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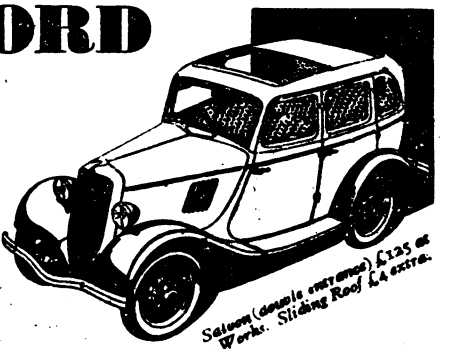

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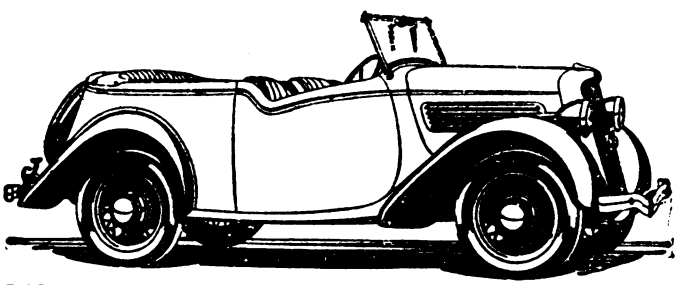

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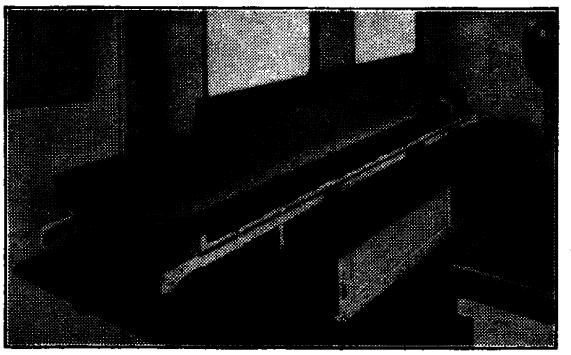

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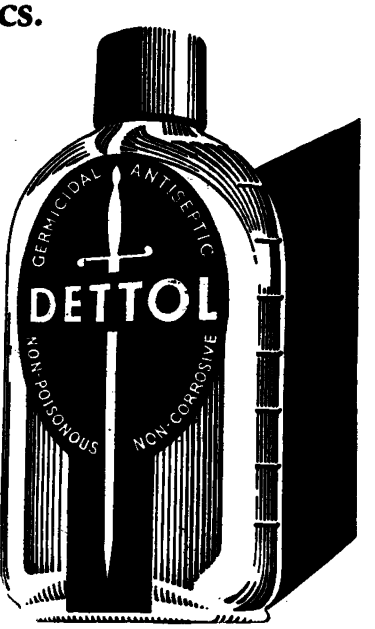

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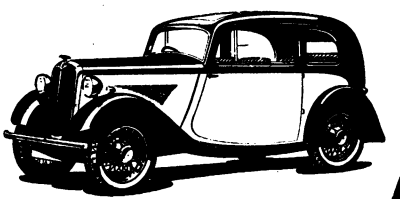

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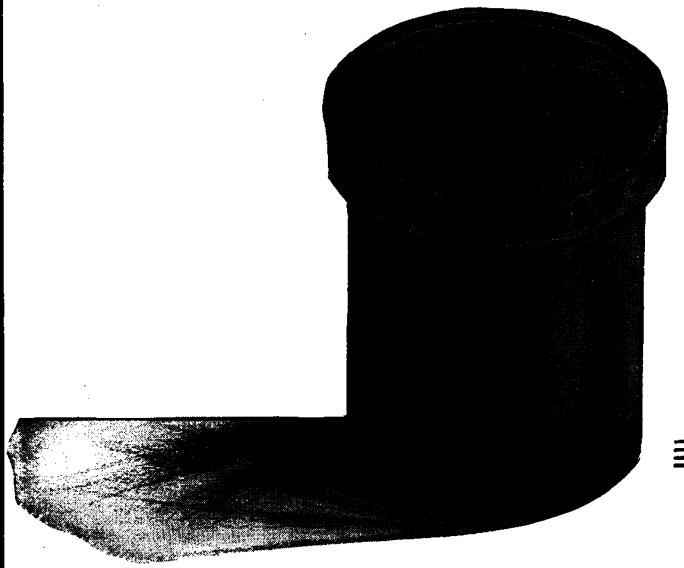

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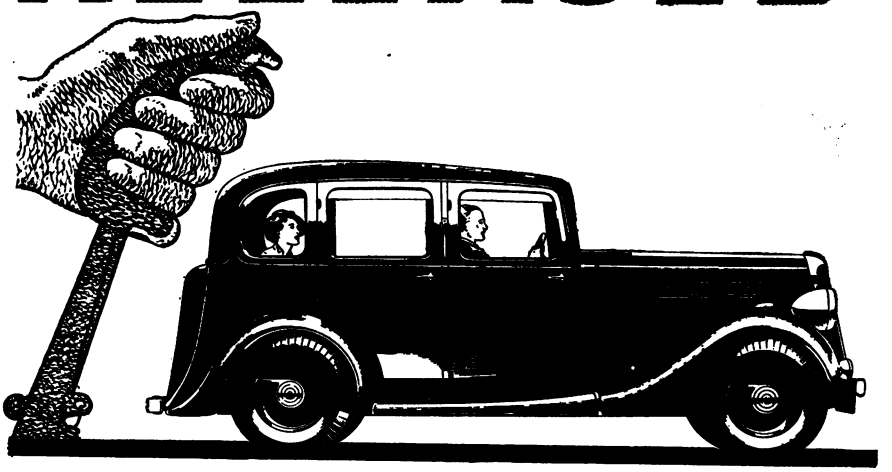

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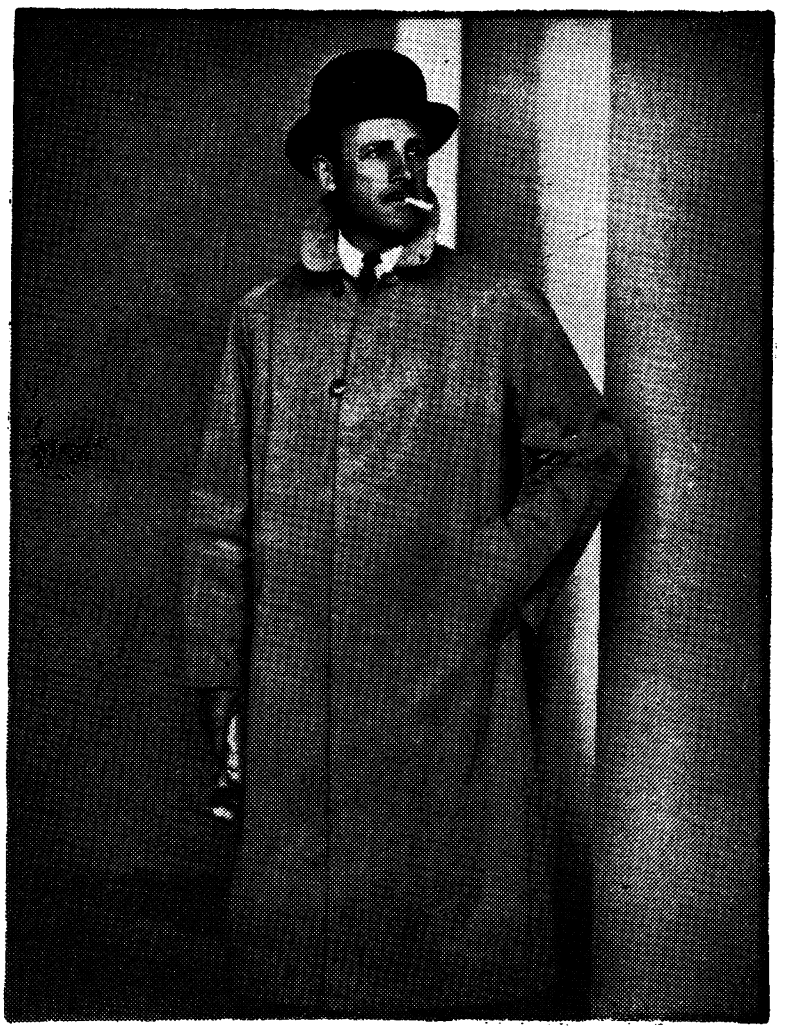

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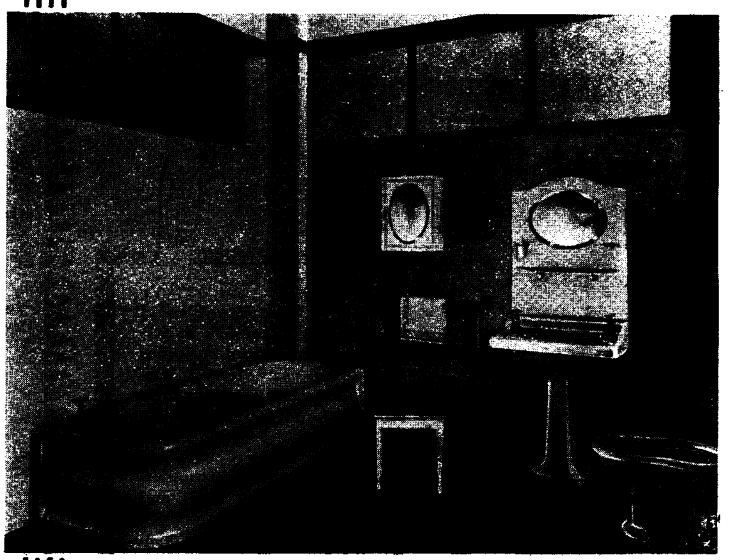

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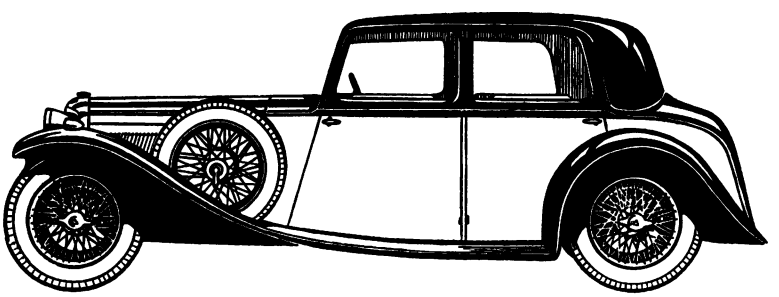

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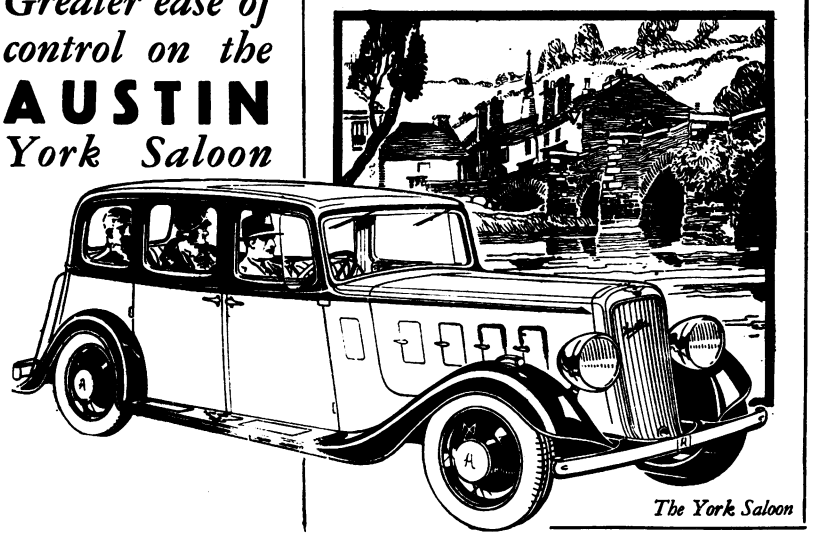

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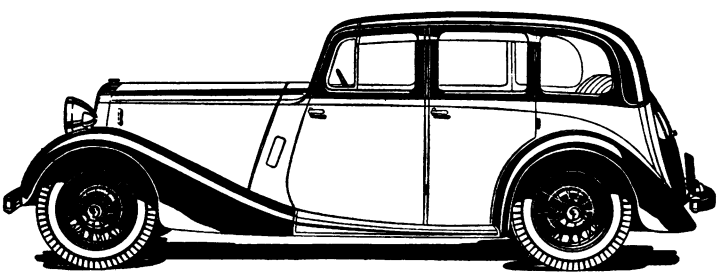

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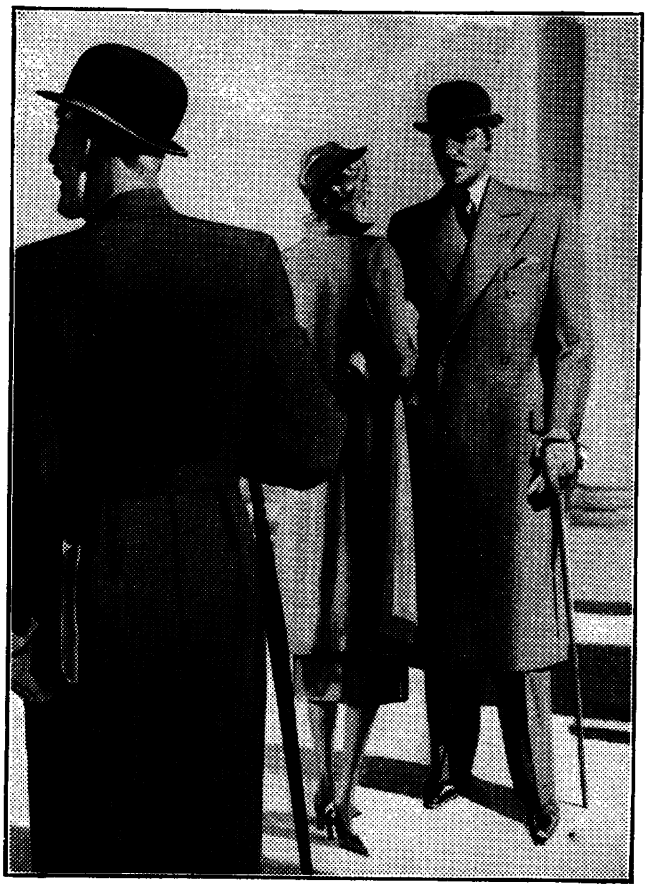

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THE
ULSTER MEDICAL JOURNAL

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VOL. IV 1st OCTOBER, 1935 No. 4

Forty Years of Gastric and Duodenal Ulcer from the Surgeon's Point of View

By S. T. IRWIN, B.A., M.B., M.CH., F.R.C.S.EDIN.
Honorary Surgeon, Royal Victoria Hospital, Belfast.

THE DAWN OF ABDOMINAL SURGERY.

LISTER'S historic discovery of the Germ Theory gained universal credence towards the end of the nineteenth century. Its acceptance marked the dawn of surgery as we know it to-day. The new principles upon which it rested were not equally applicable to all parts of the body, but the abdomen was the region most affected. Anatomical and physiological considerations stood in the way of the adoption of extensive operative procedures within the cavities of the skull and the thorax — obstacles which have been swept away by the intensive study of these areas both during the Great War and since its termination. From the outset, however, the surgery of the abdominal cavity was relatively easy; the problems demanding solution were plentiful and urgent, and the pathological lesions disclosed by laparotomy not obscure. Hence progress in this sphere was rapid and considerable. It is true that even before this time the existence of gastric and duodenal ulcer had been recognized. It was never dreamt, however, that such ulcers were as widespread as they were found by the operating surgeon to be, nor was it imagined that they could be the common cause of that type of "ordinary" indigestion which every family doctor and many housewives relieved, but rarely cured, by the daily administration of teaspoonful doses of bicarbonate of soda. Hitherto the conception of ulcer had been gained from such extreme examples as had from time to time failed to escape notice in the post-mortem room. In 1904, when a surgeon wrote "that hyperchlorhydria spelt ulcer," the remark brought down a terrific storm of abuse from the orthodox physicians of the time. In brief, the living pathology of ulcer with its associated symptomatology was only discovered when abdominal operations were rendered so safe that they became a matter of daily routine. Within a short space of time appendicectomy, operations on diseased gall-bladders and on tumours of the stomach and bowels, provided the daily adventures of surgeons in

every town, and most villages as well, and these furnished a clinical background upon which the outline pictures of ulcer of the stomach and duodenum were sketched. For the purpose of this communication, the intervening period of the forty years since Lister's time may be conveniently, if roughly, divided into four decades, each characterized by a distinctive outlook upon the problem presented by ulcers of the stomach and duodenum.

FIRST DECADE.

During the first decade, extending from 1895 to 1905, the operative surgery of the stomach and duodenum was tentative and experimental. It began by an attack upon the abdominal emergency arising from an ulcer which had suddenly perforated. The operation itself was of limited extent, consisting of a rapid closure of the perforation so as to prevent the discharge of the gastric or duodenal contents into the peritoneal cavity. The success of this operation suggested an operation for the prevention of perforation, and surgeons asked themselves why an ulcer, diagnosed on clinical grounds, should ever be allowed to perforate. Later on it was discovered that pyloric obstruction was the result of cicatricial contraction at the site of an ulcer, either of the duodenum in its first part or of the pyloric end of the stomach. Fatal hæmorrhages from the bowel occurring without other symptoms were found to be of duodenal origin and due to a pre-existing ulcer. And so the study of a subject then new began with the surgeon demonstrating the morbid anatomy and correlating it with the symptoms and signs. In Britain the van of progress was led by Mayo-Robson and Moynihan at Leeds. Newcastle-on-Tyne was early in the field owing to the co-operation of Drummond (physician) and Rutherford Morrison (surgeon), who anticipated a notable combination to become universal twenty years later. In a short time Maylard in Glasgow was writing extensively on duodenal ulcer, its symptoms and treatment. London lagged a decade behind the provincial centres, and for the first time lost its commanding position in the surgical world of these islands. It was not until 1904 that Hurst, after a visit to Leeds, began to diagnose duodenal ulcer with the help of radiology, but it took several years for the metropolitan physicians as a whole to waken up to the dramatic changes which were occurring in the attitude of the medical profession to peptic ulcer as the chief cause of indigestion.

Our Belfast School attained at an early date an outstanding position in dealing with the ulcer problem, owing to the influence of the abdominal work of A. B. Mitchell. As early as 1903, the year in which the present Royal Victoria Hospital was opened, Mitchell published his first series of twelve cases of perforation. After three failures—then, as now, due to delay in diagnosis— he had eight successful operations in his next nine cases, a truly marvellous accomplishment at a time when operation was agreed to only as a last resort, and physicians with scanty knowledge of the new pathology were doubtful of their powers of diagnosis and sceptical of the claims of surgery. This was followed by a second paper on eighteen cases of chronic gastric ulcer treated by operation, by a third on inefficient operations for gastric ulcer at the Oxford meeting of the British Medical Association, and by a fourth on "Perforative Duodenal Ulcer," read at the British Medical

Association meeting in Belfast in 1909, a record of sixteen consecutive operations without a death. A fifth paper followed in 1911 on 110 consecutive operations for gastric and duodenal ulcer. Though the numbers of cases may nowadays seem small, the papers described with Mitchell's characteristic lucidity his pioneer work in a branch of surgery still in its earliest infancy. His results were not excelled by those of any other surgeon of that time, and they gained a high reputation both for the operator himself and for the Belfast School of Surgery. In estimating their worth, it should be remembered that most of this early work was accomplished without the help either of radiology or biochemistry, now rightly regarded as essentials in the preliminary investigation of all but the most urgent of gastric and duodenal problems.

SECOND DECADE.

But we are anticipating, for Mitchell's last papers take us into our second decade.

It must be admitted that during the early years of the century an accurate knowledge of the history, the symptoms, the signs, the pathology, and the complications of ulcer had been acquired and had become the stock-in-trade of every surgeon doing abdominal work throughout the country. The general practitioner, also, who had seen a perforation operated on, could not fail to be impressed by the procedure, and eagerly looked out for his next case. Only the consulting physician failed to keep pace with the rapid progress of the subject, and failed to recognize in its true perspective a pathological entity which for several years remained outside his personal experience.

The second decade (1905-1915), therefore, found the surgeon in complete control of the diagnosis and treatment of ulcer, both of the gastric and duodenal variety. His daily work included the treatment of the dramatically sudden emergencies of perforation, the ulcer whose base had invaded a right gastric or gastro-duodenal artery, the chronic ulcer of long standing producing pyloric obstruction, and the large chronic ulcer adherent to the pancreas or liver, as well as the ulcer producing an hour-glass contraction in the body of the stomach. Thus there was great variety in the problems which taxed the surgeon's ingenuity and dexterity from day to day. It was only to be expected that no single operative measure could be generally applicable, and hence operations of great diversity were devised during this decade to meet the varying demands which were due directly to the presence of ulceration. Technique had to be developed gradually as operative experience was built up and as the results were assessed by the method of trial and error. The actual steps in this process need not delay us—through perforations to pyloric obstruction, and through the small gastric ulcer in the lesser curve to the large chronic perforating ulcer with its base wholly formed of pancreas. The result of simple closure of a perforation by the surgeon often gave an immediately good result, and rendered a patient free from pain for the first time for years. This was naturally followed by the infolding of an ulcer not yet perforated. Occasionally such a simple surgical procedure met with wonderful success, though many patients subsequently developed obstruction at the site of the suture, as did also some of those operated on for acute perforation, whilst in others the operation afforded no relief, because the symptoms

in the first instance were caused not by the anterior ulcer, but by the presence of a deeper one of the "kissing" type on the posterior surface of the affected viscus. Failure after infolding of the ulcer suggested the more radical treatment by excision, but this too was found insufficient to give permanent relief. In fact, Mitchell showed that an ulcer properly infolded disappeared and left a scar similar to that which followed excision. And so to the local treatment of ulcer had to be added some other manœuvre, either to deal with the local condition or to prevent mechanical obstruction. Where the ulcer was situated in the pyloric region, excision with end-to-end anastomosis, following the method already used for pyloric carcinoma, achieved a double purpose by getting rid of the local lesion and at the same time establishing the continuity of the intestinal canal. This operation became known subsequently as Bilroth No. 1. Technical difficulties, since overcome, made this a risky procedure, and carried at that time a prohibitive mortality. In a comparatively brief space of time, therefore, it gave place to the operation of gastro-enterostomy, which, as time went on, became established as the most generally applicable, and the most successful of all additions to the local treatment of the ulcer itself. The first gastro-enterostomy was performed by Wœlfler in Bilroth's clinic in Vienna in 1881. It was of the anterior variety, and was done for an irremovable cancer of the stomach, and was suggested to the operator by Nicoladoni, a bystander in the operating theatre at the time. Bilroth was evidently impressed, for he himself did the second gastro-enterostomy four days later, also for carcinoma, and also by the anterior route. Gastro-enterostomy was therefore not primarily suggested for the purpose of dealing with simple ulcer. The first *posterior* gastro-enterostomy was performed by Bilroth in 1885, and is now generally known throughout the world as Bilroth No. 2. The chief mechanical advantage of this method arose from the close proximity of the proximal end of the jejunum to the most dependent part of the posterior surface of the stomach, the two cavities being separated at the critical site of the anastomosis only by the transverse mesocolon. This relative position of the cavities allowed a short circuit to be effected without tension or kinking, and after various minor variations in site, size, and direction, the procedure has, more than any other single factor, revolutionized gastric surgery. The anastomosis in its earliest times was effected by decalcified bone plates (Senn), by bone bobbins (Mayo-Robson), or by the metal button (Murphy), but quite early in the century suture methods had replaced all these mechanical accessories. To summarize the progress of the study of ulcer during our second decade, it may be said that the surgical treatment of ulcer became firmly established, and that operation was performed at whatsoever stage the ulcer happened to be when the patient sought relief. In the light of our present knowledge, it was only to be expected that many failures should follow such methods, either from faulty diagnosis of the original lesion or from the occurrence of sequelæ, many of which can nowadays be anticipated.

THIRD DECADE.

The fateful years of the World War overlapped the closing years of our second decade and the opening years of our third. The demand for soldiers suddenly threw

a great floodlight upon every cause of physical unfitness. After twenty years of operative treatment of ulcer, the war found many men who had been operated on still of the age for service, as it also disclosed the astonishing frequency of ulcer, especially of the duodenal type, in men otherwise physically beyond reproach. The State in its extremity demanded men fit for full military service, and found these two groups who failed in the crucial test of fitness — first, the ulcer case not previously recognized as such, who, under the hard life either of military training or of service, found it impossible to carry on, and second, the ulcer case who had already been through the surgeon's hands and who was suffering from one or other of the sequelæ which follow in a certain proportion of cases operated on. Both these types underwent the strictest investigation at the hands of the physician, the surgeon, the bacteriologist, the biochemist, and the radiologist. Such examination, if it did not succeed in solving the problem of ulcer, showed the valuable part which each of these agencies in turn or in combination would be required to play in the final investigation of the problem, and established the modern method by which it was to be attacked. A certain divergence of views arose between physician and surgeon which has not even yet been settled, though a considerable measure of agreement has been reached. To the physician for a time it seemed that Rosenow, by harking back to the germ theory, had solved the problem of the cause of ulcer by discovering a specific micro-organism. His followers held that in any particular case it was only necessary to find the focus of infection—tooth, tonsil, appendix, etc. To deal effectively with this was to cure the patient. Hence for a time the physician and bacteriologist claimed control of the sufferer from ulcer. The results attained along these lines, however, were doomed to disappointment, though the physician *did* find out during this period that if eradication of sepsis were combined with rest, lavage, alkalies, and suitable diet, symptoms due to ulcer might be controlled, and in certain cases a potential cure could be effected.

On the other hand, the surgeon pointed out in this, our third decade (1915-1925), that many cases of ulcer failed to respond to medical treatment, and continued to suffer from pain, hæmorrhage, or obstruction, and that with the methods of operation in vogue, a considerable percentage of cures, often as large as eighty per cent., might be expected from operation—a predominating proportion of successes for an operation so radically altering the normal physiology of so important a function as that of digestion, and, moreover, that the cases least affected by medical means (obstructive) were those most suitable for surgery. In regard to the failures, the surgeon claimed that some of these were due to an operation being performed where there was no ulcer, whilst in cases of ulcer the causes of failure were now well established and could be avoided. They arose, first, in cases where through a failure in technique the stomach by emptying too quickly caused discomfort by distension of the proximal jejunum; second, some cases of the rapidly emptying type associated with achlorhydria developed anæmia; but, third, every surgeon was prepared to admit that the junctional ulcer was the real blot on gastric surgery and the chief cause of incapacity following operation. As time went on, the surgeon found himself more and more capable of selecting cases suitable for operation and rejecting those which were likely to be followed by serious symptoms.

FOURTH DECADE.

We have now reached the last ten-year period (1925-1935) of our subject. For twenty years treatment was exclusively surgical. During the next ten years physician and surgeon worked in co-operation. As a result, great strides were made in the methods of preliminary investigation, and the results of complete examination to a greater and greater extent determined treatment. The pathological lesions themselves had also altered. Whilst there were fewer of the huge ulcers of which the patients complained in the early years of the century, there appeared to be no obvious abatement in the numbers suffering from hæmorrhage or perforation.

As to the cause, we must admit at once that the cause of ulcer still eludes the student of gastro-enterology. Many interesting theories have been put forward to account for its occurrence. It may be that ulcer is the price we pay for civilization—a lagging behind of the power of adaptation in the digestive organs. McAllister of Liverpool took the view that it was a sign of atavism, and professed to find other evidences of individual maldevelopment in patients affected with ulcer. Fatigue, physical and nervous strain, faulty diet, and sepsis have all been considered as fundamental causes and given up. The recent experimental work of Dr. John Beattie, a distinguished student of this School, on central stimulation of the vagal centre in the brain, holds out the prospect of demonstrating a cranial rather than an abdominal cause for ulcer. Until the actual cause has been proved beyond dispute, prevention, the ultimate aim of medicine, cannot be expected.

If the cause of ulcer was still unknown, much had been learned of the symptoms, pathology, and treatment within the space of thirty years. Even a brief abstract of the facts and factors dealt with in the innumerable papers on the subject would not be possible within the limits of a single article. We do know, however, that whilst many individuals are prone to the disease, some suffer from it in an aggravated form. The writer found in the pyloric region of a stomach which he excised no less than twenty-eight ulcers or scars of ulcers—an indication both of Nature's power of repair (the physician's sheet-anchor) and of an unusual disposition towards the production of ulcer. Another example was that of duodenal ulcer in a boy of fourteen, verified by operation, who has since been operated on three times for perforating jejunal ulcer.

We know that no age excludes the possibility of ulcer as a cause of indigestion, though the ulcer of early life—under 20—is invariably duodenal. The writer's youngest patient in whom a duodenal ulcer was confirmed by operation was nine years of age. At least a dozen cases of perforated duodenal ulcer have been operated on at the Royal Victoria Hospital between the ages of 14 and 20 within the last ten years. At the other extreme, I have found a perforated duodenal ulcer at the age of 81.

Possibly the greatest advancement in the study of abdominal complaints made during the years since the War lies in the methods and results of diagnosis. These have succeeded in showing the great variability in the signs and symptoms due to ulceration, or rather, what is perhaps more important, on the various abdominal conditions whose symptoms simulate those of ulcer. A detailed account of the

methods used in the diagnosis of abdominal diseases will be given by others in the present issue of this Journal, and will not therefore be alluded to here. A few of the common causes of error will, however, be mentioned.

1. Cases of achlorhydric anæmia in an early stage—a not infrequent cause of chronic indigestion—can now be definitely and rigorously proven by the presence of achlorhydria combined with glossitis and characteristic blood-picture. These no longer run the risk of exploratory laparotomy, and in general, with few exceptions, it may be said that achlorhydria excludes ulcer—either primary or secondary.

2. Tuberculous lymphadenitis within the abdomen also gives rise in many cases to pain or discomfort after meals. A careful clinical examination discloses marked difference from those of ulcer — enlarged glands in the neck, axilla, or groin, absence of night pain, and tenderness over the ileo-cæcal angle—but many of these are still subjected to operation through uncertain or mistaken diagnosis.

3. Visceroptosis, especially that affecting chiefly the stomach, is a common cause of indigestion. Pain and tenderness are referred to the epigastrium. A six-hour residue found in the stomach may be attributed to organic pyloric obstruction by the radiologist, and may suggest the necessity for some sort of short-circuiting operation.

4. There is one other condition which for want of a better term may be named functional dyspepsia. It probably gives more trouble, both in diagnosis and in treatment, than any other single lesion. It may be associated with epigastric pain one to three hours after food, may wake the patient at night, may be relieved by alkalis or food, may show a high acid curve, and the through-going meal may leave a six-hour gastric residue. One, several, or all of these may be found in any given case, and yet at operation no organic lesion discovered. The physician engaged in their investigation finds them lacking in co-operation, or even contrary, and many on their own initiative or on medical advice seek surgical aid. Many have had one or more abdominal operations, and in the past some have had the futile operation of gastro-enterostomy. The mechanism of the production of the symptoms in such cases is unknown to me. The similarity of the symptoms, however, and many of the signs—radiological, biochemical, and physical—suggest that when the primary cause of ulcer has been finally determined, it will be found that the cause of functional dyspepsia also is closely allied to it.

5. When gall-stones, cholecystitis, and chronic appendicitis have been added to the above, we find a formidable list of conditions mistaken for ulcer.

In the last ten years all these and other more obvious diseases, owing to careful diagnosis on the part of the physician, have suffered less frequently than formerly from unnecessary, futile, or harmful operations.

After due examination of a patient, and having come to a diagnosis of ulcer, we still find widespread disagreement upon the question of treatment. There are still some physicians and surgeons who take extreme views of what either one or other may be capable of achieving in the treatment and cure of peptic ulcer. Most reasonable practitioners nowadays, however, physicians and surgeons alike, will be willing to admit that in all cases the first thing to obtain is an exact diagnosis.

The surgeon, when asked to advise in a case of suspected ulcer, will require answers to the following questions from the physician, to whom all such cases should in the first instance be referred :—

1. *Is an ulcer present?* Are the symptoms of which the patient is complaining due to ulcer, and can we exclude achlorhydric anæmia, visceroptosis, tuberculous lymphatic glands, functional dyspepsia, chronic disease of gall-bladder and appendix?

2. Where an ulcer is actually present, what is its position, its size, and its depth? What are its complications—severe pain, bleeding, or obstruction?

3. What has been the effect of one or more rigid courses of medical treatment followed by the best post-hospital care of which the patient is capable?

The answers to these questions fall to the lot of the physician, who calls to his assistance radiology, biochemistry, bacteriology, and hæmatology. If ulcer be found, and if medical treatment has been successful or moderately successful, and if the patient is satisfied with the results of such treatment, most surgeons will be willing to stand aside altogether. If, on the other hand, medical treatment has failed owing to the persistence of pain or of bleeding, or if the symptoms and signs of gross obstruction are still present, then the further management of the case must be surgical.

Surgical Treatment of Peptic Ulcer.

For many years the surgical treatment of most cases of ulcer, whether gastric or duodenal, meant a posterior gastro-enterostomy, with or without some local treatment to the ulcer lesion itself, such as infolding or excision with suture. No such simple and uniform solution of the problem will be accepted by the abdominal surgeon of the present day. What is the explanation of this change of view? Is it that the operation is inherently and essentially unsound, as some would have us believe? Few surgeons will admit that this is the true explanation, and it may be pointed out by way of proof of this statement that the operation is still extensively done, and that by means of it many patients have in the past been enabled to lead comfortable and even strenuous lives. The change of view is due to the fact that in the past there have been ghastly failures as well as brilliant successes following the operation of gastro-enterostomy. What have been the main causes of these failures, and what can be done to avoid them in the future? The causes of failure are threefold :—(1) In the past the operation has been done in many unsuitable cases—for example, where a functional spasm of the pylorus was mistaken for an organic obstruction. Even when ulcer is present, all types are not equally suitable for treatment by gastro-enterostomy. (2) The operator has been guilty of a technical error in the performance of the operation. (3) The operation has been followed by one or other of the sequelæ which are now known to follow gastric operations in general; these are regurgitant vomiting, microcytic anæmia, and jejunal ulcer.

With such failures and their causes in his mind, how does the modern surgeon face the problem? Can he still retain the successes of gastro-enterostomy and at the same time escape its pitfalls? Having excluded all cases of ulcer curable by

medical means alone, he will in the first instance draw a sharp line of distinction between the ulcer of the stomach and that of the duodenum.

GASTRIC ULCER.—The majority of operating surgeons now hold that gastro-enterostomy has often failed to afford relief in uncomplicated cases of gastric ulcer. For the ulcer near the pylorus with marked obstruction it is still extensively used and is a sound surgical procedure. On the other hand, for ulcer situated in the body of the stomach, the operation of choice, whether the ulcer be large or small, is a partial gastrectomy after the method of Polya or Moynihan. The occasional occurrence of carcinoma supervening on simple ulcer of the stomach affords an additional argument for this type of operation. Walton still holds, however, that for a small ulcer on the lesser curve—a very common lesion—excision of the ulcer with a posterior gastro-enterostomy gives the most satisfactory result. For the large ulcer of the chronic perforating type, he too would admit that a partial gastrectomy has surpassing advantages. In this operation the site of the ulcer will be removed. Removal, however, is not always possible owing to the close proximity of the ulcer to the œsophageal opening of the stomach. Some years ago (1928) Lake pointed out the value of a gastrectomy in which the ulcer, owing to its high position, was left behind. Many surgeons will bear witness to the phenomenal success which has followed this apparently unsound procedure.

DUODENAL ULCER.—The surgical treatment of duodenal ulcer to-day provides the battlefield upon which we find the foremost abdominal surgeons arrayed in opposite camps. Some physicians are not unwilling to rush into the fray and express opinions on a technical problem. All, however, are agreed that medical treatment, both rigid and prolonged, and if necessary repeated, should precede any thought of operation. With many patients, adequate medical treatment is no simple matter, minute directions are required from the physician and extreme diligence and patience on the part of the sufferer. Most practitioners who have to supervise the carrying out of the treatment will agree that in some cases pain persists or recurs, bleeding continues or obstruction develops, and the patient, even after a prolonged course of dieting and drugging, is still compelled to lead the life of an invalid. In such a predicament is there any hope of escape for the patient, and what surgical methods are at the disposal of the operator? They may be subdivided into two groups — (1) a posterior gastro-enterostomy, gastro-duodenostomy (Kocher), or pyloroplasty (Finney), and (2) a high partial gastrectomy. Gastro-enterostomy has been the operation of choice in this country for years, and its success has allowed very many persons afflicted with duodenal ulcer to lead active lives very little below the normal. Gastro-duodenostomy claims increased immunity from the terrible complication of jejunal ulcer. Pyloroplasty has had its chief advocates in America, owing to the skilful operative technique of its designer—J. T. Finney of Baltimore. Neither of these variations, however, has succeeded in attracting universal support in this country.

Gastro-enterostomy owes its vogue to its simplicity, its low operative mortality, and its conspicuous success in suitable cases. Only recently has it been seriously challenged in this country, mainly by the physicians, who have had to deal solely

with the failures of surgery and have no experience of the successes. Naturally, therefore, they have been struck with the frequency of jejunal ulcer as the outstanding cause of the failures, some of which have occurred after long periods of immunity. A few Continental surgeons, especially Finsterer and von Haberer in Germany, have reported that, having considered the causation of complications, they have come to the conclusion that the only logical way of preventing these is to remove completely that part of the stomach and duodenum in which ulcer is found. They have therefore wholly given up the operation of gastro-enterostomy for duodenal ulcer, and have replaced it by a high gastrectomy. In this country their views on gastro-enterostomy have been adopted and advocated strongly by Ogilvie, who doubts the value of the statistics published by those surgeons who claim a high measure of success by this popular method of short circuit. Severe criticism of the Continental views, on the other hand, has been offered by Moynihan, Patterson, Walton, and others, who, whilst willing to admit the occasional occurrence of secondary ulceration after gastro-enterostomy, reaffirm its low operative mortality and the large proportion of successes which it has already given. They also point out that where partial gastrectomy has been tried, as it has been in many cases of jejunal ulcer following a gastro-enterostomy, it has not wholly freed the patient from the danger of a further secondary ulcer arising in the jejunum adjacent to the divided end of the stomach. It must be admitted, however, that in such cases there is probably an original high tendency to ulceration, and the operation has not been of the extensive variety suggested by Ogilvie and the Continental surgeons. Time may prove that the amount of stomach removed may be the critical factor in this operation as applied to an ulcer of the duodenum in contra-distinction to that of the stomach.

The careful surgeon of to-day will probably adopt a *via media*. He will strongly disagree with Finsterer when he says that gastro-enterostomy has had its day and that in future it will have no place in the treatment of duodenal ulcer. He will also probably disagree with those who regard it as an operation of general applicability. He will use it for the ulcer of chronic type in the elderly patient in whom obstruction is the main feature. Such cases have been successful in the past, and few have suffered from the scourge of jejunal ulcer. On the other hand, where the danger of jejunal ulcer cannot be disregarded, for example in the young patient with ulcer of irritable type, with high acid curve, rapidly emptying stomach, and without obstruction, he will if possible avoid operation altogether; but if in the end, owing to pain or bleeding, he is forced to operate, he will welcome the method of Ogilvie in the hope that it will afford him and his patient a means of escape from the tragic complication of junctional ulcer, without increasing the tendency to a deficiency disease, of which microcytic anæmia is the outward and visible sign. If he obtains a result comparable to that of partial gastrectomy for gastric ulcer, both the patient and the surgeon will have just cause for thankfulness. The method will, however, have to stand the test of time before it is finally accepted without reservation as the only surgical procedure capable of adequately dealing with the problem of duodenal ulcer.

Radiology in Chronic Indigestion

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ON a subject such as this, it is very difficult to know where to start and on what lines to proceed. The scope is immense; it covers the whole field of the X-ray examination of the alimentary tract, and encroaches on that of various other abdominal organs and other regions.

W. J. Mayo has said, "Only one person in ten with gastric symptoms has a gastric lesion."[1]

Symptoms of chronic indigestion may be produced by lesions of many other organs. These may be abdominal, and even if we group the duodenum with the stomach, they may be produced by a gall-bladder lesion, by a chronic appendix, by mesenteric adenitis, by lesions of the spleen, liver, pancreas, or urinary tract, or even by lesions of the colon. On the other hand, they may be extra-abdominal, and cardiac disease, aneurysm, pleural, pulmonary, or mediastinal lesions may be the causal factor.

All of these possibilities must be borne in mind. In the diagnosis and localization of the cause, whatever it may be, radiology plays an ever-increasing part, and in close co-operation with the clinical investigations must be directed along the various lines indicated, in search of the cause of offence.

I do not propose, in an article such as this, to do more than touch on radiographic technique and the methods employed in the examination. That is of interest only to those engaged in this work.

I wish to discuss what practical aid the radiologist can offer the clinician in investigating a case of chronic indigestion, how this assistance can be best made use of, what are the powers and what the limitations of radiology in such a problem, and what is the correct outlook of the clinician towards radiology when he calls on its help.

When we think of radiology in chronic indigestion, we naturally think first of the "opaque meal examination."

The discovery of X-rays, dating only from 1896, makes radiology a mere child among the sciences. The real advance in the radiology of the digestive tract, having been made within the last fifteen or twenty years, makes that special branch younger still.

It is true that, within a year of Röntgen's discovery, Strauss[3, 4] endeavoured to gain some knowledge of the passage of materials through the alimentary canal by administering gelatine capsules, made opaque by a filling of iron oxide and bismuth subnitrate, and in 1898 the physiologist, Cannon,[5] administered bismuth subnitrate to dogs, cats, and a few humans.

Other pioneer workers of the same period were Hemmeter, Becker, Roux and Balthazard,[6] Williams,[7] and Benedict.[8]

In those days of inefficient and uncertain apparatus, with feeble X-ray production

and crude photographic material, no satisfactory results were obtained, investigations on these lines ceased, and a "silent period" followed.

In 1905 a renewal of interest followed the work of Rieder of Munich,[9] who claimed that amounts of bismuth subnitrate, much in excess of the pharmacopœial dose, could be safely administered.

Owing to the inefficient apparatus still available at this date, the work was chiefly based on fluoroscopy, but many, especially on the Continent, pursued the subject vigorously, and early work was also carried out in this country, notably by A. F. Hurst,[12] Dalton and Reid,[11] and Thurstan Holland.[10]

Most workers in these early days employed bismuth subnitrate, but certain disagreeable consequences occurred. Deaths from nitrite poisoning were reported. It was looked on with suspicion, and bismuth carbonate was substituted.

The effect of the alkalinity of this salt on the process of digestion was questioned, and Hurst advocated oxychloride.[12] The cost of this salt precluded its extensive use, and it was then found that barium sulphate, specially prepared and guaranteed free from impurities, reduced the cost, and proved equally satisfactory. This is the salt in general use to-day, though some of the older workers still consider bismuth more desirable.

The development of more powerful generating plant, employing the properties of the high-tension transformer, by Snook in 1908, and the greatly increased photographic effect from the advent of the intensifying screen, furnished further facilities for demonstrating both the normal and the pathological appearances of the digestive tract, and from that date progress was rapid, and the direct detection of gastric and intestinal lesions, as distinct from their deduction from indirect signs, became possible. This direct method was at that date mainly pursued by American workers, chief among whom may be mentioned Gregory Cole.[13] The Continental school still held to the older indirect method.

The War interrupted the progress of research. Since then advances in technique, improvements in apparatus and photographic material, have given additional power to the direct method, and the most striking development of recent years has been the intensive study of the gastro-intestinal mucous membrane, by showing it streaked with barium, demonstrated by Baastrup[14] in 1924, a line of work with which the names of Berg[16] of Dortmund, Forssell,[17] and Ackerlund[18] of Stockholm are specially associated.

This work is still new, and much has yet to be learnt, but it opens up a possible field for the detection of early and slightly marked lesions, which are totally obscured in the examination of the completely filled organ, and promises to bring radio-diagnosis to a sphere of greater and greater exactitude.

For the material for this historical synopsis, and for the references, I am indebted to Dr. A. E. Barclay's book on "The Digestive Tract."

The history of radiology in the diagnosis of gastro-intestinal disease is a short one. Its growth in the time is remarkable.

In efficient hands the accuracy of diagnosis has reached an extraordinarily high level—so high, in fact, as to constitute a danger; for a tendency has grown up,

especially among the lay public, but even also in the medical profession, to look on the X-ray examination as the final court of appeal, and to throw full responsibility for the diagnosis on the radiologist. This may be complimentary to radiology, but it is a wrong conception.

While we may grant that the radiological examination is an important link in the diagnostic chain, we must never forget that it is only a link.

Its value is altogether dependent upon its evidence being taken in conjunction with the clinical, pathological, biochemical, and bacteriological evidence, and correlated with them.

Radiology shows only one side of the picture, that dealing with variations in gross outline and density. It deals with shadows and shadow defects as evidence of physiological and pathological changes.

As Dr. A. E. Barclay[17] has said : "It is like one piece of a jig-saw puzzle which must fit, even if other parts have to be rearranged to make it do so."

The crude and commonly held idea that because the radiologist can see right through you, he must be able to see exactly what is wrong, must be corrected.

Co-operation and consultation between the clinician and the radiologist are essential for success.

This is very difficult to obtain in busy hospital work, or even sometimes in private work, but it is an ideal to be aimed at, and to secure it certain duties devolve on both parties.

As a radiologist, I feel rather diffident in referring to the duties of the clinician, but from a lengthy experience of gastro-intestinal cases referred for radiological examination, I feel a few points might be mentioned.

It seems elementary and needless to say that he should first conduct a very thorough and complete clinical examination. It is futile to endeavour to use the X-ray examination as a short cut to diagnosis. Applications sent to the X-ray department such as "Abdominal pain, X-ray please," or the more simple "Opaque meal, please," not only give the radiologist no information, but tend to raise a doubt in his mind as to whether a real clinical examination has been made.

He should consider all his clinical findings together, possibly, with biochemical findings, and decide whether a radiological examination is likely to furnish assistance. Having decided on this, he should then furnish the radiologist with an outline of the history of the case, and all the clinical facts he has elicited which would bear on the X-ray examination and diagnosis, keeping to facts, and reserving his deductions therefrom till later.

In private cases there will probably be time for further details, and in this connection I would suggest that the salient facts should be given in writing—a few words spoken directly or by telephone, when making the appointment, being often very difficult to remember when the patient arrives.

When the X-ray examination is completed, the time has arrived for the clinician and the radiologist to compare the deductions each has drawn from the facts they have observed, and by correlating and co-ordinating these, arrive at a diagnosis.

I grant this is an ideal of perfection and very difficult to obtain in the rush of

routine hospital work; but I was greatly struck, when recently in Sweden, by the fact that in all the larger hospitals at a certain fixed hour each morning the clinicians and the radiologists met for a short time in the X-ray department and discussed all the cases of interest in the previous day's work.

The radiologist must prepare himself to be a valuable partner in this collaboration. He must try to develop a clinical outlook. Radio-diagnosis is a clinical method, and the radiologist to be efficient requires a knowledge of anatomy, physiology, and clinical medicine. Without this he becomes a mere machine for producing radiograms, and however beautiful these may be, they will be of little value without the interpretative skill which is based on real knowledge, supported by clinical and common sense.

The radiogram is the radiologist's physical examination. It is wrong to speak of an X-ray diagnosis. The radiologist sees an abnormality and gives his opinion on it, but that opinion is based on his clinical knowledge and previous experience. The wider his knowledge of clinical medicine, as well as of his own speciality, the greater will be his value in the sphere of diagnosis.

The extraordinary advances in recent years in the efficiency of X-ray apparatus and photographic material, with an accompanying simplicity of manipulation, while being of immense service to the radiologist, have not been without their drawbacks.

No longer are sound electrical knowledge and photographic experience, combined with endless patience, needed to produce radiographs of merit. Anyone with a little practice can, with a modern outfit, by following instructions, produce quite good films, but the ability to do this does not make a radiologist, nor is it by its use in such a way that radiology is of real value. Clinical diagnosis can never be machine-made.

I do not mean by this that the production of the radiograph is unimportant—this is far from the case.

The late R. D. Carman of the Mayo Clinic[1] summarized the position as follows : "The X-ray examination of the digestive tract is not a mysterious art requiring extraordinary talents, nor a simple diagnostic method learnt in a day. It requires industry, experience, judgment, and care."

The danger lies in the fact that the growing ease in the production of radiograms has tempted many to dabble in that work. This is encouraged by the instrument makers, whose profits naturally depend on sales, with the result that no hospital, however small, is considered complete without its X-ray apparatus, irrespective of the ability to make proper use of it.

An American writer, Edward H. Skinner,[19] has said : "The distribution of X-ray apparatus has far exceeded the distribution of the knowledge of Röntgen interpretation."

He states that the public has been allowed to believe that the possession of apparatus carries with it sufficient knowledge to make use of that apparatus. This outlook must be changed, and the profession and the public brought to realize that

the value of an X-ray examination depends entirely on the interpretative knowledge and clinical sense of the observer brought to bear on it.

A further error to be corrected is the expectation always of a definite diagnosis, tempting the radiologist to dogmatism. Radiology is all deduction—deduction from shadow changes, of changes in the organs casting those shadows. Clinical experience will play a large part in this deductive process, but even the most mature experience and skill is often unable to give a positive answer.

The X-ray examination is another clinical examination from a different standpoint, and the radiologist must approach his work, not as a photographer, but as a clinician employing certain mechanical aids and working on certain lines.

The pioneers of radiology were, by the nature of things, men who had previously been engaged in clinical work, and reaped the advantage of this early training by the clinical attitude which they brought to bear on their special work, an attitude more rarely found in the radiologists to-day.

It may be said, if clinical knowledge is so necessary, why should not every clinician be his own radiologist?

Nothing is so befraught with pitfalls as an approach to an examination with the mind made up and the diagnosis prejudged. A preconceived opinion inevitably leads to an endeavour to read into what is seen, what the observer thinks ought to be seen; and what he has found clinically, and deduced therefrom, is bound to influence his interpretation of the X-ray findings.

It is often difficult for a radiologist to be unbiassed by what he knows the clinician suspects. It would be infinitely more difficult for the clinician himself. In addition, the patient loses the advantage of a completely independent opinion working along different lines.

This is the main argument against this principle, but, in addition, a busy clinician cannot afford the time for the perfection of radiographic technique and for making a thorough radiological investigation, nor will he have the benefits of the wider experience gained by the specialist, who will naturally see radiographically a greater number and variety of cases.

Besides the danger of starting with a preconceived opinion, the radiologist must also guard against the danger of being rushed in his examination, either with the idea of saving the patient's time or by the eagerness of the clinician for results.

A gastro-intestinal examination requires care, patience, and adequate time, and its success depends on this. Hurry and a slipshod examination is a fruitful cause of error. I feel strongly that a man striving to fit in a number of cases in too short a time cannot give the necessary individual concentration to any of them, and all will suffer.

Such rushed work is often very difficult to avoid, even in private work with patients wishing to be done at once, but more so in hospital work, where the numbers sent for X-ray investigation of the digestive tract become overwhelming.

In the Manchester Royal Infirmary the opaque-meal work has always been confined to in-patients, on the grounds that to increase the work by including out-patients would inevitably diminish the efficiency of the service to in-patients, no

radiologist being physically able to satisfactorily examine more than a limited number of such cases; and further, on the grounds that the examination of out-patients is unsatisfactory, because they cannot be trusted to strictly observe the necessary preparation, they frequently fail to re-attend for repeated examinations, and they are generally sent for another examination when admitted to the wards.

In spite of the arguments that the examination of out-patients relieves the pressure on beds, and that hospitalization is not absolutely necessary, I believe this standpoint is sound. The bulk of routine opaque-meal and cholecystographic examinations on out-patients are of little real value. Hospitalization tends to secure full advantage of clinical, biochemical, and other methods of investigation which can be correlated with the radiological findings, and in the long run both clinical medicine and radiology are better served by having a few cases thoroughly examined, than a large number run through in a routine and incomplete way.

I know one distinguished radiologist who refuses to start more than one opaque-meal case per diem, and by so doing is able to concentrate his clinical and diagnostic faculties on that case, and keep it under frequent observation, avoiding the inevitable mental confusion of trying to keep clear the clinical and radiological features of several different cases at the same time.

The next point I wish to stress, and one strongly emphasized by Dr. Barclay,[2] is that the radiologist should have a definite routine for his gastro-intestinal work, and follow it always, making necessary diversions to meet individual indications. No one routine is correct. Each radiologist must plan his own to suit his individual circumstances. By holding to a fixed routine he has definite standards of comparison, and also is less likely, on finding a positive lesion, to miss another equally positive and perhaps of greater importance in some other part of the tract.

Having completed his examination and marshalled his facts, the radiologist has next to furnish a report.

This should embody all the facts observed on screening and in the radiogram, with his conclusions and deductions therefrom.

This is the time for collaboration between the clinician and radiologist.

The positive facts each has found, are facts, and must fit in. The conclusions each has drawn therefrom must be compared and, if necessary, re-arranged, to cover these facts.

In this way, and in this way alone, can the best use be made of an X-ray examination, and the patient's interests best served.

Facts and inferences are each given their proper weight. Those that are clinical and those that are radiological each acquire their due value, and the temptation to guessing is minimized.

To sum up, I would say to the clinician : "Make as complete a clinical examination as possible, utilizing also the services of biochemistry. Furnish the radiologist with all the salient facts observed and discovered. And to the radiologist : "Approach the case as a clinician, not as a glorified photographer. Make full use of all the clinical facts learnt, avoid a preconceived opinion, have a definite routine, and take time, avoiding rush and hurry."

To both I would urge collaboration, always remembering that they are colleagues approaching the same problem along different but converging lines. Each establishes facts, the deductions from which form the foundations of the diagnosis, which in most cases in the end is really an expression of opinion.

In undertaking the radiological investigation of a case of chronic indigestion, the clinical aspects must first be carefully considered, bearing in mind what has already been pointed out, that the cause may be gastric or duodenal, but that quite frequently mesenteric adenitis, a gall-bladder or an appendical lesion may show gastric symptoms, and that even a rectal lesion or a thoracic lesion may cause reflex epigastric pain.

Lord Moynihan has said: "The most frequent site of a gastric ulcer is in the right iliac fossa"; and a confirmation of this connection is the fact that quite frequently pressure over an appendix visualized on the screen, while producing no local pain, causes pain referred to the duodenal region.

The clinical findings must give guidance as to where to start in the radiological examination.

If anything points to the urinary tract, radiograms of that should be made before the area is masked by the shadows of the ingested meal. Such an examination will also reveal calcified mesenteric glands if present, with the inference of the accompaniment of non-calcified and still actively inflamed glands.

If this examination is negative and the kidneys still suspect, pyelography, either by the retrograde method of ureteral injection or by the intravenous method, may be necessary, showing the outline of the kidney pelves and ureters, revealing abnormalities of position, outline, or shape, and in the latter method also demonstrating the secretory activity.

On the other hand, clinical evidence may point towards the gall-bladder. If so, this area can be examined directly and also after the administration of tetra-iodophenolphthalein, which is absorbed from the alimentary tract and secreted by the liver, filling the gall-bladder and throwing a well-defined shadow—the method of cholecystography.

In the early days the demonstration of gall-stones radiographically was very unsatisfactory owing to their low degree of density, but with modern technique and modern apparatus results have greatly improved, and a good proportion of gall-stones may be shown in the direct film, or even thickening of the gall-bladder walls, or dense grumous material in a gall-bladder may be visible.

The density of some calculi is so low, however, that it is not possible to show them in this way. In this case they may be shown as "negative shadows" in a gall-bladder filled with the administered "dye" in a cholecystography examination.

This method also has the advantage of giving some information as to the secretory powers of the liver and the patency of the biliary channels.

The examination of the gastro-intestinal tract itself is made by the opaque meal method, or the opaque enema in the case of the colon. When administering the meal, some of it at least should be watched passing down the œsophagus, to

exclude any abnormality there, and a rapid screen examination made of the chest, followed by radiograms if any suspicious intra-thoracic shadows are seen, remembering the late Professor Lindsay's aphorism: "When the patient complains of the stomach, think of the heart; and when he complains of the heart, think of the stomach."

The stomach itself is then examined, at first preferably with only a small amount of the opaque meal, so as to visualize small alterations which would be masked if the organ were completely filled. Localized pressure is made use of, to visualize the mucous membrane rugæ, according to the technique of Berg and of the Swedish school. As well as seeing any deformity of outline or alteration in position of the organ, the radiologist can judge the muscular tonus of the stomach and visualize the strength and rapidity of peristalsis. Care must be taken to differentiate a transient spasmodic deformity from a permanent organic one, and careful and prolonged screening and occasionally the administration of a antispasmodic drug is necessary to settle this question.

When a definite ulcer niche filled with the opaque material is seen, or a typical "filling defect" caused by a new growth invading the lumen, the radiologist is in possession of very positive signs, but the diagnosis is often much less definite: only vague irregularities may be observed, variations in tone, in peristalsis, in movements. A diagnosis cannot be made on these indefinite signs alone, but often they are of extreme value when taken in conjunction with the clinical and biochemical findings.

The duodenum is next observed. Only the first part, or duodenal cap, remains filled with the meal, which normally passes transiently and rapidly through the second and third parts. Fortunately, the vast majority of duodenal ulcers occur in this first part.

In earlier days, much stress was laid on the so-called "indirect signs" of duodenal ulcer—hypertone of the stomach, hyperperistalsis and hypermotility of the meal at six hours.

Now with advanced technique less attention is pair to these signs, and the interest centred on the actual demonstration of the ulcer niche. Much work has been done on these lines by Ackerlund of Stockholm, who by careful examination of the duodenal cap under localized pressure, at various angles, has been able to demonstrate the actual ulcer in numbers of cases in which under the older routine methods it would have escaped notice.

Frequently, owing to spasm of the pylorus, it is a matter of considerable difficulty and prolonged examination to get a satisfactory filling and delineation of the duodenal cap, and palpation while screening with pressure over the pylorus, coincident with the pressure caused by a peristaltic wave, is often necessary to get the meal into the cap.

While a very large gastric residue at six hours points to an organic pyloric obstruction, less importance is granted to delayed motility now than formerly. Atonicity or a reflex spasm may be causes of delay, and a direct demonstration of the lesion is the ideal to-day.

Gastric or duodenal diverticula or the presence of a polypus may be causes of dyspeptic symptoms, and in a routine examination these should be revealed.

The commonest cause of chronic indigestion in the intestinal tract is an appendical lesion. Many signs have been suggested at different times, as radiological evidence of such a lesion, e.g., delay in the lower coils of the ileum, hypermotility in the colon due to cæcal irritability, filling of the appendix, non-filling of the appendix, delayed emptying of the appendix, etc. The value of all or any of these is problematical. The one sign of real importance is localized tenderness on pressure over a visualized appendix.

Whether the appendix becomes filled with the meal or not, is now granted by most to be a matter of no diagnostic significance, but if it is filled, its length and position can be seen, the question of its tenderness verified, and uneven filling due to contained concretions or to spasm of its walls observed. Its mobility on pressure also gives an indication as to the presence or absence of adhesions. Many still hold that an appendix which remains filled with barium after the contents of the cæcum have been evacuated is pathological, but others have reported cases where this was noticed even for several days after emptying of the cæcum, when operation revealed a normal appendix.

This cæcal delay will follow the presence of adhesions, but the only real radiological sign of trouble is tenderness closely localized, and the other signs are of very minor importance. For this sign we require a filled appendix, and, as I have said, in both normal and abnormal cases this does not always occur.

As a rule, more satisfactory filling is obtained by an opaque meal than by an opaque enema, but if one method fails, the other may succeed. Cases vary greatly, but as a rule the best time for seeing the appendix is about twenty-four hours after ingestion of the meal, when the cæcum has begun to empty. Small doses of magnesium sulphate before examination are said to increase the chances of filling the appendix.

Mesenteric adenitis with glands in the right iliac fossa offers the great difficulty in differential diagnosis. In this the affected area is wider and glands more distributed through the abdomen. Consequently tenderness is as a rule not so localized, and there is greater evidence of delay of the meal throughout the ileum, and not only in the terminal coils.

While a lesion of the colon is not a probable cause of chronic indigestion, it has been observed. A carcinoma of the colon or a diverticulitis may give dyspeptic symptoms.

Such a lesion is best demonstrated by an opaque enema. The passage of a meal through the colon varies greatly, and as a rule only portions of it are filled at any one time. The whole colon from rectum to cæcum can be evenly filled by the enema, any deformity of outline observed and diverticula shown, filled with barium after injection, and, better still, after evacuation of the colonic contents.

A further refinement of the opaque enema examination has been recently introduced, where the colon is inflated with air after evacuation of most of the enema, and the mucous membrane rugæ well shown up still coated with traces of barium.

This is a very brief outline of the scope of radiology in the investigation of a case of chronic indigestion. As I said at the outset, my aim has not been to deal at any length with the actual radiographic methods or technique, but rather to sketch what I consider is the radiologist's rôle in this work, how he can best assist the clinician, and how the clinician can gain most advantage from his work.

Co-operation must be the keynote. The X-ray examination is not a separate examination in a watertight compartment divorced altogether from clinical medicine, nor is it a final court of appeal. It is a clinical examination, guided as to its line of procedure by clinical indications, with its results co-ordinated and fitted in with those arrived at by the clinician. In capable hands its results are accurate, but those results are not capable at all times of making a specific diagnosis, though they may be of great value as additional evidence when reviewed in the light of the clinical and biochemical findings.

When mistakes occur, they are usually due to hurried or incomplete examination, without proper preparation of the patient, or to the bias of a preconceived opinion.

Radiology in chronic indigestion is only one link in the diagnostic chain, but I think we can claim that it is a very important link.

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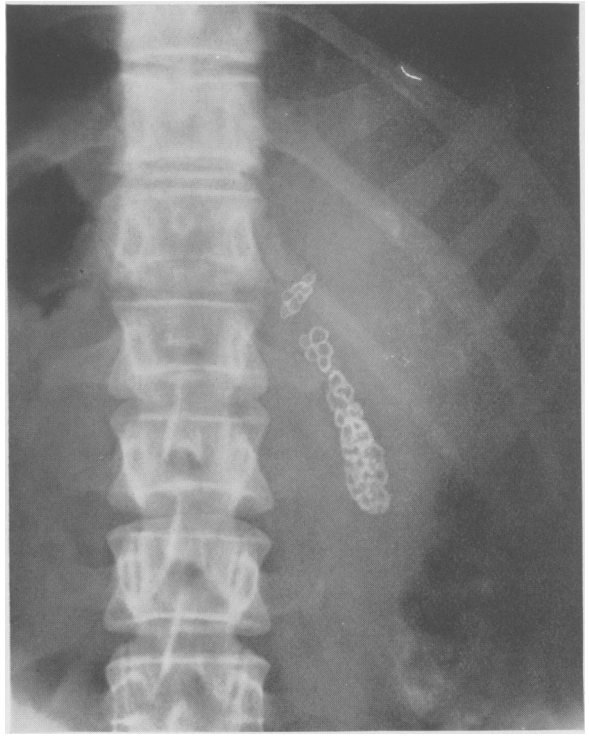
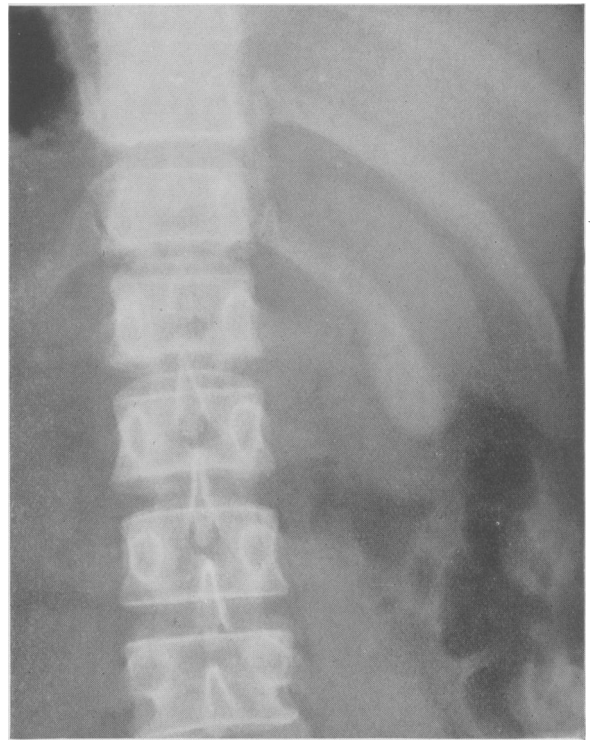
(1)



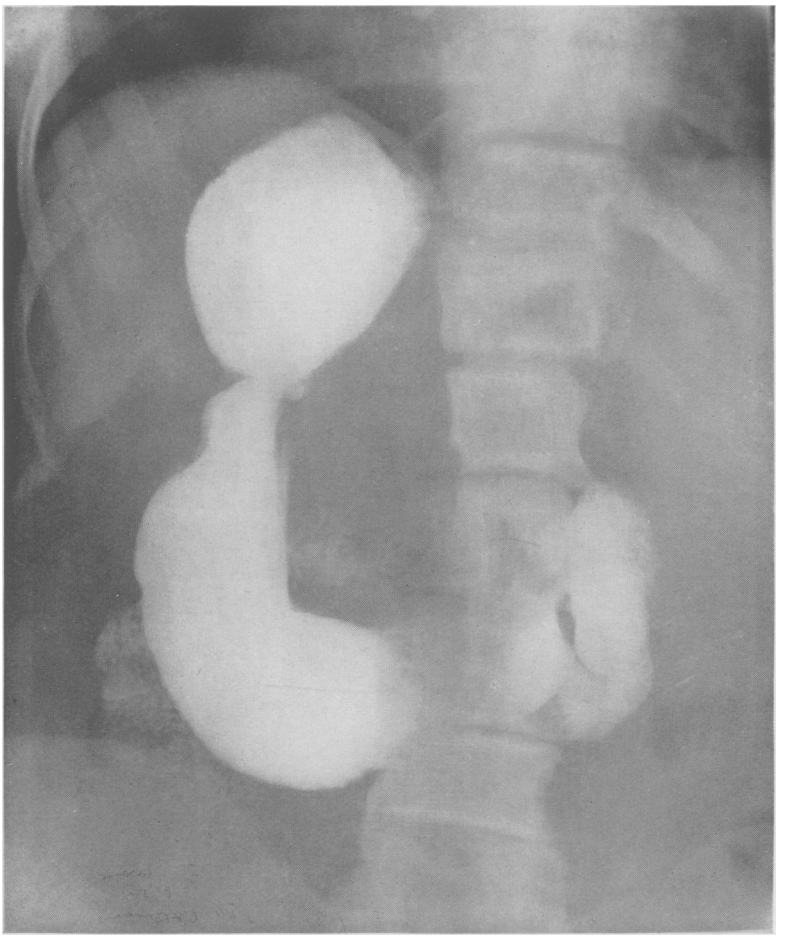
Multiple gall-stones.
Direct demonstration.

(2)



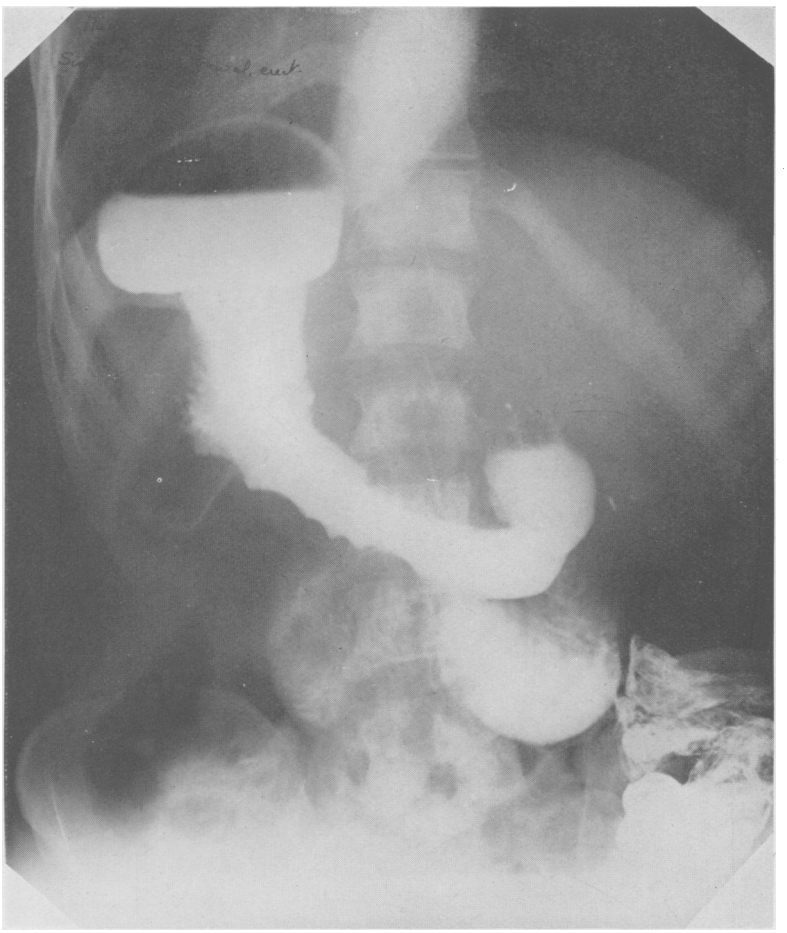
Multiple gall-stones
shown as "negative shadows" by cholecystography.

(3)



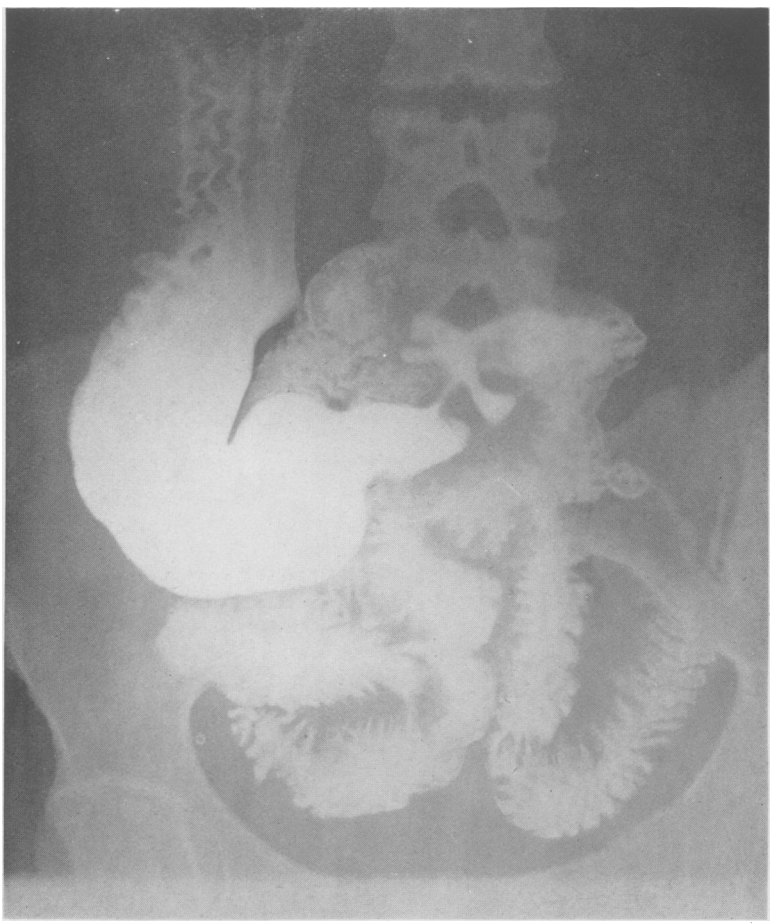
Small penetrating ulcer niche
lesser curvature stomach.

(4)



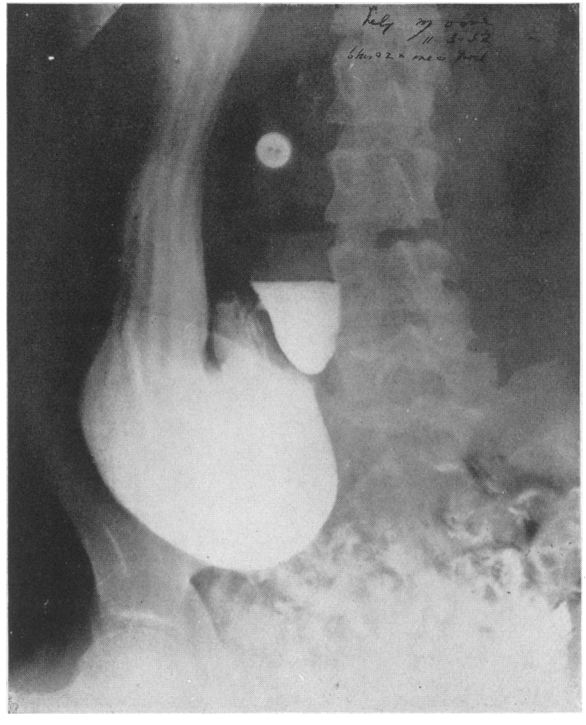
Infiltrative carcinoma (linitis plastica) involving almost all the stomach, with back-pressure delay in oesophagus.

(5)



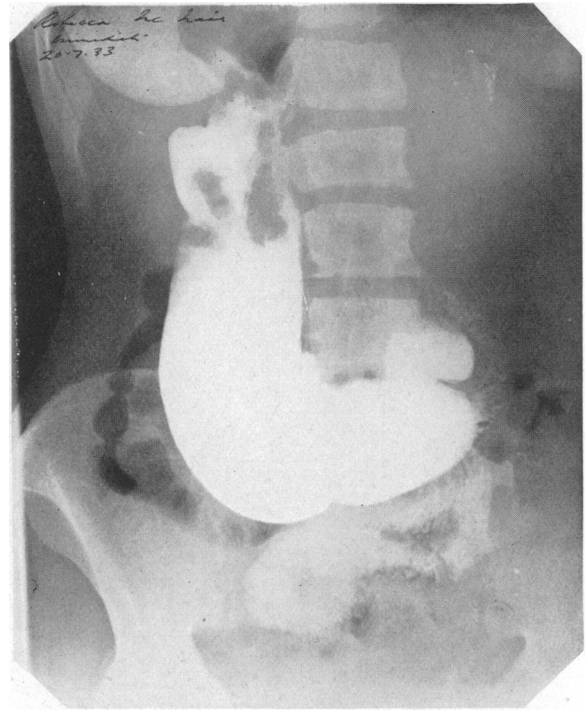
Typical deformity of "duodenal cap" in case of duodenal ulcer (note normal rugae cardiac end stomach).

(6)



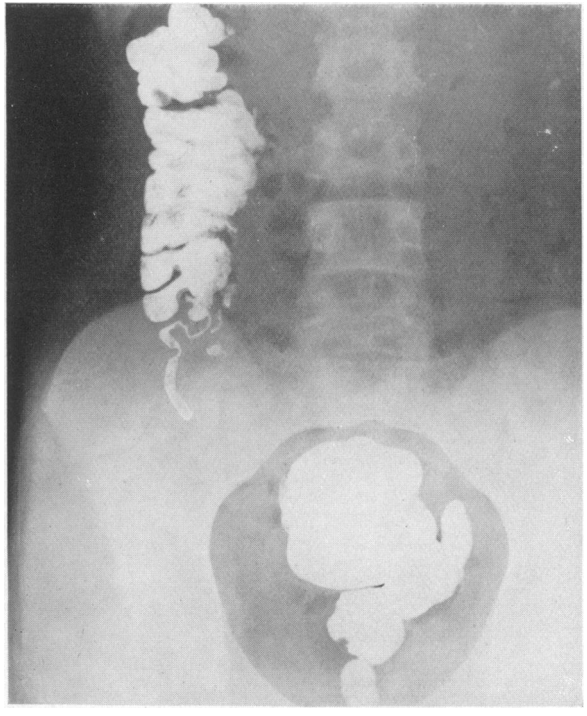
Large duodenal diverticulum.

(7)



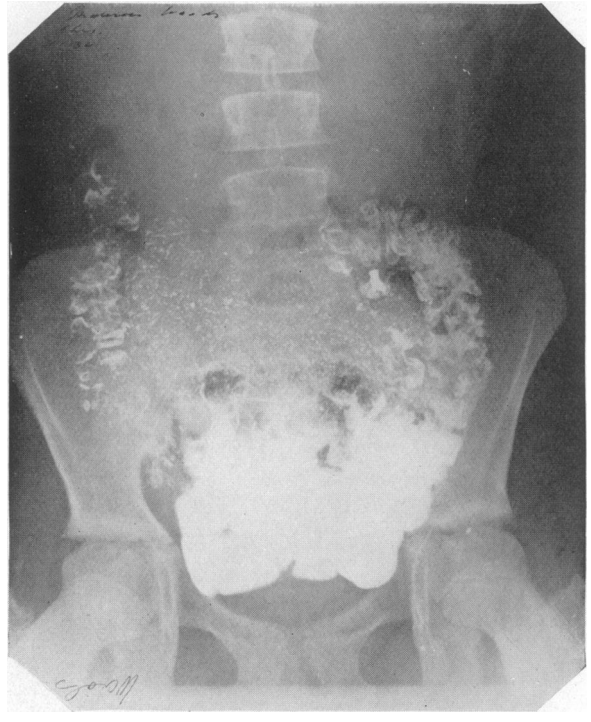
Fungating carcinoma
cardiac end stomach.

(8)

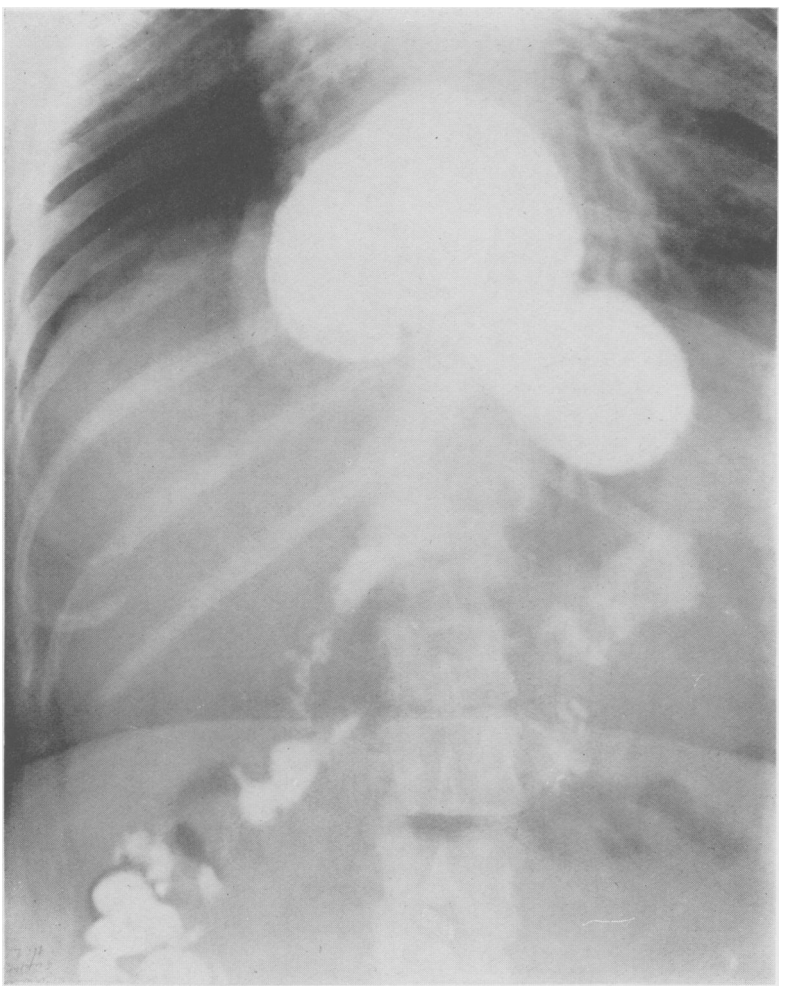


Long, tortuous, and sacculated appendix. Dyspeptic symptoms totally relieved by appendicectomy.

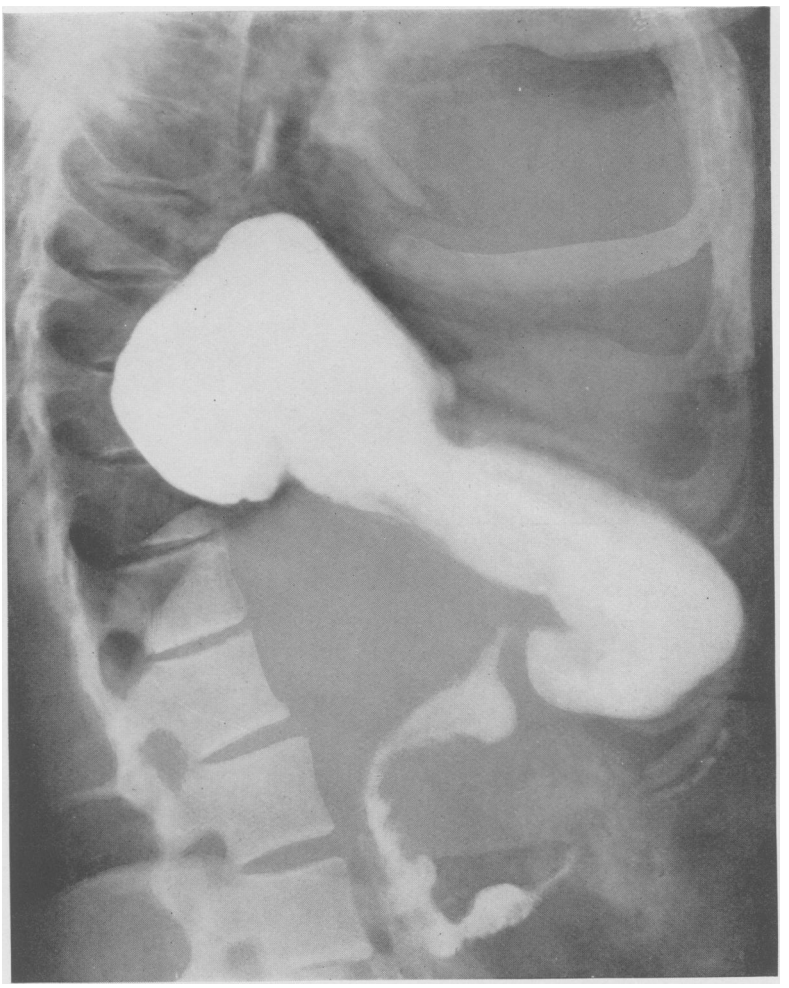
(9)



Typical "peppered" ileal stasis in case of mesenteric adenitis.



Partial "thoracic stomach"
congenitally short oesophagus.



Partial thoracic stomach
(lateral view).

The Role of Chronic Cholecystitis in the Production of Indigestion.

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THE view that chronic cholecystitis is the commonest organic cause of indigestion is widely held. In support of this contention Eusterman,[1] from a study of patients in the Mayo Clinic exhibiting gastric symptoms, states that there are sixty per cent. more cases associated with a diseased gall-bladder than with a peptic ulcer. To understand and estimate the value of such a statement, it is necessary to review shortly the physiology of the liver and the biliary tract if we are to appreciate the changes which arise under pathological conditions.

Among the many-sided activities of the liver, we will only consider those having a bearing on the subject of this contribution.

(*a*) *The storage of glycogen.* This function is possibly the most important, as in diseased conditions of the organ the blood-sugar level falls owing to shortage of dextrose. This loss of function can be temporarily restored by administration of glucose preparatory to dealing with the cause.

(*b*) *The secretion of bile salts and bile acids which assist the pancreatic ferments to split fats into fatty acids and glycerin.* Failure of this function results in excess fats in stools, due to faulty fat metabolism.

(*c*) *Detoxifying power.* Opie[2] concludes that the liver, by means of its peculiar eipthelium lining its sinusoids, fixes insoluble material and many kinds of organic particles such as bacteria.

The value of such a function in diseases due to infection arising in the intestinal tract is apparent.

Investigation into the function of the gall-bladder has been greatly assisted by the introduction of cholecystography. It is now possible to visualize the viscus and observe its size and shape, its filling and emptying, its density as illustrating its power of absorption, and its varying reactions to the ingestion of proteids, carbohydrates, and fats.

From its construction, the gall-bladder is evidently intended for the storage of only a small quantity of bile, but owing to the transference of water through its walls, concentration occurs. This concentrated bile is discharged when the neuromuscular mechanism relaxes the sphincteric action at the distal end of the common duct. A gall-bladder, therefore, to act in a normal manner, must be capable of distension and collapse, must have an unobstructed cystic duct and a mucosa able to secrete and absorb.

Following chronic infection of the gall-bladder, naked-eye changes occur, such as alteration in its colour, increase in thickness of its wall, deposit of fat in the wall, and a general fibrosis. These changes affect the adjacent liver through the

communicating lymphatics, producing a greyness of its surface and later a hardening of its structure and a rounding of its edges. As a consequence of this infection of the liver, the bile produced may be deficient in some of its normal constituents and contain abnormal ingredients, and so fail to carry out its rôle in assisting intestinal digestion and intestinal elimination. In other words, intestinal indigestion occurs.

The routes by which the biliary tract may be infected are four in number :—

(*a*) Descending through the bile stream.

(*b*) Ascending from the duodenum.

(*c*) Blood-borne.

(*d*) By way of lymphatic stream.

Bearing in mind the characteristic changes which occur in all coats of the gall-bladder in chronic cholecystitis, from the mucosa to the serosa, it is not possible to regard the biliary route as frequent, as in the absence of injury to the protective lining of the bladder, the mere bathing of its coat with a bacteria-laden fluid would not cause the extension to the deeper coats which is always present.

Considering that the liver acts as a filter between the portal and systemic blood-streams, and that predisposing causes of infection can usually be detected in the alimentary canal or pelvic viscera, it is probable that infection is most commonly conveyed by the blood or lymphatics.

A point in favour of one of these routes is that it is not uncommon to find the bile sterile, while cultures made from the wall show growths. For a considerable period it was my practice, on removing a gall-bladder, to wrap it up in sterile gauze and send it to the laboratory, where Sir Thomas Houston carried out the investigation. His valued opinion confirmed the previous statement.

Before leaving the pathology of the gall-bladder, the interesting condition known as "strawberry gall-bladder" may be referred to. This presents itself usually without the appearance of chronic inflammation. The wall may not be thickened, its colour not noticeably different from normal, while stones may or may not be present. In other words, its diagnosis prior to removal is impossible.

Boyd thinks that cholesterol is normally absorbed from the gall-bladder, and if something interferes with the normal absorption, the strawberry gall-bladder results. He thinks, moreover, that the most probable factor in preventing the normal absorption is chronic infection.

Whether the retention of cholesterol in the mucosa is due to failure to eliminate it from the bile in gall-bladder, or due to its deposit from the blood, is still uncertain, but it is believed to be evidence of faulty fat metabolism.

Taking pain, nausea, vomiting, flatulence, and eructations as the commonest symptoms of indigestion, what is their relative frequency in chronic cholecystitis?

I have examined the case sheets of 266 consecutive patients treated by myself in the Royal Victoria Hospital for affections of the biliary system during the years 1929 to 1934 inclusive, and find that the frequency of symptoms was as follows :—

Pain in upper abdomen or referred to shoulder	220
Indigestion and flatulence	143
Nausea or vomiting, or both	86
Chills or rigors	23

The pain may be situated in right upper quadrant and segmentally referred to the shoulder blade, when it is suggestive of acute inflammation or impaction of a stone, and is accompanied by some rigidity in that area and deep tenderness on expiration; or a diffuse pain over the upper abdomen accompanied by a sensation of fullness and desire to bring up wind.

Scrimger[3] offers a theory of the production of gastric symptoms on the basis of nerve distribution. "A stone in the cystic duct may cause pain through its sympathetic connections with fifth to ninth dorsal segments. At the same time vagus reflexes are set up, causing muscular spasms of stomach and intestine with increased secretion. In less acute conditions, vagus reflexes may predominate, and in the intervals there may be disturbed function of stomach or intestine, eructations of gas, hypermotility, hyperacidity, and constipation."

It appears that no definite association exists of gastric acidity in chronic cholecystitis, as hyperchlorhydria and achlorhydria have been demonstrated as being present in varying proportions of published cases.

As predisposing causes of cholecystitis, may I be forgiven for quoting the well-known alliteration: "Female, fat, fertile, and forty"; it conveys a mental picture which is suggestive.

It is accepted that this is a disease affecting women more than men in proportion of two to one or even higher, and the significance of age, sex, and pregnancy in its production lies in the greater tendency to inactivity and consequent stasis in women. This inactivity, whether due to occupation or childbearing, favours infection.

The obesity which is frequently present is not, in my opinion, a predisposing cause, but a direct outcome of the disease. As has been shown above, faulty fat metabolism exists, and no amount of exercise or regulation of diet will produce a reduction. Subjects of cholecystitis are not big eaters, and have already learnt to avoid fatty or greasy foods.

In investigating a suspected case of chronic cholecystitis, careful questioning into the personal history is of importance, as it may reveal a possible source of origin. For example, a history of peptic ulcer, of appendicitis, of colitis, of typhoid or the infectious fevers, is of extreme value.

It should be remembered that the onset of this disease is very slow, and it may be years after an infection before symptoms arise sufficiently distressing to demand attention.

It will be seen that a diagnosis may be difficult if reliance is put only on symptoms and signs, and in this connection I have purposely avoided the mention of jaundice. This sign will only be present in cholecystitis if common duct obstruction or cholangitis complicate the condition. To aid in diagnosis and help determine liver function, cholecystography is of first importance.

The outstanding symptoms of chronic cholecystitis are, therefore, pain of a characteristic type, flatulence with eructations, nausea or vomiting or both, and constipation.

Treatment.

Pre-operative.—Assuming the diagnosis to have been made and operation decided upon, it is advisable to have a careful examination of the heart carried out. Many of these patients suffer from myocardial changes which can only be discovered by the electro-cardiograph. If this examination prove satisfactory, glucose and calcium are administered for a week; oral cleansing and intestinal elimination provided for.

Operative.—The procedure adopted depends on many factors. If the subject is old and decrepit, with evident cardio-vascular or renal changes, and where the chief indications are the relief of pain and vomiting, the method which can be carried out most quickly and with the least disturbance should be employed. This is undoubtedly cholecystostomy. Later, if considered advisable, removal of the gall-bladder may be undertaken.

Cholecystectomy is the operation of choice in well-marked chronic cholecystitis, with or without stones, both in view of the satisfactory results attending it and also the removal of a potential source of cancer. In carcinoma of the gall-bladder, stones are practically always present.

There are, however, cases which both clinically and radiographically suggest loss of function, yet at operation very slight naked-eye changes are apparent. The gall-bladder may be slightly altered in colour, but thin-walled; there may be delicate adhesions to neighbouring organs and even stones may be palpated, yet we hesitate to condemn the viscus. I am now swinging round to the view that if the stones are removed and drainage provided there is a possibility of the function being restored. My attention has been directed to the recently published work of Professor Pribram,[4] who says that his experience has led him to be more conservative than formerly in resecting the gall-bladder. Asserting that the results of cholecystostomy are far from satisfactory, he has devised an operative measure which he believes will restore gall-bladder function and avoid possibility of stasis. This consists in anastomosing the neck of the gall-bladder to the side of common duct, thus preserving the pressure-regulating mechanism.

If the results following this procedure prove satisfactory, many gall-bladders now sacrificed will be saved, and the number of cases with recurrence of symptoms following resection will diminish, for, as we believe, the commonest cause of this return is spasm of the sphincter of Oddi.

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2. Graham and Others, "Disease of the Gall-Bladder and Bile Ducts."
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Pre-Cancerous Lesions of the Digestive Organs

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A PRE-CANCEROUS lesion is one which is prone to become malignant. A comparatively large number of lesions of this kind are recognized in superficial structures—for example, arsenical and X-ray dermatitis, leukoplakia vulvæ, leukoplakia linguæ, chronic cervicitis and erosion of the cervix, and "chronic cystic mastitis." Even in these lesions, however, the incidence of malignant disease is extremely variable. This variability is explained in large part by the fact that the evolution of a malignant process may be protracted over a period of twenty years or more—so long that the patient may die from some intercurrent disease before invasive growth has begun. Nevertheless, the incidence of malignant disease in these lesions is frequent enough to indicate the possibility that they are all "pre-cancerous."

The recognition of pre-cancerous lesions in the digestive system has been rather more tardy for several reasons. The accurate diagnosis of the primary condition is naturally much more difficult. This primary condition is commonly but not invariably attended by symptoms of chronic indigestion. Such symptoms are continued or may be intensified when invasive growth begins, and the transition to malignant disease is liable to be missed. In any case, the precise relationship between an internal simple lesion and a malignant growth can be verified only by surgical operation or by a thorough post-mortem examination—and then only if the growth has not obliterated the characteristic features of the pre-cancerous lesion. Speaking generally, the supervention of malignant growth in pre-cancerous lesions of the digestive organs is less frequent than in those affecting superficial structures, because the former are intrinsically more dangerous to life, and a fatal complication is more liable to develop at a comparatively early stage.

There are four important lesions of the digestive system which may fairly be described as pre-cancerous on account of the frequency with which malignant disease has been found associated with them. They are (1) chronic peptic ulcer of the stomach, (2) adenomatous polypi of stomach and intestine, (3) chronic cholecystitis and cholelithiasis, and (4) cirrhosis of the liver. One or more examples of each of these lesions in which malignant disease has supervened have been examined at this Institute during the past three and a half years, and short descriptive notes of illustrative cases are appended.

The significance of each of these lesions as a pre-cancerous formation must be assessed separately. All alike possess the greatest theoretical importance with regard to the question of the etiology of malignant disease. Theoretically, also, the recognition of a lesion as a pre-cancerous formation might be expected to mark the first step in the prophylaxis of malignant disease, and this expectation has been fulfilled in part in pre-cancerous lesions of the superficial structures. A timely gastrectomy or cholecystectomy must have achieved the same result on

many occasions, but little or nothing can be done to arrest the progress of a cirrhosis of the liver, and this lesion is fatal whether carcinoma supervenes or not.

1. Chronic Peptic Ulcer of Stomach.

Illustrative Case.—Ref. No. 166/33, R.V.H. Male, aged 46. Epigastric pain for three years, at first intermittent, but constant for last six months; no definite relation to food; relieved by vomiting; several attacks of hæmatemesis. Appetite poor; loss of weight amounting to two stones in last six months. No free hydrochloric acid in a fractional test-meal. A partial gastrectomy was performed on account of a chronic peptic ulcer.

The ulcer was situated on the lesser curvature about three inches above the pylorus. It measured approximately one inch in diameter, and was deeply excavated. The base of the ulcer was composed of dense fibrous tissue, which interrupted the muscularis completely, and the free extremities of the muscle were rounded. The floor of the ulcer was covered with a white film of necrotic debris, and its margin was regular and showed no notable thickening. Summing up, this specimen presented the cardinal characteristics of a chronic peptic ulcer on naked-eye inspection. Microscopically, these observations were verified, but, in addition, spheroidal cancer cells were seen beginning to infiltrate the margins of the fibrous base. Consequently, it was concluded that a cancer had supervened upon a simple chronic peptic ulcer, and that the lesion was in fact an "ulcer-cancer." For the sake of brevity this term "ulcer-cancer" is used in contrast to the term "cancer-ulcer," connoting a primary carcinoma of the stomach which has undergone secondary peptic ulceration.

There are three important considerations arising out of this short abstract of the clinical history. The total duration of the gastric disorder was relatively short, amounting to only three years, whereas it is not uncommon to hear a story of epigastric pain covering a period of ten or even twenty years in a chronic peptic ulcer which is eventually shown by a detailed pathological examination to be benign. Evidently the supervention of malignant disease may be hastened in certain unknown circumstances. From a clinical point of view, the change in the periodicity of the pain was probably highly significant, marking the transition from the pre-cancerous to the cancerous lesion. The absence of free hydrochloric acid is exceptional in an early ulcer-cancer, but it may have been apparent rather than real in this case on account of chronic gastric catarrh attended by an excessive secretion of mucus.

There is no doubt that a certain proportion of chronic gastric ulcers become malignant sooner or later. The frequency of this occurrence is a matter of the greatest practical importance, because it must be a deciding factor in the choice between surgical and medical treatment. It is not easy, however, to arrive at a definite conclusion on this matter, although it may be accepted that less than six per cent. of chronic peptic ulcers undergo malignant degeneration. The most comprehensive analysis has been carried out by Stewart, who examined 358 stomachs removed at operation, a few of which contained double lesions. He found

that carcinoma had developed in 17 out of 277 chronic ulcers, or 6.1 per cent., and also evidence of pre-existing simple ulceration in 17 out of 109 gastric cancers, or 15.6 per cent. Dible reached similar conclusions in a somewhat smaller series of cases, comprising 126 ulcers and 33 cancers. Of these thirty-three cancers, there were five which might fairly be regarded as having originated in simple ulcers on histological grounds alone, but in only two of them was this deduction borne out by a long clinical history. My own series of cases, accumulated during the past three and a half years, is much smaller. It contains thirty-six cancers and twenty-three ulcers. Apparently gastrectomy has not been carried out so frequently in Belfast on account of chronic ulcer as on account of cancer, unless the surgeons concerned have withheld a larger proportion of simple ulcers from examination. Within that series of fifty-nine cases there was only one certain example of ulcer-cancer, and there were two more possible but doubtful examples. The clinical expectation of ulcer-cancer was higher than pathological examination could warrant. Not very infrequently, on account of a prolonged history consistent with a diagnosis of chronic gastric ulcer, a carcinoma of the stomach has been sent for examination with a query of "ulcer-cancer?" One of these is represented by a histologically doubtful lesion, but as a rule the pathological diagnosis has been a frank cancer-ulcer. It is possible in such cases that one or more chronic ulcers have been present which have undergone healing, or it may be, as Dible has described, that a chronic gastric irritation, insufficient to cause ulceration, may simulate in its symptoms of prolonged dyspepsia those of chronic gastric ulcer, and encourage a mistaken diagnosis of ulcer-cancer. The other alternative is that the carcinoma has progressed so far as to obliterate the cardinal characteristics of an antecedent simple ulcer.

On the whole, the clinical diagnosis of carcinoma of the stomach on the one hand, or of chronic peptic ulcer on the other hand, is rarely proved to be wrong on pathological examination. Occasionally, however, it happens that a lesion which was regarded clinically as a simple chronic ulcer is proved pathologically to be an ulcer-cancer, and conversely. Generally speaking, the differentiation between a simple chronic ulcer and an ulcer-cancer can be made satisfactorily by naked-eye inspection, but the fact should not be overlooked that the naked-eye appearances are sometimes deceptive. For example, the illustrative case cited above, 166/33, presented all the naked-eye characteristics of a simple chronic ulcer in conformity with the clinical diagnosis, whereas it was indubitably malignant microscopically. Also, I recall another case, 173/33, which had every appearance of an early ulcer-cancer, as I believed, with irregular thickening of its edges and enlargement and some induration of the regional lymph-glands, and yet no trace of malignant growth was discovered in a considerable series of sections from three separate portions of the thickened margin. The glands showed only chronic inflammatory hyperplasia.

Stewart has estimated that 6.1 per cent. of the chronic ulcers in his series have become malignant, but he adds that the real significance of chronic ulcer as a pre-cancerous lesion must be much less than that figure would suggest, because many chronic ulcers heal, leaving only a scar in the submucosa and muscularis.

A chronic ulcer which has healed satisfactorily is not prone to become malignant, so far as is known at present. It is not even certain that a healed chronic ulcer is more prone to subsequent ulceration than the rest of the mucosa.

2. Adenomatous Polypi of Stomach and Intestine.

The origin of these lesions is in general obscure. It has been suggested that they are post-inflammatory, but more often than not there is no antecedent history of gastritis or colitis. On the contrary, the lesions are usually silent until malignant disease declares itself.

Illustrative Case.—Multiple Adenomatous Polypi of Stomach and Adeno-Carcinoma. No. 157/33, R.V.H. Female, aged 38; married; six healthy children. No miscarriages. There was no history of any gastric disorder.

Admitted to Royal Victoria Hospital for removal of ovarian cyst. This proved to be a pseudomucinous ovarian cystoma which had undergone malignant degeneration. Died suddenly five days later as a result of pulmonary embolism. At post-mortem examination, the lining of the stomach presented twelve polypi, and a shallow malignant ulcer (adeno-carcinoma) was found at the cardiac end of the lesser curvature. This was an accidental finding, and doubly interesting on account of the independent malignant formation in the ovary. Apparently this woman had a peculiar hyperplastic diathesis affecting two different epithelial elements, and in both situations the hyperplasia had become cancerous. There were no metastases.

Multiple polypi of the stomach are uncommon. Stewart observed only twenty-three examples in a series of 12,800 post-mortem examinations. Of these twenty-three, however, no less than seven were associatd with carcinoma—an incidence of thirty per cent.

Polypi of the colon and rectum, either solitary or multiple, are considerably more common than gastric polypi, occurring in about four per cent. of cases, and again the incidence of carcinoma approaches thirty per cent. According to Dukes, when carcinoma supervenes in one of several polypi, the remainder tend to disappear. This is not always true. A case has been described by Lockhart-Mummery and Dukes, in which three separate malignant growths and several benign adenomata were present in the rectum, and other polypi were observed in the sigmoid flexure of the colon on sigmoidoscopic examination. In one of my own recent cases, P.554, the specimen removed at operation consisted of the splenic flexure with about a foot of the colon proximal and distal to it. There was an annular carcinoma at the splenic flexure, and five polypi were counted proximal to it; they ranged in size from a pea to a walnut. The largest was situated about four inches above the carcinoma, and was more or less sessile. Microscopical examination of this large polyp revealed no invasion of the muscularis mucosæ, but the glandular epithelium showed very notable nuclear hyperchromatism and irregular hyperplasia strongly suggestive of an early malignant change. This point is important. If bleeding or any other symptom has prompted sigmoido-

scopic examination, a distal polyp may be seen which has not undergone ulceration, or, if ulceration has occurred, a small fragment from the edge of the ulcerated surface may be removed for microscopical examination. Such a peripheral fragment can be very unsatisfactory for the purposes of histological diagnosis. It may show nothing more than hyperplasia of the glandular epithelium at a time when invasive growth is already in progress in the pedicle of the adenoma. Therefore it is highly desirable that some small part of the ulcerated surface, or, better, the whole polyp with its pedicle, should be removed for examination. In any case, the prognosis cannot be regarded as favourable when it is known that something like thirty per cent. of cases of adenomatous polypi of the colon and rectum are associated with carcinoma.

In general, nothing is known with regard to the etiology of adenomata of the large intestine. There is, however, a small proportion of cases in which the formation of multiple adenomata of the colon and rectum is the expression of a constitutional predisposition to proliferation of the intestinal mucous membrane. The predisposition is inherited as a Mendelian dominant, and the condition, known as polyposis intestini, manifests itself in several members of the same family. It ends almost invariably in cancer of the rectum or colon, and that usually in the early thirties or forties. The pedigrees of thirteen families assembled and reviewed by Dukes in 1930 afford convincing proof that polyposis intestini is an inheritable disease.

3. Chronic Cholecystitis and Cholelithiasis.

Illustrative Case.—P. M., No. 54/35, R.V.H. Male, aged 58. Previous health good.

Two months before admission began to complain of sense of fullness and discomfort in epigastrium after every meal; later, of definite epigastric pain, dull and constant in character; finally, jaundice developed. An exploratory operation revealed an inoperable carcinoma of the gall-bladder. Post-mortem : The wall of the gall-bladder was much thickened; its lumen was filled with pus, and contained numerous pigment calculi; its mucosa was irregularly nodulated. The adjacent part of the liver was invaded by direct extension of the carcinoma of the gall-bladder, and more remote metastases were present in the right lobe of the liver.

The gall-bladder exhibits very considerable structural changes in chronic cholecystitis. Its wall is more or less thickened and opaque, and the bulk of this thickening is frequently due to subserous fibrosis. At the same time, the mucous glands often show a remarkable extension into the muscular coat or even beyond it into the subserous coat. This extension is apparently the result of cicatricial processes in the granulation tissue during the evolution of the chronic lesion. The displaced glandular acini are lined by a quiescent type of epithelium, and they should occasion no confusion with the invasive growth of a carcinoma. As a matter of fact, when malignant growth develops, it commonly originates in a

warty proliferation of the mucosa lining the lumen of the viscus. The hyperplastic epithelial cells show some degree of irregularity in their size and shape, while their nuclei become hyperchromatic. It may be rather difficult to differentiate between irregular epithelial hyperplasia of this type and early neoplasia, but if it is not actually cancerous, it is almost certainly pre-cancerous. In a series of seventy-five examples of chronic cholecystitis examined during the three and a half year period from January, 1932, till June, 1935, I have seen two lesions of this sort. The carcinoma originates not in the heterotopic glands within the wall, but on the surface of the mucosa. The surface of the mucosa must be more directly exposed to the action of toxic detritus within the lumen, but inflammation would appear to be merely a contributory factor in the development of carcinoma of the gall-bladder. Gall-stones are by far the most important determining factor. Eleven cases of cancer of the gall-bladder or of the extrahepatic bile ducts have been examined here since the beginning of 1932. In one case, it is not recorded whether gall-stones were present or not. Of the remaining ten cases, six were associated with multiple gall-stones either of the facetted or of the pigment variety—the varieties, in short, which are the common sequel or accompaniment of chronic cholecystitis. The proportion of cases of carcinoma of the gall-bladder and bile passages associated with cholelithiasis in this small series of ten cases is in general agreement with the much more extensive observations carried our by Gross at Leeds. She found that seventeen out of twenty-eight cancers of the gall-bladder (60.7 per cent.), and ten out of eighteen cancers of the bile passages (55.5 per cent.), were associated with gall-stones. A further analysis of her classified data enabled her to conclude that "the facetted stone is definitely associated with local lesions, neoplastic as well as inflammatory, of the gall-bladder; pigment stones are associated with cholecystitis, but not with carcinoma; while the solitary cholesterol and mulberry stones are unrelated to either of these affections."

4. Cirrhosis of the Liver (including Hæmochromatosis).

It is well known that the liver is one of the organs in the human subject in which primary malignant tumours are rare and secondary growths are comparatively common. In the great majority of cases, primary carcinoma of the liver is a sequel to cirrhosis of the organ—multilobular or portal cirrhosis on the one hand, and that peculiar cirrhotic lesion associated with an enormous deposit of iron in the liver cells and interstitial tissue, described as hæmochromatosis, on the other hand. The incidence of carcinoma is low, however, probably because other fatal complications are so liable to develop during the progress of the primary disease.

There has been only one example of primary carcinoma of the liver coming within my observation in Belfast in the last three and a half years, and it originated in a case of hæmochromatosis. The patient, a male aged 53, had not been an inmate of the Royal Victoria Hospital, and clinical notes are not available. Also a complete post-mortem examination was not carried out. The clinical features of the case had been so puzzling that no diagnosis could be made during life. A mass had been palpable in the right hypochondrium, and, post-mortem, the medical practitioner

excised an irregular portion of the liver and sent it to me for pathological examination. The macroscopical and microscopical appearances of the specimen were consistent with a diagnosis of hæmochromatosis and liver-cell cancer. Meanwhile, I have conducted post-mortem examinations at the Royal Victoria Hospital on one other example of hæmochromatosis and eight examples of portal cirrhosis. None of these nine cases has been associated with carcinoma of the liver, but in one case the epithelial hyperplasia exhibited, microscopically, an unusual degree of activity.

Primary carcinoma of the liver is classified into two main varieties, liver-cell cancer and bile-duct cancer. Liver-cell cancer is the more common of the two, and it shows a more intimate association with cirrhosis. Collating reports of published series to 1931, Stewart found that two hundred and nineteen out of two hundred and forty-six cases of liver-cell cancer (89 per cent.), and twenty-five out of forty-nine cases of bile-duct cancer (51 per cent.), were associated with cirrhosis. In his own series of one hundred and twenty-four cases of well-marked portal cirrhosis, over the age of 30, there were nine examples of carcinoma of the liver, representing an incidence of 7.3 per cent.

The general interpretation of the association between portal cirrhosis and liver-cell carcinoma is as follows. The cirrhotic lesion consists essentially in a slowly progressive destruction of the liver parenchyma around the portal tracts, accompanied by a proliferation of connective tissue as the result of the action of a toxin or poison, the nature of which is not known. When the hepatic function becomes impaired, a compensatory overgrowth of surviving liver parenchyma occurs, and this is evidenced by more or less active hyperplasia of the liver-cells, so that nodular formations resembling adenomata are formed in the liver. Eventually, by slow degrees, the hyperplastic cells acquire a more autonomous type of growth; they invade the portal veins, and declare themselves as malignant tumour cells.

These four lesions represent the principal pre-cancerous lesions of the digestive organs, in which an association with carcinoma has been definitely established. Further, it has been suggested by Hurst that chronic gastric catarrh is an important precursor of cancer of stomach, but his suggestion has not yet been substantiated. On the other hand, there would appear to be some association between chronic appendicitis and the carcinoid tumour of the appendix, of which I have seen two examples here. In these two cases, there had been symptoms of chronic appendicular disease, and the tumour was a more or less accidental finding. Fortunately it is a relatively benign formation, and rarely metastasises. The contrast between chronic peptic ulcer of the stomach and of the duodenum is most remarkable. Chronic peptic ulcer of the duodenum is not pre-cancerous, whereas Stewart has observed in his series that one out of every four chronic peptic ulcers in the pyloric antrum of the stomach shows evidence of malignant transformation.

I am indebted to all the members of the staff of the Royal Victoria Hospital for granting me free access to their clinical records, and, in particular, to Mr. S. T. Irwin, M.Ch., F.R.C.S., and to Dr. W. A. Shannon, for detailed information respecting case No. 554.

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THE DOCTOR'S LEISURE HOURS

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Remarks on Surgical Dyspepsia

By P. T. CRYMBLE, M.B., F.R.C.S.ENG.

Professor of Surgery, Queen's University, Belfast

THE recognition of surgical dyspepsia involves interpretations of signs and symptoms, a knowledge of the living pathology of the abdomen, and familiarity with surgical technique. Consequently it is only to be expected that a number of suitable cases never receive the benefits of surgery, and that a number of unsuitable cases are operated upon. The practitioner or the consulting physician may fail to recognize the *surgical* case, or may bring to operation a case devoid of surgical pathology. An even heavier responsibility devolves upon the surgeon who is called upon to recognize pathology through an incision and to apply suitable technique. What can be more tragic than the post-mortem examination which reveals only an appendix abscess in a patient treated for three years as duodenal ulcer, and what can be more unsatisfactory than the removal of an appendix for symptoms due to a lesser-curve ulcer. Three points tend to restrict the field of surgery in dyspepsia :—

1. The fact that the great majority of dyspeptics show no surgical pathology.
2. The great length of time during which the surgical dyspeptic may exist without surgical interference, a point which may delude the physician into thinking that he has cured the condition.
3. The occasional operation death.

Very little remains to be said of the signs, symptoms, and pathology, but finality has not been reached on the form or applicability of the operation. We know, of course, that the removal of a diseased or deformed appendix, the removal of a gall-stone, the removal of an inflamed gall-bladder, or the division of an adhesion, may cure a dyspepsia. We have quite decided that a dilated hypertrophied stomach, secondary to a pyloric or duodenal scar, is ideally treated by gastro-enterostomy, and most surgeons can show an occasional good result after partial gastrectomy for cancer, but non-obstructing duodenal ulcer and gastric ulcer still remain a battleground.

A large number of gastric ulcers will be found on the posterior aspect of the lesser curve, completely perforating the stomach wall and floored by the pancreas. At operation one finds the margin of the ulcer formed of cicatricial tissue, and the aperture floored by the pancreas may be anything in size from a shilling to the palm of the hand. No one has convinced me that healing ever takes place in such a condition, and therefore one must remove the pathological area or condemn the patient to continue throughout the remainder of his days with an aperture in the back of the stomach. All that a gastro-enterostomy could possibly do would be to relieve the symptoms—it could not alter the anatomical condition. There is, therefore, much to be said in favour of gastrectomy (hemi- or two-third) for the treatment of the chronic perforation of the lesser curve of the stomach, and later on the results of a series of gastrectomies will be referred to. The small non-perforating

ulcer is quite a different problem, and many surgeons would be content in this case to do a local excision plus a gastro-enterostomy. Examination of the stomachs removed by gastrectomy teaches one, however, that the ulcer is only a part of the pathology, and that the ulcer is always accompanied by a more or less extensive area of gastritis. This gastritis might disappear after the formation of the gastro-enterostomy.

The non-obstructing duodenal ulcer is the main cause of dispute at the present time. The figures of Moynihan, Sherren, and Walton show remarkably good results with gastro-enterostomy, whilst many Continental surgeons deny the efficacy of this operation and carry out extensive gastrectomy for this condition. The gastro-enterostomist claims a low operative mortality and an eighty per cent. cure, whilst the gastrectomist claims freedom from all forms of secondary ulcer, but at the price of a higher operative mortality.

Secondary ulceration, either in the stomach, stoma, or jejunum, is the chief bad after-result in gastro-enterostomy, and it is said to occur most frequently in young adults with hyperchlorhydria. Even though it does occur, it can be dealt with surgically, and later on two cases of jejuno-colic fistula will be fully reported. British surgeons still support gastro-enterostomy as the ideal measure for non-obstructing duodenal ulcer.

Secondary ulceration attacking the stomach, the margin of the gastro-enterostomy, or the jejunum, may follow a gastro-enterostomy, and an even more serious complication may arise when this ulcer erodes into the transverse colon and allows fæces to escape into the stomach. Two recent cases of jejuno-colic fistula proved amenable to operation, and show that it is not such a serious condition as has been claimed.

CASE 1.—Policeman H. O., aged 30.

History:

1923—Pain one hour after food, for one week.

1926—Similar attack, lasting for two months.

1929—Numerous more severe attacks, accompanied by vomiting and on one occasion hæmatemesis.

1930—Operation : Gastro-enterostomy and appendicectomy for duodenal ulcer.

1934—Reports diarrhœa and fæcal-smelling eructations for past five months. Motions three in the day and six at night. Loss of two stone in weight.

Operation revealed a fistula, the size of the finger, between the transverse colon and the efferent limb of the jejunum.

The fistula was entirely distal to the gastro-enterostomy.

The two portions of bowel were separated and the colic and jejunal apertures closed. There was no sign of any ulceration nor was there any surrounding scar-tissue.

Recovery was uneventful, and he has been carrying out full duty during the past year.

CASE 2.—Station-Constable T. S., aged 50.

History:

1921—Operation for perforated gastric ulcer. Simple closure.

1932—Gastro-enterostomy for long-standing pyloric obstruction.

1935—Reported with an eight-months history of nocturnal diarrhœa, loss of weight, and flax-water-smelling eructations of gas. Occasional vomiting of large quantities of thin creamy material.

Operation: The transverse colon was adherent to the efferent limb of the jejunum and the adjacent part of the gastro-enterostomy. On separating these parts, a jejuno-colic fistula the size of a florin was exposed. Superiorly the fistula included the lower angle of the gastro-enterostomy. The mucous membrane was everywhere perfectly healthy. The colic and jejunal apertures were closed and infolded.

Recovery was uneventful, and the patient has been on full duty for some months.

These two cases would encourage one to tackle such cases with more confidence, and they suggest that the colonic fæces coming in contact with the jejunal ulcer leads to healing. The reaction of the colon contents is alkaline, and this may be the healing property.

It is worthy of note that in the Middle Ages a watery extract of cow dung formed a part of certain prescriptions.

In view of the fact that there has been recent discussion on the operative treatment of gastric and duodenal ulcer, on the lines of gastro-enterostomy versus gastrectomy, it may be of value to review a series of sixty-six consecutive cases in which one has performed gastrectomy for gastric ulcer.

There were five operation deaths, giving a mortality of 7.5 per cent.

The symptoms present in the series included pain, vomiting, hæmorrhage, and loss of weight, but pain was the only symptom invariably present. The ulcer gave rise to abnormality in the opaque meal, either as a lesser-curve spur, as an hour-glass contraction, or as seven-hour gastric retention. One or two of these signs might be absent.

In all the cases the ulcer was situated on the lesser curve, mostly on the posterior surface and frequently on that portion related to the pancreas. When the ulcer has perforated the stomach wall completely, the floor of the ulcer is usually formed by the pancreas. A white fibrous ring is developed at the gastro-pancreatic junction, and this prevents any further healing. Contraction cannot take place and a new stomach wall cannot be grown.

It is only when the stomach has been divided at the pyloro-duodenal junction and separated from the pancreas that a good view of the pathological anatomy can be obtained. The mucous membrane in the immediate neighbourhood of the ulcer is the seat of gastritis, and this condition frequently extends towards the pylorus.

The operation consists of the following steps :—

1. Division of the gastro-colic ligament in relation to the portion of great curvature about to be removed.
2. Application of Martels clamp to the pylorus.
3. Division of the pylorus and closure of the duodenum.
4. Separation of ulcer from posterior abdominal wall and pancreas.
5. Application of a large Payers clamp to the stomach oral to the ulcer.
6. Proximal jejunum brought through an aperture in the transverse mesocolon, to the left of the middle colic vessels.
7. An end-to-side gastro-jejunostomy is now carried out, so that the jejunum joins the stomach at the lesser curve and leaves the stomach at the great curve. This anastomosis is made immediately distal to the large Payers clamp on the stomach. In the process of doing this the condemned portion of stomach is removed.
8. The margins of the mesocolic aperture are fixed to the stomach close to the anastomosis.

Difficulties may be encountered at each stage of the operation—

a. The middle colic vessels are in danger in division of the gastro-colic ligament. By beginning the division to the left of the middle line, in the region of the lesser sac, the vessels may be recognized and avoided. Near the pylorus the large right gastro-epiploic vessels will be encountered and will require division.

b. The infolding of the duodenal stump may prove difficult, since the gastro-duodenal vessels may be punctured or the pancreas may interfere with the infolding. These difficulties are entirely overcome by leaving the pyloric end of the stomach *in situ* as in the Devine operation. Recent research has shown that this pyloric end is of use in preventing anæmia. Unfortunately, the pyloric end of the stomach is frequently the site of the ulcer.

c. The separation of a large ulcer the size of the palm of the hand requires patience and courage. A large Payers clamp passed around the stomach above the ulcer will be of great assistance in controlling hæmorrhage and for traction.

d. A retrocolic operation may be inadvisable or impossible, owing to the absence of a mesocolon, or the shortness of the mesocolon, or the vascularity of the mesocolon. Under these circumstances the jejunum must be brought in front of the transverse colon to its junction with the stomach and anastomosed as described above or led from left to right, so that the jejunum joins at the great curvature and leaves at the lesser curvature.

Special care must be taken to obtain a watertight junction at the postero-superior part of the anastomosis, since a poor stomach wall may be found in this

region, and it is the most inacessible part. In one of my cases leakage took place at this spot and caused death.

The after-treatment is of great importance, and may turn the balance in favour of life or death. The following six points might be stressed :—

(*a*) Hæmostatic serum 2 c.c.

(*b*) Glucose saline per rectum or intravenously, if shock be present.

(*c*) Fluids only, given by the mouth, for five days.

(*d*) No aperient stronger than semproline with phenolphthaline.

(*e*) Gastric lavage for persistent hæmorrhage or obstructive vomiting.

(*f*) Alkalies by the mouth for several weeks.

After-Results of Gastrectomy.

Whilst the published lists of gastro-enterostomies run into thousands of cases, gastrectomies are few in number, so that it is difficult to compare the two operations. The surgeon should have both operations at his command, and apply to each condition the most suitable technique. The pure short-circuiter is condemned to have a very incomplete knowledge of gastric pathology, since this is mainly disclosed during the process of gastrectomy, and apart from this he will take a somewhat biassed view in favour of his method. In my opinion, gastrectomy has a very definite place in the treatment of surgical dyspepsia. The operation has a reasonable mortality (say seven per cent.), and the large proportion of cases are restored to working capacity. There is, however, a small minority in which the result is poor. There may be a temporary anæmia, which usually clears up under prolonged medical treatment, or there may be persistent pain, possibly due to secondary ulceration. I have seen one case of jejunal ulcer following hemi-gastrectomy, and the following is a short history :—

Mr. P——.

1914—Gastro-enterostomy for duodenal ulcer.

1928—Hemi-gastrectomy for stoma ulcer.

1930—Operation for jejunal ulcer. The gastro-jejunostomy had retracted high up above the mesocolon. The afferent and efferent limbs of the jejunum were compressed by the mesocolic orifice, and there was a jejunal ulcer on the efferent limb. The ulcer was infolded, the stomach pulled down into the ilio-lumbar region, and a two-inch pouch of stomach fixed there by suturing the stomach wall to the margin of the meso-colic orifice.

1935—Patient reports : "My last operation has been fairly successful, but I must be careful of the food I eat. No vegetables, potatoes, or greasy food will agree with me."

This case illustrates one of the defects of simple short circuit—the possibility of secondary ulceration. It also shows that a jejunal ulcer may develop after a hemi-gastrectomy, but that finally a fairly satisfactory result was obtained.

Abdominal Pain

By RICHARD H. HUNTER, M.D., M.CH., PH.D., M.R.I.A.,
Queen's University, Belfast.

IT has long been known that the abdominal viscera are insensitive to cutting, burning, and other forms of trauma that induce pain when applied to the somatic tissues, i.e., skin, muscle, etc. This fact led many clinicians to the belief that true visceral pain does not exist, and that the pain present in certain forms of visceral disease is caused either by inflammatory or other pathological involvement of the peritoneum. This involvement, it is said, may manifest itself either as a painful impression at the site of the lesion, or be referred to some distant area of skin or muscle.

For example: An inflammatory reaction involving the peritoneum covering the under aspect of the diaphragm may manifest itself by an area of pain, which may or may not be accompanied by hyperæsthesia, over the shoulder of the same side. The diagram shown in fig. 1 is an illustration of the path whereby the painful impulses may pass. The stimulus is received in the sub-phrenic plexus P, travels along the phrenic nerve A to the spinal ganglion B, and thence to the spinal cord, where it forms what Szemol calls a "focus of irritation" at the synapse C, at the level of the fourth cervical nerve-segment. The upper neuron D, connecting with both A and G (the peripheral nerve to the shoulder), crosses to the opposite side in the lemniscus, ascends to the thalamus E, and finally reaches the cortex of the brain F. Then, since the neuron A has never been educated to feel or localize pain directly, the cortex registers the pain along the dominant and usual pathway of neuron G, coming from the area marked with shaded lines over the shoulder.

Doubt, however, has been cast on the view that the peritoneum is the only source of abdominal pain, and many observers now believe that certain forms of pain can be appreciated by the viscera themselves.

Recently Livingstone[2] carried out a series of experiments in a case of sigmoid colostomy, which supports this view. His description of these experiments is as follows:—

"My patient was a co-operative and intelligent woman well qualified to analyze her sensations. I was unable to elicit the slightest pain or any form of sensation by stimulating the gut by a great variety of chemical and mechanical means. When the gut was strongly stimulated with the inductorium there followed a rapid blanching of the tissues, and subsequently a peristaltic contraction made itself evident. She experienced no sensation upon contracting the electrode to the gut, nor at the time of the blanching, but stated that she experienced cramp-like pain across the lower abdomen at the time the contraction was marked. She described the sensation as a 'gas-pain,' and was confident that it seemed to be within the abdomen and not in the parietes. The pain subsided gradually a few seconds after the removal of the electrode, often before the blanching had disappeared. She also experienced discomfort when a small rubber balloon within either loop of the

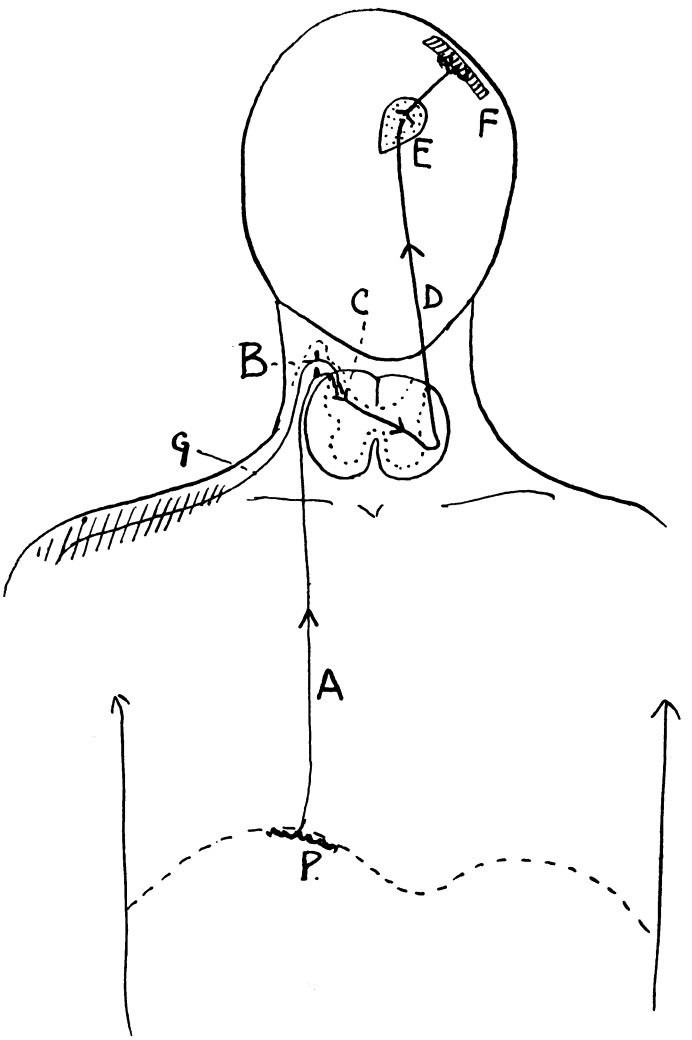

FIG. 1.

Diagram to illustrate the mechanism of referred pain. (After Capps and Coleman.)

colostomy was distended to a pressure of 80 mm. of mercury. If the pressure was increased to 100 mm. of mercury, and maintained for a few seconds, she complained of severe pain that caused her to groan and rub her hand over the pubic region. She stated that something within her abdomen felt as if it were 'about to burst.' She compared the sensation to that of a distended bladder, but knew it did not arise from that organ, and that it was more intense than any sensation she had ever had from the bladder."

Bloomfield and Polland,[3] and later Boyden and Rigler,[4] carried the investigation of this problem farther, by devising experimental methods capable of being applied directly to *normal* living men. Bloomfield and Polland obtained a number of volunteers who agreed to swallow balloons into the stomach and duodenum. The balloons were then inflated with air. The volunteers described their feelings as the experiments proceeded, and in each case complained of a deep-seated pain between the xiphoid process and umbilicus.

Boydon and Rigler's method was more refined. They induced eleven medical students to swallow the expanded metal end of a Rehfuss tube through which an induction current was passed. The metal end of the tube, attached to an insulated copper wire, acted as an electrode when in contact with the gastric or duodenal wall, and a second electrode was made of a moist pad on copper placed on the arm or leg. An electric current was sent through the wire, and its effect on the gut was observed under the fluoroscope screen, while the student described his symptoms.

The effect of the current upon the stomach was to induce a sphincteric contraction of the gut, and then an increased peristalsis distal to the point of stimulation. The effect of the current upon the duodenum could not be ascertained, because the barium passed through this portion of the gut so rapidly.

In both stomach and duodenum, contraction was usually accompanied by some degree of abdominal rigidity, depending on the strength of the current used.

The nature of the sensation that accompanied contraction of the gut ranged from" barely perceptible feelings of pressure, gnawing sensations and heartburn, to dull and severe colicky pain." When a mild current was employed, one or more seconds usually elapsed before visceral sensations were felt. Then the pain increased gradually to a climax. In the case of very strong currents causing spastic contraction of the gut, the pain was immediate.

Localization of these sensations was characterized by two general features :—

(1) The depth of the sensation, seeming to come from well beneath the abdominal wall.

(2) The definiteness with which it could be located in the upper quadrants of the abdomen, the subject always pointing to the spot with one finger.

The results of these two sets of experiments closely agree with one another in general terms, but differ from each other in details of precision. In Bloomfield and Polland's experiments the areas of pain, following inflation of the balloon in the stomach, were not sharply localized, the subject referring to the area by placing his whole hand over the mid-epigastrium, instead of pointing to the area with his

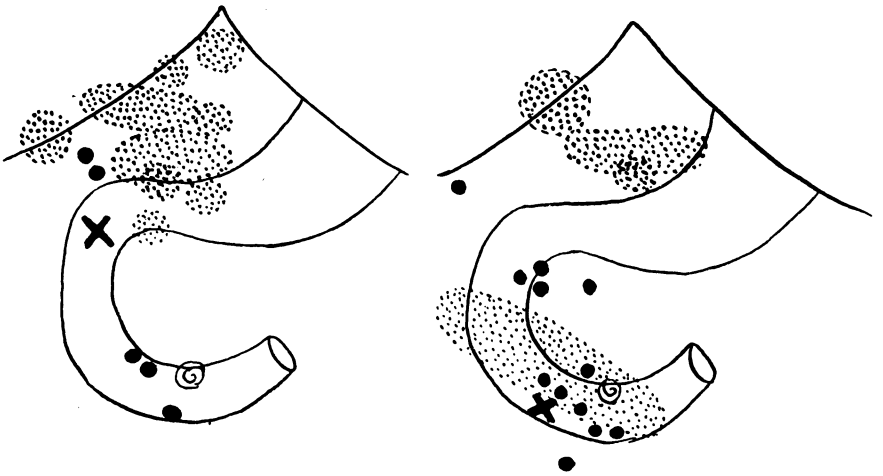

FIG. 2.

Diagrams to illustrate sites of pain following distention of duodenum by balloons (stippled areas), and pain following Faradic stimulation (large dots). X = point of stimulation. (After Boyden and Rigler.)

finger as the student did in Boyden and Rigler's experiments (fig. 2). Distention of the duodenum resulted in much more definitely localized pain, but never so localized as after faradic stimulation.

In neither case did even maximum muscle tension cause pain to be referred to the sides or back of the trunk. The latter phenomenon, it is concluded when observed clinically, cannot be caused directly by the gut wall, and must therefore be due to extension of the lesion into the mesenteries or retro-peritoneal tissues.

These, and other experiments, all support the view that true visceral pain exists.

Various explanations have been offered to account for the production of this pain. One of these is, that as pain is not a character of diseased solid organs, unless accompanied by inflammatory changes involving the surrounding peritoneum, that visceral pain may be confined to hollow organs only. It is suggested that it is produced by violent peristaltic efforts on the part of the viscus to overcome some obstruction, or to expel some solid object held within it. The pain is synchronous with the peristaltic waves, and the suggestion has been made that either the muscular contractions squeeze the afferent nerve-ends within the wall of the viscus, or that they are stretched sufficiently to give an adequate stimulus by distension of the wall of the viscus immediately in front of the peristaltic wave.

Either of these two views would offer a plausible explanation of pain, but neither of them apparently is capable of direct experimental proof. There is, however, another possible explanation of the cause of pain. It is suggested by the experiments performed by Lewis[5] on the relation which exists between the production of pain and changes in the circulation of a limb. The blood-vessels of a limb are entirely occluded, and the subject of the experiment told to perform a series of gripping movements with his hand. When this is done continuously for about

thirty seconds, great pain is experienced in the arm, and the pain becomes intolerable if the experiment is continued for seventy seconds.

The pain in this experiment could not be due to nerve-muscular tension, because it does not become accentuated during contraction, but is related to the *amount* of exercise which is performed.

One explanation for the pain might be a local anoxia due to the occlusion of the blood-vessels. An experiment, however, has been devised to show that this is not so. The arm first is exercised until the pain develops, and the time required noted. The arm is then allowed to recover, and the experiment repeated; but this time for a few seconds less than that needed to produce pain, and now, even if the occlusion of the blood-vessels is continued for five minutes, no pain develops. The oxygen content of the limb must decrease markedly during this period, but in spite of that fact no pain develops.

Lewis suggests as an explanation of these results that muscular activity liberates a pain-producing factor which passes out into the tissue spaces, and is normally removed by the blood-stream. "But," he states, "if the circulation is occluded and exercise carried out, the pain-producing substance will accumulate, and when it reaches a certain concentration will cause the sensation of pain."

In support of this view, Lewis found that when exercise is performed, with occluded circulation till pain appears, and the exercise then stopped but with the occlusion maintained, that the pain persists unchanged and does not get worse. He found, further, if at this stage the circulation is released, that the pain disappears within a few minutes.

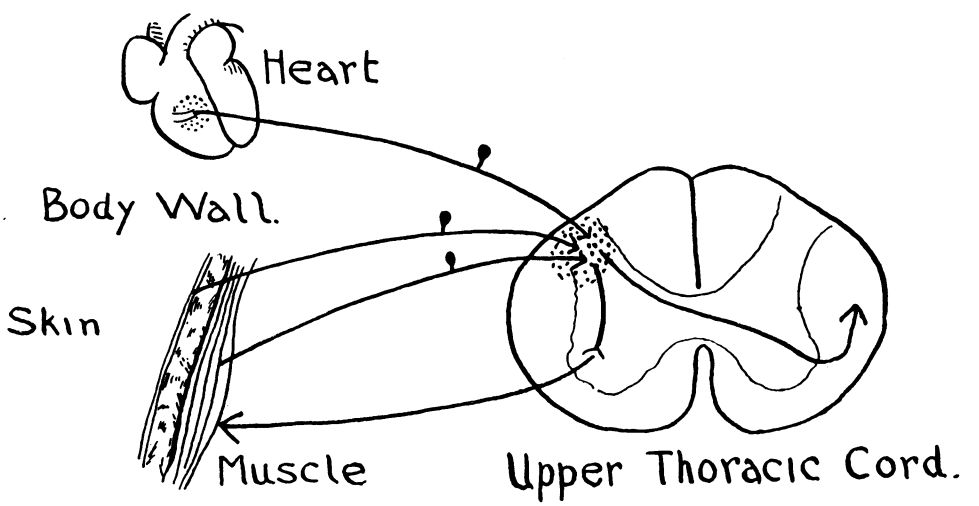

FIG. 3.

Diagram to illustrate possible neural basis of referred pain (after Wright). The dotted circle in heart indicates area of coronary occlusion. The dotted area on cord indicates "focus of irritation."

Lewis argues that the pain of coronary occlusion is produced in a similar way, as suggested by the scheme shown in fig. 3. The dotted circle in this diagram represents a region of myocardium which has become altered as a result of localized coronary occlusion. The dotted region in the posterior horn of grey matter is a so-called "focus of irritation." Afferent impulses from the heart are referred to the corresponding segments of skin and body-wall, and reflex contraction of body-wall muscle can take place. The spino-thalamic tract which crosses to the opposite side of the spinal cord conveys the afferent pain impulses to consciousness.

If Lewis is right in his deduction, the theory of a pain-producing factor could be applied to the production of pain in the intestinal tract. The dilated tube, in front of spasmodic contractions, would cause occlusion of the blood-vessels within its walls, and the pain-producing factor produced by the spasmodically contracting muscle of the tube-wall would not be carried away by the blood-stream, but remain and accumulate until it reaches the point at which it manifests itself, after irritation of the nerve-ends, by the sensation of pain.

The pain produced by electric stimulation in Boyden and Rigler's experiments could be explained in the same way. The electric current causes sphincteric contraction of the gut with blanching of the area, due to spasm of the blood-vessels, and a resultant heaping up of Lewis's pain-producing factor.

In the same way the pain found in the experiments of Bloomfield and Polland can be explained, by occlusion of the blood-vessels caused by the spasm of the gut-wall in attempting to get rid of the distended balloon.

Associated with pain in visceral disease is spasm or rigidity of the muscles of the abdominal wall. This rigidity usually involves the muscles close to the underlying diseased viscus, but it may occur at some distance from it. When it occurs at a distance from the diseased viscus, it is always found that the muscle is supplied by the same spinal nerve-segment as the viscus itself.

Morley,[6] in his book, "Abdominal Pain," brings forward evidence to show that "well-marked" muscular spasm indicates a direct involvement of the parietal peritoneum by the inflammatory process extending from the viscus. This view is ably supported by Livingstone,[7] who writes :—

"If I were to assume that reflexes of visceral origin were *solely* responsible for muscle-spasm, I should expect to be able to demonstrate the same degree of muscle resistance in the anterior abdominal wall in cases of retro-cæcal appendicitis, as in those cases in which the appendix lay directly beneath the anterior wall. Such has not been my observation. Moreover, the case of retro-cæcal appendicitis may show a spasm of the psoas muscle, which is supplied by quite a different segment of the spinal cord than that associated with the innervation of the appendix. I have come to feel that where I encounter a case of *marked* muscle-spasm in the abdominal wall, and I can satisfactorily rule out a rediculitis or local lesion in the muscle itself, I may reasonably expect to find the intra-abdominal lesion involving the parietal peritoneum in the same region that I find the muscle-spasm."

The muscle-spasm is always associated with pain, and Livingstone concludes that once the parietal peritoneum becomes involved in the inflammatory process,

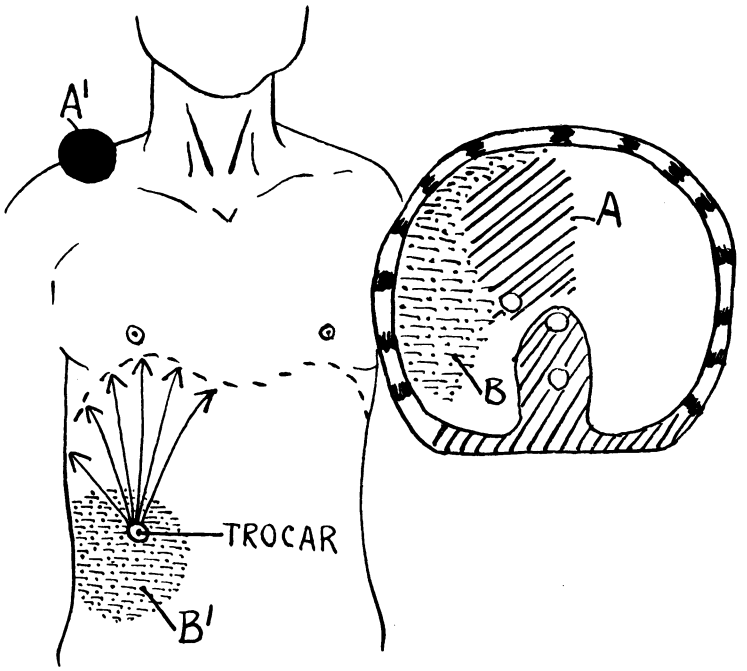

FIG. 4.

Diagram to illustrate points of referred pain from points of stimulation on diaphragm. Stimulation of area A causes referred pain at A'; stimulation of area B causes referred pain at B'. (After Capps and Coleman.)

the resulting pain will give rise to reflexes which are expressed by the spasm of the overlying muscles of the abdominal wall.

There is no doubt that the parietal peritoneum is richly supplied with afferent somatic nerve-fibres. Ramström[8] as long ago as 1908 demonstrated the presence of Pacinian bodies in the anterior portion of the parietal peritoneum. Sheehan,[9] too, proved their presence in the mesentery, and also that afferent nerve-fibres travel from them along the splanchnic nerves. Ramström showed by a series of experiments that these bodies are sensory. In these experiments an incision was made in the abdominal wall under cocaine, and the peritoneum was tested by pressure on it by a gloved hand and a spatula for evidences of pressure-sense, by cutting for pain sense, and by touching it with hot and cold instruments for temperature sense. Ramström found from these experiments that light pressure produced no sensation, but that strong pressure set up a cramp-like pain; that cutting the parietal peritoneum caused a "stitch-like" pain, and that there was no response whatever to temperature changes. In other words, that the parietal peritoneum has a deep pressure-sense; that the sense of pain is definitely present, and that temperature sense is absent.

More recently Capps and Coleman[10] carried out a series of experiments with the object of furthering our knowledge of this subject. These experiments were carried

out by a slightly more refined method than those of Ramström's. The skin over an area of abdominal wall was partially anæsthetized with ethyl chloride, and a trocar was inserted through the abdominal wall at this point. The point of the trocar was withdrawn, and through the canula was passed a long silver wire, one end of which was beaded and smooth, and the other end relatively sharp. Each end was slightly curved in order that it might more easily be brought into contact with the abdominal wall. At first patients with ascites were employed, so that the viscera would be separated from the parietes, and thus leave an uninterrupted passage for the wire, but as the technique improved, air was injected into the peritoneal cavity with the same object in view (artificial pneumo-peritoneum).

The results obtained from these experiments appear to agree with those obtained by Ramström : that the anterior median and lateral areas of parietal peritoneum are sensitive to pain; that they are sensitive to strong pressure of a smooth point, and to light pressure or lateral movement of a rough, sharp point of wire. They also found that stimulation of the parietal peritoneum can be localized with great accuracy on the abdominal wall by the patient, the error being less than an inch (fig. 4). Stimulating the diaphragmatic peritoneum shows similar results, i.e., absence of light pressure touch and presence of pain-sense. The localization of pain on these areas of peritoneum was never referred to the diaphragm itself, but was always referred to some distant part. On stimulation of the outer margin of the diaphragm, the patient complained of diffuse pain over the lower costal region and the hypochondrium, corresponding to the nerve-supply of the two parts. On stimulation of the central portion of the diaphragmatic peritoneum, the patient complained of pain over a sharply limited point along the trapezius ridge in the neck (fig. 4). These painful impulses were doubtless carried by the phrenic nerve to the cervical cord, and that the cerebral cortex registered the pain in the distribution of the cutaneous nerves of the fourth cervical nerve-segment (fig. 1).

Woollard, Roberts, and Carmichael[11] recently discussed this whole question of referred pain, as many investigators had begun to cast doubts upon it. They stimulated experimentally the central end of a divided phrenic nerve, and they found that the patient invariably complained of pain concentrated in a small area just below and internal to the acromio-clavicular joint of the same side. Infiltration of this area with novocain made no difference to the intensity or the position of the pain, thus proving that the idea of referred pain is a valid one; that it depends on events taking place in the central nervous system, and that it is not "annulled" by anæsthetizing the skin-area.

The opponents to the theory of referred pain criticized these results, as the phrenic is a somatic nerve. To overcome these criticisms, Woollard and his co-workers made a further series of studies,[12] but using the nerves of the testis. This structure is a true abdominal viscus which has migrated to a scrotal position, and a study of it therefore should answer all requirements. The method adopted in these experiments was as follows :—

The testis of one of the experimentors was drawn forwards in the scrotal sac and supported by fingers placed below it. A scale pan was then placed on the testis,

and weights placed in this pan compressed it and the epididymis between the supporting fingers and the scale pan. Known weights were placed in the pan and left there till the subject described what sensations he experienced and where he felt them. Injections of four per cent. novocain and 1 in 600,000 adrenalin were made to block the scrotal nerves, which would have interfered with the interpretation of the results. The observations thus made show that pressure on the testis produces pain in the region of the inguinal canal, in the territory of the first lumbar nerve-segment, and confirms the conception of referred pain, as first suggested by Head.[13]

The original conception of Head is that pain can be felt on the surface of the body, the occasion of which is some pathological change in a viscus, the pain in such a case being referred to the area of distribution of the cutaneous fibres of the same nerve-segment as supplies the diseased viscus. Head's observations show that, in general, neither the cervical nor the lower lumbar segments are the seat of referred pain from viscera, and from this fact it has long been concluded that the sympathetic efferent outflow is accompanied by an afferent sensory inflow, restricted approximately to the same region of the spinal cord. The segmental relations of this viscero-sensory inflow provide the anatomical basis upon which the referred pain is explained.

SUMMARY.

From the various experiments and clinical observations discussed in this paper, it would appear that abdominal pain may be a true visceral pain, the result of a diseased viscus; that it may be due to inflammatory or other pathological changes causing direct irritation of the parietal peritoneum or mesentery; or that it may be a pain referred to a cutaneous area caused by disease of either a viscus or an area of peritoneum.

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The Clinical Pathology of the Digestive Tract

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ACUTE abdominal conditions so frequently cause death, and chronic lead to so long continued ill-health and, often, dangerous complications, that it is obvious that diagnosis with a view to treatment should be made as soon as possible. It is essential that the diagnosis be as certain and exact as may be, for, in the acute case, its accuracy may be a matter of paramount importance, while, in the chronic, whether the treatment resulting be medical or surgical, it is likely to entail the patient's being away from his occupation for some weeks; if he be not on the way to recovery at the end of that time it will have been wasted.

In arriving at a diagnosis, the most important help of all is a careful account of history and symptoms. In a certain number of cases, especially in the acute, one can be morally certain of the exact pathological condition from the patient's own story, and a diagnosis which does not fit in with it should be viewed with suspicion.

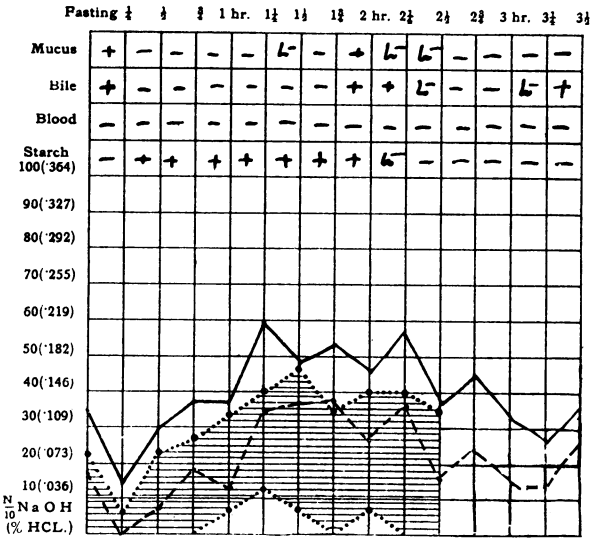
Next comes physical examination. In the acute case this is often sufficient to complete a diagnosis already considered likely, but in the chronic the assistance obtained is frequently not so great. In the latter it is advisable, if at all possible, to examine the patient on two separate occasions—when symptoms are active and again when they are quiescent. When symptoms are active, the site of pain or tenderness will be readily found. It may, however, be diffuse, covering a large area and not referable to any particular organ. If this be so, it is likely to persist and be more defined when a less active phase is present. On the other hand, if the trouble is quiescent and no symptoms are at the time complained of, physical examination may yield very scant result.

When all the information possible has been gained in these ways it should be possible to arrive at a tentative diagnosis. It is then that other methods may be called in either to confirm or negative it, and the gaze turns in the direction of radiology and the laboratory.

Of the investigations carried out by the clinical pathologist in digestive disorders, probably the most generally useful is the fractional test meal.

Fractional Test Meal.—The fractional method of investigating gastric secretion was first described in America by Rehfuss in 1914, but was not practised in this country until six years later, when the technique was introduced by Ryle. At the beginning Ryle and Bennett carried out fractional test meals on one hundred volunteer students suffering from no detectable digestive abnormality, and from the data thus obtained determined what the findings are in normal people. For this piece of work a debt of gratitude is due to them from all who are interested in the study; the limits which they placed for normal curves are generally accepted and form a standard by which aberrations may be judged.

Since then a great many investigations have been carried out by gastro-enterologists and a most voluminous literature has grown up. A certain amount of



1. CURVE FROM NORMAL PATIENT.

Free hydrochloric acid is within normal limits. Combined acid is constantly about 20. Emptying, as is indicated by the disappearance of starch, takes place in two hours. Bile is present intermittently and there is a little mucus.

The shaded area represents the limits for free HCl. (dimethyl indicator), of 80 per cent. of normal people. — — — — represents free HCl. ——— represents total acidity.

discussion has taken place from time to time, and, as might be expected, criticisms of the method have been made, some of which are :—(*a*) That the tube acts as a foreign body in the stomach. Pavlov and Beaumont proved that foreign bodies in the stomach do not alter its secretory action. (*b*) That the unappetizing nature of the meal diminishes gastric secretion. Appetite secretion is a very doubtful quantity, and many writers do not believe that it occurs at all. If it does it cannot be denied that the absence of mastication and the insipid nature of the gruel swallowed will diminish it, but this is, probably, rather an advantage, since it will render the results more comparable from one person to another. (*c*) That emotional disturbance inhibits or increases the gastric secretion during the test. The emotional disturbance is almost negligible, especially if the technique be explained to the patient beforehand and his attention distracted during the test by the reading of something interesting. The proportion of patients who have any real difficulty in swallowing the tube, or who seriously resent it, is small. In fifteen years I have come across only three patients who were unable to swallow the tube or keep it down comfortably for the required length of time. (*d*) That the secretory response is variable from day to day in the same person. When the conditions and technique of the test are exactly standardized, it has been found that there is a remarkable agreement in the curves obtained when the meal has been repeated on several occasions at intervals of a day or two. The time of emptying is very constant, and the general

conformation and height of the curve vary but little. (*e*) That the acidity is different at different stomach levels. The tube should always be kept at the same level throughout the test. In any case it would seem unlikely that, in view of the fluid nature of the gruel used and the motor activity of the stomach during digestion, very complete mixing should not take place.

There is no doubt that, whatever the theoretical objections which may be raised, in actual practice the test has proved itself of great help in clinical diagnosis. It must be remembered, however, that it only tells us what is happening during digestion and does not, as a rule, make a diagnosis. No gastric analysis should be labelled with the name of a disease, and the most it can do is to help us, when taken in conjunction with the history, clinical, radiological, and other pertinent examinations, to infer that a certain condition is likely to be present. The exception to this rule is when a gross obstruction is present and when it is generally possible to be quite dogmatic.

Occult Blood.—Another test which should be generally carried out in conjunction with the fractional test meal is the examination of the fæces for occult blood. Everyone is familiar with the black, tarry stools which follow on a large gastric or duodenal hæmorrhage. Something which is not so generally appreciated is that an ulcer, when active, although it may not give rise to as much blood as will produce a change in colour of the stools, will, as a rule, leak enough to be detectable by chemical test. Before the test is carried out the patient must be put on a special diet containing no meat, fish, fowl, or green vegetable, or anything prepared from them. As a derivative of hæmoglobin is being tested for, it must not, of course, be ingested; chlorophyll is chemically indistinguishable from hæmoglobin, and for that reason it also must be excluded.

Inquiry must also be made for bleeding gums, etc., and it is advisable that the teeth should not be brushed during the period of the test, so that there may arise no fallacy from that source.

About four days should be allowed to transpire from the commencement of the diet before the first specimen is obtained; specimens should then be examined on three or four successive days, the patient meantime remaining on the diet. The material selected should be formed, and a piece for the test taken from the centre of the mass. In this way any blood derived from low down the intestinal tract may be excluded.

In my experience the benzidin test, carried out in test tubes with reliable reagents and proper precautions, is the best for ordinary routine work.

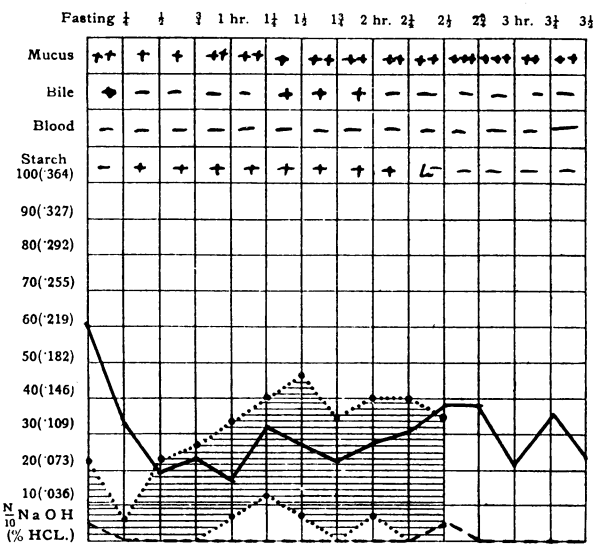
1. Gastritis.

The fractional test meal picture varies with the length of time during which the disease has been present. In the very early stages the resting juice, which normally lies between nothing and one hundred cubic centimetres in volume, may be increased and have a raised content of free hydrochloric acid. This increase of free acid may be demonstrable throughout the meal.

It is more often when the trouble has been existent for some time that a gastric analysis is undertaken. The resting juice will then be likely to be small in amount and may consist almost entirely of thick mucus, containing a considerably increased number of polymorphonuclear cells. Hypo-acidity is almost constant and, together with the large increase of mucus, is found throughout the test. Anacidity is present in about fifty per cent. of cases. Combined acid is usually increased, probably as a result of the partial combination of hydrochloric acid with mucus. In this way the hypo-acidity is also in part explainable, but, in addition, the mucus by its physical action decreases the activity of the secreting cells. It is well known that lavage of the stomach, if carried out for a period, will almost invariably cause a rise in hypo-acid curves and produce free acid in many stomachs where anacidity has previously been demonstrated. In gastritis, occult blood is occasionally found on testing the fæces, but as a rule only a faint reaction is obtained, and it is rarely persistent through a series of consecutive specimens.

2. Gastric Ulcer.

Fractional test meal findings show great variation and are often of little assistance. A great deal depends on the position of the ulcer. Those high up on the lesser curvature may give a fractional test meal normal in every respect or, perhaps, showing some hypo-acidity. On the other hand, if the site be close to the pylorus, hyperacidity is very much the rule, and a curve similar to that which is so typical of duodenal ulcer will often be obtained. Proximity of the ulcer to the pylorus seems to be the determining factor in the production of this type of curve.



2. CURVE FROM A CASE OF GASTRITIS.

Free hydrochloric acid is practically absent. Combined acidity is high. There is a large amount of mucus present in all specimens.

The lowering of the free acid curve in some cases of gastric ulcer is probably caused by the gastritis which is a constant accompaniment. The presence of excess mucus points to this, and lavage has been found to bring about a rise to normal or even a hyperchlorhydria, dispelling the mucus at the same time. Occult blood tests are generally positive when the ulcer is active, as may be judged by pain and discomfort. One may often safely secure activity in a quiescent case, for the purpose of the test, by getting the patient to eat food and do things which are known to produce symptoms.

3. Carcinoma of the Stomach.

The fractional meal findings depend on (*a*) the type of the lesion, (*b*) whether it is producing obstruction, and (*c*) whether it presents an ulcerating surface in the gut cavity.

(*a*) Many sufferers from gastric carcinoma are found to have achlorhydria, and this is especially likely in the diffuse "leather-bottle" type.

(*b*) Obstruction is generally accompanied by a marked increase in the combined acid, and when the obstruction is of high degree the findings are generally characteristic. The resting contents are then often large in amount and foul-smelling, contain charcoal and food ingested twelve or more hours previously, shreds of tissue, and, sometimes, blood. There is little or no free hydrochloric acid and no bile. During the meal there is generally achlorhydria with high combined acid, persistent starch, and no bile. At the end of three hours the greater portion of the meal will be aspirated from the stomach.

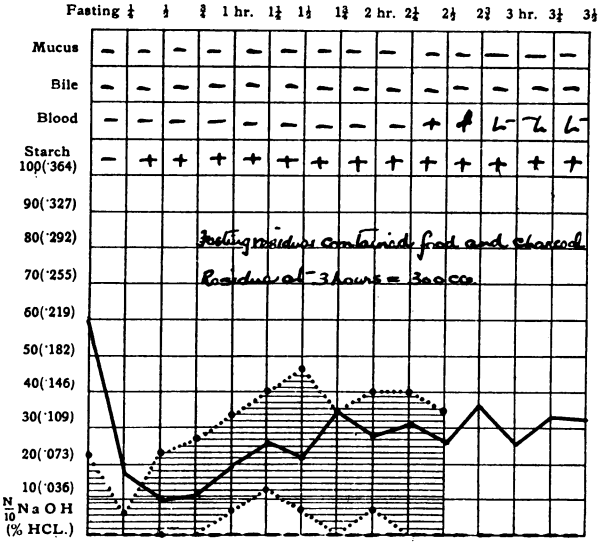
Obstruction caused by cicatrization of an ulcer does not, as a rule, give the same foul mess, and the combined acidity is not so high.

(*c*) When there is ulceration of the growth, an alkaline fluid exudes from the raw surface, and it is the combination of this with gastric juice which probably produces the high combined acidity. There is also constantly some bleeding, greater or less in amount, often giving rise to the presence of gross blood in the specimens and positive occult blood reactions on testing the stools.

4. Duodenal Ulcer.

It is in duodenal ulceration that gastric analysis is most frequently helpful in ordinary everyday clinical work. Duodenal ulcer is a comparatively common condition—much more common than gastric ulcer, for example, among cases which are seen by the consulting physician. This is surprising, for at autopsy scars of chronic ulcers in stomach and duodenum seem to be found with almost equal regularity. It is possible that either gastric ulcers are more frequently healed by simple measures or that they give rise, on the average, to less pain and inconvenience and are tolerated with less complaint.

The typical curve found in duodenal ulceration without obstruction presents the following characters. The resting juice is very variable in amount, being sometimes up to 100 c.cs. or more: it is usually perfectly clear, without mucus, and highly acid. Dilution with the gruel produces a fall of the acid to, or approaching



3. CURVE FROM A CASE OF PYLORIC OBSTRUCTION CAUSED BY CANCER.

No free hydrochloric acid. Combined acid is high. The character of the fasting residue and the large amount remaining at the end of three hours are of importance. Blood was present in the later specimens and starch throughout the test. No bile was present.

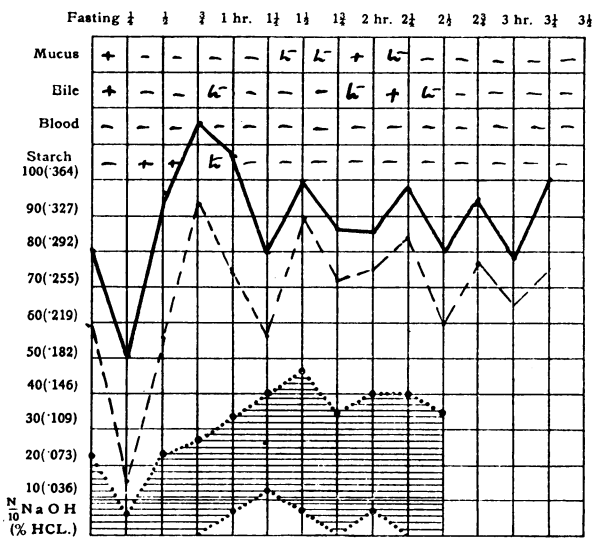
nothing, but thereafter there is a steady rise to a high figure, often 70 or 80 and sometimes exceeding 100 c.cs. Emptying, which is indicated by the disappearance of starch, is, as a rule, rapid, being often complete in an hour or little more. In spite of this there is often no fall in acidity, indicating the failure of regurgitation from the duodenum, and continued after-secretion produces a sustained high plateau which may go on indefinitely if no further food be ingested. It is important in all fractional test meals to try to obtain specimens over the full period of three hours. This is especially so when duodenal ulceration is suspected, otherwise the high acid plateau of secretion after the stomach has emptied may be missed. It is the continued hypersecretion of this highly acid juice, together with the hyperperistaltic stomach and spastic pylorus almost always associated with it, which prevent regurgitation of the alkaline duodenal contents, and so determine the high continued acidity in the stomach. For the same reason, bile is rarely present in many of the specimens or to any great amount.

Where a considerable degree of obstruction is present the picture will necessarily be modified. The resting juice will be large in amount, depending on the degree of obstruction, and may contain some of the charcoal swallowed twelve hours previously. Partially digested food may even be present. Owing to the volume of the stomach contents, and also to the gastritis which the retention gives rise to, the acid curve will not be so high. Bile is likely to be completely absent, and a large portion of the meal will be aspirated when the stomach is emptied at the end

of three hours. The bulk may, indeed, be in excess of the pint of gruel swallowed, owing to hypersecretion of gastric juice.

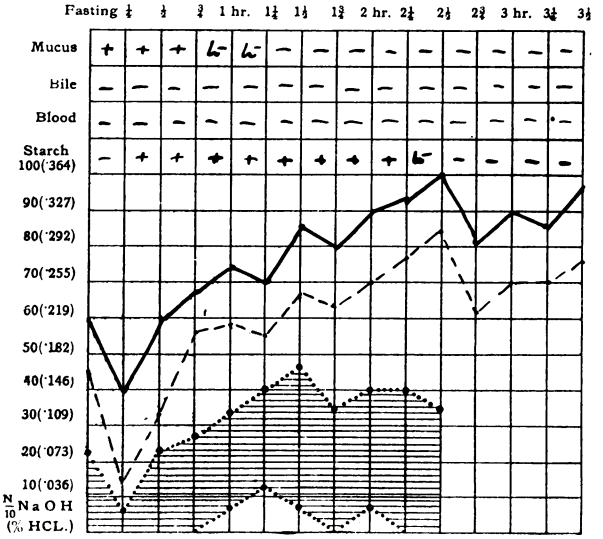
Occult blood tests can be of great help where duodenal ulcer is suspected. Disease in other organs may give rise to symptoms very similar to those produced by it—chronic appendicitis, abdominal adenitis, chronic cholecystitis, for example—and often there seems to occur a condition which, in the absence of very definite knowledge, we refer to as "duodenitis," which is almost indistinguishable clinically. This last may give very suggestive radiographic appearances, and the fractional meal is often typical of duodenal ulcer, as the same pylorospasm and hypertonic stomach often accompany it. In these cases it is of great importance to find the stools giving a series of absolutely negative reactions when tested for blood, especially if the specimens have been obtained during a period when symptoms were active. The presence of constantly strongly positive tests, on the other hand, renders ulcer very likely to be the correct diagnosis.

The test meal findings in duodenal ulcer often give help in choosing cases for the operation of gastro-jejunostomy. It has been found that the cases which give the best results are those showing slower emptying and absence of hypertonus, as indicated by a not excessively high acid curve. On the other hand, the stomach which empties in about an hour, and continues to pour out a highly acid secretion, is unlikely to do well. Emptying is likely to be even more rapid and the acid curve may become even higher after the operation than it was before. Little or no relief of symptoms will result, and conditions are ideal for the production of an ulcer at the junction. Gastro-enterostomy was originally performed only for the relief of



4. CURVE FROM A CASE OF DUODENAL ULCER WITH EXTREME HURRY.

Stomach is empty in three-quarters of an hour. Free hydrochloric acid is very high, and remains so after the stomach is empty.



5. CURVE FROM A CASE OF DUODENAL ULCER WITH NORMAL EMPTYING TIME.

Free hydrochloric acid is high, and remains so after the stomach is empty.

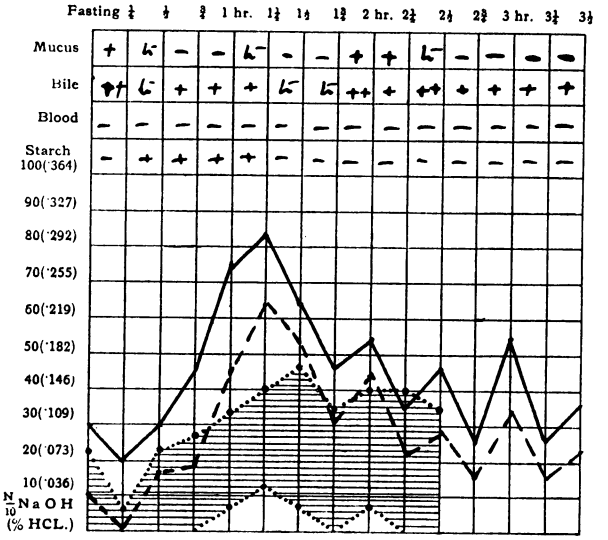
organic obstruction. The results were remarkable, and remain so where this mechanical condition, or something approximating to it, is present. The further the case departs from it the less remarkable is the result likely to be. When duodenal ulcer occurs in the highly-strung, energetic, athletic type of man, whom the Americans would describe as "a real go-getter," and in whom the fractional test meal gives a curve rising rapidly to a high peak, with early emptying and sustained after-secretion, then gastro-jejunostomy is not so likely to be successful in curing the patient, and thorough medical treatment is especially indicated.

5. Gastro-Jejunal and Jejunal Ulcer.

These when they occur are usually the sequelæ of gastro-enterostomy performed for duodenal ulcer. The fractional meal findings are likely to be similar to those of the duodenal ulcer for which the short circuit was carried out. It is often very difficult to obtain specimens for examination, however, as the stomach is very irritable and the liquid meal is rushed through the stoma almost as soon as it appears in the viscus. This difficulty will not arise, of course, if much cicatrization has produced obstruction of the stoma.

6. Cholecystitis.

There is no characteristic fractional test meal curve associated with cholecystitis. One frequently finds an achlorhydria present, and Hurst believes that it often precedes the cholecystitis, the latter arising as a result of the absence of disinfection of the stomach and first part of the small intestine by hydrochloric acid.

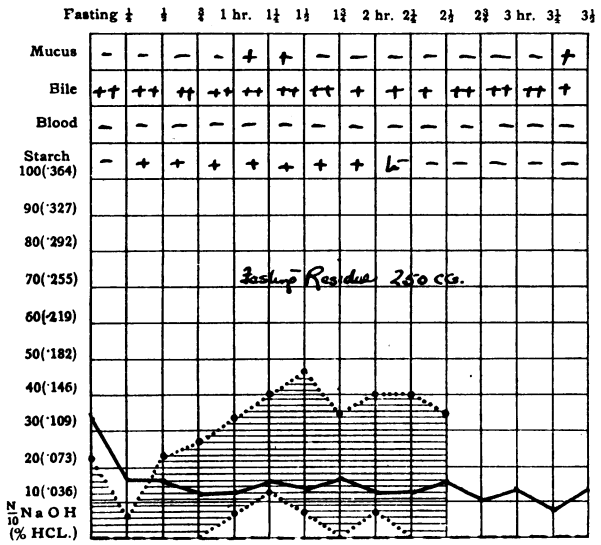


6. CURVE FROM SAME CASE AS FIG. 5, FOLLOWING GASTRO-ENTEROSTOMY.

There is a good regurgitation of duodenal contents, reducing the hyperchlorhydria both in degree and duration. The period of emptying is shortened from two and a quarter to one and a quarter hours. Bile is present throughout the test.

In normal people, when there is a normal secretion of hydrochloric acid, the contents of the duodenum are generally found to be sterile, but in its absence bacillus coli and enterococci can easily be cultured. It might be expected that these organisms would find their way back into the bile ducts and gall-bladder and, if the local resistance happened to be lowered in any way, set up a cholangitis or cholecystitis.

Duodenal Intubation.—Further information as to the presence or absence of pathological conditions in the gall-bladder may sometimes be obtained by duodenal intubation. By this means bile may be obtained for examination as it is discharged from the common bile duct. When the tube has passed into the duodenum, and reached the vicinity of the ampulla of Vater, amber-coloured bile is first syphoned off. Some solution such as saturated magnesium sulphate in water, which, when it comes in contact with the ampulla or duodenal mucosa will cause a contraction of the gall-bladder, is then injected, and as much of it as possible withdrawn again. This is succeeded by more light amber bile, probably from the common duct, to be followed in turn by dark viscid bile which comes from the gall-bladder. Of this last, careful examination should be made as to the presence and nature of deposit and micro-organisms. Frank pus or even any appreciable number of bile-stained leucocytes indicate an active inflammatory process. In the early catarrhal stage there will often be found numbers of high columnar epithelial cells deeply stained by bile and of gall-bladder origin. The presence of crystals of cholesterol, calcium or bilirubin, or of a large amount of bile-stained epithelial debris, is strongly suggestive of



7. CURVE FROM A CASE OF "VICIOUS CIRCLE" VOMITING FOLLOWING GASTRO-ENTEROSTOMY.

Free hydrochloric acid is absent. Bile is present in very large amounts throughout the test. In spite of the normal emptying time, there was a large amount of fluid present in the stomach at the beginning of the meal.

calculus formation. The importance of the epithelium, etc., being bile-stained, is that it indicates the origin of the material to be in the biliary tract, while that coming from the stomach, duodenum, or pancreas is not so discoloured.

I do not think we are on such safe ground when we come to deal with bacteriological examinations. The method by which the test must be carried out is anything but aseptic. In the mouth and pharynx the tube must be grossly infected during swallowing, and although various methods of preventing this, such as that of filling up the orifices in the bulb with paraffin wax and expelling it by air pressure when the tube has entered the duodenum, have been tried, they have not produced much improvement. When achlorhydria is present, I do not think any great reliance can be placed on cultural findings alone, and only the presence of gross infection, as demonstrated by examination of stained direct preparations, and proof by subsequent culture that the organisms are viable, can be taken as evidence of bacterial pathology. In material obtained by duodenal intubation one frequently finds a gram positive diplococcus, in appearance similar to the pneumococcus, present in large numbers, but which does not grow on culture under ordinary conditions. It is probably a pneumococcus derived from the mouth and pharynx which has been rendered non-viable, either by hydrochloric acid in passage through the stomach or by bile in the duodenum.

When hydrochloric acid is present in the stomach, one ordinarily obtains specimens from the duodenum which are not grossly contaminated, and the finding

of any viable organism in fair numbers, either on direct examination or culture of the bile on a solid medium, is very suggestive of a definite gall-bladder infection.

Just as the failure to find tubercle bacilli in a slide prepared from a sample of sputum does not exclude this infection, so negative findings from a duodenal intubation do not exclude disease of the gall-bladder; the examination merely provides one point which may help in coming to a conclusion when the whole picture is considered.

Van den Bergh Reaction.—The Van den Bergh reaction has not proved of so much practical help in the diagnosis of biliary obstruction as had at first been hoped. When an increase of bilirubin in the blood is caused by obstruction to the flow of bile, with reabsorption, it gives what is known as a "direct" Van den Bergh reaction: when it is due to an increased production of bile, as in certain blood diseases, it gives an "indirect" reaction: when damage to liver cells is the causation it gives a "biphasic" reaction. In this way it would appear easy to determine the cause of jaundice in any particular case, but, unfortunately, when obstructive jaundice has been present for more than a day or two, a secondary hepatitis supervenes, the liver cells themselves becoming deranged and giving rise to a further increase in bilirubinæmia. The biphasic reaction thus produced masks any direct reaction present, so that it is only in the first day or two that an obstructive jaundice can be distinguished from one of liver origin. That it should be so is unfortunate, as the determination of the origin of a jaundice as to whether it is primarily obstructive or hepatic is one which has frequently to be made and is often of the greatest importance. The test is of great service, however, in deciding whether there is any increase of bilirubinæmia, and, if so, whether it is of hæmatogenous origin in contra-distinction to biliary.

7. Appendicitis and Abdominal Adenitis.

Acute appendicitis is frequently diagnosable with the greatest certainty, but in atypical cases doubt sometimes arises. This seems to be especially so when the stage of abscess formation may have been reached, and it is then that a leucocytic count may be of service. When the white-cell count of the blood rises to 15,000 per cubic millimetre, with a high percentage of polymorphonuclears, it is probable that pus is present, and when it reaches 20,000 it is pretty certain to be. Unfortunately, pus does not always produce a considerable leucocytosis, so that it is only the positive finding which is of value.

In chronic appendicitis and abdominal adenitis, the fractional test meal results are very variable. When the symptoms are suggestive of duodenal ulceration, there is often obtained a curve which also might denote this condition, though marked hyperchlorhydria with rapid emptying is uncommon. More frequently the curve will be of the type slowly mounting to a moderate height, with starch still present in the stomach at three hours. In appendicitis, occult blood tests are generally negative, though in adenitis they are often positive, possibly because of an associated congestion of the mucous membrane. Abdominal adenitis is a most difficult

condition to diagnose clinically with any feeling of assurance. It most often simulates appendicitis or duodenal ulcer. X-ray examinations and laboratory tests are rarely of much assistance, but the obtaining of a well-marked tuberculin reaction in the absence of signs of any other tuberculous infection lends weight to a clinical diagnosis.

8 Pancreatitis and Pancreatic Tumours.

In acute pancreatitis the pathologist is often able to clinch a diagnosis already suspected, and so avoid a laparotomy which may easily be disastrous. The urinary excretion of diastase is in normal health remarkably constant, lying between ten and twenty units. In acute pancreatitis it practically invariably shows a considerable increase, and may rise up to two or three hundred units. As the test takes only about three-quarters of an hour to carry out, it is of great practical assistance.

Some alteration in the urinary diastatic content may be found in chronic pancreatitis, it being sometimes more, sometimes less. Frequently, however, the figure obtained is within normal limits.

Chronic pancreatitis and neoplasm generally show themselves by a failure of digestive secretion, and if the stool be examined while the patient is on an ordinary mixed diet it will be found to contain pathological amounts of starch, undigested muscle-fibre, and fats. The muscle can be seen by direct microscopic examination, as can also the starch after staining with iodine. By various dyes the fats can often be shown up also if there is a total increase, but it is better to do an exact quantitative analysis, determining total fat, unsaponified fat, neutral fat, and free and combined fatty acids. The total fat is often trebled, and a large proportion of the increase will be found in the neutral fats, owing to defective fat splitting. In obstructive jaundice the total may be equally increased, but, since the fat-splitting action is normal, the increase is in the fatty acid fraction.

9. Diseases of the Large Intestine.

In the large intestine the only two conditions in which the laboratory is directly helpful in diagnosis are malignant disease and acute diverticulitis — malignant disease only when there is ulceration into the lumen of the gut and when an occult blood reaction is obtained, and acute diverticulitis when inflammation of a diverticulum has produced pus and a polymorphonuclear leucocytosis is likely to be found.

This brings us to the end of the important pathological conditions of the digestive organs in the elucidation of which the clinical pathologist can assist. Laboratory tests can be of considerable help so long as it is realized that, for the most part, they are not of themselves diagnostic, but, like other methods of examination, simply provide points of evidence by the co-ordination and assessment of which a diagnosis may be made.

ULSTER MEDICAL SOCIETY

ANNUAL MEETING

THE annual meeting of the Ulster Medical Society was held on 23rd May, 1935, in the Whitla Medical Institute, at 5 p.m. The president, Dr. S. R. Hunter, occupied the chair.

The minutes of the last annual meeting were read and signed. The president then called on the secretary, Dr. F. P. Montgomery, to read the Council's report, which was as follows :—

The Council begs to present the seventy-third annual report of the Society.

The roll of the Society now stands as follows :—

	THIS YEAR	LAST YEAR
Hon. Fellows - - -	7	7
Life Fellows - - -	13	13
Fellows - - -	271	273
Members - - -	59	53

Total 350, against 346. In 1931 the total was 263.

During the year the Society has suffered the following losses by death :—Dr. Mark Cahill, Fellow since 1909; Dr. W. S. Gibson, Fellow since 1931; Dr. J. C. Loughridge, Fellow since 1920; Dr. Mark McDonald, Fellow since 1920; Dr. E. F. O'Connor, Fellow since 1930.

Ten meetings of the Society have been held during the year, and the attendances have been very satisfactory.

Among the speakers were Dr. A. E. Barclay, of Cambridge University, and Professor Preston Maxwell, of Pekin University.

An innovation was the reading of a series of short papers on diagnosis and treatment, which seemed to meet with the approval of the Society.

The laboratory meeting produced what was probably the finest exhibition of pathological specimens ever shown to the Society, all of them excellently classified with suitable clinical notes and radiographs.

The annual dinner was again held in the Medical Institute, and although the numbers were small, it was a most successful function.

The Hanna Golf Cup was played for over the links of the Donaghadee Golf Club, and was won by Mr. P. T. Crymble.

During last summer, the whole of the outside and inside of the Institute was painted, and repairs to the fabric were carried out and alterations were made in the living quarters of the caretaker. New curtains were provided in the lecture hall and the downstairs hall, the total expenditure being about £371. The result has been most satisfactory, and the whole Institute is now in an excellent state of repair.

Your Council has met on ten occasions, and the following attendances are recorded :—

The president, 8; Dr. Rowland Hill, 3; Dr. McCloy, 0; Prof. Lowry, 2; Prof. Thomson, 2; Dr. Monypeny, 1; Dr. Marshall, 5; Dr. Frackelton, 7; Dr. H. Stewart, 5; Mr. Calvert, 7; Dr. Kean, 9; Dr. Hunter, 2; Dr. J. A. Smyth, 4; Dr. Lyttle, 2; Sir T. Houston, 1; Dr. Allen, 5; hon. secretary, 10.

On the proposal of Dr. F. P. Montgomery, seconded by Mr. H. L. H. Greer, this report was passed. The honorary treasurer's report was then presented by Mr. C. A. Calvert, who drew attention to the apparent deficiency of about £180 on the year's working. This was mainly due to the renovation of the Institute, which cost about £371. If this expense had not been met, the year's working would have shown a clear profit. The Society is thus seen to be in a very sound financial position. The report was passed.

The honorary librarian, Dr. T. A. Kean, presented the following report, which was passed :—

Since the last annual general meeting the library has been redecorated, and the bookcases are now fitted with glass doors. As a result, the books are kept cleaner, and the appearance of the library is more pleasing.

Mrs. Fullerton has generously presented us with 216 volumes from the library of the late Professor Fullerton. Many of these books are of permanent value, and form a much appreciated addition to the library. The Council decided that this collection should be kept together and be known as "The Andrew Fullerton Collection," in memory of a distinguished and beloved Fellow of the Society.

In conjunction with the library of Queen's University, all the leading medical journals are available for the use of members. Members continue to make use of this facility.

New book-plates have been provided, and, it is hoped, the design will be found more pleasing than that formerly in use.

The election of office-bearers for the session 1935-6 was the next business, and the following were elected :—

President : Dr. Foster Coates. Vice-Presidents : Dr. Harold Grey and Dr. George Campbell (Bangor). Hon. Treasurer : Mr. C. A. Calvert. Hon. Secretary : Dr. F. P. Montgomery. Hon. Editorial Secretary : Dr. H. H. Stewart. Hon. Librarian : Dr. R. S. Allison. Council : Professor W. W. D. Thomson, Professor J. S. Young, Dr. Frackelton, Dr. Robert Marshall, Dr. James Boyd, Mr. H. L. H. Greer. Editorial Board : Professor P. T. Crymble, Professor R. J. Johnstone, Professor W. W. D. Thomson, Dr. H. J. Ritchie. Honorary Acting Editor : Dr. R. H. Hunter.

H. H. STEWART,
Hon. Editorial Secretary.

18 Malone Road, Belfast.