SOCIETIES' PROCEEDINGS.

BRITISH MEDICAL ASSOCIATION.

Meeting at Liverpool, 1912.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

JOHN MIDDLEMASS HUNT, M.B., *President*.

Abstract Report by MR. HAROLD KISCH.

After some initial observations, Dr. Hunt said: In Liverpool we have now throat departments in connection with three of our general hospitals, a lectureship in laryngology in our university, and there are seven men engaged exclusively in the practice of oto-laryngology. But much remains to be done before our speciality obtains that recognition which it has long enjoyed elsewhere. To illustrate what I mean I need only say that in this great centre of population, numbering considerably over a million, there are no beds specially set apart for the treatment of diseases of the throat and nose, except four in the Stanley Hospital, which are also used for ear cases. Personally I look forward to great changes in this respect as a result of the new Insurance Act.

Discussion on the Differential Diagnosis of Œsophageal Stenosis.

ABSTRACT OF OPENING PAPERS.

Prof. Otto Kahler firstly briefly discussed the methods of investigation formerly in vogue. He sounded a note of warning against the use of the probe. Auscultation was sometimes of use in the diagnosis. The Röntgen rays were a great help, and could be used in two ways—Röntgen illumination and radiography. The administration of bismuth salts was discussed and zirconium oxide (contrastin) recommended owing to its non-toxicity. The most suitable means of indicating stenosis was the administration of the drug in glutoid capsules of various sizes– 6, 8 or 10 mm. in diameter. Œsophagoscopy was, however, the most valuable method. He always used a Brünings' tube, the best length for the small tube being 25 cm., without a mandrin. For stenosis of the superior part of the œsophagus he preferred the patient to sit, for those in the inferior regions he favoured the lie-down position either on the back or left side. Local anæsthesia usually sufficed. The removal for examination was of the greatest use in diagnosis. Infection of the wound could be prevented by the immediate application of hydrogen peroxide or tincture of iodine, and the subsequent administration of formamint. He then mentioned the normal constrictions seen in the œsophagus during œsophagoscopy. With regard to foreign bodies, he mentioned three cases in which calcification in the thyroid had led to a wrong interpretation of skiagrams. The intense pain felt when the spot under which the foreign body lies was touched might be turned to

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account in differentiating between a foreign body, around which the parts are much swollen, and malignant disease. Foreign bodies often cause slight scratches, especially at the mouth of the œsophagus, leading to spasm and subjective stenosis. Perioesophageal abscesses, secondary to foreign bodies, might cause almost complete obstruction. Other causes of stenosis were the swallowing of caustic fluids, peptic, syphilitic and tuberculous ulcers, and after scarlet fever and diphtheria. Three forms of œsophageal tuberculosis were described by Guisez: (1) The secondary œsophageal tuberculosis arising from perforation by tuberculous glands: (2) the ulcerous form; (3) the tumour form, which could only be diagnosed by the removal and examination of a fragment. Stricture due to actinomycosis had been described, and the review concluded with a consideration of compression stenosis.

Malignant Disease and Pouches in the Œsophagus.—Mr. E. B. Waggett said: Much as we all dislike the subject, we have to remember that at least three out of four gullet cases are malignant. A case of dysphagia with regurgitation of alkaline food, emaciation, pain, recurrent and sympathetic nerve paralysis, will probably turn out to be one of malignant disease, but we cannot for a moment make a definite diagnosis on these symptoms. The one certain diagnostic sign, namely, the spontaneous ejection of a piece of growth, the nature of which can be proved by microscopic examination, is an occurrence of which I have knowledge neither at first nor second hand, though I have seen it quoted in general terms in some of the books. I need not say here that the admixture of blood with regurgitated food is by no means diagnostic of cancer, for the blood may come from dilated veins or from fissures and ulcers which are non-malignant. I am surprised to find that the general text-books still affirm that the sovereign means of diagnosis lies in the passage of the bougie. It is true that in the hands of certain surgeons the blind passage of bougies, coupled with great manipulative skill, long experience and the clinical instinct, has given reliable results with but few errors and accidents; but this blind method of diagnostic palpation has now to compete with methods of recent development. I am anxious that we should at this meeting to-day once and for all throw cold water upon blind bougieing of the gullet, for the reason that it is both unsafe and quite inconclusive. We now have two diagnostic methods, both of which can be used with safety, and one of them with certainty—the X rays and the direct method. With the X rays we can with perfect safety diagnose stenosis of the gullet and locate the position of the stenosis, determining at the same time whether the cause is intrinsic or extrinsic—that is to say, due to stricture in the true sense or to compression by neighbouring structures. A good deal of information as to the mediastinal contents is forthcoming from a simple X-ray photograph, but with the addition of bismuth porridge and the right antero-lateral posture very precise information is to be obtained. With a powerful source of X rays and a small diaphragm in use we can observe the process of deglutition in the fluorescent screen with no difficulty. Direct œsophagoscopy enables us to make absolutely sure of our case and to make a genuine diagnosis. Examination with the œsophagoscope would be ideal if it could be made without danger and without discomfort. First, with regard to danger: I make bold to say that the inspection of a stricture, malignant or otherwise, can always be made without danger, or at least with no more danger than that entailed by the local or general anæsthetic employed. A certain

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number of fatal accidents have occurred, but these are avoidable accidents. In all cases they have been due to one of two causes, namely, laceration of the diseased tissues by a guide or mandrin projecting in front of the œsophagoscopic tube, or else the attempt to pass the tube into and past the stricture. For purposes of diagnosis it is never necessary to do more than bring the end of the tube into proximity with the stricture, and in the majority of cases it is unnecessary to go nearer to the stricture than half an inch; from this distance one can with precision and safety wipe the diseased part with cotton-swabs and remove a fragment with the forceps. Secondly, with regard to the mandrin. The passage of the cricoid constriction is facilitated by its use, and it is conceivable that when the X ray has made it clear that the upper region of the œsophagus is sound, the use of the mandrin may be permissible. But no suspicious area ought to be approached except under the direct guidance of vision. The mandrin is not a necessity, and I cannot recall a single case of healthy cricoid constriction which could not be traversed without its assistance. Where the upper end of the gullet is diseased the use of the mandrin entails a grave risk. The mandrin should never be used except in those regions of the gullet which are definitely known to be healthy. The use of bougies passed under visual control is, of course, a totally different matter. Next, as to the comfort of the patient. The sense of fear and insecurity is much diminished by employing the recumbent position, either dorsal or right lateral, and by administering a dose of morphine and atropine. Œsophagoscopy is not a pleasant process, and a hurried examination made under cocaine is often useless where merely a negative result is forthcoming. If in any such case the diagnosis is worth making, it is worth while to give a general anæsthetic. With a light chloroform anæsthesia there is no danger and no after-discomfort. It is wise to keep the patient under observation for twenty-four hours. With regard to difficulty there are two classes: the easy class, where the disease is definitely in the middle and lower portions of the gullet, and the difficult class, where it is at or about the cricoid constriction. An essential point in the second class is to make out the condition of the cricoid constriction as the instrument traverses it. This can be done if two hands are taken to the work—the left hand to supply the forcible traction upon the cricoid and the right hand to advance the spatula. During its gradual progress the spatula should be rotated on its longitudinal axis, so that all parts are brought into view. The manœuvre recommended by some surgeons whereby the tip of the instrument is felt suddenly to pop through the cricoid constriction is objectionable. I hope that you will urge the necessity of this gradual sliding of the instrument through the cricoid constriction under visual control. One question remains: Is œsophagoscopy always possible? The cases in which it is impossible must be very limited. Personally I have seen but two cases, one of ankylosis of the cervical and dorsal vertebræ in an old man, another of partial ankylosis of the jaw coupled with a good set of teeth. Killian's suspension laryngoscope promises a satisfactory method of obtaining a general view of the post-cricoid region.

Year by year the technique advances; we have excellent new tubes devised by Hill in this country and other experts abroad; we have, this year, Killian's suspension apparatus.

Turning to pouches, true œsophageal pouches are practically always traction-pouches, the result of cicatrisation of diseased bronchial glands. Consequently they are generally found just below the level of the bifurcation of the trachea and on the anterior aspect of the gullet. As a rule,

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they have a wide opening and a pointed fundus, and as the latter is nearly always on a higher level than the former, food does not tend to collect in and to distend the cavity. Consequently these pouches do not give rise to symptoms, and are usually discovered accidentally. One may say that the diagnosis is made by the œsophagoscope.

We must, however, consider the pharyngeal pulsion-pouch, for though it is in fact a herniation of the lower pharynx and not of the œsophagus, it comes into notice by obstructing the latter viscus. The diagnosis is fairly easy, and it is usually made by the patient, who, as his meal progresses, experiences increasing difficulty in deglutition, associated with the development of a swelling in the neck immediately below the level of the cricoid, and usually upon the left side. The patient learns that by manipulation he can partially empty the pouch and continue his meal.

These patients often complain of the rejection of unaltered food some hours after its ingestion; it comes up in small quantities at a time, unlike the gush of regurgitation or œsophageal vomiting observed in cases of true dilatation, such as occurs above an organic stricture. Unless the pouch is kept clean by syringing after meals, fermentation takes place, with the production of foam, which may cause great discomfort and incessant cough at night. I do not remember observing this particular symptom in any but pouch cases. In conjunction with the symptoms mentioned the X ray with bismuth porridge gives invaluable evidence, but we cannot always, by the mere inspection of the photograph, assert that the case is one of pouch.

An ingenious diagnostic method has been devised by Plummer. About six yards of silk are swallowed during the twelve hours preceding the examination. A bougie, with its perforated acorn-head threaded over this silk, is passed down to the obstruction in the gullet. The silk, its lower end more or less anchored in the intestines, is now pulled taut. If the bougie remains unmoved you may diagnose stricture, but if it is raised, say a couple of inches, you may conclude that you are dealing with a pouch, and that the distance between its fundus and the lower lip of its orifice is two inches. It should always be possible to diagnose a pouch by the direct method if the tube is passed under visual control and quite slowly. The difficulty is, of course, to see the horizontal slit-like orifice of the œsophagus, which lies immediately below the level of the cricoid and sometimes under cover of the lower edge of that cartilage. The spatula passes only too readily into the pouch, and it is easy to miss the œsophageal opening unless either the introduction or the withdrawal is made with great deliberation and with the additional aid of a probe. Mosher describes the detection of the œsophageal opening in an elusive case of this sort by the use of a pneumatic œsophagoscope, whereby he obtained a view of the inflated viscus and saw the two orifices as he withdrew his instrument.

Stenoses at the Lower End of the Œsophagus, with Special Reference to those of Spastic Origin.—Dr. A. Brown Kelly.—The symptoms of obstruction at the cardiac end of the gullet are: (1) Hindrance to the passage of solids and liquids into the stomach; all degrees may exist, from the slight or momentary difficulty with meat to complete obstruction to the passage of liquids. The patient may be able to force the contents of the gullet into the stomach by swallowing movements or by drinking large quantities of fluid. (2) Discomfort or pain in the epigastric region, which the patient attributes to food and drink lying in the gullet above the stomach. (3) Vomiting, or rather regurgi-

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tation, which takes place readily, often without effort, immediately after a meal or while fasting. There is no nausea, and the food returned has its proper agreeable taste. Occasionally blood may be present. (4) Profuse secretion of saliva. Gottstein states that 150 c.cm. may be secreted in ten to fifteen minutes. (5) Loss of flesh, weakness, cachexia.

The pathological conditions at the lower end of the œsophagus which may give rise to stenosis are: Cancer of the œsophagus or cardiac portion of the stomach; mediastinal tumours and other extra-œsophageal diseases leading to compression of the œsophagus; cicatrices; idiopathic hypertrophy of the œsophageal musculature; and spastic affections.

Cancer.—Next to the cervical end, the cardiac region is the most frequent site of cancer in the œsaphagus. The chief and often sole symptom is dysphagia, which usually sets in slowly. Spasm plays an important *rôle* in most œsophageal diseases, and particularly when these are situated in the region of the cardia; consequently long before the neoplasm has attained a size capable of causing obstruction complete dysphagia may be produced by spasm.

Malignant disease in the middle portions of the œsophagus in some cases progresses very slowly and has periods of improvement. Such alternations are not characteristic of cancer at the lower end, owing to constriction of the œsophagus and the comparatively unyielding nature of the surrounding tissues. The presence of blood in vomited or regurgitated fluid, in the œsophagus on inspection, and a marked tendency for the tumour to bleed, all point to malignant disease. The appearances produced by cancer in the lowest part of the gullet, as seen by direct examination, vary greatly.

The submucous type of cancer may closely resemble certain spastic affections of the cardia, and requires detailed consideration. It is in those cases in which we are most doubtful of the diagnosis, owing to the mucous membrane being smooth and intact, that we are precluded, as a rule, from excising a piece of the tumour for histological examination. Careful observation of the endoscopic appearances is, therefore, the more necessary. The neoplasm may be at an early stage and limited to a small area immediately above the cardia. The infiltrated part of the wall may then project into the lumen, giving it a crescentic shape; it moves during respiration less freely than the surrounding parts, and its surface is smooth, stretched, intact, and very red although occasionally pale. Very soon the surface ulcerates or becomes nodular, and the true nature of the bulging is evident, or may be determined by removal and examination of a fragment. The malignant process may be more advanced, and may involve the entire circumference, producing an annular constriction. If it develop uniformly, a funnel-shaped stenosis will result. This has to be distinguished from the funnel-like constriction due to corrosive action. In the latter case the funnel is deeper, its surface is more uniformly smooth, and scarring may be seen above. When caused by cancer the surface is often nodular and ulcerated, and the mobility of the parts during respiration is diminished or absent. In both cases the œsophagus may be somewhat dilated above the constriction

The œsophagus may be normal as far as the hiatus œsophageus, and the disease causing the dysphagia may be located beneath the diaphragm. In our efforts to traverse the epicardia-cardia it is most important to carry out all manipulations with caution, and to employ no undue force in attempting to pass the œsophagoscope deeper. The danger is not so much of perforating with the point of the instrument as of tearing the

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neighbouring diseased tissue in consequence of the parts being put on the stretch.

When the cancer is in the diaphragmatic canal the difficulties of inspecting the parts are great. In the normal subject the passage of a tube into the stomach is not always easy, so that when an apparent obstruction is encountered it is necessary to allow for the natural tonus of the cardia as well as to keep in view the possible presence of a neoplasm. Owing to the limited field of view in this situation one must content oneself with the detection of very slight changes, and, if need be, examine repeatedly with the patient in different positions in order to get a characteristic aspect. If the neoplasm is in the stomach it may cause obstruction by submucous infiltration and narrowing the cardiac orifice. One looks for a tumour-like prominence or fold and particularly for an ulcerated or readily bleeding surface. Tapia has pointed out in connection with one of his cases that the respiratory movement of the fluid lying at the cardia instead of being up and down as usual is from side to side. He considers that this appearance allows of an exact diagnosis being made of the site and extent of the tumour. In the case in question the neoplasm was seen in the lowest part of the œsophagus on the right wall. This, however, was merely part of a large growth which sprang from the small curvature of the stomach and extended across beneath the cardiac opening. Its upper surface served as a resting-place for fluids, hence the horizontal oscillations.

Compression Stenoses.—Stenosis in the region of the cardia in consequence of extra-œsophageal pressure is nearly always due to a mediastinal tumour. Guisez has seen stenosis and displacement of the cardia produced by aneurysm of the aorta in one patient and by cancer of the liver in another. A tumour of the great curvature may act similarly. The œsophagoscopic appearances may be indistinguishable from those produced by submucous cancer. There is the same encroachment on the lumen by a smooth bulging of the wall. The covering mucous membrane is usually intact and normal in appearance, but may be nodular or ulcerated. The affective part does not move during respiration. The symptoms of œsophageal stenosis develop insiduously, and before they are marked the mediastinal growth has attained considerable dimensions and is evident on X ray examination.

Guisez emphasises his observation that in stenosis by compression dysphagia is quite out of proportion to the degree of obstruction. This he attributes to spasm.

Cicatrices.—The history of having swallowed a corrosive is often considered sufficient to establish the diagnosis. This is not always to be relied upon. The stenosis may develop very slowly, and years may elapse between the date of the accident and the onset of the signs of obstruction. On the other hand, it is not uncommon for patients to attribute their dysphagia to an injury from hot liquid or food, or a foreign body, and on examination to be found suffering from malignant disease. Investigations into the mechanism of deglutition have shown that solid food is carried comparatively slowly down the gullet by peristalsis, while liquids are projected rapidly or squirted to near the cardiac end by the muscles of the mouth. The facts noted in the cases that I have examined—namely, that cicatrices due to corrosive liquids are found in the lower part of the œsophagus, while those due to solids are in the upper part—are in accordance with the physiological observations mentioned. Guisez has found cicatricial stenosis in the neighbourhood of the cardia, in five patients probably due to a round ulcer, and

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in other five as a consequence of chronic œsophagitis In the latter group of cases the primary condition was spasm of the cardia, which, by causing retention of food, set up inflammation, infiltration, and concentric cicatricial contraction. A sound localises the site of the stricture, X rays give additional information as to its permeability, but only the œsophagoscope allows of a positive diagnosis being made as to the situation, nature, form, and degree of the obstruction. In old cases one usually sees a funnel-shaped constriction or diaphragm-like partition, with a more or less small aperture in the middle or to one side, and a surrounding smooth, pale, cicatricial surface. Several strictures may be superposed. If the lumen is much reduced, the œsophagus may be dilated above, and may contain food *débris* and saliva.

Idiopathic Muscular Hypertrophy.—Only very few cases of primary muscular hypertrophy of the œsophagus are on record. Elliesen has carefully studied the condition, and his case may be taken as typical. The patient was a man, aged thirty-nine, who died of tuberculous meningitis. The œsophagus, excepting above, was converted into a rigid, thick, uniform tube. The most important change was an enormous thickening, especially of the inner circular muscular layer. The external longitudinal muscular layer participated but little in the thickening. No other change could be detected. Ehlers more recently has met with a similar hypertrophy of the œsophageal musculature, which he regards as probably congenital, associated with congenital hypertrophic stenosis of the pylorus. In this case there was an immense thickening of the muscular wall, which a short distance above the cardia amounted to 12 mm. No other pathological condition was found in the œsophagus; its lumen was normal. In neither of the above cases was there dysphagia, although Elliesen's patient had to masticate carefully and eat slowly from his fifteenth year. It is possible that the dysphagia might have increased had he lived longer, and that death might have taken place from inanition, as occurred in the earlier recorded cases. Both writers discuss the ætiology of the hypertrophy, but neither can assign a probable cause. I think that cardio-spasm would explain both cases. An increase in the lumen of the œsophagus does not invariably accompany spastic closure of the cardia, and it was just because of the absence of dilatation that both authors excluded cardio-spasm as a possible cause. I am therefore not prepared to regard this condition as a distinct entity.

Paralysis.—Paralysis of the gullet is rare, but the symptoms to which it gives rise may cause it to be confused with stenosis at the cardia. The patient may be unable to swallow solids, while liquids pass easily; he may have pain behind the sternum during eating, and he may vomit frequently quantities of mucus. Our fears as to the grave nature of the ailment are set aside by the result of the examination. On auscultation the first deglutition sound may be heard if there is insufficiency of the cardia. A bougie passes readily into the stomach. The œsophagoscope is introduced with unusual ease, the parts appearing to be almost insensitive; there is no sign of disease in the gullet, and the epicardia-cardia is not unduly contracted.

Spastic Affections.—We here enter upon a subject that is highly controversial from the standpoint of the anatomist, physiologist, pathologist, and clinician.

Spasms play a very important part in affections of the œsophagus —for the moment I am speaking of the œsophagus as a whole and not of the cardiac end only. It may be either primary or secondary. We are all acquainted with secondary spasm, to which many strange and

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sometimes misleading clinical phenomena in the œsophagus owe their origin.

I may mention several examples. In œsophageal cancer not uncommonly the first symptom is sudden and complete inability to swallow, which, after a variable duration, passes off entirely. A patient with cancer at the lower end of the œsophagus may locate the dysphagia at the upper end owing to spasm. In another patient symptoms may point to obstruction at the cardia, and this may be confirmed by a bougie, whereas the neoplasm is beneath the mucous membrane in the middle portion of the œsophagus, and is still too small to interfere with sounding. The power of deglutition in patients with cicatricial stenosis may vary much from day to day, as may also the ease with which a bougie is passed, owing to spasm.

I now pass to the consideration of an affection which, in most instances, owes its origin to primary spasm—I refer to cardio-spasm.

Œsophageal Aspect of Cardio-spasm.—The normal aspect of the hiatus œsophageus varies in different persons, and even from time to time in the same person. It often appears as a cleft running obliquely forward and to the left, with a rounded fold in front and to the right, and another behind and to the left; it may also have a stellate appearance. Occasionally it opens a little, especially if a deep inspiration be taken, and scanty mucous secretion wells up. In cardio-spasm the appearance of the hiatus œsophageus may be the same as in the normal state. On the other hand, certain characteristic features may be produced by the contraction. The folds bounding the obliquely running cleft, or those radiating from the point-like lumen, may bulge unduly and produce an appearance like the os. The mucous membrane over the most prominent parts may be pale and traversed by blood-vessels, or, if irritated by retained food, may be inflamed. During the spasm there is no variation in the lumen with respiration, and no bubbling up through it of fluid or gas. Starck states that he has seen the hiatus free while spasm at the cardia produced a funnel-shaped constriction.

Having noted the points mentioned, the tube is now gently pressed onward. The parts may be found excessively sensitive, and an unusual amount of resistance may be experienced; but on waiting a little relaxation takes place, and the instrument glides along the abdominal section of the gullet and enters the stomach. Lerche states that in no case of cardiospasm is the epicardia-cardia impermeable. Starck distinguishes spasm in the diaphragmatic portion of the œsophagus from spasm at the cardia, and states that in the former he has found the closure so tight as to be insurmountable, while at the latter he has never met with muscular resistance. Spasm at the cardia may produce a stellate arrangement of folds, some of which may consist of gastric mucosa.

In carrying out these manipulations the œsophagoscope should be passed from the right angle of the mouth, and the patient's head bent to this side. The tube should be carefully kept in the axis of the gullet. Resistance at the cardia should, if possible, be overcome by cocaine, gentle pressure, and patience. It may be necessary to repeatedly alter the position of the patient before entrance to the stomach is effected. Lying on the side relaxes the crura of the diaphragm (Gottstein), and probably the right recumbent position, as recommended by Starck, is best of all.

Diagnosis of Cardio-spasm without Dilatation.—It will be gathered from what has already been said regarding compression stenoses and stenoses due to cancer at the cardia or in the diaphragmatic part of the gullet

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that the differential diagnosis of these conditions from cardio-spasm is sometimes extremely difficult; at present I am referring to cardio-spasm without dilatation.

Compression stenosis above the diaphragm can usually be recognised by the asymmetry produced; pressure below the diaphragm may alter the direction of this part of the gullet. When the abdominal œsophagus is involved, either by infiltration of its wall or by external pressure, inspection may fail to differentiate the condition from cardio-spasm. If difficulty is experienced in entering the stomach, in spite of the adoption of the manipulative measures recommended, it is best to suspect the presence of malignant disease. Repeated examinations should then be made at intervals, with the object of detecting the development of irregularities of ulceration in the neighbourhood of the cardia. Any attempt to pass the œsophagoscope into the stomach should be made with great gentleness. It is necessary not only to keep the instrument completely under the control of the eye and hand, but to bear in mind that extremely vulnerable diseased tissue may be in the immediate vicinity. If the state of the patient do not allow of delay, let gastrostomy be performed, and settle the diagnosis later.

Dilatations of the Œsophagus.—Stenoses at the cardia are liable to be followed by dilatation of the part of the œsophagus immediately above. When the stenosis is due to cicatricial contraction, extra-œsophageal compression, or malignant infiltration of the wall, the dilatation, if present at all, is, as a rule, insignificant. In cardio-spasm it may also be *nil*, but on the other hand it may be immense. A marked dilatation, with symptoms of obstruction at the lower end of the œsophagus, is therefore strongly suggestive of cardio-spasm. The cases in which a differential diagnosis is so difficult and to which reference has just been made are those of cardio-spasm without dilatation.

Angulation and Kinking of the Œsophagus.—The œsophagus not infrequently undergoes marked deviation, sometimes with elongation; the elongation may be associated with, or independent of, dilatation. The normal length of the œsophagus is about 25 cm.; in a case of Luschka's it measured 46 cm. When the elongation is such as to cause the gullet to approach the hiatus diaphragmaticus obliquely, kinking may be produced and symptoms of dysphagia may arise. It is probably only under such conditions that the crura of the diaphragm play a part in causing obstruction. In the later stages of cardio-spasm with dilatation the dysphagia therefore is chiefly mechanical. The recumbent position is then specially unfavourable for deglutition, as the food gravitates to the deepest part of the sac and cannot get into the stomach.

Kovács and Stoerk have recently studied the condition of the œsophagus in enlargement of the heart. They show that as a result of enlargement, especially of the left auricle, the œsophagus may be markedly compressed and deviated. The bend, which may approach a right angle, passes to the right and backwards, and the lowest part of the gullet, in order to reach the opening in the diaphragm, must cross the aorta almost horizontally. They also prove by X rays that ingesta may be delayed above the bend. Starck has found the mucous membrane in this situation very inflamed, a condition which he assumed to be due to the temporary retention of food.

Bending of the œsophagus is recognised during direct examination by the difficulty or impossibility of keeping the lumen of the gullet opposite the end of the œsophagoscope even when the tube and patient's head are bent unduly to the right or forward.

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Symptoms.—Occasionally a dilatation of the œsophagus is found at a necropsy without there having been any symptoms to suggest its presence during life. As a rule, however, symptoms are manifested. These may set in suddenly and be very marked, some form of dysphagia being the most prominent; or they may develop insidiously, so that years may elapse before the affection causes trouble. A characteristic symptom of dilatation is regurgitation. This is unaccompanied by nausea or retching, and may be brought about merely by increase of intra-thoracic pressure without active contraction of the gullet. The regurgitated fluid consists of undigested food and mucus, is usually alkaline, but if long retained may be slightly acid; it contains lactic acid, but no hydrochloric acid or pepsin.

It is not surprising that patients complaining of vomiting and epigastric pain, amongst other symptoms, are regarded as suffering from disease of the stomach. A fair number of cases are recorded in which gastro-enterostomy was performed for persistent vomiting, and at the *post-mortem* examination the disease was found in the œsophagus and not in the stomach. In cases of violent, cramp-like pains in the epigastrium, Gottstein emphasises the importance of keeping in view not only affections of the stomach and gall-stones, but also spasm of the cardia. In all of his cases of cardio-spasm the disease had been treated for years as ulceration of the stomach or gall-stones.

Rumination.—In connection with regurgitation reference should be made to merycism or rumination. Some of the published reports of cases of rumination strongly suggest that the patients had stenosis at the cardia with regurgitation. The correctness of the diagnosis in these cases becomes more doubtful when no mention of the latter condition is found, and when at the *post-mortem* examination of some ruminators dilatation of the lower part of the œsophagus is stated to have been present.

Well-marked cases of regurgitation and rumination are easily distinguished, but in some individuals the symptoms may not be characteristic, and the distinction may be difficult. In rumination heredity often plays a part. The process may set in at any age, even in childhood. It begins from a few minutes to half an hour after meals; mouthful after mouthful of the food is returned, chewed, and swallowed; and this may go on for an hour or until next meal. The food is pleasant, but may become sour if the rumination continue longer than two hours; it contains hydrochloric acid and pepsin.

Diagnosis.—Various tests have been devised to prove that the œsophagus is dilated, and that it can retain a large quantity of fluid without allowing any to escape into the stomach. Thus a stomach-tube is passed and 200 c.cm. of methylene-blue solution poured into the stomach. The tube is then removed, and the patient drinks 200 c.cm. of milk. Again the tube is introduced a distance of 30 cm. and unchanged milk is obtained, and on being passed to 50 cm. blue-coloured fluid returns. The capacity of the dilated œsophagus can be conveniently measured by Lerche's œsophagometer. X rays give valuable information.

Endoscopy surpasses all other methods in the extent and accuracy of the information it affords regarding œsophageal dilatations. When the œsophagoscope is in the lower third of the gullet, instead of being able to overlook the entire or greater part of the circumference, we find, if the patient is seated, that more or less of the wall passes out of sight and that we are looking into a large cavity. During expiration the dilatation will be at its smallest and the walls best seen. If the patient is recumbent during the examination, a fold of the overlying wall may dip down and

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cover the end of the tube and present no respiratory movements. It will also be found that in order to bring into view opposite points in the circumference of the gullet the tube must make a considerable excursus. The lining membrane of the dilatation is crossed by transverse folds and is inflamed or superficially ulcerated; usually it contains fluid, food, *débris* and mucus. The inspection must, lastly, discover any malignant tumour that may be present. The association of diffuse dilatation and cancer has been noted by several observers.

Ætiology of Œsophageal Dilatations.—The first important contribution towards the elucidation of dilatation of the œsophagus was made by Zenker in 1878. This writer published reports of a series of cases in which the patients died with symptoms of severe dysphagia, and at the necropsies œsophageal dilatation was found, but no stenosis; he thought the condition was due to diminished power of muscular contraction. In 1881 Strümpell, in discussing a similar case, raised for the first time the question of spasmodic closure at the cardia as a cause; he dismissed this, however, in favour of the view that there was kinking at the hiatus œsophageus and secondary dilatation in consequence. V. Mikulicz in 1882 made the first œsophagoscopic examination of the affection; he attributed it to spasm at the cardia, and therefore termed it "cardio-spasm." In almost all cases of so-called idiopathic dilatation of the œsophagus there is spastic closure at the cardia, as indicated by the symptoms, appearance, abnormal resistance, and the fact that cure is effected in a large proportion of cases by stretching the cardia. The study of the ætiology of dilatation of the œsophagus is thus inseparably bound up with that of cardio-spasm. Before proceeding to consider the theories that have been advanced to explain these associated conditions it is necessary to discuss certain objections that have been raised to the existence of primary spasm of the œsophagus.

Does Primary Spasm of the Cardia Exist?—Dr. William Hill, whose exceptionally large experience in œsophagoscopy entitles his utterances on the subject to our respect, has recently published a paper denying the possible existence of primary spasm at the cardiac end of the œsophagus. In support of his contention he asserts that the circular fibre musculature at the extreme lower region of the œsophagus is weak, and, if anything, weaker than elsewhere, and quotes passages from Prof. Macalister's "Text-book of Human Anatomy" to show that there is no special muscular sphincter in this region.

In order to obtain first-hand evidence as to the condition of the musculature at the junction of œsophagus and stomach, Dr. Williamina Abel, Carnegie Research Fellow in the Physiological Department of the University of Glasgow, has made a number of dissections. These confirm the correctness of Prof. Macalister's description as quoted by Dr. Hill. On the other hand, nothing was found in any of the dissections or in the anatomical works consulted to justify Dr. Hill's statement that the circular fibre musculature was specially weak in this region. I would point out, however, that "the modern supporters of the spasmodic theory" do not seek to explain spasm at the lower end of the œsophagus by the presence of a sphincter. The few writers who have employed the term "cardiac sphincter" have probably done so rather as a matter of convenience than from any desire to account for the pathogenesis; had they used the expression "sphincter-like appearance" in reference to the hiatus œsophageus no objection could have been raised. The true explanation of cardio-spasm will probably be found to lie in a disturbance of innervation; to this important aspect of the question Dr. Hill has barely referred.

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Mention will be made immediately of the various theories that have been advanced in this connection, but I may conveniently preface them by recounting a few physiological facts, which will also serve to prove that spasm at the cardia is possible.

From experimental work it has been found that stimulation of the peripheral ends of the cut vagi causes contraction of the whole œsophagus but dilatation of the cardia; this is essentially the normal process of deglutition. On the other hand, section of the vagi without stimulation is followed by dilatation of the lower part of the œsophagus and contraction of the cardia; this corresponds to the supposed condition in cardio-spasm. The vagi thus control the muscular action—the peristalsis—of the œsophagus and supply a special dilator branch to the cardia. Tonic contraction at the cardia is maintained by the autonomous nervous system, which consists of a fine plexus situated between the two muscular coats under control of ganglia which are independent of the central nervous system. Normally, this tonic contraction is inhibited during deglutition by impulses conveyed along the vagi, and the cardia opens to allow the food to enter the stomach. Secondary closure of the cardia is due to reflex action excited by the acidity of the gastric juice.

Returning to the clinical aspect, the question that next suggests itself is: Have we a correct conception of spasm as it exists at the cardia? We are accustomed to associate in our minds with the term "spasm" a condition of violent muscular contraction, but if we read the reports of a number of cases of cardio-spasm we find that almost invariably the obstruction at the cardia is stated to have yielded on waiting a little. Some writers assert that the cardia-epicardia is always permeable in cardio-spasm. In this connection I might mention that even in animals with both vagi cut, when the spasm is undoubted, it can be overcome comparatively easily by increasing the intra-œsophageal pressure. At one time I myself held a different opinion, and in consequence experienced a mishap that I shall always regret. In a patient with great dysphagia, but with no sign of disease in the œsophagus excepting firm closure at the hiatus, my efforts to overcome what seemed to me to be strong spasm caused a tearing of neighbouring diseased tissue with fatal result. Secondary spasm may certainly have been present in this case, but the main resistance encountered was due to subdiaphragmatic carcinoma. Dr. Hill states that from his endoscopic observations spasm of a really unyielding nature is not found. Has he not been looking, as I did, for what is non-existent? It might be advisable for all of us to remember that in cardio-spasm the spasm is of a yielding character and little more than a state of non-relaxation.

Theories as to the Causation of Diffuse Dilatation of the Œsophagus: (1) *Primary Cardio-spasm.*—The first explanation of œsophageal dilatation, as already stated, was offered by v. Mikulicz, who attributed it to the retention of food, etc., above the spastically contracted cardia. In support of this view it is pointed out that a stenosis of comparatively short duration, as in cancer, is occasionally found associated with a certain amount of dilatation. In cardio-spasm, which may go on for years or even decades, great dilatation need, therefore, excite no surprise. The muscular hypertrophy of the walls of the distended organ suggests the presence of obstruction to be overcome, as in valvular disease of the heart. Meltzer goes a step further. He believes in the presence of cardio-spasm, and attributes it to inhibition of the cardia dilator. In consequence the cardia, instead of relaxing during deglutition, as it normally should, remains firmly closed. Rosenheim opposes the theory of primary cardio-

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spasm because marked and extensive dilatation of the œsophagus above anatomical strictures, both benign and malignant, is comparatively rare, and further, because obstruction at any part of the œsophagus to the passage of ingesta produces, if the muscular strength of the organ be well maintained, regurgitation, but no dilatation.

(2) *Primary Atony.*—Another group of observers maintains that the dilatation is primary. Rosenheim, the chief exponent of this theory, attributes dilatation to atony of the œsophageal musculature, followed by overstretching and distension of the walls, and regards the spasmodic closure of the cardia as secondary and produced by the irritating action of stagnating collections of food on the altered mucous membrane. Quite recently the atonic theory has received support from Holzknecht. His X-ray researches have led him to recognise in a large proportion of those suffering from nervous dysphagia a characteristic condition which he regards as due to an atonic state of the œsophageal musculature. When very thick semi-solid food is swallowed normally it passes through the gullet as a closed column, the length of a finger, in about seven seconds. In the affection in question, on the other hand, each portion of semi-solid food is pushed onward by that next swallowed until a continuous stream courses through the gullet. A variable interval, often a quarter of an hour, may elapse before all reaches the stomach. The dysphagia may be overshadowed by various neurotic symptoms—for example, cramp-like feelings in the throat and chest, retching, attacks of choking, and occasionally inanition may result.

Starck admits the possibility of atony leading to dilatation, but states that no case has been proved, for almost without exception muscular hypertrophy is found at *post-mortem* examinations. This has not been my experience, for in a comparatively small material I have met with thinning twice, in one case to a very marked degree. A case of Lerche's reported this year promises to throw light on the nature of the disease, and to open up new possibilities in its treatment. A piece of banana had stuck in the patient's throat; afterwards she experienced delay in the passage of food through the upper part of the chest, and subsequently was unable to get anything into the stomach. X rays showed a sausage-shaped dilatation. There was not much obstruction to food at the lower end of the œsophagus, but it passed slowly through the upper part. The galvanic current was applied to the interior of the œsophagus in this region, which was undoubtedly in a state of atony. The electrode consisted of four thin steel felt-covered springs. After twenty applications there was surprising improvement, and after ten more the epicardia-cardia was stretched. Two months later the œsophagus was found, on examination, almost normal. More than a year after treatment the patient was feeling perfectly well. In this case, according to the author, the primary disturbance was atony of the upper thoracic part of the œsophagus, which delayed the passage of food and thereby irritated the epicardia-cardia to spasm.

(3) *General Neurosis.*—The view that simultaneous dilatation of the œsophagus and spasm of the cardia is due to a general neurosis was advanced by Kraus as a result of his observations of a case in which, after death, atrophy of both vagi was found. He considers that it reconciles the two apparently antagonistic theories already described. To explain his standpoint I may again remind you that if vagal transmission is interfered with the œsophageal walls will relax, while the cardia will remain closed and obstruct the entrance to the stomach.

These facts justify one in speaking of a paralytic dilatation of the

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œsophagus; they also explain why after death dilatation is found, but no anatomical stenosis. On the other hand they fail to explain the hypertrophy that exists in a large proportion of cases. If the musculature of the œsophageal wall is paralysed in accordance with Kraus's theory, it will not undergo hypertrophy.

Kraus does not assert that atrophy of the vagi is always present in œsophageal dilatation; in fact he refers to cases in which these nerves and their œsophageal branches were demonstrated to be normal; he desires rather to lay weight on the importance of taking the vagi and their connections into consideration.

(4) *Neurosis of the Autonomous Nervous System.*—The action of the autonomous nervous system in maintaining tonic contraction of the cardia has just been referred to, and it is easily conceivable that the tonus of this system may be increased so that normal impulses conveyed by the vagi are too weak to inhibit, and cardio-spasm results.

In Kaufmann's case, which was the first of the kind reported, of the four nerves which receive sympathetic supply and which react similarly pharmacologically—namely the vagus, oculo-motor, chorda tympani and pelvic (erigens)—three were affected.

Last year Heyrovsky reported a case of this nature. The patient was an exceedingly nervous woman who had increased tendon reflexes, absence of corneal and pharyngeal reflexes, and symptoms of heightened tonus of the autonomous nervous system, as indicated by cardio-spasm, dermography, abnormally profuse sweating, eosinophilia, hyperacidity and marked sensitiveness to pilocarpin. There was no slowing of the pulse. A beneficial effect from atropin could not positively be shown, as other therapeutic measures were employed.

Further evidence pointing to the nervous origin of cardio-spasm is afforded by the nature of the exciting causes and the temperament of some of the patients. A number of cases date from an injury received; others are traceable to the violent vomiting of pregnancy or sea-sickness. Such causes are capable of producing local lesions involving centres independent of the central of the nervous system.

You will recall the fact that after the entrance of food into the stomach the cardia is reclosed by the acidity of the gastric juice. It is easily conceivable that hyperacidity may give rise to undue contraction of the cardia or interfere with its normal dilatation. Hyperchlorhydria is probably, therefore, a potent exciting cause of cardio-spasm. The onset of the disease has been occasionally traced to psychical disturbances, and its course has been found to be influenced by emotions, moods, exertion, etc. In such cases the patient was frequently neurasthenic or hysterical. The bolting of imperfectly masticated or large pieces of food is supposed to be capable of producing œsophagitis and consequent spasm and atony.

(5) *Anatomical Abnormalities.*—Having disposed of the innervation theories of cardio-spasm, I shall now very briefly refer to certain other explanations that have been advanced to account for dilatation at the lower end of the œsophagus.

Luschka has described dilatation limited to a small part of the œsophagus above and below the diaphragm, which he has termed respectively the fore-stomach and cardiac antrum. The fore-stomach has been proved by Zenker to be congenital, and Fleiner has seen it in the living and dead subject as a dilatation resembling a sac or diverticulum. The condition may long remain latent until for some reason its function is disturbed. In these cases, however, the dilatation or fore-stomach is circumscribed and is quite distinct from the diffuse dilatation we have been considering.

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The former may therefore be regarded as a rare variety. The congenital origin of this small class throws no light on the ætiology of the typical diffuse dilatations ordinarily met with. Congenital narrowing of the cardia has been offered as another explanation of diffuse dilatations. The circumference of the normal cardia is stated to be 6·9, 5, and 5·5 cm. by v. Hacker, Leichtenstern, and Stierlin respectively. Stierlin reports a case of great dilatation in which the cardia was only 2·8 cm., and refers to other similar cases.

In connection with congenital abnormalities the investigations of Mehnert may be mentioned. This observer found thirteen physiological narrowings of the œsophagus; the spindle-shaped segment between each two he termed an enteromere. The fore-stomach corresponds to Mehnert's eleventh œsophageal enteromere; while the narrowing of the cardia just referred to might be attributed to the persistence of the thirteenth physiological constriction. The fact that other developmental defects were present in some of the cases supports the view of a congenital origin.

Conclusions.—In conclusion, I venture to express the following opinions: Under the term "cardio-spasm" several distinct pathological conditions have been included. Those due to developmental or anatomical abnormalities should allow of differentiation and correct classification. The existence of primary cardio-spasm is undeniable. Cardio-spasm alone accounts for the *ante-mortem* and *post-mortem* conditions found in some cases. In others it affords an insufficient explanation, and further investigation is demanded. The disease is sometimes located in the extrinsic nervous system, as manifested by degenerative changes in the vagus or its œsophageal branches. The most frequent seat of the lesion is probably the intrinsic nervous system, and the disturbing cause an injury, hyperacidity, or other influence exercising a harmful action on the ganglia. A fuller knowledge of the physiology of the autonomous nervous system is likely to assist in elucidating the pathology of cardio-spasm.

Discussion.

Dr. William Hill (London) gave a lantern demonstration of slides illustrative of œsophageal stenosis. In the course of it he said that pulsion diverticula, or pressure pouches, were purely peri-œsophageal, and caused dysphagia by extrinsic pressure on the cervical œsophagus. The dissections of Keith, Killian and others showed that they were localised ectasia of the post-cricoidal pharynx, the mucous, submucous and fibrous coats forming the wall of the hernia. The diverticulum passed out between the oblique and fundiform fibres of the crico-pharyngeus (inferior constrictor) muscle. The mouth of the diverticulum could always be demonstrated both by endoscopy and radioscopy (with bismuth paste) on the posterior wall of the post-cricoidal pharynx. Killian was wrong in describing these diverticula as arising immediately above the mouth of the gullet as the fundiform fibres of the crico-pharyngeus intervene, and he was equally wrong in describing this fundiform bundle of fibres as the sphincter of the mouth of the œsophagus, as it was a part of the pharyngeal musculature and sphincteredthe deep pharynx. These diverticula were purely pharyngeal in origin, and could not be correctly described as œsophageal or as pharyngo-œsophageal as is often done. The mouth of the gullet was situated at the lower border of the crico-pharyngeus (inferior constrictor) muscle, and had no specialised sphincter development of its own either at its mouth or at its termination.

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The term "cardio-spasm," commonly applied to phreno-cardiac stenosis, was usually, if not invariably, inaccurate. The speaker had failed to find evidence of primary spasm, whether paroxysmal or continuous, in the region of the cardia; and the assumption that long-continued primary spasm could give rise secondarily to a permanent anatomic spastic contracture at the cardia was unproved. Deglutitory cramp of a short paroxysmal character and more or less painful was often observed as a secondary symptom in acute and chronic gullet stricture and also when large foreign bodies were impacted. But was there any convincing evidence that deglutitory cramp was ever primary anywhere in the gullet, or that it produced secondary functional stricture of the cardia? The term "cardio-spasm" had been in the past loosely applied to undoubted anatomic—that is, organic—narrowings of the lumen in the region of the cardia, as well as to almost every case of phreno-cardiac dysphagia of uninvestigated or unknown ætiology and pathology.

Dr. Walker Downie (Glasgow) held that primary spasm of the cardiac orifice did as a matter of fact occur. This opinion was based on a case which he had seen some years ago in consultation with a physician in the Western Infirmary of Glasgow. The patient was a man over sixty years, who had much difficulty in swallowing, and most of the food swallowed returned after an interval, and he was losing flesh steadily. A large bougie could be passed readily down to the cardiac orifice of the stomach, beyond which it or any smaller one could not be passed. Some time later the patient died. At the *post-mortem* examination, performed by the pathologist of the hospital, the œsophagus was found to be thoroughly healthy, with no malignant disease of its walls nor in any of the neighbouring structures, and the conclusion then formed was that the difficulty was due solely to spasm.

The President agreed with Mr. Waggett that the blind passage of a bougie should not be practised, but did not consider that there were a sufficient number of men able to pass the Killian tube, and that under these circumstances the passage of a bougie was the lesser evil.

Dr. Harris P. Mosher (Boston, U.S.A.) said the X-ray plate was often misleading in the diagnosis of œsophageal pouch. He had had two cases where the œsophagoscope proved an apparently typical plate to be wrong. The use of a widely open tubular speculum made it possible to learn the condition of the œsophagus in the cricoid region, and to dispense with the mandrin for the introduction of the tube. He preferred making examinations under a general anæsthetic, because he was trying to get a view of anything, and wanted that view to be as large as possible. In his examinations a tube 20 mm. in transverse diameter was used, and he felt that at times even larger tubes might be employed. The larger the tube the larger the field, and the greater the field the more accurate the diagnosis which it afforded. A negative examination under cocaine meant that everything had to be done over again. Not only was a more exact diagnosis possible under a general anæsthetic, but the treatment which could be carried out was far more efficient. In the dilatation of fibrous strictures, for example, one dilatation under ether or chloroform would accomplish what it took months to secure under local anæsthesia.

Mr. Jobson Horne (London) could not accept, as of general application, the proposition that the use of the œsophageal bougie should now be condemned. The time had not arrived for regarding the œsophagoscope as fully developed or the bougie as an obsolete instrument. The œsophagoscope at present was in only a few experienced hands, and a

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good deal of useful work could be done still with the bougie in conjunction with the X rays. The physiological, functional, and malignant forms of stenosis of the gullet, he considered, might be co-ordinated, as the sites of occurrence were commonly the same in all three. Of the diseases causing stenosis of the œsophagus, syphilis, he considered, ranked second to malignant disease, but relatively it was by no means so frequently met with. Tuberculosis of the œsophagus, comparatively speaking, was rarely met with, excepting in the last stages of pulmonary phthisis. Owing to the enforced recumbent position, the ulceration occurred on the posterior wall, and the cicatrisation seldom reached the stage of stenosis.

Dr. Bronner (Bradford) thought it was not always necessary or advisable to give a general anæsthetic for the use of the œsophagoscope. If the bougie were olive shaped and passed with care, there was not much danger to the patient; in fact, much less danger than the use of the œsophagoscope in unskilled hands. Strictures of the upper part of the œsophagus were frequently of non-malignant nature and due to fissures of the mouth of the œsophagus.

Mr. C. W. M. Hope said general anæsthesia was in almost all cases not necessary when examining the œsophagus. Except in a few cases of post-cricoidal growth, he found local anæsthesia exceptionally satisfactory.

Sir StClair Thomson (London) could not support Mr. Waggett's plea for the abolition of the bougie, for in many years' practice he had never had any anxiety from the careful use of the solid bougie, and yet in his early days of œsophagoscopy he had had many moments of anxiety. Fortunately he had never had, nor been associated with, a fatal accident from direct examination, but one of his cases had alarming emphysema of the neck with damage to the cricoid cartilage. Therefore he thought that teachers should insist on the fact that œsophagoscopy was a much more delicate and dangerous proceeding than direct tracheoscopy. Of course, once acquired, œsophagoscopy was a safe and sound procedure, and he should abolish the use of blind bougieing.

Mr. Herbert Tilley (London) thought that, while the mandrin was quite unnecessary in the hands of the practical œsophagoscopist, it would not be wise to deprive the beginner of its aid in enabling him easily to enter the upper regions of the gullet To pass the metal tube into this region required considerable practice, and only by this could the beginner learn the amount of pressure necessary to overcome the resistance in the cricoid region; in his days of inexperience the mandrin would probably be safer than the metal tube. The speaker also thought that the olive-headed bougie was not entirely useless or to be discarded, for nothing so effectually cured cases of functional dysphagia as the passage of such an instrument. He was still inclined to regard the condition as a functional spasm of the musculature in the upper gullet; if not, how would Dr. Hill explain the sense of resistance and its sudden giving way to the bougie with complete cure of the dysphagia? He had had many opportunities of using radium in large doses and properly screened for malignant stricture of the gullet, but at the Radium Institute they had never seen a cure of any form of squamous epithelioma affecting the mucous membrane of any part of the alimentary tract, although very great relief to dysphagia followed the dilatation of, and insertion of the radium capsules into, the malignant stricture. Some of this relief was probably due to mechanical dilatation of the stricture.

Mr. Waggett, in reply, said he wished to emphasise the following points: (1) The blind passage of the bougie left the surgeon in ignorance

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as to the nature of the obstruction: (2) it was therefore desirable that at least one surgeon in every hospital should be able to pass the œsophagoscope with safety through the healthy portions of the gullet; (3) it was unnecessary for diagnostic purposes to pass the instrument into and past the diseased area, and it should not be difficult to train students in this country, as was done in other countries, to pass the instrument with perfect safety, under direct visual control, to within a quarter of an inch of the lesion.

Dr. Brown Kelly, in his reply, maintained that the obstruction at the cardia in so-called cardio-spasm was due to spasm and not to paresis. This he had shown was caused either by abolition and inhibition of the cardia tonus or by the action of the autonomous nervous system in overpowering the dilator fibres of the vagus. In many cases of cardio-spasm there was epigastric pain, but this was not essential. In his opinion cardio-spasm did not imply violent contraction, but rather a condition approaching one of non-relaxation.

Dr. Hill, in reply, said that, according to observations with his deep pharyngoscope, Prof. Kahler and others were wrong in describing the upper view of the closed mouth of the œsophagus as appearing as a slit. The partially closed post-cricoidal pharynx presented a transverse slit-like lumen during the passage of the endoscope, but the true closed mouth of the gullet appeared as a dimpled pucker or rosette. Exner used radium salts enclosed in glass tubes, whereas their radium salts were enclosed in platinum screens of 2 mm. thickness, with an outer rubber covering 1 mm. thick. Moreover, there were other refinements of technique, so that the radium tube remained accurately in the cancerous area, so that no cauterisation took place, as was probable, at all events, in Exner's earlier applications. Dr. Brown Kelly's very interesting experiments and observations on section of the vagi seemed to him to support very strongly his (the speaker's) views that functional phreno-cardiac dysphagia was of the nature of atony, for Dr. Kelly showed that after section of the vagi the whole thoracic gullet was actually dilated—that is, atonic and paralysed—and it could scarcely be argued that the phreno-cardiac region was presumably in a state of spasm whilst the rest of the gullet was admittedly atonic. Those who loosely use the term "spasm" made no distinction between paroxysmal deglutitory spasmodic cramp and long-continued tetanus, and they did not appear to realise that primary spasm without subjective sensations presented great difficulties. Moreover, they made no distinction between hypothetical functional spasm and an alleged post-spasmodic anatomic contracture. Dr. Downie's 24 cases, recorded as "spasmodic," out of a total of no less than 100 cases of alleged gullet stricture, could not be accepted as unequivocal evidence, as the diagnosis had been made by the old methods of investigation in most instances. In answer to Mr. Tilley's remarks, Dr. Hill pointed out that the relief afforded by bougieing was no presumptive evidence of spasm, as that procedure would merely accentuate spasm, whereas bougieing admittedly relieved paresis by mechanical stimulation, and was the accepted treatment for the relief of soft inflammatory tumefaction, as well as for tighter anatomic strictures.

(*To be continued.*)

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