An Address

ON

THE SPHINCTERS OF THE ALIMENTARY CANAL AND THEIR CLINICAL SIGNIFICANCE.

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BY

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THE sphincters of the alimentary canal play a very important part in digestion. Disturbances in their functional activity are not at all uncommon and give rise to a variety of symptoms of great interest.

In order to understand the nature and significance of these pathological disturbances in function an accurate conception of the anatomy and physiology of the sphincters is essential. Anyone wishing to study the structure of the sphincters of the alimentary canal will, however, be disappointed to find how inadequately they are described in even the latest editions of the standard textbooks of human anatomy. Thanks to the work of Cunningham and of Symington respectively the pyloric and anal sphincters receive sufficient attention. But we get no clear idea of the anatomy of the cardiac and ileo-caecal sphincters, and indeed many authors entirely ignore the existence of one or both of them, whilst the pelvi-rectal sphincter is never mentioned.

The fundamental conception concerning the physiology of the sphincters depends upon the classical experiments of Bayliss and Starling¹ carried out in the laboratories of Guy's Hospital twenty-five years ago. They showed that peristalsis consists of a co-ordinated reflex, in which a wave of contraction, preceded by a wave of relaxation, passes down a hollow viscus. When this is extended to the action of sphincters it means that a sphincter should relax at the approach of each peristaltic wave. The conditions which modify this co-ordinated action will form the chief subject of my address to-night.

THE CARDIAC SPHINCTER.

(a) Anatomy and Physiology.

The point where the oesophagus joins the stomach is called the cardia, but much doubt has been expressed as to whether a cardiac sphincter exists at all. Poulton and Payne² have, however, clearly demonstrated that the lower end of the oesophagus in rabbits exerts a definite sphincter action. They also state that on cutting longitudinally through the cardia into the oesophagus after death in man the lower two or three inches can often be seen to have a thicker muscular coat than the part immediately above. From a radiological study of the act of swallowing in normal people and in patients suffering from achalasia of the cardia, we have come definitely to the conclusion that the last inch or more of the oesophagus, comprising the whole of the abdominal portion, and sometimes a very small part of the thoracic portion, has such a totally different function from that of the rest that it deserves to be regarded as a sphincter. This has recently been confirmed by observations made during a laparotomy performed for the relief of achalasia of the cardia and by a study of the oesophagus in stillborn infants. The relatively long cardiac sphincter in the latter is clearly differentiated from the rest of the oesophagus by the greater thickness of its muscular coat and by the pallor and prominent longitudinal rugae of its mucous membrane (Fig. 1). When swallowing is not taking place, the postural tone of the circular muscle fibres of the cardiac sphincter is such that it has no

FIG. 1.—Stomach of stillborn full-time infant, showing cardiac sphincter, C.S., with thickened circular muscle coat, and deep, pale, longitudinal rugae. D, Diaphragm, attached to centre of sphincter. P.S., Pyloric sphincter.

potential lumen, in contrast with the cervical and thoracic oesophagus, which have a potential lumen at least an inch in diameter, although when empty their walls collapse and so completely obliterate it. Consequently the sphincter offers a definite resistance to the passage of food, whereas the rest of the oesophagus offers no resistance at all.

The vagal and sympathetic nerve fibres which supply the oesophagus end in connexion with ganglion cells situated between the circular and longitudinal muscular coats, where they constitute a plexus, similar to the plexus myentericus of Auerbach in the intestines. Keith³ has shown that this consists mainly of nodal tissue, similar to that of the auriculo-ventricular bundle in the heart, and only to a small extent of nerve cells and fibres. The nodal tissue reaches its greatest development in the part of the oesophagus immediately adjoining the stomach; this furnishes additional anatomical evidence for the existence of a specialized cardiac sphincter, though in adults there is often no obvious thickening of the circular muscle coat in this region, corresponding to that of the pyloric, ileo-caecal, and anal sphincters. The distinction is probably due to the fact that the function of the cardiac sphincter differs from that of the others in being concerned in the prevention of regurgitation of gas and fluid from the stomach into the mouth, and only to a very minor extent in the regulation of the onward passage of the contents of the oesophagus. It is consequently never called upon to resist a degree of pressure anything approaching that which the other sphincters are able to withstand.

When swallowing takes place the intrapharyngeal pressure rises; this propels the food into the oesophagus, along which it is actively conveyed by a peristaltic wave; our observations with the x rays fifteen years ago showed that this occurs almost as rapidly when a man stands on his head as when he is in the normal erect position. When the food reaches the extreme lower end of the oesophagus

FIG. 2.—Diagrams of the passage of a bolus of barium-containing food (B) through the oesophagus (OO), as seen with the x rays, in successive seconds after swallowing. S, Spine; H, heart; D, diaphragm; C.C., cardiac sphincter; F, fundus.

its progress can be seen with the x rays to be arrested owing to the resistance offered by the closed sphincter (Fig. 2). Immediately afterwards the sphincter relaxes and the food slowly trickles through it into the stomach, but even now the lumen of the sphincter is very much less than that of the rest of the oesophagus. The relaxation of the sphincter as each peristaltic wave travelling down the oesophagus reaches it is typical of the activity of every other sphincter.

Under normal conditions, when the whole of a meal has been eaten, the cardia remains closed and so prevents the regurgitation of food, gastric juice, and gas, which would otherwise occur as a result of the positive pressure in the stomach and the negative pressure in the thorax. The intragastric pressure always remains about the same, as the tone of the muscular coat of the stomach adapts itself accurately to the volume of its contents. When the pressure is suddenly raised as a result of rapid aerophagy or of the evolution of carbon dioxide after swallowing sodium bicarbonate, the resistance offered by the cardiac sphincter becomes insufficient and gas is expelled by the mouth.

(b) *Achalasia of the Cardia (so-called Cardiospasm).*

Nearly every pathological museum contains one or more specimens of an enormously dilated oesophagus with a greatly hypertrophied muscular coat. The condition is a remarkable one, for no obstruction at the cardia has ever been discovered after death to account for the dilatation and hypertrophy. In the absence of organic obstruction some form of functional obstruction must be present, as otherwise it would be impossible to explain why, in spite of the violent peristalsis which eventually leads to hypertrophy of its muscular coat, the oesophagus should be so greatly dilated and always filled with a large quantity of food and watery mucus.

Since Mikulicz⁴ in 1882 first suggested that the functional obstruction is due to spasm of the cardia the condition has been commonly known as cardiospasm. It is obvious that if a spasm were present it would offer considerable resistance to the passage of a bougie, and that if a bougie had once passed through the contracted cardia it would be so firmly gripped that its withdrawal would require considerable force, just as spasm of the anal sphincter offers considerable resistance to the finger and grips it tightly when once it has passed into the rectum. But using a rubber tube filled with mercury instead of a rigid bougie, I found that it dropped so easily into the stomach that it was impossible as a rule to detect any resistance when the cardia was reached. With its end in the stomach it could be moved freely up and down and finally be withdrawn without the slightest drag upon it, such as the grip caused by spasm would produce. These observations afford good evidence that no spasm is present. The anatomical evidence is equally conclusive. It is obvious that spasm, continuing for as many years as patients with this condition may live, would give rise to hypertrophy, corresponding with the hypertrophied sphincter ani which results from the spasm caused by a chronic anal ulcer. But a careful examination of many specimens has shown that the last inch of the oesophagus, the part within the abdomen corresponding with what I have already referred to as the cardiac sphincter, is not dilated and there is no hypertrophy of its muscular coat. I recently saw the most striking confirmation of this when my colleague, Mr. R. P. Rowlands, exposed the abdominal part of the oesophagus in a case of enormous dilatation of the oesophagus, which was presumably accompanied by great hypertrophy of its wall, as the condition had been present for seven years and we had often watched extraordinarily vigorous peristaltic waves passing down to the cardia. The part exposed was of normal diameter, and its wall, far from being hypertrophied, was remarkably thin.

Though as long ago as 1895 Rolleston⁵ had suggested that the dilatation of the oesophagus might be due to a "failure in the co-ordinating mechanism by which the cardiac sphincter is relaxed in swallowing," the cardiospasm theory held undisputed sway until, in 1910, a consideration of the facts I have just described led me independently to the same conclusion.⁶ I believe that the functional obstruction is due to the fact that the peristaltic waves which pass down the oesophagus in swallowing are not followed, as they should be, by relaxation of the cardiac sphincter. It is not spasm, but absence of relaxation, or achalasia (*a*, not; *χαλᾶω*, I relax), of the cardiac sphincter which causes the obstruction.

The resistance offered by the unrelaxed cardiac sphincter can be overcome by a pressure of about eight inches of water. Consequently when a meal is taken no food passes into the stomach until the contents of the oesophagus form a column eight inches high (Fig. 3). Then, whenever anything is eaten or drunk, the cardia opens and allows food to pass till the height is again eight inches, when it closes once more. The patient, after losing a great deal of weight, finally reaches equilibrium, as sufficient food passes into the stomach at each meal to keep him alive on a low level of metabolism. He learns to empty his oesophagus by a voluntary effort, which is quite distinct from vomiting, on going to bed, as liquid would otherwise regurgitate into his mouth, and sometimes also two or three times in the day if the accumulation gives rise to serious discomfort.

Very little is known as to how achalasia of the cardia develops. I believe some cases, especially those beginning after middle life, are strictly analogous to heart-block, being the result of degenerative disease, or, in rare cases, of syphilis, of the nodal tissue, which Keith has shown is so well developed at the cardiac sphincter. One patient of mine had tabes, and the achalasia of the cardia was probably analogous to the much more common tabetic disorders of micturition. Rolleston⁵ and Looser⁷ have each

FIG. 3.—Diagrammatic drawings of oesophagus in achalasia of the cardia. A shows column of barium-containing food, 8 in. high, above closed cardia; B, after additional quantity of food swallowed, so that the taller column, being heavier, opens cardia and allows surplus to enter stomach; C, return to condition of A after surplus food over the 8-inch column has entered stomach.

described a case following diphtheria; in these there may have been a vagal neuritis, the achalasia being analogous to what sometimes occurs in animals after division of both vagi. In one case degenerative changes were found by Kraus⁸ in the vagi after death, and in another Pollitzer⁹ found the vagi buried in a mass of enlarged glands. Most of the remaining cases are probably the result of some gastric or other abdominal disorder, which leads to reflex inhibition of relaxation of the cardia, corresponding with what often occurs at the pyloric and ileo-caecal sphincters. One of my patients had mitral stenosis and a very dilated left auricle, which may have been the source of the reflex achalasia.

I have found that the regular use of a large mercury tube (24 gauge) almost always gives great relief, and is often followed by permanent recovery. The patient learns to pass it on himself, at first keeping it in position for ten minutes before each meal, then gradually for shorter periods and less frequently, until finally he passes it only once a day, once a week, or only at long intervals whenever he feels that there is some slight return of obstruction. In the only case I have seen in which the mercury tube gave insufficient relief Mr. R. P. Rowlands divided the thin muscular coat of the entire cardiac sphincter longitudinally, and the result has been quite satisfactory, though the patient still has slight dysphagia. Treatment by forcible dilatation with Plummer's hydrostatic apparatus is painful, and has been followed on a few occasions by fatal peritonitis. We tried it twice without success before Mr. Rowlands operated on the patient already referred to.

(c) *Waterbrash and the Morning Sickness of Alcoholics.*

In the condition I have just described the cardiac sphincter never relaxes. I believe that waterbrash is also due to achalasia, but in this case relaxation is only inhibited at certain times. Waterbrash consists in the sudden evacuation of large quantities of watery alkaline fluid. The fluid is saliva which has been secreted in excessive quantities as a reflex result of the presence of excess of hydrochloric acid in the stomach—as, for instance, two or three hours after a meal in a patient with hyperchlorhydria associated with duodenal ulcer. The saliva is swallowed, but accumulates in the oesophagus, as relaxation of the cardiac sphincter is inhibited by increasing the acidity of the gastric contents, as Cannon¹⁰ found experimentally in 1908. The evacuation of the fluid is not associated with nausea and is recognized by the patient as being quite different from vomiting; it is produced, in fact, by the same voluntary or semi-voluntary process as the evacuation of the contents of the dilated oesophagus in achalasia of the cardia. This explains the otherwise inexplicable fact that waterbrash is sometimes immediately followed by the vomiting of very acid and partly digested food. the alkaline

saliva having been separated from the acid food by the closed cardiac sphincter.

The morning sickness of alcoholics is also due to emptying of the oesophagus and not true vomiting. Mucus, secreted by the inflamed pharyngeal and oesophageal mucous membrane, accumulates in the oesophagus during the night; it is not actually swallowed, but trickles down, so that the cardiac sphincter does not relax. The colourless mucus first brought up is often followed by bile-stained fluid from the stomach, as the chronic gastric catarrh may cause actual vomiting.

THE PYLORIC SPHINCTER.

(a) *Anatomy and Physiology.*

The pyloric vestibule is separated from the duodenal bulb by the pyloric canal, a channel about half an inch in length, which is normally closed by the tonic action of the well developed sphincter surrounding it (Fig. 4). Under

FIG. 4.—Three sections through the same specimen to show structure of pylorus. (After Symington.) A, Section through pyloric vestibule of contracted empty stomach. B, Section through pyloric canal. C, Section through duodenal bulb, showing pyloric orifice as seen from duodenum.

general anaesthesia and after death the sphincter relaxes, and the canal can no longer be clearly distinguished from the vestibule, except in infants; consequently our knowledge of the structure and functions of the pyloric sphincter under normal conditions has been derived to a great extent from radiological observations. Until recently Cannon's theory of the "acid control of the pylorus" was universally accepted. Acid in the stomach was supposed to cause relaxation of the sphincter, whereas acid in the duodenum caused its contraction; thus the duodenal contents were prevented from becoming too acid, as no further chyme could pass the pylorus until that already present in the duodenum had been neutralized by the alkaline pancreatic and intestinal juices and bile. That the matter was not so simple seemed, however, to be clear from the fact that the stomach emptied itself at the normal rate or unusually fast in cases of achylia gastrica, in which there is no acid in the stomach to stimulate the opening of the pylorus. The recent investigations of Baird, Campbell, and Hern¹¹ at Guy's Hospital on healthy men with one tube in the stomach and another in the duodenum show that the acid control of the sphincter is only one of several factors controlling the activity of the pylorus. Carlson and Litt,¹² experimenting on anaesthetized animals, found that the introduction of sodium bicarbonate into the duodenum is as effective a stimulus to closure of the pylorus as hydrochloric acid, and that acid in the stomach had no effect at all. These observations do not prove that Cannon's conclusions from his radiological experiments on healthy cats were erroneous, but that his generalization was too sweeping.

The investigations of Thomas and Whealon¹³ and of Carlson and Litt¹² have shown that the pyloric sphincter is supplied with both motor and inhibitory fibres both by the vagi and the sympathetic nerves, the character of the response to any stimulus depending upon the tone of the sphincter at the moment. The work of various other investigators proves, however, that the main effect of the vagus on the pyloric sphincter is to cause relaxation, and the main effect of the sympathetic is to cause contraction.

The pyloric canal opens under normal conditions on the approach of every peristaltic wave from the very beginning of digestion, before free acid has appeared in the stomach, until the last trace of chyme has been evacuated, whether this is very acid, as in constitutional hyperchlorhydria, or actually alkaline, as in constitutional achylia gastrica. In this way chyme is pressed through the still narrow canal into the duodenum at a uniform rate.

(b) *Achalasia and Spasm of the Pylorus, and the Origin of Hunger Pain.*

The presence of an ulcer in the duodenum frequently upsets the normal pyloric mechanism by giving rise to a

protective reflex, which results in the inhibition of the relaxation which should occur as each peristaltic wave approaches the sphincter. This does not happen until the later stages of digestion, as it is only when the gastric contents have become highly acid that they act as a sufficient irritant to the exposed nerve endings in the ulcer to cause reflex pyloric achalasia. The irritation by acid does not itself cause pain, as extreme hyperchlorhydria may occur in healthy people with no indigestion, and hyperchlorhydria is just as marked in patients with duodenal ulcer between their attacks or after the ulcer has healed as in the presence of a severe attack. Moreover, I have poured dilute hydrochloric acid of a strength greater than ever occurs in hyperchlorhydria into the empty stomach of normal people and of patients with ulcer without causing any pain.

The pyloric achalasia caused by a duodenal ulcer is at first intermittent, but after a time it becomes almost continuous. Consequently the evacuation of the stomach, which begins with the usual rapidity characteristic of the hypertonic gastric diathesis, in the absence of which a duodenal ulcer rarely if ever develops, becomes abnormally slow in the later stages of digestion. After a time the stasis becomes more marked owing to fatigue of the weakened muscular coat of the stomach, intervals of complete inertia alternating with periods of great peristaltic activity. At the same time the tone of the pyloric vestibule begins to give way, the resulting dilatation to the right of the middle line being in striking contrast with the hypertonic condition of the rest of the stomach, but later still the whole of the stomach becomes dilated. The dilatation and ptosis which may therefore occur in chronic duodenal ulcer are secondary, and are due to the loss of tone and delayed evacuation caused by the pyloric achalasia. If the stomach had been examined at any earlier stage, hypertonus and rapid evacuation would have been found, and these characteristics of the hypersthenic stomach gradually return under successful medical treatment.

The secondary delay and dilatation I have described as a sequel of achalasia have often been erroneously regarded as the result of pylorospasm. Although there is no doubt that in the early stages achalasia accounts for everything, when the inflammation round an active duodenal ulcer invades the pyloric canal spasm of the sphincter may actually occur. During periods of active peristalsis, when the x rays show that nothing is passing through the pylorus, it is still possible to squeeze some of the opaque meal into the duodenum by pressure on the stomach so long as the obstruction is due simply to achalasia. But sometimes nothing can be pressed through; in all probability spasm is then present.

It is important to remember that other conditions give rise to similar disturbances in the functional activity of the pylorus, so that the recognition of these disturbances does not by any means prove that a duodenal ulcer is present. The most common is a gastric ulcer, especially if it is situated in the near neighbourhood of the pylorus. But even a lesser curvature ulcer, situated a considerable distance away, occasionally produces so much disturbance in the activity of the pyloric sphincter that considerable stasis results and pyloric obstruction may be simulated. Disease of the gall bladder, and much less frequently chronic appendicitis, may give rise to reflex achalasia. The disturbances in gastric digestion which result are indistinguishable from those caused by achalasia secondary to a duodenal or gastric ulcer. These clinical facts correspond with the results of the experiments of Carlson and Litt, which showed that stimulation of any sensory visceral nerves may increase the contraction of the pylorus.

Observations with the x rays have proved that the hunger pain of duodenal ulcer occurs when the stomach is nearly empty and disappears when evacuation is complete. This corresponds with the only time that peristalsis can increase the pressure within the pyloric vestibule, as each wave then separates the contents of the small part of the stomach distal to it almost completely from the contents of the large part proximal to it. If the pyloric sphincter relaxes, the pressure spends itself in evacuating part of the chyme into the duodenum, the rest passing back as an axial reflux

stream (Fig. 5, A). But if the sphincter remains closed owing to the achalasia caused by the irritation of the ulcer by the very acid gastric contents two or three hours after a meal, it can only spend itself to a slight extent dynamically

FIG. 5.—Section through pyloric vestibule (P.V.) and duodenal bulb (D) in normal man (A), and in a patient with a duodenal ulcer (B), showing how pain is caused in the latter by pyloric achalasia. *Pe, Pe*, Peristaltic wave, approaching pyloric sphincter (P.S.). *s*, Spasm in bulb caused by ulcer.

by producing an exceptionally active axial reflux stream (Fig. 5, B). Consequently the tension on the muscular coat of the extreme pyloric end of the vestibule is greatly increased. This is the actual cause of pain, the rise in tension on the individual muscle fibres being the adequate stimulus required to produce the afferent nervous impulse, which calls forth the sensation of pain as well as the

associated reflex symptoms of tenderness and rigidity in the corresponding segment of the rectus abdominis muscle. Our investigations several years ago proved that the mucous membrane of the alimentary canal is insensitive to tactile, thermal, and painful stimuli; that neither the normal mucous membrane nor the surface of an ulcer is sensitive to hydrochloric acid of a strength greater than that ever present in the stomach, either in health or disease; and that tension in the muscular coat is the sole cause of the sense of fullness and, when it is sufficiently intense, of the sense of pain. The conditions responsible for the occurrence of achalasia of the pylorus, together with the fact that the tension in the vestibule can only be increased by peristaltic waves when the bulk of the gastric contents is very small, account for all the distinguishing features of the hunger pain of duodenal ulcer. They explain why it begins when the stomach is nearly empty, but differs from simple hunger in ceasing when evacuation is complete, and why relief is obtained by vomiting, and by all kinds of food or drink, which increase the bulk of the gastric contents and at the same time reduce the acidity of the gastric contents and so more or less overcome the achalasia. Sodium bicarbonate not only acts in both these ways, the evolution of carbon dioxide increasing the bulk of the gastric contents and the neutralization of hydrochloric acid overcoming the achalasia, but the sudden rise in the general intragastric pressure caused by the evolution of carbon dioxide at the same time stimulates the whole stomach to contract: the consequent further rise in intragastric pressure overcomes the resistance offered by the unrelaxed but not abnormally contracted pyloric sphincter, and forces some of the gastric contents into the duodenum. Simultaneously gas is forced through the cardia into the oesophagus, from which it escapes by belching. The pressure in the pyloric vestibule is thus relieved and the pain disappears. Many patients, not realizing that the gas has been produced by the action of the acid on the alkali, regard their relief as due to the sodium bicarbonate making it in some way possible for them to get rid of an accumulation of wind.

Other alkalis relieve the pain less completely and less rapidly, though they may neutralize the acid just as efficiently. In the case of magnesium oxide this is because no gas is evolved, and in the case of bismuth carbonate the carbon dioxide is liberated so slowly that it dissolves in the gastric contents as soon as it forms, so that there is no rise in intragastric pressure and no belching occurs.

When air is swallowed it acts in the same way as the gas given off by the action of acid on sodium bicarbonate. The patient misinterprets his discomfort and thinks it is due to flatulence. The repeated attempts he makes to bring up wind only lead to aerophagy. The sudden rise in intragastric pressure caused by the rapid swallowing of air stimulates the stomach to contract; this results in the noisy belching of air and the simultaneous expulsion of

chyme into the duodenum, as Meunier¹⁴ has demonstrated by actual observations with the x rays. The relief that aerophagists always claim to get, which would otherwise be inexplicable, is in this way explained.

When pylorospasm is present it acts in exactly the same way as achalasia, as even extreme spasm of a sphincter does not by itself cause pain, as the tension on the muscle fibres themselves is the same whether they are contracted and short or relaxed and long.

When a gastric ulcer or disease of the gall bladder causes pyloric achalasia the pain produced is identical in character with that of duodenal ulcer. As the exciting cause is different, the time relations, the factors which give relief, and the history of the attacks are generally distinctive. These characteristics depend not on the way the pain originates, which is the same in each case, but on the different ways in which the achalasia of the pylorus is induced.

(c) *Symptoms of Pyloric Obstruction caused by Achalasia of the Pylorus.*

A year ago a man of 53 came under our observation with an eight years' history of recurrent pyloric obstruction; the pain and very severe vomiting always subsided with a diet of small dry meals. When his abdomen was watched after taking an ordinary mixed meal, the stomach was seen and felt to stand out from the surface of the abdomen as a firm mass, along which passed very powerful peristaltic waves. The evidence of pyloric obstruction seemed so clear that I asked Mr. Clayton-Greene to operate, although a previous exploration had revealed nothing abnormal. No organic disease was found, and the pylorus appeared to be perfectly healthy, but the wall of the stomach was definitely hypertrophied. In view of the absence of obstruction nothing was done, and the patient has remained perfectly well ever since by keeping strictly to small dry meals. It seems probable that this was a case of achalasia of the pylorus, and that if the dietetic treatment had not proved successful, a pyloroplasty would have given relief. It is, of course, a surgical law that no operation should be performed on the stomach in the absence of organic disease, but I think that the very rare cases like the one I have described, in which the clinical and radiological evidence shows that pyloric obstruction is present either continuously or intermittently, the question of pyloroplasty deserves consideration if careful dieting fails to give relief.

(d) *Hypertrophic Pyloric Stenosis of Infants.*

The pyloric obstruction present in the hypertrophic stenosis of infants, whatever may be its cause, results in abnormally vigorous peristalsis, and this leads to hypertrophy of the muscular coat of the stomach, particularly the pyloric end, where the waves attain their greatest strength. The obstruction is primarily functional, as many quite typical cases, in which definite hypertrophy of the sphincter has been recognized by palpation, have been cured completely by lavage and dieting, so that it is clear that the hypertrophy itself is not the cause of the obstruction. The obstruction must therefore be due either to achalasia or spasm of the pyloric sphincter. If achalasia were the cause, the violent peristalsis and the contractions of the stomach which can give rise to projectile vomiting would be sufficient to overcome it, and pressure over the stomach, when visualized with the x rays after an opaque meal, would cause some of the chyme to pass through into the duodenum, but this does not in fact occur. It is clear that the obstruction is due to something which produces a much more powerful resistance than simple absence of relaxation. The only possible explanation is spasm. If, as I believe, John Thomson is correct in regarding the spasm as primary, this would explain the development of hypertrophy of the sphincter, and it is the spasm which is overcome by successful medical treatment.

The cause of the spasm, which apparently originates at birth, as the symptoms often begin in the first week of life and always within the first two months, is still quite obscure. It is always associated with hyperchlorhydria, and the condition is several times more common in boys than in girls. It therefore seems possible that the tendency to spasm of the pylorus is the expression in an extremely

exaggerated form of the constitutional condition which I have called the hypersthenic gastric diathesis, which manifests itself by hypertonus, hyperperistalsis, and hyperchlorhydria, and occurs much more commonly in men than in women. The diathesis is often present in several members of a family; this would explain the rare instances in which two and even three or four members of the same family have hypertrophic pyloric stenosis.

THE ILEO-CAECAL SPHINCTER.

(a) Anatomy and Physiology.

The valve-like structure which separates the ileum from the caecum was discovered in 1579 by Caspar Bauhin, who believed that it allowed the free passage of the contents of the ileum into the caecum, but prevented regurgitation in the opposite direction.

Though Mason Good¹⁵ in 1822 described the ileo-caecal junction as a muscular structure, the function of which

FIG. 6.—Section through end of ileum and caecum (after Spalteholz) to show structure of ileo-caecal sphincter. (After Rutherford.) I, ileum; C, caecum; A.C., ascending colon; I.C.S., ileo-caecal sphincter; A, appendix.

was "to moderate the flow" from the small intestine into the colon and "to prohibit a regurgitation of the faeces" from the caecum into the ileum, his work was completely forgotten, and the separation of the contents of the small from those of the large intestine was still universally regarded as a result of the mechanical action of Bauhin's "valve" until 1903, when Keith³ clearly demonstrated the existence of a strong ileo-caecal sphincter. His observations, and the more recent ones of Rutherford,¹⁶ show that the sphincter is formed by the fusion of the circular and longitudinal fibres of the

end of the ileum and those of the adjoining caecum, with the addition of some independent specialized circular fibres at its apex (Fig. 6).

Rutherford in 1914 made some important observations on the human ileo-caecal sphincter through a large caecal fistula. He found that it formed a smooth, scarlet, hemispherical papilla, about 1.8 cm. in diameter, which projected about 1 cm. above the pink and folded mucous membrane of the caecum (Fig. 7). The orifice was closed and formed a dimple in the centre of the papilla. During periods of activity a peristaltic wave could be seen through the relaxed wall of the caecum to traverse the end of the ileum at regular intervals. When it reached the caecum the orifice opened owing to relaxation of the sphincter, which at the same time caused the papilla to become larger and less prominent.

FIG. 7.—Appearance of papilla formed by the ileo-caecal sphincter in man, as seen from the interior of the caecum during life. (After Rutherford.)

About a drachm of fluid faeces, sometimes mixed with flatus, emerged. The sphincter then closed, and became again more firm and prominent. No sign of the fraenula described by anatomists was ever observed.

In 1904 Elliott¹⁷ had demonstrated that the tone of the sphincter depended upon its sympathetic nerve supply, as section of the splanchnic nerves was followed by its permanent relaxation, with the result that the contents of the small and large intestines mixed freely with each other. On the other hand, direct stimulation of these nerves, or indirect stimulation by means of adrenaline, caused the sphincter to contract tightly at the same time as the movements of the rest of the intestine were inhibited. Stimulation of the vagi and pelvic nerves had no effect. Keith³ has found that nodal tissue forming Auerbach's plexus is as remarkably developed at the ileo-caecal sphincter as it is at the cardia.

The main function of the ileo-caecal sphincter is to prevent the contents of the ileum passing too rapidly into the caecum. Alan Newton of Melbourne and I¹⁸ found with the x rays that the barium-containing chyme reaches the end of the ileum an hour or more before any appreciable quantity enters the caecum, and that the ileum is often still full four or five hours after the last traces of barium have left the stomach. Consequently an accumulation of chyme occurs in the terminal ileum, where it actually remains for a greater period than in the stomach. During the whole of this time active segmentation, which leads to thorough churning of its contents, can be seen in the last few inches of the ileum, but there is very little peristalsis. It is evident that the ileo-caecal sphincter has exactly the same effect on the contents of the terminal ileum as the pyloric sphincter has on the contents of the stomach; it controls the onward passage of the chyme so that sufficient time may elapse for digestion and absorption of the food to be complete. This physiological ileal stasis is so well regulated that the chyme which reaches the caecum contains only very small quantities of nutrient material in solution.

The ileo-caecal sphincter relaxes each time a peristaltic wave passes down the last few inches of the ileum. Though these waves begin a short time after the chyme first reaches the ileo-caecal junction, they are at first very infrequent. As a result of the gastro-ileal reflex, which I first described fifteen years ago, active peristalsis occurs in the extreme end of the ileum, the sphincter relaxes, and chyme enters the caecum whenever food enters the stomach.

It was at one time thought that the ileo-caecal sphincter had an important function to perform in preventing the regurgitation of faeces from the colon into the ileum. But we now know that under normal conditions there is no anti-peristalsis in the proximal part of the colon, so that there is never any tendency for the contents of the caecum to pass back into the ileum. When, however, retrograde movement of the contents of the colon occurs in cases of obstruction, and after distension of the bowel with an enema, the sphincter is not capable of withstanding any considerable degree of pressure from the side of the colon, as the x rays show that part of an opaque enema given to normal individuals at no greater pressure than eighteen inches of water frequently passes from the caecum into the end of the ileum.

(b) Pathological Ileal Stasis.

So many ill results have been ascribed to ileal stasis that one requires a certain amount of courage to express a doubt as to whether it is ever of the slightest importance, except in cases of organic obstruction near the ileo-caecal junction due to a growth, tuberculosis, or definite adhesions the sequel of acute appendicitis. But in my experience intestinal poisoning is due far more frequently to the artificial diarrhoea produced by aperients than to intestinal stasis; the only form of intestinal stasis which gives rise to symptoms is that involving the caecum and ascending colon; and, if in exceptional cases this is associated with well marked ileal stasis, it is the colonic rather than the ileal stasis which is the more important. Cases of true small intestine poisoning, such as the streptococcal infection of the duodenum constantly present in Addison's anaemia, are secondary to gastric achylia and not to colon infection, and they are never associated with ileal stasis.

When ileal stasis occurs in the absence of organic obstruction, it is generally due to reflex achalasia of the ileo-caecal sphincter caused by chronic appendicitis, exactly corresponding to pyloric achalasia in duodenal ulcer. Kinks of the terminal ileum are doubtless interesting developmentally, but they are of no clinical importance, as they never lead to sufficient narrowing of the lumen to obstruct the passage of the fluid contents of the ileum, for peristalsis is wonderfully adapted for propelling the contents of a muscular tube round bends, whether they are normal flexures or so-called kinks. If a kink caused true obstruction it would not be seen with the x rays, because the distal segment would be empty. The so-called "controlling appendix" is never the direct cause of ileal obstruction; it may control the passage of faeces into the caecum by causing achalasia of the ileo-caecal sphincter, but only if it is chronically inflamed.

THE PELVI-RECTAL AND ANAL SPHINCTERS.

(a) Anatomy and Physiology.

In his *New Views of the Process of Defecation*, published in 1833, James O'Beirne drew attention to the important function of the flexure, formed where the movable pelvic colon joins the fixed rectum at an acute angle, which is best described, I think, as the pelvi-rectal flexure.

FIG. 8.—Frozen section through pelvi-rectal flexure, showing thickening of circular muscle layer forming termination of pelvi-rectal sphincter (P.R.S.). P.C., Pelvic colon; R., rectum; Pe, Pe, peritoneum; s, sacrum. (After Symington.)

O'Beirne from the results of digital examination of the rectum and of experiments with a rectal tube, that under normal conditions the faeces do not pass beyond the pelvi-rectal flexure, but collect in the pelvic colon until immediately before defaecation. I believe that this is due to the presence of a true pelvi-rectal sphincter, which resembles the cardiac sphincter rather than the pyloric and anal sphincters in depending upon its postural tone being sufficiently great to obliterate its lumen rather than upon any considerable development of a special band of circular muscle fibres. W. J. Mayo¹⁹ has pointed out that the folds of mucous membrane are disposed in a longitudinal direction in the last two inches of the pelvic colon, in contrast with their circular arrangement in the remaining portion; this suggests that the lumen of the last two inches is normally obliterated owing to a sphincter action of the circular muscle fibres analogous to what I have described in connexion with the abdominal portion of the oesophagus (Fig. 9). The sphincter's action is considerably assisted by

FIG. 9.—Diagrammatic section through last 3 inches of pelvic colon, rectum, and anal canal, showing pelvi-rectal sphincter (P.R.S.) with its longitudinal folds, ending at pelvi-rectal flexure (P.R.F.). A.R., Ampulla recti; A.A., anal canal, surrounded by internal sphincter ani (I.S.A.) and external sphincter ani (E.S.A.), and strengthened by levator ani muscles (L.A.). H¹, H², H³, the three folds of Houston.

lectures showed that the sense of fullness produced by distension of the colon immediately above the pelvi-rectal flexure was referred to the front of the abdomen just above the pubes, in striking contrast to the perineal sensation—the call to defaecation—felt on distension of any part of the rectum. When the call to defaecation meets with its natural response, the pressure in the rectum is further increased by the voluntary contraction of the diaphragm and abdominal muscles; this sets up the reflex act of defaecation—a wave of peristalsis down the rectum followed by active relaxation of the powerful sphincter ani, which causes the faeces to be evacuated from the rectum through the narrow anal canal.

Whether there is a true sphincter at this point is still uncertain, but Symington's sections through the frozen body certainly show a definite thickening of the circular muscle fibres at the junction (Fig. 8), and Reeves found a definite narrowing of the lumen of the colon at this point in 80 per cent. of 46 cadavers.

Observations by direct palpation and with the x rays and the sigmoidoscope confirm the conclusion drawn by the very acute angle formed where the dependent pelvic colon joins the fixed rectum, especially in the erect position.

The gastro-colic reflex which occurs when food enters the stomach results after breakfast in a wave of peristalsis passing along the distal part of the large intestine; the obstruction at the pelvi-rectal flexure is overcome, presumably as a result of relaxation of the sphincter, and the pelvic colon empties itself into the rectum. The sudden distension of the muscular walls of the rectum gives rise to a sensation in the perineum which is recognized as the call to defaecation. The observations

recorded in my Goulstonian

(b) *Hirschsprung's Disease, the Megacolon of Adults, and Achalasia of the Pelvi-Rectal and Anal Sphincters.*

The condition described in 1886 by Hirschsprung of Copenhagen, in which children, most often boys, from early infancy become progressively more constipated owing to the failure of the dilated and hypertrophied colon to empty itself, is much less rare than was at first believed. Severe cases generally end fatally before the age of 8, but investigations with the x rays and the sigmoidoscope show that patients suffering from a milder form of the condition may grow up in sufficiently good health to lead normally active lives. One of my patients was, indeed, an Oxford rowing blue. It is remarkable how greatly dilated the colon may be without causing any protrusion of the abdominal wall, and how, as in three of my cases, a hugely dilated loop of pelvic colon may actually be situated above the liver without giving rise to any symptoms. There does not appear to be any clear distinction between the cases of megacolon in adults, who complain of nothing beyond a moderate degree of constipation, and the fatal cases of Hirschsprung's disease in young children. In the former the hypertrophy of its muscular coat has made it possible for the colon to overcome the obstruction at the pelvi-rectal flexure or anal canal sufficiently to prevent the accumulation of enough flatus and faeces to cause a dangerous degree of dilatation. In the latter the hypertrophy has not kept pace with the dilatation, and colon failure—analogous to heart failure in mitral stenosis—finally occurs with fatal results.

The lower limit of the dilated colon is with equal frequency at the pelvi-rectal flexure and at the sphincter ani. It has always been recognized that no organic obstruction is present in these cases, although the violent peristaltic contractions of the colon which can be seen through the thin abdominal wall and the hypertrophy of the muscular coat which results from this show that some functional obstruction must be present. The obstruction must, I believe, be due, in the cases in which the dilatation begins at the pelvi-rectal flexure, to achalasia of the pelvi-rectal sphincter, when, at the commencement of defaecation, a peristaltic wave passes down the pelvic colon and reaches the flexure in the process of emptying the pelvic colon into the rectum. In the cases in which the rectum is dilated a similar condition of achalasia affects the sphincter ani, which fails to relax when the peristaltic wave which should evacuate the rectum reaches it. In both varieties the condition of the colon is strictly comparable to the dilated and hypertrophied oesophagus, which, I have shown, is caused by achalasia of the cardia.

Anal achalasia never calls for surgery. It is quite easy to keep the colon empty by lavage, as a tube can always be readily passed through the sphincter. This should be done every day at first, then every two or three days, and finally, only if an accumulation appears to be forming. An attempt should be made at the outset to diminish the resistance offered by the unrelaxed anal sphincter by forcibly dilating it, in spite of the fact that it is never found to offer more than the normal resistance when a finger is inserted into the rectum, and that no hypertrophy of the sphincter is ever present, in contrast with that which follows the long-continued anal spasm caused by a chronic anal ulcer.

In pelvi-rectal achalasia also the colon must be emptied by daily lavage. But in some cases the water which reaches the dilated pelvic colon remains in it, and nothing is evacuated. An attempt should then be made, with the patient in the knee-elbow position, to manipulate a tube, through which water is running, past the pelvi-rectal

FIG. 10.—Composite drawing of a megacolon, due to anal achalasia in a woman of 43, made from radiographic observations after an opaque meal and an opaque enema. C, Cecum; A.C., ascending colon; T.C., transverse colon, ascending limb of which passes to S.F., splenic flexure, outside D.C., descending colon, which merges into the hugely dilated pelvic colon, P.C.; this reaches above the liver, which is shown shaded, and finally joins R, the dilated rectum.

flexure. If this does not succeed, a sigmoidoscope must be passed and the rubber tube introduced through it. If this also fails, the abdomen must be opened. On pulling the distended colon upwards, a tube can be passed into it from the anus, and the bowel is then emptied. After this has been done it may be possible to pass the tube daily in the manner already described, as the obstruction originally caused by the pelvi-rectal achalasia is always increased by the kink produced by the greatly dilated pelvic colon where it joins the undilated rectum. If, however, this eventually becomes impossible, an anastomosis should be made between the undistended rectum and the terminal ileum, or, preferably, the under surface of the extreme end of the pelvic colon. It is, however, exceedingly difficult to do this, and it may even be quite impossible owing to the small size of the rectum and to its situation deep in the pelvis. In such cases nothing short of excision of the whole of the dilated colon is likely to give relief.

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RADIOLOGICAL EXAMINATION IN ORGANIC DISEASES OF THE COLON:

THE OPAQUE ENEMA METHOD.

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THE clinical evidence presented in most cases of colonic disease is meagre, variable, and often unreliable.

The sigmoidoscope will give direct evidence if the lesion is within its reach, but unfortunately this does not often happen. At its best, this form of examination only covers the last few inches of the colon itself. On account of the low malignancy of the majority of cancerous growths of the colon, their early diagnosis is of great importance, and any method of examination whereby direct evidence may be obtained before operation is valuable.

The radiological examination, using some opaque fluid as the medium, if carried out correctly, will give reliable and definite information in diagnosing the presence of an organic lesion of the colon. It is, of course, not always possible to distinguish between certain types of lesion, such as tuberculosis and carcinoma of the gut, but the fact remains that by a radiological examination the disease may be detected and located, which is of considerable surgical importance.

The radiological methods at command are: (1) the opaque meal given by the mouth and followed through to the anus—at one time the only routine in use, and (2) the opaque enema. Both methods have their advocates, but those who have had the opportunity of examining a number of colonic cases will have no hesitation in deciding which of the two is the more reliable. I entertain no doubt that the opaque enema is the only practical way of making a detailed examination of the lumen of the large gut. It is, in fact, as valuable as the opaque meal in the detection of gastric lesions.

Advocates of the opaque meal examination may say that a colonic obstruction can be detected in this way, but this is certainly not correct in a number of mechanical obstructions due to new growths. At its best it is unreliable; I have seen cases of colonic carcinoma where there has been no delay or obstruction in the passage of the meal from

the mouth to the anus, and yet there has been complete obstruction to the opaque enema. The reason for this has not been satisfactorily explained, but the fact remains.

The two chief disadvantages attached to the opaque meal method are: (1) That the examination is prolonged; several days, even weeks, may be required before the colon clears itself, and the final result, even then, is unreliable. (2) The second disadvantage is that colonic haustration or segmentation is very marked, the uneven distribution of the opaque salt rendering a detailed examination almost impossible.

The advantages of the opaque enema are that the examination is rapid, the whole gut being filled from rectum to caecum in a few minutes; that the almost complete absence of haustration renders small filling defects of the lumen easily detectable; and that if the enema is properly administered there is practically no discomfort to the patient.

The disadvantages are few and insignificant, compared with the data obtainable. My colleagues say it is a "messy" job, but except for occasional accidents not a drop of the opaque substance used should escape outside the body. Excluding accidents, a "mess" is indicative of faulty technique. The alleged discomfort for the patient has already been dealt with. It is a curious fact that the large majority of patients display no concern whatever, even when the colon is fully distended from rectum to caecum. This is possibly due to the preparatory wash-outs.

METHOD OF EXAMINATION.

Unless the opaque enema examination is carried out with careful attention to technical details it may lead to serious errors in diagnosis, discomfort to the patient, as well as to the operator, and its value as a diagnostic agent will be lessened.

Preparation of the Patient.

As our object is to inject a foreign substance in an organ which normally is already full, a clearance of its contents is essential. In other words, the colon must be free of faeces. Anyone who has seen the opaque fluid obstructed by a hard mass of faeces will realize the importance of efficient clearance, as its appearance is remarkably like the obstruction of a new growth.

Castor oil is the most reliable aperient. This should be administered at least thirty-six hours before the examination, for unless the effect has passed off by the time operations begin there is a risk of the opaque enema being returned. Light diet follows the aperient. A plain water enema is administered the night before and a second on the morning of the examination. This procedure will ensure an empty colon. The medical man in charge of the case should fully realize the importance of this preliminary preparation indicated by the radiologist. Serious errors in diagnosis may otherwise be made.

Apparatus.

A douche-can of the usual pattern, in which the outlet is about an inch from the bottom, is the most suitable. Any sediment that may collect can thus settle down without blocking the tube. I have tried several other patterns, but this type is the best. About four feet of thick-walled rubber tubing is attached to the can and to the end a Higginson's syringe is added, the valves of the syringe being removed, as they would soon become blocked. The use of the bulb of the syringe will be discussed later. The acorn-shaped rectal nozzle made in metal will be found to be the most suitable. It throws a shadow, so that its correct position can be verified before the injection takes place, and its shape renders it less liable to slip out.

The douche-can, which is placed about two feet above the patient, is now filled three-quarters full ($1\frac{1}{2}$ pints) with the barium emulsion (Ramul No. 3) warmed to body temperature. The clip which is placed on the tube to control the flow is released and the emulsion run up to the nozzle; this excludes the air. The clip is now closed. The end of the nozzle only is smeared with a small quantity of vaseline. If the whole is lubricated there is a tendency for it to slip out during the injection. The patient, suitably attired, lies on his back, with a towel under the buttocks and a pad of cotton-wool under the sacrum. With nervous patients,