A Clinical Lecture ON DUODENAL ULCER.

DELIVERED AT THE LONDON SCHOOL OF
CLINICAL MEDICINE.

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So much attention has been directed recently to ulcerative conditions of the duodenum that the opportunity seems favourable for discussing with you a disease which has hitherto somewhat eluded accurate clinical observation, because its symptomatology has been but imperfectly described.

Modern surgery has now introduced methods of treatment, the advocacy of which makes the early recognition of duodenal ulceration more than ever a question of primary importance. Tuberculous and cancerous ulcers of the duodenum are not included in the following summary, which applies only to ulcers of a simple or non-specific type. Duodenal ulcer occurs more frequently in men than in women, and, though it may be met with at any age, it is most common at or about middle life.

In most instances the ulcer is situated in the first part of the duodenum, above the entry of the common bile duct and close to the pyloric ring. It is usually circular in shape, but may be oval or elliptical, and has a punched-out appearance, with inflammatory changes at the borders very similar to what we find in the more common gastric cases. From the situation of the ulcer at that portion of the duodenum between the pylorus and the bile-duct papilla, which is the only part of the intestinal canal subjected to contact with the acid gastric contents before they are mixed with and neutralized by the secretions of the liver and pancreas, it seems not unlikely that the projection of this acid fluid against a vulnerable spot may play an important part in causation. Bacterial infection probably also enters largely into the etiology.

Duodenal ulcer may be met with either in an acute or in a chronic variety, but it must be remembered that, though acute conditions such as haemorrhage or perforation may be the first definite manifestations of the disorder, yet the ulcer may have existed in a chronic form for a long time previously without giving rise to any definite symptoms. It has been estimated that about one-fifth of all the cases observed are latent over a longer or shorter time, sometimes many years, before they produce signs and symptoms sufficiently characteristic to admit of their definite diagnosis.

The leading and characteristic features of duodenal ulcer are deep-seated and localized pain in the right hypochondrium; dyspeptic symptoms occurring several hours after a meal and relieved by taking more food; and melaena or haematemesis. The pain is of varying intensity, and is situated to the right of the ensiform cartilage about the level of the tip of the eighth rib, whence it frequently radiates through to the back and reveals itself by a tender spot to the right of the last dorsal vertebra. On deep pressure the right rectus muscle stands out with more or less firm resistance. This local tenderness, accompanied by rigidity of the rectus muscle, is so striking that, without its presence, the diagnosis of duodenal ulcer must always be doubtful. The subjective pain, which is of a burning, gnawing character, comes on from three to four hours after a meal, is usually associated with acid eructations, and is always relieved by the ingestion of food. The pain gradually increases until it reaches a climax and is accompanied by a sense of distension in the stomach, which is temporarily relieved by eructations of gas. It comes on earlier after a light liquid meal than after one that is more substantial. It not uncommonly wakes the patient out of his sleep about 2 or 3 o'clock in the morning. The descriptive title of "hunger pain" has been suggested because it is usually first complained of when the patient is beginning to feel hungry. Its commencement is often heralded by a copious secretion of saliva. The appetite is generally excellent. Vomiting is the exception rather than the rule, and, when it occurs, fails to relieve the pain so completely as it does in gastric ulcer. The general health

is not seriously interfered with, and the disorder is subject to long remissions, during which the patient is both subjectively and objectively apparently perfectly well. Melaena, when it occurs, may be so slight as to be unperceived, or it may occur profusely once only after a sudden feeling of faintness accompanied by colicky pain. In some instances a copious haemorrhage from the bowel is the first intimation of serious illness, but as this occurrence may result from many other affections besides duodenal ulcer, an accurate diagnosis can be arrived at only by a process of elimination of other possible explanations. In the most serious group of cases perforation occurs as the first event; it reveals itself in the usual way by sudden acute pain, which is often referred to the right iliac fossa, and is quickly followed by rigid abdominal muscles, vomiting, small rapid pulse, subnormal temperature, sweating, and all the other well-known evidences of collapse.

A duodenal ulcer may continue over a very long period of time and be so obscure that it is unsuspected until narrowing of the duodenum from cicatricial changes produces a train of developments, including dilatation of the stomach, similar to those met with in stenosis of the pyloric orifice, from which they may be indistinguishable. Or, if the ulcer happens to be situated over the orifice of the common bile duct, its cicatrization may cause obstruction to the discharge of bile into the bowel and so be the cause of a persistent jaundice similar to that met with in cases of impacted gall stone. In cases of long duration, in which dyspeptic symptoms have been more or less continuous, there may be very considerable loss in weight; and, if there has been active haemorrhage, anaemia may be a striking feature of the condition. Apart from actual cicatricial contraction, another not uncommon result of continued irritation from a chronic inflammatory process is the formation of adhesions, which may involve not only the duodenum itself, but the neighbouring gall bladder or stomach as well, and so give rise to a confused series of symptoms, which it is often difficult to explain. The urine may be normal, but it sometimes shows a trace of albumen, sometimes has an excess of bile, sometimes contains indican, and not infrequently gives a positive pancreatic reaction.

In the diagnosis of duodenal ulcer the conditions with which it is most liable to be mistaken are gastric ulcer, neurasthenic gastralgia, gall colic, and the gastric crises of locomotor ataxia. In gastric ulcer the pain comes on immediately or shortly after food, according to its situation, and is at once relieved by vomiting; the local tenderness, if any, is situated in the epigastrium; the appetite is poor, and whatever slight desire there may be for food is checked by the dread of the pain that will follow its ingestion, it being a point of some diagnostic importance that the degree of pain is frequently in direct ratio to the quality and quantity of the food taken; when bleeding occurs it is generally, in the first instance, in the form of haematemesis. In gastralgia the pain comes on suddenly at any time and irrespective of food, by which it may be even relieved; pressure over the epigastrium or the eructation of wind generally relieves the discomfort; haematemesis or melaena never occurs; the patient is usually of the neurasthenic type. In gall colic the onset of the pain is more acute, and its intensity is more severe; it is always accompanied by nausea or vomiting, and often, though not always, followed by jaundice; the liver is frequently enlarged, and there is tenderness on pressure over the gall bladder; there may be a history of previous attacks, and of the recovery of gall stones from the faeces. Cases have been recorded of duodenal ulcer, confirmed by operation, in which the attacks of pain so closely resembled biliary colic, that they necessitated the patient being put to bed with hot bottles and the administration of morphine. In the gastric crises of locomotor ataxia their true nature will be determined by the presence of ocular symptoms, a history of previous lightning pains and the absence of knee-jerks. Gastric and duodenal ulcers originate in much the same way and present similar anatomical characters. Their symptoms may resemble one another so closely as to make a definite diagnosis impossible in many instances, while, as has been already pointed out, the complexity of the symptoms which may arise when the morbid condition is one of such a nature as to involve the gall bladder or pyloric end of

the stomach as well as the duodenum itself, is such as to make definite differentiation a matter of impossibility. And yet, despite this uncertainty, there are not wanting at the present day those who advocate an abdominal section in the sure confidence of finding a duodenal ulcer if the patient presents the following symptoms: Sudden hæmatemesis or melaena; a history of recurrent gastralgic attacks with intervals of apparent good health; pain in the stomach region coming on three or four hours after a meal and relieved by food; flatulence and regurgitations of acid fluid from the stomach; tenderness and increased resistance to pressure in the right hypochondrium.

Such a combination of symptoms no doubt affords strong presumptive evidence in favour of duodenal ulcer, but does not justify the wholesale condemnation of every patient presenting such a picture to the operating table. One surgical authority goes the length of saying that it is "not necessary to the attaining of an accurate diagnosis that any examination of the patient be made." The meaning of this statement presumably is that the patient's own statement of his sensations is sufficient to warrant the opening of his abdomen without the formality of any physical examination. This same authority states, of a patient who was sent to him because of an inguinal hernia: "As he entered my room I was struck with his blanched appearance, and my diagnosis of duodenal ulcer was made before he reached a chair." To the ordinary observer such a process of lightning diagnosis is, to say the least of it, startling.

The following case well illustrates some difficulties that are not uncommonly met with:

A robust, energetic, full-blooded, gouty man of 54 years of age, had never been ill throughout the length of a busy and over-strenuous life, except for a mild attack of enteric fever which he went through when between 20 and 30 years of age. From the time of that illness he suffered every now and again from attacks of what he believed to be indigestion, which he ascribed to more work than usual, and from which he always recovered if he gave himself mental and physical rest, and curtailed the supplies of food, alcohol, and tobacco. He had been a free liver, he smoked to excess, indulged daily in three large meals, dealt generously with himself in the matter of alcohol in all its varieties, and seldom went to bed before the early hours of the morning. He was distinctively of the neurotic type, and flattered himself that he was able to keep his health in perfect order by physical exercises of one sort and another, and by periodic courses of energetic and even violent massage. The only evidence of organic disease he presented to physical examination was a marked diminution in the area of liver dullness, a tendency to hæmorrhoids, and an occasional trace of albumen with a persistency of phosphate in the urine. He slept perfectly at night, digested everything he took between the times of his gastralgic attacks, increased gradually in weight, and had no tenderness either in hypogastrium or epigastrium on the deepest pressure. His gastralgic attacks recurred at varying intervals, and, when he allowed them to be medicinally treated, were always rapidly relieved by citrate of caffeine, phenacetin, and codeia. On a certain Saturday he overtook himself on a golf links, took a large luncheon between his games, and in the evening indulged in an abundant dinner. After going to bed he found himself unable to sleep, and, feeling his stomach uncomfortably overloaded, he proceeded to subject himself to severe physical exercises and deep abdominal kneadings. This produced faintness, which was followed during the night by a moderate hæmatemesis and the following day by considerable melaena. He became sufficiently alarmed to take proper care of himself, rapidly became well, and has remained so ever since. Here is a picture which at first sight seemed to justify the advice given him to have a gastro-enterostomy done without delay, as beyond all doubt he was suffering from a duodenal ulcer, which, to permit of such an amount of hæmorrhage, must be deep, and therefore more than likely to perforate. But there is another side to the story, and before committing such a case to the risks of an operation which might well enough prove to be unnecessary the following points are worthy of attention: The patient is a man of the distinctive neurotic type; the history of his life, and such physical signs and symptoms

as he presented, all pointed to the existence of a slow cirrhotic process, which had mostly affected his hepatic tissues; his attacks had been so far typically gastralgic, and had yielded at once to antineuralgic remedies; the hæmorrhage he had suffered from could be fully explained by the state of his liver, *plus* excessive exertion, *plus* too much food and drink, *plus* unwise manipulation of his abdomen; there had been a complete absence of hypogastric tenderness or muscular resistance to pressure over the duodenal region; the gastralgic attacks had only occurred after—and not impossible in consequence of—unusual and excessive physical and mental strain; there was an entire absence of dilatation of the stomach, or of other evidence of cicatricial contraction of the duodenum, such as might reasonably have been expected if, over so many years, he had been suffering from an ulcerative process. All these considerations surely afford good reasons for patience and further observation, and, to my mind, justify an attitude of scepticism as to the urgency, or even necessity, for surgical intervention at the present juncture of affairs. Such an attitude of reserve is fortified by the consideration of yet another case: A gentleman of 51 years of age had suffered for many years from indigestion and pains in the abdomen, which were ascribed by different doctors to duodenal ulcer, gastralgia, gall stone, and simple gastric catarrh. He was treated on many plans, and underwent the usual cure at Carlsbad, but though he got better for a time his discomforts always returned. Eventually he was operated upon in the belief that the true cause of his symptoms must be gall stones. The gall bladder and ducts were, however, found to contain no stones, though he was told that many adhesions had been found round and near the gall bladder. After his operation he was better for a time, but soon he began to experience nausea, heartburn, and pain in his stomach, which always came on three or four hours after a meal or during the early hours of the morning, was accompanied by acidity, and was immediately relieved by food or a dose of bismuth and soda. He had recently lost fully a stone in weight. There was no tenderness in his right hypochondrium, but he had definite though slight dilatation of his stomach, and his conjunctivæ were suspicious of a mild degree of icterus. The urine was found to give a marked pancreatic reaction to Cammidge's tests, but contained neither sugar nor albumen. On further inquiry it transpired that at the time of the operation the adhesions were found to be the accompaniment of a mass of glands which were regarded as probably tuberculous, and which, spreading from the root of the mesentery, involved the head of the pancreas. Here again is a case where the symptomatology was so obscure that a definite diagnosis was impossible, and where the operation revealed an unexpected explanation in the discovery of glands which so interfered with the normal activity of the pancreas as to set up a mild form of pancreatitis. Incidentally it may be remarked that this case bears witness to the value of Cammidge's tests, the positive reaction they afforded being fully confirmed by the surgical history, which was only obtained subsequently. It further raises the important question of how far the symptoms which are now attributed to duodenal ulcer may not frequently be, in some degree, the result of pancreatic insufficiency. A distinguished physician has put it on record that "the pancreatic C reaction has been found in all my cases of duodenal ulcer demonstrated at the operation in which it has been looked for, and in the majority of those in which a duodenal ulcer was inferred from the symptoms." And an equally distinguished surgeon has confirmed this experience and has stated that the pancreatic test "is common both to duodenal ulcer and to cholelithiasis, both of which are associated with duodenal catarrh." We are still without any well-defined clinical picture of the subjective phenomena which result from a chronic pancreatitis. Cammidge's tests applied to the urine and faeces, even if taken alone, are of the highest suggestive value, though they only become essential links in the chain of evidence when they confirm clinical observation; they then convert a tentative into a definitive diagnosis. The pancreas, when disturbed only by a chronic process, may be quite impalpable, and may yield no sensations of pain, either subjective or objective; the sclerotic changes which have invaded its interstitial tissue

may not have encroached upon the islands of Langerhans, and the urine may therefore give no positive reaction to Fehling's test, such as would arouse our suspicions; the appetite may remain vigorous, and any digestive disturbance complained of may be apparently entirely unassociated with the intake of food or drink; and, to outward appearance, the patient may look well and hearty. But may it not be true that many cases which are now regarded as due to suppressed gout, inactive liver, gastralgia, acid dyspepsia, or duodenal ulcer, are really instances in which the pancreas is either wholly or partially responsible for the disturbance of health, which, though definite, is too ill-defined to admit of a positive diagnosis?

Let me quote yet another striking case in support of such a hypothesis:

An active man of 52, of the gouty type himself and belonging to a family many members of which had suffered from acute gout, who had worked hard and lived abstemiously, and whose only previous illness of consequence was a mild attack of enteric fever fifteen years previously, had suffered periodically over some years from recurrent attacks of what were regarded as gastro-hepatic catarrh due to gouty disturbance. These attacks were always of the same type; they came on after unusual work or worry, were characterized by nausea, anorexia, headache, somnolence, pronounced mental depression, slight icterus, colicky pains in the upper region of the abdomen, and a profuse discharge of lithates in urine containing an excess of bile. They lasted over several days and cleared up under the influence of rest, a semi-starvation diet, and alterative medicines. A final attack, however, supervened in which to the same train of symptoms there was added a marked and gradually deepening jaundice with pains which were more severe than any he had experienced on previous occasions, and with a nausea that went the length, several times, of active sickness. Remedies failed to afford relief, and eventually he developed pyrexia to the accompaniment of a succession of mild rigors. Under the impression that he must be suffering from gall stones, with a superimposed cholecystitis, his abdomen was opened. No gall stones were found, but his pancreas was hard and swollen though not apparently haemorrhagic. His gall bladder was drained and he made a satisfactory recovery. Since the date of his operation he has resumed active work, and kept well, but he still has occasional attacks similar to, though less severe than, those he previously suffered from, and he has developed a mild glycosuria which is kept in check by careful attention to diet, etc.

It is of interest to mention, in passing, that in the case of this patient, his attacks, when they occur, are now speedily remedied by the administration of colchicum and alkali, together with repeated small doses of rhubarb and blue pill. This raises the very significant possibility of his pancreatic troubles being possibly, even probably, gouty. And why should not the poison of gout lay siege to the interstitial tissues of the pancreas as readily as to those of the liver or kidneys? Indeed, why not even more readily, when it is remembered what an important part the digestive juices and internal secretions of the pancreas play in the processes of active metabolism? Is it not conceivable that what, for want of a better or more accurate name, we call suppressed gout may not in time prove to have for its etiological foundation active changes in the pancreatic tissues—changes which, though of minor and passing importance at first, gradually end in the establishment of cirrhosis of a more or less widespread distribution? May it not even ultimately turn out that true sthenic gout itself is closely related to active pancreatic changes in the first instance? As bearing upon such a possibility, I need hardly remind you of the frequency with which glycosuria develops in later life among men who in their earlier years have suffered from gout, and how it is apt to be associated with a minor degree of albuminuria—suggestive evidence surely of interstitial changes in the pancreas analogous to similar changes of an interstitial type in the kidneys!

It is admitted that ulcer of the duodenum is sometimes a multiple lesion; that it is often associated with gastric ulcer, and with ulcerations elsewhere in the course of the gastro-intestinal tract. In view of this fact, may not the ordinary duodenal ulcer turn out to be a mere local expression of some dyscrasia, gouty or other, and not a

disease entity at all? From such a standpoint it is not surprising that duodenal ulcers tend to recur, nor that operation, even if temporarily ameliorative, is often inefficient as a measure of permanent cure.

In an illuminating lecture delivered at the London Hospital in February last, the author, who is one of the physicians to the hospital, expresses the opinion that "gastric or duodenal ulcer is what we commonly term a diathesis, that is, a natural proneness of the body to a given disease, and that the actual visible ulcer itself is an acute, usually virulent attack of the disease or outbreak of the diathesis, just as an acute attack of gout or rheumatism is an acute outbreak or attack of gout or rheumatism in a person prone to these diseases."

Without the evidence afforded by operation many such cases must obviously remain obscure, but it seems to me that we have not yet arrived at sufficient accuracy of diagnosis to justify certain claims that have been made for surgical interference. In some cases such as the last I have related, surgical assistance is essential; and there may be others, less acute than this, in which draining the gall bladder and bile ducts, or the performance of a gastro-enterostomy, may prove a preventive measure, so far especially as the pancreas is concerned, of the utmost value; but to insist that every patient presenting symptoms of a certain type and sequence, which may or may not be caused by ulceration of the duodenum, should be forthwith exposed to the risk of an abdominal section is going too far. And I am clear, from personal observation, that the symptomatology of duodenal ulcer is by no means so definite as some of the protagonists of this modern surgical method of treatment would have us believe. In the first case I have mentioned to you the patient may, or may not, have a duodenal ulcer, but, to my way of thinking, his case is one to be carefully watched, but not to be interfered with surgically until more precise indications arise, or until his comfort and health are becoming so seriously impaired as to render operation imperative. In the second case operation only brought to light an unexpected explanation of the symptoms, but failed to do more than afford such relief as came from breaking down some adhesions. I do not suggest that, in this case, operation should have been declined; under the circumstances it was the one course left open which was likely to be of use; my contention is that the symptoms which are so confidently regarded by some authorities as indubitably due to duodenal ulcer may occur in, or be so closely simulated by, other pathological conditions as to make such cocksure certainty in diagnosis unreliable.

In the course of a recent discussion at the Royal Society of Medicine, one of the speakers related the case of a patient on whom he operated who had all the characteristic symptoms of duodenal ulcer, "including periodicity of attacks, and hunger pain, etc., and yet no duodenal ulcer was present."

He also referred to others in which, in his experience, the same result had been met with. An infection originating in the upper part of the gastro-intestinal tract must, one would imagine, often find its way up the ducts to the gall bladder and liver on the one hand and to the pancreas on the other, and more especially would this seem likely to happen in conditions where the duodenum had become functionally disturbed even before the process of ulceration was an accomplished fact. Not only has operation often failed to reveal the ulcer whose existence was confidently predicted beforehand, but many cases have been published which prove that operation, even in admitted cases of the disease, have failed to afford relief: notably in a paper communicated to the BRITISH MEDICAL JOURNAL of this year, the writer tabulates five cases of duodenal and three of gastric ulcer, in all of which the results, so far as symptoms were concerned, were so unsatisfactory that the condition of the patient was no better after than before surgical intervention was resorted to. Until differential diagnosis enables us to eliminate many possible sources of fallacy, it will be most in the interests of patients suffering from suspicious symptoms to keep them under careful observation, and within reach of skilled surgical assistance, but to allow them to exhaust the possibilities of medical treatment before adding to the risks of their condition that of a major operation.

As regards treatment, it must be borne in mind that if an ulcer actually exists its healing must be a slow and

somewhat tedious process, and that, if it be a mere dyscrasial phenomenon, it is liable, even if wholly or partially healed, to recur on every renewal of dyscrasial activity. The way in which many cases of supposed duodenal ulcer become well when carefully managed makes it seem likely that in some instances either no accomplished ulceration, or ulceration of modified extent only, is present. We are all familiar with the difficulties met with in the diagnosis of stomach ulceration, and of the large proportion of cases at first labelled "ulcer" which get completely well under rest, regulated diet, and suitable medicinal treatment. It would require some courage to advocate the opening of the stomach on the provocation only of a set of symptoms that might, but also that might not, be the result of an ulcer somewhere on its mucous surface, and yet nothing less than this is the procedure which the duodenal enthusiast asks us to adopt. In suspected cases the patient should limit for the time being his active work, and must carefully avoid all exercises or active games that throw special stress on the abdominal muscles. In the event of a haemorrhage having occurred, the rest obviously must be complete and in bed. Under such circumstances the food should be bland, easily digested, and given at stated intervals in limited quantity. A semi-starvation diet is recommended by some authorities; but though this may be advisable in acute cases for a few days, it need never be pushed the length that is sometimes found necessary in gastric cases, because ingestive discomfort is comparatively slight and unimportant.

After the first acute phase of the illness is over the diet may be reasonably generous, but it is well to remember that tea, stimulants, uncooked vegetables or fruit, and sugar are best done without, and that starchy food should be taken in moderation. The following plan of dietary will be found to suit many cases: A cupful of hot water to be slowly sipped before breakfast every morning; for breakfast, café-au-lait sweetened with saxon, toasted protein, or caseoid bread with butter, eggs poached, boiled or scrambled, or white fish. During the forenoon a cupful of thin clear meat soup, which it is advantageous to thicken with about half an ounce of gelatine. For luncheon well-cooked fresh veal, mutton, or lamb, cooked green vegetables, preferably in the form of a purée; stewed fruit, sweetened with saxon; some form of starchless bread or biscuit, thickly spread with sour milk or lactic acid cheese; Perrier, Bilin, Gieshühler, or other natural alkaline water. In the afternoon boiled milk, or cocoa infused from the nibs; toasted bread and butter, simple tea-cake. For dinner, fish, plainly cooked and without rich sauce; egg entrée, sweetbread or calf's head, chicken or other form of bird, a small quantity of potato in the form of chips or ribbons, a simple omelette or a savoury. A small quantity of brandy or whisky, if desired, with the meal, and a cup of black coffee after it. Farinaceous puddings, bread, other forms of meat, etc., may be gradually added to the dietary as improvement takes place.

It is of elementary importance to make sure that the buccal cavity is clean, and to this end all decaying stumps should be removed and carious teeth cleaned and stopped. An antiseptic mouth and throat wash should be used freely at least twice a day, both as a gargle and also to brush the teeth with.

As regards medicines, my experience is that most cases are speedily relieved by a combination of bismuth and carbolic acid, to which, if the pain is at all severe, a suitable dose of morphine may be added. The following is a useful formula: Carbonate of bismuth, 40 grains; pure carbolic acid, 2 grains; solution of hydrochlorate of morphine, 15 minims; mucilage of acacia, *quantum sufficit*; compound infusion of gentian to make up 1 oz. To be taken in water from two to three hours after every meal.

To counteract the constipation which usually results from large doses of bismuth, the following or some such pill should be given at bedtime; Calomel, $\frac{1}{2}$ grain; extract of rhubarb, 2 grains; extract of cascara sagrada, 1 grain; capsicine, $\frac{1}{2}$ grain.

When all active symptoms have been subdued, the bismuth mixture may be replaced by such a tonic as this: Finkler's papain, 2 grains; carbolic acid, 2 grains; strychnine, $\frac{3}{16}$ grain; codeia, $\frac{1}{4}$ grain; and extract of rhubarb,

$\frac{1}{2}$ grain. To be put in a capsule and taken after meals three times a day. And the aperient pill may be replaced by a morning dose of Carlsbad salts, when required. When haemorrhage has occurred, it is usually an indication for the administration of iron when the tonic stage is reached.

Treatment by sterile horse serum, together with a dry and mostly meat diet, has been advocated recently, but, though I have not resorted to it in cases of suspected duodenal ulcer, I have given it a fair trial in gastric ulceration, but without any result sufficiently striking to confirm the claims advocated for serum as the most valuable of all methods of management.

The leading indications for surgical interference would seem to be: The occurrence of perforation; repeated melaena or haematemesis, especially if associated with pain and resistance to pressure over the right hypochondrium; cicatricial contraction of the duodenum, causing dilatation of the stomach; resistance of the symptoms to all forms of medical treatment; and recurrent attacks of cholecystitis, accompanied by evidences of pancreatitis.

TUBERCLE BACILLI IN THE FAECES IN TUBERCULOSIS.

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It has been commonly supposed and taught that the discovery of the tubercle bacillus in the stools is diagnostic of tuberculous enteritis. Until recently it had hardly been conceived that the tubercle bacillus might be found in the faeces of persons suffering from other forms of tuberculosis. The observations which follow show how frequently they occur in the faeces of patients affected with pulmonary tuberculosis. Their occurrence in the stools would indeed seem to be much more constant than in the expectoration.

It may be recalled that last year Rosenberger¹ described the presence of tubercle bacilli in the blood in different forms of tuberculosis. More recently Willson and Rosenberger² announced that they had found tubercle bacilli in the stools of 100 tuberculous patients, including early cases, and in 21.5 per cent. of 1,033 clinically normal persons. According to these observers, the bacilli obtain access to the blood stream and are carried from the affected focus to the intestinal wall, through which they are excreted, and so are finally discharged in the stools. Klose³ has likewise found that in almost every case where tubercle bacilli were found in the expectoration they were also found in the faeces.

The significance of the constant discharge of tubercle bacilli from the alimentary tract is immense, both from the point of view of clinical medicine and of public health. On this account it seemed to us desirable to make further observations on the subject, and these are now recorded.

Uhlenhuth and Xylander⁴ have recommended the use of "antiformin," a mixture of alkali hypochlorite and alkali hydrate, for the purpose of distinguishing acid-fast from other bacteria in sputum. Acid-fast bacteria can resist this disinfectant when diluted to 20 per cent. for two to five hours, while other bacteria and organic matter generally are speedily dissolved. Uhlenhuth mixed sputum with antiformin in sterile Petri dishes. We used instead conical precipitating glasses. A small piece of faeces, about a cubic $\frac{1}{2}$ in. in size, is placed in a conical glass, and to this some 20 c.cm. of antiformin, which has been diluted with water to 15 per cent., is added. The faecal matter is well broken up and mixed with the fluid, and thereafter as much again of the dilute antiformin solution is added. This is further mixed, and allowed to stand for about an hour. A white curdy precipitate appears on mixing, and in the course of an hour settles into a white layer which varies in breadth. Beneath the white layer some unchanged faecal matter remains, and above the white layer the fluid is of a clear yellow or brownish colour. A residue, including tubercle bacilli, which have been set free by the destruction of the other