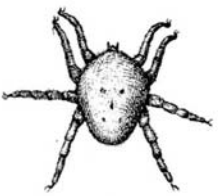
in America, has observed a nympha to abandon its host's ear 35 days after the larva had been introduced, and in other cases the nymphæ remained attached after 98 days had elapsed. Nymphæ have also been observed at Onderstepoort to remain in the ears of their hosts for 3 months. When engorged they leave their host and crawl about in search of some sheltered spot where they can change into adults, which takes place in from 7 to 31 days. They then fertilise, and then the females commence laying eggs, after which they die."



× 3.

When a tick has got into a human ear the patient at once realises that there is something alive in his ear, and comes to a doctor with the diagnosis already made that he has a tick in his ear; and when the ear is examined there is usually little difficulty in seeing either the tick or some of its legs. In one case in a child of five which I saw the whole inner end of the meatus was blocked with the engorged tick, and had I not suspected a tick I might readily have taken the obstructing object for a polypus. Except in this case I have never experienced any difficulty in catching the tick with forceps and removing it entire, and though there was no difficulty in the child's case the tick's body had to be burst before it could be removed.

Beyond the irritation caused and redness and swelling of the tissues little temporary trouble is produced, and apparently no permanent injury. In calves, sheep and goats the irritation seems at times to produce death.

SOCIETIES' PROCEEDINGS.

ROYAL SOCIETY OF MEDICINE.—LARYNGOLOGICAL SECTION.

December 6, 1919.

President: Dr. James Donelan.

(*Continued from p.* 344.)

Discussion on Dilatation of the Œsophagus without Anatomical Stenosis (*continued*).—**A. Brown-Kelly.**—*Treatment*: The preliminary part of the treatment of cardiospasm is carried out by aid of direct inspection. If the œsophagoscope is to be passed for the first time a general anæsthetic should be given, otherwise the nervous strain caused by the examination may aggravate the disease. In introducing the tube more resistance than usual is encountered at or near the mouth of the gullet. This experience, together with the fact that these patients often

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

refer their dysphagia to the cricoid region and not to that of the cardia, indicates that obstruction at the lower end of the gullet may be associated with spasm at the upper end, just as pyloric stenosis sometimes gives rise to reflex spasmodic stricture of the œsophagus.

If the patient is lying on his back with the head slightly lowered, which level is the best position for the examination of the cardiac end of the gullet, a quantity of cloudy grey fluid runs out of the tube as soon as it enters the gullet. More may be drained off by further depressing the head and shoulders. The masses of soft food in the lower parts are then removed and the walls of the dilatation inspected.

The hiatal and subhiatal regions[1] are now examined. These are in the axis of the œsophagus and easily found if the dilatation is spindle-shaped. Most dilatations, however, are flask-shaped, and in advanced cases there may be considerable sagging of the wall to the right and downwards, so that the hiatus comes to be situated on the lower part of the left lateral wall while the fundus of the dilatation is at a lower level. Under these conditions, instruments introduced along the axis of the œsophagus impinge on the floor of the sac, and to reach the epicardia they must be directed well to the left and passed beneath the great bulging produced by the heart. It seems to me that œsophagoscopy in such cases affords a particularly favourable opportunity of studying movements of the heart.

The first point to note for future use in regard to the hiatal gullet is its distance from the upper teeth. In reports of cases of cardio-spasm striking measurements of the gullet are sometimes given, but I am not aware of any statement to the effect that its abnormal length is a constant feature of this disease. The chief conditions affecting the length of the normal gullet are sex, height and age. In the male of medium height the distance from the upper incisors to the cardia is 40 cm. on an average and in the female 38 to 39 cm. The following table shows the sex and height of the patient and the distance to the hiatus—*i. e.* to a level in the gullet 3 to 4 cm. above the cardia. It will be seen that while no individual is above medium height and several below it, the gullet in most is considerably elongated; this lengthening was greatest in the severer cases. The increase in length becomes more striking if to each of these measurements at least 3 cm. be added, which is the distance of the hiatus from the cardia (Table I).

TABLE I.—HIATAL ŒSOPHAGUS.

Case.	Sex.	Age.	Height.	Distance from upper incisors.
1	F.	19	5 ft. 2½ in.	37 cm.
2	F.	60	5 „ 4 „	42 „
3	F.	56	—	—
4	M.	58	5 „ 7 „	48 „
5	M.	33	5 „ 5 „	47 „
6	M.	24	5 „ 7½ „	47 „
7	M.	18	5 „ 4 „	37 „
8	M.	59	5 „ 7 „	43 „
9	M.	41	5 „ 6 „	43 „
10	M.	22	5 „ 5½ „	44 „
Average distance in males				40 cm.
„ „		females		39 „

The aspect at the hiatal œsophagus (or hiatus œsophageus as commonly but wrongly termed) next calls for attention. Some writers maintain that it is normal; others that it is normal, but with the folds of mucous membrane more firmly pressed together so as to close the lumen; others

[1] Dr. Wm. Hill's terminology has been largely adopted in this paper.

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

that it is peculiar to cardiospasm and may differ in slight and severe cases; and lastly, there are several who, in discussing this disease, have omitted to describe the hiatal œsophagus, probably having come to no conclusion as to its typical appearance, or regarding this as not different from the normal.

In most of my cases the lumen of the hiatal œsophagus was surrounded by a stellate arrangement of folds of mucous membrane (Figs. 1 and 2); in others it was V-shaped, or merely a slit with a prominent cushion in front and behind. The size of the lumen was constantly changing, enlarging with inspiration and contracting or closing with expiration. (All the patients had $\frac{1}{100}$ gr. of atropine injected hypodermically before being anæsthetised.)

The appearances and movements just mentioned were noted while the œsophagoscope was about 3 cm. above the hiatal level. From this viewpoint the aspect of the hiatal œsophagus in my cases of cardiospasm was therefore normal. But as soon as the instrument was introduced a little farther and came into contact with the parts around the hiatus, this at once was closed, and remained closed until the tube was withdrawn a few centimetres, when the rhythmical opening and closing of the epicardia recommenced. This sequence of events could be produced over and over



Fig. 1.

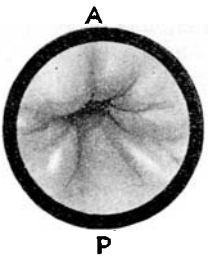


Fig. 2.

again in five of my cases; it was absent in one in which the patient had almost recovered, and was not observed in the others, which were examined before this heightened reflex activity had attracted my attention. In patients suffering from affections other than cardiospasm in whom the sensation has been tested, it has invariably been found that the opening and closing of the epicardia continued in spite of its being touched by the tube, and ceased only when firm pressure was applied to the parts immediately surrounding the orifice; even then the movements occasionally persisted. The number of examinations I have made of patients with and without cardiospasm in reference to this feature is still too small to warrant the statement that it is pathognomonic of cardiospasm.

Various writers mention that in cardiospasm firm closure of the gullet at the level of the hiatus follows any attempt to pass a tube through it, but they do not contrast the normal aspect and movements it presents when untouched with the spasmodic closure it undergoes from slight and distant irritation, nor do they appear to have met with the hyperæsthesia as constantly and over as large an area of the gullet as I have indicated; and certainly none of them, so far as I know, has ascribed the cardiospasm to this hyperæsthesia.

These observations help to reconcile the apparent discrepancies in the descriptions of the hiatal œsophagus in cardiospasm, and show that the varying statements really correspond to the varying conditions of

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

the opening. Further, they carry us a step onward in our knowledge of the nature of the disease by proving the presence of a hyperæsthetic region for some distance above the hiatal level. Irritation of this region is seen to produce contraction or spasm of the œsophageal wall, and not compression such as might be caused by the action of the crura of the diaphragm. Illustrations of the œsophagus at the hiatal level in cardiospasm that have been published also give the impression of contraction and not compression. If these observations and deductions prove correct we may discard the term "cardiospasm" and adopt that of "suprahiatal hyperæsthesia." Both, however, merely mark stages in the progress of our knowledge of the pathogenesis of œsophageal dilatations, as did the other names that have been successively applied—namely, idiopathic, paralytic or atonic, spasmogenic and cardiospastic. We are still ignorant of the primary cause of the disease; whether the next clue is to be sought in an undue irritability of the autonomous nervous system, and this in turn in a disturbance of some internal secretion, or of certain constituents of the blood, must meantime remain in the realm of speculative pathology.

The drugs chiefly recommended for the affection under consideration have been: bromides, valerian, belladonna, atropine and papaverin. I have not used the first two as none of my patients has been of a neurotic type. Belladonna or atropine I have occasionally tried on the assumption that the underlying disturbance was vagotonia, but without obtaining any appreciable improvement. Papaverin is said to be of assistance in slight cases.

The systematic washing out of the dilated gullet with an alkaline solution will be useful if the lining membrane is inflamed or otherwise unhealthy, but I have found it necessary only exceptionally owing to the stretching treatment employed having nearly always been followed by cessation or amelioration of regurgitation. On the other hand, some patients use a stomach-tube frequently in order to evacuate the contents of the dilatation before going to bed, or to open the cardia, or even to introduce food into the stomach if the spasm is specially troublesome.

The food taken should be thoroughly masticated or soft and swallowed slowly. The disease is said to have been caused in some instances by bolting food. The patient should neither eat nor drink anything likely to irritate the wall of the dilatation.

At one time the local treatment consisted chiefly in the passage of sounds and bougies. In slighter cases this may suffice. Thus, two of my patients have been cured for seven and two and a half years respectively by having had metal or ivory olives passed twice or thrice (Tables II, III, and IV).

TABLE II.—E. B——, AGED NINETEEN.

1911.	
June 1 . .	Dysphagia for seven years; subsisted for past year on biscuits, Benger's food, eggs, etc.; regurgitates very little; weight 6 st. 11 lb. 12 oz.
June 28 . .	Passed small olives.
July 5 . .	Passed larger olives.
July 12 . .	Can swallow meat.
August 1 . .	On ordinary diet.
September 27 .	Very stout; being overfed; weight, 9 st. 9¼ lb.
1912.	
October 2 .	Very well; weight, 9 st. 11 lb.
1918.	
September 3 .	Has had no recurrence of dysphagia or regurgitation.

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

TABLE III.—F. T——, AGED SIXTY.

1916.	
June 21	Dysphagia for one and a half years; occasionally complete for four or five days; has lost 1½ st. since onset.
June 30	Food measured (*see* Table IV).
July 5	Olives passed.
July 8	Deglutition improved; no regurgitation; olives passed.
July 12	All food swallowed; no regurgitation.
1918.	
November	Can eat anything; never regurgitates; has gained 3 st. since June 21, 1916.

In most cases, however, the amount of stretching produced by even the largest bougies or olives is insufficient to dispel the cardiospasm. An important contribution to the treatment of this affection was made in 1898, when Dr. J. C. Russel,[1] of Southport, described the condition we have under discussion, and recommended for its cure the stretching of the cardia by means of silk-covered rubber bags of increasing size. Unfortunately this paper was generally overlooked. Plummer, however, refers to it, and gives Russel the credit he deserves.

Five years later v. Mikulicz demonstrated the benefit obtained by overstretching the muscles around the cardia. He accomplished this at first with two fingers, later with specially constructed forceps, introduced through the stomach.

In order to obtain the same effect without gastrostomy Gottstein and Geissler devised an instrument for introduction through the mouth consisting of a tube fitted at the lower end with an hour-glass-shaped distensible bag. When the constricted part of the bag was embraced by the cardia, water or air was injected until the desired amount of dilatation had taken place. The success that attended the use of this instrument led to the construction of various modifications. In some of these the distensible bag has been retained, as in Plummer's apparatus. In others it has been replaced by two or more metal blades, which can be separated to the desired extent by a graduated screw (Abrand, Mosher) or a dynamometric spring in the handle (Lerche, Brünings).

TABLE IV.—F. T——, AGED SIXTY.

June 30, 1916.

Breakfast.		oz.	*Dinner.*		oz.
7 a.m.	Porridge	6	12.30 p.m.	Soup	3
	Milk	4		Chicken and potatoes	2
7.30	Water	4		Jelly and milk	4
				Cup of milk	6
	Total	14		Total	15
	Regurgitated	8		Regurgitated	8
	Retained	6		Retained	7
Tea.			*Supper.*		
3.30 p.m.	Tea	5	7 p.m.	Milk	6
	Bread and butter	1		Water	3
	Total	6		Total	9
	Regurgitated	2		No regurgitation.	
	Retained	4		Retained	9

Totals: Swallowed, 44; regurgitated, 18; retained, 26.

[1] *Brit. Med. Journ.*, 1898, i, p. 1450.

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

Guisez in 1911 had used Gottstein's balloon in fifteen cases and had obtained improvement in all. He advised that the bag be distended until its diameter was 6 to 7 cm. He had found that the capacity of the dilatation diminished rapidly after treatment.

Plummer, who probably has had greater experience than anyone in the treatment of this affection, employs a dilator connected with a water tap and furnished with a gauge indicating the amount of pressure. This is gradually raised at successive treatments until satisfactory results are obtained. In one case unusually high pressure caused rupture of the œsophagus. In 1912 he published his results in ninety-one cases of diffuse dilatation of the œsophagus without anatomic stenosis. Of these, seventy-three were completely relieved of dysphagia, eleven were not completely cured, four had died and three could not be traced. Most of the patients with extreme inanition needed only one treatment to effect a cure. In three patients he found that the dilated œsophagus had returned to the normal size.

Chevalier Jackson considers that it is difficult to place dilating bags accurately by blind methods, and therefore prefers a mechanical divulsor, such as Mosher's, which can be introduced through the œsophagoscope and the action of which can be regulated by the sense of touch. His 53-cm. œsophagoscope is passed into the stomach and afterwards the divulsor through it. Under guidance of the eye the œsophagoscope is withdrawn until the expansile portion of the divulsor in exposed and its greatest diameter placed in the hiatal œsophagus. It is then expanded to about 20 to 25 mm. and kept in for from five to ten minutes. As divulsion is painful he advises ether anæsthesia. He finds from one to six divulsions at intervals of a week necessary.

I have used Gottstein's instrument almost exclusively. The method adopted is as follows: Having obtained during the œsophagoscopic examination the distance from the upper teeth to the hiatus, this is measured from the middle of the bag along the stem of the instrument and marked with a thread or a sliding piece of rubber tubing, which also serves to protect the bougie from being bitten. Before introducing the instrument the contents of the gullet are removed by suction, or, if thick, washed out. Patients unaccustomed to the procedure will at first be cocainised, but they quickly learn to dispense with this. The dilator is then passed. It may meet with slight obstruction at the hiatus, but if gentle pressure be maintained for a few seconds it glides onwards. When the measurements indicate that the bag is in position it is gradually distended by water. In the instrument I use, four to six syringefuls are usually injected before the patient begins to complain of pain, whereupon the injection is stopped. Sometimes the distended bag is left *in situ* for a few minutes, after which the water is allowed to escape and the apparatus withdrawn.

A method, by means of X rays, of determining whether the bag is in position, is to place a small lead square or circle over the site of the stream of bismuth passing from the gullet to the stomach, and afterwards to use a dilator the lower part of which contains a lead rod. The latter is watched crossing the lead window and the distance estimated. It will usually be found to pass considerably to the left in entering the stomach. One or a few dilatings may produce a cure, or improvement lasting for months. In more obstinate cases I have repeated the dilatation at intervals of a few days over a period of several weeks, but the benefit obtained has not been proportionate to the amount of treatment. The dysphagia as a rule is immediately relieved, and the day following

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

the first stretching the patient eats a hearty meal such as he has not enjoyed for months or years. He quickly gains weight and vigour, and may remain well indefinitely or for a variable period (Table V).

TABLE V.—H. M——, AGED FIFTY-EIGHT.

1913.	
May 14	Dysphagia seven years; weight under 10 st.
May 27	Passed thick *stomach-tube.*
June 18	Swallowing soft food; no regurgitation for ten days.
August	Weight, 10 st. 8½ lb.
1914.	
May 3	Dysphagia returned three weeks ago; complete for one week. *Dilator*, two and a half syringefuls.
May 17	*Dilator*, three and a half syringefuls.
May 18	Greatly improved.
1916.	
May 3	Well until fortnight ago; all regurgitated since.
May 15	*Dilator*, six syringefuls.
May 31	Better than for years.
1917.	
February 28	Very well; no regurgitation; weight 11st. 12 lb.
November	Died of pneumonia; took food well till last illness.

TABLE VI.—Mrs. M'A——, AGED SIXTY-TWO

1914.	Total swallowed	Regurgitated.	Retained.
October 25	97 oz.	86 oz.	11 oz.
October 26	36 „	26 „	10 „
	Cardia dilated.		
October 30	89 oz.	16 oz.	73 oz.
„ 31	108 „	17 „	91 „
November 1	113 „	11 „	102 „
November 2	105 oz.	22 oz.	83 oz.

The regurgitation also ceases, or is distinctly diminished (Table VI). Sometimes, however, there is retention without regurgitation, and the presence of food and fluid in the dilatation may escape notice unless looked for. The contents of the œsophagus may be removed by the Senoran's suction apparatus; and the quantity obtained from time to time gives an indication of the progress towards recovery. Another method of determining the presence and amount of retained fluid is to cause the patient to swallow a capsule three-quarters full of bismuth which floats. In one of my patients, from whose gullet about 300 c.c. of fluid could usually be withdrawn, the capsule could be seen about the level of the episternal notch moving up and down with respiration. Although the amount of retained fluid in this case was so considerable, the patient suffered but little inconvenience from it and often was unaware of its presence.

There appears to be no doubt that the dilated œsophagus occasionally returns to its normal size; several authorities have made observations bearing on this point.

I shall say nothing about gastrostomy excepting to recommend it for patients who have been reduced to a moribund state by prolonged fasting, and in whom an examination with the œsophagoscope and dilatation treatment would meantime be contra-indicated.

The only death I have had directly due to cardiospasm was that of a woman, aged fifty-six, who had suffered from dysphagia for twenty years. Four weeks before death the obstruction at the cardia had suddenly become almost complete. She was first examined by me five days before her death, when, on introducing the œsophagoscope, a large

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

quantity of bismuth in suspension was drained off: this had been swallowed six days previously for X-ray purposes. A tube was passed into the stomach under direct observation, and left *in situ*. After having been fed through this for two days she withdrew it. She was then able to swallow a little fluid naturally. The same evening she died of heart failure. At the *post-mortem* examination the gullet was found to be greatly dilated. There was no anatomic stenosis at the lower end and no sign of malignancy anywhere (fig. 2, p. 365).

S. G. SHATTOCK: At times the œsophagus will pass entirely through the right crus, and it is conceivable that under such circumstances an anatomical variation might lead to obstruction. In one such case which I have seen, however, dissection showed that the œsophagus above this abnormal entry was not dilated.

With regard to spasm: There have been put as alternatives that spasm may be one cause of this obstruction, and that the other may be inco-ordinated action, that is to say, a want of co-ordination between the descent of the contents of the œsophagus and the dilatation of the cardiac orifice. If we accept the observations of Dr. Brown-Kelly—and I think they are most important—we have a very simple and satisfactory solution of the cause of the obstruction.

I have long held myself that a hyperæsthetic condition of the mucosa furnishes the most satisfactory explanation of hypertrophic stenosis of the pyloric canal; so that the mere passage of food over the membrane excites a spasm in the muscle, which may completely prevent the passage of food. That, therefore, is a theory congruous with Dr. Brown-Kelly's observations upon the œsophagus. There is one difference, anatomically, in the case of the pylorus: there is an overgrowth of muscular tissue at the site of obstruction. That we do not see in œsophagectasia. In an excellent preparation at St. Thomas's Hospital, *e. g.* in which a man, after thirteen years' obstruction, died of inanition, there is no muscular thickening whatever at the cardia. This may, therefore, in the minds of some, still make for the theory of neuro-muscular inco-ordination; for the closure beyond the area of excitation *might* have been due, not to an active spasm, but to a failure of the co-ordinated dilatation.

When we cast about for somewhat analogous conditions, one of the nearest perhaps is that of the urinary bladder. There are certain cases of idiopathic dilatation of the urinary bladder, male and female, in which one can find no trace of urethral obstruction. I have examined some such, and they have been described also by others, and what is more, they occur in the fœtus. They might be thought to be cases of infection and of spasm incited in the urethral muscle by the passage of urine over an inflamed mucosa, but the fact that the conditions may occur before birth will exclude such a view. There is one example in Guy's Hospital Museum, another in the College of Surgeons, of female children whose bladders at birth were greatly distended without discoverable cause of obstruction. That some such are due to neuro-muscular inco-ordination may be guessed from the phenomenon of what Paget called "stammering" bladders, where, after the escape of a few drops of urine, a complete block will ensue, which may last long enough to lead to painful distension. In reflecting upon this subject[1] I considered the possibility of there being not a *hyper*æsthetic but a *hypo*æsthetic state of the vesical mucosa (pharyngeal hypoæsthesia or complete anæsthesia is

[1] *Proc. Roy. Soc. Med.*, 1908-9, ii (Sect. Path.), p. 98.

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

well known in hysteria). The view seemed to me, moreover, applicable as an explanation of megacolon, where the colon slowly dilates without any trace of organic obstruction, and this commencing in the pelvic portion where there is no anatomical sphincter. In connection with this subject I devised the following experiment: Into the bladder of a female cat a little cocaine solution was injected through a fine catheter. After a few minutes the bladder could be indefinitely distended with salt solution, none of which escaped by the side of the instrument, as it did before the local anæsthetic was used. Obviously, one of two things had happened: either the mucosa had been rendered anæsthetic and had failed to furnish the proper reflex for the excitation of the detrusor urinæ, the sphincter remaining co-ordinately contracted, or the cocaine had reached the muscular tissue and produced motor paralysis. And this leads me to conclude by suggesting that one way of discovering whether we are dealing with an extraneous factor like the constriction, actual or virtual, of the diaphragm, or with the intrinsic factor of proper spasm or achalasia, might consist in the use of this local anæsthetic. I am sure the skill of laryngologists would enable them to cocainise the cardiac orifice or the cardiac end of the œsophagus. You will either anæsthetise the mucosa, or, better still, paralyse the musculature of the cardiac orifice. And then, if it were found that the obstruction was removed, Dr. Hill's view that all these cases are due to phrenic conditions would, I think, be negatived.

I may add this further criticism: that if the obstruction is really where Dr. Hill suggests it is, a study of the preparations on the table will show that the dilatation later on proceeds down to the actual orifice of the stomach; on his view this should not be so. For let us assume that the dilatation started at the diaphragm; as it proceeded it would overcome the obstruction, and on reaching the cardia the obstruction should be removed. But that is not the case. The dilatation, in these preparations, ends only at the gastric orifice, and we must conclude, I think, that we are dealing either with spasm of the cardia or with a form of obstruction due to inco-ordination.

The PRESIDENT: Before Dr. Brown-Kelly replies, will Dr. Hill say what is the value of the researches which have been made on the innervation of the œsophagus? Seven or eight years ago I heard Dr. Guisez deal with this subject at the Hôtel Dieu, and he mentioned that the dilatation of the cardiac end of the œsophagus was due to the special dilator branch of the vagus. On referring to Guisez's book I find that it was Oppenchowshi, of Dorpat, who demonstrated the special dilator nerves of the cardiac end, and it was he also who showed the action in constricting the opening of the branches of the sympathetic through the great splanchnic. That seems to be the mechanism which is irritated and which produces the closure of the orifice in these cases. In Mr. Harmer's case we saw that the presence of a very small foreign body in the œsophagus will excite spasm quite out of proportion to the size of that body. In this discussion there has been no allusion to the superior constrictor of the œsophagus—I mean the circular fibres around its upper end, and their share in the mechanism—which is only second in importance to the closure of the lower end in these dilatations. Nothing was said as to the mode of opening of the mouth of the œsophagus, except that it was passive to boluses of food, whereas it has now been demonstrated that an active dilatation takes place in anticipation of the bolus through the action of dilator filaments.

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

W. Hill (in reply): Mr. Shattock, Mr. Howarth and others have failed to grasp my meaning as to what takes place at the hiatus of the diaphragm during the last stage of the deglutitory act. In the same way that the opening up of the upper orifice of the gullet is dependent, not on any intrinsic œsophageal action, but on the pulling away of the larynx and cricoid plate from the spine by the hyoid and tongue muscles, so the opening up of the lower region of the gullet—the phreno-cardiac portion—is not mainly brought about by the intrinsic œsophageal musculature, but is mainly actuated by the musculature of the crura forming the margins of the hiatus. I draw on the blackboard an inferior view of the hiatal region at rest when it appears as a long lanceolate slit enclosing the collapsed laterally compressed gullet. I maintain that whether or not there are inhibitory nerves which bring about dilatation of the cardia, the intrinsic gullet musculature is powerless to make the margins of the hiatus in the diaphragm assume a circular or elliptical form, which is a necessary antecedent to the expansion or patency of the previously collapsed and compressed gullet. If this phrenic mechanism is at fault it would explain functional stenosis at the hiatal level. There is no proof that hypertonic contraction or spasm occurs in these cases, for the endoscopic appearances are normal. As regards the drawings of Starck and Guisez, their pictures when they are not within the limits of normality are purely fanciful. Achalasia may possibly be a factor in functional stenosis at the hiatal level, but in my view the phrenic factor is necessarily of far greater importance, and has been too long ignored.

S. G. Shattock (in further comment): One can imagine—indeed one is disposed to believe—that those phrenic fibres which surround the opening are inhibited in their contraction as the œsophageal contents pass. It is so in the case of the urethra. When the bladder contracts to empty itself the compressor urethræ relaxes, but the urethra does not expand, as Dr. Hill would suppose in the case of the œsophagus; it merely relaxes. But given such an inhibition, there is no reason why the aperture should be made round instead of remaining as an inert slit.

A. Brown-Kelly (in reply): I have endeavoured to deal with the subject from a practical standpoint. In 1912, at Liverpool, I discussed the various theories that had been brought forward with regard to the ætiology of this affection. The opinions I have expressed to-day are founded on observation, and it remains for you to put them to the test.

The Pathology of Œsophagectasia (Dilatation of the Œsophagus without Anatomic Stenosis at the Cardiac Orifice) was illustrated by a unique series of specimens collected by **Irwin Moore.**

Remarks by Prof. S. G. Shattock: The following comments, which have received the approval of the two openers of the discussion on œsophagectasia, have been drawn up with the object of indicating what conclusions are suggested from a study of the specimens exhibited at the meeting.

The Thickening of the Muscular Coat.

As shown in the specimens, this varies in degree in different cases; and even in the same specimen, in different zones, or at different levels of the dilatation. There is none in which the muscular wall is atrophic —*i. e.* absolutely thinner than the wall of the normal œsophagus. In some the amount of muscular thickening is not merely proportional to

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

the increased capacity of the tube, but is absolutely above the normal. The overgrowth of muscle is to be viewed as of a secondary or compensatory kind, brought about by the obstruction below. In all, therefore, a paralytic condition of the tube may be excluded as an explanation of the dilatation; although, of course, such an ætiology might obtain from disease, either peripheral or central, of its vagal supply; for it may be so produced experimentally. That an atrophic condition, however, might be merely a secondary phenomenon is not to be ignored in this connection. As long as the blood supply of the dilating tube remains good, the muscular overgrowth would be main tained; when this fails, as from disease of the supplying arteries immediate or remote, the dilatation would proceed with atrophy. These results would be strictly comparable with those which occur in the muscular wall of the bladder in obstruction due to enlargement of the prostate.

Comparison between the Normal Pyloric and Cardiac Canals.

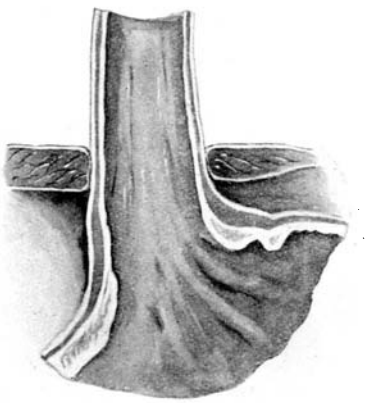


Fig. 3 a.—Normal cardia from a well-developed young male adult (the same subject as Fig. 3 c). The crura of the diaphragm are shown in their position; they lie immediately above the sphincter.

One of the interesting things arising out of the examination of the specimens is, that in one there is a well-defined and pronounced hypertrophy around the terminal part of the œsophagus. The overgrowth is confined to the circular fibres.

[On the ground that there is evident hypertrophy of the phrenocardiac portion, Dr. Hill would not include this as a case of functional stenosis without anatomic lesion.]

The only other specimen suggesting an overgrowth of the cardiac sphincter is one from the London Hospital, figured by Sir Hugh Rigby in Choyce's "System of Surgery." The figure there given is somewhat misleading, however, in that the parts have not been straightened out to show their proper relations. When this is done, it is seen that the thickened circular muscle lies almost entirely above the actual constriction, and in the wall of the lowest part of the *dilated* œsophagus. In this specimen, the thickening of the circular muscle, if regarded as a sphincter hypertrophied in thickness and in length, is now incorporated in the dilated termination of the œsophagus. This apparent anomaly can only

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

be explained by supposing that the chief part of the sphincter, which may at one time have closed the cardiac orifice or canal, has slowly given way under the distending force from above. It is therefore remarkable that the musculature of the terminal portion of the canal below has sufficed to maintain the obstruction.

[It may be observed, in passing, that the sphincter vesicæ in the cat's bladder, although insignificant in size, is sufficient to prevent the distended bladder being emptied by firm pressure made with the hand,

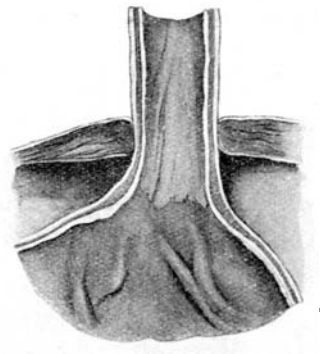


FIG. 3 B.—Normal cardia from a well-developed female of middle age. The white œsophageal epithelium is sharply demarcated from the gastric; the sphincter embraces portions of both areas.

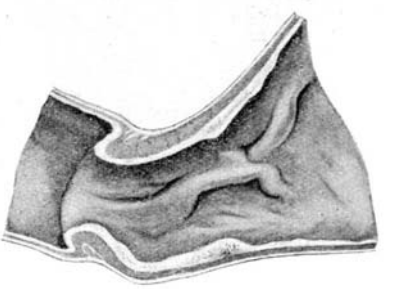


FIG. 3 C.—Normal pyloric canal from a well-developed male adult.

Natural-sized drawings from specimens specially dissected and prepared by Prof. S. G. Shattock, F.R.S.

when the extrinsic action of the urethral muscle in front is excluded by the passage of a catheter through the urethra but not actually into the bladder.]

As the exact position and dimensions of the cardiac sphincter are matters of importance in connection with the subject of œsophagectasia, two anatomical specimens have been specially prepared, male and female, and accurately drawn in order to elucidate the subject. Fig. 3 A (p. 373) is from a well-developed young adult male, who died after an operation for cerebellar tumour. The parts were carefully dissected after removal; a narrow cylinder of wet cotton-wool was lightly drawn through the lower part of the œsophagus from the stomach; and the specimen, laid on cotton-wool, was hardened in formol solution, and afterwards bisected

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

in the coronal plane. Fig. 3 B is a similarly prepared specimen from a well-developed female of middle age, who died suddenly from pulmonary embolism after hysterectomy.

One thing noticeable in making the preparations was the ease with which the cardia could be displaced through the opening between the crura, the connection of the structures concerned being of the most delicate kind. Attention may be drawn to the thickness of the crura as well as to their close apposition to the lower end of the œsophagus, the relative positions of the different structures being carefully preserved. A study of the drawings will show that the thickening of the circular

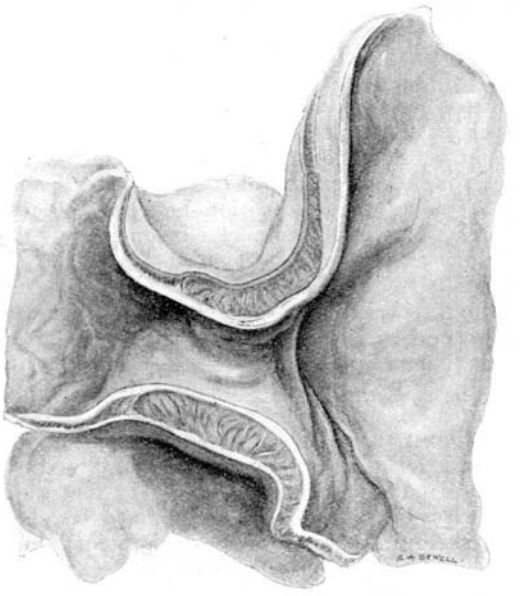


FIG. 4.—Hypertrophic stenosis of the pyloric canal, from an adult. (In the Museum of St. Thomas's Hospital.) Natural size.

fibres forming the sphincter embraces part of the stomach as well as the end of the œsophagus, and that it lies quite below the diaphragm. In Fig. 3 B the dividing line between the œsophagus and the stomach is sharply brought out by the greater opacity of the thicker, squamous-celled epithelium of the former. The sphincter, moreover, is not a simple ring, but a fusiform thickening of sufficient length to justify the use of such a term as the cardiac canal rather than that of cardiac orifice. If compared with the pyloric sphincter (Fig. 3 c), the latter has an abrupt termination on the distal aspect, where it produces an annular elevation within the lumen; but on the proximal side the thickening of the circular muscle tails off around the pyloric canal, much as in the case of the cardiac sphincter.

If overgrowth of unstriped muscle be taken as an indication of abnormally forcible contraction, the dilatation in the case from University College referred to may be ascribed to cardiospasm [in the sense,

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

Dr. Hill remarks, in which Mikulicz employed the term—viz. hypertrophic stenosis following on a primary functional spasm].

The hypertrophy in this case is remarkably like that met with in hypertrophic stenosis of the pyloric canal as seen in infants; and more so when this stenosis is found in the adult. Of the last there are two examples in St. Thomas's Hospital Museum. In both the mucosa is intact; the muscular thickening is confined to the circular fibres; and in one, certainly, the stomach has been dilated from pyloric obstruction. Of No. 943F a drawing has been introduced (Fig. 4). It came from a man, aged sixty-one, admitted for mitral stenosis; he had had malaria, and there was a history of rheumatic fever; nothing in regard to gastric trouble is mentioned in the notes. In the case of the University College specimen, the patient was fifty-five years of age; symptoms had existed for eighteen years. In Sir Hugh Rigby's case the patient was forty-two, and symptoms had been present "for many years." Whether hypertrophic stenosis of the pyloric canal may not arise at a later date than usually assumed is a matter needing further observation; if so, the two conditions would be still more parallel. No similar overgrowth of the cardiac sphincter is present in any of the other specimens. In explanation of these, a want of co-ordination between the propulsive action above and the relaxation of the sphincter below may be the more correct explanation, and it is this which the term "achalasia" is designed to connote; for, clinically, there is no palpable hindrance to the passage of the bougie through the cardia. When the two phenomena are compared, there is, perhaps, no very great difference between them. In the second case the obstruction is due to the *tonic* contraction of the cardiac sphincter; and in the first, the contraction is augmented, or *hypertonic*—*i. e.* it is a proper cardiospasm. Nevertheless it conduces to accuracy to draw the distinction if there are grounds for making it.

Next, in regard to the part played by the diaphragm. Whatever can be distinctly conceived is possible, and it may be that the neuromuscular fault in certain cases lies in the diaphragm and that the obstruction is extrinsic. The skiagrams undoubtedly demonstrate the presence of an undilated segment of the œsophagus above the stomach, the upper limit of which segment corresponds with the superior surface of the diaphragm. This, it may be submitted, however, does not prove that the diaphragm is the *cause* of the obstruction. The obstruction, one may still think, is below at the cardia, the tone of the diaphragm disallowing the dilatation of the included part of the tube. When, in the course of time, the increasing pressure of the accumulation above overcomes this extrinsic support, then, as shown by the skiagrams and specimens, the dilatation involves the whole of the canal as far as the sphincter, which nevertheless remains capable of holding up the œsophageal contents. The term "phreno-cardiac segment," as used by Dr. William Hill, is very appropriate as applied anatomically in the present discussion. That an inhibitory relaxation of the diaphragm immediately around the œsophagus accompanies the dilatation of the cardiac sphincter is not improbable, judging by analogy from what occurs in other positions; and it may prove that failure in the second is accompanied with failure in the first, although, *per se*, the tone of the diaphragm would be ineffective as a cause of obstruction.

That a reflex may arise in the stomach, which will prevent the entry of ingesta, may be taken as established, and is illustrated by such a case as the following: A patient was in St. Thomas's Hospital suffering from

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press

a gastric ulcer. Following a bismuth meal a skiagram showed that after much had passed into the stomach, the material was held up in the œsophagus, which became dilated, the dilatation terminating inferiorly immediately above the diaphragm. Here the achalasia arose as a reflex transmitted from the stomach. There is no evidence, however, that gastric conditions, either organic or functional, are necessary ætiological factors in producing neuro-muscular œsophagectasia. On the contrary, the direct observation made by Dr. Brown-Kelly proves that the reflex fault may lie in the œsophagus, since the experimental irritation of the mucosa above the cardia, in place of being followed by relaxation of the sphincter, as it is normally, was followed by its closure.

Lastly, amongst the drawings there is inserted one showing a distinct and wide-spread muscular hypertrophy, especially of the circular layer, but unaccompanied with dilatation; the heart was much dilated, and weighed 18½ oz. A similar condition is shown in a second specimen from Guy's Hospital Museum, described by Dr. Newton Pitt.[1] Here the heart was likewise enlarged. The muscular thickening of the œsophagus was most marked an inch above the cardiac orifice. Such results have been attributed to obstruction caused by the pressure of the enlarged heart. Aortic aneurysm may undoubtedly produce œsophageal obstruction and some local dilatation of the canal above.

In connection with muscular hypertrophy of the œsophagus without dilatation, it is enough to point out that it *may* represent a completely compensated achalasia, or even cardiospasm—a possibility already conceived by Dr. Brown-Kelly.[2] This could only be established by finding such hypertrophy unassociated with aortic aneurysm, enlargement of the heart, or other extrinsic organic obstruction.

ABSTRACTS.

Abstracts Editor—W. Douglas Harmer, 9, Park Crescent, London, W. 1.

Authors of Original Communications on Oto-laryngology in other Journals are invited to send a copy, or two reprints, to the Journal of Laryngology. *If they are willing, at the same time, to submit their own abstract (in English, French, Italian or German) it will be welcomed.*

PHARYNX.

Researches on the Pharyngeal Reflex.—A. Croce. "Arch. Ital. di Otol.," xxx, No. 4.

The author examined a large number of people with a view to determining the constancy or otherwise of the pharyngeal reflex. The sensitiveness of the pharynx was supposed to be diminished in the case of hysteria. Croce examined 64 cases of hysteria, 24 of aphonia, and 10 mutes. In none of these was there any anæsthesia of the pharynx and the normal reflex was present in all. Eighty-eight healthy persons were also examined and the reflex found present in all. The reflex is weak in thyroid hypertrophy and in old age. Diseases such as tabes cause abolition of the reflex. *J. K. Milne Dickie.*

[1] *Trans. Path. Soc.*, 1888, xxxix, p. 107.
[2] *Brit. Med. Journ.*, 1912, ii, p. 1047.

https://doi.org/10.1017/S0022215100021459 Published online by Cambridge University Press