms of the perforation from those of the peritonitis, which latter are the ones on which we depend for a diagnosis. Escape of gas is apt to take place so gradually that it is hardly a symptom to wait for before making a diagnosis, notwithstanding the fact that its presence is almost pathognomonic. I have seen cases with little tympany even hours after the perforation had occurred.

Besides the above local symptoms referable to the abdomen or seat of lesion, we have other symptoms serving to corroborate the diagnosis. We should not lay too much stress on them, as the local symptoms are, without these, enough on which to place the strongest reliance and base a sufficiently strong presumption.

Vomiting may or may not be present; in fact, it is not a symptom in more than 7 to 10 per cent. of the cases reported.

Blood pressure, measured by the sphygmomanometer, according to the reports of Crile and the Johns Hopkins School, furnishes a most valuable means of diagnosis. The cases are, of course, too few as yet to say with absolute certainty what the exact value of a rapid rise in blood pressure as thus indicated is, but from my experience I am inclined to believe this instrument will or should come into as general use as the stethoscope.

Crile, in five cases at Lakeside, made a diagnosis by this means, all verified by autopsy or operation. In one case a second perforation was also indicated by a rapid rise in blood pressure. With subsidence of peritonitis the blood pressure fell rapidly to normal again.

Leucocytosis has been shown to be of value only in connection with the other symptoms. This is probably accounted for by the rapid outpouring of leucocytes into the peritoneal cavity as a result of the infection. As McCrea and Mitchell have shown, even with other symptoms it may be misleading, two of their cases with this evidence and other symptoms presenting no perforation at operation. Armstrong, with an experience of 34 operated cases, says it is not to be relied on. Elsberg says: "It can be used only with circumspection as a diagnostic symptom, while the absence of a leucocytosis does not exclude the possibility of a perforation."

Pulse and temperature, of all the signs of perforation, are the least valuable. The character of the pulse is a very valuable sign, but the rate is hardly to be depended on. Like temperature, it means very little in many instances, and both are so frequently misleading that no reliance can be placed in either as even fairly constant from a diagnostic standpoint.

Facial expression may not be altered, except to indicate pain. Not infrequently, however, the expression is pinched, anxious and indicative of a very grave change in the patient's condition. When there is much shock this is especially true, and we have the expression so graphically described by Warren in this condition, when, as Gross says, the "machinery of life has been rudely unhinged."

Shock may be very marked or absent or so slight as to pass unnoticed. When marked it is a symptom of value only as indicating a most desperate case. I wish here again to draw attention to shock as a symptom not of perforation *per se,* but rather of a rapid and profound poisoning. The text-books lay entirely too much stress on this symptom of perforation. In my own experience in the one case operated on it was never a symptom, and I have seen other cases in which it was entirely absent. Without bleeding, this symptom is one either of rapid sepsis, with consequent obstruction or of early impending death. Notwithstanding this, it is not an indication for delay in treatment.

Of all these symptoms, the most important are pain, tenderness and rigidity.

TREATMENT.

To obtain the best results, the operation should be done promptly within certainly the first twenty-four hours; better still, as soon as a probable diagnosis is made. Of the six recoveries reported by Armstrong, five were operated on within thirteen hours after the first symptom, and Cushing believes that with careful watching and early diagnosis, 50 per cent. to 60 per cent. should be saved. In other words, we should operate if possible before the peritonitis becomes too general and too virulent. As lives have been saved, however, even in very late cases, who can say when a patient is beyond hope of recovery? One life in 100 or even in 1,000 is worth saving.

Some will say that cases may get well without operation. True, but as Fitz says: "Since perforation may take place without any suggestive symptoms, and since suggestive, even so-called characteristic symptoms, may occur without any perforation having taken place, it must be admitted that recovery from such symptoms is no satisfactory evidence of recovery from perforation."

It is also true that sometimes we may make mistakes and find no perforation, but we must remember that a peritonitis may exist without perforation which may demand celiotomy to rescue the patient. Murphy has especially called attention to this and reported several cases. In these, while there is no perforation, the ulceration has extended through all the coats except the serosa, and we have the bacteria finding their way through this thin wall into the peritoneal cavity. The symptomatology may then be precisely the same as in perforation; in fact, exclusive of the hole in the bowel, the pathology and outcome are similar. Chevalier says: "Operate even when the diagnosis is not fully decided,"

which means, of course, to operate promptly. Do not wait for all the positive and confirmatory evidences of the trouble or the golden opportunity will have been lost. Nor should we be too greatly perturbed about shock when present, as it is in these cases no indication for delay. Some of the best results have been in patients operated on in shock.

As to the technic of the operation, I would only say, do not try to do too much. Do not lose time in useless tinkering and fussing. Do the work quickly—best through an incision in the right linea semilunaris—close the opening in the bowel, and be sure to amply drain the cavity. If the opening is not readily found, let it be and drain extensively. To prolong the operation needlessly is bad surgery. If the patient is a bad subject for a general anesthesia, use a local anesthetic.

Escher has recommended stitching the edges of the intestinal opening to the parietal incision, and Le Compte advises damming off the infected area, particularly when there are multiple openings near each other, with a gauze wall. An artificial anus presupposes only one perforation. The mattress Lembert suture is looked on as the best. Extensive flushing and leaving some of the normal saline solution in the belly, with of course drainage, is, I believe, the preferable way to overcome the peritonitis, although Finney and others continue to advise dry mopping of all infected areas.

In conclusion, I wish to read the following table of Elsberg and some additional cases I have found in the literature bringing the reported cases to date:

REPORTED CASES OF OPERATION TO APRIL, 1903.

	Adults.	Children.	Total.
Cases operated on	264	25	289
Recoveries	59 (22.4%)	16 (64%)	75 (25.9%)
Deaths	205	9	214 (74.1%)

ADDITIONAL CASES.

	Cases.	Recoveries.
Armstrong	8	5
Crile	4*	2
Chevaller and other Parisian surgeons	13	5
Frank	1	0
Harte	13	3
Méry	1	0
Total	40	15 37.5%

*One unoperated.

Of these last, 14 are reported as children with 5 recoveries, or 35.57 per cent.; 26 adults, 10 recoveries, or 38.46 per cent.

My own case was as follows:

A man, 32 years of age, in the third week of typhoid fever, living in the country, was seized with a sharp pain in the right iliac fossa, which disappeared after copious movement of the bowels. Two hours later his temperature became very slightly subnormal, and continued so or normal for twenty-four hours. When seen by me 60 hours later his temperature was slightly above 99 F., pulse between 86 and 90, though it rapidly rose to 112. There had been no vomiting, and distension of the abdomen had been present for only a short time after the sudden pain. The patient presented a good appearance, without pain or tenderness, and was not depressed. Tympany was present over the entire abdomen as far as the fifth intercostal space, with marked rigidity and absence of liver dullness. The pulse was hard and characteristic of peritonitis. Diagnosis of perforation was made, with prognosis of death, with or without operation. The patient asked that he be given the slight chance by operation. On opening the abdomen it was found filled with pus and fecal matter and the intestines distended above a point about eighteen inches above the perforation, below this point the intestine was flat. The perforation was closed with difficulty, the cavity washed out and five drains inserted. The patient was put to bed in fairly good condition, and next morning had a pulse of 130, no vomiting or pain. He died on the second day.

CONCLUSIONS.

From the foregoing we can make the following summary:

1. Perforations are to be expected in about 2.5 per cent. of all cases of typhoid fever.

2. Prompt surgical intervention is the best and only logical treatment.

3. Early diagnosis is most desirable, and will be the means of greatly reducing the mortality, as 55 to 60 per cent. would recover.

4. Diagnosis, sufficiently early to achieve these results, can only be made by careful watching, treating all cases as serious ones and a proper interpretation of the early, even the preperforative, symptoms, the suggestions of Cushing as to this stage being of value. At the first indication of this stage have the surgeon in consultation and be prepared to operate.

5. The sphygmomanometer should come into general use as an important aid to diagnosis.

6. More cases die from delay than from errors in surgical technic. Therefore, in doubtful cases, though a mistake may be made and no perforation found, operate.

7. No case, unless dying, is so desperate as to be beyond some hope of saving. So in operating, lose no time and be sure to drain.

8. Get into the abdomen quickly and get out more quickly.

(NOTE.)—Since this paper was written, Dr. W. D. Haggard of Nashville has reported, in a paper read before the American Association of Obstetricians and Gynecologists, three cases, with three recoveries.—L. F. (See THE JOURNAL A. M. A., Oct. 17, 1903, p. 979.)

A SIMPLE, ACCURATE AND RAPID METHOD OF LOCALIZING FOREIGN BODIES IN THE ORBIT.

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When the eye has been injured and there is a possibility that a foreign body remains, the very first procedure is to determine, if possible, the exact location of the object in reference to the eye. If the foreign body can not be seen with the ophthalmoscope we must resort to the *x*-ray, and should the result of such examination be positive, we then proceed to localization before any means whatsoever are tried for its removal.

As the vast majority of the foreign bodies in eye cases (iron, steel, copper, lead, glass, etc.) are revealed by the Roentgen ray, we can usually be certain of a positive result if the foreign body be present. The circumstances attending the injury often determine the probable substance of the foreign matter present. Fortunately most of such injuries to the eye are produced by pieces of iron or steel, and as these objects are attracted by a magnet we have thus an invaluable method for their removal.

Since the advent of the incomparable giant magnet of Haab, great advancement has been made in the successful treatment of these cases, but I do not at all agree with those who advocate using the Haab magnet in an attempt to determine whether or not an eye contains such a foreign body. I do not consider it good surgery or good sense to attempt to remove a piece of steel from the eye before its exact location is known. This being so, it is most desirable to have a method of localization