may have fallen short, I can assure you that clinically they were very presentable pictures of three distinct gastric conditions—malignant tumour, gastric ulcer, and a subacute gastritis. I was fortunate in getting histories of infection in the majority of my cases—I say fortunate, as you all know the difficulty of obtaining a medical history of any kind from a native, to whom the acquisition of a venereal disease is a mere incident. From the histories and from a consideration of all the collateral circumstances in those from whom no history could be obtained, I was led to the conclusion that all of them were the result of the acquired disease. The youngest, and also the earliest after the date of infection, was a female, 28 years of age, with a clear history of infection six years previously.

The comparative rarity of the definite tumour formation may be judged when I say that in thirty-five years I have encountered it only seven times; all were males, and only one European. In three the symptoms of obstruction with dilatation of the stomach were very definite; in one, the European, there were signs of a partial obstruction, and in the remaining three there were no signs of obstruction whatever. In the European the swelling lay in the middle line, and yet it produced some obstruction as only fluids could pass. This can be explained only on the hypothesis that, by its size, it caused a distortion of the gastric planes and thus obstruction. In the other non-dilated cases the site of the tumour was to the right of the middle line, so that, of the seven cases, six were in the pyloric region.

I am unable to add anything to our knowledge of the pathology of these conditions as I have had no fatal case, but writers on this subject are fairly well agreed as to the changes to be found. These are two—syphilitic endarteritis and gummatous formations. Of the latter three forms are described—small discrete gummata scattered all over the mucous membrane, flat diffuse gummata, and gummatous tumour. I do not think there is much room for doubt that these tumour cases were of the latter variety. Although I have had no death I believe myself justified in stating that the course of these cases would differ little from that of the most acute type of malignant disease. In none, excepting the European, did the history of gastric trouble extend beyond six months. The condition of the dilated cases was grave enough to warrant a belief that they were within measurable distance of their end, and, what with the absolute anorexia, the abeyance of gastric function and the cachexia, I do not think that the course of the non-dilated cases would have been much longer than the dilated. The European dated the beginning of his symptoms back twelve months. Early diagnosis is therefore a desideratum, and to that end it would be a prudent measure, having diagnosed an organic lesion of the stomach, either to confirm or eliminate syphilis by a Wassermann or Noguchi test.

The only symptom which might give a pointer is the nature of the pain. While there do occur cases of malignant disease of the stomach which throughout their course are "silent," they are the exception. Pain in malignant disease is usually a prominent symptom, occurring erratically, radiating widely, always severe, often indeed agonizing, disappearing completely for a time. Pain in gastric syphilis is never at any time pronounced, but it is always there, dull and constant, sometimes better, sometimes worse, especially at night, and limited to a zone extending right round the body a little below the level of the diaphragm. There is of course always the possibility of such cases merging into malignancy, as old syphilitic lesions do in other parts of the body, and it would not surprise me if the case shown to-night should, in its half-treated condition, follow that course.

The second group of cases, those mimetic of gastric ulcer, are not as a rule difficult of diagnosis. Though vomiting is a symptom, the excessive vomiting in the illustrative case is unusual, and unless of spinal origin not easily explained. The symptoms common to all were thickening, sometimes almost amounting to tumour formation, in the epigastrium, tenderness on palpation, vomiting, and the presence of blood in the vomited matter. Taking the three cardinal symptoms of gastric ulcer, pain, vomiting, and haemorrhage, and discussing them seriatim, it is possible to go some distance towards establishing a diagnosis. Pain in gastric ulcer is brought on, or much aggravated by, eating, and is relieved to a corresponding extent by vomiting. It is usually located in one spot, a "point of pain," is intense, and there is generally a point of referred pain, usually in the back. In ulcerated gumma of the stomach, which I take to be the pathological condition, there is a constant substratum of pain, not much

aggravated by eating, but greatly so by the effort of vomiting, radiating laterally, and forming a girdle of pain which, as usual, is worse at night. Vomiting is common to both conditions.

Haemorrhage in gastric ulcer is usually copious, coffee-ground, or bright red, according to its time of retention. In gummatous ulcer the blood comes up only with food, is in small clots, and bright red only at the end of a severe effort of vomiting. There is a possibility of a true peptic ulcer developing in a stomach which is the site of syphilitic endarteritis.

The third class—those resembling subacute gastric catarrh—are more common than is generally assumed, at least among Asiatics, and they range from those having only subjective symptoms to those in whom the local condition verges on tumour formation.

Some assistance in diagnosis may be had from test meals, as all the cases in which it was given had a pronounced hypochlorhydria. They are too few to generalize upon, but it is significant that the condition was the same in all.

To summarize briefly, there are two symptoms and three signs. The symptoms are, first, anorexia—I know of no condition in which it is so absolute, excepting in some early cases of malignant disease—and second, the nature of the pain. The signs are, first, enlargement of the liver (and occasionally also of the spleen), with smooth outline, present in three-fourths of all cases; secondly, rapid development of cachexia; and thirdly, stigmata of syphilis.

The real test is the empiric, the therapeutic, and that may be applied both from its negative and positive aspects. It seems to be immaterial what care is expended or with what ingenuity drugs and diets are tried and changed, the result is *nil*. On the other hand, when specific treatment is begun, the change is immediate, often dramatic. In the pre-salvarsan days, from which period the most of my cases are taken, the prescription of choice was calomel $\frac{1}{2}$ grain, with sodium bicarbonate 10 grains, and heavy magnesium carbonate 10 grains, given thrice daily with hot water on an empty stomach, and continued until signs of mercurial saturation appeared. After the first day or two, as soon as mercury began to be absorbed, improvement began and was generally very rapid. There was always the possibility that local action shared in bringing about the improvement, but now that is eliminated and negated, as intravenous injection of novarsenobenzol acts on the stomach condition immediately.

SOME PRINCIPLES OF AFTER-TREATMENT IN ACUTE ABDOMINAL DISEASE.*

BY

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THIS paper is founded on the results of trying the more modern methods of after-treatment in a series of just under 300 cases of acute abdominal disorders, on which I have operated in the last two years. During the same period I had the opportunity of observing many cases operated on by others, and, since they were seen, it is impossible to avoid taking them into consideration.

An attempt has been made to follow the results of *post-mortem* examinations and the appearances observed at second operations, and to bring these findings to bear on technique and on after-treatment.

In surgery some older methods are being abandoned for those we believe to be better; but in nearly all conditions it is being increasingly recognized that the operation is but a part of the treatment. It is also becoming manifest that, apart from the particular manipulation called the operation, the great therapeutic agent is rest. It is astounding to realize how little we have advanced in basic principles of treatment since the time of Hilton. With all our elaboration of technique, the almost universal objective of surgery is to provide the patient with an easier task in overcoming the damaging agent.

At the conclusion of an emergency laparotomy the patient is left with a double task. He has to repair the ravages of his disease, and to overcome those portions of the damaging agent which the surgeon has left behind. He has also to repair the injuries inflicted by the surgeon, who, unlike the

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cells and fluids of the body, cannot accomplish his task without inflicting damage.

The dual repair involves many processes—excretion of toxic substances, manufacture of antibodies, multiplication and migration of cells, besides the patient's essential metabolism. Practically the only way in which we can hope to influence any of these processes is by securing for the patient physiological rest. The medical attendant, therefore, when all goes well, holds a purely watching brief. He must be prepared to act solely as a trained observer. He must be familiar with the living pathology of acute abdominal disorders, and must have a constant mental picture of what is going on in "this abdomen at this time." He must be able to diagnose at the earliest possible moment any variation from the normal convalescence. His is the responsible—and often thankless—task of deciding when a surgeon is to be called in again. He has the difficult task of doing nothing amidst the sometimes silent opposition of nurses and relatives. The treatment after operation for an acute abdominal condition is, therefore, rest; but there are certain indications for a departure from this waiting attitude.

Shock and Toxaemia.

Without discussing the genesis of shock in detail, it may safely be said that it is produced by one or more of the following factors, and that, little as we knew about it seven years ago, further knowledge makes its explanation more difficult:

1. Trauma and exposure, especially of abdominal viscera.
2. Mental and emotional impressions; pain or unfelt painful stimuli.
3. Loss of fluid from the body by haemorrhage, vomiting, or diarrhoea.
4. Loss of heat.
5. The absorption of certain poisonous substances, toxins—bacterial and non-bacterial—for example, histamine.

Its salient features are mental torpor, "pinched" facies, cold extremities, usually, but not invariably, a rapid pulse rate, and quite late a falling blood pressure. Shock is usually most severe some hours after the damage is accomplished. A slow pulse is not unusual in a patient otherwise in a state of shock. It is usual to find a patient whose peptic ulcer has perforated with a slow, full pulse, 60 to 70, yet he is cold, clammy, and evidently suffering from severe shock. One would feel more confidence in making the diagnosis from the feel and appearance than from a consideration of the chart. Patients whose blood pressure falls steadily during an operation seem to have but small chance of surviving.

Treatment.

In the absence of exact knowledge of its pathology we are compelled to treat symptoms. Thanks to Crile, we know how we may prevent severe shock. These methods are based on the elimination of noxious stimuli, maintenance of body heat, and replacement of lost fluid. These, too, are our sheet anchors after shock has been produced. What the patient requires is: *Rest*, mental, physical, and physiological. We can give him this with an adequate dose of morphine. Do not let us withhold this drug, and if it is to be of any use it must be given in full dose. Let the patient's rest and the state of his pupils be the guide to dosage. *Warmth.* The best way is by means of the electric hot-air bath; where this is not available, hot blankets and bottles. The supply of heat should be continued up to but not quite as far as the sweating point. *Fluid.* This serves three purposes: (1) It maintains the volume of the blood; (2) it can supply heat; (3) it diminishes thirst and provides a vehicle for the excretion of toxins. We can give whole blood if a suitable donor is available; it must be given intravenously, and the citrate method or that by means of Kimpton's tube are the most convenient methods. We may give gum solutions (Bayliss), which should be given intravenously. The effect of these is more sustained than the next. We can give various isotonic solutions, of which I think glucose (1 oz. to a pint) is the most satisfactory. Intravenous, subcutaneous, rectal and oral methods are available. If the patient can take fluid by the mouth—and here lies the great virtue of gas and oxygen anaesthesia—it seems infinitely more pleasant and just as satisfactory to give fluid in that way.

The Question of Stimulation.

Drugs such as strychnine, digitalin, ergot, adrenaline, and pituitary extract may all have a pressor effect on the vasomotor system. All available evidence goes to show that this

effect is transitory, and is succeeded by a fall to a lower blood pressure than existed before the administration. It is therefore inadvisable to use them in the treatment of shock.

Posture.

Raising the foot of the bed and bandaging the extremities should aid the circulation of blood in the vital centres. Practically the position may do good in severe shock and can do no harm. It is, however, very rare for a patient to need more than rest as supplied by morphine, and warmth as supplied by hot bottles.

Toxaemia.

In the absence of exact knowledge of the chemistry of the circulating toxins we know of no drug or combination of drugs which can deal with the complex chemical poisons we know toxins to be. It is the patient who must elaborate the drugs which are required. We think that it is the spleen, the liver, and the endocrine system which are concerned with antitoxin formation; but we cannot give any orders to these viscera. What we can do is purely passive. We can supply rest. We can, by reducing protein intake, reduce the exogenous urea formation—again giving rest. We can reduce the work thrown on the excretory organs. We can supply a vehicle for excretion. The indications are therefore to abolish protein intake, to allow rest and sleep, and to give fluid.

General Management of the Case.

All the indications we have so far examined demand rest. Rest and sleep are essentials, and for their attainment comfort is necessary. In many cases we cannot secure comfort without giving morphine, and the indications and contraindications for its use must be discussed. Morphine adds a burden to the excretory organs and lowers their efficiency, it masks symptoms and signs which should be apparent, it makes for diminished intestinal movement, and is said to increase intestinal distension. On the other hand, morphine is very often the only drug which will secure rest and sleep, and the patient who does not rest will very likely die.

The first objection is real and must be faced. It is a disadvantage which I believe is outweighed by its advantages. It is a disadvantage that might be equally urged against the use of anaesthetics. The second objection is also true, and offers the sole contraindication. We must not give morphine while trying to decide for or against a second operation, but the decision should be made quickly. The third objection contains a truth and also a fallacy; morphine diminishes intestinal movements, but I hope to show that this is desirable. It is in cases which present complications, such as obstruction, adhesions, peritonitis, and the like, that the repeated administration of morphine is called for. These conditions will produce distension without morphine. In a straightforward case morphine is not required after the first twenty-four hours. In the first hours a night's sleep works a startling change. Morphine is, nevertheless, indicated if the patient be restless and in pain after twenty-four hours so long as we are confident that further operation is not indicated.

The next thing that makes for comfort is the unrestricted intake of fluid by the mouth. Quantity need never be restricted, but it is wise to let each drink be small until we are sure that vomiting is not being induced by it.

The avoidance of all unnecessary interference with the patient makes for comfort. Enemas, feeding, and injections are particularly disturbing if repeated. Sometimes patients are almost treated to death. As soon as possible let the patient be nursed in the position of greatest comfort. There should be as few dressings as possible, tubes should be removed early, and syringing and all fiddling with the wound avoided. After an operation for appendicitis patients do best in Fowler's position, and on the right side, but they can safely be got out of this somewhat uncomfortable position after forty-eight hours. Good nursing as regards washing, oral cleanliness, and arrangement of bedding and bed rests, adds enormously to comfort. When there is vomiting after the first twenty-four hours the stomach tube is the best way of stopping it; it is uncomfortable, and, if the patient will not co-operate, painful; but once a patient has experienced the extraordinary relief of having an empty stomach he will desire its repetition. It is quite frequently a life-saving measure.

Dressings.

A clean case wants dressing when the stitches come out, or when for any reason it is desirable to inspect the abdomen.

A suppurating case wants dressing only as often as pus soaks through to the outside of the dressing. Fomentations should be avoided as far as possible. They are apt not to be sterile, and are indicated only where there is a spreading cellulitis of the abdominal wall which we desire to localize as an abscess which can be drained.

Drainage.

In this connexion facts oppose much that is traditional. It is possible to drain the whole peritoneum for but a short time—about twenty-four hours. Experimental work and numerous necropsies show that any foreign body, such as a drainage tube, is isolated by plastic lymph adhesions within a very few hours. This statement is true for a dying individual; it is likely to be even more true for one who is going to survive his infection.

The foreign body, having been shut off by the above-mentioned layer of lymph, continues to drain the sinus it has produced for itself, and to drain this sinus only (Fig. 1). The discharge soon ceases once the offending tube is removed. Sometimes in removing the tube a portion of the barrier is torn; a gush of pus is the result (Fig. 1). The drainage tube is therefore useless after the first twenty-four hours (approximately). It is also dangerous; I refer only to that portion which projects into the peritoneum; the barrier of lymph is invaded by cells which convert it into granulation tissue. The granulation tissue becomes young fibrous tissue; eventually it contracts and becomes mature fibrous tissue. I have thus traced the genealogy of a fibrous band of adhesions. The longer the tube is left, the more complete is the evolution towards fibrous tissue and the denser the resulting band. In peritonitis the evolution towards true fibrous tissue (permanent) seems to have three common sites where it progresses to completion. These are: the vicinity of drainage tubes, of a residual abscess, or of traumatism or blood clot—for example, scar of uterine stump. I will quote three illustrative cases.

CASE I: Perforated Duodenal Ulcer.—S. K., male, 51. Operation twelve hours after perforation. Peritoneum flooded with bile-stained glairy fluid. Perforation closed, and a posterior no-loop gastro-enterostomy was done. Drainage tube through a stab wound above the pubes. The operation took thirty-five minutes, and he left the theatre in good condition. The tube was left in five days. Ten days after operation he developed obstruction. At his second laparotomy the peritoneum was clear of adhesions except in the track of the drainage tube. Here there was a dense, well defined band adherent to and obstructing the small gut. This was divided, and the wound closed. He died twenty-four hours later, and necropsy was refused.

CASE II: Perforated Duodenal Ulcer.—F. W. B., male, 40. Operation four days after perforation, which was minute "leaking." There was marked lymph formation over the whole of the right flank. Perforation closed, and a posterior gastro-enterostomy done. Drained, two tubes. He made a good recovery, and was discharged from hospital. Three months later he was readmitted with intestinal obstruction of four days' standing. At operation there were dense adhesions both where the tubes had been and to the abdominal scar, none elsewhere. He died the same night. None of my other cases of perforated peptic ulcers (16) have shown evidence of the formation of adhesions. They have either been closed without drainage or the tubes have been removed within forty-eight hours.

CASE III: Acute Appendicitis, Diffuse Peritonitis; Operation, Appendicectomy.—N. B. S., female, 5. Drainage through a supra-pubic stab. Tube out in forty-eight hours. Tenth day a second laparotomy on account of intestinal obstruction. Small gut greatly distended and adherent to a small residual abscess on the upper surface of the sigmoid mesentery—remote from the situation of the drainage tube. The rest of the peritoneum was clear of adhesions as far as was seen. Gut was freed and oversewn and the abscess drained for forty-eight hours. She made an uninterrupted recovery.

These three cases illustrate what I have found to be the common sites at which dense adhesions are liable to form. They point strongly to the danger of prolonged drainage.

The essential difference between an abscess cavity in the peritoneal cavity and a generalized peritonitis is that the latter has the power of isolating a foreign body by adhesions. An abscess cavity is lined by granulation tissue which has not got this power. The whole peritoneum, or a really large part

of it, cannot become an abscess cavity; the patient dies long before this point is reached.

The whole matter of length of drainage comes therefore to the question how long an inflamed peritoneum can delay its power of isolating a foreign body. The healthy peritoneum isolates the least irritating of foreign bodies in about twelve hours or less. I have never seen a diseased peritoneum which had not done so in twenty-four hours, and I find it very hard to believe that it can be delayed beyond about forty-eight hours. If you believe that this power can be delayed three or four days or longer, then for just so long is prolonged drainage defensible. We really want a pathologist with vast experience to tell us, first, what is the longest time he has seen a tube left in a living patient without its having been shut off, and secondly, what is the average time in cases of diffuse peritonitis. The latter figure should form our routine, the former our maximum, only to be used in desperate cases. To drain longer would be unjustifiable. When there is a localized abscess lined by granulation tissue the case is different; here the tube should be shortened gradually as the abscess collapses. In diffuse peritonitis it should be cut to a length, which is that of the thickness of the abdominal wall, after forty-eight hours.

The next point to be considered turns on the frequency of

residual abscesses where drainage is curtailed as to time. It is difficult to determine from so small a number of cases. My own impression from my own and from reading the notes of and seeing other cases treated by more prolonged drainage, is that they are not more frequent; drainage, however prolonged, does not eliminate the risk of residual abscess. The residual abscess is so often remote from where the tube has been that it is difficult to see how any drainage short of "kippering" the patient could eliminate it. The usual fate of a residual abscess is to resolve without interference by the surgeon. The history of appendix abscess as well as recent experience and present doctrine all combine to prove this. It was formerly the fashion to wait until an appendix abscess resolved. Quite 60 per cent. did resolve. After a period of operating on everything

it is now recognized almost universally as good practice to wait under certain defined conditions—namely, a history of longer than four days, an improving patient, a palpable decreasing tumour, and when immediate operation can be performed should the improvement not be maintained. This is most emphatically not a doctrine of waiting in the early stages of an attack of appendicitis. Most of the residual abscesses which occur after operation will disappear if the patient is watched under conditions of semi-starvation and rigid non-interference by purges, etc.; but if increasing or if reasonable suspicion exists that it is causing the formation of obstructing bands, it should be appropriately drained.

The foregoing remarks apply with equal force to the maintenance of the Fowler position, except that here it is only a comfortless position and not a dangerous complication which is at stake. Any peritoneal exudate which has not gravitated to the pelvis in forty-eight hours must be dealt with *in situ* by the peritoneum. Fortunately for the surgeon, in the majority of cases the peritoneum is capable of dealing effectively with what he leaves behind.

Acute Dilatation of Stomach.

The frequency of this condition is difficult to determine. Cases which reach the stage of exhibiting a complete clinical picture usually die whatever is done for them. On the other hand, we are hardly justified in claiming that every case whose vomiting is cured by gastric lavage is a case of acute dilatation. One is almost justified in saying that the surgeon who never sees a case treats it most efficiently. Pathologically it is a paralytic dilatation of the stomach most commonly occurring after abdominal operations, but sometimes when there is no other disease in the abdomen. Whether a mechanical factor exists after the dilatation has commenced

FIG. 1.—a, Tube; b, aponeurosis; c, muscle; d, peritoneum; e, gut in section; f, barrier of lymph separating abscess from tube; g, abscess; h, granulations adherent to i, damaged gut liable to be obstructed as h contracts.

is a matter of little but academic interest. Clinically, there are repeated large vomits and belchings of wind, relieved temporarily by the passage of a stomach tube, marked distension of the upper abdomen, chiefly to the left of the mid line, and great prostration.

Treatment.—The important thing is that further operation on the stomach is almost uniformly fatal. Repeated gastric lavage, combined with the prone position over a pillow with the head low, saves a few cases. If anaesthetic vomiting persists for more than twenty-four hours, there is a grave suspicion that this condition is commencing, and if there is distension, eructations, and a rapid pulse, no time should be wasted in conjecture. If a stomach tube be passed it will often be found that a patient who is apparently emptying his own stomach every few minutes still has a reserve of 30 to 40 oz. of filthy fluid in it. The stomach should be gently washed out again and again with sodium bicarbonate (60 grains to the pint) through a large funnel, until the wash is clear. If the patient is greatly relieved this should be repeated every four or six hours, until the stomach contents fall to less than 4 oz. on passing the tube. All feeding by the mouth should be stopped and rectal or subcutaneous salines given. The passage of a stomach tube sounds and looks a brutal procedure; but the relief is so great that patients will often demand its repetition. Provided that the tube is soft its passage is safe where a gastro-enterostomy has been done—far safer, in fact, than leaving a dilated stomach.

Ileus Paralyticus.

This is a paralysis of the motor mechanism of the gut which may be due to (a) toxæmia, (b) over distension, (c) trauma and exposure. Most textbook descriptions advise calomel, eserine, pituitrin and saline purges, etc.; croton oil is not often advocated. Ileostomy and caecostomy are also advised.

First, ileus, apart from peritonitis, is rarely found either at second operations or *post mortem*. This means either that it is a rare condition, or that cases of it usually recover; both statements are probably true. The common finding, both at operation and *post mortem*, is a widespread plastic peritonitis,

FIG. 2.—A, B, Line of mesentery; x, portion of abdomen in which upper third of small intestine usually lies; y, portion where middle third usually lies; z, portion where lower third usually lies.

which is a mechanical bar to peristalsis; not that there is localized obstruction complete at any one point and to be relieved by operation, but, as it were, a brake applied over a considerable length of intestine. Cases diagnosed as ileus are therefore more truly described as post-operative peritonitis. All the cases of paralytic distension I have had the opportunity of accurately observing have been in association with peritonitis, usually primary, sometimes secondary to obstruction. When the condition of the small gut is observed during operations for appendicitis, it is in the immediate vicinity of the iliac fossa that the gut is most distended. From a consideration of the usual lie of the gut in the abdomen this means the lower third of the small intestine (Fig. 2). It is justifiable to assume that the gut dilates because it is inflamed, and that the dilated condition is the resting condition in response to inflammation. The pathological picture with which we have to deal in an ordinary case of appendicitis is therefore that the lowest coils of the ileum are inflamed and distended, and the gut progressively approaches the normal as the stomach is approached (Fig. 3). It is rational to attempt to empty this, but normally, and in fact nearly always, it empties itself. The precise action of the various methods we have at our disposal shows that the difficulties are greater than in dilated stomach.

1. *Direct Emptying of the Dilated Gut by means of a Tube.*—This may be done at the primary operation, but patients recover without this drastic measure too often to make it part of the routine in operations for severe appendicitis. A drainage operation may be performed after the condition has been diagnosed; caecostomy, ileostomy, entero-anastomosis, all have their advocates. My own small experience of these operations is not satisfactory (two cases with two deaths after ileostomy). Theoretically, caecostomy drains the gut below an obstruction. Ileostomy is in theory sound; perhaps I have been unfortunate, but I have never

seen it save a life, and I have seen absolute rest save several lives.

2. *Measures which Depend on Stimulating the Gut.*—It must be remembered (Fig. 3) that the paralysed gut is already receiving its normal stimulus—distension—in excess and is not responding. All the measures which stimulate the gut—calomel, salines, castor oil, eserine, pituitary—are bound to act most effectively on the normal upper coils of small intestine. Whether they have any effect on the paralysed gut below is very questionable. Their effect will be to force more fluid into the distended cesspool near the ileo-caecal valve. Those who have used Moynihan's tube will know how impossible it is for force from above to empty coils of distended gut. I therefore believe that any stimulation can only add to the difficulties near the ileo-caecal valve, and until the passage of flatus indicates that the tone of the small intestine is recovering all purgation is absolutely contra-indicated. The best way of restoring the tone of the gut is to give it rest by administering morphine and withholding food—the great initiator of peristalsis—until flatus is passed.

Enemas.—An enema can reach the lowest coils of the small gut, and a single effective enema may do good. If it be retained, a second or third can only add to the patient's discomfort. The bowels act because the patient is getting well. He does not get well because his bowels act. If his bowels do not act for a week and he is comfortable there is no occasion to worry. What generally happens when a patient is left alone is that he passes flatus spontaneously about the second or third day. On the fourth or fifth he has a natural call to stool and passes a profuse liquid stool; after that he can have food and a purgative. If he is distended, the effect of a single small turpentine enema may be tried; it may aid him to pass flatus or a motion. Should it be retained or returned unchanged, this must be accepted as an indication that the muscle of the small gut is not yet ready to pass on its contents; he should be left entirely alone except to give morphine if he be restless. The experiment may be repeated after twenty-four hours. Lockhart-Mummery has shown that the so-called high rectal tube, unless passed with the aid of a sigmoidoscope, coils itself up in the rectal ampulla. The cautious use of pituitary extract seems to help a little, but it should never be given in repeated doses, and it acts probably only in the cases that would have recovered without it. The important part of the treatment is to leave the patient alone, give morphine when it is called for, and restrict food without cutting down fluid.

Intestinal Obstruction.

Sometimes it is difficult to decide whether a patient is suffering from paralytic distension due to peritonitis, or from intestinal obstruction due to a band. Organization and contraction of adherent bands of lymph must take place before they can distort the intestine sufficiently to produce obstruction, and this takes six to ten days. This time is an important factor in making a diagnosis.

The cases which show evidence of ileus paralyticus are particularly prone to develop obstruction about this time. The reasons are, I think, that the cause, peritonitis, is present, that it is easy to leave an acute kink in gut which is distended, and there is no peristalsis to straighten out any such kink. It is well to be on the watch in such cases, and to waste no time in exploring again where the slightest confirmatory physical sign exists.

FIG. 3.—a, Empty, healthy small gut, readily excitable by food or purgatives; b, distended, inflamed, kinked and inexcitable gut; c, appendix—commonest primary cause; d, fluid faeces—highly toxic if under pressure; left alone, usually no evidence of toxæmia.

Patients who have a mechanical obstruction nearing completion suffer from attacks of spasmodic colicky pain with short intervals of relief. If this fits in with the time factor the presence of obstruction should be suspected. Visible coils of gut are almost diagnostic of obstruction, and if peristalsis is either visible or audible the diagnosis is no longer in doubt. Vomiting is a late sign of obstruction. It means that the distension has reached the upper coils of gut. It is matter for congratulation if the diagnosis is made and the second operation performed before the patient has vomited.

Lastly, though the results of an enema and the passage of flatus are strong negative evidence that obstruction is not complete, they are not absolutely so. The worst post-appendix obstruction I ever operated on had had a "good result" that morning. In this case the small gut was so distended that it blew out and burst like a bicycle tyre when the abdomen was opened.

Post-operative Peritoneal Infection.

Repeated dark vomits, distension, hiccough, and marked toxæmia, are evidence that the patient, as a whole, and his peritoneum in particular, are failing to cope with the virulence or numbers of the organism. Further operation holds out little prospect of doing any good. There is no one collection of pus that can be effectively drained. The gut is friable and will not bear the gentlest handling, and stitches will not hold if it is torn. The only chance for such patients, and it is a slender one, lies in absolute rest, morphine, and rectal or subcutaneous salines with glucose.

Remittent pyrexia should lead to examination of the abdomen, digital examination of the rectum, and examination of the bases of the lungs. If a residual abscess is diagnosed it is well to wait a little and watch progress unless the patient is going downhill. The majority get smaller and finally resolve. If the abscess gets bigger or the patient deteriorates it must be drained in the appropriate manner. It is best not to await rupture into the rectum or vagina. Such cases do better if deliberately and adequately drained.

A CASE OF PRIMARY CARCINOMA OF THE LUNG.

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THIS case was under continuous observation by one of us in the wards of Addenbrooke's Hospital for four months before death, and in the earlier stages simulated tuberculosis with somewhat interesting and rapid alterations in the physical signs. The correct diagnosis was only reached when definite enlargement of glands above the left clavicle was found to have taken place. Owing to the length of time the case was under observation it seemed to us to be worth putting on record.

Primary carcinoma of the lung is an uncommon disease. Hamman¹ states that 105 cases occurred in 46,169 autopsies, a percentage of 0.2; figures given by Ewing² show that 1.0 per cent. of primary cancers occur in the lung.

Primary growths are classified in three groups according to their origin: (1) From the bronchial epithelium; (2) from the bronchial mucous glands; (3) from the lung substance. The type of tumour found in group (1) seems to be localized to the larger bronchi and not to spread diffusely, to break down easily and form bronchiectatic cavities, being histologically essentially columnar-celled. Tumours of type (2) are characterized by abundant mucous secretion derived from the special type of cell of which the growth is composed. They also tend to be localized round the larger bronchi. The third group comprises the diffuse cancers with widespread destruction of lung tissue by rapidly spreading infiltration. The type of cell found in these varies, even in different parts of the same tumour, from columnar to flattened epithelium-like cells. The greater mass of cells are usually cubical in shape. The point of origin of tumours in the third group is difficult to determine, as their growth is rapid and diffuse. The cubical type of cell points to an origin either from the bronchioles or from the lining cells of the alveoli, whose shape often becomes cubical in collapsed lung. The matter is, however, of academic importance. Our case falls within the third group, the tumour being diffuse and the preponderating cells being cubical in type.

Clinically, carcinoma of the lung presents itself as a chronic pulmonary inflammatory disease, as a chronic pleurisy with effusion, or as a local patch of permanent consolidation; the usual diagnosis is pulmonary tuberculosis, fibroid phthisis, or unresolved pneumonia. The symptoms which suggest growth are rapid loss of weight and strength, pain, dyspnoea, and peculiarities in the physical signs, which are frequently unilateral. Pain is by no means always present, but severe continuous pain in the absence of pleurisy, or a constant boring pain with indefinite or unusual physical signs, should arouse suspicion. Fever, though always present in some degree, is usually much less than is found in tuberculosis rapidly advancing; the temperature may not rise above normal for days or even weeks. Sputum may be absent for some time, but in the later stages is frequently abundant; it is usually mixed with blood, and has been likened to currant jelly. Persistence of bloody sputum for weeks and months is very suggestive. The pleura is so commonly involved that pleural effusion is one of the chief signs of the condition; it is often smaller in amount than the physical signs would lead one to anticipate, and it is frequently tinged with blood; but the commonest cause of blood-stained pleural effusion is not malignant disease, it is tuberculosis (Horder³). Pneumothorax is a not uncommon occurrence in the course of the disease. Most of these points are illustrated in our case.

A. T. R., aged 27, a french polisher, was admitted to Addenbrooke's Hospital, Cambridge, on December 11th, 1920. He complained that for the past three weeks he had been troubled by shortness of breath, cough, and blood-spitting. He had previously had good health; he had, however, been discharged from the army in March, 1918, for deafness following shell concussion; for this disability he was pensioned. At the time of his discharge he had a cough, which disappeared after treatment for a few weeks in hospital.

He was a poorly nourished man, weight 7 st. 9 lb., a little dyspnoeic, not cyanosed. The physical signs were those of pleurisy, with a little effusion and patchy consolidation of the lower lobe of the right lung; the condition was thought to be tuberculous. During the first fortnight in hospital there was some loss in weight, a daily evening rise of temperature to just under 100°, and an increasing pulse rate. He had a little cough, with the daily expectoration of thin blood-stained sputum in which tubercle bacilli could not be found. Towards the end of December the patient had lost 6 lb. in weight, the evening temperature was rising to just over 100°, the pulse rate still increasing and the physical signs extending.

He was now placed on a balcony under open-air conditions, and at the end of three weeks had improved; he had regained 6 lb. in weight, there was little cough or sputum, though this was still blood-tinged; the evening temperature was not over 99° F. for fourteen days. The physical signs were much the same in the right lung; there were only occasional rhonchi in the left.

On January 25th, 1921, well marked signs of pneumothorax were observed; there was no pain or distress and the onset had evidently been insidious. A skiagram showed a right hydro-pneumothorax, and that the heart and aorta were displaced towards the right. A few days later blood-stained fluid to the amount of 4 c.cm. was withdrawn from the right thorax; the fluid was sterile and contained polymorphonuclear and mononuclear cells in equal quantity. In the middle of February the patient, who had become much emaciated, complained of constant gnawing pain in the right hip and thigh; in spite of treatment this continued almost without remission to the end.

The pneumothorax slowly disappeared, leaving the whole of the right side of the chest flat and immobile, dull to percussion, with very feeble breath sounds; the heart was displaced towards the right. Early in March enlargement of the left cervical glands was noted; they were soft, painless, and not tender; they rapidly increased in size, and those on the right side became palpable and visible. Towards the end of the month hoarseness, stridor and oedema of the face commenced and the superficial veins of the upper thorax became engorged. These signs of obstruction increased, and the patient died on April 15th, 1921. During the last eight weeks the temperature was normal or subnormal.

At the necropsy the whole of the right lung was found to be tightly adherent to the thoracic wall; the lobes were indistinguishable, almost the whole lung being involved in a hard white fibrous growth, from the cut surface of which thin white material could be easily scraped. The growth was most dense at the hilum and was found to involve the wall of the bronchus almost up to the bifurcation. The only portion of the lung comparatively normal was the extreme lateral part, which was collapsed. The left lung was entirely free from growth, and was emphysematous and congested. The left pleural cavity contained about a pint of milky fluid, chylous in nature and probably due to involvement of the thoracic duct; the pleural walls were in a condition of gelatinous oedema. Bronchial and tracheal glands were involved on both sides, forming a continuous mass with the greatly enlarged glands in the neck. The lumen of the superior vena cava was much narrowed by constriction. On the upper surface of the liver were four small nodules of growth; two of these were half an inch across and two less. The posterior abdominal glands were enlarged, but none were more than half an inch in diameter; on section they showed a similar appearance to that of the main growth.

Microscopically the lung substance is replaced by a mass of cubical cells which, though in most places irregularly arranged,