MEDICAL GRAND ROUNDS

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A PATIENT WITH DYSPHAGIA

In the last two years (April 1975 - February 1977), 59 patients with dysphagia were referred to our GI Service for endoscopic evaluation. The following endoscopic diagnoses were made:

TABLE I

Endoscopic Diagnosis of Dysphagia in 59 Patients

Diagnosis	No. of Cases
Esophagitis and/or stricture	14
Esophageal ulcer ± stricture	5
Achalasia, spasm, dilated esophagus	8
Carcinoma	6
Polyps	1
Other causes	26
(rings: 10; hiatus hernia: 8; normal examinations: 8)	

This may not represent a true distribution of the causes of dysphagia seen at PMH, as many patients with obvious motility problems were not

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endoscoped. Since we have followed the dictum, "dysphagia is never functional", all patients with unexplained dysphagia who were referred to our service were endoscoped. In any case, almost half of our patients with dysphagia were reported to have esophageal rings, hiatus hernia or a normal examination. The purpose of my presentation is to discuss some of these "other" causes of dysphagia.

I thought the best way for me to do this is to tell you a tale of a New York millionaire who could not swallow. This tale is of special significance for me as it marked the beginning of my interest in esophageal disease.

Case Report:

NYM, 57 y/o ♂

Had suffered from episodes of dysphagia for over 20 years. The dysphagia was intermittent, occurring 3 to 7 times a year. The episodes always occurred with solid food; meat, particularly roast beef and steak, were the usual offenders. Several times the episode occurred during social dinners and this was particularly embarrassing. Usually the patient was able to wash his bolus down with water, but on a few occasions he had to induce regurgitation and bring out the bolus. He believed that nervous tension precipitated his attacks, but he could not say for sure if he swallowed larger, improperly chewed boluses while under tension. He never had any trouble with liquids or soft foods. He had no weight loss, and he maintained good health otherwise. There was no history of heartburn or chest pain except during the episodesof dysphagia.

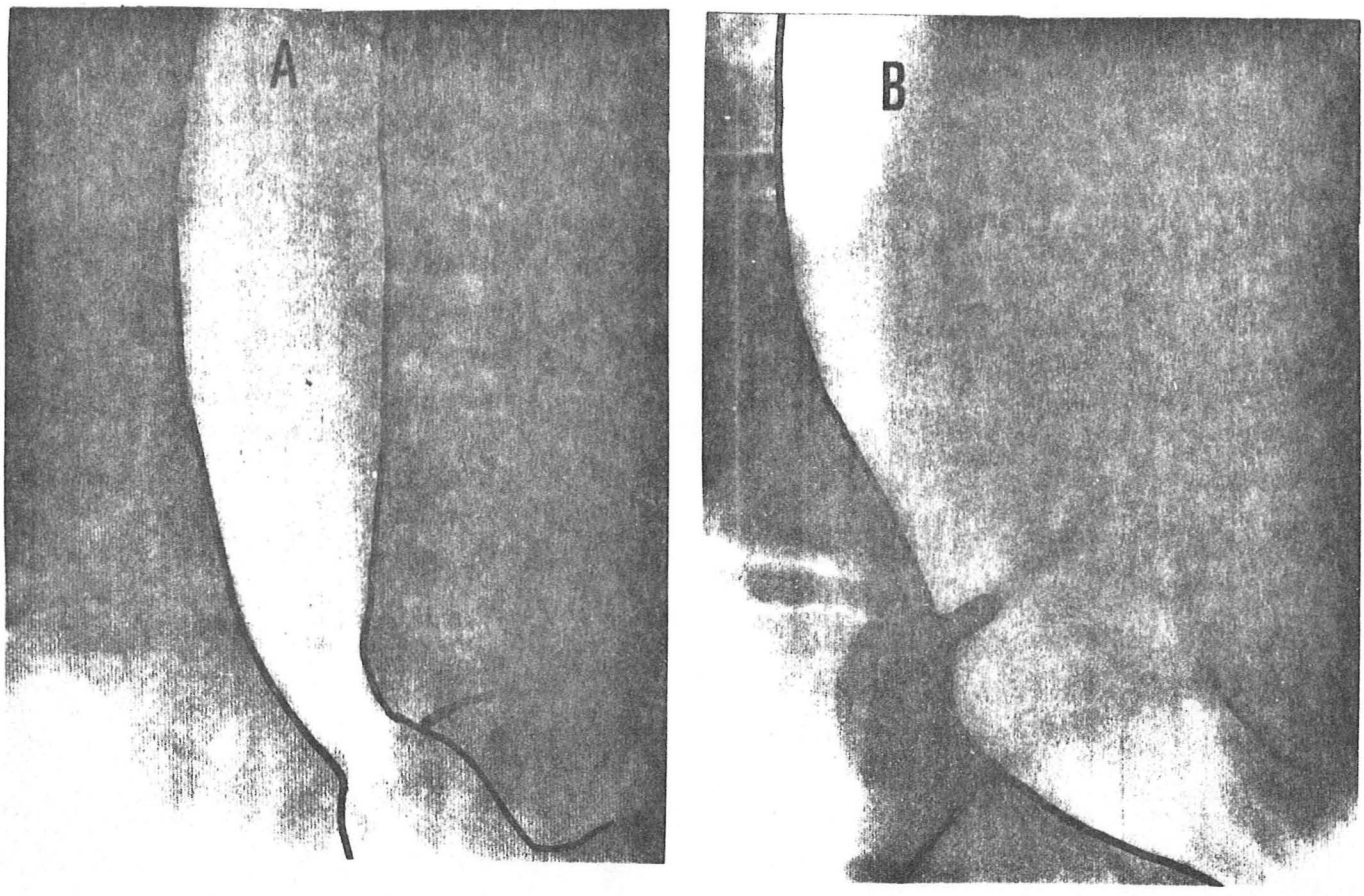
The patient was initially seen by an internist. A barium swallow was reported to be normal, and he was treated for "nerves". About 5 to 6 years after the initial symptoms, several episodes occurred at close intervals, and therefore the patient was endoscoped with a rigid scope. On endoscopy, the esophagus was found to be perfectly healthy without esophagitis or stricture. The patient was treated with anticholinergic agents with relief of his symptoms. The symptoms, however, recurred and then the patient was reinvestigated by a thoracic surgeon, who found that the patient had a small hiatus hernia and suggested hiatus hernia repair. The patient was now feeling well, and therefore refused surgery. In the subsequent years he visited many experts "all over the country".

The list of the diagnoses that he collected for his condition and the representative barium swallow are shown on the following page.

List of Diagnoses Made:

Psychogenic dysphagia
Hiatus hernia with mucosal plication
Esophageal web
Peptic stricture
Contractile lower esophageal ring
Lower esophageal ring
Steakhouse syndrome

Fig. 1. Representative barium swallow of the patient



By the time the patient came to New Haven, it was obvious that the patient had a ring-like defect in the lower esophagus causing his dysphagia. The main problem was to find what that ring was.

Recognition of Two Types of Rings

In March 1953, Ingelfinger and Kramer first described a clinical entity which ironically came to bear Schatzki's name. Ingelfinger and Kramer described 6 patients with intermittent dysphagia who were previously diagnosed as having cardiospasm, peptic stricture, diaphragmatic hernia or psychoneurosis. These diagnoses, however,"could not be substantiated, and in each instance dysphagia was thought to be due to a constriction-ring in the lower esophagus".

One of their patients had a typical history and x-ray appearance of this syndrome. The patient underwent surgery, and the lesion was described as a benign narrow stricture at the cardia. On microscopy, a localized thickening of muscle covered entirely by squamous epithelium was found. Ingelfinger and Kramer concluded that the constriction-ring was due to localized hypertrophy of the circular muscle. They called it a contractile ring and believed that episodic increase in contraction was responsible for episodic dysphagia in their patient.

In June 1953, Schatzki and Gary described 5 patients with episodic dysphagia, similar to those described by Ingelfinger and Kramer. They, however, believed that the ring was a passive fixed structure which does not vary in diameter or location in the same person.

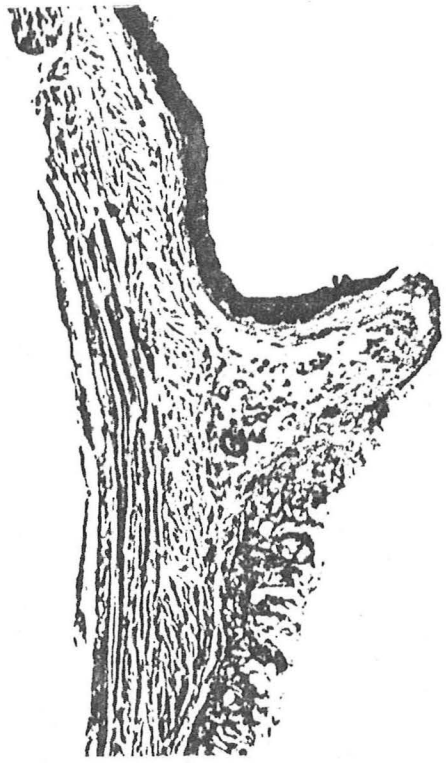
One of their patients who had a typical history and x-ray appearance (as shown) died of an unrelated cause. Careful examination at autopsy showed a thin annular mucosal fold constricting the lumen. In contrast to the findings in the patient of Ingelfinger and Kramer, this ring was found to be covered with squamous epithelium on the upper and columnar epithelium on the lower surface. The muscle layer did not contribute to the ring. Schatzki believed that the ring represented the junction between the stomach and esophagus, and that the radiographic demonstration of a ring above the diaphragm was evidence for the presence of hiatus hernia.

In the subsequent 15 or 20 years, around 100 papers were published on this entity, but the original disagreement regarding the location and nature of the lower esophageal ring remained unresolved. However, careful studies had shown that wide asymptomatic rings also occurred and that they were quite common, occurring in over 10% of the unselected patients undergoing UGI. Moreover, several radiologists suggested that these lesions were "pseudorings", being mere plications of redundant mucosa.

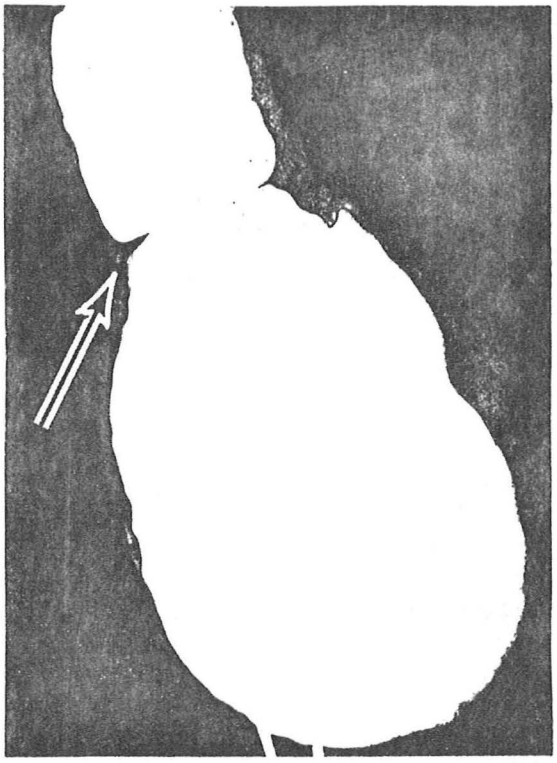
We felt that if these rings are actual anatomic defects, then their nature and location can only be defined by proper anatomic and pathologic studies. Moreover, if the rings are as common as 10% in unselected patients, we should be able to find them in unselected autopsies. Such a proposal was hampered by a reluctant pathologist, who said that he himself had suffered from episodes of dysphagia due to a lower esophageal ring for over 10 years and that he was doing careful autopsies for over 30 years and had never seen a ring. However, he agreed that he had not done a proper study by properly distending the gastroesophageal area, and that pathologists had not recognized lesions such as hypertrophic subaortic stenosis when they were not carefully and specifically looked for. Therefore, he agreed to cooperate in the study.

The lower half of the esophagus and upper part of the stomach along with a 1-cm to 2-cm rim of attached diaphragm was removed from the body as one piece. A ligature was tied around the circumference of the stomach about 10 to 15 cm distal to the gastroesophageal junction. A mixture of barium sulfate and formalin was then injected through the open end of the esophagus. A ligature was then tied around the esophagus. Radiographs of the gastroesophageal area were then obtained. The specimen was fixed by immersion in formalin. The specimen below shows a transverse annular constriction, not unlike the rings seen in the esophagus filled with barium in vivo.

The specimen was then opened, and details of the squamocolumnar junction and the presence of any circular transverse ridges were noted. Sections of the ridges were taken for histologic study, as shown below.



HISTOLOGICAL SECTION THROUGH A RING



BARIUM-FILLED SPECIMEN

Fig. 2

A mucosal ring is so called because it is a transverse fold of mucosa that encircles the entire circumference of the esophageal lumen. The core of the mucosal ring is formed by a variable amount of connective tissue, muscle fibers of muscularis mucosa and blood vessels. There is little contribution from the muscle wall, and there is no evidence of inflammatory

reaction (Fig. 2). The upper surface of the ring is covered by squamous epithelium, and the undersurface by the glandular epithelium. In six cases the mucosal junction lay at the apex of the ring, and in two, the squamous epithelium rolled over the apex of the ring to line a variable extent of its undersurface. In one specimen, the squamocolumnar junction extended onto the upper surface of the ring for a short distance.

In contrast, another kind of annular constriction was called a muscular ring because it is formed by a localized annular thickening. The submucosa and the muscularis mucosae remain normal, and their thickness is unchanged. There is no evidence of an active or healed inflammatory process. The muscular ring is covered with squamous epithelium on either side.

The radiologic features of the two types are well illustrated in one of the two specimens showing both muscular as well as mucosal rings. The muscular ring appeared as a wide constriction, whereas a mucosal ring appeared as a thin web-like constriction.

Moreover, note that when both rings are present, the muscular ring is present rostral to the mucosal ring.

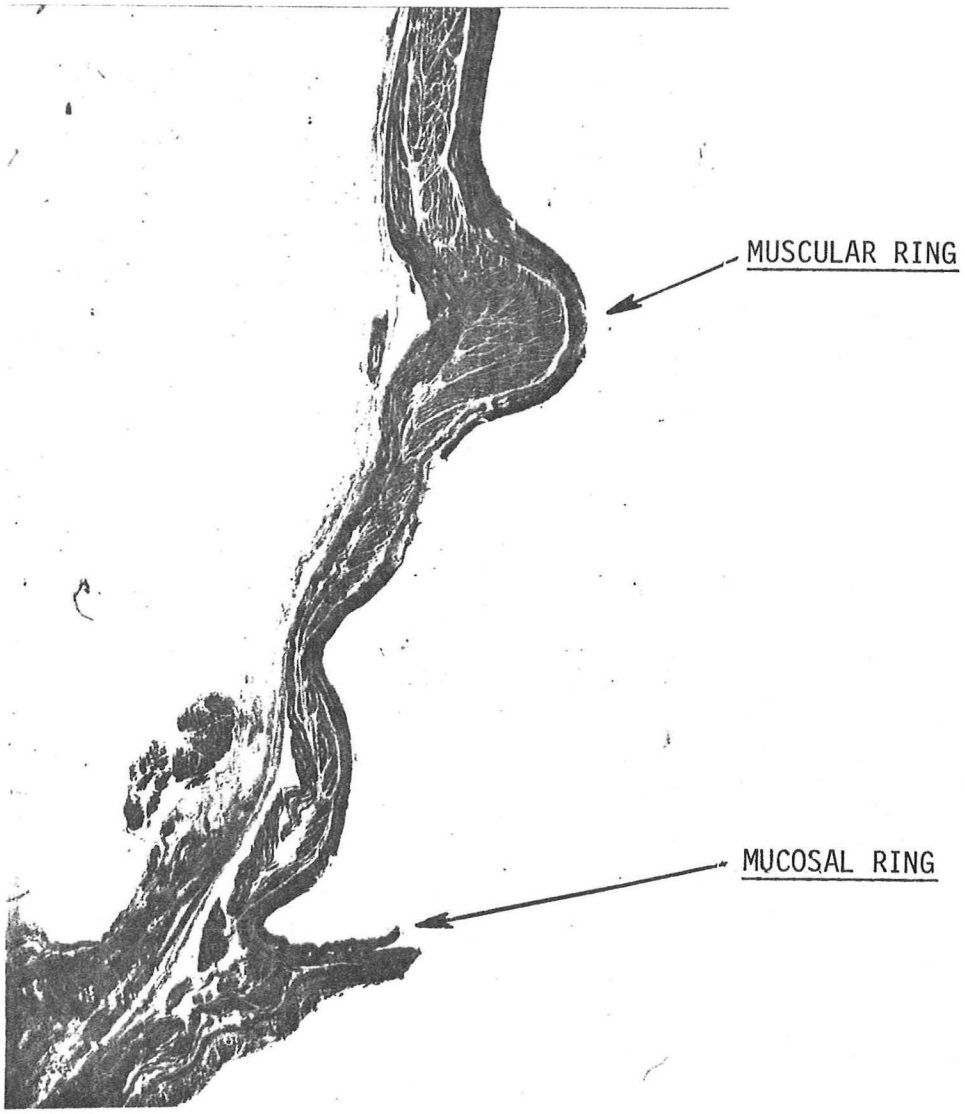


Fig. 3

The location of the rings was studied in relation to the squamocolumnar mucosal junction, insertion of the phrenoesophageal membrane and also the specialized fibers in the inner circular muscle layer. The mucosa was dissected away from the muscular wall along the submucosa and the relation of the ring to muscle thickness noted.

All mucosal rings were located at the squamocolumnar mucosal junction and their location corresponded to the location of constrictor cardiae and the lower insertion of the phrenoesophageal membrane. The muscular rings were all located proximal to the mucosal junction, and it appeared to be a prominent inferior esophageal sphincter described by Lerche and as suggested by Ingelfinger and Kramer.

These studies provided morphologic support to suggest that both Ingelfinger and Kramer as well as Schatzki and Gary were correct. The muscular ring corresponded to the ring described by Ingelfinger and Kramer, and mucosal ring corresponded with the ring described by Schatzki and Gary.

THE MUCOSAL RING (SCHATZKI RING)

Pathogenesis:

Three theories have been advanced for the pathogenesis of mucosal ring.

Plication theory. This concept holds the ring to be a mere pleat of mucosa. In this view, the esophagus shortens for some unknown cause and pulls the stomach into the thoracic cavity to produce a hiatal hernia. The theory suggests that the esophageal mucosa is loosely attached to the underlying tissues except at the squamoglandular junction, where it is strongly adherent, and so, as esophageal mucosa becomes redundant in relation to the shortened esophageal muscle, a ring is formed. For such a theory to be correct, the two surfaces of the ring should not be adherent to each other, correction of a hiatus hernia alone should eliminate the ring, and the ring should not be seen as a fixed structure at operation or at autopsy. Anatomic studies have shown that apart from firm circumferential attachment at the squamocolumnar junction, the esophageal mucosa is also firmly attached to the underlying muscles in the grooves between the longitudinal mucosal folds. The ring can be clearly seen and felt as a fixed, web-like lesion at operation both from above and from below.

Inflammatory theory. Barrett proposed that the ring was nothing more than a thin peptic stricture as a result of gastroesophageal reflux, whereas Templeton thought that it represented mucosal peptic esophagitis and ulcer with annular contractions similar to the hour-glass contraction in the stomach. The frequent if not invariable association with hiatus hernia likewise convinces many physicians of the causal role of acid peptic reflux in the genesis of the ring.

The principal objection to this theory lies in the absence of any history of preceding reflux and a notable lack of evidence of frank esophagitis and ulceration. Barrett argued that the ring, once formed, prevented further reflux, but this should be all the more reason for a prominent history of peptic esophagitis in the period before the detection of the ring, a story that is usually lacking. Furthermore, there is no correlation between the size of the ring and any gastroesophageal reflux, as there might have been if the ring had been implicated in peptic esophagitis. Some strictures at the lower end of the esophagus are the result of peptic esophagitis, but for important therapeutic purposes these should be differentiated from mucosal rings.

Developmental theory. The anatomical studies strongly support a developmental origin of the lower esophageal ring. The most telling argument against the congenital origin of the mucosal ring has been the fact that in most patients symptoms are first noted only after the age of 40. In some cases, nevertheless, a history of minor episodes of swallowing difficulty can be traced to childhood. Furthermore, it is not uncommon for many long-standing obstructing lesions of the upper gastrointestinal tract to come to attention first in the fourth through sixth decades of life. For example, partial pyloric antral diaphragm and duodenal diaphragm, usually accepted as a more obviously congenital lesion, may not be noted until middle life. An annular pancreas that has gone undetected in infancy likewise may give rise to symptoms only in the fourth and fifth decades. The late onset of symptoms from a mucosal ring may well be related to decrease in elasticity of the ring as a result of repeated trauma, or simply of the stiffening of old age. A congenital origin of the mucosal ring is supported by the relatively unchanging state of the ring once it has been detected; most rings change very little over a considerable period of observation. If the ring were concenital in origin, it would be difficult, however, to explain its frequent association with hiatus hernia; moreover, an occasional ring should be seen below the diaphragm. Of course, a ring is difficult, if not impossible, to demonstrate when it is present in the diaphragmatic hiatus or below it, since proper distention of the area is then not easy. Schatzki has told us of a woman in whom the ring could be seen above the diaphragm during a pregnancy, but the ring disappeared in the post-partum period, along with the hernia that accompanied it.

The squamocolumnar junction is usually considered to take a zigzag line, a course that would make it difficult to site a smooth circular ring precisely at the squamocolumnar junction. We have noted a considerable variation in the pattern of squamocolumnar junctions. Out of the 60 specimens, a zigzag line was present in 51, islands of gastric mucosa in one, and an almost straight squamocolumnar junction in the remaining eight specimens. Squamocolumnar mucosal rings seem to be found largely in patients with almost straight squamocolumnar junctions. Such data tend to support the theory that the ring is congenital in origin or, at least, that it requires a preformed developmental background for its genesis.

Prevalence:

Data on the prevalence of asymptomatic and symptomatic rings found on

radiologic studies are summarized below:

Asymptomatic Rings:

Poppel, 1950	-	.2% (in 500)
Schatzki and Gary	-	4.6% (in 368)
Kramer, 1956	-	6% (in 100)
Keyting, 1960	-	12.8% (in 1,131)
		14% (in 100, asymptomatic subjects)

Symptomatic Rings:

Schatzki and Gary	-	1 in 200 UGI
Keyting	-	1 in 500 UGI
PMH	-	1 in 184

Rings in Autopsy Specimen:

Zaino et al.	-	10% (in 80)
Goyal et al.	-	9% (in 100)

The mucosal ring may well be the commonest cause of dysphagia that comes to the practitioner's attention; its frequency at radiologic examination has varied, however, from 0.2 to 14 per cent as a result of differences in the technic, criteria and enthusiasm of the observer. Thus, in the 1950's, when the ring was first being recognized, Poppel et al. found one ring in 500 unselected radiologic examinations, whereas 4.6 per cent of 368 patients examined by Schatzki and Gary showed the lesion. Since that time, rings have been deleted with a higher and almost ever increasing frequency, from 6 to 14 per cent on routine barium-meal examination. It is probable, however, that in some of these statistics, rings other than mucosal rings were included. Many of the rings so detected cause no clinical trouble, and those producing symptoms are less common. Schatzki and Gary having found only 0.5 per cent of their patients undergoing study of the upper gastrointestinal tract to have a ring that caused dysphagia.

CLINICAL FEATURES

Age of onset of symptoms. The symptoms of obstruction first appear after the age of 40 years in most patients. Peak incidence occurs after the age of 50 years, and very few patients develop symptoms below 35 years. Rarely, a symptomatic mucosal ring may occur in infancy and childhood.

Sex distribution. Although in initial studies the ring appeared to be more frequent in men, subsequently both sexes are found to be equally affected.

Symptoms:

Dysphagia. Episodic dysphagia to solids is the hallmark of this syndrome The description of a classical episode of dysphagia by Ingelfinger and Kramer remains unsurpassed: "The dysphagia was described as a painful 'sticking' sensation quite sharply localized under the lower portion of the sternum. The attacks occurred only during eating, and the patients had no doubt that a portion of the offending food became 'stuck in the swallowing tube'. The intensity of the distress ranged from moderately severe to agonizing and was at times accompanied by considerable anxiety. Some patients wondered if they could continue to breathe or if they were about to suffer a heart attack.

The sensation of maximum distress and of complete esophageal occlusion lasted from a few minutes to several hours; then the patient felt as if the bolus was 'working through' the obstructed area leaving an aching pain which slowly subsided. During moderate attacks, the patients tended to drink water in an effort to force the retained food out of the esophagus into the stomach. During more severe episodes they were desperately afraid of swallowing anything until they sensed that some release of the obstruction was taking place. Following subsidence of the attack, some patients felt comfortable."

Some patients have less impressive symptoms. A patient may note merely that food sticks when he attempts to swallow a particularly large mouthful, and he may overcome this by taking a drink of water without realizing for some time that anything is really wrong. Many patients who show rings on barium swallow have no dysphagia at all.

Schatzki's careful study showed that the symptom of dysphagia was related to the diameter of the ring. Rings smaller than 13 mm were always symptomatic, whereas only a proportion of patients with ring diameters between 13 and 22 mm were symptomatic. Rings with size over 25 mm were not symptomatic.

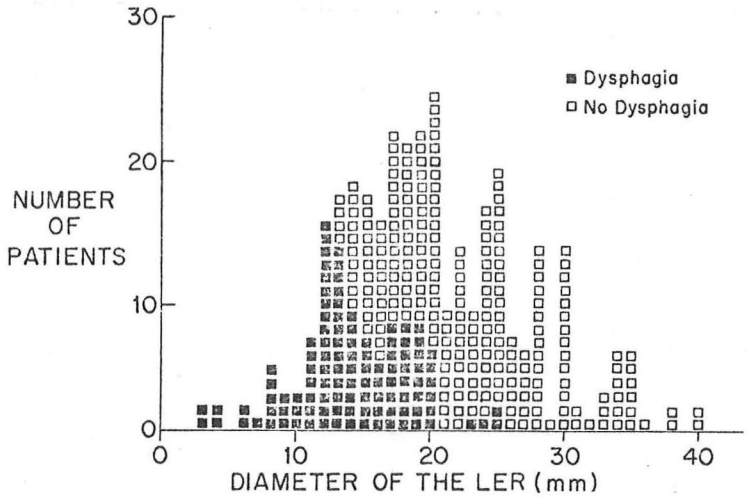


Fig. 4

The duration of dysphagia at the time of presentation is usually long, around 2-3 years, but may vary from the same day to many years (15-20 years).

The presentation of the course of dysphagia is also variable, as shown below:

Presentation of Dysphagia

Episodic (classical syndrome)	=	25 (63%)
Progressive	=	10 (25%
Acute obstruction	=	5 (12%)

(Sealy and Young, 1964)

Many patients who present with progressive dysphagia or acute food impaction give a previous history of episodic dysphagia.

The episodes of dysphagia, in most patients, are precipitated only if the patients eat certain types of solid food - namely, bread, meat, particularly roast beef and steak - particularly when these foods were swallowed hastily, without chewing properly...for example, during a business or otherwise important conversation, after a few drinks and an aching tooth. A steakhouse provides an ideal situation for such circumstances. Therefore, it is no wonder that many such episodes occur in steakhouses, and this syndrome has been called the "steakhouse syndrome".

Obstruction of the esophagus with the development of acute aphagia, a bolus of food securely lodged above the ring, is the commonest complication that causes the patient to present in the emergency room.

In rare instances, neglected impaction or very vigorous attempts at swallowing may lead to esophageal rupture.

Weight loss. Progressive weight loss is unusual because the symptoms are intermittent, and the patient can maintain calorie intake by soft liquid food.

Chest pain. Chest pain is uncommon and usually occurs only when the esophagus is completely obstructed by a bolus. Then, it is retrosternal, in the lower part of the chest, and radiates into the neck or arm.

Heartburn and reflux. Gastroesophageal reflux is considered to be infrequent in patients with mucosal ring. Barrett suggested that the ring acted as a barrier against reflux. On the other hand, Postlethwait and Sealy pointed out that the symptoms of reflux were not uncommon. In 33 of 91 cases collected from four series, the patients had symptoms of reflux. In our experience, troublesome reflux symptoms are not common in patients with mucosal ring. We believe that prolonged symptoms of reflux before the onset of dysphagia should arouse suspicion of annular stricture.

DIAGNOSIS

The diagnosis of a lower esophageal ring is easy when it is considered, but it is too often missed, usually because the presence of a lower esophageal ring is not thought of. In the past, such patients traveled from physician to physician seeking relief, carrying esophagrams and reports of negative esophagoscopic examinations with them. The episodic nature of the dysphagia, together with the apparent good state of health and the well preserved weight, leads the unwary physician to make a diagnosis of neurosis or psychogenic dysphagia. Ramsey cites a patient who felt so harrassed by the diagnosis of neurotic dysphagia as to commit suicide, because he had severe dysphagia and lived on barley water and baby food for eight years while no cause for his trouble could be found.

Barium swallow. Radiologic examination is the definitive diagnostic investigation, but unless extraordinary care is taken with the examination, many rings will be overlooked. The radiographic features of the ring and the technique described in the original paper by Schatzki and Gary remain unparalleled. The success of the examination depends upon maximum distention of the lower esophagus, and the plethora of maneuvers that have been considered are all designed to produce this effect.

Unless the ring provides a higher degree of stenosis, it will not be seen on an examination made with the patient erect. The examination is carried out with the patient recumbent and can be performed satisfactorily with the patient either prone or supine. Every effort must be made to obtain a very large bolus of the barium mixture. The patient is encouraged to fill his mouth completely before swallowing. The examiner then requests as deep an inspiratory effort as possible. This has the effect of closing the esophageal hiatus in the diaphragm. Furthermore, the cupola of the diaphragm is retracted, and the ring is more easily visualized against the lung field. It is possible to assist in the retention of the barium in the esophagus in several ways. The patient may be examined in a prone oblique position with a bolster under the abdomen to raise the intra-abdominal pressure, or supine with the right side elevated from the table - a position in which the examiner can provide manual compression on the upper abdomen. The Trendelenburg position and the Valsalva maneuver have also been used.

The mucosal ring, a smooth, symmetrical, web-like defect no more than 2 to 4 mm in thickness, is seen each time the lumen of the esophagus is distended beyond the size of the ring. The ring diameter remains constant on repeated examination, and sometimes the ring is seen to funnel a little. For the diagnosis of a simple mucosal ring, the remainder of the esophagus must give no evidence of ulceration, fibrosis or rigidity. These rings are usually described as located 4.0 to 5.5 cm above the diaphragmatic shadow. However, this distance is not relevant, for the diaphragmatic shadow changes its relation to the esophageal hiatus with the posture of the patient as well as with the phase of respiration.

The esophageal hiatus in the diaphragm is difficult to identify radiographically. The ring, however, is almost always seen above the pinched-off segment of barium. The ring moves in relation to the vertebral body with respiration. The ring also appears to move upward away from the diaphragmatic shadow as the area below the ring distends with barium.

Role of swallow with solid food or marshmallows. In their original paper, Ingelfinger and Kramer noted that in 3 of their 6 patients, the lesion was not identified by routine barium swallow. In these 3 patients when, in addition to barium, a small amount of food (incriminated in development of symptoms) was given, the true lesion was detected, and the patient experienced the usual symptoms. They thought that the lesion became more prominent after a solid bolus. Some have suggested barium-containing capsules, tablets or barium-soaked marshmallows as being helpful in the diagnosis of rings and other obstructive lesions of the esophagus. Schatzki and Gary, however, were able to demonstrate rings in all their patients with careful study with liquid barium. It appears that a key factor in detecting the obstructing lesions in the esophagus is proper distention of the esophagus

Endoscopy. In the past, mucosal rings were not ordinarily seen through the esophagoscope for two reasons: the usual scope of 9 to 12 mm may slip by even a stenotic ring; and the lower esophagus cannot be dilated with air insufflation. In some patients the examination was considered negative even though some difficulty was experienced in passing the area interpreted as cardia. With increasing awareness, a ring has been identified endoscopically in an increasing number of patients, partly also because of the availability of the wider fiber esophagoscope, through which the lower esophagus can be inflated. Apart from identifying the ring, the esophagoscopic examination is also valuable in excluding esophagitis, other abnormalities and sometimes also in treatment of these rings.

Esophageal manometry. Esophageal manometry studies are not useful in diagnosis. The ring is an inactive diaphragm of tissue, and its presence therefore cannot be recognized at manometry. The lower high-pressure-zone pressures in these patients have been reported to be low or almost normal.

CLINICAL COURSE

Schatzki followed 66 patients with rings. His findings at the end of 5 years are summarized on the next page.

TABLE II

Followup of Rings (5 Years)

	No Change	Deteriorated	Improved
Diameter			
Symptomatic (30)	19	11	0
Asymptomatic (36)	27	8	1
All (66)	46	19	1
Symptoms			
Symptomatic (30)	12	4	14
Asymptomatic (36)	32	4	--

(Schatzki, 1963)

MANAGEMENT

Acute obstruction

Acute obstruction is a medical emergency. Acute esophageal obstruction usually stimulates profuse salivation. Moreover, the patient usually tries drinks to wash down the impacted bolus. This fluid may be aspirated in the tracheobronchial tree causing spells of choking, coughing and cyanosis. Therefore, it is best he insert a Levin tube nasally to the obstruction for continuous suction of liquid esophageal contents. This helps in prophylaxis against aspiration, preparation for roentgenographic study and definitive treatment. Chest x-ray should be obtained to look for presence of bone in the impacted meat and to look for any evidence of mediastinal air suggesting esophageal rupture.

There are two ways to get rid of impaction. In vivo dissolution can be done with papain solution. Papain should not be used if x-ray shows evidence of bone along with meal and/or esophageal rupture. After the esophagus has been emptied with a nasogastric tube, 20 ml solution of 5 tablets of papase in water is instilled in the esophagus, and the patient is kept upright. This should be followed at 20- to 30-min intervals by repeated instillation of papain solution. This is frequently followed by passage of the bolus into the stomach after 2-4 exchanges of papain. If papain is not available, meat tenderizer may be used instead. Generally, if the bolus does not move after 6 exchanges of papain (3 hours), the effort is best abandoned, and plans should be made for transesophagoscopic removal.

Healthy mucosa is not digested by papain, and if the patient is known

to have a lower esophageal ring, this treatment can be used. However, if the esophageal mucosa is ischemic and damaged due to carcinoma, it can be digested by papain, and that is bad.

Treatment of the Ring

Explanation and reassurance. Symptoms occur when a bolus lodges at the ring so that the primary problem is a purely mechanical one. For this reason, symptoms can almost always be avoided if the patient avoids coarse food and carefully chews good food. Indeed, in some patients that may be all that is necessary. Drug treatment with anticholinergics, muscle relaxants or tranquilizers has not been of help as could be anticipated.

Transesophageal Rupture of the Ring

The ring may be stretched easily by transesophageal dilatation or by division through an esophagoscope.

Endoscopic rupture. In 1956, Delmonico reported successful treatment of a symptomatic ring by forcible rupture with esophagoscope. Two patients were relieved when the edge of the ring was punched out with an esophageal forceps. Friedman identified and successfully treated a ring by rupturing it under direct vision, using an Olympus esophagoscope. Since then many reports of successful treatment by this technique have appeared.

Rupture with Hurst bougies. This relatively simple procedure has achieved an undeservedly bad reputation as ineffective in the past, largely, we believe, because the bougies employed have not been wide enough to stretch the mucosal ring adequately. For example, Wilkins and Bartlett reported failure of bougienage in one of their patients, but since they used only a No. 26 bougie, with a diameter of 9 mm, no relief would have been anticipated. In contrast, Carter successfully treated four patients with a No. 48 bougie, corresponding to a diameter of 16 mm and one therefore calculated to stretch a tight ring. We suggest that at least a No. 48 bougie be employed whenever dilatation of a mucosal ring is attempted.

Our own technic differs somewhat from that used in most other clinical situations requiring esophageal bougienage in which it is customary to dilate the esophagus slowly and gradually. In the patient with a lower esophageal ring we ordinarily pass a small bougie of No. 16 to No. 18 caliber first to accustom the patient to the procedure; thereafter, we routinely use a No. 40 to 48 without any intervening sizes. The rapid passage of a large bougie presumably stretches or ruptures and dilates the lower esophageal ring forcibly and suddenly.

If symptoms are not relieved after dilatation, however, it is important

to ascertain fluoroscopically that the dilator has actually passed through the ring.

Dilation with a Mosher bag. Even more effective dilatation may be accomplished by means of inflatable bags such as those employed in achalasia. Adams successfully treated two such patients, and Mossberg stretched a ring 17 to 33 mm in diameter with a Mosher pneumatic dilator. Several patients with rings have been successfully treated with Mosher bags. Disadvantages of the Mosher bag treatment include the requirement of fluoroscopic control, the generally lesser experience with this procedure and the fear that an occasional esophageal perforation may occur.

Response to dilation. If the ring is adequately stretched or otherwise ruptured, bougienage should not really be required more than once, Postlethwait and Sealy treated 11 patients with Hurst bougies up to size 44 Fr in three treatments over a week. They followed their patients for a period of 4 months to 5 years and reported good results in all, except two who suffered recurrent dysphagia after relief lasting two and three years. Recurrence of symptoms months or years after adequate dilation presumably results from restenosis.

Most clinicians would agree that the radiologic evidence of a ring almost never disappears even if the patient is rendered asymptomatic. This is not surprising in view of the fact that a ring size up to 40 mm can be recognized radiologically, and it is unlikely that anyone ever dilates the esophagus up to that degree (corresponding to 120 Fr size). Moreover, the final ring diameter is less than that of the bougie employed because of the elastic properties of the ring.

Unfortunately, there are few objective data on the outcome of dilatation of the ring. If the ring is adequately stretched or otherwise ruptured, relief should be long lasting. Recurrence of symptoms, however, is common, and repeated dilatations may prove necessary. But the usefulness of repeated dilatation is difficult to evaluate because of size of the bougies employed, lack of fluoroscopic control and careful assessment of ring size after treatment by radiologic means. In few well studied cases, the ring size increases appreciably after dilatation, and then, with time, the diameter decreases, indicating restenosis. No data on the frequency of restenosis after dilatation are available.

One complication that may follow stretching of the ring is heartburn as a result of gastroesophageal reflux and peptic esophagitis, but the frequency and degree of reflux have not been documented by objective evidence.

Operative therapy. Several different operative procedures have been carried out, but they are not necessary.

Various operative procedures performed are listed below:

1) Hiatal hernia repair alone
2) Hiatal hernia repair with digital rupture
3) Hiatal hernia repair with partial excision
4) Hiatal hernia repair with circumferential excision
5) High vagotomy
6) Resection with reanastomosis

It is really amazing that the patients have been reported to have done well ("and living happily ever after") after all the above types of operations,including high vagotomy.

The claims should be accepted with some reservation. For example, in one patient hiatus hernia repair alone was reported to completely abolish the ring, and the x-rays were (fortunately) published. Careful examination revealed that the postoperative barium swallow did not distend the lower end of the esophagus enough to demonstrate a ring, even if it was present.

Now let us turn to the consideration of the muscular ring and annular peptic stricturs, because these two lesions are frequently confused with the mucosal ring. In this consideration, only the differentiating features of these entities which distinguish them from a mucosal ring will be considered.

MUSCULAR OR CONTRACTILE RING

Pathogenesis and pathology. The muscular ring is characterized by an annular area of muscle thickening. This thickening or prominence does not occur just anywhere in the esophagus, as thought by Schatzki, but it regularly occurs in the region corresponding to the inferior esophageal sphincter (described by Lerche), as first suggested by Ingelfinger and Kramer. A muscular ring is best considered as a prominent, unrelaxed,inferior esophageal sphincter and marks the upper part of the lower esophageal sphincter. (The lower esophageal sphincter area comprised the region of the inferior esophageal sphincter and the ampulla or the vestibule distal to it.)

The pathogenesis of muscular prominence is not known. It may represent actual muscle hypertrophy, but in many instances it simply represents abnormal relaxation, and the observed muscle hypertrophy may be more apparent than real. The relationship of this ring to achalasia, if any, is not known, but a prominent and persistent muscular ring is frequently associated with diffuse esophageal spasm. It may also occur in association with scleroderma and hiatus hernia, and then it can be confused with a peptic stricture.

Prevalence. Frequency of muscular rings is not known; they appear to be less frequent than the mucosal rings.

Clinical features. Schatzki and Gary thought that a muscular contraction is never symptomatic, even though Ingelfinger and Kramer had already well documented a symptomatic patient with a muscular ring. The clinical features of a muscular ring are quite indistinguishable from those of a mucosal ring. However, chest pain occurring even without the episode of dysphagia is suggestive of a muscular ring as it indicates the associated diffuse esophageal spasm, but sometimes the chest pain is simply due to hyperactive lower esophageal sphincter.

Barium swallow. The hallmark of a muscular ring is the change in its size and shape from moment to moment. Therefore, it may be impossible to distinguish it from a mucosal ring from a single film. However, the muscular ring is usually wider, its width varying from 3 mm to 3 cm.

Motility studies. Many of these patients have very high basal sphincter pressure. The sphincter relaxes normally, or near normally, but the relaxation is followed by a high amplitude and long-lasting after contraction. The syndromes of "hypertensive lower esophageal sphincter" or "hypercontracting lower esophageal sphincter" may represent patients with muscular rings.

Treatment. Muscle relaxants such as nitrates and sublingual nitroglycerine may be helpful in some patients. Some patients improve with dilation with Hurst bougies, while others were relieved after an extramucosal myotomy.

RING-LIKE PEPTIC STRICTURE (ANNULAR STRICTURE)

Pathology and pathogenesis. True peptic strictures vary in width; usually they are 1-4 cm wide, but others are long, extending over several centimeters in the esophageal wall on one hand, and thin and web-like on the other.

The annular peptic strictures almost always form at or near the squamocolumnar mucosal junction. These strictures may follow a peptic ulcer of the esophagus and may be likened to an hourglass contraction. These patients always show radiographic and endoscopic evidences of esophagitis, ulceration and rigidity in infiltration in the esophageal wall. Histologically, mucosal ulceration, dense connective tissue due to scarring and infiltration with cells are seen.

Prevalence. The frequency of true annular strictures simulating a

lower esophageal ring is not known, but they are less frequent than the mucosal rings. Paulson, from Baylor, reported on 30 annular strictures. Larrain found that 7 of their 21 patients with peptic strictures had a width of 1 cm or less, and among them 3 had a width of 5 mm or less. (Other patients had a width of 11-20 mm = 8, 21-30 mm = 4 and 31-39 mm = 2 patients.)

Clinical features. These features are similar to those with mucosal rings. However, heartburn and reflux may be more prominent accompaniments.

Barium swallow. This may show evidence of associated ulceration and the rigidity and lack of normal distensibility of the esophageal segment above and below the ring.

Esophagoscopy. Esophagoscopy shows esophagitis and ulceration. In a patient with a true mucosal ring with associated reflux esophagitis, the differentiation between the mucosal ring and annular stricture may be impossible.

Manometric studies. These studies generally show very low sphincter pressure.

Management. The management of annular strictures is difficult and generally less rewarding than that of the mucosal rings. These lesions show considerable resistance to dilation and may require initial dilation with metal olives. Dilations should be done gradually and persistently. Mosher bag dilation is dangerous and should never be attempted in these patients. A rigorous antireflux therapy must accompany dilations. Some of these patients have been successfully treated with an antireflux operation with or without instrumental dilation of the stricture.

The comment regarding the dilation of esophageal strictures that, "all these patients die sooner or later - killed by a bougie", is not really applicable to our current techniques of dilation.

OTHER LESIONS CAUSING RING-LIKE DEFECT IN THE LOWER ESOPHAGUS (TERMINAL 2-4 cm SEGMENT)

Indentation caused by sling fibers. This is not circumferential and is asymmetrical present only on the left side. This should not be confused with the lower esophageal ring.

Circumferential leiomyoma or neuroma. In one of the cases described by Schatzki and Gary, the lower esophageal ring was due to a chain of small leiomyomas arising from the inner circular muscle layer. A circumferential leiomyoma, particularly when associated with hiatus hernia, may simulate a ring. At operation such a leiomyoma may be confused with a muscular ring, but leiomyoma can be distinguished from the surrounding muscle tissue with ease. A neuroma may also produce a lesion resembling the lower esophageal ring.

Cartilaginous rings. Several cases of cartilaginous rings due to abnormal persistence of rests in the lower esophagus have been described. Most of these cases occur in children, but some are first recognized in adulthood.

Vascular rings. Warden described a vascular ring in a 16-month-old child that produced a short area of narrowing of the lumen about 2 cm above the diaphragm on x-ray examination. Esophagoscopy revealed a constriction at the site, and biopsy was reported as showing chronic esophagitis. At operation, however, an aberrant right eighth intercostal artery passed anterior to the esophagus about 2 cm above the diaphragm. The artery was ligated and divided, resulting in complete radiographic and and symptomatic cure. This case emphasizes that particularly in children the cause of lower esophageal ring should be carefully evaluated by proceeding with dilation.

Annular carcinoma. An annular carcimona can simulate a muscular ring as the defect is wide. However, the fixed nature of the lesion should provide a differentiating point between the two.

RING LESIONS AND WEBS IN OTHER PARTS OF THE ESOPHAGUS

On clinical grounds alone, the lesions in the lower esophagus cannot be differentiated from the lesions in other parts of the esophagus with certainty. Although localization of the site of dysphagia by the patient is helpful, it is not always accurate.

For the purposes of description, lesions in the esophagus, except segments of 4 cm at either end, are designated as the mid-esophageal webs or rings. The upper esophageal webs or constrictons include those in the upper esophagus within 4 cm of the cricoid cartilage.

WEB-LIKE RING STRICTURES OF THE MIDDLE ESOPHAGUS (∿ 20 cm)

These webs are uncommon. Out of 58 cases of stenotic esophageal webs at all locations, Shamma and Benedict found two cases of mid-esophageal webs. Several types of webs have been recognized.

Congenital webs. These are covered over by squamous epithelium on both surfaces. They produce symptoms soon after birth or when solids are first introduced. These webs may be associated with lower esophageal rings.

Barrett's esophagus with squamocolumnar mucosal ring. These are very similar to Schatzki's ring, except they are present in the mid-esophagus near the aortic arch.

Peptic strictures. Annular peptic strictures with or without a peptic ulcer in the mid-esophagus almost always indicate the presence of columnar-lined lower esophagus (Barrett's esophagus).

Inflammatory stenosis. Certain skin diseases concomitantly involve the esophageal mucosa. For example, benign mucous membrane pemphigus may produce a web-like scar in the esophagus.

Carcinoma. Annular submucosal carcinoma is rare, but can occur. It produces a wide ring and does not produce a membranous stricture.

Clinical Features.

In infants, usually there are no symptoms until 5 to 11 months of age when the child is given solids to eat. Then the dysphagia is intermittent and usually the body weight is well maintained. Once established, the symptoms vary little. Some patients can avoid symptoms by chewing carefully or by consuming liquid diets. Out of the two patients reported by Shamma, one was a 72 y/o female who had episodic dysphagia to solids for six years.

The mid-esophageal webs may occur in association with mucosal rings. Dr. Eisenstein showed me a case of a 22 y/o man who had "intermittent dysphagia for all his life". He frequently had trouble swallowing a pill. He had learned to chew carefully, avoid hard foods and subsist on a soft liquid diet. He had lost no weight, a previous x-ray of the esophagus was reported to be normal and he was told that he had a "nervous esophagus". Studies at Presbyterian Hospital revealed mid and lower esophageal rings. The rings were identified endoscopically and sectioned, and the patient had prompt relief of symptoms and is doing well (now after 8 months). We are aware of two other reported patients who had mucosal rings in association with mid-

esophageal webs. One patient, a 39 y/o man, had solid food dysphagia for 8 years before a correct diagnosis was made (Shamma et al., 1958). Another patient, a 46 y/o man, had intermittent dysphagia for 20 years before a correct diagnosis of the rings was made.

Clinically, the history of patients with mid-esophabeal webs is similar to that of lower esophageal rings. The location of the ring cannot always be determined by the location of dysphagia as described by the patient, as many patients with lesions in the lower esophagus may refer to the upper chest or the suprasternal area as the site of dysphagia.

The squamocolumnar mucosal webs in the mid-esophagus do not present any distinctive clinical features. Heitman et al. (1967) reported the case of a 66 y/o male with painless dysphagia to solid food for 6 months. Studies revealed that the ring was lined by squamous epithelium above and columnar epithelium below. The esophagus distal to the ring was lined by columnar epithelium. Because of the special clinical importance of recognizing patients with Barrett's esophagus, it is important that all mid-esophageal webs have histological studies.

Wider annular strictures associated with Barrett's esophagus are more common. These should be recognized because, like the lower esophageal peptic stricture, they require more rigorous and careful technique of dilation.

Benign peptic strictures unassociated with Barrett's esophagus simply do not occur in the mid-esophagus. The presence of squamous epithelium distal to a mid-esophageal stricture should raise a question about annular carcinoma, along with other clinical entities.

UPPER ESOPHAGEAL WEBS AND RELATED LESIONS

Upper esophageal webs are one of the three causes of related appearances seen in the pharynx and upper esophagus listed below:

1) Post-cricoid impression
2) Cricopharyngeal indentation
3) Upper esophageal web

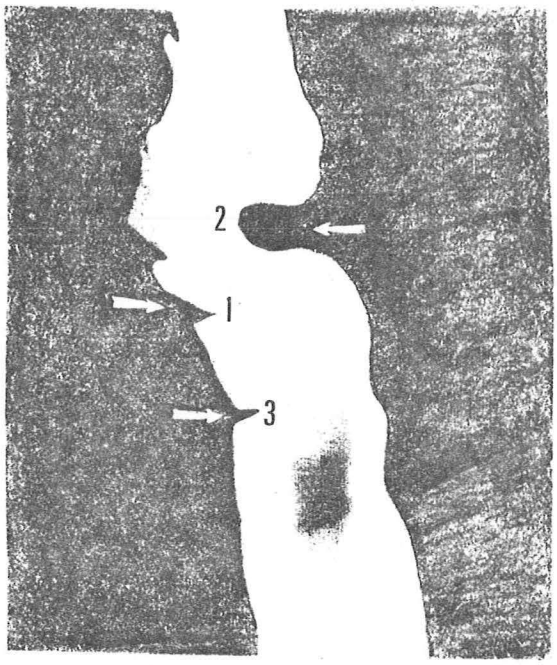


Fig. 5

The post-cricoid impression

This name is given to a wide spectrum of defects in the anterior portion of barium column immediately posterior or below the cricoid cartilage. These defects can take various forms as shown in Figure 6. Pitman and Frazer suggested that these defects are due to the laxity of the mucous membrane over

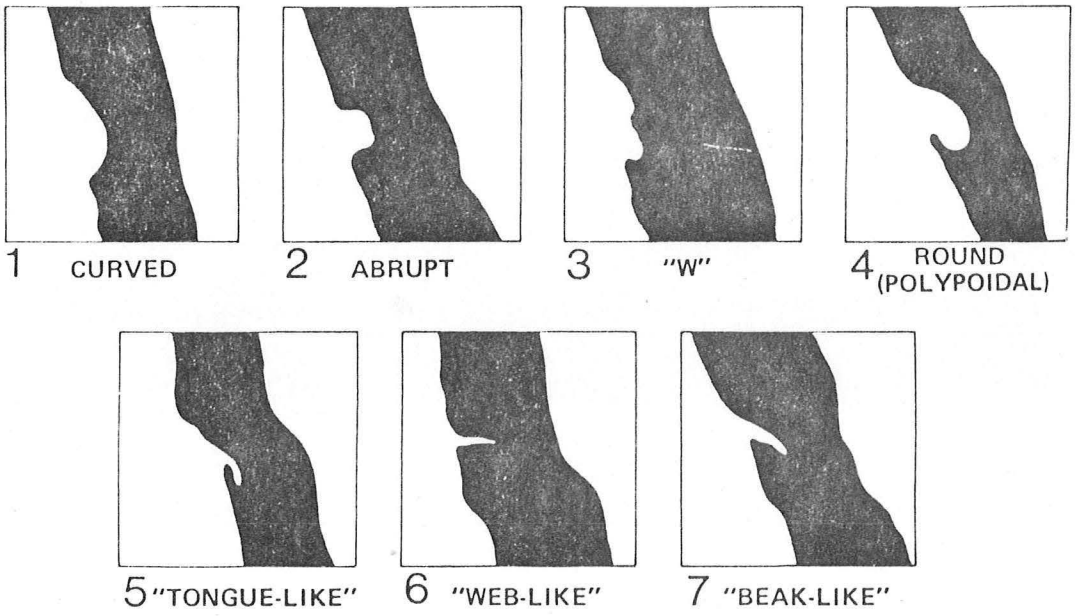


Fig. 6

the normally varicose submucosal veins in the post-cricoid region. They recognized post-cricoid indentation in 86% of patients with dyphasia. But 90% of the control subjects without dysphagia also had similar defects. Clements et al. also reached similar conclusions; they found such defects in 62% of patients with 74% of patients without dysphagia. It is therefore important to appreciate that they are normal variants.

Web-like post-cricoid impression should not be confused with upper esophageal webs and the mass-like defects should not be confused with carcinoma or other tumors. The hallmark of post-cricoid impression is its change during deglutition.

Cricopharyngeal Indentation

A posterior indentation of the esophageal lumen is often observed at a level corresponding with the location of the cricopharyngeus. It is called esophageal lip or spasm or hypertrophy. However, pathological and clinical significance of this indentation is controversial.

In the resting position, the cricopharyngeal muscle is in a stage of tonic contraction, assuring closure of the proximal esophagus and separation of the respiratory system from the digestive tract. On deglutition, after initiation of pharyngeal peristalsis, the cricopharyngeal muscle relaxes, permitting the advancing food bolus to enter the esophagus without resistance. Seaman has postulated that incoordination of pharyngoesophageal motility may suffice to explain this posterior esophageal defect and that it is not necessary to attribute the cricopharyngeal indentation to spasm or muscle hypertrophy. If the cricopharynx fails to open at the proper time or if it contracts prematurely, a defect in the barium column would be readily apparent.

It is noteworthy that most often patients with this defect show no evidence of dysphagia. However, if the defect is more pronounced and accompanied by difficulty in swallowing, this entity is usually referred to as cricopharyngeal achalasia.

TABLE IV

Prevalence of Cricopharyngeus Indentation

Author	No. of Cases	Indentation	With Symptoms
Siebert et al.	50	29 (58%) *7 (14%)	0
Seaman		(5%)	
Clements	100	(18%)	1

*Marked

Siebert et al. and Johnston, among others, believe that cricopharyngeus indentation is of no clinical importance. On the other hand, Crichlow as well as Seaman consider the cricopharyngeal indentation as a pathologic phenomenon reflecting neuromuscular dysfunction. The latter author believes that patients may adapt themselves to abnormal swallowing and therefore may not display manifestations of dysphagia.

"Cricopharyngeus achalasia" is thought to be an important cause of dysphagia in patients with neuromuscular disease of the pharynx and in postlaryngectomy patients. Moreover, cricopharyngeus myotomy is reported to afford relief of dysphagia in these patients. Some observers also believe that cricopharyngeus abnormality, other than causing dysphagia, is also responsible for the production of Zenker's diverticulum. Manometric studies usually do not demonstrate the suggested abnormalities in cricopharyngeus relaxation. In brief, the clinical and pathological importance of cricopharyngeus indentation is not properly understood.

Patients with globus sensation in the throat, interestingly, do not show radiographic evidences of prominent cricopharyngeus indentation, but manometric studies show that these patients have very high cricopharyngeal pressures.

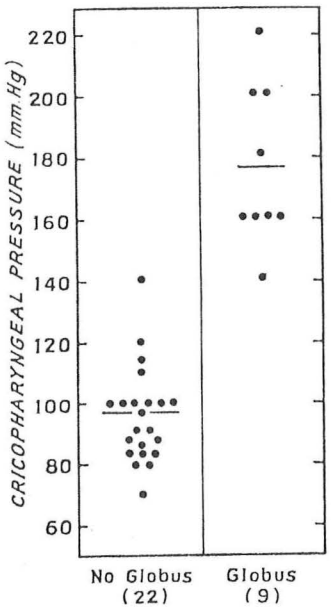


Fig. 7. Cricopharyngeal sphincter pressures in patients with globus sensation

Neither post-cricoid impressions nor cricopharyngeus indentation are part of Plummer-Vinson syndrome.

Cervical Esophageal and Hypopharyngeal Webs

These are thin, shelf-like defects measuring 1-2 mm in thickness. They protrude for various lengths into the anterior aspect of the esophageal lumen. They are usually semilunar, extending only a little on the side walls, so that they may not be visible on anterio-posterior projection. Rarely are they circumferential. They may be present in the hypopharynx or the cervical esophagus. Sometimes multiple webs occur.

Frequency of hypopharyngeal webs. The advent of cine fluoroscopic examination of the pharynx and esophagus has led to a more frequent diagnosis of upper esophageal webs. For example, at Columbia Presbyterian Hospital in New York only 9 webs were diagnosed in 10 years (1950-1960). In contrast, 55 webs were recognized in one year (1972-1973). More recent studies report a frequency 5-15%. In a study of 1,000 consecutive, unselected patients, Mosher et al. found one or more webs in 5.5% of the patients. Clements et al. reported a frequency of 8% (in 100 patients). A small number (5-6%) of these webs are truly annular rings and are therefore more likely to be directly responsible for dysphagia.

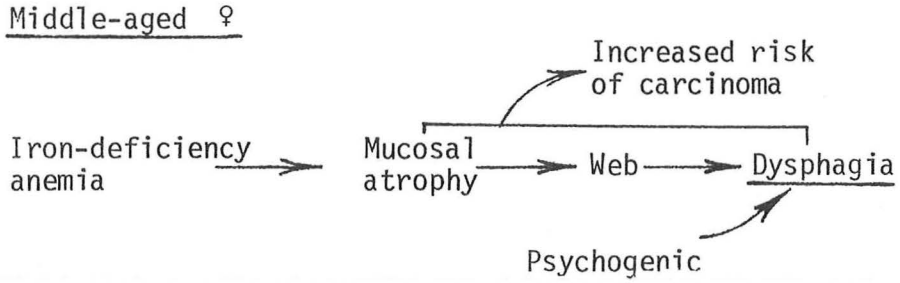
Observer error in radiological diagnosis of hypopharyngeal webs. Elwood et al. asked 8 expert radiologists (most of whom had published papers on hypopharyngeal webs) to participate in a study of observer error in radiological diagnoses of the webs.

Sets of copies of barium swallow films of 132 female patients with post-cricoid dysphagia were made. The number of subjects in whom a web was detected by different radiologists varied from 6 to 59 per cent. There was complete agreement on the presence of a web in only 3% of the patients. Moreover, although a web was considered to be stenotic in 14 subjects by at least one radiologist, all radiologists agreed on its presence in two patients, and in 10 cases only one examiner reported stenosis to be present. This study demonstrates the problem of a reliable diagnosis of webs.

PLUMMER-VINSON, PATERSON AND BROWN KELLY SYNDROME

In 1922, Vinson from Mayo Clinic described a syndrome of pharyngeal dysphagia, anemia and frequent splenomegaly in middle-aged women. Interestingly, (and unlike events during the discovery of the lower esophageal ring), Vinson pointed out that Plummer had recognized the syndrome, even though he (Plummer) never wrote the paper. The syndrome now bears the name of both of them. They considered dysphagia to be hysterical and not related to any organic lesion. A similar syndrome was recognized in England by Paterson and Kelly, and therefore in Europe this is popularly known as Paterson-Kelly syndrome. Over the years, dysphagia has come to be believed to be due to upper esophageal webs which are thought to be the result of iron deficiency. Recently this concept has come under scrutiny.

PATHOGENESIS OF THE SYNDROME OF PLUMMER-VINSON



Webs as a cause of dysphagia in P-V syndrome. In 1919, Brown Kelly reported that some midle-aged women with dysphagia had atrophic oropharyngeal mucosa, and they also had webs, bands and folds in the pharynx. They found that rupture of these bands caused relief of dysphagia.

Waldenstrom[11] and his associates were the first to demonstrate esophageal webs roentgenographically in patients with Plummer-Vinson syndrome and suggested that webs were the cause of dysphagia. These authors, however, did not present any studies in control subjects. Moreover, they appeared to have included webs as well as other post-cricoid impressions.

Recently some authors have considered presence of a web to be a prerequisite for the diagnosis of this syndrome. If so defined, 100% of patients with this syndrome would have webs. Such a definition, of course, leaves out a majority of patients with iron deficiency and dysphagia in middle-aged women - the original description of the syndrome.

Moreover, it is questionable if a web is the cause of dysphagia in these patients. Out of 108 patients who were found to have webs on cine studies done for all causes, 32 had dysphagia, while 76 had no dysphagia (Seaman, 1976). The size of the webs was similar in the two groups. In another study webs were found in 9% of the patients with dysphagia of undetermined cause, and 8% of the patients without dysphagia.

Prevalence of webs in patients with chronic iron-deficiency anemia. Chisholm et al. observed a strong association between iron-deficiency anemia and webs. They found a frequency of 10% in these patients and 0% in 98 healthy subjects. They also suggested that those patients with iron deficiency who had mucosal changes and thyroid disease were more likely to have webs. Seaman compared the incidence of iron-deficiency anemia in 51 patients with webs with 405 controls and found no difference. Clements et al. in a recent study found an 8% incidence of webs in patients with iron-deficiency anemia as well as in controls.

In summary, it appears that there is no increased association between iron deficiency and the pharyngeal or upper esophageal webs, and it appears unlikely that iron deficiency leads to web formation.

Iron-deficiency anemia and dysphagia. Anemia was considered as a part of this syndrome, but its importance was emphasized by Scandanavian workers who considered iron-deficiency anemia as the cause of dysphagia and called this condition "sideropenic dysphagia".

Wynder and Fryer, however, found normal serum iron levels in 62% of 109 patients. Some patients have been described in whom treatment of iron-deficiency anemia cured dysphagia. However, in others symptoms recurred despite iron therapy.

Elwood et al. found 108 women and 21 men who had unequivocal pharyngeal dysphagia. These patients did not have increased prevalence of iron deficiency.

P-V syndrome and hypopharyngeal carcinoma. Paterson was aware of the frequent association of this syndrome with carcinoma of the hypopharynx and upper esophagus. This association was emphasized by Ahlbom.

Wynder et al. compared the frequency of Plummer-Vinson type of dysphagia in patients with hypopharyngeal carcinoma. They found that the Plummer-Vinson type of dysphagia was present in 70% of patients in Sweden with pharyngeal carcinoma, whereas not a single patient with pharyngeal carcinoma from Memorial Hospital in New York gave a prior history suggestive of P-V syndrome.

These studies show that Plummer-Vinson syndrome may indeed exist in certain parts of the world and not in the others. In its fully developed form this syndrome is rare, if not non-existent in the United States. Moreover, the real common denominator in patients of this syndrome may be "chronic unexplained dysphagia in middle aged women".

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