Zeitschrift	**Surgery a monthly journal devoted to the art and science of surgery**
ISSN	**0039-6060**
Band/Heft	**JUN 41(6)**
Jahr	**1957**
Autor des Artikels	**BARRETT NR**
Titel des Artikels	**The lower esophagus lined by columnar epithelium.**
Seiten	**891-894**

SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

VOLUME 41
JANUARY—JUNE, 1957

ST. LOUIS
THE C. V. MOSBY COMPANY

1957

SURGERY

VOL. 41 JUNE, 1957 NO. 6

Original Communications

THE LOWER ESOPHAGUS LINED BY COLUMNAR EPITHELIUM

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DEFINITIONS

THE ideas discussed here are not based upon statistics nor upon a large collection of specimens; they are the results of thinking about a few unusual cases of esophageal disease. As such they may be rejected or modified in the light of future experiences. Some may be worried because I have changed my opinion relating to certain matters, but progress is not static and there is no subject which does not yield more knowledge as the depths are sounded.

This paper concerns a condition whose existence is denied by some, misunderstood by others, and ignored by the majority of surgeons. It has been called a variety of names which have confused the story because they have suggested incorrect etiologic explanations; congenital short esophagus, ectopic gastric mucosa, short esophagus, and the lower esophagus lined by gastric epithelium are but a few. At the present time the most accurate description is that it is a state in which the lower end of the esophagus is lined by columnar epithelium. This does not commit us to ideas which could be wrong, but it carries certain implications which must be clarified.

The literature about esophageal disorders is confused because common words have different meanings in the minds of different writers. To avoid misapprehensions on this score a few simple terms will be defined as to the sense in which they are used in this paper. The *esophagus* is the narrow passage through which food and drink pass from the pharynx to the stomach. *Gullet* is an alternative word which is useful because it has no exact anatomic significance. The *cardia* describes the mechanism (flap valve or whatever it be) which prevents reflux from the stomach into the esophagus. The word does not refer to the site at which the gullet widens out into the stomach nor to a hypothetical muscle sphincter which some believe separates the two.

Lecture given at the Mayo Clinic and at Professor Owen H. Wangensteen's Surgical Clinic at the University of Minnesota in Minneapolis, Minn., in November, 1956.

Received for publication Jan. 25, 1957.

This paper is based upon the proposition that the condition under discussion is importantly different from *sliding hiatal hernia.* The latter is an acquired deformity, which may be present at birth, in which a part of the stomach has migrated from below the esophageal hiatus into the mediastinum. The esophagus has in fact become, temporarily or permanently, short, and a narrow tube of true stomach is elevated in consequence. As the description of the condition under discussion proceeds, the anatomic and clinical differences between it and hiatus hernia with short esophagus should be clarified beyond doubt.

Allison[1-3] has said that sliding hiatal hernia and a lower esophagus lined by columnar epithelium often exist together in one and the same patient. In such a person the proximal alimentary canal would be constituted in this way: pharynx, esophagus lined by squamous epithelium, esophagus lined by columnar epithelium, sliding hiatal hernia, and stomach.

The novelty of this anomaly of the lower esophagus is denied by those who remind us that it has been recognized for half a century under the title of "islets of ectopic gastric mucosa in the esophagus." At the risk of tedium this point demands absolute definition.

According to the Oxford Dictionary the word "ectopic" means out of place. In this sense an inguinal hernia contains tissues which are out of place, but surgeons do not refer to these structures as being ectopic. The words "ectopic" and "heterotopic" describe islands of epithelium found in parts of the alimentary canal where they are unexpected. Before the turn of the century pathologists had described patches of gastric epithelium as occurring at almost all levels in the gut but particularly in Meckel's diverticula. These patches were prone to pathologic changes and it was assumed, though never proved, that similar changes could be invoked to explain any lesion of the esophagus involving columnar epithelium. This at least is the sense in which I interpret the early writings of authorities such as Chevalier Jackson.[9] In these writings the pith of the matter was overlooked in so far as the esophagus was concerned. We know now that the best reason for splitting these hairs is to distinguish between those conditions which allow acid or alkaline digestive juices to bathe unprotected squamous epithelium and those, on the other hand, which are harmless because the unusual cells secrete no ferments.

The fact is that columnar epithelium can appear in the gullet as it traverses the mediastinum in three entirely different circumstances. The first is a sliding hiatal hernia; the second is the condition described in this paper, namely, that in which the lower part of the gullet is lined by columnar epithelium extending upward for a long or a short distance above the esophagogastric junction; and the third is true islets of ectopic columnar epithelium.

It may be apt to summarize the facts about islets of ectopic gastric epithelium in the esophagus. Taylor has carefully examined and reviewed this problem. He defines ectopic epithelium as a state in which a portion of the normal mucous membrane is replaced by a mucous membrane foreign to the

part but normal to some other reach of the alimentary canal. Such anomalies are common and occur in two varieties, congenital which may be superficial or deep and acquired.

Congenital superficial epithelial heterotopia is a process of substitution of the epithelium only. Nothing deep to the muscularis mucosae is involved. In the esophagus this is a common occurrence. In 1904, Schridde[18] found islets of glandular tissue resembling cardiac gastric glands in 70 per cent of the bodies he examined. He stressed that the islets were situated in the postcricoid region and that they could generally be seen with the naked eye (varying in size from ⅛ to ¾ inch in transverse diameter). They presented as smooth, velvet, depressed areas which were apt to be mistaken for erosions and were more often found on the back than the front of the gullet. He believed that they secreted gastric juice but that they were harmless because the ferments were neutralized by saliva. These findings were confirmed by others, notably Rector and Connerley[16] who, in 1,000 consecutive autopsies performed on children, found 89 examples of heterotopia in the esophagus. Twenty-six of the patches contained parietal cells but none had caused pathologic changes.

Congenital deep epithelial heterotopia involves the growth of epithelial structures in the esophagus deep to the muscularis mucosae and includes conditions such as gastric cysts and adenomyomatous tumors.

The acquired heterotopias are commonly found in the stomach and beyond and are manifest in the edges of healing ulcers (peptic, dysenteric, and tuberculous), ulcerative colitis, and other inflammations. They matter in the present discussion because if for any reason the squamous lining of the lower esophagus were to be destroyed it could, theoretically, be replaced by columnar cells. Moreover, the replacement could be patchy and simulate islets of heterotopia.

THE PRESENT ANOMALY

When the lower esophagus is found to be lined by columnar cells, the abnormally placed mucous membrane extends upward from the esophagogastric junction in a continuous, unbroken sheet. There is no question of ectopic islets. The extent of the anomaly varies from a few centimeters to the upper esophagus. This deformity is not associated with or complicated by other anatomic changes: that is (1) the external appearances and the muscular anatomy of the stomach and the esophagus are normal; (2) there is no anatomic change in the mediastinum; (3) the blood supply from the aortic segmental arteries and the left gastric artery is normal; and (4) the crus of the diaphragm and the peritoneal reflections in the neighborhood of the hiatus are normal.

THE ETIOLOGY

At present the explanation of this anomaly is not known. The following observations are suggestions which bear upon it.

Comparative Anatomy.—The mucous membrane of the esophagus and of the stomach varies in its distribution in different species of mammals. It is principally in the carnivora that the epithelium changes abruptly from squamous

to columnar precisely at the level of the "cardiac valve." And in this group the esophagus widens into the stomach at the same place as that at which the epithelium changes.

The anatomy of the mucous membrane of the foregut depends upon the food habits of the animal concerned. Those which have teeth in the mouth and which eat meat are arranged as we are; but in ruminants and species which are edentulous in the mouth, the stomach not only masticates the food but stores it before digestion.

Thus it comes about that in most vertebrates there is no "cardiac valve" mechanism and an important part of the stomach is lined by horny squamous epithelium. The extreme example is ornithorynchus (the duck-billed platypus) in which the squamous epithelium of the esophagus continues onward as far as the pylorus. But an important area of the stomachs of animals as widely different as the horse, the cow, the rat, and the rabbit is covered by squamous cells.

The points which are relevant to our discussion are these: (1) The precise limits of this or that epithelium is of no moment in most mammals in which a cardiac sphincter would be an unnecessary luxury. (2) This anomaly of the lower esophagus cannot be explained as a reversion to a more primitive zoologic type. (3) The tendency in the mammalian world is for squamous epithelium to continue onward toward the pylorus, not for columnar epithelium to grow up the esophagus.

Histology of Human Specimens.—Confusion has crept in again here, for although there is no doubt that in these cases the mucosa lining the lower esophagus looks, in every way, identical to the stomach (being velvety in texture, bluish in color, and raised in typical rugae), there are those who argue whether or not it is stomach.

The facts are these:

The thing which importantly distinguishes the stomach of a man from the esophagus is its mucosa. The mucosa of the stomach is formed of special tall columnar cells which are prolonged deeply into simple or branched tubules whose components have undergone further differentiation into cardiac, fundal, and pyloric glands. These glands secrete hydrochloric acid, mucus, pepsin, and secretin.

Functionally the columnar mucous membrane in these esophageal cases secretes little if any acid, pepsin, or secretin.

Histologically the upper part of the esophagus is normal and there is an obvious and abrupt transition between this and the unusual segment at the bottom. Just below this boundary, whose margin is corrugated, the columnar cells are flat and arranged in shallow, tubular glands amongst which lie mucus secreting units. There are no oxyntic cells and the structure of these glands resembles the normal deep esophageal glands.

Lower, in the esophagus the simple tubular crypts give place to more typical gastric mucous membrane. Scattered oxyntic cells appear. At any level in this abnormal segment, perfectly formed patches of squamous epithelium occur.

These findings, which are similar to those described by Allison and Johnstone,[3] suggest that the abnormal epithelium, despite its looks, does not function exactly as stomach and probably secretes little digestive juice.

Embryology.—Until recently differences of opinion have existed as to the development of the esophageal epithelium and of its glands in man. To elucidate these problems transverse sections of human embryos, at all stages in development between 3 mm. and 230 mm. C.R. lengths, were cut by Johns (1952).[10]

From the 3 to the 16 mm. stage the epithelium from one end of the gullet to the other is stratified *columnar* in type.

Between the 23 and 34 mm. stages it becomes two-layered and subsequently changes to multilayered although the cells remain columnar. This epithelium proliferates to such an extent that the lumen of the esophagus is actually, or nearly, obliterated and the channel is ultimately re-established by vacuolation. The vaculoles are largest above the level of the tracheal bifurcation and persist until the embryo is about 70 mm. long. Similar vacuolation occurs in the embryonic esophagus of the pig, rabbit, rat, and hedgehog.

After the 70 mm. stage, islets of ciliated columnar epithelium, which arise from the basal layer of the preceding stratified columnar lining, appear in the middle of the gullet; they extend, coalesce, and ultimately line the whole tube.

In the 130 mm. embryo the ciliated columnar lining is replaced by stratified squamous epithelium, and this change also begins in the middle reach of the esophagus and extends upward and downward. The last segment to lose its ciliated columnar epithelium is the upper end of the gullet, and traces of these cells have been described as persisting at birth.

During the development of a human embryo the sequence of changes in the epithelia which line the foregut might be arrested or deviated from normal. Thus it should not surprise us to find columnar, ciliated, transitional, or squamous epithelium anywhere between the mouth and the duodenum.

Acquired Heterotopia.—One of the facts which are difficult to explain is why this deformity always involves the lower esophagus. No specimen has as yet been described in which the whole of the gullet is lined by columnar cells. The explanation could be that if the cardiac valve of a normal person were to become incompetent and if the lower esophagus were, as a result, to be bathed for a long time by digestive gastric juice, the squamous epithelium could be eaten away and totally replaced by more quickly growing columnar cells. This concept might explain the site of the deformity, the fact that many cases occur in patients who have an incompetent cardia due to a sliding hiatal hernia, and the fact that many patients are elderly and have a history of heartburn dating back many years. But defects, produced experimentally in the squamous epithelium of the esophagus of the dogs, heal by squamous regeneration.

None of these suggestions smooth away all difficulties and the etiology remains open to speculation.

PATHOLOGIC CHANGES

Surgeons who have studied the histology of specimens removed at operation have found that the greater part of the unusual epithelium consists of simple tubular glands which secrete mucus but which include few true gastric elements. There are some oxyntic cells at the lower end of most specimens and these may be situated proximal to the junction between the gullet and the stomach. Thus the abnormal segment may sometimes be subjected to the harmful effects of its own gastric juice; but in speculating about this point there are other factors concerned.

The junction between the squamous and columnar epithelia in these cases is generally situated at about 20 to 25 cm. from the incisor teeth and not just above the diaphragm as it is in most sliding hiatal hernias; and the higher up the gullet a lesion is the less the risk that the squamous epithelium will be digested because the surface is more adequately protected by saliva and mucus.

Is there, in these patients, a "cardiac valve" mechanism, and if so where is it? My observations suggest that two types of cases occur. The first is the patient who has the lower part of the gullet lined by columnar epithelium and no pathologic lesion at the esophagogastric junction. On a barium swallow and when examined radiologically, this individual has what appears to be a normal gullet. The "cardiac valve" is in its usual place and the mechanism does not permit reflux; that is, the "cardiac valve" is a long way below the level at which the change-over in the epithelium occurs, and between the valve and the change-over there is a segment of gullet lined by abnormal epithelium. The fact that a "cardiac valve" can behave normally in such cases indicates that the esophagogastric angle is more important to this function than the exact histology of the epithelium at the level of the valve.

The second is that described by Allison and Johnstone[3] who reported that in most of their patients the lesion was complicated by the presence of a typical sliding hiatal hernia below the abnormal mucous membrane. In these there was no "cardiac valve" and free reflux of gastric contents was usual. In the past these cases have been diagnosed as straightforward examples of sliding hiatal hernia. In both types a variety of different lesions can occur singly or in combination.

Reflux Esophagitis.—Esophagitis is not so commonly the presenting abnormality as it is in cases of sliding hiatal hernia, but it occurs and may be initiated by reflux of gastric contents or as a result of secretions from the oxyntic cells which exist in the lower esophagus. It could also be due to both factors operating simultaneously.

The various pathologic changes which have been found follow the pattern described by Allison.[1-3] The lesions may be superficial, involving only the squamous epithelium, or they take the form of ulcers and leukoplakia. The ulcers are generally situated at the point where the squamous epithelium ends and the lower margin of the defect abuts upon the columnar epithelium. Ulcers which are due to esophagitis do not perforate or cause massive bleeding by

eroding a large blood vessel, but they can cause anemia because of a persistent small loss of blood. In some circumstances the inflammation may spread more deeply than the mucous membrane.

Esophageal Stricture.—When this occurs low in the mediastinum as a complication of sliding hiatal hernia, the strictures are due to reflux esophagitis and are caused by dense fibrous tissue which permeates the muscular coats of the esophagus, occasions mediastinal adenitis, and fixes everything to the adjacent structures. A few strictures occur which are like a cord of string tied in the submucosa and, as Allison says, these can be shelled out locally without interrupting the continuity of the gullet. It is also stated that, upon occasion and particularly in children, ascending esophageal fibrosis can develop and occlude a long length of lumen.

I assume, but am not convinced, that when the lower esophagus is lined by columnar epithelium and when a stricture develops high in the mediastinum at the junction of the squamous and columnar elements, the processes described above can be operative. But the outstanding feature of the specimens we have examined to date is not principally scar; it is muscle hypertrophy. Starting at the level where the epithelium changes, there is a striking increase in the circular muscle of the gullet. This accretion tapers off below the stricture over a distance of 2 inches or more. Whatever may be its cause it is not comparable with the hypertrophy proximal to obstructions elsewhere because it is most accentuated at the level of and beyond the stricture. Histologically the hypertrophied muscle bundles look normal and are not importantly invaded by inflammatory cells. The appearance of the tissue contrasts strongly with the base of a typical peptic gastric ulcer. Inside the muscularis mucosae, which is usually intact, there is generally a layer of submucous fibrosis with ulceration of the luminal epithelium; and the whole mass, which feels at operation hard like a cancer, is adherent to the adjacent structures in the mediastinum. Another point is that the blood vessels which nourish the submucosa and mucosa pass inward from the surface of the esophagus and negotiate this thick layer of muscle. Many of these vessels are thrombosed in the specimens we have examined.

These details could be important for the following reasons:

These strictures are not simply composed of scar. In many the bulk of tissue is circular muscle fibers.

It is generally assumed that the sequence of events which initiates an esophageal stricture of this type is peptic digestion leading to inflammation; that is, the inflammation spreads from within, outward; and as a secondary effect it produces the appearance of muscle hypertrophy because the gullet is shortened like the belly of the biceps in contraction. This explanation does not satisfy me because, in these cases, the esophagus is not short. Could it be that an entirely different process is at work? Suppose for the sake of argument that in some instances the first thing to go wrong is hypertrophy of the muscle. We know that wherever in the body a muscular tube changes its nerve supply, as does the esophagus between its upper and lower reaches, at that place it is

likely to develop muscular disorders such as hypertrophy, leiomyomata, conditions of spasm, neurofibromata, and so forth. If there should prove to be a basis of reason in this thought, the possibilities of treating these strictures, assuming them to be a muscular anomaly aggravated by sepsis, would be different from current practice.

ULCERATION

Peptic Ulcer.—Cases have been described of patients who have developed peptic ulcers in the abnormal segment of the lower esophagus. These ulcers have nothing in common with the shallow and transient esophageal erosions described above. They occur at a level below the change-over in the mucous membrane and they behave as do gastric ulcers in the stomach. Two kinds of peptic ulcer have been found.

Acute peptic ulcers: Acute peptic ulcers are not uncommon. It may be that these defects are the result of post-mortem changes in some instances, but they could be real ulcers. Their presence could account for some cases of unexplained hemoptysis. Allison has raised another interesting point. It is not known whether such an ulcer would heal by covering of columnar epithelium. It might heal by squamous metaplasia and if so, could account for the islets of squamous epithelium which have been found among the columnar cells. Such a speculation emphasizes the ever-recurring difficulty as to which lesions of the gullet are acquired and which are congenital.

Chronic peptic ulcers: Chronic peptic ulcers in the esophagus were described by Rokitansky, but the issue has been confused because it was formerly stated that they had arisen in isolated patches of ectopic gastric mucous membrane.

The differences between these ulcers and esophageal ulcers, on the one hand, and gastric ulcers, on the other, were first emphasized by Barrett,[4] in 1950, and for this reason the chronic peptic ulcer which complicates the lesion we are discussing and which is situated in what appears to be the gullet has been called Barrett's ulcer in the literature.

Barrett's ulcers are chronic gastric ulcers. They generally occur in middle-aged or elderly people and they invade and destroy the muscle coats of the esophagus. The adjacent tissues are edematous in life and as the ulcer penetrates through the wall of the gullet the mediastinal tissues become involved by fibrosis and inflammatory adenitis. The ulcer is liable to produce fatal hemorrhage by perforating a large blood vessel or mediastinal and pleural suppurations. Malignant change has also been described as having originated in such an ulcer, but the evidence is not conclusive.

The risk of a stricture forming is not great, because these ulcers grow in the line of the gullet and seldom involve the whole circumference. Thus the fairway is not usually obstructed and dysphagia, of which the patient may complain, is generally due to spasm above the lesion and to local edema. One may generalize and say that a benign stricture discovered in the lowest part of

the gullet is not likely to be due to a gastric ulcer. On the other hand, a stricture demonstrated at or above the aortic arch is almost certainly situated above an esophagus lined at its lower end with columnar epithelium.

Allison and Johnstone have stated that stenosis at the lower end of the gullet is not likely to be due to stenosis in connection with a Barrett's ulcer.

Carcinoma occurs immediately above the level of the change-over in the mucous membranes, in which case it is squamous in type and in the lower part of the esophagus when it is columnar. There are no special features which distinguish these lesions histologically from those growing in the normal gullet or stomach, but one important misconception needs correction. If a carcinoma starts at the normal anatomic esophagogastric junction and if it is columnar, it is difficult to say whether it has originated in an abnormality of the type under discussion in this paper or whether it is an example of a direct upward extension of a gastric cancer into the esophagus. The point is of academic interest at the level of the diaphragm but if a columnar cell growth is discovered at the level of the arch of the aorta, how is one to interpret this finding? In the past it has been said that the inference in such a case is that a gastric cancer has grown up the gullet from the level of the diaphragm. If this were always true the prognosis as regards resection would be hopeless because of the extent of the tumor. But it is seldom true. The usual explanation of such a finding is that the patient has developed a columnar cell cancer de novo in columnar esophageal mucosa. Some of these columnar cell cancers are localized and favorable for resection. The explanation that a columnar growth could have originated in an esophageal mucous gland is possible but has not been substantiated by an actual specimen. To prove this contention would involve demonstrating a columnar cell cancer entirely surrounded by squamous epithelium, and Carrie[7] has likened this to the unicorn which many have described but only one has seen.

DIAGNOSIS

When the lower part of the gullet is lined by columnar epithelium, the patient will have no signs or symptoms unless a pathologic lesion is present. In such a one, a barium swallow would not reveal an abnormality either in the part which is assumed to be normal gullet or in the stomach. That is, columnar epithelium in the lower esophagus does not affect normal peristalsis or cast a typical shadow of rugae in roentgenograms.

The diagnosis will only be made when a pathologic process has been added to the abnormality of the mucous membrane.

The symptoms of esophagitis and its complications are the same as those which occur when this process is due to a sliding hiatal hernia, except that the pain is often felt higher up in the thorax and referred to the middle of the sternum. Dysphagia and regurgitation of food and saliva with mucus are common, and anemia due to slow bleeding is a feature.

The symptoms due to Barrett's ulcer or to its complications are not specific, but the pain is more likely to be related to the taking of food than to the effects of posture. It is not usually associated with belching of wind, heartburn, or abdominal discomfort. These patients may complain of a severe, boring pain

localized to the back if fixation to the aorta is occurring. If, on the other hand, there is a perforation into the mediastinum, the signs and symptoms mimic those which are characteristic of a perforated gastric ulcer in the peritoneal cavity. I have had experience of a case in which the ulcer caused, at first, a pericardial effusion and, later, death due to suppurative pericarditis. If a major blood vessel such as the aorta is eroded, the patient dies of massive hemorrhage. Loss of weight is a common complaint, and symptoms such as dysphagia are often of long duration. There may be periods of remission in which it is possible that a measure of healing has occurred, or spasm and edema may have subsided for the time being. In the first series of cases which I published, the patients had died as a result of the complications caused by their ulcers. None had been diagnosed correctly or treated in life.

Radiologic Findings.—Johnstone has described the radiologic findings when the lower esophagus is lined by columnar epithelium. He has emphasized that, unless a pathologic lesion is present in the esophagus, there is no special pattern caused by the columnar mucosal folds which suggests the diagnosis. The superficial lesions due to esophagitis cannot be seen and peristalsis is not abnormal. A benign stricture at the level of the aortic arch should suggest that the lower esophagus is lined by columnar epithelium because sliding hiatal hernias are not usually as large as this.

The "cardiac valve" mechanism in these cases will be incompetent if a hiatal hernia is present as well; but if it is not, there will probably be a mechanism which prevents reflux situated at the expected place, that is, below the diaphragm where the gullet appears to enter the stomach. These cases lend support to the proposition that the most important single factor in competence or otherwise of the "cardiac valve" is the angle at which the gullet enters the stomach. I have had 2 patients who, in spite of symptoms, were at first thought to have nothing abnormal because a competent cardia was seen in the usual place below the diaphragm. Both had gullets lined with columnar epithelium at the lower ends and esophagitis at the level of the aortic arch.

If a Barrett's ulcer is present in the lower part of the gullet, Johnstone believes that a correct diagnosis can generally be made with a barium swallow. He says, "When a deep ulcer crater can be demonstrated close to the cardia, there is strong presumptive evidence of its gastric origin. Such craters may be more than a centimeter in diameter. The walls are clear-cut, and in one case terracing was observed. Undermining of the margins has also been seen, but the edges appear regular and smooth. The crater is usually single, but in two instances a second crater has been found. . . . They are more often on the posterior or the posterolateral wall, and they tend to spread longitudinally rather than to encircle the lumen. In some the crater lies in the stenotic segment, but in others the lumen is scarcely narrowed and there is little obstruction. Periesophageal infiltration is not uncommon, and soft tissue shadows may be seen at the base of the ulcer." It is not always possible to distinguish radiologically between such an ulcer and a carcinoma.

Esophagoscopy.—Esophagoscopy is an essential preoperative investigation in all patients suffering from disease in the esophagus.

The diagnosis should be suspected if the change-over of the mucous membrane is found at a high level. As the epithelial transition is sharp, there should be no difficulty in marking its point unless there is local inflammation. If there is a doubt, pinch biopsies will settle the matter. In most of my cases the change has been at about 20 cm. from the incisor teeth, and this is higher than one expects it to be if the diagnosis is a sliding hiatal hernia.

A fact which suggests that the lower part of the gullet is lined by columnar epithelium is that the level at which the change-over of the epithelium occurs at endoscopy does not tally with the observed level of the cardiac valve mechanism radiologically.

The diagnosis is clinched if columnar epithelium is found above a pathologic lesion in the gullet. If the patient has a stricture at the esophagogastric junction, the radiologist is likely to call it a carcinoma and the fixity of the parts may suggest the same mistake to the endoscopist even though a negative biopsy has been obtained. The moral of this is that if a good piece of tissue has been obtained for biopsy and if the histologic report is against carcinoma, the possibility of a benign stricture should be entertained. Such strictures occur at all levels in patients who have never swallowed caustics.

The diagnosis of Barrett's ulcer at endoscopy is difficult not only because these ulcers often develop beyond an esophageal stricture but because, through an esophagoscope, they look like carcinoma.

Thoracotomy.—In some cases the diagnosis will not be confirmed until the structures in the lower mediastinum and in relation to the right crus of the diaphragm which forms the esophageal hiatus can be inspected at thoracotomy. The presence or absence of a peritoneal sac and the anatomy of the hiatus and of the left gastric artery are the deciding factors.

TREATMENT

The lower esophagus lined by columnar epithelium has not been recognized long enough for anybody to be dogmatic about treatment. The following observations represent ideas rather than techniques of proved value.

It is reasonable to inquire what is the practical importance of being able to distinguish between a gullet partially lined by columnar epithelium and a sliding hiatal hernia? Both produce similar diseases and all one need know is that there are two conditions which may be different in origin, but which apparently result in the same defects. It is possible to make a case against this argument.

1. In sliding hiatal hernia the abnormal segment is a part of the true stomach. In this condition it is neither true stomach nor esophagus. How it may behave physiologically and pathologically is impossible to predict.

2. When the lower part of the gullet is lined by columnar epithelium the "cardiac valve" (if one exists) lies beyond the point where the epithelia change. After a sliding hiatal hernia has been reduced, the "cardiac valve" and the change-over in the epithelia correspond in level.

3. In sliding hiatal hernia, competence of the "cardiac valve" is attained by restoring the parts to their normal anatomic positions. Symptoms due to the abnormality under consideration cannot be cured in this simple way because there is no hernia to reduce and because the inflammation in the gullet is not always due to incompetence at the "cardiac valve."

4. The strictures, which occur as a result of the lower gullet being lined with columnar epithelium, are situated higher in the mediastinum than those due to a sliding hiatal hernia. This increases the technical difficulties of reconstituting the alimentary canal after excision.

5. Barrett's ulcer is rare as a complication of sliding hiatal hernia. But peptic ulcers occur in paraesophageal or rolling hernias. It is relatively common if the lower part of the gullet is lined by columnar epithelium, and its presence raises difficult problems of treatment.

6. It is probable that the majority of columnar cell carcinomas arising in the gullet (as opposed to the cardiac end of the stomach) grow from a segment whose lining is abnormal. The treatment of cases of this type has not been specifically considered by surgeons as yet. The prognosis in relation to radiotherapy and excision may not be the same as it is when the growth is squamous or truly gastric in origin.

Various contingencies arise, some of which will be discussed:

1. The patient complains of heartburn and on investigation the barium swallow is normal. No esophagitis is found at esophagoscopy, but the lower esophagus is lined by columnar epithelium.

In such circumstances I would tend to assume that the secretions from the lower part of the gullet were not the cause of the symptom and that the heartburn was due to intermittent reflux from the stomach. If medical measures did not produce relief, the object of performing an operation would be to improve the competence of the "cardiac valve," and the presence of the abnormal epithelium in the gullet could be ignored. Thus, some operation such as that advocated by Lortat Jacobs designed to accentuate the esophagogastric angle might be tried.

2. The patient complains of heartburn and has esophagitis above a stretch of gullet lined by columnar cells.

This could be due to reflux of secretions from the stomach or to secretion from oxyntic cells in the abnormal epithelium of the gullet. Hence, if an operation is to be performed, the lower end of the gullet must be separated from the zone of esophagitis. In practice this means resecting the gullet between the inflamed area of squamous epithelium and the stomach. The chief difficulty of this operation is to know how best to reconstitute the alimentary canal.

3. The patient has dysphagia due to a benign stricture at the change-over of the epithelia.

The best treatment of these cases is open to question because, as has been indicated above, there are a variety of different causes of esophageal obstruction which are all called strictures, and there is no accurate clinical way of deciding which is which preoperatively. For instance if the semblance of a permanent stricture is produced by spasm and edema due to inflammation or digestion of the mucosa, it might be reasonable to treat the patient medically, to allay the inflammation by using cortisone, to quiet the spasm with appropriate drugs, and perhaps to dilate the constriction through an endoscope. If the stricture is due to a localized submucous ring of fibrous tissue, Allison tells us that the ring can be excised and the parts returned to normal. If it is predominantly caused by hypertrophy of the smooth, circular, muscle fibers, some operation of the type devised by Rammstedt for pyloric stenosis might suffice. But if the stricture is principally due to fibrous tissue, one may regard the matter in the way Wangensteen[25] does and advocate subtotal gastric resection and pyloroplasty with subsequent dilatations of the stricture or take the view that nothing short of excision of the stricture itself is adequate to the occasion.

Whenever a stricture of the type we are considering is excised, the details of how to restore continuity arise. Morris[13] recommended end-to-end anastomosis of the esophagus as the simplest solution. Sweet and his associates[21] agreed with Valdoni[23] and have preferred esophagogastrostomy, while Ellis[8] has devised an operation in which he removes the stricture and the lower esophagus, divides the vagi, excises the pyloric half of the stomach, and re-establishes continuity by anastomosing the esophagus to a tube of stomach fashioned from the fundus and anastomosed at the other end to the duodenum. It is obvious that the parts must be amenable to surgery if they can sustain these various attacks.

In the United Kingdom, surgeons such as Allison, Brain, Davidson, myself, and others at first preferred esophagojejunostomy as a solution to the problem, but during the last 3 or 4 years we have been performing esophagojejunogastrostomy with improved clinical results. Merendino and Dillard[11] have shown that in experimental animals the free loop of jejunum interposed in this operation between the esophagus and the stomach is not subject to digestion from gastric juice, that it retains its peristalsis, and that it acts in some measure as a barrier against reflux.

In heralding this "new" operation let us remember that, before 1900, Tavel of Berne suggested using an isolated segment of small bowel to perform a gastrostomy because the transplant retained its peristalsis and prevented leakage. This observation was confirmed by Jean Charles Roux[17] who based his operation of gastroenterostomy en Y upon it, and in 1907, published his technique for by-passing a stricture of the esophagus in a child by using a free loop of jejunum in which arterial arcades had been so divided that the intestine reached from the neck to the stomach.

4. The patient has a Barrett's ulcer.

Surgical treatment is eventually essential in these cases but not before every effort has been made to establish temporary healing. Bed rest and a jejunostomy for feeding are good preliminaries. These ulcers probably behave like gastric ulcers and some can be induced to heal. As they heal, the edema in the surrounding tissues subsides and a patient who has complained of dysphagia may swallow again. To operate before taking these precautions is to invite technical difficulties. There is no information as to whether an ulcer which has healed will remain quiescent, and the feeling at the moment is that it should be removed. To achieve this the lower part of the gullet must be mobilized and the mass of inflammatory tissue around the ulcer itself makes this step difficult and, sometimes, dangerous. Before dissecting the gullet away from the aorta, the latter should be mobilized above and below the ulcer so that it can be temporarily clamped if serious bleeding should occur. After the lower part of the esophagus has been freed, this must be excised and the continuity of the alimentary canal restored.

5. The patient has a combination of pathologic lesions.

In these circumstances there will seldom be any alternative but to resect the whole of the abnormal area; and, if a sliding hiatal hernia is present as well, this must be reduced in order to achieve a competent cardia or excised with the lower part of the gullet.

6. The patient has a columnar cell carcinoma in the esophagus.

If the growth is situated at the lower end and in proximity to the stomach, the treatment will be as advised by Allison,[1-3] Sweet,[21] Wangensteen,[24] or Tanner for carcinoma at the cardia. In many cases the most the surgeon can offer is that he can so arrange matters that the patient can swallow. There is no justification in opening the chest in such a case, finding the growth to be inoperable, and saying nothing can be done. Whatever is found the patient's dysphagia must be relieved. This at least can be done.

If the carcinoma is at the level of the aortic arch or higher, there is less experience to draw upon. Two points suggest themselves. It may be that some of these cases will respond to radiotherapy better than one hopes, and this kind of treatment could be a valuable preliminary to surgical excision. Such problems are, at this moment, being reviewed by Smithers.[19] The other point is a surgical one. What should one excise? The whole of the lower esophagus and most of the stomach and its mesenteries or, more simply, the growth and the tissues in its immediate neighborhood? Either operation could involve plastic procedures such as reconstruction of a bronchus and perhaps replacement of a segment of the gullet by a suitable graft. Morson[14] and others have discussed these problems.

SUMMARY

It is suggested that the lesion under discussion should be called the lower esophagus lined by columnar epithelium, and that it is probably the result of a failure of the embryonic lining of the gullet to achieve normal maturity. The

differences between this condition and sliding hiatal hernia are underlined, and some of the reasons for distinguishing between the two are discussed. The pathologic conditions which complicate the anomaly are enumerated and the methods of diagnosis are described. The necessity for treatment and the various problems involved are recorded.

The present knowledge concerning the lesions associated with columnar epithelium in the gullet has come at the time when surgeons are beginning to feel that the pathology and treatment of esophagitis have been mastered. It may be that the importance of this new topic will be to stimulate research once again.

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