drug is claimed only to be an amoebicide, this result is what might be expected. The other three preparations exhibit a common action, varying little in degree, which is of considerable interest. They destroy all *B. coli* in a dilution of 1 in 200, and a large proportion of *B. coli* at 1 in 2,000. On the other hand, they destroy only a proportion of streptococci at 1 in 200, and at 1 in 2,000 streptococci actually multiply. This is a selective action paralleled by the well known property of phenol of inhibiting the growth of *B. coli* in dilution of approximately 1 in 200, at which streptococci will grow vigorously, a property which has been found of considerable use in routine bacteriology. This suggests that all these three preparations are closely allied in action, and presumably in composition, to phenol.

To those who regard the predominance of streptococci in the faeces as accountable for chronic ailments such as arthritis, this action on the part of these drugs will hardly commend itself. But before applying such findings to the presumed effect of these drugs on the human subject, it must be asked in what dilution they were present in the intestinal tract when taken in ordinary doses. This cannot be determined unless it is known whether they are absorbed, combined, or destroyed, or unless a method be devised for estimating them in the faeces themselves. The latter, having in view that their composition is unstated, is hardly feasible. But some indication as to their activity, if any, in the colon may perhaps be obtained by comparing the results of the two above series of experiments *in vivo* and *in vitro* respectively. The latter clearly show that in certain dilutions three of these antiseptics exert a selective action on coliform bacilli.

If, in experiments on the human subject, it were shown that although the total numbers of organisms in the stools were unaffected the proportion of streptococci to coliform bacilli was increased, it might be presumed that this change had been brought about by the action of the antiseptic, and therefore that its dilution in the bowel is something in the neighbourhood of 1 in 2,000. That this is probably not so is shown by the following figures, which refer to the same series of experiments as in Table I.

TABLE III.—Average Percentage of Streptococci in Faeces before and during Administration of "Intestinal Antiseptics."

Antiseptic.	Average Percentage of Streptococci.	
	Before Administration.	After Administration.
Dimol	34	54
Kerol	4	3.9
Izal (1925)	31	36
Izal (1925)	46	37

In only one case, that of dimol, is there an appreciable increase in the number of streptococci in the stools during treatment, and even this is not sufficient, as an unconfirmed observation, to indicate with certainty that the drug is exerting its selective action *in vivo*, although the fact that it possesses this power *in vitro* to a greater extent than either kerol or izal suggests that this may be the case. Kerol and izal do not appear to affect the relative proportions of the two types of organism at all.

If this comparison is justifiable, therefore, it would appear that these drugs do not reach the colon in an active form and sufficient concentration to exert the effect which they are shown to produce on faecal organisms *in vitro*.

Conclusions.

The only conclusion which can with certainty be drawn from these experiments is that the drugs investigated, when given by the mouth, do not appreciably reduce the total number of living aerobic organisms in the faeces. Whether it can therefore be said that they are therapeutically useless is a matter upon which it would be rash to pronounce an opinion. But it may reasonably be submitted, in view of these findings, that specific and detailed evidence as to the alleged action of these drugs should be published in support of the claims which are constantly made for them. It is possible that data exist upon which these claims are founded, obtained by other methods, which traverse the deductions suggested by the experiments here described. If not—if, in fact, these claims are made without due investigation of the results obtainable in the living

subject by the administration of these drugs—then it may reasonably be asked whether any indication exists for their employment.

Summary.

The experimental results obtained may be summarized as follows:

1. Four preparations, of which three are described as intestinal antiseptics, were found to exert no appreciable effect on the total numbers of living aerobic organisms in the faeces when administered by the mouth in adequate doses.

2. Of the same preparations the three referred to above were found to exert on faeces *in vitro* an antiseptic action much greater for coliform bacilli than for streptococci, the concentration of the drugs being considerably higher than that in the intestinal tract which presumably follows their administration by the mouth. The fourth preparation had no evident action whatever in any dilution employed.

This work formed part of a research undertaken for the Gillson Scholarship of the Society of Apothecaries. I am indebted to them, and to Dr. Geoffrey Evans, whose patients were some of the subjects in the investigations.

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GASTRIC PAIN.*

BY

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NORMAL digestion should be without sensation except for such feelings as those of satisfied hunger and a renewal of energy. Unpleasant sensations such as discomfort and pain after the ingestion and during the digestion of food are symptoms of the abnormal. The abnormality may be in the patient's consciousness in that he is in such a state of nervous sensitiveness that the ordinary actions of digestion are felt by him and are unpleasant. But even in these cases the general condition of debility affects the digestive apparatus and produces faulty working. Gastric pain is associated in our minds with the patient who complains of indigestion because there is something wrong with the stomach.

There is no more difficult task in medicine than to unravel the tangle of symptoms which drive a dyspeptic to consult us. Of these symptoms discomfort and pain are prominent. The investigation of the symptoms—that is, the taking of the history—is of the greatest value as an aid to diagnosis. It is to surgery, and not to physiology and medicine, that the credit is due for elucidating the disorders of digestion. Physicians at one time were content to treat indigestion with alkalis and, if that failed, with acids; since surgical measures have come to the fore our knowledge of gastric disorders has increased and the methods of investigation have become more numerous and more trustworthy.

The tripod on which a gastric diagnosis rests is: (1) the clinical examination, of which the history is by far the more important part; (2) the test meal; (3) the radiological appearances of an opaque meal. Of these the radiological findings can be the most definite. An ulcer can in some cases be demonstrated and we then have direct evidence. But most of the radiological evidence is indirect, and is concerned with the observations of position, filling, emptying, and peristalsis. The test meal is valuable, but is more variable as the gastric secretions may vary from day to day and are subject to influences from outside.

The clinician, therefore, still has work to do in examining the patient. The physical examination in gastric disorders seldom reveals any gross sign. There may be a tumour, or visible peristalsis, or a dilated stomach with a splash; but usually rigidity of the abdominal muscles is the only objective sign. At the same time it must be remembered that a full examination must be made, which includes

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the lungs, the nervous system, the urine, and, last but not least, the rectum.

It is well to let the patient say all that he will about his pain after giving him a start. When he has finished his spontaneous account, the examiner can arrange it and amplify it from the answers to leading questions.

Pain must be the subject of many questions. First, its situation is important. Among the organic or surgical dyspepsias left-sided pain suggests gall-bladder trouble or gastric ulcer, and right-sided pain duodenal ulcer. Epigastric pain is a symptom of any gastric disorder.

The next point is severity. Different patients estimate pain in different ways. The robust man who has never been ailing regards any abdominal pain as severe, while a woman, who is used to abdominal pain, will look upon it as a nuisance that will pass away if endured patiently. So, in estimating severity, we must form our opinion of the patient while we listen to the description. A few special questions may help us. Did it make you feel sick? Did it make you sick? Did it make you sweat? Did you faint or feel faint? What are you willing to go through to avoid a recurrence?

Next comes the relation of pain to food. It is well to draw up a time-table of meal times, filling in the usual time of the onset of pain, and to ask also if the pain occurs at night and wakes the patient with any regularity. It is necessary to distinguish the difficulty in getting to sleep, which may be due to flatulence or worry, from the waking up at 2 a.m. on account of pain. It can safely be claimed that freedom from pain between what the patient calls "attacks" is the most important symptom in a gastric or duodenal ulcer, and to a smaller extent of other organic dyspepsias of non-malignant origin.

The patient complains of attacks of indigestion and pain which recur. During the intervals he is fit and has a good appetite. The attacks tend to become more frequent and severe and the intervals shorter and less comfortable. At this stage he stops buying soda-mints and other remedies, and consults his doctor. This condition of periodicity in dyspeptic attacks is the most definite single point in favour of a non-malignant organic lesion. Any patient who gives such an account of his symptoms should go through a complete investigation by test meal and opaque meal. Regularity of pain is a striking feature in some cases. It is greatest in cases of duodenal ulcer, and less in gastric ulcer. It is more changing in its times in dyspepsia due to gall stones, and in its severity in that due to chronic appendicitis. How relief is obtained is important. If vomiting occurs, does it relieve? Has medicine given relief? Does lying down make any difference? Do all measures have apparently no effect?

Before trying to explain the cause and significance of gastric pain, it will be advisable to mention other painful conditions occurring in the dyspeptic.

There is in some cases hyperaesthesia of the skin of which the patient may be conscious. He may say that the touch of the bedclothes gives him a peculiar sensation. But more often this hyperaesthesia is found only on testing the sensibility of the skin. It is a peculiarity of sensation due to the increased sensibility of the spinal segment which innervates both the disordered viscus and the area of skin.

Tenderness is a more definite phenomenon. It is entirely objective and is produced by pressure. The best way to discover tenderness is to palpate the abdomen with varying degrees of pressure and to ask the patient to say at once if the examination of any spot is painful. Tenderness is not of simple origin. It may be due to the irritation of nerve endings in the subperitoneal connective tissue by the action of some irritant process such as inflammation, or by a mechanical factor such as spasm of stomach or intestine, or pressure may be painful when the abdominal muscles or segments of them are in a state of tonic contraction. This rigidity can be regarded as protective, the contracted muscle fibres shielding the troubled organs below. It is obvious that these two causes of tenderness may be present at the same time. But rigidity, and tenderness due to muscular contraction, are the commonest objective signs in the various forms of dyspepsia, the upper segments of the recti muscles being most frequently affected, especially the right. Pain and tenderness may

be due to the irritation of nerve endings when an organic lesion such as ulcer invades neighbouring tissues. But having mentioned these forms of sensation that can be called painful we are still left with the pain of which the patient originally complains.

This pain is the true visceral pain, and may be present in organic and functional disorders of digestion. It is not due to a sore mucous membrane. The sensations of pain, heat, and cold are not referred to consciousness as such from the nerve endings in the mucous membrane of the gastro-intestinal tract, except in a very minor degree. These forms of sensation are only found fully developed in surfaces covered with a stratified epithelium. Columnar and cubical epithelium are without, or almost without, these forms of sensation. This statement can be illustrated by stimulating the mucous membrane in a case of colostomy. Touch, pin-prick, heat, and cold do not impose themselves on the consciousness of the patient. But if it be dragged on gently with forceps the patient will at once tell you that he feels something, and if you drag hard enough he will complain of pain.

Further, in many cases of organic dyspepsia, such as duodenal and gastric ulcer, a notable feature is its periodicity. The ulcer at certain times is not associated with pain. It is reasonable, then, to regard the mucous membrane as not being the sensitive tissue, but to look elsewhere.

It is beyond dispute that when pain is felt nerve units are abnormal or are abnormally stimulated. In gastric visceral pain we have to seek for something which is capable of producing pain and is behaving abnormally. Of the three coats of the stomach the mucous coat is ruled out, and the peritoneal coat is more concerned with tenderness, an ordinary somatic painful sensation which is elicited by palpation. There is left the muscular coat. We have seen that traction on the intestine in colostomy will evoke a painful sensation if sufficiently strong. The bulk of the evidence obtained by radiological examination is the record of the position and the observed movements in stomach and duodenum. It has been shown radiologically that hunger pains coincide with movements of the stomach. We know that stretching and over-contraction of skeletal muscle are painful, and the same is true for smooth muscle.

If we attempt to correlate the pain of dyspepsia with the actions of the muscular coat we get a very reasonable explanation. Take, for example, a case of chronic ulcer of the lesser curvature of the stomach; the usual time for pain is twenty to thirty minutes after food; its arrival in the stomach produces functional activity with a secretion of acid juice. The muscle fibres or the nerve endings of the muscle fibres are stimulated by the contents of the stomach, and a painful contraction takes place. The contraction may be painful either because it is a cramp or because there is a pull on subperitoneal nerves by contracting muscle in the base or edge of the ulcer. When an ulcer is present in the pre-pyloric region the pain will occur later after food, usually one to one and a half hours.

In duodenal ulcer the pain is so late after food that it is dated as being before food; it is relieved by the intake of food, and can rightly be called hunger pain. It is usual for there to be an excessive secretion of acid juice in duodenal ulcer. Hunger pain is a duodenal symptom, not necessarily of ulcer. An explanation of the pain would be that the acid juice in the duodenum causes the pyloric sphincter to contract painfully. It is not an entirely satisfactory explanation, but the hypertonic contraction of the sphincter can be regarded as one cause of pain in duodenal ulcer. This type of pain is a symptom of duodenal trouble, usually associated with hyperchlorhydria. Its repetition after free intervals is the point in favour of duodenal ulcer, especially if haemorrhage has been noticed.

It is obvious that we must seek further to discover why the muscular coat should behave so as to produce pain. Possibly the presence of acid in the stomach is the exciting cause of the abnormal and excessive muscular contraction in gastric and duodenal ulcer. When food is taken into the stomach the resting juice is diluted so that the strength of the acid is lowered. Next the presence of food stimulates a flow of acid juice, and both the amount and the strength of the acid are increased. At the same time the digestive

movements of the stomach have started on their course. This would explain the interval between eating and the onset of pain in gastric ulcers.

In duodenal ulcer the percentage of acid in the gastric juice is nearly always high. The pyloric sphincter relaxes in the presence of acid on its proximal side, and contracts if acid remains unneutralized on its distal side. If the strength of the acid is excessive the contraction of the sphincter is overdone and prolonged. The results of gastro-jejunostomy are said to be the most satisfactory in cases of duodenal ulcer. Perhaps this is because the operation allows a more free regurgitation of alkaline juice into the stomach, and provides an exit that is not guarded by a sphincter.

So that in gastric and duodenal ulcers the explanation of pain and its time-table is the behaviour of the smooth muscle under the influence of acid. In one case the acid stimulates the base of the ulcer, and in the other a highly acid juice produces a cramp of the sphincter.

These symptoms are such as are described by the patient as being characteristic of his trouble in its early days. By the time he presents himself for examination the symptoms are usually more confused, and, during the worst periods, the pain is constant and is bad at all times, soon and late after food. It is necessary, then, to ask the patient to go back and describe the symptoms of the earlier days.

If the ulcer causes a deforming scar, thereby producing either hour-glass stomach or pyloric obstruction, pain will be produced by the stretching of the stomach wall and will only be relieved by vomiting, lavage, or emptying the stomach naturally. A change, therefore, in the character of the pain and other symptoms must be looked for and taken into account. When the history reveals a change of this kind in a case of long duration, the evidence is very strong indeed and a definite diagnosis can be made with great accuracy.

Alteration in the character of the pain will also occur when the base of an ulcer becomes adherent to neighbouring structures, such as pancreas, gall bladder, liver, and crus of the diaphragm. When an ulcer of the lesser curvature becomes adherent to the pancreas, constant pain through to the back will be felt. If the crus of the diaphragm is invaded the pain will be in the shoulder. The same pain will be felt in inflammation of the gall bladder as soon as the diaphragm is affected, though the more constant pain in gall-bladder trouble is through to the scapula.

But these pains are additional and are either referred or due to the implication of subperitoneal nerve endings. The vast majority of dyspeptics, however, have nothing so simple as an ulcer. A very large number complain of pain as soon as food enters the stomach. This may be a temporary affliction or more lasting. It is found in patients who have a tired-out or ill used stomach. At its worst the pain does not go until the patient has vomited. In character the pain is more often a dull aching, and not the violent cramp-like pain such as occurs with duodenal ulcer. It can be regarded as a stretching of atonic muscle fibres. In such patients there is atony of the circular and longitudinal fibres, due either to the general weakly condition of the patient or to some recent misuse of the stomach. The presence of food stretches the muscles and produces discomfort or pain. In the worst cases the strong oblique muscle fibres are also affected and then the symptoms are more lasting. In such cases there is a general dropping of the stomach.

In many of these cases there is a low acidity of the gastric juice. There may also be slow emptying of the stomach. These people may truly be said to have weak digestions. But in some, in which enteroptosis can be demonstrated, there is an increased acidity, and there may be rapid emptying. Here the symptoms may suggest duodenal ulcer, but the absence of the intervals of good digestive health, and the immediate improvement on putting the patient to bed, and other minor points will help to differentiate. It is not necessary for the enteroptotic patient to have indigestion. Many eupeptics have a low-placed stomach. But if some cause of indigestion occurs the presence of ptosis is against the patient's chances of satisfactory relief.

The majority of dyspeptics who seek relief belong to the class of atonics. Very often they are feeble in other ways, and their poor nutrition enhances their general as well as their gastric debility. The surgeons call the condition "medical dyspepsia." They are quite right to avoid operations on such subjects, for they are likely to do them more harm than good. But it is rather sad that the physicians should be left to deal with the most thankless and intractable kind of case. It is still more sad when the physician is called in to make the best of a patient who has had a series of operations, including gastro-jejunostomy, appendicectomy, freeing adhesions, etc. However, a good deal can be done for these atonic patients by putting them in bed with the foot of the bed raised, by giving small feeds frequently, by fitting an abdominal belt, and by massage.

Cancer of the stomach may be productive of pain of the visceral type, but pain must not be regarded as a necessary symptom of cancer. If the cancer cells infiltrate the wall of the stomach, making it stiff and rigid as in the leather-bottle stomach, the capacity of the stomach is incapable of adapting itself to the presence of food. The entry of food may put the wall of the stomach on the stretch and produce instant pain. Or a carcinomatous ulcer may be associated with pain in the same fashion as a non-malignant ulcer by infiltrating neighbouring tissues. But many cases of cancer are painless. Possibly the absence of acidity is responsible for the absence of pain. But the majority of patients who have pain immediately after food have a tired-out or ill used stomach which is in a condition of atony.

Substernal pain, or "pain in the chest"—a symptom of indigestion commonly found in constipated young women and in sufferers from flatulence—is concerned with the cardiac sphincter. Substernal pain may be due to dilatation of the oesophagus because the cardiac sphincter will not relax to allow food to pass into the stomach. In the flatulent, pain is produced by the pressure on the cardiac sphincter until it opens. When the cardiac sphincter opens and the gastric tension is lowered, relief of the pain follows at once. Here again the wrong behaviour of muscle can be blamed for the pain.

In all the cases described it would seem that the visceral pain of indigestion is to be attributed to the condition and the behaviour of the muscular wall of the stomach. If, then, we wish to explain to ourselves the meaning of digestive pain we must express our explanation in terms of muscle. In the disorders of digestion there is infinite variety. A few examples only have been taken to show the significance of the predominant symptom—pain.

In the opinion of the patient pain is of the greatest importance, and if we can understand how the pain is produced we are in a better position to treat it and to prevent a recurrence. We can arrive best at this understanding if we take into consideration the state and behaviour of the smooth muscle of the stomach.

NEPHRECTOMY IN INFANCY.

BY

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ALTHOUGH the removal of a kidney during the first twelve months of life is seldom indicated, nephrectomy may be a life-saving measure. Renal sarcoma in the very young is well known and may produce a tumour of considerable size which is often bilateral. Urinary symptoms are generally absent and attention is first drawn to the lesion by the presence of an abdominal swelling. The general health remains good until pressure of the growth causes obstruction either to the ureter or the adjacent bowel.

Opinions differ as to the origin of these tumours, some holding the view that they are extrinsic to the kidney, while others claim that they are entirely renal. If the latter view is correct, it is strange that haematuria, the common manifestation of all other neoplasms of the kidney, should never be observed, more especially as carcinomatous