Remarks ON INDIGESTION FROM A SURGEON'S STANDPOINT.*

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To discuss indigestion among the common complaints is obviously correct, because it is probable that nearly everyone from time to time experiences sensations due to temporary upsets in the complicated co-ordination of gastrointestinal physiology; but the fact that some of these upsets are due to surgical disorders, and may necessitate an operation of more or less severity, raises them above the plane of minor ailments, though they may still be common complaints.

The title of this address suggests that indigestion may be divided into strict categories—that there is a type with which the surgeon must deal, a type for the physician, and doubtless another for the gynaecologist. But is that really so? I would rather say that the standpoint—that is, the point from which we view the patient—is the same for all medical men, and that the time has gone by when the surgeon was merely a craftsman obeying the order of his superior, the physician. Nowadays we all, whatever our label, insist on making our own diagnosis. We are all equally equipped to investigate conditions, however obscure; we use the same diagnostic tools, and, in most cases, come to the same conclusions. We still differ, though to a constantly diminishing extent, on treatment; but with an increasing appreciation, not so much of the nature of a disorder as of the stage at which it has arrived, we must inevitably reduce the number of those cases where differences still exist. We surgeons can take some credit for insisting on what has well been called the living pathology—that is, for exposing to the eye and permitting the minute examination of diseased organs at an early stage—a study more fruitful than is an examination, after death, of structures so altered by prolonged disease that the early signs are unrecognizable. I would mention, as an example, how the knowledge that has resulted from the histological examination of the gall-bladder, removed at an early stage of the disease, has led to a correct estimation of the effect that cholecystitis has on the general health, and to a realization of how much more important the disease of the gall-bladder is than the stones it may happen to lodge. Or, as another example, how a critical examination of displaced viscera has led us to modify the views at first held on visceroptosis. Of course, it may be years before some ingrained beliefs are abandoned—such as, for instance, that hyperchlorhydria is a disease, and that it is due to overwork, or that recurring abdominal pain in children is due to “bilious attacks,” or that gall-stones can be cured by medical treatment. But, gradually, knowledge is spreading that surgery has done much to explain the early stages of many abdominal complaints, and that certain abdominal disorders, as far as their treatment is concerned, are purely surgical from the very beginning.

DISORDERS OF THE STOMACH.

When one talks of indigestion, one thinks at once of the stomach, and certain questions arise which require an answer. A. Is the stomach itself diseased, or is it acting in sympathy with some other diseased organ? B. If the stomach is at fault, is it because of some physiological variation or of a pathological change? C. If a pathological change, is that amenable to medical treatment, or must some direct attack be made on it by a surgical operation?

A. We know that a perfectly healthy stomach may be so affected by disease in other viscera that its disordered

action may simulate a definite lesion. The best known instances are the dyspepsias in lesions of the appendix and in gall-stones. It is accepted that gastric pain is due to disordered muscular action, and is really an exaggeration of normal sensations, such as hunger, which are associated with hypermotility. The presence of some irritable focus, such as a chronic appendicitis or cholecystitis, excites a hyperperistalsis in the stomach, and pain results. It is for the diagnostician to distinguish between this condition and a similar condition due to a local lesion, for it is obvious that treatment directed to a referred condition must be ineffectual while the causative lesion is still active.

It is interesting to note that appendicitis will more readily excite symptoms of duodenal ulcer in persons of the duodenal diathesis—stalwart, well-developed men, in whom hypermotility and hypersecretion are the rule—and symptoms of gastric ulcer in the hyposthenic type with a narrow chest, poor physique, and a tendency to visceroptosis, in whom gastric motility and secretion are both on a lower plane. Our first duty, then, is to distinguish between actual and referred symptoms, and everything depends on a correct answer to this first question.

B. Investigation has proved that the stomach is very responsive to internal and external stimuli. The flow of gastric juice is excited or inhibited by many causes, some of them physical, others mental, and even the level of the gastric curvature may vary at the call of the emotions, so that the “sinking feeling” has a real physical basis. Too much reliance cannot, then, be placed on decimal variation in gastric acidity, but, if there is a pathological change in the stomach, the investigation of gastric secretion may be of real assistance. We know, too, that a small proportion of healthy and otherwise normal people have no free hydrochloric acid in their gastric secretion, but whether that renders them more liable to develop diseases associated with low gastric acidity—for example, pernicious anaemia or gastric cancer—is not known. We do know that they have a normal appetite and good digestion, in spite of this apparent anomaly. It is very doubtful whether variations in gastric secretion give rise to symptoms, and to distinguish between physiological variations and pathological variations requires other means of investigation, among which the x-ray barium meal examination and the chemical examination of the stools have chief place.

C. Having determined that a pathological lesion is present, the question arises as to the prospect of permanent cure, and the choice of methods to ensure it. It is here that the medical view and the surgical view diverge. Until quite recently the disappearance of symptoms was looked upon as evidence of cure, but with more exact methods of examination we know now that, while in practically every patient with gastric or duodenal ulcer confined to bed, and treated on medical lines, all the symptoms will disappear in a few days, the ulcer takes many weeks to heal, and may even remain unhealed. We know also that from time to time the x rays will demonstrate the gradual diminution in size and eventual disappearance of really large crater ulcers under medical treatment. The question we have to answer is: What type of ulcer gives a reasonable chance of cure by medical means, and what are the chances of such a cure being permanent? We know that all superficial ulcers of the stomach and duodenum are associated with remission of symptoms, sometimes for long periods, but that there is a tendency to recurrence. It is probable—and I think I have proved it by seeing many of these conditions during abdominal operations—that during a remission the ulcer really heals. Have we any assurance that a superficial ulcer healed under medical treatment will be less likely to recur than one which heals without treatment? There can be no answer to the question until we can collect the after-histories of thousands of contrasted cases. With regard to crater ulcers, the matter is less controversial. Remissions in these cases are less the rule, and these ulcers behave like all other ulcers with a more or less fixed base in their reluctance to heal and their tendency to break down again. In spite of the gratifying progress seen by the x rays in some of these cases treated medically, I am convinced that the proportion of recurrences is so high that medical treatment is not ideal for these cases, even if we overlook the fact that these are

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essentially the cases in which grave complications occur. Of these complications, perforation has lost much of its terror since early intervention became the rule, but haemorrhage is still a serious sequel, and one that is too often beyond surgical skill. I have at present in my wards a patient who had on two occasions been entered for admission for a duodenal ulcer, and had twice refused the bed owing to remission of symptoms. The ulcer perforated a few days ago, and he has had a hard fight for his life.

Another point bearing on the choice of treatment arises from one's views on the etiology of gastric and duodenal ulcer. The opinion is widely held that these ulcers are not primary, but are secondary to some septic focus elsewhere; and, in their turn, the teeth, the tonsils, the nasal sinuses, the gall-bladder, the appendix, and indeed the whole intestinal tract, have been suspected of being the source of the infection. I have been impressed by this theory, as, in the days when I treated lesser curve ulcers by excision only, I saw many recurrences at the site of excision—a very natural sequel if all I had done was to remove a result and leave the cause behind. I have also seen patients with duodenal ulcer, upon whom I have performed gastro-jejunostomy, fail to obtain relief until the appendix or the gall-bladder has been removed at a subsequent operation. In accordance with this belief, it has long been an accepted line of treatment by physicians and surgeons alike to look for, and, if they are present, deal with all forms of sepsis in the upper respiratory and alimentary tracts as a preliminary to treating the ulcer. But it is impossible by any means other than a laparotomy to obtain a real knowledge of the condition of the gall-bladder or appendix; nor is it possible to get rid of these two common foci of infection by any means except their removal by operation. If, then, there is reason to suspect that an ulcer in the stomach or duodenum is associated with a lesion of the appendix or gall-bladder, a permanent cure can be hoped for only by operation.

LESIONS OF THE APPENDIX.

I need not apologize for taking up your time by discussing the dyspepsia associated with and due to chronic appendicitis. The old controversies as to whether the appendix, crippled by an attack of acute inflammation, could, when apparently quiescent, give rise to symptoms, have all died down, and there is general agreement that an abnormal appendix can give rise to dyspeptic symptoms. Indeed, it is probably a frequent cause of the type of dyspepsia in children commonly labelled "bilious attacks." But the symptoms may be anomalous; they may be indefinite, or they may point to a different organ, and so we undoubtedly see mistakes made in the diagnosis of appendix dyspepsia. Many times has the appendix been removed, and a duodenal ulcer or cholecystitis left undiscovered and untreated. These mistakes do and will occur in spite of all our care; but their number may be reduced by the strictness of our investigation, and perhaps particularly by our realizing that such mistakes are possible.

What symptoms are we to rely on for a diagnosis of chronic appendicitis? Some cases are quite easy. There is a history of characteristic attacks with intermissions during which the patient may, or may not, be free from discomfort. But it is in the cases where there have been no definite attacks that difficulty arises. Indeed, some surgeons maintain that the chronic appendix does not exist unless there have been definite acute attacks. I believe that there is a type of chronic appendicitis, characterized by sclerosis and thickening of the organ with obliteration of the lumen from the tip, which can arise independently of acute attacks of inflammation. This type of appendicitis gives rise to a train of symptoms referred to the stomach and accompanied by some minor local discomfort.

Probably the commonest error is to confuse appendicitis and visceroptosis. We know now that variations in level of the abdominal viscera occur in quite healthy people; but it is not difficult to diagnose pathological visceroptosis. The symptoms are typical; the patient herself is typical. But the question we have to answer correctly is this: "I know this patient has visceroptosis; I know that all

her symptoms could be due to that disorder; but can I be certain that the fixed pain she complains of in the right iliac fossa is not due to a coexisting disease of the appendix?" We have all seen scores of cases of visceroptosis in which the removed appendix gives every sign of chronic inflammation. And so the appendix is removed and the patient starts on the long downward path of repeated operations. Two months ago a patient was referred to me who had had eleven operations; last week I saw a woman of 35 who had begun her operative life—if you might so call it—at 18, and had had six more operations since then. Each operation seems to give temporary relief, but is soon followed by what is euphemistically called "adhesions," and another operation is done. There is no doubt that there is real difficulty in excluding disease of the appendix in cases of agreed visceroptosis; but my advice would be that, in spite of an occasional brilliant success from anchoring operations, visceroptosis is not a disorder which is likely to benefit from surgery.

Unfortunately, the barium x-ray examination rarely helps much in the diagnosis of appendicitis. Occasionally it is possible to visualize the appendix and to prove that it is adherent and tender, but, unless the appendix is visualized, no opinion can be given, and we know that many of these atrophic sclerosed appendices are quite free from adhesion. I should sum up the question by saying that local pain, and even tenderness in the appendix area, unaccompanied by "indigestion," must not be accepted as evidence of appendicitis in the absence of a history of acute attacks.

DISEASE OF THE GALL-BLADDER.

Chronic cholecystitis presents an even more difficult problem. I do not refer here to cases complicated by the presence of gall-stones, for many of these give rise to a characteristic train of symptoms which makes diagnosis easy. It is the type where a low-grade streptococcal infection deep to the mucosa leads to gradual thickening and distortion of the gall-bladder, often with adhesions to the neighbouring structures. This pathological condition has been discovered and investigated purely by the efforts of the surgeons, who have been able to see the early changes in the gall-bladder, and, by removing the organ, to examine it histologically and bacteriologically at all stages of the disease. We know that the infection is generally due to a streptococcus; that it is blood-borne; that its seat is deep to the mucosa, which for a long time is unaffected; that it leads to gradual changes in the appearance of the gall-bladder; and that it may have effects on the general health much more widespread than its effect on the digestive apparatus. The healthy gall-bladder is a bright sky blue in colour, a colour which extends down to the cystic duct, which is enclosed in a fatty envelope. When the gall-bladder is diseased, the first change is seen near the cystic duct, where the fatty envelope seems to be spreading towards the fundus. The gall-bladder loses its bright colour, the last to disappear being at the fundus, and at the same time it becomes thickened and contracted until it reaches the stage, so often seen in the post-mortem room; of a yellow, shrunken, distorted organ. With this occurs a local hepatitis, so that the gall-bladder becomes intimately adherent to the liver, instead of being attached to it by a loose areolar bed.

The symptoms are not too clear-cut in the absence of stones, but, in most cases, the patient complains of an almost constant aching and dragging in the right hypochondrium, not referred to the angle of the scapula as frequently as when stones are present. Loss of appetite with flatulent dyspepsia, and a disinclination to take fatty foods, are common—taking food excites the pain, especially if adhesions are present—and sometimes there is definite loss of weight. From time to time mucus may form and lodge in the cystic duct, giving rise to a modified kind of colic. The only clinical sign of any diagnostic value is the occurrence of local tenderness elicited by Murphy's method, but occasionally one finds the liver enlarged.

There are two methods of investigation which may, as technique becomes standardized, lead to a great advance in the diagnosis. The x-ray examination of the gall-

bladder after administration of the dye tetraiodophenolphthalein has already proved its value. The dye may be administered intravenously or by the mouth in capsule. The former method is certain, but has technical difficulties; the latter is less certain, as one is not always sure that the dye is absorbed, but it is easy. In both cases the patients must be carefully prepared. The dye is excreted almost entirely in the bile, and should appear in the healthy gall-bladder four hours after intravenous administration, or twelve hours after oral administration. The shadow, light at first, becomes denser as the bile becomes concentrated, and repeated photographs are taken; a meal rich in fats is then given, and the gall-bladder empties. The patient is without food for at least fifteen hours. *Failure to obtain a shadow* after intravenous administration shows something amiss in the biliary apparatus; after oral administration, it may mean only a failure of absorption, and is a demand for intravenous administration. *Mottling of shadow* suggests calculi, which are often defined very clearly. *Irregularity of contour* suggests adhesions, and *variations in density and time of appearance and disappearance* suggest functional derangement. A wide experience is necessary to assess the value of this last sign.

The other method is one employed and perfected by Dr. Vincent Lyon of Philadelphia, who has demonstrated this method to me on several occasions. It involves the use of a duodenal tube, which is passed through the stomach into the first part of the duodenum; a saturated solution of magnesium sulphate is introduced through the tube, and in a few minutes the contents of the duodenum are withdrawn by suction. The magnesium sulphate relaxes the sphincter of Oddi, and Dr. Lyon maintains that he is able to collect the bile from the common duct, from the gall-bladder, and from the hepatic ducts into separate receptacles, and examine each specimen microscopically, chemically, and bacteriologically. I have seen many results of these experiments, and I am satisfied that from them a great deal can be learned about the condition of the biliary passages.

The treatment of an infected gall-bladder is removal. There is no certainty that any form of medical treatment has any avail. Many spas, such as Carlsbad, Neuenahr, and Harrogate, make claims to cure cholecystitis, but it is doubtful whether the improvement, if any, is more than temporary. Drainage of the gall-bladder is useless in view of the site of the infection. Cholecystectomy is said to be a dangerous operation, but in experienced hands there is hardly any danger, and, so long as the condition is uncomplicated, the risk is limited to post-operative chest complications, which can generally be avoided by careful choice and preparation of the patient.

INFECTION OF THE MESENTERIC GLANDS.

There is one form of indigestion to which I have drawn attention that is worthy of notice. It results from infection of the mesenteric glands, and is generally tuberculous in character. Though it usually affects children, no age is exempt, and I have now under my care a case in a man of 24. The glands are infected from the intestine, probably by milk, and though the disease is at first local, the glands may later on become foci for widespread tuberculous disease elsewhere. The symptoms are due to disordered intestinal peristalsis, and consist of short-lived colicky pains not well localized, brought on by exercise, and relieved by heat and pressure. The pain is so severe that children will cry out. Vomiting is unusual. There is no sign of intestinal ulceration. The lesion can be distinguished from appendicitis by the character of the pain, which may occur frequently, but lasts only a few minutes or less. There are never attacks lasting two or three days with pyrexia and abdominal rigidity, such as one sees in appendicitis. Between the attacks the patient is quite well; but, as the condition progresses, caseation with adhesions may occur, and complications of the minor obstruction type may arise, while recovery from the attacks is not so complete, with the result that the patient's nutrition suffers. Three stages are recognized: enlargement of the glands, probably in the early period a simple infection; then caseation; and finally calcification. The caseation stage is the danger stage, as at this period the serous covering is often

affected, and adhesions may form, leading to obstruction; while, later still, with the giving way of the gland capsule, widespread peritonitis with massive adhesions leads to a condition in which there is little hope of cure. The glands first affected are those in the ileo-caecal angle, and the predisposing cause is probably a pathological appendix. Treatment is surgical, and consists of laparotomy and removal of the appendix. If any caseating glands are found they should be removed, the greatest care being taken to reperitonize the bed from which they have been removed. Subsequent treatment is on ordinary sanatorium lines, avoidance of milk, unless its bacterial cleanliness is assured, and obtaining either actual or artificial sunlight.

DYSPEPSIA OF THE MIDDLE-AGED.

In conclusion, I would say a few words about the surgical dyspepsia of late middle-age. Here there is only one essential—namely, to disprove the possibility of cancer before any treatment is instituted. This is sometimes very difficult, but failure to do so leads to the disaster of inoperability, which one sees too often in abdominal cancer. The cases one must be most suspicious of are those where symptoms occur for the first time after the age of 50, and in all cases the onset is most insidious. We are not yet in a position to state, even approximately, the life-history of cancer, but I think we shall find eventually that it is much longer than is generally supposed, and that there is a premonitory stage marked by symptoms which at present elude us. We have long suffered under the tyranny of the dead-house, and our textbook symptomatology is that of the terminal stages. Take, for instance, a subject germane to our present inquiry, that of cancer of the stomach. We are still asked to make our diagnosis on certain symptoms—wasting, vomiting, pain, and a tumour—which are all late symptoms, and which, if one only knew the early symptoms, should never occur. We are beginning to recognize some of the early symptoms of cancer of the stomach, of which one may enumerate loss of appetite, loss of energy, a heavy feeling in the epigastrium affected by food, flatulence, and belching—all symptoms which precede pain and loss of weight. In the same way cancer of the colon should no longer be diagnosed by pain, loss of weight, and signs of obstruction. There is a preliminary stage of loss of energy, and variations in the accustomed regularity of bowel evacuations, which might make one suspicious and lead to a minute investigation. Cancer of the pancreas, again, is rarely diagnosed before the occurrence of jaundice. There is only one remedy for this, and it lies in the hands of the family doctor. What we need is a series of cases, collected by general practitioners, of malignant diseases proved by operation, in which all the incidents, however trivial, for which the doctor has been consulted for two or three years before the known onset of cancer are reported and analysed.

Suppose, now, you have a case of cancer of the stomach. You look up the patient's history. You find that, three years before, he came to you saying that he had had a little indigestion. He had, perhaps, been working over his desk, and thought he had got a bit of cramp. A little later he turns up with a story that he has been eating something that disagreed with him, and had had a poor night with epigastric pain. A few months later he says he is not feeling "quite the thing," and could you give him a tonic? Or take a case of cancer of the colon. You find in your notes that perhaps, two years before, the patient had come to you saying that, although he had always had a regular action of the bowels once a day, it had become irregular, and he had had to take a mild aperient. A few months later he turns up saying: "A funny thing happened to me last night. I got a pain in my stomach, and found I was full of gas. It did not last long, but it upset me." And, later still, he complains of occasional diarrhoea, or increasing constipation, and there is another request for a tonic.

If cases were tackled in this way, there would gradually arise, and, I hope, creep into our textbooks, a knowledge of a series of minor incidents which are common to cancer cases, and from which we might learn enough to make us watchful and apprehensive of the occurrence of cancer. We could then adopt those methods of minute investigation which we rely on to detect the early signs of disease.