heart. Here is where the great difficulty in interpreting variations of rate comes in. The sino-auricular node without vagus influence would cause an increase in the heart's rate. When, then, we find an increase in rate, we have to ask whether it is due to the sino-auricular node being freed from vagal activity, or to an increase of the sympathetic influence, or to a direct effect of some agent (temperature, toxins, drugs) on the sino-auricular node itself. These are the types of questions that require settling before we can understand the action of remedies or the symptoms of disease.

We know already a little, but that little indicates that there is much more to be known. We know, for instance, that the increased heart rate that follows the administration of atropine is due to a suspension of vagal activity, and that the increase in rate is different from that which follows a rise of temperature or effort, or the administration of alcohol. It is also known that digitalis may have a definite effect on the vagus, stimulating it and so restraining the activity of the sino-auricular node.

Varied as are the effects of drugs diseased states have effects as distinctive, for agents of disease affect the reflex system in diverse places and diverse ways, and it is by the recognition of these diverse modifications that the different diseased states can be recognized. We know that the reaction is different in pneumonia from that in typhoid fever, and these again from that in rheumatic fever. During some infections the conducting system may be affected, as shown by the temporary presence of partial heart-block.

But there is more than that, for the agent of disease modifies or counteracts the action of drugs. Thus, though in the normal heart atropine increases the rate, yet in certain diseased states it may slow the heart's rate. In some forms of disease digitalis will slow the heart rate, in other diseased states it is without effect. The recognition of these facts explains the reason of the confusion which reigns in the therapeutic field. A great deal of work has been done in testing the action of drugs on the healthy heart, but it has not been realized that the diseased condition modifies the effects of the drug. When it is realized that it is chiefly on some part of the reflex process that most drugs act the importance of a better understanding of this process will become apparent, all the more so when it is further realized that it is also by the modification of some parts of this process that many symptoms of disease arise. If these points are grasped it will be seen that in the sick man the agents of disease have affected the parts on which the drugs act, and therefore we can state as a principle which must be recognized in the investigation of the action of drugs that *the effect of a remedy is modified according to the nature of the disease*. It is the neglect of this principle which keeps therapeutics in the state of confusion in which they are at present.

Other reactions have to be considered. R. M. Wilson has lately drawn attention to a factor that has not been recognized—the secondary reaction to a stimulus. In a balanced reflex, when an act is regulated by two nerves acting the one in opposition to the other, the excitation of one is accompanied by the diminished action of the other. When the excitation ceases the nerve whose activity was diminished may greatly increase its activity. Thus a temporary slowing of the heart by stimulation of the vagus may be followed by a great increase in the rate of the heart by secondary reaction of the sympathetic (a reflex stimulation of the sympathetic by the vagus).

Abnormal Rhythms.

If the understanding of the reflex system is of importance with the normal rhythm, it is evident that with an abnormal rhythm it is of still greater importance. The various places where the heart beat may start are different in structure and in function from that of the normal starting place (the sino-auricular node), and in consequence the effects upon the ventricles are different. This was so fully dealt with in the previous article that I need not dwell upon it here, save to refer to the differences which follow effort in the cases, say, of the normal rhythm, auricular fibrillation, and heart-block, and to the difference of the effect of drugs like digitalis. Moreover, the fact that the stimulus which is produced by the cells of the contracting system—as in auricular fibrillation, flutter, and the idio-ventricular rhythm—is different from that which arises in the cells of the conducting system calls for a reconsideration of the mechanism of these abnormal rhythms and of their relation to the various agencies (effort, drugs, heat, toxins of disease) which act upon them.

SUMMARY.

1. The mechanism concerned in regulating the heart beat belongs to the vital process included under the term "reflex."
2. The cells taking part in this mechanism belong to two groups: an extrinsic, connected to the nervous system by the vagus and sympathetic nerves, and an intrinsic, which includes the sino-auricular and auriculo-ventricular nodes, and the Purkinje cells in the bundle and auricles and ventricles.
3. Agents of disease (toxins) which modify the rate and rhythm act on different parts of this mechanism, and certain disease agents produce characteristic effects.
4. Drugs that influence the rate and rhythm of the heart act in the same way, some of them also producing characteristic effects.
5. As toxins act on the same parts as drugs the effects of drugs will be modified by the effects of the toxins.
6. Before the effects of a remedy can be investigated it is necessary to know if the toxins have not already produced an effect which will interfere with the action of the remedy.
7. The kind of knowledge which disturbances of the reflex process reveals is limited to the variations in rate and rhythm, and they give no information regarding the functional efficiency of the heart.

THE PEPTIC ULCER.

WITH A REVIEW OF TWELVE MONTHS' SURGICAL TREATMENT (1921).

BY

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To obtain control over any disease it is necessary to possess means which can be safely employed to eradicate it when established, and a knowledge of its etiology such as will enable us to prevent its occurrence or recurrence. Surgical experience has made a very substantial contribution towards the mastery of peptic ulcer. It has contrived means of dealing radically with ulcers which are safe, at least in practised hands and when employed with discrimination. In the course of arriving at its technique it has thrown a great deal of light on the conditions which determine peptic ulceration, and which, if undisturbed, will tend to its recurrence. I will argue later that with the assistance of radiology it has now actually reached an understanding of the immediate cause of ulcer, and has determined, although not exhaustively, the ulterior causes.

Among the contributions which radiology has made to medicine none is more brilliant than that which it has made to the diagnosis of gastric disorders. Without its assistance the diagnosis of ulcer uncomplicated by penetration or haemorrhage or stenosis was only a matter of suspicion. Now suspicion can be put to a test in which the margin of error is, in my experience, extremely small. I wish to express my gratitude to my colleagues, Dr. Oram and Dr. Mather, to whom I owe the reports on the cases which I propose to review.

Of these cases, 48 in number, on whom I operated in 1921, 41 were men, 7 women. In 34 instances the ulceration was gastric, in 8 duodenal, and in 6 jejunal. These figures do not include other simple disorders of the stomach, such as those which depend on adhesions or those due to pylorospasm without ulcer, but I wish to refer shortly to these two classes.

Adhesions Involving the Stomach.

One is impressed with the gross disturbance of function and the simulation of visceral disease which may be caused by such adhesions. I have opened the abdomen on four occasions during the year in cases of this kind, the patient in each case being incapable of following any occupation. In all four the adhesions were chiefly between the stomach and the anterior abdominal wall deep to an operation scar. According to the histories obtained the earlier operation had been as follows:

1. For gastric ulcer (evidence of operation on pylorus).
2. For perforated gastric ulcer (suture).
3. For gastric ulcer (gastro-enterostomy).
4. For perforated duodenal ulcer (suture).

Infection is the cause of such adhesions. When formed between the stomach and the abdominal wall they may cause

pain of severe intensity associated with the period of digestive movement, and pain is also characteristically complained of when exercise is taken. In dealing with them the omentum is of service; after separation the whole area concerned is covered by omentum fixed by sutures in whatever directions it may be desirable. The fourth case deserves special notice.

A man aged 46 on whom, at the age of 31, I operated for perforation of a duodenal ulcer. In 1918 I operated again and found an ulcer on the lesser curvature of the stomach; a posterior gastro-enterostomy was then done. In June, 1921, he was again admitted complaining of sharp stabbing pain in the left hypochondrium. I found the gastro-enterostomy opening in good order, the ulcer of the lesser curvature healed, and no ulceration elsewhere. The stomach was, however, attached to the anterior abdominal parietes, and also to the liver. These adhesions were dealt with as detailed above, and when I saw him six months later he told me that he had been free of all pain or discomfort.

Pyloric Spasm.

Cases of pyloric spasm and pyloric hypertrophy do not strictly belong to my subject. In four cases seen during the year ulcer was suspected either on the ground of clinical signs or of x-ray report, or of both. In all four a pyloric incision was made to demonstrate definitely whether ulcer was present or not. The operation was completed as a pyloroplasty. Relief of symptoms and increase of weight have followed in each case. Ulcer was simulated clinically in all four; in two the radiological diagnosis was also ulcer, in one the report said simply "marked delay," and in the fourth the stomach was reported normal in outline and function.

PEPTIC ULCER.

The forty-eight cases of peptic ulcer fall into six groups:

Group 1.—Perforated duodenal ulcer; two cases. Treated by infolding suture, gastro-jejunostomy, and pelvic drainage.

Group 2.—Ulcer of the lesser curvature, treated by ulcer excision alone or by excision and gastro-jejunostomy. This group includes three cases only. For reasons which will be discussed later ulcer excision alone is not a satisfactory operation.

The first case illustrates the disappointing result of this operation, an experience which has been recorded by other surgeons. It was adopted in this case with a view to shortening the operative procedure as much as possible, but other experience has shown me that it is never advisable on this ground and that there are better means of dealing with patients who are much reduced in strength by their disease. In this case, eight months later, I supplemented the ulcer excision by gastro-enterostomy. Gastric pain had persisted and vomiting was frequent. At this second operation, while no ulceration was found, the stomach was seen to be somewhat deformed at the site of the excision and this was apparently the cause of his symptoms. Since the supplementary operation he has done well.

In the second case of the group the ulcer was excised on account of repeated serious haemorrhage. A blood transfusion made operation possible; in the floor of the ulcer was a large vessel showing a perforation temporarily plugged by clot.

In the third case the operation was chosen, as in the first, because the general condition of the patient did not seem to admit of gastroectomy. Excision was, however, supplemented by gastro-jejunostomy at the same time and from this combination better results are obtained than by excision alone.

Group 3.—Comprises ten cases treated by posterior gastro-enterostomy alone. In six instances the lesion was duodenal ulcer, in three gastric ulcer, in one pyloric stenosis.

Group 4.—Comprises two cases in which the operation was transgastric suture of the ulcer. In one case I had to deal with an ulcer near the cardia in a stomach which lay high up under the diaphragm. The stomach was small and the whole of it lay under the ribs, and the ulcer area was inaccessible except by gastrotomy.

The other case is somewhat remarkable in that both I and another surgeon opened his abdomen, the clinical and radiological diagnosis being ulcer, and found none. Six months after the operation, which I performed and in which adhesions appeared to be the cause of his symptoms, he was readmitted after a massive haemorrhage. I found an ulcer high on the lesser curvature, difficult of access, and also penetrating. It was sutured after gastrotomy.

The clinical result in these two cases has been satisfactory for the time being, but the proceeding is probably not radical enough for a permanent result. The condition of these two patients is, however, surprisingly good, as far as complaint is concerned.

Group 5.—Includes the cases, twenty-five in number, who were treated by partial gastrectomy. In three cases the operation was a "sleeve" resection. In the remainder the operation was what is most commonly described as the

posterior Polya or Reichel operation—that is to say, resection of the stomach from the pylorus to beyond the ulcer-bearing area, closure of the duodenum, and anastomosis of the stomach wound to the jejunum through a mesocolic gap.

Group 6.—This group consists of six cases of jejunal ulcer. In these cases no one procedure is always suitable. The ideal procedure, as carried out in one case, is as follows: The pylorus was divided and the duodenum closed; the stoma was resected and the gastric and jejunal wounds left by this resection were closed. The pyloric half of the stomach was then resected and the stomach wound was anastomosed to a jejunal loop at a point about eighteen inches from the jejunal commencement, the anastomosis being retro-colic. The other procedures were:

- (a) Two cases. Resection of the stoma; gastro-jejunostomy in Y.
- (b) One case. Resection of stoma; end to-end anastomosis of jejunum.
- (c) Two cases. Resection of stoma; gastric and jejunal wounds closed.

Simple resection of the stoma with closure of the stomach and jejunal wounds is obviously only suitable when no ulcer or stenosis exists elsewhere. If there is ulceration or stenosis in the stomach I think the operation is best completed by resection after the proceeding of Polya, or by a long loop gastro-jejunostomy. I do not like the Y gastro-jejunostomy; it is technically difficult after resection of the stoma, and has the worse fault of giving no safeguard against a reappearance of jejunal ulceration.

Among the forty-eight cases of the several groups there was no mortality, but two cases gave rise to some anxiety.

The first was a difficult case of partial gastrectomy for ulcer toward the cardiac end of the lesser curvature. A cigarette drain was used. On the sixth day there was a definite leakage of gastric secretion; jejunostomy was performed and mouth feeding stopped. The sinus closed in twenty-four days.

The second case was one of sleeve resection for mid-gastric stenosing ulcer. A fortnight after operation he showed abdominal distension which proceeded to complete obstruction on the eighteenth day. A volvulus of the ileum was found and released.

The necessity for operative treatment of peptic ulcer, apart from complication, arises from its resistance to other forms of remedy. The conditions of a peptic ulcer may be said to be three:

1. The ulcer involves the visceral wall alone; it is non-penetrating, non-stenosing.
2. The ulcer is penetrating.
3. The ulcer is stenosing.

Opinions do not differ as to the necessity for operation in the stenosing ulcer; few will expect a favourable result in penetrating ulcer without operation, but there is evidence that many non-penetrating ulcers will heal under general and local medical treatment. Up to the present it is not possible to base such treatment on an etiology that is demonstrated and acknowledged. That peptic digestion plays a definite part in the etiology is admitted, but pathologists are still searching for some antecedent condition rendering the gastric mucous membrane vulnerable to attack by the gastric juice. It is by no means certain that there is any such general antecedent lesion, or, in fact, that there are any causative factors other than an altered gastric secretion and an altered gastric rhythm. Any theory of the causation of this ulcer must explain its incidence in stomach, duodenum, and jejunum. The jejunal ulcer is illuminating. It shows that the normal jejunal wall submitted to the direct action of the gastric juice may suffer destruction. I am aware that many surgeons believe that only a jejunal wall damaged by the presence of a foreign body, such as an unabsorbable suture, is liable to peptic attack. There is undeniable evidence to the contrary, and the six cases here recorded add to it. In one of the cases a fragment of silk was found near the ulcer site, but not actually at the ulcer; in the other five cases there was no such foreign body. It is said that damage done by a clamp may be responsible for the ulcer, but two considerations negative this being a usual factor—the one that jejunal ulcer does not commonly occur at the clamp line, the other that many jejunal ulcers make their appearance long after any possible clamp injury must be presumed to have disappeared. As far as jejunal ulceration is concerned there is evidence that, given a certain chemical state of the gastric secretion, the normal jejunal mucous membrane will succumb to peptic digestion. I submit that the same holds good for the stomach and duodenum, and that if we have regard to the physiology of the gastric secretion, and more particularly to its intimate relations to pancreatic secretion,

the occurrence of gastric, duodenal, and jejunal ulceration, under conditions which disturb these relations, is explained.

The stomach consists of two segments, a fundal segment and a pyloric segment. The chemistry of these two segments may differ materially at any given moment; the fundus is occupied with salivary digestion in the early stages; the gastric secretion poured out from border cells and gland cells accumulates in the pyloric segment, and it is here that peptic digestion takes place. Langley compares the peptic activity in the pyloric segment with that in the fundal segment as 73 to 1. The pylorus is not a closed orifice during normal digestion. Quite early in the process small quantities of gastric juice and food pass into the duodenum, and in the cells of the duodenal mucous membrane the hormone of pancreatic secretion is formed. There is also a regurgitation of pancreatic secretion into the stomach. Rehfuess, using trypsin as an indicator of this regurgitation, has shown that it is regularly associated with normal digestion, and that the trypsin value is high in the presence of low acidities and low when the stomach contents are of high acid concentration. Boldyreff believes that gastric acidity is lowered to an optimum by this passage of pancreatic fluid. However that may be, pancreatic secretion, which on the one hand is evoked by the gastric secretion, on the other hand acts as a regulator of the activity of peptic digestion. There is thus a correlation, diphasic, between gastric secretion and pancreatic secretion. *This correlation is dependent on an intact pyloric mechanism.* We must not indulge the notion that it is the free acid of the gastric juice which is the noxious agent in producing gastric or duodenal ulcer. Free hydrochloric acid is absolutely innocuous in the concentrations in which it is met with in normal or pathological stomachs. It is merely a rough indicator of peptic activity; in fact, it can hardly be said to be even this, for the ratio of peptic power is not proportional to the acidity. What we can say is that, generally speaking, peptic attack only occurs in an acid medium.

Now we have been shown by the radiologist that in both gastric and duodenal ulcer there are certain departures from the normal in the motor functions of the stomach and the pylorus; that in gastric ulcer the stomach is characteristically hypotonic and retains its contents for an undue length of time; that in duodenal ulcer the stomach is hypertonic, and that there is hurry in emptying. In both cases there are certain departures from the normal in the pyloric mechanism. Hurst says that these motor aberrations are present before ulcer appears and persist after it has ceased to be clinically apparent. In other words, that they are primary, or at any rate precede and are independent of the presence of ulcer. What will be the effect of such aberrations on the correlation of pancreatic and gastric secretion? If the stomach is hypotonic, if there is pyloric spasm and stasis, there will be a failure in the hormone stimulation of pancreatic secretion, and on the other hand a failure in the regurgitation of pancreatic fluid into the stomach. Under such conditions the wall of the organ will be subjected to an unduly prolonged contact with a gastric juice which is unchecked and unregulated by pancreatic regurgitation. The gastric wall suffers damage under these conditions, and the damage may end in ulceration.

If, on the other hand, the stomach is hypertonic, if the pyloric sphincter allows, not the normal intermittent discharge of small quantities of acid peptic fluid into the duodenum, but the early flooding of the duodenum with peptic fluid before pancreatic secretion is active, the duodenal wall is then subjected to gastric juice of unchecked peptic activity. It is therefore in the duodenal wall that we find the ulcerative lesion when the functional aberration is that of hypertonicity and hurry. In an analogous way may be explained the incidence of jejunal ulcer after anastomosis. It occurs when the peptic activity of the gastric juice is high, and when for some reason the pancreatic secretion, which normally checks this activity, defaults.

It would appear, then, that we can find in derangements of the gastro-pancreatic correlation a sufficient explanation of the incidence of peptic ulcer (given a peptic secretion of high activity), and also reasons for its varying site. We have at present incomplete knowledge of the conditions which set up these primary functional disturbances; they themselves do not appear to be dependent on the chemistry of the gastric secretion, for hypersecretion and hyperchlorhydria are usual both in the hypotonic stomach of gastric ulcer and the hypertonic stomach of duodenal ulcer. We do know, however, that visceral disease elsewhere—for example, in the appendix and gall bladder—may be connected with such functional

gastric disorders, and that the latter may disappear after surgical treatment of the focal disease.

In treatment we have regard to three main points:

1. The ulceration itself.
2. The activity, or excessive activity, of the gastric secretion, and the default of the mechanism which normally controls this.
3. The disturbances of motor function which precede and account for both.

Surgical treatment deals with ulcer (1) by methods of removal which leave no embarrassing deformity behind; (2) by anastomosis, which provides a permanent check on the peptic attack of the gastric juice; (3) by removing any focus of disease elsewhere.

All the cases reviewed here had had a lengthy period of medical treatment extending from many months to many years. I am aware that under the term "medical treatment" I include different types of treatment more or less thorough. Very high claims were made by physicians for what I may call the "satisfaction" method, the basis of which is the Lenzhartz diet. I do not think that physicians are now satisfied with the permanence of results obtained by this method. That which is in most favour to-day is the "neutralization" treatment identified with the name of Dr. Sippy of Chicago. Dr. Sippy summarizes the claims he makes for this method in the *New Oxford Medicine*, vol. iii, p. 169. They are too long for quotation. I have been unable to find clinical records of a large series of cases treated by this method and followed over long periods afterwards. This is the most satisfactory evidence, and is most valuable if late results are recorded in the patients' own words.

The surgical remedy for duodenal ulcer is posterior gastro-jejunostomy. The anastomosis between stomach and jejunum must measure three inches in its long diameter, and it must encroach on the pyloric segment of the stomach. Some surgeons add to the gastro-jejunostomy an excision of the ulcer, others add occlusion of the pylorus. Failure to obtain permanent relief by gastro-jejunostomy is so exceptional that to add either the one or the other appears to be unnecessary. I have employed occlusion of the pylorus on many occasions; I do not think it has any effect on the result in duodenal ulcer, and I am doubtful whether it is physiologically sound. The activity of pancreatic secretion is likely to be checked if the gastric contents are excluded from the duodenum.

In gastric ulcer healing is most certainly brought about by removal of the ulcer. Can gastro-jejunostomy be relied on to so remedy the stasis in the stomach and the peptic activity of the gastric juice that ulceration will not recur? Certainly ulcer excision alone is not sound, and gastro-enterostomy alone cannot be relied on, and even in combination the results are by no means uniformly good. I may instance a case of ulcer of the lesser curvature for which, some two and a half years ago, I did posterior gastro-jejunostomy. This anastomosis is still anatomically satisfactory, but the ulcer did not heal, and I subsequently excised it. Relief was only temporary. The patient then naturally transferred himself to another surgeon, who removed the pyloric portion of the stomach as far as the anastomosis, and in this pyloric portion was a new ulcer. Yet gastro-enterostomy is a remedy of value even in inveterate gastric ulcer, but it is not the remedy of choice; a more radical procedure is advisable. That more radical operation is partial gastrectomy. The term includes two different procedures, either—

- (a) Sleeve resection with gastro-gastrostomy; or
- (b) Resection of more or less of the stomach, including the ulcer, closure of the duodenum, and anastomosis of the stomach and jejunum.

Sleeve resection I believe to have only a limited field; it is the operation of choice for mid-gastric cicatrized ulcer. In the presence of active ulcer it is open to the same objection as simple ulcer excision, in that it does nothing to modify the chemistry of the gastric secretion.

Partial gastrectomy combined with a gastro-jejunal anastomosis does two things: in the first place, it removes the ulcer and the ulcer-bearing area; in the second place, it renders the stomach incontinent and prevents finally the accumulation of a noxious gastric secretion.

In all the cases recorded the method employed was the posterior Polya or Reichel operation. I can imagine the anterior method to be a necessity if what remains of the stomach is too far removed from the mesocolic gap to sew the margins of this gap to the stomach wall; but this was possible in all these cases, and in none have there been signs of anastomosis

constriction such as Dr. Charles Mayo has recorded and which led him to prefer the anterior operation.

This operation is well borne. In my view the most important points in the technique are: first, such infiltration anaesthesia as to give satisfactory relaxation; second, sound closure of the duodenum; third, great care in suture of the stomach to the jejunum with small suture gaps; fourth, a watchful haemostasis throughout. In the cases recorded the post-operative recovery was free from complications, from undue shock, from haemorrhage, and from vomiting. The post-operative treatment is as follows: Sterile water is given at once and the patient is encouraged to suck formalin tablets—they allay thirst and keep the mouth clean; on the second day milk, sterilized and alkalized; on the third day an egg, beaten up and divided, is added, and on this day medicine is started for the purpose of controlling the activity of the gastric juice—it contains bismuth carbonate, magnesia ponderosa, and tinctura belladonnae. As the diet is increased powders are given in addition, consisting of calcium carbonate and magnesia.

This operation is the one to be chosen in inveterate penetrating gastric ulcer. Whether it is feasible in a given case cannot be decided until the abdomen is opened. Probably in 10 or 15 per cent. of the cases in which it is desirable it will prove to be not feasible. Some alternative will then have to be adopted. The real alternative is a transgastric operation (excision, or cauterization, or suture, or a combination of these), together with gastro-jejunostomy. If the stomach is so fixed that a posterior gastro-enterostomy is difficult, there is the valuable alternative of pyloroplasty—Finney's operation. This operation, which I should perhaps call gastro-duodenostomy, aims at relieving stasis and modifying the chemistry of the gastric secretion, as does gastro-jejunostomy.

The best contribution to the study of the treatment of gastric ulcer, whether that treatment be surgical or medical, is the collection of careful records. We want to appreciate the immediate risks of operative procedures, the early results, the late results. We want to know much more as to the persistence of the favourable results that are claimed for methods of medical treatment. Impressions and opinions are of little value to the argument; the question requires the same kind of handling as a laboratory problem, and similar methods of recording and analysing results.

THE TREATMENT OF PERFORATED GASTRIC AND DUODENAL ULCER.

BY

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THE treatment of perforated gastric and duodenal ulcer is entirely surgical, and the prognosis largely depends on the time when operation is performed. Recovery is contingent on early diagnosis and prompt surgical treatment, for delay almost inevitably leads to a fatal termination. During a period of two years I operated upon a consecutive series of 46 cases of acute perforated gastric and duodenal ulcer at the Manchester Royal Infirmary. Four patients died following the operation, giving an operation mortality of under 9 per cent.

There is still considerable difference of opinion as to whether an ulcer which has perforated will, after operation, give rise to further trouble, and whether gastro-enterostomy is necessary. I have endeavoured to throw some light on this question by following up the after-histories of the cases in this series. The following are the details of the cases:

Sex and Age.

Forty-four of the patients were males and two females. It is remarkable to note the preponderance of the male sex. The youngest patient was 19 years of age, the oldest being a man of 71. The greatest number of perforations occurred between 30 and 60 years of age.

History of Previous Indigestion.

This could be elicited in most cases. In four patients perforation was the first sign, no previous gastric disturbance being present. In a considerable number of the duodenal cases the symptoms of previous indigestion were very slight.

Situation of the Ulcer.

Forty of the ulcers were duodenal or pyloric in situation. In only five cases were the ulcers found to be gastric. There was, in addition, one case of perforated gastro-jejunal ulcer.

Hours Perforated and Operative Results.

The time that had elapsed before operation is shown in the following table, together with the mortality rate:

Hours Perforated.	Duodenal Perforations.			Gastric Perforations.			Total Mortality.
	No. of Cases.	Died.	Mortality.	No. of Cases.	Died.	Mortality.	
1-12 hours	24	0	Nil	1	1	100 %	4 %
12-18 ..	6	0	Nil	—	—	—	Nil
18-24 ..	4	0	Nil	3	1	33 %	14 %
Over 24 ..	6	2	33 %	1	0	Nil	28.6 %
Total ...	40	2	5 %	5	2	40 %	9 %

This does not include one case of perforated gastro-jejunal ulcer which recovered.

Mortality.

Four patients died following operation, giving an operative mortality of 8.7 per cent. for the series of 46 cases. Thirty-four cases of perforated duodenal ulcer were operated upon within twenty-four hours and all recovered. Six were operated upon after this period and four recovered; the patients who succumbed were both in poor condition when submitted to operation, and died in thirteen and eighteen hours respectively following operation, from shock. Of the five cases of gastric ulcer, two died subsequently from pulmonary complications. One died on the fifth day following operation from acute oedema of the lungs, the second succumbed on the tenth day after operation from bronchopneumonia. Autopsies were held on both these cases. The ulcers were found to be large chronic spreading ulcers near the pyloric end of the stomach. Microscopical examination showed that both the ulcers were malignant, one being a glandular carcinoma, whilst the other was a spheroidal-celled carcinoma.

Death occurred in one of the patients subsequently from acute appendicitis. He had made an uneventful recovery following a perforated duodenal ulcer and had been discharged from hospital. He was readmitted shortly afterwards with a perforated appendix, and died following operation for this condition. All the remaining cases are, to the best of my knowledge, alive at the present time.

OPERATIVE TREATMENT.

Immediately the diagnosis of perforated ulcer has been made laparotomy should be performed without delay. Time should not be spent in attempts to improve the patient's condition; shock can best be relieved by early operation, and a successful result depends essentially on prompt treatment.

The technique of the operation is now so well known that it requires no special description. The perforation is closed with fine thread sutures passed at some distance from the edges of the ulcer. In cases of duodenal perforation the sutures are passed from side to side and thereby avoid narrowing the lumen of the gut. When tightened these infold the ulcer and a piece of omentum is then turned up and stitched over the suture line. I have always found it possible to close the ulcer successfully by suture. Any method other than suture must be attended with a high mortality owing to the danger of leakage.

The peritoneal cavity is carefully swabbed out with gauze swabs wrung out of very hot saline. Some surgeons advocate irrigation in preference to swabbing, but irrigation adds the diffusion of the extravasated material and increases the risk of subphrenic abscess.

The operation lasts twenty minutes, rarely longer, and subcutaneous saline may be administered whilst it is in progress with advantage. Drainage is seldom employed. In perforation of under eighteen hours' duration it is never necessary. After twenty-four hours when there is gross soiling it may be advisable. This is best done through a suprapubic stab incision and the tube removed as soon as possible.

After-Treatment.

The Fowler position is adopted as soon as the patient is returned to bed; rectal salines are given at four-hourly intervals for two days. For the first forty-eight hours the patient is only permitted small sips of water by the mouth; fluids in increasing quantities are given later. An enema is given daily, no purgative being allowed till the seventh day. The stomach is thus kept at rest as far as possible, and opportunity given for the healing of the ulcer to start. After discharge from hospital the patient is instructed to take a careful diet for six months. Sodium bicarbonate should be taken regularly for a time, as this appears to diminish any tendency to hyperchlorhydria, and defective teeth are attended to. This post-operative treatment, so often neglected, undoubtedly assists in obtaining satisfactory results after operation.