compared to the effect of a blow upon the pit of the stomach, though why the shock of abdominal injury should be so especially profound I will not undertake to explain. In other cases the patient lives a day or two, yet never rallies. A man, 40 years of age, who was under treatment for a chronic ulcer of the stomach, became suddenly, at 8 o'clock one morning, pale, pulseless, and delirious (lipopsychia); he had evidently undergone a great shock. Throughout the day he continued to be delirious; consciousness never fully returned. Considerable reaction took place; his skin became hot, his pulse full and frequent; he retched a little, but did not vomit. The same night he became comatose, and so died at 8 o'clock next morning. I regret being unable to say whether the urine was suppressed or not. On examining the body the ulcer was found to involve the pylorus, and to have perforated on the duodenal side thereof. The contents of the stomach were in the peritoneal cavity, but there were no signs of peritonitis, neither excessive vascularity, nor exudation of lymph. This kind of perforation closely resembles other abdominal diseases in which lipothymia predominates. You, Sir, have remarked how perforation of the duodenum will simulate Asiatic cholera in its algid stage; the physicians who attended the Duchess of Orleans came finally to the opinion that she died of cholera morbus; and Dr. Wm. Sedgwick has discussed this topic very fully with especial reference to the suppression of urine.

LATENT PERFORATION.

Lastly, the perforation may be latent; that is to say, unattended by any symptoms adequate to the diagnosis. Not that symptoms are utterly wanting; this is seldom the case; there are more or fewer of those so often mentioned—pain, vomiting, lipothymia, and the rest—but they are rendered obscure by occurring in the course of a disease marked by no less serious disorders. The contrast between a person when in good health and when suffering from perforation of the peritoneum is great indeed; but when a patient has been exhausted by four or five weeks of severe typhoid fever, the occurrence of perforation may be a comparative trifle, so far as symptoms are concerned; a little pain in the belly, a little vomiting, attracts no attention. Or there may be no pain, no tenderness, no vomiting; nothing but a sudden defervescence. Or, again, a change in the patient's look is sometimes the chief sign. During a state of typhomania, in particular, the gravest intercurrent lesions often happen unsuspected.

But perforation, latent so far as symptoms are concerned, will sometimes yield one or more of the physical signs of peritoneal tympanites. For instance, in the later stage of typhoid fever, a rapid and great distension of the belly, such as to stretch the skin and make it shine, and attended by disappearance of the liver dullness to percussion, is a tolerably trustworthy token of intestinal perforation, and is sometimes the only token. The liver dullness may disappear before the abdomen becomes distended.

INCISION FOR THE PURPOSE OF DIAGNOSIS.

To conclude by referring once more to puncture of the peritoneum as a means for ascertaining the presence of inflammatory effusion. A small incision would seem to be justified by conditions such as these: First, the patient is suffering from acute peritonitis, and the indications of poisonous infection of the blood are becoming more and more marked; or next, there are reasons for believing that perforation of the peritoneum has occurred. In either case, unless we can bring relief, the patient must die. A small incision will not make him worse; nay, to drain the peritoneal cavity affords a chance of recovery; the only objection lies in the natural dislike to an operation, however small. I wish I could believe that chloroform sleep did no harm, but I fear that this drug has a depressing effect upon persons suffering from acute peritonitis. However, suppose a small incision made, and we find that the peritoneum contains pus or air, beyond simple drainage two further courses lie open to us; we may follow John Hunter's suggestion, and wash the belly out with warm water, a proceeding more serious than simple drainage, or we may enlarge the incision and search for the cause of the peritonitis, a proceeding highly dangerous, for you will, I think, agree with me when I say that operations of this kind have seldom any other effect than to hasten the patient's end. A simple puncture, if it do

not save the patient's life, will at least bring the satisfaction of knowing that the nature of the disease has not been misunderstood.

PERITONITIS: ITS NATURE & PREVENTION.

*Being the Main Argument of a Paper read before
the Harveian Society of London.*

By LAWSON TAIT, F.R.C.S.ED., L.R.C.P.ED.

THE FUNCTIONS OF THE PERITONEUM.

THE peritoneum, for many reasons which need not here be discussed, cannot be regarded as a gland nor yet a mere lymph sac, but its importance in the human economy is so great, that I am disposed to rank it almost next in order to the brain. Not only does it appear in the very earliest stages of embryonic formation, but, if we exclude gross mechanical lesion such as acute hæmorrhage, cardiac valve rupture or other failure, destructive hæmorrhage at the base of the brain, there is hardly any such rapidly and certainly fatal disease as simple inflammation of the peritoneum. Opportunities of studying the facts of the peritoneum are very common, so that our fundamental ignorance is not very creditable. The mere gross anatomy of the peritoneum is usually dismissed in textbooks by an explanation that its peculiarities are for the purpose of allowing the free movement of its involved organs, but I published a number of cases¹ in which there were such variations of the peritoneum (in one case no peritoneum at all), that it is perfectly certain patients may live with very material modifications of this freedom of movement. This is also proved by the fact that peritoneal adhesions, even of the most intricate and extensive kind, seem to give little or no inconvenience of themselves. The peritoneum is developed very early in embryonic life, and many other very important structures are separated from it in the changes. In a certain line of development its functions seem to lie chiefly in the direction of assisting a proper ovulation, but bit by bit it comes to be apparent that all the great organs of the body, except those of the nervous system, seem to require to be involved in its folds in a very curious and singularly invariable way. This does not exclude the heart and lungs, though in the higher vertebrates these have a further and more specialised separation. It is impossible to regard such a fact as this without a suspicion being aroused that there is some definite physiological purpose to be served by its curious plications. If we contrast two such viscera as the bladder and the stomach, we find the former but slightly related to the folds of the peritoneum compared to the latter, yet the movements of distension and contraction are relatively greater in the bladder than in the stomach, therefore mere facility of movement is no explanation of the great cavities.

The bladder is very slightly an organ of secretion and absorption. It has a very scant arrangement of adenoid tissue in its walls. The stomach is essentially an organ of absorption and secretion, and quite half its bulk is gland tissue, and therefore we may find that the arrangement of the peritoneum is associated with glandular function. Certain it is that all the great organs which are regarded as having functions preparatory to and essential to the general nutrition of the body, and most of those regarded as excretory, have this curiously-involved relation to this mysterious and complex membrane, with the doubtful exception of the pancreas. Further, the membrane itself has a series of strange and invariable plications and redundances of surfaces, the omenta, mesenteries, and various appendices which, should its function be that of active secretion or absorption, clearly must be of great service. Then, finally, its surface everywhere is excessively vascular; hyperæmic, as it were, its minute anatomy shows abundant arrangement of stomata, like those on the leaves of a tree, eminently suggestive of active work of some kind. It is most profusely supplied by nerves which are not derived from the motor tract. Normally these nerves convey no sensation, and there-

¹ *Journal of Anatomy*, 1874.

fore it may be assumed that they are nerves for controlling or directing some special function.

In addition to what I have said, I have to point out that one great anatomical fact concerning the peritoneum has had, so far as I can discover, no kind of importance attached to it in its pathological consideration; in fact, it is not alluded to at all save in the anatomical textbooks. It is that all the organs actively engaged in the primary preparation of the systemic nutrients are not only enclosed in the peritoneum, but all the blood gathered from these great organs is collected by a series of minute venous radicles exposed voluminously and immediately under the peritoneal epithelium to any influences which may arise in it. In addition to this, the whole of the blood prepared in or by these organs is mixed together in the portal vein and carried into the liver, where it undergoes its final change previous to the oxidation which seems essential for tissue nutrition everywhere. In the liver there is removed from this mixture of primary digestion a substance which is returned to the intestines—bile. This interesting material has been the subject of countless researches and experiments, but we do not know yet any one satisfactorily established fact about it, save that in excess it acts as a purgative.

All the statements concerning its functions, which have been based on experiments on the lower animals, have been absolutely invalidated by the results of those cases where the operation of cholecystotomy in human beings has resulted in a permanent and complete biliary fistula. In such cases, by reason of the occlusion of the common duct, the bile has been shewn to be not in the least essential to the human economy. It does not act as a preventive of decomposition of the chyme, it is not necessary to the digestion of anything not digested without it, it is not absolutely necessary either as a laxative or the reverse; its absence does not lead to slow starvation as in dogs, for patients steadily gain weight and maintain good health when deprived entirely of their bile for years. We also know that no kind of drug seems in the human subject to excite or diminish its secretion, and its more rapid or slower flow is in no way influenced by any part of the process of normal digestion, so far as my own observations have gone. These conclusions were made and published twelve years ago, and have been amply confirmed since by Mayo Robson. The bile, in fact, would seem to be an effete material, which requires to be passed through the intestines a second time to undergo either complete and unaltered elimination, or to be subjected to some secondary change. Chemical examination of the intestinal contents seems to establish the latter of these alternatives with considerable probability. A few other facts we do know about bile with perfect certainty; for example, that a very small quantity, if dislodged upwards into the stomach, will immediately, and as long as it is there, completely arrest the function of that important organ. It is also known that certain errors of diet, as we call them, will so paralyse the pylorus, increase the quantity of bile, or in some other way put the mechanism out of gear, that bile will pass into the stomach instead of passing downwards through its ordinary duodenal route. When this happens the patient becomes bilious, as it is called. For any mild and temporary form of such condition, everybody knows that the best remedy is something like a blue pill and a black draught, in spite of Bennett's famous experiments and the wholly erroneous conclusions he drew from them.

THE CAUSES OF PERITONITIS.

A consideration of all these elementary facts (elementary, though there is not one of them which in my mind does not possess transcendent importance), and many others, some to be discussed later on, and others not to be referred to for want of time, made me brush aside all the theories of peritonitis which had up to that time been fashionable. The result was that I was led entirely to oppose an acceptance of that theory which now occupies largely the professional mind: I mean of course the universal influence attributed to micro-organisms. Peritonitis is not a lesion which fits into the germ theory of disease at all, and no hypothesis based on the facts of the decomposition of dead infusions in the physiological laboratory helps me to understand the facts of peritonitis, as I have it under my eyes almost every day of my life.

NERVOUS INFLUENCE IN INFLAMMATION.

No theory of inflammation will ever fit the facts of the process which has not its basis in the mechanics of the tissues, and there is one element of which we have as yet far too little knowledge to enable us to solve the mystery: I mean the nervous system and its method of action in those perverted processes which we call disease. The mere difference in the process of inflammation in proportion to nerve supply is enough to suggest this, and the extreme rapidity of the process in the peritoneum must be, in its initial steps at least, due to the disproportionate supply of that class of nerves which do not govern movements, but which are nerves controlling function until they are disturbed, and then they become nerves of sensation. Of the real work and influence of these nerves we know very little, but if we look at the face of a patient, even as soon as twelve hours after the onset of acute peritonitis, even before pain has become a leading feature of the case, we shall be easily led to the conclusion that nerve disturbance is doing more to kill her than microbic invasion. As to the general influence of disturbed innervation in the inflammatory process we need only point to the familiar example of bedsores in cases of fractured spine.

SEPTIC PERITONITIS.

That there is such a thing as septic peritonitis I admit as a possible secondary result of the acute suppurative metritis of the poisoned child-bearer; and I have only to look back to our ghastly experience of the poisonous influence of the festering ovariectomy stump in the days of the clamp for a second illustration. But septic peritonitis, save where definable from evidence wholly extrinsic to the condition of the peritoneum, is an etiological entity which exists only in the mind of the pathological metaphysician. There are no symptoms, there are no physical signs, there are no *post-mortem* appearances by which peritonitis which is septic can be substantially differentiated from peritonitis which is not septic; unless we bury our heads in the sand declaring, as some germ enthusiasts do, that all peritonitis is and must be septic. But this is a vicious argument in a circle which as serious logicians we need not regard. I have also to complain that this term septic, even as generally applied, but specially as applied to peritonitis, has become a veritable shibboleth to the minds of a serious proportion of the profession. It has also become a real danger in the practice of our art, for it constantly intervenes as a real stumbling block to progress.

THE GREAT OMENTUM.

One of the many curiosities and puzzles of the peritoneum is the great omentum. I am disposed to regard it as the arbiter of the intraperitoneal currents. That there is an ebb and flow in the peritoneum I am quite sure, though I have not yet unravelled the secrets of its tides. The great serous sac holds in it, like a dressing bag, a great mass of serous covered organs which may, any and all of them, secrete into its cavity, or absorb from it. Possibly they may carry out one process at one time and the other at another. The omentum, I think, does so certainly and for this reason. In a series of patients suffering from peritoneal dropsy due to the disease which we now call peritoneal papilloma, a disease of epithelial origin, sometimes malignant and sometimes not, a number of cases will be found in which the effusion may be made to come and go as the observer wishes, by merely keeping the patient in bed or out of it. In others it cannot be altered, and these are the cases in which, on exploration, it will be found the great omentum is chiefly or very largely involved. As everybody now knows, the practice of opening and cleaning out all such cases has resulted in the curing of about half the cases, and the removal of huge masses of neoplasm, all formerly regarded as cancerous, and which in all probability would have been all fatal in their issue. One incidental result has been met with in the conclusion just indicated, that the great omentum possibly has a very important function, hitherto unsuspected; in relation to this, bear in mind that the huge venous system of the omentum joins the other systems from the great digestive organs, in pouring its blood into the portal vein—a fact in itself extremely suggestive.

WASHING-OUT V. SPONGING.

I have now to unfold, with greater brevity and want of detail than the occasion deserves, the process of evolution of

my present views of peritonitis, beginning as it did in 1878, when, by the final dismissal of the clamp, we abolished for ovariectomy the extraperitoneal treatment of the pedicle. Up to that time everything was so obscured by disaster and anxiety that real light upon anything was impossible. As the mortality of ovariectomy rapidly diminished, the fear of the peritoneum disappeared, and then came the second step in the revolution, when Bantock and I simultaneously raised a protest against the tapping of ovarian tumours. For several years later, however, we still got the old tapped cases, with their formidable adhesions and other horrors, and those cases died, and died, I suspected, chiefly from the damage done to the peritoneum by the sponging necessary to cleanse it. I gave up the use of sponges as far as possible, and took to washing the cavity out by dashing in large quantities of warm water, gradually evolving the neater and more effective method by the trocar nozzle. This again brought upon me strong condemnation from various quarters, but I went steadily on as I found my cases recovering, until it became evident that practically no case was hopeless, however bad it looked, providing the bleeding was stopped and the peritoneal cavity was properly washed out and drained. When I hear some authorities say that they never wash out and never need to drain, I merely conclude that they lose cases which I save, and that they put their ill results down to the omnipotent germ.

As a part of the same argument, I have to record my observation that the opening of the abdomen in cases of ascitic effusion due to the papillomatous alteration of the peritoneal surface, the careful washing of it out and its subsequent drainage, if carefully and tenderly performed, is an operation as absolutely devoid of risk as an operation can be; yet I never trouble my head about excluding germs. I have seen very few deaths in some hundreds of cases, and not one in which the result was reasonably attributable to the operation, certainly not a single instance in which it resulted from the onset of acute peritonitis.

NERVOUS ORIGIN OF PERITONITIS.

I therefore conclude that peritonitis, after abdominal operations and injuries, begins in a stimulus, just as in the web of the foot of the frog. I do not think that there can be a doubt that nerve injury enters largely into the explanation of this. Next comes the period of mechanical congestion and stasis, and at this point we enter upon a consideration of the clinical symptoms of peritonitis; and here let me say once for all that I absolutely exclude from my own arguments and that I will not admit for the arguments of anyone else any deductions made from observations which are not absolutely verified by some kind of intraperitoneal investigation, either *ante* or *post mortem*. The peritoneal cavity is a region in which the unexpected is so constantly turning up that nothing but actual physical experience inside it can be admitted as serious argument.

THE SYMPTOMS OF PERITONITIS.

The question now comes in order what are the symptoms of peritonitis? My answer is that they are very numerous, very various, and very serious, but that there are none, no not one, nor any group of them, upon which absolute reliance can be placed.

TEMPERATURE AND PULSE.

Of late years we have had much reliance placed on the records of temperature and pulse for clinical purposes, and in many diseases their value is undoubted. But for peritonitis they are not only untrustworthy, but may become positively misleading. Some years ago I performed a trifling operation on the uterus. It was only the insertion of a solid piece of sulphate of zinc, and I had done it in this patient some ten or twelve times before. On the last occasion it made her ill for some days, and two of us watched her with great care. As we had a perfectly, or almost perfectly, normal temperature and pulse record, we absolutely refused to regard the ailment as serious, and we were not prepared for a quite sudden termination of the case about the tenth day. On *post-mortem* examination we found about a pint of pus in the abdomen, and I do not suppose I have ever had so damaging an experience as was then made for me by my ignorance of the fact that acute fatal peritonitis may run its course and yet the temperature never rise a full degree above normal, nor the pulse be increased beyond 90 beats in the minute. I

have been more alive to the fact since; yet, on the contrary, I can show records after operations of the temperature at 106° and the pulse at 160—even at 200, where recovery took place and the question of peritonitis was never seriously entertained. Do not for a moment imagine that I recommend you to break your thermometers and discontinue your pulse glasses, but do not put exclusive faith in them.

ALTERATION OF FACE.

There are two signs so constantly present in peritonitis that I regard them always with anxiety, even though their presence is not to be taken as infallibly indicative of the inflammatory danger. I mean alteration of the patient's face and distension of the intestines.

The alteration of the patient's face is deceptive, because many fidgety people, and those who bear pain badly, will put on a face indicative of danger when there is none; and the habit many practitioners have of saving their patients' pain after an operation by the administration of opiates is a source of great danger, for it masks this facial alteration at the very time when its aid is most needed. Pain, though by no means a pathognomonic sign of peritonitis, is pretty constantly and markedly present at the onset. An opiate, therefore, conceals the first and most readily appreciated indication of danger. Sometimes, however, an opiate must be given after an operation, and then I always give it by the hypodermic needle, as that avoids the constipating effect of opium by the mouth and leaves me my great means of assistance ready for use—I mean purgation. The alteration of the face I most fear is not one of pain, but of anxiety, accompanied by a tendency to chatter and ask questions. If a patient will be quiet and not talk she is pretty sure to recover. If she persistently chatters she is pretty sure to die. On relief of the symptoms, which practically means getting the bowels to act, the face becomes placid and the patient quiet.

INTESTINAL DISTENSION.

The symptom which I most dread is intestinal distension, and that all my clinical aids, assistants, and nurses are constantly on the watch for. I never saw fatal peritonitis in which distension was not a prominent feature. I never said that every case of intestinal distension after an operation represented a case of peritonitis, yet this is the manufactured view which an enthusiastic young assistant surgeon of a well-known metropolitan hospital set himself as a task to combat some two years ago. But as all cases of peritonitis have that as a symptom—and an early symptom as well as a very distressing one—my rule has always been *obsta principii*, and my empirical rule has been a most successful one. My mortality from peritonitis has dropped *pari passu* with my complete establishment of the practice of anticipating the peritonitis as soon as I see distension. According to my own rule I am, of course, not entitled to say that there was peritonitis in any of these cases, or that it was cured by purgation; but when I see cases with all the symptoms which are ever seen in peritonitis, and find the symptoms yield in those in which my purgative treatment is efficient and in time; and, on the other hand, when I see similarly affected cases in which time has been lost and purgation not effected, and see these die, and then peritonitis is found on *post-mortem* examination, I am strongly disposed, as is the fashion in medicine, to argue *post hoc ergo propter hoc*, to believe that such cases do have peritonitis, and that most of them recover. Here I may be wrong, but this I do know, that the moment we see distension we purge, and if we succeed in purging the patient recovers; if not she dies. Therefore I am content to say that at least we prevent peritonitis, and it is for that purpose that my routine treatment is directed.

THE CAUSE OF DEATH IN PERITONITIS.

At this point I am brought back to my physiological beliefs about the peritoneum, and I say at once that the cause of death in peritonitis is a disturbance of the ebb and flow of the serum stream of the peritoneum, and the disturbance of the function of the liver. In fact, the liver is the lethal organ in peritonitis.

In any ordinary bilious disturbance, say what is known as the bilious vomiting of the crapulous, the first thing which happens, if I read the phenomena aright, is an over-secretion

of bile. How this is arrived at I do not know, but there are two results from it which are invariable. The first thing is that the usual course of the bile is diverted; the pylorus seems to be temporarily paralysed; bile flows into the stomach, and thence is speedily ejected. The second result is—and sometimes the two are not concurrent, but are alternative—the bile passes downwards and excites a bilious diarrhoea.

In peritonitis the first serious symptom—the symptom which when fully established after the occurrence of distension generally means a fatal issue—is bilious vomiting. At first the character of the vomit is that of simple gastric catarrhal flow, faintly coloured with bile; this rapidly alters to such an extent as to show that more and more bile is passing into the stomach, and the quantity of the ejection rapidly increases. If a resolution of this very fatal symptom is brought about, the quantity of bile diminishes, and finally disappears. If a favourable issue is not to be the result, the vomit rapidly alters in character till the too familiar coffee-ground matter, which is due to hæmorrhagic flow from the liver, appears. All through the course of the case the general distension of the abdomen increases, till it acquires a drum-like hardness, the bowels remain obstinately quiescent, and the patient dies.

The course thus described is not one without exception in almost every particular, but it is an accurate description of the great bulk of deaths from peritonitis after operations, and the course of the cases generally dates from the first appearance of distension (towards the close of the second day) till the final issue between the close of the fourth and fifth days; and so constant has been my experience of time in this matter that for years I have regarded the fourth night as the critical time for all abdominal sections, except those involving the use of the clamp in hysterectomy, and these never can be regarded as free from the risks of peritonitis till the stump has come off and the wound nearly healed.

These critical times have an indication to me of very great importance, to which I shall again recur, and to which I give far more close heed than I do to any question of theoretical sepsis, for its practical value is far greater.

At the *post-mortem* examination of fatal cases of acute traumatic peritonitis very little variation, save in degree, can be found. The main features are the uniform occupation of the intestines by vast quantities of gas and some fecal fluid in which there is a marked absence of bile, as shown clearly by naked-eye appearances, and proved incontestably by chemical analysis. There can be no question that a most serious disturbance is due to the misdirection of the bile current. The coats of the intestines are thickened and the muscular fibres of the intestine blurred in their outline by hæmic stasis and its resulting effusion. The stomata of the peritoneum are very prominent, and crowded with cells which appear as if they were streaming out. The stigmata are greatly exaggerated in size, and the minute blood vessels present all the characters familiar in the frog's web. If I had only a knowledge of what might happen in such structures with such alterations, but without the clue afforded by the clinical history of peritonitis, I should say that the intraperitoneal flow must have been checked, and the muscular coats of the intestines paralysed. As to the latter, I do not think for a moment there can be a doubt that it is one of the initial processes in peritonitis, and largely accounts for the distension. That the intraperitoneal flow is checked is indicated by two things—first, that the peritoneum is dry, that is, free from effusion, just in proportion to the acuteness of the processes, till in a very chronic case, which will probably recover, you may be called upon, as I have been, to remove pints of purulent fluid from the abdomen.

The second point suggestive of this conclusion is that we might urge the increased quantity of biliary fluid thrown off by the liver as being due to the increased quantity of fluid passed into the portal circulation by the arrest of the intraperitoneal flow. The ejection of the bile from the stomach may also possibly aid the mechanical stasis in the intestine, and its consequent distension by depriving the muscular coats of a natural stimulus to vermicular action, for that is suggested as one at least of the subsidiary functions of the bile, though it is quite certain that this action goes on without the bile in normal digestion.

Further, at *post-mortem* examinations you may find lymph and purulent effusion in the peritoneal cavity to a greater or less extent, this again varying with the time occupied by the course of the case. You rarely see much if the case has not gone over the fourth day, and you will find pints if the case has lasted a fortnight.

For a proper *post-mortem* investigation of a case of peritonitis, microscopic examination of the structure of the liver is essential. It will be found engorged and evidently suffering from great changes, its normal tissue seeming to have undergone disintegration. The exact microscopic condition, especially after the whole organ has been well washed out, is a field of inquiry which I am certain would yield results worth much closer investigation than I have been able to give it. The altered effect of the liver function upon the blood which passes into the pulmonary circulation is also a direction in which investigation has yet to be made.

PROGNOSIS IN PERITONITIS.

I have now a list of considerably over three thousand consecutive abdominal sections of which the mortality is something close upon 8 per cent., and the great bulk of this mortality has been due to peritonitis. I have also been called to a large number of fatal cases of peritonitis where the causes have not been associated with my own work. A very large proportion of my own patients have, in my opinion, recovered from operations followed by traumatic peritonitis more or less severe. I should say, therefore, that it must be a long way under the mark when I express my conviction that I have seen over a thousand cases of this serious disease. If I were asked what exists in my mind as the most potent conclusion concerning it, and the most important element for the patient, I should answer "time." If the grave symptoms are all matured on the fourth day after an operation, a fatal issue is pretty certain. If they hang over till the sixth or later, the chances of the patient's recovery increases in a geometrical ratio, always excepting cases of hysterectomy, and therefore it is that I have a corollary, confirmed absolutely by empirics, that taking time by the forelock in dealing with peritonitis is everything. It is no use talking about treating peritonitis; you have not a chance as a rule; you must prevent it. Therefore, my policy is always to get control over the vermicular movements of the intestinal tubes before the mechanical stasis of the inflammatory process has rendered it impossible, and, whether my theory be correct or not, my practice has had most satisfactory results.

THE PREVENTION OF PERITONITIS.

Years ago I was driven to the determination to discontinue absolutely the use of opium by the mouth after abdominal operations, for two reasons: First, its action on the intestines is most certainly to modify and even suspend vermicular movement; and secondly, it masks the real condition of the patient. One dose of morphine, given under the skin immediately after the operation, for the double purpose of relieving pain and modifying shock, is all that my patients ever get, and the great bulk of them do not get that.

Another matter regarding which my convictions were altered very early in my practice was that strange and unaccountable thirst which inevitably follows the opening of the peritoneum, even in a mere exploratory incision as much as in a protracted and severe operation. Cut down through the abdominal wall to the peritoneum, without damaging the membrane, and there is no thirst—at least, it is rarely seen, and is then very transient. But open the peritoneal cavity and you have an invariable symptom—intolerable thirst lasting for twenty-four to sixty hours. At first I thought that this was a clear natural indication that the patient should have plenty of fluids and plenty of ice. But I have long since come to regard ice as one of the things which should be banished absolutely from the sick room. It never acts in any other way than to increase thirst, and the best thing to allay thirst after an abdominal section is warm water. Gradually I found it wise to diminish the quantity of fluid after an operation to the vanishing point, and now I keep my patients for as nearly forty-eight hours as may be in absolute starvation—this being modified, of course, by age and previous exhaustion. I have, indeed, come to regard this thirst, not as an indication for the administration of

fluids, but, on the contrary, as one indication that the traumatism of the peritoneum has altered its flow and ebb in some strange way, and that the less fluid there is about the sooner will the natural rectification of the process be effected. Certain it is that since I have adopted this policy, sickness after operations—I do not mean mere anæsthetic sickness, but the dreaded sickness of the second and third day—has almost disappeared, whereas formerly it was almost a routine symptom.

Again, if sickness sets in on the third or fourth day, or at any time after, all food (and in that I include water) is absolutely stopped for 12 hours or even longer if necessary. I was driven to this by the uniform experience that all drugs generally employed for the arrest of vomiting were, under the circumstances of my practice, absolutely futile; that any food given came back only altered by biliary admixture. Further, I was influenced by the perfect certainty that nothing could possibly be digested or absorbed by the stomach so long as bile was being poured into it. Let me advise everyone dealing with abdominal operations to permit nothing vomited by the patient to be thrown away till he has seen it. This is a rule for my nurses, of which I permit no breach, for I often discover the most risky over-feeding of patients during their convalescence by zealous nurses or foolish friends. It is here that most especially is shown the great value of the treatment of such cases in a private hospital over the undisciplined conduct to which they may be subjected in a private house. The friends of patients are always the source of infinite danger and give far more trouble than the patient herself. Let me tell just one story about this out of dozens that I might give you.

CASE I.—A patient upon whom ovariectomy was performed was provided with a hearty meal of biscuits and port wine unknown to me and against my general instructions, for her misguided friends "were sure she was so weak and reduced that she never could pull through such cruel starvation." The result was of course basins full of stuff, at first a purple mixture of flour and grape juice, and then of pure bile in a stream almost continuous for twelve days. At the end of that time the wound—an exceptionally large one, for the tumour had been practically solid—was widely open, and I had fished out, bit by bit, sloughs of what seemed to be the whole of both recti muscles. When the vomiting stopped there was a sheet of peritoneum about 8 inches by 6, under which the intestines could be seen wriggling, and that was all that remained between the patient and another world. Fortunately the slender barrier held good, the wound slowly healed, and the patient ultimately recovered, but she has now an enormous intestinal protrusion.

I have therefore a belief that the starvation and withholding of fluid prevents the mechanical stasis of the circulation in the intestinal coats, which appears to me to be the initial stage of the fatal process of peritonitis, and this preventive measure I endeavour to assist by stimulating the peristaltic movement. If this mechanical stasis does occur it originates always in the transverse colon, and I cannot help seeing in this the agency of the great omentum, and therefore my first efforts are directed to this part of the canal in the form of stimulant enemata. I have tried a vast number of different kinds of enemata—some suggested by my own thought and others suggested by ingenious friends—but I have always gone back to soap and turpentine. It is the business of any nurse watching one of my abdominal sections to note every six hours a set of four conditions—the pulse, the temperature, the occurrence of distension, and the passage of flatus *per anum*. So soon as the latter is freely and naturally established our anxiety ends, though our watchfulness is unremitting. The want of such passage for twenty-four hours after an operation, especially if accompanied by the slightest suspicion of distension, is dealt with without fail by the nurse herself, on her own responsibility, by the administration of a turpentine enema, unless she has been especially warned not to give it for some special reason, such as a rent in the rectum. If the turpentine does not answer the nurse reports, and a mild saline purgative is ordered—generally a seidlitz powder—and this is repeated every four hours until it acts. If there should be any especial anxiety, such as the occurrence of vomiting or the presence of a clamp, 5 grains of calomel are given instead of the seidlitz powder.

If the distension increases we never rest till we have had the bowels moved, and then our anxiety is nearly always at an end and our efforts rewarded by recovery. But above all things there must be no time lost, and nobody who may be called into consultation by anxious friends must be permitted to write a prescription and try some favourite mixture. So

soon as this is threatened I repudiate all responsibility, and retire from the case.

There is nothing in all this that I have not published years ago, but I am bound to take some trouble to repeat it on a favourable opportunity such as this, because it has been grossly misrepresented and misunderstood. I have never said that the purgative treatment will cure peritonitis, for peritonitis, once it is completely established, is a practically incurable disease, and almost uniformly fatal. But when it is completely established I do not know. Of this I am certain, granting a large number of cases going on to peritonitis, after operations or from any other cause, if you subject the patients (not the peritonitis) to the purgative treatment, the number who will go on to incurable peritonitis will be absolutely fractional compared to what will be the result if they are left alone or submitted to any other treatment which has come under my notice.

When called in to a case of well-established peritonitis I always urge a trial of this treatment, because the stage may not have passed at which it may still be effective, but the chances are that it has. I generally find that the time has been lost, and that the irresistible tendency of our profession must be to treat the prominent symptom (pain) with the only drug on which implicit reliance can be placed (opium) has put the purgative treatment beyond the reach of trial.

TIME AS AN ELEMENT IN PROGNOSIS.

I have already said that time is the most important element in a case of peritonitis from another point of view, and to that I now purpose drawing your attention. Briefly, it is my opinion that the outcome of a case of peritonitis depends far less on the severity of the symptoms than on the time over which they run. Thus symptoms may be quite parallel in two cases, and yet one will die on the fifth day and another will go on to the twelfth and get well, the symptoms lasting all the time with almost initial severity. The same thing is known about tetanus. My experience of this peculiarity of peritonitis is that it is not limited to traumatic peritonitis, but is seen in every kind, including that which we know as idiopathic peritonitis. Let me give in very brief outline a few very noteworthy experiences I have had in this particular.

CASE II.—Many years ago a young domestic servant was sent down to me from a mansion in Cornwall with an ovarian tumour, and was sent most improperly without an instruction from her mistress that the children to whom she was acting as nurse had just recovered from scarlet fever. Five days after the operation I discovered that she was desquamating, and then unearthed a clear history. By the time the discovery was made I had performed eleven more abdominal sections, and had carried the infection to every one of them; the event occurred in the days when I used complete Listerian precautions, spray and everything else. Three of my nurses were also infected, but fortunately all the fifteen cases recovered, though one of the nurses was maimed for life by a stiff ankle-joint. Only one of the twelve abdominal sections displayed anything more than the ordinary symptoms of mild scarlet fever, but that exception was a most remarkable one. She was a congenital imbecile patient who was sent to me by Dr. Connor, of the Lancaster County Asylum, with the sanction of the Commissioners in Lunacy, for the removal of a large ovarian tumour. She rapidly developed all the signs of peritonitis, and on the fourth day had profuse and irrepressible bilious vomiting, so that I fully expected her death on the fifth day. But she did not die. The symptoms went on without any kind of remission, especially the bilious vomiting, so that soon the poor creature's head had to be bolstered over a slop pan, and an excruciated tract from the angle of the mouth down the chin and some way down the neck marked for two or three weeks the acridity and continuity of the bile stream. I went to her bedside three times daily for eight weary days, fully expecting that every visit would be my last to her. On the night of the twelfth day I said to the nurse I will not come in the morning, as the patient's hands were cold and her pulse imperceptible, and I felt perfectly convinced that she must die during the night. The nurse was a good Catholic, and the patient in her estimation, even as an imbecile, deserved consideration for another world. From her own *débutier*, and with fulness and devoutness of intention, the nurse performed that rite, necessary in her belief for eternal salvation. The vomiting immediately stopped, and next afternoon I found the patient actually taking food. She made an easy but somewhat protracted recovery.

In this case it would be an endless task to recite the remedial attempts which had been made. They were all futile. Enemata never extracted a cubic inch of flatus from the patient's intestine, and no kind of dietetic or therapeutic expedient could be effective owing to the persistent stream of bilious fluid ejected from the stomach. An occasional hypodermic injection of morphia eased the patient, but its depressing effect was so great and its benefit so temporary that even that was given up. What brought about the successful issue? I confess I do not know. Either it was the miraculous

effect of the religious rite, or it was lapse of time. But the cases that go on for so long as this nearly always get well. The fashionable sceptic and the scientific critic will, of course, say that this was not a case of peritonitis. But how could he get over such cases as the following, examples of which I can quote in considerable number:—

CASE III.—A. B., aged 17, placed under my care by her uncle, a well-known medical man, on account of a large tumour supposed to be ovarian, but which at the operation turned out to be a huge cystic tumour of the left kidney, which I removed by abdominal section, tying the pedicle, and closing the wound. The patient was all right when I went from home on the third day, leaving her in charge of a highly valued assistant, who knew my views and loyally acted up to them. High temperature and pulse, with severe distension, displayed themselves on the morning of the fifth day, but no vomiting, purgatives and turpentine enemata were persevered with, but without effect, and when I got back on the fifteenth day after the operation I found her pinched, wasted, with high temperature records and an enormously distended abdomen. But my accustomed touch at once discovered that the distension, though it had a tympanitic note on the surface, was mainly ascitic effusion, and therefore I immediately reopened the abdomen, giving vent to about 20 pints of the most stinking pus I ever encountered. I washed well out and drained, and she made a speedy and most satisfactory recovery. She is now a well grown and perfectly healthy woman.

Will the critics refuse to accept this as a case of acute traumatic peritonitis? If not, I have of course to give up the discussion, only asserting that in my opinion it was. Why did she get well? I cannot say unless I recognise the influence of time, for my experience is that if she had matured a tablespoonful of that pus in her peritoneal cavity on the third day after her operation she would have died on the fifth; and another conclusion in my mind, as deep-rooted as the alphabet of the language I speak, is that no germ theory will explain death in the one case and recovery in the other. Nothing I know will explain the difference in the results, of which time is the only factor at present discoverable by our limited vision.

CONCLUSION.

The practical outcome of my empirical experience is this, that the purgative treatment of peritonitis or threatening peritonitis, or of anything which seems to be likely to turn out peritonitis, if it be promptly brought to bear on the case, will gain the all-important time, and often it gains such time as will call into action that influence which will eventually turn the scale in the favour of the patient.

INJURY TO THE PANCREAS: A CAUSE OF EFFUSIONS INTO THE LESSER PERITONEAL CAVITY.

Read in the Section of Surgery at the Annual Meeting of the British Medical Association, held in Nottingham, July, 1892.

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In looking over recently recorded cases of pancreatic cysts which have been treated by surgical operation, three points have forced themselves upon my mind. (1) The large number which have followed abdominal injury; (2) the almost general absence from these reports of any reference to the condition of the lesser peritoneal cavity; and (3) the striking similarity between these cases, and the two cases of effusion into the lesser peritoneal sac which I am about to relate.

The main object of this paper is to call attention to the relationship which exists between injuries of the pancreas and accumulations of fluid in the lesser peritoneum, a relationship which, so far as I can ascertain, has hitherto attracted but little notice.

My attention was first called to the subject in the year 1878, when, on making a *post-mortem* examination, I was surprised to find the middle two-thirds of a gangrenous pancreas detached and free in a collection of fluid contained within the lesser peritoneum. About four years later I saw in the practice of a friend a similar case. I could find but scant reference of any kind at this time to affections either of the pancreas or the lesser peritoneum, and none whatever to the particular condition just referred to, but since then our knowledge of pancreatic disorders has been much added to, and gangrenous pancreatitis has become a well recognised, although rare, disorder. Injuries of the pancreas—uncomplicated by serious and usually fatal damage to other struc-

tures, like the liver, spleen, and stomach—are perhaps even rarer, but I have met with two cases of the kind.

CASE I.—W. G., aged 20, was admitted under my care into Queen's Hospital, Birmingham, on May 22nd, 1888. Shortly before admission he had been knocked down by a horse at a race meeting, and on recovering consciousness complained of being hurt in the abdomen. When brought into hospital he was much collapsed, had been sick, and was suffering severe pain in the upper part of the abdomen, but exhibited no external marks of injury. He vomited many times during the first two or three days, and in the vomit an occasional fine thread-like streak of blood was noticed. His temperature registered 100° on the day after admission, but soon became normal, and after the fifth or sixth day his symptoms had entirely gone, except for a little tenderness on pressure in the epigastric region. He left hospital at his own request on May 31st, and for a few days kept fairly well, but then began to suffer from attacks of epigastric pain, which lasted two or three days, and were sometimes accompanied by vomiting. These attacks occurred about once a week until July 27th, when, because of a more severe attack, he was admitted into the General Hospital, Birmingham, under my friend, Dr. Simon, who has kindly placed his notes of the case at my disposal. The lad had lost flesh, and was anæmic. His tongue was red and slightly coated with brownish fur. He had a fair appetite and no nausea or vomiting; the pain was not increased by food. The abdomen was tense and prominent over the epigastrium, and tender on pressure. Liver dullness normal, lower part of belly soft and free from tenderness. Pulse 100; urine 1010, alkaline, and contained phosphates, but no albumen. The pain ceased on the following day. On July 30th he had an attack of severe abdominal pain, which lasted ten hours, but on August 18th he was discharged, having made no further complaint. His temperature throughout had been subnormal, varying between 96.8° and 98.2°.

On August 29th he had another sudden and severe attack, with vomiting, a few blood streaks being again seen in the vomit, and at night he observed a swelling in the left hypochondrium. He was readmitted to the hospital, and it was noticed "that the pain was gnawing in character, and was much easier when he sat up in bed." A tumour could be seen and felt in the epigastrium and left hypochondrium, tense and rounded, reaching from the left costal arch down to the umbilicus and into the upper part of the left lumbar region. (See Fig. 1.) It was dull on

Fig. 1.

percussion and tender to pressure. Next day the tumour could not be defined, but there was increased resistance and tenderness in the umbilical region, and the painful symptoms had passed away. Two days later the pain returned; all food was withheld from the stomach, and he was nourished by peptonised enemata. Twenty-four hours later it was noted "a good night; pain gone; no tumour to be felt; stomach note over former site of tumour." Two days later he was free from pain, but the tumour could be again distinctly felt. A hypodermic needle was passed into it, and a dark-coloured fluid withdrawn. An aspirator needle was then inserted, and 29 ounces of fluid taken away. No chemical examination of this is recorded. On the following day he had more pain in the belly, and the sickness returned. Two days afterwards the tumour had filled again, and was tense and tender. The pain increased during the next twenty-four hours, and the vomiting became more frequent. On September 11th the abdomen was opened by an incision in the left hypochondrium. A cavity was found containing a further quantity of dark brown fluid. This cavity was washed out carefully, but the boy died exhausted a few hours subsequently. The temperature throughout had never been above 98.4°, and the morning preceding the operation it was 97.2°. A *post-mortem* examination was made next day by Dr. G. F. Crooke, from whose notes the following account is abstracted:

"On opening the abdomen, signs of recent peritonitis were found. Situated behind the stomach, and co-extensive with its posterior surface, was an enormous encysted hæmatoma, its lower edge reaching further than the stomach and to within 1½ inch of the transverse umbilical line. It was removable with the stomach, and was filled with 8 or 4 pints of dark blood containing old coagula; thus the comparatively empty and collapsed stomach appeared to rest posteriorly upon a large sac (the lesser peritoneal cavity), which was distended with altered blood. Regarding the structures entering into the composition of the sac walls, the folds of the lesser omentum were so altered that it was hardly possible to tell what they were, but it appeared as if the hæmorrhage might have occurred from some ruptured vein in the gastro-splenic omentum, found its way inwards, and gradually become encysted between the peritoneal coat of the stomach posteriorly and the peritoneal covering over the pancreas. No special examination of the pancreas for injury was made."

In connection with this case I wish to call attention to the following points: First, the injury to the belly and the sym-