cutaneous level. Here the two threads are firmly tied together and cut short, the knot being buried. Finally, the edges of the tiny incision are brought together with a single suture of horse-hair and sealed with collodion. The resulting scar is almost invisible, and yet the end of the muscle slip is quite firmly secured.

Muscle transplantation is still on trial. (See also JOURN. OF LARYNGOL., RHINOL., AND OTOL., May, 1920, p. 155.)

THE END.

SOCIETIES' PROCEEDINGS.

ROYAL SOCIETY OF MEDICINE.—LARYNGOLOGICAL SECTION.

December 6, 1919.

President: Dr. JAMES DONELAN.

Discussion on Dilatation of the Œsophagus without Anatomical Stenosis.[1]—**William Hill.**—Diffuse dilatation of the œsophagus without anatomic organic narrowing in the region of the cardia is usually in its early stages, at all events of the spindle-shaped type, though later lateral lobulations may develop, especially near the phrenic level, where there is sometimes an abrupt obtuse termination of the dilatation. The ectasia is in most cases limited to the thoracic gullet, though in very advanced ones the cervical œsophagus, the pharyngo-œsophageal orifice, and even the lower pharynx are much enlarged. In a few observed cases some elongation of the gullet has been demonstrated in addition to great increase in calibre, the viscus then taking a sigmoid course in the chest. There is stasis of ingesta due to its passage being distinctly impeded at the level of the diaphragm, but when the dilated gullet in these cases is removed and examined *post-mortem* the lumen of the phreno-cardiac segment is found to be not subnormal, and in certain and rare cases, to be alluded to later, it may even here be actually enlarged. We have to do generally, therefore, with a normal potential lumen of the gullet at the phrenic level and immediately below it, in combination with a functional stenosis and arrest of food at the phrenic level and ectasia above.

In some instances the cause of this stenosis is obvious—*e. g.* in cases of either hernia or of eventration of the diaphragm in which the stomach assumes a thoracic position considerably higher up than normal under the left ribs, thus producing angulation or kinking of the subphrenic œsophagus.

Dilatation of the whole of the gullet, including the phreno-cardiac portion, has exceptionally been observed in cases of hour-glass constriction of the stomach and in marked pyloric stenosis of long standing. This latter group, like the previous group—viz. that due to upward displace-

[1] We regret that lack of space prevents the insertion of any more than a portion of this interesting discussion.—ED., JOURN. OF LARYNGOL., RHINOL., AND OTOL.

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ment of the stomach—can be differentiated by the aid of the X rays from the ordinary type of dilated thoracic œsophagus, in which there is neither external pressure nor angulation nor intrinsic anatomic narrowing of the lumen below, and in which the inability of the food to pass freely through a potentially normal lumen appears to justify the term "functional stenosis." The latter condition presents a constant X-ray picture as the bismuth meal in well-marked cases of œsophagectasia is seen arrested at the phrenic level, not passing immediately into the stomach. The same picture is, however, observed in fibrous stricture and other forms of true anatomic stenosis of the phreno-cardiac gullet, and the differential diagnosis can only be certainly made by endo-œsophageal inspection of the phrene-cardiac region by means of large-sized endoscopic tubes. I employ an œsophagoscope 18 mm. in diameter for choice, and if such tubes can be passed into the stomach there is no anatomic stricture. Blind bougieing has been much relied on in the past in order to ascertain the non-anatomic character of the stenosis; and when a large bougie passes readily into the stomach it is strong presumptive evidence in favour. But a sense of stricture due to spasmodic gripping of the bougie behind the cricoid sometimes leads to error. When a soft rubber hollow bougie of large calibre filled with mercury is seen with the aid of the X-ray screen to pass without impediment into the stomach this test can be relied on as pointing to the absence of an organic stricture; but if the gullet presents a truncated and lobulated dilatation at the phrenic level the nose of the bougie may fail to hit off the entrance to the phreno-cardiac gullet; the test then fails to differentiate between a functional and an anatomic organic stricture in this region, so that the superiority of the œsophagoscopic method as a certain method of diagnosis and applicable to all cases is evident.

Most physicians, surgeons and radiologists, even at the present day, speak of the site of the stenosis being at the cardia; we, however, of course know that the X-ray findings show that the arrest of the bismuth is at the level of the diaphragm. Chevalier Jackson was, I think, the first to call attention to the correct level, and he discarded Mikulicz's term "cardiospasm" as applied to these cases, and substituted that of "phrenospasm" in the first edition of his book. Cardiospasm is a term that was, and still is, employed in reference to two types of obstruction near the lower end of the gullet, viz. (1) to cases of hypertrophic anatomic—*i. e.* organic—stenosis with ectasia of the thoracic gullet above, which are supposed to have originated in pure hypertonic spasm of the cardiac circular fibres and with subsequent hypertrophic muscular narrowing of the lumen; and (2) to cases of spasm of these fibres without subsequent hypertrophic anatomic stenosis. Whether the first sequence actually occurs is now being questioned in some quarters, but many hold not only that it does occur, but that long-standing cardiospasm is *always* followed by hypertrophy locally and ectasia above, and that the absence of hypertrophy in functional non-anatomic stenosis necessarily excludes true cardiospasm. Some—*e. g.* Brown-Kelly—however, who up to recently employed the term "cardiospasm," were careful to explain that they only meant a state of non-relaxation of the circular fibres of the cardia which should normally occur as a part of the neuro-muscular co-ordinate act of swallowing. Hurst, believing that a true spasm leads to hypertrophic stenosis, therefore substituted the term cardiac "achalasia" for that of "cardiospasm" where only functional stenosis was inferred from the free passage of a large mercury-

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filled tube. This terminology is more acceptable to many than the old one of cardiospasm, and it may perhaps be that this explanation of want of relaxation is a correct one of what occurs in some few cases, though personally I have not satisfied myself that the stenosis has been at the cardiac level in any of my patients. The term "cardiospasm" I have discarded for the same two reasons, and in addition it may be pointed out that the special mark of a hypertonic spasm in the alimentary canal is painful cramp or colic, which as a primary phenomenon is not evident in my experience, though frequent enough as a secondary symptom when food is impacted or arrested.

Holding that there is no convincing evidence that either cardiospasm or cardiac achalasia ever exist as primary phenomena, and seeing that the level of stenosis is not at the cardia but at the level of the hiatus œsophageus of the diaphragm (where the gullet is embraced by the crura, which constitute a potential extra-œsophageal sphincter), I regard these terms as likely in course of time to become obsolete. In reference to the phrenospasm hypothesis of Jackson, it may be said that as a secondary symptom we are all familiar with it, but if Jackson's former views had been correct then we should have had to assume in the majority of these functional cases a primary spasm of the crural fibres without the usual mark of diaphragmatic spasm, viz. hiccough. I do not know if Jackson has advanced matters very much in his new edition by throwing over the term "phrenospasm" and substituting that of "hiatal œsophagismus," meaning apparently thereby both an accentuation or spasm of the normal tonic contraction of the crura surrounding the gullet at the hiatus œsophageus of the diaphragm, and also of the circular fibres of the gullet itself at that level. As a mercury-filled tube in these cases passes readily through the gullet at the hiatal level there can certainly be no powerful hypertonic spasm of the crural fibres or of the circular fibres of the gullet itself, and no very obvious evidence of any distinct obstructive tonic contraction which is assumed by him to occur here.

All these hypotheses, be it noted, proceed on the assumption that general ectasia of the thoracic gullet is secondary to primary obstruction of one or other portions of the phreno-cardiac segment of the œsophagus brought about by muscular action or overaction.

I tentatively suggested some years ago that there might possibly be a block or impediment to the free passage of food due to an opposite cause—viz. neuro-muscular paresis and absence of co-ordinate *active* opening up of the phreno-cardiac gullet during the act of swallowing. At rest the gullet is collapsed, not open at the hiatal level, being embraced and possibly gripped by the crura; at the same time the hole for the passage of the gullet through the diaphragm (hiatus œsophageus) is a narrow lanceolate slit; during the act of swallowing the hiatus enlarges and its margin assumes a circular form, and a wider cross-section brought about by phrenic and possibly sympathetic nerve action on the muscular crura; and instead of a collapsed œsophageal lumen at the hiatal level the gullet here follows the expanded hiatus, and thus we have an open tube. The exact sequence of events and the factors involved in this neuro-muscular effect, resulting in the active opening up of the phreno-cardiac gullet during the act of swallowing, has not been satisfactorily elucidated, but paretic abeyance of this normal act of active opening up of the lumen by muscular action would necessarily lead to a less free passage of food. The difficulty in the way of securing acceptance for an alleged strictly localised paresis in this circum-hiatal

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neuro-muscular mechanism is further increased by postulating that the derangement producing such chronic and far-reaching and gross results is primarily of a purely functional character, seeing that it occurs equally in males and females and not especially in neurotic subjects. On the other hand, there is no evidence of a primary organic paralysis of the phrenic nerves which would account for such interference with the co-ordinate act of swallowing at this level, such as would tend to stenosis and secondary dilatation of the gullet above. If a localised neuro-muscular "block" really occurs at this level, however loth I am to fall back on the functional neurosis explanation, I can find no other likely pathogenesis at hand. I regard the neurosis as extra-œsophageal rather than instrinsic—*i. e.* it is not in the actual œsophageal musculature, as has been generally assumed by others, but probably referable to the crural fibres surrounding the hiatus.

Mr. Shattock, in a discussion on a paper by Batty Shaw and Woo,[1] states that he is led to the hypothesis that œsophagectasia "is due to an inco-ordination of the nervous impulses transmitted by the vagus during deglutition, which impulses should normally cause contraction of the tube above and an active dilatation of the cardia." This explanation nearly corresponds with that of H. D. Rolleston and of Hurst. Whilst agreeing that the essential primary cause in these cases is of the nature of neuro-muscular inco-ordination, I regard the enlargement by crural action of the hiatus in the diaphragm (for the œsophagus) as of much more importance than mere relaxation of the cardia. If the peccant mechanism is really in the diaphragm, then Mr. Shattock is wrong in asserting that dilatation of the œsophagus is idiopathic and due to sympathetic and vagal vagaries; it would largely be secondary to an extra-œsophageal phrenic factor in the complex co-ordinate act of deglutition.

The older authorities, even up to and including Zenker and Morell Mackenzie, felt no difficulty in regarding dilatation of the thoracic œsophagus without anatomic stenosis below as a primary idiopathic condition, and due to a sort of myasthenia or diminished contractile power of the musculature; as, however, hypertrophy rather than muscular atrophy is a fairly constant feature except at the site of greatest dilatation, where there may be partial atrophic thinning of the muscular coats, this hypothesis was generally abandoned even before the introduction of X rays demonstrated that fairly strong, though ineffectual because not water-tight, peristaltic contractions occurred when bismuth paste was swallowed. This shows that the circular fibres remain active, so primary atony is apparently ruled out. It is interesting to note that Zenker and von Ziemssen, in their well-known contribution on "Diseases of the Œsophagus," not only regard the ectasia as primary in cases where there is no anatomic stenosis immediately below, but they do not suggest any purely functional stenosis below as entering into the pathogenesis; spasm they describe as a separate morbid entity, but state that it does *not* lead to subsequent ectasia. They state that in addition to stagnation ectasiæ above an anatomic organic stricture there are also "rare cases with considerable and occasionally enormous ectasiæ in which there is no underlying stenosis whatever," thus apparently excluding functional stenosis not only of a spasmodic, but also of a paretic nature. The circular fibres are supposed to be innervated by the sympathetic, and it is curious to note that so far back as 1895 H. D. Rolleston suggested that

[1] *Proc. Roy. Soc. Med.*, 1916–17, vol. x (Sect. Med.), pp. 16, 17.

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the paresis primarily affected only the longitudinal fibres which are supplied by the vagus, and that this vagal paralysis "would allow dilatation of the tube to occur, and at the same time by interfering with the opening of the cardiac sphincter would induce hypertrophy of the circular muscular coat."[1] How paralysis of the longitudinal fibres innervated by the vagus exactly interferes with the opening of the lower end of the gullet, and alternately what part, if any, the longitudinal fibres play in the active opening up of the phreno-cardiac œsophagus, Rolleston does not explain, but I have long thought that there is nothing inherently improbable in the suggestion that the longitudinal fibres of the gullet and the longitudinal and oblique fibres of the stomach possibly play some accessory part in the opening-up during deglutition of the, at other times, closed phreno-cardiac gullet.

Jackson has pointed out that with a large, dilated atonic stomach filled with gas there is sometimes dysphagia, which is at once relieved when the patient has got rid of the flatus by eructation; and I have observed that in cases with phreno-cardiac stenosis, whether organic or functional, some patients testify that they can swallow better after they have succeeded in relieving the wind in the stomach by belching. These facts have led to the suggestion that affections of the stomach other than those noted earlier in my remarks may, by producing angulation of the subphrenic gullet, be the primary cause in some cases of œsophagectasia without intrinsic anatomic stenosis. In several of my own cases there has been not only a history of chronic gastric disorders, but in some marked gastroptosis has been seen by the X rays. But Shattock states that there is no *post-mortem* evidence of angulation of the subphrenic gullet in these cases, and I may add that, though I have looked for it, I have never seen such angulation by the X rays; the bismuth passes either in a straight or else in a slightly curved stream, not in the least approaching a kink. Whilst hesitating, then, to accept the hypothesis that gastric disorders (especially where there is dilatation and fermentation) are a primary cause of functional stenosis of the lower gullet, followed by ectasia above, I admit that these disorders aggravate the condition when established, and that treatment of the stomach, especially by lavage, relieves to some extent the dysphagia and is an important part of the treatment.

Dr. Brown Kelly will deal more especially with the question of treatment; but successful methods of treatment sometimes give clues which help to clear up obscure points in ætiology, and I must make some remarks from that point of view. As functional stenosis has been held to be due to paresis, it might be expected that the therapeutic application of electricity would supply some corroborative information. This is not so. The faradic current only acts on striped muscle, which does not extend much beyond the upper third of the gullet. The use of the ordinary constant galvanic current is contra-indicated in a moist tube like the gullet on account of the danger of electrolytic action. The sinusoidal current is safe, but in the only case in which I tried it it failed. It is generally held that the alleged functional stenosis at or near to the lower end of the gullet with ectasia above is best and more or less successfully treated by the same methods of dilatation of the phreno-cardiac portion as are appropriate to unequivocal organic strictures. Personally I have not obtained the uniformly gratifying results recorded by others by bougieing and other dilatation methods

[1] *Trans. Path. Soc. Lond.*, 1896, vol. xlvii, p. 39.

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in cases where the diagnosis of absence of anatomic stenosis was unequivocal—*i. e.* where a mercury-filled tube easily passed into the stomach, clearly demonstrating that the lumen of the phreno-cardiac gullet was not merely potentially but actually normal. This test appears to me not only to demonstrate a normal lumen, but to exclude intrinsic spasm and probably also achalasia of the circular fibres of the gullet, both at the hiato-phrenic and cardiac levels, and also spasm and achalasia of the crural fibres (at the hiatal level of the diaphragm). The effect of dilatation by large bougies, hydrostatic and pneumatic bags, and by the passage of endoscopic tubes of large calibre, is of course very beneficial in cases wrongly assumed to be purely functional, but in which actual intrinsic anatomic narrowing is present; but the milder form of the same treatment—viz. bougieing—might be expected to prove useful in paretic conditions by exercising a stimulating effect; but it is hardly to be expected that such mechanical stimulation, more especially by bags and divulsors, would relieve spasms and other contractile derangements. I agree that the "bougie effect" sometimes does temporarily improve to a substantial extent some cases of non-anatomic stenosis, but the improvement in my experience is more often only slight in unequivocal cases of functional stenosis, and such improvement as is obtained I regard as due to stimulation and relief of the *inertia* of the crural neuro-muscular mechanism in the region of the hiatus, by which the collapsed hiatus and gullet are induced to open up widely at this site. When Sir StClair Thomson was advised last year in this Section to dilate up with large bougies a case of œsophagectasia with a widely open phreno-cardiac gullet as seen with the œsophagoscope in position, he pathetically inquired, "What am I to bougie?"

Mr. Shattock suggestively asks if "there may not be a fault on the side of the mucosa which fails to supply a proper afferent stimulus" for the requisite active opening up of the lower portion of the gullet. He, however, makes no suggestion that the inertia or block is localised at the hiatal level and concerned with the impaired action of the crural fibres of the diaphragm as I hold, but he accepts the usual view that there is wrong action of the circular fibres of the cardia supposed to be innervated by the sympathetic nerves of the plexus gulæ. The improvement in the relief of dysphagia in my cases I have attributed just as much to accessory methods of treatment as to such dilating methods as the passage of bougies.

Bags and divulsors I have discarded as there is no evidence forthcoming that either actual inflammatory rigidity or adhesions of the margin of the hiatus exists in these cases. I teach the patient in the early stages of treatment to pass all food directly into the stomach by means of a soft rubber stomach-tube, when the orifice of the phreno-cardiac gullet is easily hit off, or failing that, by a gum-elastic tube. He or she is taught to wash out the dilated œsophagus, and also the stomach when there is dyspepsia, before taking a feed though the tube, and if the obstruction is so great as to lead to a considerable accumulation of saliva in the gullet this is removed between feeds as well, so as to relieve strain and help the relaxed walls to gain tone. This lavage further helps to cure the sodden state of the mucosa, and to prevent the formation of erosions and ulcers which tend to result from continued stasis of stale food. After three weeks short drinks are permitted by the mouth, but any fluid remaining after ten minutes is removed by the patient with the rubber œsophageal tube. The improve-

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ment is sometimes so marked that soft food and even ordinary well-masticated meals can be swallowed either directly into the stomach or else they pass more gradually through without much delay. Care must be taken to eat slowly and little at a time, and the presence of stasis must be frequently tested by the patient by suction or lavage through the œsophageal tube. I have never known a real cure, but the improvement may be so great that the disability is undetected by others present when meals are taken, and the patient swallows fairly normally provided he does so carefully, deliberately and forcibly. In some cases the relief is only moderate, possibly due to the treatment not being religiously adhered to; relapses at all events are frequent.

Some cases improve scarcely at all under any method of treatment, and in these instances Mr. Shattock has suggested that it might be justifiable to excise the subphrenic gullet, and make an end-to-end junction between the dilated thoracic œsophagus and the stomach. I think the fixing of a non-slipping vulcanite or rubber intubation apparatus in the phrenocardiac gullet would be a feasible procedure if made in two portions, the lower one inserted and joined up through a gastrotomy, and it would be less dangerous than excision. I have tried Guisez's apparatus, but it slips up out of position just as does a Symonds' funnel here.

In conclusion, let me state that I fully realise that there is still a possible doubt as to whether œsophagectasia without anatomic stenosis below is a primary or a secondary condition, and it cannot be claimed that any of the explanations given, including my own, are anything more than unproven hypotheses and perhaps all very wide of the mark. I think, however, that the phrenic factor in the deglutitory act to which I called attention in 1911 cannot be ignored, as appears to be the case at present.

Ætiology.

With so much obscurity as to the essential nature of these ectasiæ, even as to whether there is an underlying functional stenosis or not, it necessarily follows that nothing much is really known as regards the predisposing and exciting causes. There is no sex predomination, and only a small proportion of the patients affected can be classed as neurotic. The condition is not limited to any period of life. Some few cases observed in the very young have been held to be congenital. Zenker is probably wrong in his statement that "ectasiæ seem to be developed more frequently in youth than at any other time," even if those following cicatricial stenosis are included in the statement. There is no reliable information regarding the exciting causes of the condition. Claims made by reporters of individual cases of the supposed influence of lifting heavy weights, imbibing large draughts of hot water, and the impaction of hot dumplings and other ingesta are not convincing.

Symptomatology.

I have devoted so much time to the obscure question of pathogenesis that I feel deterred from dealing at length even with the commoner symptoms, much less the occasional and exceptional ones. Suffice it to say that the commoner ones are more or less identical with those met with in other varieties of ectasiæ, viz. those developed above a cicatricial stenosis; the X-ray pictures are also identical, and the essential difference is only brought out *by the œsophagoscopic findings and by*

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bougieing, on which the differential diagnosis ultimately depends. Dysphagia varying in degree is nearly always a late symptom in œsophageal stenosis of any kind, as the lumen can be considerably reduced without interfering much with deglutition. In this form of functional dysphagia, however, some difficulty is noted comparatively early before marked secondary dilatation has occurred. It is often intermittent in the early stages, with quite prolonged periods of normal deglutition followed by relapses.

Later regurgitation is added to mere dysphagia. The dilated gullet when overfilled from above and unable to empty itself sufficiently quickly below necessarily tends to cause an overflow into the pharynx, and when this is accompanied by peristaltic actions the accessory muscular actions usually associated with gastric vomiting may be called into play, and thus we get what is known as œsophageal vomiting. Regurgitation is often involuntary, but on account of the discomfort felt in the overloaded chest the patient frequently reinforces the involuntary act by a voluntary one. Coughing and even deep breathing may lead to regurgitation when the gullet is filled or nearly filled with fluid or other form of nutriment.

The questions of waterbrash and of rumination occasionally observed in œsophagectasia I am precluded from dealing with at length now.

A dilated gullet which is at the same time over-filled is made evident to the patient by symptoms varying from mere discomfort to evident distress and up to genuine pain. The pain is of the nature of a secondary spasm, or cramp, or colic, or angina, and is brought about by hypertonic muscular action—*i. e.* violent peristalsis in an ineffectual effort to force impacted food through the stenotic lower end of the gullet. Violent pain may last from a second or two to fifteen minutes. With the throat mirror frothy fluid can often be seen in the pyriform fossæ, and where there is increased salivation, as often happens in well-established cases, much saliva which has been first swallowed and later regurgitated in a frothy form has frequently to be expectorated.

As regards loss of weight it is exceptional to find marked and dangerous emaciation, even in cases of long standing, though a few patients have been gastrostomised for symptoms bordering on aphagia, and cases have even been allowed to succumb to asthenia from starvation. In these instances a proper investigation should have established the correct diagnosis, and led to the employment of so simple a remedy as the stomach-feeding tube long before such a state of affairs had become established. There is usually a history of some loss of weight in long-standing cases even when the patients look healthy and well covered; a patient often states that he lost a stone or two to begin with in the course of six or twelve months, and then managed by careful deglutition to maintain a constant, though below par, weight for succeeding years. When the amount of nourishment which gradually trickles through from the dilated gullet into the stomach is large compared with the amount regurgitated, the patient may manage to maintain normal weight and strength for three or four years or even more after the onset of the dysphagia. The main point is that there is not usually in functional stenosis the steadily progressive loss of weight such as is met with in organic (*i. e.* anatomic) stricture.

(Dr. Hill showed a number of skiagrams illustrating functional phreno-cardiac stenosis.)

(*To be continued.*)

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