Remarks

ON

PERITONITIS AND ITS TREATMENT.

Founded on an Address delivered to the Trowbridge Division of the British Medical Association

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"THE treatment of peritonitis has always been a fruitful topic of discussion and dissension and will probably remain so, as the final chapter on this subject has not yet been written."¹ Such, gentlemen, were the opening words of an American professor of surgery when giving an address on the treatment of peritonitis. They are unfortunately as true to-day as they were when they were spoken just a year ago by Dr. John Gibbon of Philadelphia. I make no excuse, therefore, for reproducing them and for making them serve as the text of my discourse to you to-day.

In the whole domain of medicine and surgery there is scarcely any disease so serious, so different in its clinical manifestations, so dangerous when wrongly treated, and yet so difficult to treat according to fixed rules. Peritonitis may be a mere local inflammation of comparatively little importance which quickly subsides under simple medical treatment or no treatment at all. More often it is a much more serious affection demanding prompt and careful medical or surgical treatment. Only too often does it come before our notice in the form or at a stage when neither physician nor surgeon can do much, if anything, for its relief. Acute peritonitis, subacute peritonitis, chronic peritonitis, general peritonitis, local peritonitis are all mere degrees of the same process, and yet how different as a rule is the treatment required for each. Not only so, but a form of treatment which is to be strongly recommended at one stage or in one form of peritonitis may be most harmful and disastrous in its results if employed at another, and perhaps not very far removed, stage or form of the same disease. But underlying our treatment of all forms of peritonitis there are certain broad principles that should guide us in our endeavour to select the best possible means of treatment in any individual case.

It will be my intention this afternoon to discuss some of these principles and to endeavour to apply them to the different forms of peritonitis which we are in practice called upon to treat. The subject of peritonitis is, however, such a vast one that I must ask you to allow me to limit myself and to deal only with acute peritonitis. With the various kinds of chronic peritonitis and with the minor and slighter forms of local peritonitis I am not especially concerned to-day. And you will not be surprised if as a surgeon I dwell more upon the operative than upon the non-operative forms of treatment. Not that I wish it to be thought that a surgeon always necessarily treats peritonitis by operation. On the contrary, I believe that there are many cases of acute peritonitis that for one reason or another are best treated without operation. It is the duty of a right-minded surgeon to be ready to operate in any case in which it seems to him best for the patient that he should operate. But it is equally his bounden duty to abstain from operating if in the case before him he thinks that operation will do harm rather than good.

Peritonitis—inflammation of the peritoneum—what is it? Why is it so dangerous, so much more dangerous than inflammation, say, of a muscle, or lymphatic gland, or even of a joint? The very term peritonitis is a misleading one. It is not the mere inflammation of the peritoneum that is dangerous. This inflammation is in itself often exceedingly beneficial and protective. But peritonitis is dangerous in proportion as it is associated with *absorption* from the peritoneal cavity. It is the absorption of toxins from the peritoneal cavity, not the inflammation of the peritoneum, that is dangerous. The peritoneal cavity is, as we all know, a vast lymphatic space from which absorption of toxins readily takes place.

Let us consider for a moment what we mean by peritonitis. It is the inflammation of the peritoneum produced by the presence of bacteria. These may in some cases

have come from the blood-stream, as when peritonitis occurs, as it occasionally does, in the course of an acute septic lesion of some other part of the body. Some of the cases of pneumococcal peritonitis secondary to pneumonia may perhaps be taken as an illustration. The bacteria may be introduced directly from without, as in the case of an accidental septic wound of the peritoneum or in the course of an operation performed without due attention to surgical cleanliness. But in the great majority of cases the bacteria are introduced into the peritoneal cavity from within by passing through the wall of one of the hollow abdominal viscera, the appendix, the intestine, the stomach, gall-bladder, Fallopian tube, &c. Not uncommonly the poisonous contents of the viscera are introduced through a visible opening in the wall of the viscus. Such is the case in the so-called perforative peritonitis. Perforation of a gastric ulcer, of an ulcer of the intestine, of the gall-bladder, are familiar illustrations of which we must all have seen many examples.

In appendicitis the amount of septic matter that suddenly escapes into the peritoneal cavity is generally quite small and often there is no visible opening in the appendix. Even if there is a definite visible opening the amount of septic matter that escapes is often small—perhaps only a drop or two, for example. The perforation of an appendix is usually preceded by adhesive inflammation of the lumen of that organ, which is apt to shut off the cavity of the appendix from that of the cæcum, so that all that escapes is what was in the appendix at the time of operation. This I have been able to demonstrate time after time in the post-mortem room when as surgical registrar I had abundant opportunities of doing post-mortem examinations on cases of appendicitis. By far the most serious and dangerous forms of peritonitis produced by appendicitis are those in which the perforated appendix has previously been distended and filled with a considerable quantity of foul septic fluid and those in which it is not the appendix itself that suddenly ruptures but an abscess that has slowly and quietly formed outside the appendix and which ruptures secondarily through its wall. These are the two conditions that are commonly found in cases to which the term fulminating appendicitis is applied. I am aware that this term is often applied, I think erroneously, to any case of acute appendicitis which happens to be rather more severe than usual. In the great majority of cases of acute appendicitis, then, the amount of poison that escapes is small. It is not more than the peritoneum itself can deal with. Most cases of acute or subacute appendicitis, therefore, even if not treated by operation, recover spontaneously, as all of us know who are old enough to remember the days when appendicitis was never treated by immediate operation.

But now let us take the case of a perforation of a large hollow viscus like the stomach or the cæcum. Here a perforation is rarely preceded by adhesive inflammation. The perforation occurs suddenly and is immediately followed by the pouring out into the peritoneal cavity of a vast quantity, often many ounces, of septic material. If the stomach perforated during digestion the quantity poured out is very great. If it happens that the stomach be empty at the time then the amount poured out, although usually considerable, is much less than during digestion, and these are the cases of perforated gastric ulcer in which symptoms are less severe and pronounced. It is in the case of sudden perforation of distended intestine, however, that we see the most violent and profuse invasion of the peritoneal cavity by a large quantity of horribly poisonous fluid material. Everybody here must have seen at one time or another one of those distressing cases of chronic obstruction of the large intestine which has been allowed to progress without relief by operation. A middle-aged or elderly patient gets obstruction of the bowels; he becomes distended, constipation persists, days, even weeks, elapse without relief. He has had little pain, very likely he is walking about, he probably vomits only occasionally, but the tension of his abdomen continually increases. Then suddenly he is seized with violent abdominal pain, he becomes collapsed, and in a few hours he is dead. The cæcum has suddenly perforated. At the post-mortem examination many ounces, perhaps half a pint or more, of thin liquid fæces is found spread out all over the peritoneum. The man has died from acute septic poisoning, the poison having been rapidly absorbed from the previously healthy and non-adherent peritoneum.

A similar train of symptoms is seen when a large intra-peritoneal abscess bursts secondarily into that part of the

¹ John H. Gibbon of Philadelphia, in *New York Medical Journal*, April 7th, 1906. No. 4384.

general peritoneal cavity that has not been shut off by adhesions. Now it is just in these the most rapidly fatal forms of peritonitis that we see the least amount of visible peritonitis. I have done a post mortem in a case of this kind and have seen with the naked eye practically nothing that could anatomically be called peritonitis. In such a case there is perhaps a little injection of the peritoneum, but little or no lymph has been poured out and the surface of the peritoneum is quite smooth and shiny. But this is seen only in the most extreme cases where death has occurred within a few hours. And it is seen as a rule only in the case of elderly people in whom the resistance of the peritoneum to bacterial invasion is less than it is in younger subjects. But if the dose of poison is much less and the patient lives for a day or two, then we see a very different state of things. The peritoneum is intensely inflamed, much sticky lymph has been poured out, pus is seen to be smeared all over the peritoneal cavity or to be collected in places into definite abscesses; much thin purulent fluid may be found in the peritoneal cavity. In such a case the peritoneum has made a gallant attempt to shut off or to get rid of the poison. But it has failed; there was too much poison. The patient has died of toxæmia. The peritonitis could not stop the toxic absorption.

And so I come back to the point from which I started when I said that peritonitis is in itself beneficent and protective, acting as it does as a barrier against the further absorption of septic matter. It has happened to myself on many occasions to have had the opportunity of seeing how much septic matter can be dealt with and destroyed by the peritoneum. More than once have I opened the abdomen and seen an abundance of septic matter, lymph, and even pus smeared widely over the peritoneal cavity. A few weeks later the same abdomen was opened for some further operation and little could be seen except a few adhesions. The septic matter had been wholly destroyed and got rid of.

Now let us consider for a few moments the clinical symptoms that may be observed in a case of commencing peritonitis due to the sudden escape of toxic material from a perforated viscus such as the stomach. In the first place there is a more or less severe pain which is usually at first localised to the seat of the perforation, but may be more or less general or may be referred to the region of the umbilicus. The peritoneum is normally insensitive to mechanical injury. This may easily be demonstrated in the course of such an operation as gastrotomy or colotomy when performed, as it sometimes has to be, without an anæsthetic. In such circumstances we all know that the stomach or colon may be pricked or cut without the patient feeling any pain. But the peritoneum is sensitive to such stimuli as the application to it of an irritant poison such as septic material. A sudden severe and persistent pain, accompanied by tenderness and rigidity of muscles over the affected area, is often sufficient for the diagnosis of a perforation of the stomach. It is frequently the duty of a surgeon to operate for perforated gastric ulcer upon such evidence alone. Nausea and vomiting usually follow soon afterwards, but it is not necessary or desirable to wait for the later symptoms such as rapid pulse and elevation of temperature. These latter indicate septic absorption and show that the patient has reached a stage less favourable for operative interference.

Now what does nature do when a sudden invasion of the peritoneal cavity by bacteria takes place? There is immediately a rush of blood to the affected area. The abdominal veins are capable of holding a very large quantity of blood, and a very large quantity of blood is now needed for the great fight which is to take place between the blood plasma and white corpuscles on the one hand and the bacteria on the other. The abdominal viscera then, and especially the great omentum, become engorged with blood. This filling up of the abdominal veins leaves less blood in other parts of the body. Hence in severe cases we have collapse. The arteries are empty and the pulse is feeble. I am aware that other factors, such as reflex inhibition of the heart, may play a part in the production of shock. But I lay special stress on this cause of the shock and feeble, rapid pulse that we may meet with in peritonitis because it ought to teach us that the proper mode of treatment is to replenish the empty vessels by giving the patient fluid, plenty of fluid, to replace that which has gone off to the abdomen. But I shall return to this later.

Concerning the two important symptoms, elevation of pulse and temperature, I need say but little. Everybody

knows that of the two the pulse is by far the more important. The temperature may be normal or subnormal in a fatal case of peritonitis. If the pulse is not greatly raised, and especially if its rate be not steadily increasing, there is as a rule no immediate cause for anxiety. Think well of a case of peritonitis in which the pulse-rate remains steadily below 100.

I come next to a symptom which is present in all but the very early stages of peritonitis, and that is *distension* of the abdomen. The intestines become filled with gas. Why this should be so is by no means clear to me. That it is not altogether due to the growth of bacteria is obvious. I presume that it is in some way connected with injury to the nerves of the intestines, although why this should lead to the actual formation of gas I do not know. On a small scale we see this same rapid distension of the intestine with gas when we apply a clamp to the small intestine in the course of an ordinary gastro-enterostomy. Within a few minutes of the application of a single clamp to the loop of jejunum, the latter which was empty and collapsed becomes distended. When the time comes for opening the intestines it is found to be filled with a mixture of mucus and bubbles of gas. A question I have often asked myself without being able to answer satisfactorily is, How far is this distension protective and beneficial? Certainly many cases of slight peritonitis are accompanied by great distension and the patients nevertheless get quite well without operation. On the other hand, it is quite certain that the presence of much distension adds very greatly to the difficulties and danger of the case if any large operation be undertaken.

We know but little about the conditions that regulate the absorption of septic matter from the peritoneal cavity. That great absorption and destruction of septic matter does take place there is no doubt. What becomes of it? It is probably excreted directly into the stomach and perhaps into the intestines and thus got rid of by vomiting and defæcation. Possibly distension plays an important part in this absorptive and excretive process. Certainly free purgation has an excellent effect in draining away septic products in many early cases of peritonitis. It is not, however, always possible or judicious to employ this remedy, because the movements of the intestines may in certain stages of peritonitis do much harm in disturbing adhesions and disseminating poisons over healthy peritoneal surfaces. In later stages when the intestine becomes paralysed and unable to contract, distension is a sign of very bad omen.

There is a class of case in which great distension of the whole abdomen occurs early in connexion with the escape of a small quantity of septic matter into the peritoneal cavity. There is little or no tenderness, the patient's general condition is good, there is no vomiting, but the pulse is generally rapid. In such cases it is generally best not to do any large operation but to be content with giving plenty of fluid. I may illustrate this by a case of acute appendicitis in a young lady whom I saw with Dr. H. Savory at Bedford. There was so much distension that I was doubtful whether she had not already got a generalised peritonitis. A small incision an inch long showed that the peritoneum was not everywhere affected. A small gauze drain was inserted, the distension gradually disappeared, and a few weeks later I removed the appendix with ease and safety, the patient making an excellent recovery. Any attempt to remove the appendix during the period of great distension would almost certainly have been attended by grave disaster. In more severe cases of the same class there may be vomiting as well and the patient may be so ill that the surgeon is tempted to perform a large and immediate operation. Two years ago I saw with the late Dr. William Travers of Kensington a case of what had been supposed to be one of acute intestinal obstruction. The abdomen was greatly distended and very tense and tender and there was much vomiting. After a few hours of waiting the vomiting subsided, then in the course of a few days the distension subsided completely, and later still I removed the appendix by an easy and safe operation.

A few words about the effusion into the peritoneal cavity of serous fluid which occurs in so many cases of acute peritonitis. It is well known that blood serum (or plasma) is bactericidal. The fluid is poured out to help in destroying bacteria. We see this in the cellular tissue when an infected area is surrounded by cedema. According to Sir A. E. Wright, the bactericidal power of serous fluid diminishes in the immediate neighbourhood of an infected area. Its bactericidal power becomes exhausted. Consequently the

letting out of this fluid by operation leads to the effusion of a further amount of fresh serum and the destruction of more bacteria. Hence the beneficial effect of drainage of inflamed areas. The beneficial effects of draining away the fluid in chronic tuberculous peritonitis is thus explained. One other point I should like to allude to and that is the effect of a dose of toxin upon the opsonic power of the blood plasma. A dose of toxin of sufficient size produces at first a lowering of the opsonic index—that is, the patient enters into what is called the negative phase with regard to that particular bacillus. If, then, a further dose of toxin is immediately administered, as is necessarily done to a greater or less degree by the traumatism of an operation, then the negative phase may be still further increased even to the point of death. But after an interval of a few days, about three or four, the opsonic index is greatly raised to far above the normal and operations at this stage can be performed with very much greater safety.

The particular form of bacillus with which we most often have to deal in cases of perforative peritonitis is, of course, the colon bacillus, and it is to this form that my remarks more especially apply. There are, of course, various other kinds of bacilli that play their part in the production of peritonitis, but I do not propose to discuss them to-day. Much has been written of late about the virulence of the bacteria and its effect upon the production of peritonitis. Without wishing to detract from the value of observations upon the virulence of bacteria in particular cases, I would say that I think that of late undue stress has been laid upon the *quality* of the bacteria and too little stress upon the *quantity* of poison that is the cause of a perforative peritonitis. A tolerably extensive experience of peritonitis both in the post-mortem room and at the bedside, an experience extending over a period of more than five-and-twenty years, has convinced me that, other things being equal, it is upon the *quantity* of poison that is suddenly effused into the abdomen rather than upon the quality that the severity of the attack of peritonitis depends; and this is especially true of the peritonitis connected with appendicitis, that most common cause of peritonitis.

Now as to treatment. I have gone at some length into matters of pathology and symptoms because a correct knowledge of the state of affairs before us has a most important bearing upon our treatment. The first point then for us to decide is the diagnosis. The more early and the more exact the diagnosis the better shall we be able to treat an individual case. The next point is to give a prognosis. What is likely to be the natural course of the illness if left to itself. What is likely to be the effect of the various modes of treatment that we can offer. The nature of the perforation, if there be one, the quantity of poison effused, the character of the poison, and especially the *quantity*, all effect powerfully the prognosis in an individual case.

The condition of the patient when first seen and the severity of the symptoms are often enough to enable us to decide at once that the prognosis is extremely grave and perhaps that an immediate operation offers the best prospect of recovery. In peritonitis there is much truth in the saying that prevention is better than cure. We shall doubtless all be agreed that in a case of perforation of a gastric ulcer the sooner an operation is performed the better for the patient. In the case of sudden perforation of an appendix, or of an appendix abscess, the same course is sometimes the wisest one to adopt.

But it must be remembered in connexion with acute appendicitis that *it is only in a minority of cases that there is any actual or visible perforation*. Of 460 consecutive cases of acute appendicitis published by Ochsner, in 255, considerably more than half, there was no perforation. The case for immediate operation is then very different from that of perforation of a stomach. But a peritonitis due to the escape of a small quantity of poison may spread steadily and the patient gradually pass into a condition in which operation is attended with grave risk. Here then is a difficulty, which some surgeons attempt to solve by teaching that all cases of early appendicitis should be operated upon *if seen early*. There is much to be said in favour of this view. But, practically, we surgeons do not see cases of acute appendicitis very often in this very early stage. And if one adopt the rule, also laid down by some surgeons, of operating upon all cases as soon as they are seen we should very often operate on patients at an unfavourable stage, at a stage when the mortality is admittedly high, at a stage when, in

my opinion and that of many surgeons, it is usually best to defer operation, for a time at least. At this later stage much may already have been done by nature in a case of intra-peritoneal inflammation by shutting off and localising the poison. The risk of an operation at this somewhat later stage is that we may undo much of the good that has already been done.

It is absurd to pretend that operations, especially large operations, upon patients suffering from acute peritonitis do not carry with them much risk. What we have to consider is whether the risk of the operation with its necessary attendant evil is, or is not, more than counterbalanced by the good it is likely to do. The harm that a large operation may inflict is twofold: shock from manipulation and exposure of viscera and further absorption of septic matter by diffusion and absorption of poisons already more or less shut off by adhesions and omentum from the circulation. But, on the other hand, what good does an operation do? We can do two things by operation, and practically all operations for peritonitis do these two things only. Firstly, we can, by closing a perforation or removing a septic focus, prevent the further entrance of poison into the peritoneum. Secondly, we can to a limited extent, but to a limited extent only, remove the poison that is already in the peritoneal cavity. I think that most surgeons will agree that the removal of septic matter from the peritoneum by operation is never complete. We can remove a great deal; we can often remove so much that the peritoneum itself can deal with the little that is left. I have in past years seen the abdomen washed out in the most thorough manner with gallons of fluid, but the septic matter was not all removed and the patient, though operated upon in quite an early stage of peritonitis, has nevertheless died of sepsis. What we cannot do by operation is to remove from the blood the poison, bacteria or toxins, that have already entered it. But we can to a certain extent get rid of these poisons in the blood by other means—namely, by encouraging excretion of them through the kidneys and in other ways. And this is done mainly by the administration of fluid in one way or another.

Now for purposes of treatment I should like to divide all cases of peritonitis into four classes.

I. Those in which there is no perforation of any viscus and in which there is no collection of fluid in the peritoneal cavity. The early stage of a peritonitis supervening after an abdominal operation in which no hollow viscus has been opened may be taken as an example. The amount of poison in the peritoneal cavity is in such a case very small. In such cases free purgation by salines and the administration of large quantities of fluid either by the mouth or, better, by the rectum, or in severe cases by the subcutaneous tissue, or even into a vein, is, in my opinion, the best form of treatment. This treatment aims at eliminating by bowels and kidneys the toxins that have already entered the circulation and puts the peritoneum in the best condition for destroying the bacteria that remains. Opium should be avoided in such cases. If this treatment fails to arrest the course of the inflammation it may be desirable to drain the peritoneal cavity if there is any evidence that there is free fluid in the peritoneal cavity.

II. The second class is that in which we know, or strongly suspect, that there is a recent perforation of a hollow viscus, such as the stomach or intestine. Here an immediate operation should be performed with the object of closing the perforation and preventing further extravasation into the peritoneal cavity. When once the diagnosis has been made the sooner the operation can be done the better. If it can be done before symptoms of peritonitis have become well marked so much the better. A perforated gastric or duodenal ulcer affords the best example of this type of case. A minority of cases of acute appendicitis, the fulminating group, come into the same class. The very hopeless cases of perforation of a distended cæcum may also be treated in this way, but it is very rarely, if ever, that operation can in these cases be done soon enough to be of any use.

In the cases of this class it is prevention of peritonitis rather than the cure of peritonitis that is aimed at. With regard to the best method of dealing with the material that has already been effused into the peritoneal cavity much difference of opinion prevails. Some advocate thorough flushing of the whole peritoneal cavity, others prefer local sponging, others again trust mainly to drainage. In the less severe cases operated upon early it is sometimes permissible to close the abdomen without drainage. For my own part, after a tolerably extensive experience of all these

methods, I think that as a rule the less that is done to the peritoneum by washing or sponging the better, and that free drainage usually affords the best means of getting rid of the extravasated septic material. But it is impossible to treat all cases according to any fixed rule. In this second class of cases it is best, in my opinion, to avoid purgatives at all stages, to give at first nothing at all by the mouth, but to give fluid, if necessary, by the rectum or subcutaneously. In many of the less severe cases the administration of water by the mouth does no harm.

III. The next class is a very important one and includes the great majority of the cases of appendicitis that we see in practice—at any rate, in surgical practice. These cases are usually seen at some later stage than those of the two classes I have already mentioned. This class includes those in which there is considerable peritonitis involving much of the peritoneal cavity, with more or less tendency to the concentration of the inflammation in one region. The septic fluid, sero-pus or pus, is more or less localised. Most of the cases of acute appendicitis with abscess belong to this class and many of those without abscess. There is usually a good deal of distension of the abdomen with rigidity and tenderness, but the latter is more marked in one part of the abdomen than elsewhere. Nausea and vomiting are present if food is taken. The pulse and temperature are probably both above the normal. Whether the patient looks very ill or not depends not so much on the amount or nature of the septic material within his abdomen as upon the amount of septic absorption that is taking place from the septic focus. A patient may have a horribly septic focus in his abdomen, a perforated or gangrenous appendix surrounded by very offensive pus, and yet he may be suffering very little from absorption if the septic focus is well shut off by omentum and adhesions and is not under conditions of great tension. I desire to draw your attention especially to this point because one sometimes hears a surgeon speak as if the mere existence of such a septic focus were in itself a sufficient reason for its immediate removal by operation. The operation for its removal may be far more dangerous than leaving the septic focus where it is; at any rate, for a short time until a more favourable period for operation has arrived. Ochsner's opinion and statistics strongly support this conclusion.

Now how are we to treat these cases? Our natural surgical tendency in such cases is to remove the septic focus as soon as possible, and a great many surgeons, I think the majority of the surgeons who write upon appendicitis, teach that this should be done. In some cases it is undoubtedly good treatment to do so. But in many cases I venture to think that immediate operation is very bad treatment and does immense harm. When we are brought face to face with one of these cases we ought, I think, to ask ourselves first of all what is likely to be the course of events if we do not operate at once? Secondly, what good and what harm may we do by operating at once? Thirdly, if we do operate what manner of operation should we do, for there are several?

The ordinary course of events in the majority of cases in my experience is that the symptoms increase in severity up to a certain point and then recede as the septic matter becomes more localised or as it is completely absorbed. When localisation has taken place then an operation should be undertaken for the evacuation of the pus, or in cases where there is no abscess, for the removal of the appendix or other primary source of infection. In a minority of cases, in my experience a small minority, *provided that the case be properly treated* throughout, the inflammation will continue to spread and the patient will either die or will pass into the next class of case which I propose to discuss.

It is exceedingly hard in a given case to give an accurate prognosis. The duration of the illness, the stage which it has reached, the severity of symptoms, and especially the increasing severity of symptoms are the points upon which which we must endeavour to base our prognosis and consequently our treatment. But the great majority will improve and pass into a more favourable condition for subsequent operation if not interfered with by operation at this acute stage. If it is decided to abstain from operation at this stage it is extremely important that other treatment should be carried out rigidly. The most important point is *not to give purgatives* or large enemata and *not to give food by the mouth*. The patient can do very well without any food for a day or two, and if it be necessary to give him nourishment this can be administered quite well by means of small nutrient enemata. If the patient is thirsty he should be given fluid

and the most convenient way of doing this is to inject considerable quantities *very slowly* by the rectum. Care should be taken not to distend the rectum, but to let the fluid run in so slowly that it is absorbed as fast, or nearly as fast, as it enters. If the patient is not thirsty there is no need to give much water. He should be kept as quiet as possible and should be placed in such a position that the thorax is considerably higher than the pelvis. Any fluid within the abdomen will then tend to gravitate towards the iliac fossa or the pelvis, situations in which it can subsequently be most easily dealt with.

For some years I have treated cases of acute appendicitis of this class and the next on these lines. The number of cases I have had to deal with is no doubt smaller than the number that come to surgeons who have written much on appendicitis. I suppose, too, that I must have been singularly favoured by good fortune, for in the last two years I have lost but a single case of acute appendicitis, either in hospital or private practice—a boy who came into hospital on the second day of a very acute attack. He was not operated upon and he died two days later. A necropsy showed a considerable abscess which had burst, evidently before admission to hospital, and which had set up a general peritonitis. A stout old lady of over 60 years of age whom I saw with Dr. Charles Drage of Hatfield also died from peritonitis without operation. She was very fat and in bad condition and her peritonitis may possibly have been due to appendicitis, although I think it was due to another cause. There was no post-mortem examination. The boy might perhaps have benefited had he been operated upon when I first saw him, although even then his chance of recovery would have been poor. The old lady was one upon whom no prudent surgeon with any regard for his patient could have operated with the least chance of success. On the other hand, I can point to a considerable number of cases of acute appendicitis with peritonitis which I have had to treat within the same period, upon none of which did I do any immediate operation and all of them have recovered. Some underwent no operation at all; most had the appendix subsequently removed in a quiet interval; others had abscesses opened at a later stage at periods varying from a day or two to several days after I first saw them.

But I have no desire to dwell upon my own cases. I prefer to bring forward the very large and detailed series of cases published by Dr. Ochsner,² who, like so many American surgeons, has had an unusually large experience of appendicitis. He deals with 1000 cases of appendicitis and his results are tabulated as follows: Chronic appendicitis or interval operations, 540; mortality, .5 per cent. Acute appendicitis without perforation, 255 cases; mortality, 1.9 per cent. (of these six entered the hospital within 36 hours of the attack). Acute appendicitis, perforated or gangrenous, without abscess, 55 cases; no deaths (of these five entered the hospital within 48 hours of the attack). Acute appendicitis, perforated, with abscess, 117 cases; mortality, 3.4 per cent. Acute appendicitis with diffuse peritonitis, 33 cases; mortality, 30 per cent. Of the total number of 1000 cases 22 died. "Of the 255 acute appendicitis cases without perforation, 200 were operated upon immediately on entering the hospital and 55 were treated by gastric lavage and absolute prohibition of food and cathartics of any kind by the mouth, the nutrition being accomplished by means of small enemata. Of the 55 cases, perforated or gangrenous, but without abscess, 21 were operated on at once and 34 were first starved until they seemed in a safe condition for operation. In most of these cases the appendix was completely surrounded by the omentum and held away from all other abdominal structures. Of the 117 cases of acute appendicitis in which abscess had formed, 39 were operated on at once and 78 were treated by prohibition of all nourishment and cathartics by the mouth until their condition seemed sufficiently improved to make the operation appear safe. Of the 33 cases entering with diffuse peritonitis resulting from perforated or gangrenous appendicitis, all were treated at first with gastric lavage and exclusive rectal feeding. Of this class a number were in a dying condition when they arrived at the hospital, which accounts for the high mortality in this class of cases. Seven of these cases were not operated on for the reason

² A Review of the Histories of One Thousand Consecutive Cases of Appendicitis operated on at the Augustina Hospital during the thirty-three months from July 1st, 1901, to April 1st, 1904, by A. J. Ochsner. A full abstract of this paper will be found in the Journal of American Medical Association, Dec. 17th, 1904, from which I quote.

mentioned, but they are counted among the deaths in order to include the entire mortality of all cases treated."

Many cases of acute abdominal inflammation are first seen by the surgeon when the distension and acute symptoms have already begun to subside and in many of these operation may at once be undertaken with advantage. There is often danger in waiting too long and allowing pus to disseminate, although the danger is not usually so great as that of operating too soon in these cases. The treatment of localised intraperitoneal suppuration scarcely comes within my province to-day. But I might just say that one or other of two classes of operation should be done according to the stage which the suppuration has reached and the situation of the abscess. Either the abdomen should be freely opened, the region of the abscess carefully walled off with gauze *before* the abscess is opened; or else, if the abscess is in contact with the abdominal wall over a considerable area (and palpation affords the best means of determining this) a simple incision into the abscess within the area of adhesions is sufficient. The danger of the first course is that the abscess may unwittingly be opened before the gauze packing has been placed in position; the peritoneal cavity then becomes suddenly flooded with a large quantity of septic material and very grave risk is incurred. This is a not uncommon cause of death in unskilful hands. Frequently this method of opening the abscess is the only one that is available, as in cases where the pus is not in contact with the abdominal wall in such a manner that it can be opened by simple incision. The danger of the second course is that in the simple incision of an abscess, such as that of the appendix, the area of limiting adhesions may be transgressed and the non-adherent and consequently absorbing peritoneal cavity opened up and flooded with septic pus in the manner just described with equally disastrous result. I show you here six diagrams which I published some years ago in THE LANCET.³ They are of cases of appendix abscess which came under my notice in the post-mortem room when I was surgical registrar. Each shows the area over which there were adhesions between the abscess and the abdominal wall. I have now added to each the incision that was actually made by the operator and it will be seen that in all the cases the non-adherent peritoneum was opened with fatal result.

A very excellent method of opening localised collections of pus, and one that I frequently employ, is that of making an incision through the posterior fornix of the vagina in women or through the rectum in males. But in order to do this with safety there must be a sufficiently large collection of pus, and ordinary care must be taken not to wound other important anatomical structures. It is frequently far safer to drain by the vagina than to do so through a deep incision through the anterior abdominal wall. The one thing that must not be done in the opening of an intra-abdominal abscess is to cut into it without due regard to the protection of the non-adherent peritoneal cavity. Either the general cavity must be carefully protected by gauze packing or advantage must be taken of adhesions already present if disaster is to be avoided.

IV. I come now to the fourth class of cases, those in which there is a general diffuse peritonitis with general distension of the whole abdomen and purulent fluid diffused over a large portion or the whole of the abdomen. I would here point out that diffusion of septic matter over the *whole* abdomen is not so common as is often thought. The post-mortem experience that I gained during the years when I was surgical registrar to a large London hospital taught me that many cases which clinically seemed to be cases of general peritonitis involving the whole peritoneal cavity were often found post mortem to be really cases in which the peritonitis was localised to a portion only of the abdomen, perhaps two-thirds or three-fourths only. Generally some small portion of the peritoneal cavity was not involved.

Of the symptoms of this form of peritonitis I need not say anything as they are sufficiently well known to all of us. With regard to the appropriate treatment there is much difference of opinion. Dr. Gibbon⁴ of Philadelphia in the paper I have before alluded to says that "surgeons in their attitude towards a generalised peritonitis may be put into three classes. Those who do nothing in the way of operative interference but simply keep the patient quiet and the stomach empty until the inflammatory process and exudates

have become localised, and if the patient lives until this has taken place they then operate. The next class is made up of those who believe in immediate and radical surgical intervention, consisting in freely opening the peritoneal cavity, breaking up all adhesions, opening all pockets and thoroughly irrigating the entire peritoneum. The third and more recent class believe that the abdomen should be immediately opened, the cause of the inflammation sought for and removed, and the abdomen thoroughly drained with the least possible disturbance of viscera." Dr. Gibbon goes on to say that it has always seemed to him that "the failure to get the good results reported from certain lines of treatment is because we do not carry them out as specifically as is recommended by their advocates: in other words, we, inclining possibly to the operative treatment in employing the Ochsner method, do it half-heartedly without expecting a good result, whereas Ochsner carries it out to the letter and expects good results. We have all seen a very incomplete and injurious irrigation of an abdomen done in cases of general peritonitis. Such treatment, unless it is carried out thoroughly and every part of the abdomen irrigated, can be of little benefit if it is not definitely harmful, and this accounts for the difference in results obtained at the hands of those who are enthusiastic over irrigation and of those who do not look upon it with the same confidence."

The Murphy method as described by Gibbon "consists in making a small opening in the abdomen, closing the perforation or removing the appendix as the case may be, the introduction of a large drainage-tube into the pelvis, placing the patient in a sitting posture of from 35 to 45 degrees, and the administration of a quart of salt solution every two hours per rectum." Gibbon quotes Murphy's words as follows and they seem to me to be of the greatest importance: "It is a fatal mistake," says Murphy, "to mop, wash, or handle the intestines or peritoneum. Then, too, the simple opening of the abdomen relieves the pressure under which the pus rests, and we know all that is necessary to stop absorption from a pus accumulation in any part of the body is to open the cavity and relieve the pressure. It is injurious and even dangerous to milk or even handle an acute abscess; it is doubly so to handle an infected peritoneum, but the pressure should be relieved by opening it, allowing it to drain, and exposing the anaerobic flora to the air." Gibbon describes eight cases of his own in which he had adopted the Murphy method and he had five recoveries and three deaths. He expresses himself as a warm convert to the Murphy method. He further states that he had heard Murphy say in October, 1905, "that he had had 29 cases of diffuse or general peritonitis in which he had employed his method with but a single death, this one occurring from pneumonia on the sixth day."

Like most surgeons, I suppose, I have tried all three methods at different times, and I am inclined to the belief that of the three the Ochsner method is the one that is most suitable for the great majority of cases of diffuse peritonitis. For the very worst class of case, in which no delay is possible, and they are, I think, few in number, the Murphy method is undoubtedly good. I entirely agree with those who condemn the very large operations which are so often performed in this country. The mortality of these operations, so far as I have seen and so far as I can judge from those who have published detailed statistics of their cases, has been literally terrific.

I think I notice at the present day a tendency amongst surgeons of large experience to abstain more and more from these very large operations. We are beginning, I think, to realise more and more fully that many very bad and apparently hopeless cases of general peritonitis will recover if the surgeon will abstain from doing any large operation. In this connexion I should like to refer to an admirable paper by Mr. J. Rutherford Morison, a well-known advocate of extensive operation in early cases of appendicitis. He gives details of a large number of cases treated with excellent results. But speaking of general peritonitis he says: "General peritonitis of more than 24 hours' duration is not likely to be benefited by operation, for the favourable chance has been lost; indeed, an operation not seldom hastens the end and may be the cause of death," and he goes on to say, "it is not without surprise that I have observed recovery in such cases without operation." In this paper he gives details of 15 cases of general septic peritonitis. Ten of them were treated by operation, with eight deaths, while five of them were treated without operation, with only one death! The numbers are small, but I think significant,

³ THE LANCET, Dec. 30th, 1899, p. 1858.

⁴ The Treatment of Diffuse and General Peritonitis with Special Reference to the Murphy Method, New York Medical Journal, April 7th, 1906, p. 706.

and Mr. Morison has recently told me that with regard to this particular class of cases he has largely given up his former practice of operating upon them.

A few words about the posture which a patient with suppurative peritonitis should be made to assume. For a long time, as I have already said, I have been in the habit of raising the shoulders of my patients so that fluid in the peritoneal cavity may gravitate towards the pelvis where it can be most easily and safely dealt with. Murphy carries this practice much further and according to Gibbon and Fowler he "has been so much impressed with the advantage of the sitting posture that he now has cases of peritonitis brought to the hospital in the ambulance, anæsthetised, placed on the table, and operated upon in this position." I feel sure that Murphy is entirely right and I wish that I could persuade all those who send cases of peritonitis to the Royal Free Hospital to adopt the same precautions.

And now, in conclusion, let me add a few words about that most fruitful source of peritonitis, I mean, of course, acute appendicitis. It may seem to you an extraordinary thing for me to say, but I say it with deliberation and with a profound conviction of its truth, that the very high gross mortality in acute appendicitis which undoubtedly exists in this country (and in other countries as well) is largely due to the fashion of indiscriminate, excessive, and injudicious operating which not long ago was at its height but which, I venture to think and hope, is now showing signs of subsiding. Appendicitis may or may not be more common than it used to be. Personally I do not think it is, although no one can deny that it is more often diagnosed and I have no doubt that deaths from it are much more common than they used to be. It is not unusual to hear it stated that modern operations for appendicitis have resulted in a great saving of life. I believe that, on the whole, the exact converse is the case. If we were all to go back to the old treatment of appendicitis that was in vogue 25 years ago, and that is still in vogue in many parts of the country, there would be a lesser mortality from appendicitis than is the case at present. This is a very different thing from condemning all operations for acute appendicitis, which I am far from doing. Nor do I wish to detract from the excellent work that is done by many surgeons of large experience who advocate, and perform, frequent operations for appendicitis and publish very often brilliant results. I am thinking rather of the far larger number of operations performed by those who do not publish their results, performed by those who do not discriminate sufficiently between the cases that should be operated upon and those that should not be operated upon. It is the doctrine of "Here is a septic focus, let us take it out now at all costs," that, in my opinion, does so much harm. It is for more discrimination in the choice of an operation, in the kind of operation, in the stage at which it should be performed, that I would venture to plead.

ACUTE YELLOW ATROPHY OF THE LIVER FOLLOWING OPERATION FOR INTESTINAL OBSTRUCTION.

By C. E. CAMPBELL-HORSFALL, M.B., CH.B. VICT.

ALTHOUGH from time to time cases of acute yellow atrophy are placed on record, yet it remains one of the rarest diseases met with and its causation as inexplicable as ever. But cases following operations are rarer still and with but one exception have all ended fatally. Perhaps before discussing the case which is the subject of this paper it would be well briefly to review the general features of acute yellow atrophy. Osler defines it as jaundice associated with marked cerebral symptoms and characterised anatomically by extensive necrosis of the liver cells with reduction in volume of the organ.

Etiology.—This is one of the rarest diseases. There are not more than 300 cases on record. It generally occurs between the ages of 20 and 30 years, but cases have occurred in quite young children and even in infants. It is more common in women than in men and seems to have a remarkable connexion with pregnancy. It has followed fright or profound mental emotion and is sometimes secondary to other ailments—typhoid fever, sepsis, syphilis, gonorrhœa, and phosphorus, antimony, and arsenic poisoning. Although the symptoms produced by phosphorus

poisoning are very similar to those of acute yellow atrophy the two conditions are not identical.

Morbid anatomy.—The liver is greatly reduced in size, looks thin and flattened, and sometimes does not reach more than one-half or even one-third of its normal weight. It is flabby and its capsule is wrinkled. On section the colouring is mottled, there being red and yellow areas of softened liver tissue. The organ may cut with considerable firmness. Microscopically the liver cells are seen in all stages of necrosis and in spots appear to have undergone complete destruction. The bile ducts and gall-bladder are empty. Hunter considers that it is a toxæmic catarrh of the finer bile ducts, similar to that which is produced after poisoning by toluylene diamine or phosphorus.

Symptoms.—Commences usually as a gastro-duodenal catarrh and at first the jaundice is thought to be of simple nature. This generally lasts a few days and then severe symptoms set in, headache, delirium, trembling of the muscles, and in some instances convulsions. Vomiting is a constant symptom and blood may be brought up. Hæmorrhages occur into the skin or from the mucous surfaces. Pregnant women may abort. With development of head symptoms jaundice usually increases, coma sets in, and gradually deepens until death. The temperature is variable, sometimes afebrile and in some cases marked pyrexia occurs. Usually the temperature rises just before death to 105° or 107° F. The pulse is usually rapid and the tongue is coated and dry. The individual enters into a typhoid state. The urine is bile-stained and contains tube casts. Leucin and tyrosin crystals are not constantly present. The stools are clay coloured from the absence of bile. Cases are almost invariably fatal, there being but few instances of recovery. Nineteen recoveries have been collected by Weising and Ballin. No known remedies have any effect on the course of the disease.

As a sequel to operations Max Ballin has collected ten cases from the literature, a table of which I append below, including the one that came under my own observation. All of these cases, with the exception of Ballin's own case, died.

The following are the notes of the case under my care. On the evening of May 31st, 1904, I was called to see the patient, a young married woman. She complained of severe pain in the lower part of her body and had vomited several times. She had not been feeling well for some time, and two days before I saw her she had had a slight shivering fit. When I saw her she looked anxious and was in evident distress. She had a pulse of 100 and her temperature was 100.5° F. On further examination I found great tenderness over the abdomen in the lower part, most marked in the left iliac region. No tumour could be felt and there was no marked dullness. The abdomen moved readily with respiration. I elicited the following further facts. She had been perfectly regular until her last period, which occurred a fortnight previously to the present attack, when she only lost a small quantity of blood, and this not until six weeks after her previous period. She had been very much troubled with constipation which had latterly become much worse. She also suffered from intense dysmenorrhœa, being utterly prostrated during the menstrual flow. A vaginal examination revealed marked fixation of the uterus, but otherwise it was negative. The rectum was empty. The patient was kept in bed and a small dose of calomel was administered. On the next morning there had been a slight watery action of the bowels. The temperature was 99°, the pulse was 100, the pain was more severe, and the vomiting continued, there being nothing distinctive about the vomit. I saw the patient later in the day, when her condition had not improved. A definite mass could be now felt below and to the left of the umbilicus. Vomiting continued, though not fæcal, and the bowels refused to act. There was no marked distension of the abdomen and no visible peristalsis was present. The pulse was still rapid but the temperature was normal. As the symptoms pointed to intestinal obstruction I decided to operate.

The patient was removed to a nursing home, and on the afternoon of the same day chloroform was administered and the abdomen was opened in the middle line below the umbilicus. On opening the peritoneal cavity a little serous fluid escaped. There were marked signs of an old peritonitis, the pelvic viscera being matted together. The cæcum had been dragged over and was fixed on the left side of the abdomen together with the appendix, which was firmly adherent to the cæcum in its entire continuity. The appendix