

# The Cavendish Lecture

ON

## A NEW THEORY OF THE CAUSATION OF ENTEROSTASIS.

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MR. PRESIDENT AND GENTLEMEN,—Only those of us who had completed our medical studies before 1895 are fully aware of the extent to which our knowledge of the living human body has been revolutionised by the discovery and application of the Roentgen rays. Before 1895 we had no accurate information regarding the rate of progression of food along the alimentary tract; we are now in a very different position, thanks to the labours of a younger generation, which has applied and developed Roentgen's discovery.

We now know that food is normally delayed at certain points of the alimentary canal; at the junction of the œsophagus there is a mechanism which causes a temporary delay; there is another at the junction of the stomach and duodenum which effects a more prolonged delay of the gastric contents; there is evidence for the existence of a regulating mechanism near the duodeno-jejunal junction; there is certainly a mechanism at the lower end of the ileum which regulates and retards the passage of contents from the small to the large intestine. Along the great intestine there appear to be three points at which delay may be regarded as normal: (1) Near the commencement of the transverse colon, leading to the filling of the cæcum and ascending colon; (2) at the recto-colic junction, leading to accumulation in the pelvic colon; (3) above the anal canal, leading to accumulation in the rectum. We also now know that the degree of delay at those points differs from one person to another, but after making all allowance on the score of individual variation there remain a considerable percentage of cases in which the delay at one or more of these points is so long that it must be regarded as pathological. We have been in the habit for some time past of speaking of abnormal delay above the ileo-colic junction as "ileal stasis"; clinicians also speak and write of "duodenal stasis," "gastric stasis," stasis of the proximal colon, and stasis of the distal colon; we may therefore legitimately admit œsophageal and rectal stasis to the same category. In the present state of our knowledge we may regard all of those enterostatic conditions as morbid exaggerations of normal delays which take place in the passage of alimentary contents, and may conveniently group all of them under the name of "enterostasis."

It is to the condition of enterostasis I propose to devote the Cavendish lecture of this year of war—1915—and to lay before you certain observations and inferences which appear to throw light on its nature and causation.

### "NODAL" TISSUE IN THE BOWEL.

Some years ago I again took up a research on the ileo-cæcal region of the bowel, with no thought of elucidating the condition I have described to you as enterostasis, but with the definite object of throwing more light on that system of tissues in which the heart beat arises, and by which it is propagated throughout auricles and ventricles. If No. 4799.

we were to find the beginnings of such a system, it was clear by then that the heart, a highly specialised hollow complex muscle, was not the organ we should find them in. The bowel, because of its structure and manner of action, seemed very likely to yield information regarding the early history of the "nodal" and "bundle" systems of the heart. I selected the ileo-cæcal junction of the rat's bowel as the site of my initial inquiry, because I knew that lively and complex movements arose at that junction, and inferred that if a special tissue were present in the bowel, representing the nodal tissue in which the heart beat takes its normal origin, it should be found at that site. I further inferred that if "nodal" tissue did occur there it must be directly interposed between the muscle fibres, on the one hand, and nerve fibres and nerve cells on the other; it must be of the nature of neuro-muscular tissue. It is difficult to unravel the true nature of nodal tissue in the heart of man or of the higher mammals, but in the heart of the monotreme echidna, Dr. Ivy Mackenzie and I were able to convince ourselves that there was a true and direct union between nerve and muscle fibres in the sino-auricular node.

A series of sections through the ileo-cæcal junction of the rat's bowel revealed a collar of peculiar tissue, with two extensions which passed into the anterior and posterior walls of the cæcum. In the collar or ring could be recognised nerve cells and nerve fibres, but there was also present a third element—numerous branching cells, not connective tissue in nature—with processes which united with muscle cells, on the one hand, and with the processes from true ganglionic cells on the other. I regarded these intermediate cells as a possible representation of the nodal tissue of the heart.<sup>1</sup> It was also clear from an examination of the series of sections that the ileo-colic collar was merely a local specialisation of the myenteric (Auerbach's) plexus.

### STRUCTURE OF THE MYENTERIC PLEXUS.

I therefore began a systematic examination of Auerbach's plexus, using the methods we had found most successful while investigating the nodal system of the heart—namely, fixing specimens by a mixture of formalin and alcohol and staining with van Gieson's reagent. A number of fortunate circumstances gave me material for investigation. Sir W. Arbuthnot Lane, Sir Berkeley Moynihan, and Professor J. W. Smith gave me specimens which they had removed for the relief of intestinal stasis, and which they had preserved immediately after removal by operation, so that the tissues were perfectly preserved. I owe a special debt to my friend, Dr. W. C. Mackenzie, who placed at my disposal an extensive series of preparations showing the microscopic structure of the alimentary tract of Australian mammals, particularly of the monotremes—platypus and echidna. Professor William Wright, who had noted independently the peculiar structure of the myenteric (Auerbach's) plexus, gave me the loan of a series of undescribed preparations from the alimentary tract of the elephant. Mr. H. Wilson, prosector to the Royal College of Surgeons, provided preparations made from the alimentary tract of many vertebrate animals, so that it was possible for me to found my deductions on a fairly wide series of observations.

<sup>1</sup> For a preliminary description of the ileo-cæcal collar see British Journal of Surgery, 1915, vol. ii., p. 576.

The total result was to convince me that the myenteric plexus is not a simple structure composed of merely nerve cells and nerve fibres, but one of a composite texture. There are ganglion cells in the plexus and there is an ample network of particularly fine fibres. But there is also an abundant third element in the plexus—the branching intermediate cells which have been already mentioned, and which appear to become continuous with processes of certain groups of muscle cells on the one hand and with the branched processes of ganglionic cells contained in the nerve fibres of the plexus. Indeed, it is often difficult to say whether a certain intermediate branched cell is to be regarded as a muscle cell or as one of the cellular elements of the plexus, so alike are they in form and in staining reaction. In the ileo-colic junction of all mammals I met with an elaboration and modification in the myenteric plexus; in the wombat, for instance, in which the cæcum is highly developed, the ileo-colic specialisation of the myenteric tissue is very evident. On looking into the literature of the subject I found that Koelliker, in one of his earliest researches, had recognised a cellular element in the myenteric plexus which was different from any form of cell occurring in other nerve plexuses. I found further that Dr. L. R. Müller,<sup>2</sup> in an account published four years ago on the innervation of the bowel, states definitely that the myenteric plexus differs from a true nerve plexus both in structure and in staining reaction. He found, as I have done, that the methods which are usually effective in demonstrating the structure of true nerve tissue do not serve to bring out all the structural elements which go to make up the intramuscular plexus in the bowel.

#### FUNCTION OF THE MYENTERIC PLEXUS.

I therefore adopted the following conception as a working hypothesis: that the myenteric plexus represents a nodal and conducting system. I regard the intermediate branched cell as capable of assuming the appearance of either a nerve element or of a muscle element. In the heart, for instance, I hold the opinion that the sino-auricular and auriculo-ventricular nodes represent unmodified or slightly modified parts of its original myocardial nerve-plexus system, while the auriculo-ventricular bundle also represents a part of the original plexus of the heart, but a part in which the intermediate cell element had assumed more or less perfectly the form of a muscle cell and had almost replaced the nerve fibres and ganglion cells of the original plexus. Further, I was prepared to believe, on the ground of my anatomical observations, that, as in the heart, the efferent nerves, which influence the contraction, tone, and rhythm of the bowel, would not end directly in the musculature of the bowel, but in the tissue of the myenteric plexus, and that it was through the intermediation of the intermediate cell elements of the plexus that the splanchnics and vagi effected their influence on the movements of the bowel. In reaching this conclusion I was largely influenced by the observations made by Dr. T. R. Elliott<sup>3</sup> when he discovered that adrenalin had the same effect on the lower iliac tract as stimulation of the splanchnic nerve. In both cases he found that the ileo-colic sphincter contracted, while the rest of the iliac musculature was inhibited. The adrenalin acted, so he inferred, on some element interposed between the terminals

of the splanchnic nerve and the muscle cells. The intermediate cells of the myenteric plexus answer very well to the element postulated by Dr. Elliott.

#### MANNER OF DEVELOPMENT.

If I am right in presuming that the myenteric plexus represents in the intestine a system which corresponds to the nodal and conducting system of the heart, then it is also to be expected that both systems should be developed in a corresponding manner. The bundle system of the heart, it will be remembered, arises from the muscle-forming stratum which lies immediately beneath the endocardium. An examination of the developing human intestine shows that the myenteric plexus arises in a corresponding manner. A section across any part of the intestine of a human foetus in the fourth month of development shows the two muscular coats—longitudinal and circular—separated by an intermediate cellular layer, which is clearly myogenic in nature, for one can see muscle cells being differentiated from it, and being added to the longitudinal coat on the one hand and to the circular coat on the other. It is out of the same intermediate cellular layer that the myenteric plexus is developed. One may suppose that the true ganglionic cells found in the fully developed plexus are immigrant neuroblasts derived from the neural cord, but the other cells of the plexus, the intermediate branched cells, I presume to be native to the embryogenic muscle layer and really to represent immature muscle cells. Thus, a similarity in the manner of development favours the theory on which I am proceeding—that the bundle system of the heart and the myenteric plexus of the intestine represent corresponding functional structures.

#### RESULTS OF EXAMINATION OF VARIOUS PARTS OF THE ALIMENTARY TRACT.

My next step was to examine other parts of the alimentary tract where peristaltic movements are known to arise. Radiologists are almost unanimous in the opinion that the peristaltic waves which sweep along the pyloric part of the stomach arise in the distal or lower part of the body of the stomach. The only exception to this statement I have seen is one made by Dr. L. Gregory Cole<sup>4</sup>—viz., “that when food is in contact with the cardiac end of the stomach contractions begin near the fundus.” A series of sections were therefore made of the stomach of a full-time child in search of a nodal centre in which the pyloric movements might take their origin. The search revealed the fact that the myenteric plexus was well developed in the pyloric division of the stomach and along the lesser curvature, but elsewhere in the fundus and along the greater part of the body of the stomach it was much less abundant. The search did not reveal any localised development of the plexus at or near the site where gastric movements appear to take their origin.

#### *The Cardia of the Stomach.*

This search in the stomach proving a disappointment, it occurred to me that the ileo-colic collar I have described might be merely part of the sphincteric mechanism which lies at the juncture of the small and great intestines. I therefore commenced an examination of the various sphincteric regions of the alimentary tract. One of the regions examined was that of the cardia of the stomach, the sections made involving the lower end of the oesophagus and adjacent part of the stomach. The sections which Mr. Wilson made from this region of

<sup>2</sup> Dr. L. R. Müller, *Deutsches Archiv für Klinische Medizin*, 1911, vol. cv., p. 1.

<sup>3</sup> *Journal of Physiology*, 1904, vol. xxxi., p. 157.

<sup>4</sup> *Archives of the Roentgen Ray*, December, 1911.

the stomach of cats and rats showed a distinct modification of the musculature and myenteric plexus just distal to the ring which marks the cessation of the oesophageal epithelium and the commencement of the gastric lining. At that site there was a definite development of neuro-muscular junctional tissue—just such an area as might serve as a nodal centre for the stomach. The structural modification at this site in the human stomach is less definite than in the rat and cat; nevertheless, one can be certain of its presence.

My belief that there was such a nodal centre at the gastro-oesophageal junction of the mammalian stomach was confirmed by an examination of Dr. W. C. Mackenzie's preparations. In the echidna, for instance, I found very similar tissue to that seen in the sino-auricular node of the same animal. Dr. Mackenzie and I found that by mechanical stimulation of the site of the nodal centre of the living stomach of a dead rabbit we could provoke, after a very short latent period, a definite form of gastric movement of more than passing interest. The movement commenced with a contraction of the gastric musculature in the immediate neighbourhood of the cardia, spreading along the smaller curvature towards the hiatus angularis, when it apparently ceased. The fundus and the greater curvature were not involved. We noted also that each cardiac movement, of the kind just described, was followed by a peristaltic wave along the pyloric part of the stomach—the movement with which radiologists are familiar. There was a distinct interval between the cardiac and pyloric contractions, but there could be no mistake about the sequence—the pyloric movement was a direct result of the initial cardiac one. I am of opinion, then, that there exists, at the gastro-oesophageal junction, a main nodal centre at which the rhythmical contractions of the stomach are initiated.

#### *The Duodenum.*

Another minor investigation drew my attention to the peculiar form and distribution of the myenteric plexus in the duodenum. Mr. Wilson had made a series of sections across the termination of the common bile duct and ampulla of Vater, so that one could study the arrangement and nerve-supply of the sphincter which surrounds the termination of the common bile duct and pancreatic duct. This sphincter is apparently concerned in regulating the flow of bile and pancreatic juice. Amongst the sections there was a series made from a human foetus in the fifth month of development. I was struck by the rich development of the myenteric plexus—or tissue which gives rise to the plexus—in the wall of the duodenum in the neighbourhood of the ampulla of Vater. One could trace the nerve tissue from the solar plexus forming unions with it; one could see, too, that the ampullary sphincter was supplied by a direct extension from the myenteric tissue of the duodenum. An examination of the first or horizontal part of duodenum showed a poor and scattered development of the myenteric plexus; its strands appear to be direct extensions from the richer plexus in the sphincteric region of the stomach. In the second part of the duodenum the myenteric plexus is richly developed, especially on the area of the duodenum lying against the head of the pancreas above or proximal to the entrance of the common bile duct.

Radiologists are well aware that the first part of the duodenum shows only a limited degree of contractility; propulsion waves appear to arise in the

second part of the duodenum. Although I have found no limited area of nodal tissue which might serve as a main centre for the duodenum, yet I am of opinion that it will be found that the richer site mentioned above—proximal to the entrance of the common bile duct—serves as such a centre.

#### *The Colon.*

A systematic examination of the specimens sent to the Museum of the Royal College of Surgeons by Sir W. Arbuthnot Lane and Sir Berkeley Moynihan gave me a knowledge of the distribution of the myenteric plexus in the great bowel and the various lesions it is subject to in cases of colitis or of intestinal stasis. I observed that the plexus was very plentiful in the distal part of the transverse colon, in the descending and iliac colons. It is well known to be highly developed in the rectum. Nowhere did I find any special plaque or area of nodal tissue, but everywhere—particularly in the descending colon—there was an abundance of intermediate cells. I was inclined to correlate the tonic properties of the transverse and descending colons with the richness of their myenteric plexus. I noted, too, that in cases of colitis there was the same form of fibrosis of the muscular coat as one sees in the myocardium of diseased hearts—a fibrosis in which meshes of the myenteric plexus are often involved. I noted, further, that in cases of colitis and of intestinal stasis there were structural changes in the cellular elements of the plexus.

#### THE RHYTHM OF DIFFERENT INTESTINAL SEGMENTS.

My search for a nodal system along the alimentary tract had reached the stage just described when I had the good fortune to visit Dr. W. B. Cannon in his laboratory at Harvard University, where I found him using pieces of living bowel as agents for estimating the quantity of adrenalin in any given solution. He told me of a research lately carried out in his laboratory by Dr. Alvarez, who found that the commencement of the second part of the duodenum dominated the rhythm for the whole duodenal loop. When the duodenal loop was cut out in segments and each segment kept alive in Locke's solution, the rhythm or beat was fastest in the segment from the commencement of the second stage and slowest in the segment from the end of the loop. On my theory the upper segment had the greater amount of nodal tissue, and was therefore the pacemaker of the duodenal rhythm.

Dr. Alvarez found that the first segment of the jejunum has a slightly higher or faster rhythm than in the last part of the duodenum, but that from the first part of the jejunum to almost the last part of the ileum the rate of rhythm decreased. At the last part of the ileum, if a piece of the ileo-cæcal junction were left attached to it, the rate actually increased, but only if the ileo-cæcal junction were left attached. That fact interested me, because it was explicable if I were right in regarding the ileo-colic collar as a nodal centre—as a pacemaker for the cæcum and ascending colon.

#### SPHINCTERIC MECHANISMS.

Before reading Dr. Alvarez's researches I was seeking for an explanation of the various conditions I have described under the name of enterostasis in a derangement of certain sphincteric mechanisms which are distributed, for functional purposes, along the alimentary tract. My conception of the organisation of these mechanisms was founded on the workings of the sphincters of the bladder and of the anus. The sphincters

of the urethra and anal canal are inhibited whenever the contents of the bladder or rectum are brought in contact with the mucous membrane lining them. One could conceive these sphincteric mechanisms being damaged and deranged in many different ways. Everyone knows that a pill or tabloid may be arrested at the cardiac orifice of the stomach. Its arrest is very probably due to the fact that it cannot enter the orifice as a fluid bolus can, and thus bring about the inhibition of the sphincter at the cardia. We are becoming aware that the relaxation of the pyloric sphincter may be influenced by many different conditions. It is true that there is no structural differentiation of a sphincter in the terminal part of the duodenum, and yet I am certain that duodenal distension and stasis is not an infrequent condition.

Dr. A. C. Jordan and other radiologists have published many photographs demonstrating the condition. I agree with Dr. A. F. Hertz that duodenal stasis is not due to a "kinking" at the duodeno-jejunal junction, for all radiograms I have examined of this condition show that the blockage is near the junction of the second with the third parts of the duodenum, at a point where "kinking" or a mechanical form of obstruction could not be produced. At the point where duodenal blockage takes place there is neither an anatomical sphincter nor an arrangement of parts which could produce an obstruction by mechanical means.

#### ILEAL AND COLIC STASIS.

In ileal stasis, the most common and most serious of enterostatic conditions, there is clearly some obstructive force at work in the neighbourhood of the ileo-cæcal junction, delaying the passage of the iliac contents. There is no well-marked anatomical sphincter there comparable to the pyloric sphincter, but beyond doubt there is, in the terminal part of the ileum, an elaborately organised sphincteric mechanism, and there are also peritoneal bands and folds, both of embryonic and post-embryonic origin. One can perceive that the sphincteric mechanism might become disordered, or that in the upright position of the body the peritoneal bands might produce a mechanical obstruction; in either case, an ileal stasis would result. A critical examination of radiograms taken from cases of ileal stasis does show a number in which the bismuth shadow in the terminal part of the ileum is reduced to a thread, or is empty as if there was a spastic condition of the terminal iliac sphincteric tract, but I have not seen a single radiogram showing a sharply marked obliteration of the terminal ileum, such as might be caused by the constricting or dragging action of a peritoneal band or adherent appendix. In the majority of cases of ileal stasis there is neither evidence of a spastic sphincter nor of an obstructing band. There is clearly some other factor at work, which we have not yet taken into account.

Stasis of the proximal colon—of the cæcum and ascending colon—may be explained by an appeal to mechanical means or to a functional derangement of the colic musculature, or to a combination of both of these factors. Sir W. Arbuthnot Lane is persuaded that the delay in the proximal colon is mainly the result of mechanical means, of prolapse of various segments of the colon with the formation of peritoneal bands and the production of obstructing kinks. Radiograms have failed to produce evidence of obstruction situated at bands or kinks; there is no hypertrophy of the muscular coats above

the site of such bands or kinks; when acute flexures of the bowel are produced experimentally, stasis does not result (Murphy and Cannon); those familiar with comparative anatomy know how much the bowel may be inflected, twisted, coiled, or acutely folded without any obstruction resulting. On the other hand, the evidence is fairly complete which points to a gross derangement in the action of the musculature of the transverse, descending, and iliac colons in cases of stasis of the great bowel. There is a general agreement among clinicians that in most cases of colic constipation there is a marked increase in the tonus of these segments of the colon; in many cases the muscle has become almost spastic in its manner of working.

#### INTESTINAL RHYTHM.

It will be thus seen that I do not think either mechanical conditions or even derangements of sphincteric mechanisms can give an adequate explanation of all the phenomena of enterostasis. But when we transfer from the heart to the alimentary tract not only the anatomical and physiological data relating to its nodal and conducting system, but also our knowledge of cardiac pathology of heart-block or auricular fibrillation, of extrasystole, and of delay in conductivity, we seem to reach a more rational explanation of the motor derangements of the alimentary tract. One has to remember a fact with which physiologists are familiar—namely, that all the non-striated muscle of the body, the musculature of the alimentary tract, of the bladder, uterus, and ureter, has the same property as cardiac muscle—a power of rhythmical contraction which it continues to exercise as long as it is alive. I do not know who was the first to show that any piece of excised intestine manifested that property; my own knowledge of it was derived from the well-known paper published by Bayliss and Starling in 1899.<sup>5</sup> They demonstrated the intrinsic beat of bowel muscle; they found that every piece of bowel possessed a double rhythm or beat—a rapid one in which contraction waves arose at the rate of 10 to 20 a minute, and a slow and concurrent one in which the contraction waves arose at the rate of one or two a minute. They further showed that the slow beats gave rise to true peristaltic waves. The more rapid and slight rhythm was concerned with division and mixing of the intestinal contents. It is also probable, as Mall<sup>6</sup> originally indicated, that the more rapid and rhythmical contraction is directly concerned in the maintenance of the portal circulation. The musculature of the whole intestinal tract has its pulse. The appendix, although it has a peculiar arrangement of the myenteric plexus and evidently possesses a peculiar mechanism for becoming filled and emptied, has also a double rhythm of the same kind as is found in the rest of the alimentary tract.<sup>7</sup>

#### Rhythmical Zones.

The researches carried out by Dr. Alvarez<sup>8</sup> in Dr. Cannon's laboratory carry our knowledge of the intestinal rhythm another step onwards. His investigations were concerned with only two parts of the alimentary tract, but so far as they relate to these parts they have a definite significance. The inherent power of rhythmical contraction, as

<sup>5</sup> Journal of Physiology, 1899, vol. xxiv., p. 99.

<sup>6</sup> Johns Hopkins Hospital Bulletin, 1896, vol. i., p. 37.

<sup>7</sup> See Observations on the Movements of the Isolated Appendix, by James A. Gunn and R. H. A. Whitelocke, British Journal of Surgery, 1914, vol. ii., p. 92.

<sup>8</sup> American Journal of Physiology, 1914, vol. xxxv., p. 177; 1915, vol. xxxvii., p. 267.

measured by the number which arise per minute, decreases as one passes along the duodenal loop; at the commencement of the jejunum there is an enhanced power which decreases as one descends to the terminal part of the ileum. At the ileo-colic junction we enter a new zone, with a higher initial rhythm than that of the terminal part of the iliac zone, for from Dr. Alvarez's experiments I infer that the ileo-colic collar represents the pacemaker of the proximal colon. How far the proximal colic zone extends in the human bowel one cannot as yet tell, but it probably stretches beyond the hepatic flexure into the transverse colon, reaching a point corresponding to that in the colon of the cat and dog, where antiperistaltic waves are known to arise. Antiperistaltic waves are only possible when a distal nodal centre has gained or possesses an ascendancy in excitability over the centre which lies at a higher proximal point in the alimentary tract.

So far I have sought to distinguish four rhythmical zones in the intestine—a duodenal, a jejuno-iliac, a proximal colic, and a distal colic. But on the imperfect evidence already available there are grounds for presuming two more—a gastric and œsophageal. I am of opinion that the nodal tissue round the cardia acts as the pacemaker of the stomach; the gastric rhythm appears to arise there and spread along the lesser curvature to the pyloric part of the stomach, where the gastric zone ends.

#### *Inhibition of Peristaltic Waves.*

Why is it that the gastric waves do not spread into the duodenum and dominate the rhythm of that segment of the bowel? Between the stomach and duodenum there is every form of bridge required for the conduction of an impulse; there is a direct connexion of musculature and of myenteric plexus, and yet physiologists and radiologists agree that gastric waves do not pass beyond the pylorus. They are inhibited or arrested there. I think the peculiar nature of sphincteric muscle explains that arrest. It is well known that in a certain stage of contraction a muscle passes into a refractory period. John Hunter recognised that the peculiarity of a sphincter was that its normal condition was one of contraction. A muscle in a state of contraction must be a muscle with an almost continuous refractory period—a muscle which will serve as a natural block for contraction waves. Dr. Alvarez observed that the duodenum only manifested its inherent fast rhythm when freed from the pylorus; the pyloric sphincter evidently exercises an inhibitory action on the first stage of the duodenum; radiologists are familiar with its open distended state. Its functional condition can be explained on the well-known Bayliss-Starling law; contraction of one segment of the bowel causes inhibition of contraction in the segment just distal to it. The pylorus is the contracting segment, and the first stage of the duodenum the inhibited one.

It is clear that the excitability and contractility of the œsophagus diminish as the stomach is approached; as at the pylorus the peristaltic waves which propel the food downwards do not pass into the musculature of the stomach, but are arrested or inhibited at the cardia.

#### THEORY OF INTESTINAL STASIS.

I have now laid the basis of the theory on which I seek to explain the nature of enterostasis. In passing along the alimentary tract food is propelled through a series of zones or segments, each

furnished with its own pacemaker and its own rhythmical contractions. In the heart we find two such zones—an auricular and a ventricular; in the normal heart the sino-auricular node is the master pacemaker. But a block or imperfection in conduction may occur between the two zones of the heart, with the result that "back-pressure"—a venous stasis—is produced. Now, seeing the similarity between the cardiac and alimentary motor mechanisms, we do not seem over-presumptuous if we suppose that irregularities may occur in the nodal and conducting system of the alimentary canal—irregularities of the same kind as are known to occur in the heart. When such irregularities or blocks do occur we should expect to find them at the points where one rhythmical zone or area passes into the succeeding zone. That is exactly what we do find. We find a block where the œsophagus joins the stomach; we find another where the gastric zone ends and the duodenal begins; we find it where the duodenal zone passes into jejunal and where the jejuno-iliac passes into the ileo-colic. We find a block may occur at any point of passage from a lower to a higher rhythm. At several of these junctional points sphincters are situated and I do not deny that the mechanism of such sphincters may become disordered and cause stasis of the alimentary contents, but it will probably be found that a disturbance in the action of a sphincter is but part of the disturbance which affects the entire rhythmical segment to which it belongs.

Further, it is clear that to obtain an orderly propulsion of the food along the whole length of the alimentary canal those various rhythmical zones must be closely coördinated in their action, and there is a growing body of evidence, both experimental and clinical, that points to a very close coördination by means of a complicated system of reflexes. Disturbance in any one segment upsets the rhythm in all the segments. Bayliss and Starling observed that distension of the duodenum inhibited the action of the ileum; surgeons are familiar with the fact that a duodenal disturbance upsets the rhythm of the stomach. From the facts already mentioned it is easy to see that disturbance in the excitability and rhythm of the pacemaker of the cæcum will be reflected to the lower ileum. One can understand, on the hypothesis I place before you, how stasis in the great bowel may be followed by ileal stasis, duodenal or gastric stasis, or how a disturbance of the conductivity or excitability of any of the rhythmical zone may ultimately give rise to stasis in all.

I should do Sir W. Arbuthnot Lane, who has forced medical men the world over to investigate and attempt to relieve a condition which causes nearly as much suffering as even a great war, less than justice if I expected you to adopt the hypothesis of intestinal stasis I have laid before you in place of his simpler mechanical explanation. I know he will not mind me describing his explanation of enterostasis as the "drag, band, and kink" theory. My hypothesis, you will find, has certain advantages over his. In the first place, I bring into the foreground the musculature of the alimentary tract, which is recognised by all as the sole propelling power in the intestinal wall. In his theory a defect in the musculature of the bowel takes a very minor part in the causation of stasis. Further, my theory is the more in harmony with the appearances observed by clinicians and pathologists, and because it rests on a better basis of anatomical and physiological fact I believe it will ultimately win.